DIAGNOSIS OF PRIMARY AND CONCOMITANT FLEXOR ENTHESOPATHY IN THE CANINE ELBOW

Evelien de Bakker

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Promotor:
Prof. Dr. B. Van Ryssen

Co-promotors:
Dr. I. Gielen
Prof. Dr. J.H. Saunders

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Department of Medical Imaging and Small Animal Orthopaedics
Faculty of Veterinary Medicine
Ghent University
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Evelien de Bakker
Department of Medical Imaging and Small Animal Orthopaedics
Faculty of Veterinary Medicine
Ghent University

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“Om te kunnen schrijven, moet je op dat moment denken dat je geweldig goed bezig bent – zoals je om een kuil te graven met elke schep moet kunnen vinden dat de kuil dieper wordt. Zoniet leg je beter je schep weg en ga je wat anders doen”.

Susan Smit, uit de column “Voltooid”.
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Dankwoord

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LIST OF ABBREVIATIONS

cm centimeter
CT Computed Tomography
DICOM Digital Imaging and Communications in Medicine
3D 3-Dimensional
dors dorsal
FCP Fragmented Coronoid Process
FE Flexor Enthesopathy
g gram
HDP $^{99}$mTc-disodium oxonate
HiSPECT High resolution Single Photon Emission Computed Tomography
IEWG International Elbow Working Group
IOHC Incomplete Ossification of the Humeral Condyle
IV IntraVenous
kV kiloVoltage
mA milliAmpère
MBq MegaBecquerel
MCD Medial Coronoid Disease
MHz MegaHertz
ml/kg milliliters per kilogram
mm millimeter
mmol millimol
MRI Magnetic Resonance Imaging
ms milliseconds
NMR Nuclear Magnetic Resonancy
OCD OsteoChondritis Dissecans
PACS Picture Archiving and Communication System
sag sagittal
STIR Short inversion – Time Inversion Recovery
T Tesla
TE Echo Time
TR Repetition Time
trs transverse
T1W SE T1-Weighted Spin Echo sequences
T2W SE T2-Weighted Spin Echo sequences
UAP Ununited Anconeal Process
μ-SPECT micro Single Photon Emission Computed Tomography
PREFACE

The elbow joint is a frequent localization for thoracic limb lameness in medium-sized and large breed dogs. The most important canine elbow disorder is elbow dysplasia, which includes medial coronoid disease, osteochondritis dissecans, ununited anconeal process and incongruity. Diagnosis is based on the primary radiographic changes or secondary subtrochlear sclerosis and osteoarthritis, often combined with the findings of computed tomography or arthroscopy. An unrecognized cause of elbow lameness is represented by changes of the medial humeral epicondyle and the attaching flexor muscles. In the past these medial humeral epicondylar changes were described as 'ununited medial epicondyle' but a more appropriate term is flexor enthesopathy, including bony lesions as well as soft tissue lesions. It can be visualized radiographically by the presence of a calcification or fragmentation in the area of the flexor muscles and their attachment to the medial humeral epicondyle or by the presence of a spur at the caudal edge of the medial humeral epicondyle. Little attention was given to this problem as only limited clinical consequences were seen. However, in our experience, lameness caused by these medial humeral epicondylar lesions occurs on a regular base. When no other elbow disorders are diagnosed, the term primary flexor enthesopathy is used. The challenging type of primary flexor enthesopathy is characterized by the absence of clear radiographic changes. In these cases the diagnosis of flexor enthesopathy may be missed and the distinction from discrete medial coronoid process lesions may be difficult.

On the other hand, flexor pathology is often seen in the presence of elbow dysplasia, mostly in chronic cases of medial coronoid disease. In those cases elbow dysplasia is considered as the primary problem and treatment is aimed at fragment removal, since it is not known whether the concomitant flexor enthesopathy has any clinical consequences. Knowing that radiography is not always able to demonstrate the medial coronoid process lesions, the distinction between primary flexor enthesopathy and elbow dysplasia with concomitant flexor enthesopathy requires additional diagnostic procedures.
SECTION I

GENERAL INTRODUCTION
Part I

MEDIAL HUMERAL EPICONDYLAR LESIONS IN THE CANINE ELBOW: A REVIEW OF THE LITERATURE
MEDIAL HUMERAL EPICONDYLAR LESIONS IN THE CANINE ELBOW: A REVIEW OF THE LITERATURE

Summary

Radiographic changes at the medial humeral epicondyle were originally reported as ununited medial epicondyle by Ljunggren et al in 1966, characterized by the presence of loose ossified bodies either on the medial side of the elbow joint or distal to the medial humeral epicondyle. Since then several clinical papers reported similar lesions, but used different terms: dystrophic calcification of the flexor tendon origins, traumatic avulsion of the medial humeral epicondyle, medial humeral condylar osteochondritis dissecans and development of a preformed ossification centre. Bony spur formation at the caudal edge of the medial humeral epicondyle was described as another radiographic finding, although less frequently compared to calcification near the medial humeral epicondyle. Since the pathological changes in dogs seem to have similarities to certain enthesopathies in man, the term ‘flexor enthesopathy’ was recently suggested to describe the disorder in dogs. Up to now, the aetiology and clinical significance of these lesions are poorly known. This part gives an overview of the veterinary and human literature in an attempt to explain the aetiology and to suggest a diagnostic protocol and treatment plan.
Introduction

Thoracic limb lameness in dogs is often localized in the elbow. Several developmental disorders are recognized as a cause of lameness, including fragmented coronoid process, osteochondritis dissecans of the humeral condyle, ununited anconeal process and incongruity. These disorders are grouped under the term elbow dysplasia and have been well documented in the literature (1-3).

Ununited medial humeral epicondyle is a lesser-known condition and has been historically classified as elbow dysplasia (2). It was considered as a failed fusion of the medial epicondyle ossification centre to the humerus, characterized by the presence of loose ossified bodies either on the medial side of the elbow joint or distal to the medial humeral epicondyle (4). Calcified bodies similar to those described as ununited medial epicondyle have been reported over the past years. The most frequently described appearance is a calcified structure near the medial humeral epicondyle (2, 5-12). One report describes spur formation at the caudal part of the medial humeral epicondyle (8). Several terms have been suggested to describe these lesions: dystrophic calcification of the flexor tendon origins, traumatic avulsion of the medial humeral epicondyle, medial humeral condylar osteochondritis dissecans and development of a preformed ossification centre (5-7, 9-12).

Up to now, the precise cause of medial humeral epicondylar lesions in dogs has been poorly understood and therefore it is difficult to define a correct term. It can also be questioned if all lesions can be classified under one term. Certainly the most frequently used term 'ununited medial epicondyle' seems incorrect, since several reports indicate that there is no radiographic evidence of a failed fusion between the medial epicondyle and the humerus (9, 10, 12).

Medial humeral epicondylar lesions can cause lameness, but may also be asymptomatic since they have been described as an incidental finding (4). Therefore interpretation of the radiographic changes may be difficult and this may lead to inadequate treatment. The pathological changes diagnosed in the canine elbow seem to have similarities to certain disorders in human medicine. Of note are Little Leaguer’s elbow and Golfer’s elbow, both of which are characterized by comparable images to those seen with medial humeral epicondylar lesions in dogs (13, 14). Since these conditions have been
well documented in human literature, this information can be used to further investigate medial humeral epicondylar lesions in dogs.

The purpose of this part is to describe the different forms of medial humeral epicondylar lesions reported in veterinary literature. The descriptions are abundantly illustrated by images from the Department of Medical Imaging and Small Animal Orthopaedics except for a few illustrations printed with the authors’ permission. By reviewing the reported cases and comparing them to similar disorders in human medicine, an attempt is made to explain the aetiology and to propose a diagnostic protocol and treatment plan.
**Development of the elbow joint and anatomy of the flexor muscles**

The elbow joint is a complex synovial hinge joint formed by the distal part of the humerus and the proximal part of the radius and the ulna. It is supported by strong collateral ligaments and the tendinous origins of several muscles, which originate from the medial humeral epicondyle (15).

The distal end of the humerus develops from three centres of ossification: one within the capitulum, one in the trochlea, and another in the caudal portion of the medial epicondyle. The medial humeral epicondylar epiphysis forms the medial humeral epicondyle, from which many carpal and digital flexor muscles originate. Radiographically, the centre of ossification for each part of the humeral condyle appears during the second to third week after birth. The ossification centre of the medial epicondyle appears four to eight weeks after birth (depending on the breed) and fuses to the distal humeral physis at approximately 10 weeks (2, 15, 16) (Figure 1).

![Figure 1: Medio-lateral elbow radiographs, illustrating the development of the medial humeral epicondyle of A) a Mixed Breed at 6 weeks of age, B) a Border Collie at 3 months of age and C) a mixed breed at 4 months of age. (1: Two separate centres of ossification of the humeral condyle are superimposed. 2: The large ossification centre of the medial humeral epicondyle is partially ossified in B) and completely ossified in C). 3: The proximal radial epiphysis. 4: Separate ossification centre for the olecranon process)
The muscles of the flexor group originate from the medial humeral epicondyle and attach to it through a short tendinous part (17). The flexor carpi radialis muscle arises from the medial humeral epicondyle and inserts on the proximal palmar surface of metacarpalia II and III. It runs caudal to the pronator teres, which arises from the medial humeral epicondyle and inserts on the medial side of the radius (17). The superficial digital flexor muscle is located caudal to the flexor carpi radialis muscle, arises from the medial humeral epicondyle and inserts with four tendons on the proximal palmar part of the middle phalanx of digits II, III, IV and V (Figure 2) (17).

The origin of the flexor carpi ulnaris has two separate muscles; an ulnar and a humeral head. The short bellied ulnar head arises from the caudal part of the olecranon and inserts distally as a tendon on the accessory carpal bone (17). The humeral head originates proximally from the medial humeral epicondyle, deep to the ulnar head, and inserts distally as a tendon on the accessory carpal bone (17). The deep digital flexor muscle has three points of origin: the medial humeral epicondyle, the proximal part of the ulna and the medial part of the radius. All three heads of the deep digital flexor muscle have one common distal tendon which divides into five tendons and inserts on the palmar side of the distal phalanx of digits I, II, III, IV and V (17) (Figure 3).
Medial humeral epicondylar lesions

Different types of lesions have been described and different terms have been used, suggesting a different aetiology. Nevertheless, most reports describe similar lesions. Based on the terminology the authors used to describe the pathology, an overview of the reported lesions is given.

Ununited medial epicondyle

The first report on calcified bodies near the medial humeral epicondyle mentioned an eight-month old German Shepherd dog in 1966 (2). Islands of bone (1.5 cm long and 0.5 cm wide) caudal and distal to the medial humeral epicondyle were diagnosed on lateral and craniocaudal radiographs (2). On histopathology, the calcifications consisted of normal cancellous bone, which had been attached to the humerus by fibrocartilage tissue (2). This lesion was suggested to be an ununited medial epicondyle and was considered as a form of elbow dysplasia (2). However, in later reports several authors demonstrated the absence of radiographic evidence of failed fusion of the medial epicondyle to the humerus, thus suggesting that ‘ununited medial epicondyle’ is an incorrect term to describe those calcified bodies near the medial humeral epicondyle (9, 10).

In another report, the existence of an ununited medial epicondyle is sustained; however it is explained as a form of osteochondrosis in which fragments of the cartilage avulse with the attachment of the muscles. The rationale in believing that osteochondrosis is the underlying problem is that an ununited medial epicondyle is often bilateral and is often seen without trauma (4) (Figure 4).

A recent study reported on the appearance of an ununited medial epicondyle in a group of related Labrador Retrievers, diagnosed at 6 and 8 years of age. The authors pointed out that this lesion does have a hereditary character however with unknown clinical relevance (18). No other reports on the inheritance of this condition were found. According to the latest guidelines of the International Elbow Working Group, an ununited medial epicondyle is no longer included in the elbow dysplasia scoring system (19).
Figure 4: Different examples of ‘an ununited medial epicondyle’. A) Medio-lateral flexed projection of an ununited medial epicondyle (white arrow). B) Medio-lateral extended projection of a large ununited medial epicondyle in another dog (black arrows). C) Craniocaudal projection of the same dog in image B. The densities seen in site b on the craniocaudal view are not obvious on the medio-lateral projection (white arrowheads). On the craniocaudal view, the densities in site a are obscured by the humerus but are evident on the medio-lateral projection (black arrows). From Piermattei et al., 2006.

Preformed ossification centre

Calcified bodies similar to those described in the reports on an ununited medial epicondyle, which were located medially at the origin of the humeral head of the deep digital flexor muscle, were diagnosed in the elbow joint capsule (5) (Figure 5). Histopathological examination had revealed bone trabeculae centrally, degenerated cartilage at the end of the calcified body and columns of irregular cartilage infiltrated by fibrous tissue towards the tendon of the humeral head of the deep digital flexor muscle (5). Instead of describing this lesion as an ununited medial epicondyle, it was suggested that this calcified mass had developed from a preformed ossification centre, comparable to a sesamoid bone. The calcified body was considered as a possible cause for the development of osteoarthritis (5).
Part I: Review of the literature

Figure 5: Craniocaudal radiographs illustrating a 'preformed ossification centre': a calcified body (white arrows), found bilaterally in the elbow joint capsule, located medially at the origin of the humeral head of the deep digital flexor muscle. From Grondalen et al., 1976.

**Traumatic avulsion of the medial humeral epicondyle**

Several papers reported on calcified bodies near the medial humeral epicondyle as a traumatic avulsion fracture of a part of the epicondyle. A few case reports describe traumatic avulsions in immature dogs presented with acute lameness (6, 7, 9, 10). On radiographs, an irregularly shaped calcified mass below the medial humeral epicondyle and a mis-shapen medial humeral epicondyle can be diagnosed (6) (Figure 6). Histologically, the bony fragment is covered by cartilage at the level of the tendino-osseous junction (7). It is suggested that when a fragment of the immature medial humeral epicondyle is separated by trauma, it continues to grow with nourishment from the attaching flexor muscles (9). Traumatic avulsion in the immature dog would presumably happen before the fusion of the growth centre with the distal humeral physis at 10 weeks (9, 16).
In human medicine a similar condition has been described as Little Leaguer’s elbow. It involves young children (baseball players) in which valgus stress across the elbow results in injury to the weak apophyseal plate of the medial humeral epicondyle. This results in a complete avulsion of the medial humeral epicondyle, while in dogs only a small part of the medial humeral epicondyle is separated (13, 20, 21) (Figure 7).

Traumatic avulsion of the medial humeral epicondyle can also appear in mature dogs. Two different presentations are described: acute cases associated with a recent trauma and chronic cases without evident trauma (7). On radiographs a bony fragment is separated from the medial humeral epicondyle. A unilateral appearance of the lesion suggests a traumatic cause while a developmental cause is more likely in bilateral lesions. To differentiate between both disorders, radiographs of both elbows are recommended (7).

Figure 6: Craniocaudal radiographs illustrating ‘an avulsion fracture of the medial humeral epicondyle’, indicated by white arrows. A) Three month old Sheltie Crossbred Dog; B) Nine month old Labrador Retriever; C) Six month old Labrador Retriever. From Culvenor et al., 1982 (A and B) and Vaughan, 1979 (C).
Figure 7: Craniocaudal radiographs illustrating different injury patterns of the medial humeral epicondyle in an immature human elbow. A displaced fracture of the medial humeral epicondyle (A) and apophysitis (B) (white arrows). From Klingele et al., 2002.

**Dystrophic calcification of the flexor muscle origins**

Another frequently described theory for the existence of calcified bodies is dystrophic calcification of the origin of the flexor muscles. According to several case reports, the calcified bodies were located mainly in the tendinous origin of the flexor muscles (5, 9-12) (Figure 8). This led to the presumption that the calcified bodies were metaplastic changes of tendon tissue, which connects the flexor muscles to the medial humeral epicondyle. This presumption was supported by the histological examination, which revealed bone trabeculae changing into tendon tissue or surrounded by fibrocartilaginous tissue. The histopathological diagnosis was tendinitis ossificans with reactive new bone proliferation (5, 9, 10, 12).
Figure 8: Different examples of ‘dystrophic calcification of the flexor muscle origins’ visible on a craniocaudal (A-C) and a medio-lateral extended (D) projection (white arrows). Note the different size and localization of the calcified bodies: a small-sized calcified body above the joint space (A) and a medium-sized calcified body below the joint space (B) and a craniocaudal and lateral view of a large-sized, elongated calcification extending from the medial humeral epicondyle to below the joint space (C and D). From Meyer-Lindenberg et al., 2004.

Because the joint capsule has extensions under the flexor carpi radialis muscle and the deep digital flexor muscle, it may also be involved in the pathology (9, 15). All flexor muscles can be involved, but the deep digital flexor muscle seems to be predisposed (12).

The real cause of the development of dystrophic calcifications is still unknown and most papers report it as a solitary elbow problem (2, 5, 7, 8). However, in some studies elbow dysplasia, mainly incongruity and fragmented medial coronoid process with chronic inflammation, was associated with the calcifications (9, 10, 12). In two studies describing a total of five dogs, incongruity was found in three dogs and in one study of 26 joints a fragmented medial coronoid process was diagnosed in six joints (9, 10, 12). Whether chronic arthrosis can lead to dystrophic calcifications remains uncertain (9, 12). In a study of 26 elbow joints with calcifications, only 13 had moderate to severe osteoarthritis. The remaining 13 elbow joints did not have any osteoarthritis, or just a mild form (12).
Another possible cause of dystrophic calcification within the flexor muscles is increased stress \((9, 10, 12, 14, 22)\). In human medicine, overuse injuries are well known and are described as enthesopathy or insertional tendinopathy. Tendinopathy refers to an overuse injury of the tendon close to the insertion on the bone, while enthesopathy is defined as a pathological change affecting the enthesis, at which the tendon attaches to the bone \((14)\). ‘Tennis elbow’ is known as lateral epicondylitis or enthesopathy of the tendons attaching to the lateral humeral epicondyle. It occurs more frequently than ‘Golfer’s elbow’ or medial epicondylitis, which is located around the medial humeral epicondyle. Both these conditions are caused by repeated microtrauma and stress and are considered as overuse injuries with a multifactorial origin: besides intrinsic factors such as anatomical variations, malalignment problems and muscle weakness, there are extrinsic factors of which excessive loading is the main pathological stimulus for degeneration \((23, 24)\). The enthesis is vulnerable to overuse injuries due to the stress concentration at the hard-soft tissue interface \((14, 22)\).

**Spur formation at the caudal part of the medial humeral epicondyle**

Bony spur formation or enthesophytes are bony outgrowths extending from the skeleton into a tendon at its enthesis \((14)\). Bony spur formation at the caudal aspect of the medial humeral epicondyle has not received attention as a clinical problem since it has only been reported once in veterinary literature. In this latter paper, four cases of bony spur formation are described \((8)\) (Figure 9). The authors suggested that trauma to the superficial digital flexor muscle insertion led to a local bony proliferation, and therefore the disorder was described as a traumatic enthesopathy. Histology of the resected medial humeral epicondylar spur revealed woven and compact bone with clusters of chondrocytes, surrounded by dense and loose fibrous connective tissue \((8)\). It was believed that in the early stage of this disorder pain and lameness may occur without the obvious presence of enthesophytes \((8)\). In two of the four described cases, simultaneous osteoarthritic changes were present. It was therefore unclear whether the medial humeral epicondylar spur was a manifestation of osteoarthritis or the primary problem causing osteoarthritis \((8)\). In human literature, it is suggested that osteophyte and enthesophyte formation are linked and that they both represent a
skeletal response to stress. While osteophytes develop to adapt to a changed loading on synovial joints caused by injury or disease, enthesophytes represent a comparable adaptation at the enthesis (14). Because an enthesophyte consists of fibrocartilage at the tip, it is believed that bony spur formation is an extension of normal enthesis growth in the direction of the tendon reflecting the orientation of the fibrocartilage cells (14, 25).

Figure 9: A large ‘epicondylar spur’ on the medial condylar ridge of the humerus in a 5-year-old Golden Retriever (white arrow). From May et al., 1988.
Clinical data

According to literature, there does not seem to be a breed predisposition for medial humeral epicondylar lesions. The reported breeds are German Shepherd Dog, Bernese Mountain Dog, Rottweiler, Newfoundland, Labrador Retriever, Collie, Basenji, Bassett Hound, Sheltie, Dalmatian and Airdale Terrier (2, 7-10, 12, 18). Medial humeral epicondylar lesions seem to occur in dogs of all ages (7, 9, 10, 12). Traumatic fragmentation was seen in a number of dogs before 10 weeks of age, after which the medial epicondylar epiphysis fuses with the humerus (2, 9). Medial humeral epicondylar lesions may cause lameness or may be asymptomatic (4). The presence of a calcification or spur at the medial humeral epicondyle is often considered as a coincidental finding without any clinical significance (4, 8). However, several reports attributed lameness to the presence of these lesions (2, 5, 7-12). Other clinical findings are aspecific and comparable to those seen with any other elbow pathology: a painful and swollen elbow joint, painful and limited flexion and extension of the elbow sometimes associated with crepitation, and in chronic cases atrophy of the shoulder muscles (2, 7-10, 12).
**Diagnostics**

Radiographic examination is the first step to visualize the bony changes of the elbow joint. The medio-lateral flexed and craniocaudal projections should be examined carefully to determine the presence, shape and location of calcified bodies and the presence of spur formation (4, 8). According to the guidelines of the International Elbow Working Group a spur is described as a sign of osteoarthritis (26). A calcified body is most frequently diagnosed on the craniocaudal projection and is located caudoventral, medial or distal to the medial humeral epicondyle at the level of the joint space (4, 12) (Figure 8). The calcified body may be missed on the medio-lateral projection because of superimposition of the humerus and radius (4). Care should be taken not to confuse the calcified body with a displaced osteochondritis dissecans flap (27, 28). However when the fragment is visible on the medio-lateral projection, the flexed and extended projection can be used to determine whether the fragment hinges dorsally on the flexed projection, demonstrating the localization within the flexor muscles (9). Since the presence of concurrent elbow problems has been described, the joint should be inspected for other lesions to exclude them as a possible cause of lameness and to make sure that lameness is indeed related to the radiographic lesion of the medial humeral epicondyle (9, 10, 12).

Ultrasonography of both elbows is recommended to evaluate whether the flexor muscles are involved in the process. In man, it is a commonly used technique (29). The main ultrasonographic findings of medial epicondylitis in man are pre-insertional hypoechoic swelling, outward bowing and thickening of the common tendon of the flexor muscles (the pronator teres, the flexor carpi radialis, the palmaris longus, the superficial digital flexor and the flexor carpi ulnaris). The tendon appears to be heterogenous with decreased echogenicity and focal or diffuse areas of irregular fibrillar appearance and ill-defined margins with partial or complete tears. Additionally, cortical irregularities at the medial humeral epicondyle (spur formation) and intratendinous calcifications can be detected (29, 30, 31). Comparison between both elbows is necessary in order to notice subtle differences (31). Advantages of ultrasonography are the ability to examine soft tissue, which is poorly visible on radiographs, real-time assessment of joint and tendon movement under manipulation.
and lack of ionizing radiation (32). Limitations are operator-dependence, a long learning curve and the need for high resolution ultrasound equipment (31).

Magnetic resonance imaging (MRI) is another diagnostic tool to confirm the involvement of flexor muscles in medial humeral epicondylar lesions, commonly used in human medicine (33-35). The MRI findings of medial epicondylitis are increased signal intensity within the common flexor tendon on both T1-weighted and T2-weighted images, tendon thickening, small joint effusions and periostitis (34, 36) (Figure 10). Advantages include visualization of the anatomy in multiple planes, superior soft-tissue detail and avoidance of ionizing radiation exposure. Disadvantages are the need for general anaesthesia and the high costs of the equipment (33).

![Figure 10: An axial T1-weighted spin echo MR image (A) and an axial fat suppressed T2-weighted fast spin echo MR image (B) of a human elbow joint with medial epicondylitis. A) Abnormal intermediate signal intensity within a thickened common flexor tendon origin (white arrow). B) Abnormal intermediate to high signal intensity within a thickened common flexor tendon origin (white arrow). A high signal intensity is also visible in the superficial subcutaneous tissue at the lateral side of the elbow (white arrowhead), which most likely represented inhomogeneous fat suppression. From Kijowski et al., 2005.](image_url)
In a clinical study ultrasonography was compared with MRI for diagnosing epicondylitis in man (23). The sensitivity for detecting epicondylitis ranged from 64% to 82% for ultrasonography and from 90% to 100% for MRI. Specificity ranged from 67% to 100% for ultrasonography and from 83% to 100% for MRI. Ultrasonography seems adequate for diagnosing epicondylitis in the majority of the patients, allowing MRI to be reserved for patients with symptoms whose ultrasonographic findings are normal (23).

Although computed tomography (CT) is not used as a standard diagnostic tool in human medicine to diagnose epicondylitis, it can be of interest in diagnosing lesions of the medial humeral epicondyl and the flexor muscles using either a bone or soft tissue window (37). As with MRI, CT requires general anaesthesia, but image acquisition is faster and the images are more detailed (37).
**Treatment**

Asymptomatic cases radiographically discovered as incidental findings in canines are supposed to be left untreated, although follow-up studies of those cases are not available. Symptomatic cases can be treated either conservatively or surgically. Due to the small number of cases and the short follow-up period, the results of the treatment should be interpreted with caution.

**Conservative**

The optimal medical treatment for medial humeral epicondylar lesions has not yet been documented in veterinary literature. A treatment of four weeks of pentosan polysulphate sodium\(^a\) did not have any effect (10). Prednisolone\(^b\) and Mefenamic acid\(^c\) were reported to be effective, but lameness recurred when treatment was stopped (8). In human medicine, conservative therapy is the first step in treating medial epicondylitis. It has been described as highly successful, although there is no information about the long-term results. The treatment is initiated with the application of ice to the affected elbow for a period of 15-20 minutes three to four times a day, combined with oral nonsteroidal anti-inflammatory medication for a period of 14 days. In cases of little response to this treatment, a period of night splinting combined with local corticosteroid injection around the affected tendon insertion is suggested. As soon as the symptoms have improved, a guided rehabilitation program can start (38).

**Surgical**

In dogs, traumatic avulsion of the medial humeral epicondyle is treated surgically by fixating the chip with a lag screw (6). Only when the fragment is too small or brittle it is suggested to remove it. Post-operative external support by using a supportive bandage, a modified Robert-Jones bandage or a half splint is recommended in order to prevent excessive elbow abduction and hyperextension of the digits (7). Information on the results is limited: in two dogs in which a lag screw was used, lameness had
disappeared at six months and one year respectively after surgery. One dog was still sound two years after removal of the chip (7).

In contrast to dogs, treatment of Little Leaguer’s elbow in humans consists of complete rest, ice packs and analgesic medication; surgery however is rarely necessary (13). Minimally displaced fractures can be treated with splint immobilization, and fractures with more than 5 mm displacement should be treated surgically by internal fixation (13). Several papers report on the surgical treatment of lesions other than a traumatic avulsion in dogs that do not benefit from conservative therapy (8, 10, 12). A standard surgical procedure for the removal of the calcified bodies has not been described in veterinary literature, although all papers report on a similar surgical approach. Via a medial incision the superficial and deep fascia are separated, the origin of the different flexor muscles is dissected, the involved flexor muscle is identified and the calcified body is isolated and removed (Figure 11). Any bony spur formation is removed using a rongeur. The remaining muscle or tendon stump is sutured to the proximal part of the involved muscle (5, 8-10, 12).

Figure 11: Surgical removal of the pathologic tissue in a canine elbow affected by medial humeral epicondylar lesions. A) Via a medial incision the superficial and deep fascia are separated and the superficial digital flexor muscle is visible. B) The white pathologic tissue within the flexor muscle is removed (held up by the bracket forceps).
Because the calcified bodies can be associated with other forms of elbow dysplasia, the arthroscopic or surgical inspection of the elbow joint for cartilaginous or bony fragments is recommended (9, 10, 12). A good outcome has been reported following surgery. The largest and also most recent study reports on the treatment of 22 elbows: 15 dogs did not have any lameness, five dogs were only lame after heavy exercise and two dogs were still lame after an average follow-up period of 18 months (12). Three dogs with spur formation and one dog with spur formation and calcified bodies improved significantly after an average postoperative period of four months (8). The need for surgical removal of the calcified body and the good outcome of that treatment can be questioned since no long-term follow-up studies have been done. However, in a study about mineralization of the supraspinatus muscle, reformation of the fragment five years after surgical removal has been described without recurrence of lameness (39).

Figure 12: Surgical treatment of medial epicondylitis in man. A) A skin incision overlying the medial humeral epicondyle reveals cutaneous and ulnar nerves. The dotted line indicates the incision site of the common flexor-pronator mass. B) The distal region of the common flexor-pronator origin is visible with the pathologic tissue (held by the forceps). C: Reattachment of the common flexor-pronator origin to the medial humeral epicondyle. From Ciccotti et al., 2004.
In human medicine a standard surgical procedure for medial epicondylitis consists of the excision of the pathological tissue, stimulating the healing response by improving local vascularity, reattaching any elevated tendon to the medial humeral epicondyle and repairing the remaining defect (Figure 12). Post-operatively a splint is placed (38). The prognosis seems good: a review of 35 human patients with medial epicondylitis who underwent surgical treatment reported 88% good to excellent results after an average follow-up period of six years (40). Another report of 26 patients mentioned 87% success after an average follow-up period of seven years (41).
Conclusion

Pathological changes in the area of the medial humeral epicondyle are most frequently described as calcified bodies near the medial humeral epicondyle while spur formation is only mentioned in one report. Several terms have been used to describe these lesions: ununited medial epicondyle, dystrophic calcification of the flexor tendon origins, traumatic avulsion of the medial humeral epicondyle, medial humeral condylar osteochondritis dissecans and development of a preformed ossification centre. The varying description and illustrations (Figure 4 to 8) of the reported cases suggest a different aetiology, although there are several similarities between the different classifications. Histopathological examination of the resected calcified tissue revealed similarities as well: all calcifications or fragments contained bone surrounded by cartilage or fibrocartilaginous tissue, as well as a spur consisting of compact bone with clusters of cartilage. However, histopathological findings do not explain the cause of the problem.

A comparison of the findings in dogs with a similar pathology in man supports either a traumatic cause or an overuse lesion with an insidious onset in most cases. Although in some cases a developmental problem could be the cause, the term 'ununited medial epicondyle' does not reflect the aetiology of the different forms and should therefore be replaced by a more appropriate term. The term 'flexor enthesopathy' is proposed to describe the presence of pathological changes within the flexor muscles and their attachment to the medial humeral epicondyle, without referring to the cause of the problem.

Changes at the medial humeral epicondyle are often considered as clinically unimportant lesions, although they were the cause of lameness in the reviewed reports. It is also the authors’ experience that these lesions should be included in the differential diagnosis of elbow problems as a primary cause of lameness. When the first radiographic screening reveals medial humeral epicondylar changes, further diagnosis should include ultrasonography and eventually should be followed by magnetic resonance imaging.
Conservative treatment does not appear to be successful in dogs, whereas in the majority of reported cases surgical removal of the calcifications led to a significant improvement. In man, however, conservative treatment is the standard treatment. The good results may be explained by the early diagnosis and appropriate treatment. An early and correct diagnosis might improve the results of conservative treatment in dogs. A study of a large series of dogs affected with medial humeral epicondylar lesions should reveal more information on the diagnosis and management of the different types (Section III, Chapter 2-8).
Footnote

a Cartrophen; Biopharm Australia: Bondi Junction, Australia
b Prednoleucotropin; Berk Pharmaceuticals: Eastbourne, UK
c Ponstan; Parke-Davis: West Ryde NSW, Australia
References


Part I: Review of the literature


Part II

PRIMARY FLEXOR ENTHESOPATHY OF THE CANINE ELBOW: IMAGING AND ARTHROSCOPIC FINDINGS IN EIGHT DOGS WITH DISCRETE RADIOGRAPHIC CHANGES
PRIMARY FLEXOR ENTHESOPATHY OF THE CANINE ELBOW: IMAGING AND ARTHROSCOPIC FINDINGS IN EIGHT DOGS WITH DISCRETE RADIOGRAPHIC CHANGES

Part II: Discrete primary flexor enthesopathy

**Summary**

The aim of this part was to describe the radiographic, ultrasonographic, computed tomographic (CT), magnetic resonance imaging (MRI) and arthroscopic findings in eight dogs with elbow lameness caused by primary flexor enthesopathy. Eight client-owned dogs were included in this clinical study. In all dogs, lameness was localized to the elbow joint by clinical examination. Radiographic examination, ultrasonography, CT and MRI were performed prior to arthroscopy. In seven dogs surgical treatment and subsequent histopathology were performed. Primary flexor enthesopathy of the medial humeral epicondyle was diagnosed in eight dogs (13 joints) by combining the minimal radiographic changes with specific ultrasonographic, CT, MRI and arthroscopic findings at the medial humeral epicondyle. In all elbow joints any other pathology could be excluded. Histopathology of the affected tissue revealed degeneration and metaplasia of the flexor muscles. The most important cause of elbow lameness in dogs is medial coronoid disease. Primary flexor enthesopathy at the medial humeral epicondyle, which is an unrecognized condition, can be considered a possible cause of elbow lameness in the dog. Diagnosis is based on specific imaging and arthroscopic findings. Since medial coronoid disease often presents with minimal radiographic and arthroscopic changes, primary flexor enthesopathy of the medial humeral epicondyle should be considered as a differential diagnosis in these cases, in order to make the correct treatment decision.
Introduction

The elbow is a common location for thoracic limb lameness in medium and large breed dogs. The most frequent diagnosis is fragmented coronoid process, based on the history and the clinical and radiographic examination. Typical radiographic findings are fragmentation, deformation or an unclear outline of the medial coronoid process, often accompanied by sclerosis or secondary osteoarthritis (1). However, in a considerable number of cases diagnosis of fragmented coronoid process is challenging because of minimal radiographic changes (2-5).

Ununited medial epicondyle has been reported as a rare problem and is often considered as a clinically insignificant finding (6). It was originally reported in 1966 after which several clinical reports were written, discussing and disagreeing on the terminology, origin, clinical significance and prevalence of this condition (7-16). Most reports described the condition as a separate bony fragment, an avulsed fragment, or a calcified body of the flexor muscles. A German report of 2004 described the radiographic and arthroscopic findings in 26 joints (23 dogs) with a calcified body ranging in size from 5x3 mm to 30x12 mm, mainly visible on the craniocaudal projection (16). One report described a 'spur' at the caudal edge of the medial humeral epicondyle as the only finding and suggested that pathology of the flexor muscles might be present without radiographic changes at an early stage (17). This disorder was called enthesopathy because of the pathologic changes within the flexor-bone connection, called the 'enthesis' (18, 19).

A review of the recent publications and orthopaedic handbooks illustrated that ununited medial epicondyle receives little attention as a cause of elbow lameness and it is not mentioned on the list of differential diagnoses (6). Attention is exclusively given to fragmented coronoid process, osteochondritis dissecans of the humeral condyle, ununited anconeal process and elbow incongruity (20, 21). One recent report described the coincidental diagnosis of ununited medial epicondyle and elbow osteoarthritis in a litter of Labrador Retrievers and suggested a higher incidence than presumed, although no clinical importance was attributed to the finding (22). Furthermore, ununited medial epicondyle is no longer considered as a disorder belonging to the elbow dysplasia complex because of its low prevalence and low clinical impact (23). However in the author's experience, clinically significant lesions in the area of the medial humeral
epicondyle are diagnosed on a regular basis. They are visible not only as a clear fragment or calcified body, but also with minimal radiographic lesions. In a subsequent series of 200 radiographic elbow images, changes of the medial humeral epicondyle were seen in 40% of the cases (24) (Section III, Chapter 1). A calcified body near the medial humeral epicondyle was diagnosed in 5% of the 200 elbows. In half of these cases the calcified body was diagnosed as a sign of primary flexor enthesopathy. A spur or irregular outline of the medial humeral epicondyle without the presence of a fragment was seen in nearly 25% of the 200 elbows. In most cases this was an expression of osteoarthritis, but in 2% of the total number of elbows the changes were an expression of primary flexor enthesopathy (24).

This part describes the discrete radiographic and specific ultrasonographic, computed tomographic (CT), magnetic resonance imaging (MRI) and arthroscopic findings in eight dogs with elbow lameness, admitted because of a suspected fragmented coronoid process and diagnosed with pathologic changes of the medial humeral epicondyle and the attaching flexor muscles.
Materials and methods

Medical records of eight dogs, evaluated and treated over a period of five years, were analyzed. In all dogs a complete history and results of a clinical examination were available as well as the radiographic, CT and arthroscopic images (13 joints). In three dogs (five joints) additional ultrasonographic and MRI images were available. In case of surgical treatment, histopathology was carried out.

Clinical examination

Clinical examination included inspection on walk and trot and palpation to define the range of motion, joint distension, and pain reaction. Detailed scoring of lameness was done by assignment of grades on a scale from zero to ten, a system which has been described for equine lameness evaluation (25, 26).

Imaging

Radiographic examination with the dog sedated included three standard projections of both elbows: the mediolateral flexed and extended projections and 15° oblique craniolateral-caudomedial projection. Ultrasonography of the medial aspect of the elbow was performed in three dogs (five joints) with a linear, 10-15 MHz probea. Computed tomography was performed for all patients using a helical CTb with 1 mm slice thickness and a bone reconstruction window of both elbows. MRI of the elbow was performed in three dogs (five joints) in transverse, sagittal and dorsal planes with a 0.2 Tesla, permanent magnetc using T1-weighted, T2-weighted and STIR sequences. Arthroscopy was performed with a 2.4 mm arthroscope via a standard medial approach (27).
Treatment

Treatment consisted of one single intra-articular administration of 0.5-2 mg/kg bodyweight Methylprednisolonacetate combined with 6 weeks restricted exercise in one dog (two joints). Seven dogs (10 joints) were treated by partial removal of the affected muscle, followed by 6 weeks restricted exercise with walks on leash and administration of non-steroidal anti-inflammatory drugs (7 dogs, 10 joints). Broad-spectrum antibiotic drugs were administered during 5 days postoperatively. Two affected contralateral joints were left untreated.

Treatment results were assessed with a questionnaire to the owners inquiring about the lameness status, activity level and need for medication and by a clinical examination three months to four years after treatment.
Results

The diagnosis of primary flexor enthesopathy was made in eight dogs. Five dogs were affected bilaterally. Breed, sex and age are listed in table 1.

<table>
<thead>
<tr>
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<th>Treatment</th>
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Table 1: Distribution of breed, sex and age, uni- or bilateral lesions and uni- or bilateral treatment in eight dogs affected by primary flexor enthesopathy of the elbow. (M: Male, F: Female)

Clinical examination

Lameness had a gradual onset in five dogs and an acute onset after exercise without obvious trauma in three dogs. Non-steroidal anti-inflammatory drugs had been administered in all dogs resulting in a temporary improvement or no improvement at all. On clinical examination the dogs showed a mild to moderate degree of lameness (grade two to six on a scale of ten), varying elbow distension and pain reaction on hyperextension and a full range of motion. In all cases, a firm, well defined swelling could be palpated on the medial side of the elbow joint, caudodistal to the medial humeral epicondyle. Pressure in that region was not particularly consistent in eliciting signs of pain.
Imaging

In all 13 joints, discrete radiographic changes were detected. Radiographic changes of the medial humeral epicondyle were seen unilaterally in three dogs and bilaterally in five dogs. The medial humeral epicondyle showed a discrete spur in 11 joints (Figure 1: A, B). In two joints of the same dog, the medial humeral epicondyle was sclerotic with a small radiolucent area (Figure 1: C).

Figure 1: Radiographic findings (mediolateral flexed projections) in three joints affected by primary flexor enthesopathy. A) Bernese Mountain Dog of 3 years old showing a discrete spur (white arrow) on the medial condylar ridge of the humerus with mild osteoarthritis and subtrochlear sclerosis (case 6). B) A 1.5-year-old Boerboel with a discrete spur (white arrow) at the medial humeral epicondyle (case 4). C) Great Swiss Mountain Dog of 3.5 years old with sclerosis and a radiolucent area at the medial humeral epicondyle (white arrow) (case 8).

Radiographic signs of osteoarthritis were absent in twelve joints. One joint showed mild osteoarthritis (grade 1 according to the International Elbow Working Group) (Figure 1: A). In all dogs the medial coronoid process was well delineated and had a normal triangular shape. Subtrochlear sclerosis was absent in all joints, except for the one joint with signs of mild osteoarthritis (Figure 1: A). The medial part of the humeral condyle was round and smooth and there was no clear step or enlargement of the joint space suggesting the presence of incongruity. The absence of incongruity was confirmed by CT.
An ultrasonographic examination was performed in five affected joints (three dogs). A mild to moderate outward bowing of the flexor carpi ulnaris muscle and presence of fluid around and within the affected muscles (the superficial digital flexor muscle, the deep digital flexor muscle and/or the flexor carpi ulnaris muscle) were seen in all joints. The echogenicity of the deep digital flexor muscle or flexor carpi ulnaris muscle was decreased in four of the five joints and the flexor muscles (the superficial digital flexor muscle, the deep digital flexor muscle or flexor carpi ulnaris muscle) were thickened in three of the five joints (Figure 2).

Figure 2: Ultrasonographic image in a longitudinal plane of a 3.5-year-old Great Swiss Mountain Dog (case 7) with pathologic changes of the flexor carpi ulnaris muscle (1): moderate outward bowing (broad white arrow) and local decreased echogenicity (small white arrows) can be noticed. The deep digital flexor muscle (2) and superficial digital flexor muscle (3) show a normal appearance.
CT was performed in all dogs. The medial humeral epicondyle was sclerotic and showed a periosteal reaction in all affected joints (Figure 3: A, B). The medial coronoid process did not show any signs of subchondral bone cracks, fissure or fragmentation, which was afterwards confirmed during arthroscopic inspection (Figure 3: C). The medial aspect of the humeral condyle did not show any heterogenity or stripes as signs of kissing lesions and there was no step on the sagittal view indicating incongruity (Figure 3: D).

Figure 3: CT transverse slices in bone algorithm (A and B at the level of the humeral epicondyles, C at the level of the medial coronoid process) of a 5-year-old Samoyed (case 1) with bilateral lesions. A) Spur and sclerosis at the medial humeral epicondyle of the right elbow (white arrow). B) Sclerosis and periosteal reaction at the medial humeral epicondyle of the left elbow (white arrow). C) Intact medial coronoid process (black arrowhead), minimal osteoarthritis at the medial side of the ulna (black arrow) (left elbow). D) Corresponding radiographic image of the left elbow, showing a spur (white arrow).
MRI was performed in three dogs (five joints). The sagittal T2-weighted sequence revealed swelling and a hyperintense signal around the proximal aspect of the flexor muscles extending in the muscle bellies in all joints. This signal was confirmed as being a fluid signal on the fat suppressed STIR sequence (Figure 4).

Figure 4: Radiographic image (mediolateral extended projection, A) and MRI images (B-D) of a 3.5-year-old Great Swiss Mountain Dog (case 7). A) Minimal spur (white arrow). B) T2-weighted transverse image showing a hyperintense signal within the deep fibers of the flexor tendon origins (white arrows) and an increased signal change of soft tissue. C) STIR sagittal image demonstrating a hyperintense fluid signal within the flexor muscles at the level of the attachment site to the medial humeral epicondyle (white arrows). D) T2-weighted sagittal MRI image showing a hyperintense fluid signal at the level of the attachment site of the flexor muscles (white arrows).
Arthroscopy was performed in all affected joints. At the insertion of the flexor muscles a considerable number of ruptured fibers and thickened remnants were visible in all joints. The short tendinous part of the muscle was not seen as one solid structure but looked segmented and fibrillated (Figure 5: A and B). An erosion near the medial humeral epicondyle was seen in half of the affected joints while the remaining part of the joint surface, including the medial coronoid process and the humeral condyle, was intact (Figure 5: C). Synovitis was present in all joints but was locally more pronounced around the origin of the affected muscles.

Figure 5: Arthroscopic views of primary flexor enthesopathy in a 3.75-year-old Great Swiss Mountain Dog (case 7). A) Segmented and fibrillated aspect of the attachment of the flexor carpi ulnaris muscle (black arrow), localized synovitis and discrete erosion (white arrow). B) Remote view of the attachment of the flexor muscles (black arrow), anconeal process (white arrow) and medial aspect of the humeral condyle (black asterisk). C) Intact medial coronoid process is visible (white asterisk). The equal level of the radial head and medial coronoid process suggests congruity of the joint.

The diagnosis of ‘affected’ of the contralateral joints was based on radiographic, CT and arthroscopic findings and, when carried out also with ultrasonography and MRI. Not only there was fluid accumulation, but also thickening and typical arthroscopic signs of insertion tendinopathy.
**Surgical findings and treatment**

Surgical treatment was performed in three unilateral cases and two dogs had bilateral surgery with a period of one year between both surgeries. Two bilaterally affected dogs had unilateral surgery. One bilateral case was treated with a bilateral injection of 0.5-2 mg/kg bodyweight Methylprednisolone acetate, four years between both joints.

During open surgery the affected part of the flexor muscle appeared to consist of thickened, hard and white fibrous tissue located in one or more flexor muscles and connected with the surrounding tissue and joint capsule (Figure 6: A). After transection the structures had the aspect of swollen, partially ruptured and unorganized tendinous tissue (Figure 6: B).

![Figure 6: Aspect of the affected tissue during open surgery. A) White, thickened proximal part of the flexor carpi ulnaris muscle (white arrow). B) Transection shows the thickened fibrous tissue (white arrow).](image)
Follow up

All dogs returned to complete function following treatment within one to six weeks. According to the owners the dogs regained full activity and did not need any further medication. Two bilaterally affected dogs with unilateral surgery never showed clinical problems of the untreated side. On clinical examination the dogs did not show any lameness, the treated joints were not distended and radiography did not show any development of osteoarthritis.

Histopathology

The removed tissues consisted of dense collagenous tissue for the major part. The distal end of the samples showed normal muscle fibers. At the proximal end, synovial villi were present. The collagenous tissue in all cases showed a varying degree of disorganised collagen bundles, presence of young fibroblasts, multiple tortuous small blood vessels and, in two cases, local cartilagenous metaplasia (Figure 7).

Figure 7: Histopathological image demonstrating local cartilagenous metaplasia of the flexor muscles. (Hematoxylin and Eosin staining, magnification 40x)
Discussion

This study draws the attention to flexor enthesopathy as a differential diagnosis for elbow lameness with discrete clinical and radiographic changes. Furthermore, flexor enthesopathy is a poorly known elbow disorder and cases with obvious radiographic changes are often considered clinically unimportant. The imaging and arthroscopic findings of eight dogs with primary flexor enthesopathy showing discrete radiographic changes are described. At first presentation, the combination of elbow pain and minimal radiographic changes suggested the presumptive diagnosis of a fragmented medial coronoid process. However, further diagnostic imaging and arthroscopy excluded medial coronoid problems and instead lead to the detection of lesions of the flexor muscles attaching at the medial humeral epicondyle.

In dogs, the 'ununited medial epicondyle' is known as a disorder characterized by a bony fragment or calcified body in the area of the medial humeral epicondyle (6). Since there was no fragment or calcification in the described series, the term 'ununited medial epicondyle' was considered inappropriate. To indicate the disorder, we chose the term 'flexor enthesopathy' adopted from human medicine (19). In human medicine, the 'enthesis', which represents the tendon-to-bone-connection, has been receiving great attention lately, enthesitis or enthesopathy being considered as an important cause of locomotion problems. The 'Golfer's elbow' and 'Tennis elbow' are examples of this problem (18, 19). In this paper, the term 'primary' is used, because it refers to the absence of underlying pathology in the elbow. This is in contrast with other reports that describe lesions at the medial humeral epicondyle in the presence of fragmented coronoid process and incongruity (8, 12, 16, 22). In these joints it is unclear whether flexor enthesopathy is the primary cause of the elbow problem or secondary to another elbow disorder. Although several reports have described a radiographically visible fragment or calcification as the cause of lameness, it is often considered as a clinically unimportant finding (6, 22, 23). Only one report described a 'spur' as the only sign of flexor pathology (17). A spur at the medial humeral epicondyle is often considered as a sign of osteoarthritis and in that case it is not recognized as a primary problem. However, when a joint is affected by osteoarthritis, new bone formation is found at several locations within the joint. In this series no osteophytes at other locations than
the medial humeral epicondyle could be demonstrated except in one case with a mild degree of osteoarthritis.

In the light of 'obscure' elbow lameness, it is important to draw the attention to primary flexor enthesopathy, in order to consider it in the differential diagnosis when radiographic changes are not obvious. Recent publications attribute obscure elbow lameness to fragmentation of the medial coronoid process (FCP), often seen as discrete lesions (2, 4, 28). Recently the term 'Medial Coronoid Disease' (MCD) was introduced, because in 17.6% of cases no fragmentation or fissure was found (29). Subtotal coronoidectomy was advised, even in cases where no clear lesion of the medial coronoid process during arthroscopic inspection was visible. Other recently published papers illustrate the difficulty of the diagnosis of medial coronoid disease since many dogs show only minimal clinical and radiographic signs of elbow pathology (2, 4, 30). Since the described cases in this series have a similar profile as dogs affected by FCP/MCD (2, 4, 29) a lesion of the flexor tendons should always be considered in the differential diagnosis when 'obscure elbow lameness' is present, in order to make a correct diagnosis and treatment decision.

The age of the dogs in the presented series varied between two and seven years with a mean of 3.5 years. This is older than the typical age for medial coronoid disease, which is known as a developmental disorder. However, several recent papers describe the occurrence of medial coronoid disease in older dogs (2, 4, 5, 29, 31).

A wide range of imaging techniques was applied in this study. Because radiographic changes were minimal, additional imaging techniques were necessary to demonstrate pathology in the suspected region and to exclude other pathology. CT enabled the demonstration of bony changes, while arthroscopy confirmed pathology in the area of the medial humeral epicondyle. Both techniques were also helpful in excluding medial coronoid lesions, which was the most probable diagnosis in these cases. Only in a later stage of this series the authors became more familiar with the disorder and decided to apply other techniques that enable the visualization of soft tissue structures. This explains why ultrasonography and MRI were not used in all cases. High-resolution ultrasonography enabled an excellent visualization of the flexor muscles and their
attachment. The outward bowing, swelling and localized ruptures were easily recognizable. MRI demonstrated swelling and an increased signal that confirmed pathology of the affected flexor muscles. Probably not all imaging modalities are necessary to visualize flexor enthesopathy and to distinguish clinically important lesions from coincidental findings. It was not the object of this study to compare the different imaging modalities for the diagnosis of flexor enthesopathy. For that purpose the number of patients is too small and this series does not reflect the average presentation. The next chapters describe larger studies including more evident cases of flexor enthesopathy with visible fragments or calcifications in order to define the value of each imaging modality and the clinical significance of each pathologic finding.

The diagnosis of primary flexor enthesopathy was thus based on the presence of a variety of specific changes and exclusion of medial coronoid changes by using different imaging modalities. Five contralateral joints were diagnosed as affected since the same findings were noticed. Although the presence of fluid is the most significant factor to indicate an active process and chronic scar tissue will not necessarily lead to clinical problems, no clear differences could be noticed in this limited series.

Two types of treatment derived from human medicine were performed: intra-articular injection of corticosteroids or surgical removal of the affected tissue (6, 32-34). All dogs became sound after treatment. It was not the intention of this paper to describe and evaluate the most appropriate treatment. In the future, a larger study will be carried out to indicate the optimal treatment protocol for flexor enthesopathy.

This study draws the attention to an unrecognized elbow problem, leaving several questions to be answered. It is the author's opinion that primary flexor enthesopathy represents an infrequent yet important option in the differential diagnosis of elbow problems in medium and large breed dogs. Cases of primary enthesopathy may show minimal radiographic changes and hence suggest medial coronoid disease as the cause of lameness. A correct diagnosis of flexor enthesopathy can only be obtained by combining the radiographic findings with other imaging techniques and arthroscopy to confirm suspected lesions of the medial humeral epicondyly and the attaching flexor muscles and to exclude medial coronoid disease.
Footnote

a MyLab 30; Esaote: Firenze, Italy
b CT Prospeed; GE: Milwaukee, Wisconsin, USA
c Airis Mate; Hitachi: Tokyo, Japan
d Richard Wolf GmbH: Knittlingen, Germany
e Moderin 20 mg/ml; Pfizer A.H: Brussels, Belgium
References

Part II: Discrete primary flexor enthesopathy


SECTION II

SCIENTIFIC AIMS
SCIENTIFIC AIMS

Flexor enthesopathy refers to lesions of the flexor muscles and their attachment to the medial humeral epicondyle. This unrecognized elbow disorder is radiographically characterized by the presence of a calcification or fragmentation in the area of the medial humeral epicondyle and the attaching flexor muscles or by the presence of a spur at the caudal edge of the medial humeral epicondyle. Since additional information about this condition is rather limited, the general aim of this study was to further unravel the aetiology, clinical significance, diagnosis and treatment.

Radiographic changes of the medial humeral epicondyle and the attaching flexor muscles have mainly been considered as clinically unimportant or coincidental findings and have mostly been regarded as an expression of osteoarthritis. The real prevalence and meaning of these lesions have never been investigated. The first aim of this study was therefore to determine the frequency and radiographic aspect of medial humeral epicondylar lesions and to evaluate the association with osteoarthritis in a series of affected elbows.

Lesions consistent with flexor enthesopathy can be the primary cause of elbow lameness, however the majority of the lesions are seen concomitant with other elbow disorders. To introduce the new terms ‘primary’ and ‘concomitant’ flexor enthesopathy, the second aim of this study was to give a description of both forms of flexor enthesopathy based on the presence of flexor changes and the presence or absence of other elbow disorders using different diagnostic techniques. The obtained definitions can be used as a basis for further detailed studies.

The detection of flexor enthesopathy on the one hand and the distinction between primary flexor enthesopathy and elbow dysplasia with concomitant flexor enthesopathy on the other hand are essential to make the correct treatment decision. Until now, the differentiation between both forms of flexor enthesopathy was only based on the presence or absence of other elbow disorders. Therefore, the third aim of this study was to determine whether specific pathologic changes of the flexor muscles and their attachment could be used as other parameters to distinguish both forms. For this
purpose, a detailed analysis of specific flexor enthesopathy characteristics was performed with radiography, ultrasonography, scintigraphy, CT, MRI and arthroscopy of joints affected with primary flexor enthesopathy, concomitant flexor enthesopathy, elbow dysplasia and normal joints. The results of these studies should enable the establishment of a reliable diagnostic protocol.
SECTION III

RESULTS
Chapter 1

RADIOGRAPHIC FINDINGS OF THE MEDIAL HUMERAL EPICONDYLE IN 200 CANINE ELBOW JOINTS
RADIOGRAPHIC FINDINGS OF THE MEDIAL HUMERAL EPICONDYLE IN 200 CANINE ELBOW JOINTS

Summary

The aim of this chapter was to determine the frequency and radiographic aspect of medial humeral epicondylar lesions as a primary or concomitant finding and to evaluate the association with osteoarthritis.

Medical records of dogs diagnosed with elbow lameness were reviewed. Inclusion criteria for this study were a complete clinical examination, a complete set of digital radiographs and a final diagnosis made by computed tomography or magnetic resonance imaging and arthroscopy. Changes of the medial humeral epicondyle were recorded and correlated with the radiographic osteoarthritis and final diagnosis.

Eighty of the 200 elbows showed changes of the medial humeral epicondyle. In 12 of these 80 elbows, changes of the medial humeral epicondyle were the only finding within the joint and these elbows were diagnosed with primary flexor enthesopathy. In the remaining 68 elbows, other concomitant elbow pathology was found. In those cases of concomitant epicondylar changes higher grades of osteoarthritis were recorded, while most elbows with primary flexor enthesopathy showed a lower grade of osteoarthritis.

Changes of the medial humeral epicondyle are often considered clinically unimportant and are regarded as an expression of osteoarthritis. This study showed the relatively frequent presence of medial humeral epicondylar changes of which the majority was considered concomitant to a primary elbow problem. When changes of the medial humeral epicondyle are the only pathologic finding (primary flexor enthesopathy) they should be considered as the cause of lameness and not as a sign of osteoarthritis.
Introduction

The elbow joint is a frequent origin of thoracic limb lameness in the dog. The most common disorder affecting the canine elbow joint is elbow dysplasia, a disorder which includes medial coronoid disease, ununited anconeal process, osteochondritis dissecans of the humeral condyle and incongruity (1-3). Diagnosis is based on the primary radiographic changes, or secondary subtrochlear sclerosis and osteoarthritis, often combined with the findings of computed tomography or arthroscopy (4-9).

Lesions of the medial humeral epicondyle and the attaching flexor muscles can equally cause elbow lameness, distension of the elbow joint, a limited range of motion and elbow pain (10, 11). These findings are comparable to the abnormalities present in joints affected by medial coronoid disease (4, 8, 12). Careful palpation of the medial side of the elbow joint, caudodistal to the medial humeral epicondyle, often reveals a firm well defined swelling, which is absent in joints with medial coronoid disease (10). Radiographic changes of the medial humeral epicondyle are often considered to be concomitant findings with limited clinical importance, although they have been mentioned as a cause of forelimb lameness in several, mostly older case reports (2, 11, 13-18). Radiographic changes at the medial humeral epicondyle were first described as an ununited medial epicondyle and were temporarily classified under the elbow dysplasia complex (2). This condition consisted of one or more loose ossified bodies either on the medial side of the elbow joint or distal to the medial humeral epicondyle (19). A failed fusion of the medial epicondyle ossification centre to the humerus was considered to be the cause. In later papers calcified bodies similar to those described as an ununited medial epicondyle were reported and different terms were used to describe these calcifications: dystrophic calcification of the flexor tendon origins, traumatic avulsion of the medial humeral epicondyle, medial humeral condylar osteochondritis dissecans and development of a preformed ossification centre (2, 13-18, 20, 21). Bony spur formation (enthesophytes) at the caudal edge of the medial humeral epicondyle was described as another radiographic finding at the medial humeral epicondyle. Spur formation as a sign of elbow disease has been less frequently described compared to calcified bodies near the medial humeral epicondyle (10, 15). A spur is considered to be an osseous outgrowth, extending from the skeleton into a tendon at its enthesis (22). Spur formation has been described concomitant with osteoarthritis, but it remains
unclear whether epicondylar spur formation is a manifestation of osteoarthritis or the primary problem causing osteoarthritis (15). In recent studies the term “flexor enthesopathy” was suggested referring to primary pathological changes within the flexor muscles and their attachment to the medial humeral epicondyle (the 'enthesis'). However, the precise cause of the disorder has not been reported (10, 11).

Lesions of the medial humeral epicondyle can occur as a single problem, but the presence of concurrent elbow problems has been described (16). Both for calcified bodies and spur formation, the challenge is to recognize the changes at the medial humeral epicondyle as a sign of primary flexor enthesopathy and to make the distinction between clinically important and unimportant lesions using additional diagnostic methods.

The purpose of this chapter was to make an inventory of the radiographic changes of the medial humeral epicondyle in order to determine the frequency of medial humeral epicondylar lesions and to evaluate the radiographic aspect as a primary finding or concomitant to other elbow diseases and its association with osteoarthritis.
Materials and methods

Consecutive files of dogs with elbow lameness diagnosed between 2008 and 2010 were reviewed. To be included in this study, a complete clinical examination and a complete set of digital radiographs (flexed and extended mediolateral and 15° oblique craniolateral-caudomedial projections) were required. The final diagnosis had to be confirmed either by computed tomography, magnetic resonance imaging or arthroscopy depending on the type of elbow problem. According to these requirements, the files of 117 dogs corresponding to 200 elbows were included in this study.

The radiographic images of these elbows were reviewed by the first author (EdB), a Board-certified ECVDI diplomate (HvB) and an experienced orthopedic surgeon (BVR) on a workstation, using the appropriate software. The medial humeral epicondyle and surrounding soft tissues were evaluated and findings were classified as irregular outline, calcified body, spur formation or both spur formation and a calcified body. Other abnormalities of the elbow joint were also recorded; these were mainly the presence of medial coronoid disease, osteochondritis dissecans, neoplasia as well as elbow osteoarthritis classified into 4 grades according to the criteria defined by the International Elbow Working Group (23).

After evaluation of the radiographs and the data obtained by further diagnostic methods (computed tomography, magnetic resonance imaging and arthroscopy), the elbow joints were divided into three groups: elbow joints without lesions of the medial humeral epicondyle, elbow joints with only lesions of the medial humeral epicondyle or surrounding soft tissues (“primary flexor enthesopathy”) and elbow joints with lesions of the medial humeral epicondyle associated with other elbow disorders (“concomitant flexor enthesopathy”).
Chapter 1: Radiographic findings of the medial humeral epicondyle

Results

Two hundred elbows met the criteria for inclusion in this study. In 120 elbows no radiographical changes of the medial humeral epicondyle were seen. In 80 elbows four types of radiographic changes at the medial humeral epicondyle were diagnosed: an irregular outline of the medial humeral epicondyle, spur formation, a calcified body and a combination of spur formation with a calcified body. In 12 of these, the radiographic changes of the medial humeral epicondyle were the only findings and these elbows were diagnosed with “primary flexor enthesopathy”. Ultrasonography, scintigraphy, computed tomography, magnetic resonance imaging and arthroscopy demonstrated pathology of the flexor muscles and their attachment to the medial humeral epicondyle in these elbows and excluded other elbow pathology. In the remaining 68 elbows, other concomitant pathology was found, including medial coronoid disease (71%), osteochondritis dissecans (7.5%), tumour (1%) and osteoarthritis (0.5%). These elbows were classified as “joints with concomitant medial humeral epicondylar lesions”.

Figure 1: Distribution of the final diagnosis in the 200 subsequent elbows and in the 80 elbows with medial humeral epicondylar changes. MCD: joints with medial coronoid disease, including medial compartment disease and joints with concomitant incongruity. FE: joints with primary flexor enthesopathy. OCD: joints with osteochondritis dissecans, with or without medial coronoid lesions and incongruity. UAP: joints with an ununited anconeal process and concomitant other lesions. IOHC: joints with incomplete ossification of the humeral condyle.
Of the total of 200 elbows, 170 elbows were radiographed because of the lameness examination and 30 elbows had postoperative follow-up radiographs. All radiographs belonged to different dogs. The 30 postoperative elbow radiographs showed medial humeral epicondylar lesions in 25 joints. Four of those had been diagnosed with primary flexor enthesopathy and showed similar lesions before surgery. The other 21 had been diagnosed with medial coronoid disease and did not show medial humeral epicondylar lesions pre-operatively. Of those follow-up radiographs, 5 were without medial humeral epicondylar lesions. Four had been diagnosed with medial coronoid disease and one with ununited anconeal process. The distribution of the final diagnosis within the total of 200 elbows and within the 80 elbows with changes of the medial humeral epicondyle is given in figure 1.

Four types of radiographic medial humeral epicondylar changes were diagnosed (Figure 2).

![Figure 2: Distribution of changes of the medial humeral epicondyle within 12 elbows with primary flexor enthesopathy (dark red) and 68 elbows with concomitant other elbow diseases (light red). (Values in parentheses indicate number of elbows).](image)
The two most frequent types were spur formation with or without the presence of a calcified body. Spur formation without a calcified body was the most frequent finding in joints with concomitant epicondylar lesions, while spur formation combined with a calcified body was the dominant type of lesion in joints with primary flexor enthesopathy (Figure 2-4). Calcified bodies in cases of primary flexor enthesopathy were always accompanied by spur formation (Figure 2 and 4).

![Figure 3: Lateromedial flexed projections illustrating a spur (white arrows) in a 7-year-old Border Collie with primary flexor enthesopathy (A) and a 9-year-old Belgian Shepherd Dog with concomitant flexor enthesopathy (B). A) The medial coronoid process is normal with mild osteoarthritis (black arrow) and mild subtrochlear sclerosis (white arrowhead). B) The medial coronoid process is unclearly delineated (black arrowhead) with osteoarthritis (black arrow) and subtrochlear sclerosis (white arrowhead).]

The third type was the presence of a calcified body without changes of the medial humeral epicondylar outline which was a less frequent finding exclusively seen as a concomitant epicondylar lesion (Figure 2 and 5).
Figure 4: Radiographic (extended and flexed mediolateral, and 15° oblique cranio-lateral-caudomedial projections) (A-C), CT (D-F) and arthroscopic (G-I) images of a 3-year-old Great Swiss Mountain Dog diagnosed with primary flexor enthesopathy. A-C) On radiography, primary flexor enthesopathy is visible as a calcified body (white arrowhead), spur formation (black arrow) and an irregular outline of the medial humeral epicondyle (white arrow). A mild degree of osteoarthritis is visible (black arrowhead). D-I) The absence of a medial coronoid process lesion in this dog is shown on the CT images and arthroscopic views. D-F) On the CT images (bone algorithm, D and E), the medial humeral epicondyle is sclerotic with an irregular outline (black arrow). A clear calcified body is visible within the flexor muscle group (white arrowhead). A small osteophyte is seen on the medial coronoid process (black arrowhead). After intravenous injection of contrast (2 mg/kg Iopromid®), clear contrast captation within the flexor muscles is visible (soft tissue algorithm, F) (black arrow). G-I) Arthroscopy shows a normal medial coronoid process (white arrow), an erosion at the attachment site of the flexor muscles to the medial humeral epicondyle (black arrow) and ruptured fibers of the flexor tendons near their attachment to the medial humeral epicondyle (white arrowhead).
Chapter 1: Radiographic findings of the medial humeral epicondyle

Figure 5: Flexed and extended medio-lateral and 15° oblique craniolateral-caudomedial projections (A-C) of an 8.7-year-old male Golden Retriever diagnosed with concomitant flexor enthesopathy. A-C) An unclearly delineated medial coronoid process (white arrowhead), osteophytes (black arrow) and subtrochlear sclerosis (black arrowhead) can be seen. A calcified body is visible caudal to the medial humeral epicondyle on the lateromedial projections (white arrow), but is not visible on the 15° oblique craniolateral-caudomedial projection.

The fourth type of lesion was an irregular outline of the medial humeral epicondyle, which appeared as a less frequent primary finding (Figure 2, 4 and 6). An irregular outline of the medial humeral epicondyle and spur formation were seen on the mediolateral flexed projection. Calcified bodies were diagnosed either on the lateromedial (flexed and extended) or 15° oblique craniolateral-caudomedial projection. The mediolateral projection revealed 13 elbows with a calcified body, the 15° oblique craniolateral-caudomedial projection showed 16 elbows with a calcified body. Only in 4 elbows a calcified body was visible on both the mediolateral and the 15° oblique craniolateral-caudomedial projections (Figure 7).
Figure 6: Radiographic images (lateromedial flexed projections) of an irregular outline of the medial humeral epicondyle in an elbow with primary flexor enthesopathy (A) and an elbow with a fragmented medial coronoid process (B). A) Four-year-old male Great Swiss Mountain Dog with a normal medial coronoid process, no osteoarthritis and no subtrochlear sclerosis. The irregular outline of the medial humeral epicondyle is marked by the white arrow. B) Ten-year-old male Bichon Frisé with an ill-defined medial coronoid process (black arrowhead), osteoarthritis (black arrows), subtrochlear sclerosis (white arrowhead) and an irregular medial humeral epicondyle (white arrow).

Figure 7: Lateromedial extended projection (A) and 15° oblique craniolateral-caudomedial projection (B) of a 7-year-old female Rottweiler with primary flexor enthesopathy illustrating a calcified body on both projections (white arrows).
The general osteoarthritis distribution for the 200 elbows was no osteoarthritis in 28%, grade 1 in 23%, grade 2 in 28% and grade 3 in 20%. The correlation between the degree of osteoarthritis and the presence as well as the absence of medial humeral epicondylar lesions is illustrated in figure 8.

**Figure 8:** Distribution of osteoarthritis (according to the guidelines of the International Elbow Working Group) within primary flexor enthesopathy (dark red column), elbows with concomitant epicondylar lesions (light red column) and elbows without medial humeral epicondylar changes (pink column). (Values in parentheses indicate number of elbows)
Discussion

Lesions of the medial humeral epicondyle have not been well documented in literature. Calcified bodies in this area are mostly considered to be a coincidental finding, although several mostly older papers reported those lesions as a cause of elbow lameness (2, 13-21). Osteophytosis or a ‘spur’ is usually interpreted as a sign of osteoarthritis (15, 23). Our study demonstrated that both types of medial humeral epicondylar lesions can be diagnosed frequently and can be clinically relevant in a limited but not insignificant number of cases. In order to define how frequently lesions of the medial humeral epicondyle are diagnosed and how these lesions are related to other primary elbow disorders and osteoarthritis, 200 files of dogs with elbow lameness were analysed.

The final diagnosis in our series of elbows reflected the generally accepted occurrence of elbow pathology. Although it would be ideal to have each diagnosis confirmed by a full work-up protocol including computed tomography or magnetic resonance imaging and a final diagnosis by arthroscopy, this was not always allowed by the owner. Lesions of the medial coronoid process were the most frequent diagnosis. Since computed tomography has a sensitivity and specificity of 88% and 85% respectively for the detection of fragments of the medial coronoid process, the distribution of lesions can be considered quite accurate, even in the absence of an arthroscopic confirmation (24, 25). Medial coronoid disease was the most frequent final diagnosis in our study, representing 77% within the total of 200 elbows and 60% within the 80 elbows with medial humeral epicondylar changes. This is consistent with other reports in the literature where fragmented medial coronoid process is also the most frequently diagnosed elbow disorder (26).

Osteochondritis dissecans and ununited anconeal process are other well-known elbow disorders but were diagnosed considerably less frequently in this study (7% and 2.5% respectively within the 200 elbows and 6% and 0% within the 80 elbows). This finding equally reflects the findings in other reports in the literature (1, 3, 8-9). In 5.5% of the 200 elbows in our study osteoarthritis was found without the presence of a primary disorder confirmed by computed tomography and in some cases arthroscopy. In half of the cases it involved the contralateral side in unilateral lameness.
The diagnosis of primary flexor enthesopathy was based on the radiographic findings and confirmed with additional diagnostic imaging techniques. Ultrasonography, scintigraphy, computed tomography, magnetic resonance imaging and arthroscopy demonstrated pathology of the flexor muscles and their attachment to the medial humeral epicondyle and excluded other elbow pathology. The abnormalities in these elbows were diagnosed as “primary flexor enthesopathy”, a term adopted from human medicine (22). It refers to pathological changes within the flexor muscles and their attachment to the medial humeral epicondyle. The enthesis is receiving great attention in human medicine, because enthesitis or enthesopathy is considered to be an important cause of locomotion problems, such as Tennis elbow and Golfer’s elbow (22). The percentage of elbows diagnosed with primary flexor enthesopathy in our study was 6%, which is comparable to the percentage of osteochondritis dissecans (7%) and higher than the percentage of ununited anconeal process (2.5%). This low but not negligible percentage draws attention to primary flexor enthesopathy as a cause of canine elbow lameness.

In our study, 40% of the elbows showed radiographic signs of medial humeral epicondylar changes. Logically, these changes were diagnosed at a high prevalence in joints with primary flexor enthesopathy, as pathology of this disorder is typically located in the area of the medial humeral epicondyle. However in two cases of primary flexor enthesopathy, the only finding was an irregular outline. This should make the clinician aware that the problem may also be present in the absence of clear radiographic changes. The highest percentage of medial humeral epicondylar changes was seen in elbows diagnosed with medial coronoid disease. In these elbows the epicondylar changes were considered concomitant to the main joint problem and treatment was only addressed to that problem. A different approach was described in another study of 26 joints (23 dogs) with calcified bodies near the medial humeral epicondyle (16). In that series, the calcification was surgically removed in 22 elbow joints. However 11 joints were simultaneously treated for medial coronoid disease, osteochondritis dissecans or ununited anconeal process (16).
Changes of the medial humeral epicondyle, observed in the present study, were divided in four types: an irregular outline of the medial humeral epicondyle, calcified bodies in the surrounding tissues, osteophytosis or ‘spur formation’ at the medial humeral epicondyle and a combination of spur formation with a calcified body. The less pronounced types were an irregular outline of the medial humeral epicondyle and spur formation. These changes can be considered as a logical finding in joints affected by any elbow disorder and may be explained as a sign of osteoarthritis. Spur formation was the most frequent finding in joints with concomitant flexor enthesopathy. However, in joints affected by primary flexor enthesopathy, this finding referred to the primary disorder. A more obvious finding was a calcified body diagnosed with or without a medial humeral epicondylar spur. In cases of primary flexor enthesopathy calcified bodies were always seen in combination with a spur. This may illustrate the active involvement of the attachment site (enthesis) of the flexor muscles in the disease process. This finding proves that a spur should not always be considered as a sign of osteoarthritis as is suggested in the guidelines of the International Elbow Working Group (23). A calcified body plus spur was the most frequent finding in joints with primary flexor enthesopathy. This is confirmed by the previous reports, all mentioning a calcification except two (2, 10, 13-18, 20, 21). According to these previous reports, calcified bodies are most frequently diagnosed on the 15° oblique craniolateral-caudomedial projection and can be missed on the mediolateral projection because of superimposition of the humerus and the radius (11, 16, 19). The results of our study show that a considerable number of calcified bodies were seen only on the mediolateral projection. Only 4 calcified bodies were visible on both projections, therefore both projections are necessary to diagnose calcified bodies in the area of the medial humeral epicondyle.

Since changes at the medial humeral epicondyle are often considered as a sign of osteoarthritis, the findings were compared with the degree of osteoarthritis. Our study shows that in the group of joints with concomitant epicondylar changes the highest percentage of medial humeral epicondylar lesions was found in joints with a high grade of osteoarthritis (grade 3). This was different for the group with primary flexor enthesopathy, where most changes were noted in joints with a lower grade of osteoarthritis (grade 0-2). In the group of elbows without medial humeral epicondylar changes lower grades of osteoarthritis (grade 0-2) were diagnosed more frequently than
in the group of elbows with concomitant flexor pathology. These findings suggest that concomitant epicondylar lesions are correlated to a high degree of osteoarthritis, while primary epicondylar changes are not. In the latter, the changes illustrate the primary problem involving the flexor muscles and their attachment, which can in return cause secondary osteoarthritis. On the other hand, not every elbow joint with osteoarthritis shows a radiographic change at the medial humeral epicondyle, suggesting that the medial humeral epicondyle is not a standard localization for osteoarthritis.

As previously mentioned, the percentage of medial coronoid disease as the definitive diagnosis in the total of 200 elbows was very high. The diagnosis of medial coronoid disease also included radiographs taken after arthroscopic removal of the coronoid lesion. The number of these postoperative radiographs was significantly higher in the group with concomitant medial humeral epicondylar changes (21 of 48 elbows) compared to the group without medial humeral epicondylar changes (4 of 94 elbows). Apparently arthroscopic treatment of a medial coronoid problem can induce the development of a spur or a calcified body near the medial humeral epicondyle. Considering this and knowing that osteoarthritis progresses after arthroscopic removal of medial coronoid disease this finding supports the theory that concomitant medial humeral epicondylar changes are related to the development of osteoarthritis (27-29).
Conclusion

Radiographic changes at the medial humeral epicondyle are a frequent finding in elbow disorders of which medial coronoid disease is the most important one. However, these changes may also be a sign of primary flexor enthesopathy. This specific area of the joint should be evaluated carefully to detect the lesions in the first place and to interpret them correctly in order to make the right treatment decision.
Footnote

a Eklin Medical Systems: Santa Clara, California, USA
b OsiriX Imaging Software; Pixmeo: Geneva, Switzerland
c Ultravist 300; Bayer Schering Pharma AG: Berlin, Germany
References


Chapter 2

DESCRIPTION OF PRIMARY AND CONCOMITANT FLEXOR ENTHESOPATHY OF THE CANINE ELBOW:
INTRODUCTION OF NEW TERMS
DESCRIPTION OF PRIMARY AND CONCOMITANT FLEXOR ENTHESOPATHY OF THE CANINE ELBOW:

INTRODUCTION OF NEW TERMS

Summary

The aim of this chapter was to report the characteristics of two forms of flexor enthesisopathy, primary and concomitant, based on different diagnostic techniques. Over a period of 3 years, a prospective study was performed on dogs admitted for the complaint of elbow lameness. Based on the radiographic findings a selection of dogs underwent a complete series of different imaging modalities. With each technique, pathology of the medial humeral epicondyle and the presence of other elbow disorders were recorded. All joints with signs of flexor pathology apparent with at least 3 techniques were selected. A distinction was made between primary flexor enthesisopathy and concomitant flexor enthesisopathy based on the absence or presence of other elbow disorders.

Twenty-three joints were diagnosed with primary flexor enthesisopathy and 20 joints with concomitant flexor enthesisopathy. In 43% of the joints with primary flexor enthesisopathy and in 75% of the joints with concomitant flexor enthesisopathy, pathology at the medial humeral epicondyle was demonstrated by all techniques. All joints with concomitant flexor enthesisopathy had a diagnosis of medial coronoid disease and/or osteochondritis dissecans.

Pathology in the region of the medial humeral epicondyle is a sign of flexor enthesisopathy. It may be present as the only sign in a joint with primary flexor enthesisopathy or concomitant with other elbow pathology. In both groups, flexor lesions can be demonstrated with different imaging techniques. The distinction between primary flexor enthesisopathy and concomitant flexor enthesisopathy is based on the presence or absence of other elbow pathology, mainly medial coronoid disease. Recognizing both forms is important for a correct treatment decision.
Introduction

Forelimb lameness in medium and large breed dogs is most frequently located in the elbow. The most common cause of elbow lameness is fragmented medial coronoid process, also named medial coronoid disease because of the varying types of lesions (1-4). Pathology in the region of the medial humeral epicondyle, radiographically demonstrated by a calcified body or spur formation, is less frequently regarded as a cause of elbow lameness (5, 6). Medial humeral epicondylar lesions were first reported as an ununited medial epicondyle in 1966 (7). Other reports followed describing similar lesions, which were named differently: dystrophic calcification of the flexor tendon origins, traumatic avulsion of the medial humeral epicondyle, medial humeral condylar osteochondritis dissecans and development of a preformed ossification centre (6, 8-13).

In a recent review article, we have suggested the term “flexor enthesopathy” to describe the different lesions in the region of the medial humeral epicondyle, since it involves both the flexor muscles and their attachment to the medial humeral epicondyle (5). It was adopted from human medicine where enthesopathies are frequently diagnosed in patients suffering from locomotion problems (14).

In a recent study of 200 elbow joints, a high prevalence of radiographic medial humeral epicondylar changes was demonstrated (15). In that series, 6% of the joints were diagnosed with primary flexor enthesopathy and 34% with concomitant flexor enthesopathy. In the primary form, flexor enthesopathy was considered as the only cause of lameness because no other elbow disorders were found. Another recent study reported eight dogs affected by primary flexor enthesopathy with clinically significant lesions in the region of the medial humeral epicondyle and the presence of minimal radiographic changes (4). It was stated that flexor enthesopathy is not always easy to recognize and that it should be considered as a differential diagnosis when the signs of medial coronoid disease are discrete or absent (4). In contrast to the primary form of flexor enthesopathy, the concomitant form was found simultaneously with other elbow disorders such as medial coronoid disease and osteochondritis dissecans (11, 15, 16). It is not yet determined to what extent these concomitant flexor lesions contribute to the lameness of the dog and if additional treatment is necessary (4, 15).

Our suggested treatment of both forms is different: in primary flexor enthesopathy joints are injected with 0.5-2 mg/kg bodyweight Methylprednisolonacetate or the
affected flexor muscle is surgically transected, similar to the treatment of medial epicondylitis in humans (4, 17). Our approach to the treatment of joints affected by concomitant flexor enthesopathy was limited to the surgical treatment of the primary elbow disorder by fragment or flap removal, without treatment of the flexor muscles (15). This is in contrast to a study including 26 elbow joints diagnosed with calcifications near the medial humeral epicondyle, which described the simultaneous treatment of medial coronoid disease, osteochondritis dissecans or ununited anconeal process with the surgical removal of the calcification in 11 joints (11).

In order to make a correct treatment decision, the detection of and differentiation between both forms of flexor enthesopathy should be possible. Especially in case of primary flexor enthesopathy, incorrect surgery of the medial coronoid process should be avoided. Since radiographic changes in the medial humeral epicondylar region can be minimal, inconclusive or even absent, additional imaging techniques are necessary (4).

The purpose of this chapter was to report the characteristics of two forms of flexor enthesopathy, primary and concomitant flexor enthesopathy, based on the combination of different diagnostic imaging modalities.
**Materials and methods**

Over a period of three years, a prospective study was performed on dogs admitted for the complaint of elbow lameness. The prospective study was conducted in accordance with the guidelines of the Animal Care Committee of the University of Ghent. Clinical examination included inspection on walk and trot and palpation of the elbow joint to define the range of motion, joint distension and pain reaction. Detailed scoring of lameness was done by assignment of grades on a scale from zero to ten, a system which has been described for equine lameness evaluation (18, 19). Joints were considered subclinically affected when lameness was not present and the joint was not painful. The radiographic findings were the basis for selection of the dogs: suspicion of changes of the medial humeral epicondyle with or without lesions of the medial coronoid process or painful elbows without clear radiographic changes. The selected dogs underwent the complete series of different diagnostic imaging techniques, including ultrasonography, scintigraphy (HiSPECT), computed tomography (CT), magnetic resonance imaging (MRI) and arthroscopy. With each technique the medial humeral epicondyle and the attaching flexor muscles of both elbows were evaluated and the presence of other elbow disorders was recorded.

Radiographic examination included three projections of both elbows, the mediolateral flexed and extended projection and 15° oblique craniolateral-caudomedial projection, which were performed while dogs were sedated.

Ultrasonography of the medial aspect of the elbow was performed while dogs were sedated and positioned in lateral recumbency with a linear, 10-15 MHz transducer. Grayscale images of the common flexor muscles were acquired in the longitudinal plane from the insertion on the medial humeral epicondyle to the musculotendinous junction.
Chapter 2: Primary and concomitant form of flexor enthesopathy

Figure 1: Radiographic images of primary (left) and concomitant (right) flexor enthesopathy demonstrating a spur (white arrow), irregular outline of the medial humeral epicondyle (white arrowhead) and calcified body (broad black arrow). Unclearly delineated medial coronoid process (small black arrow) and moderate subtrochlear sclerosis (black asterisk) is seen in concomitant flexor enthesopathy (right). Both signs can also be seen on the left elbow, but in a minimal degree.

Figure 2: Ultrasonographic images of primary (A-C) and concomitant (D-G) flexor enthesopathy. A) Loss of fiber structure of the flexor carpi ulnaris muscle (white arrow) with a normal appearance of the superficial digital flexor muscle (1). B) Moderate outward bowing (white arrow) of the flexor carpi ulnaris muscle (2). C) Large calcified body within the superficial digital flexor muscle (white arrow). D) Irregular outline of the medial humeral epicondyle (white arrowhead). E) Normal appearance of the superficial digital flexor muscle (1) and loss of fiber structure of the flexor carpi ulnaris muscle (white arrow). F) Outward bowing of the deep digital flexor muscle (white arrow) and normal appearance of the superficial digital flexor muscle (1). G) Large calcified body within the flexor muscles (white arrow).
Scintigraphy (HiSPECT) was performed with dogs under general anaesthesia two to three hours after the injection of hydroxymethane diphosphonate (mean 22 MBq/kg). A conventional triple head gamma camera, adapted with 3 multi-pinhole collimators (6 holes, 3 mm, resolution 2.4 mm) was used. The resolution of this system allows recognition of specific anatomical areas in the elbow (20). Dogs were positioned in lateral recumbency with the examined forelimb extended. Data were acquired for 20 minutes in step-and-shoot mode (10 steps, 36° angular step, 120 seconds per step, matrix 256x256). Activity in the medial humeral epicondyle and medial coronoid process regions was visually scored from one to three based on the still images and 3-dimensional movie.

Figure 3: Lateromedial HiSPECT images of primary flexor enthesopathy and concomitant flexor enthesopathy. A) Primary flexor enthesopathy characterized by focal increased bone tracer uptake in the region of the medial humeral epicondyle (grey arrow). B) Concomitant flexor enthesopathy showing increased bone tracer uptake in the region of the medial humeral epicondyle (grey arrow) and in the region of the medial coronoid process (black arrow). (H: Humerus; U: Ulna; R: Radius)
Computed tomography was performed with dogs under general anaesthesia with a single-slice helical CT scan using a bone and soft tissue reconstruction window for both elbows. Contiguous views (2 mm thick) were obtained from the proximal part of the ulna to 3 cm distal to the radial head, parallel to the humero-radial joint space. In the region of the radio-ulnar joint, 1 mm thick views were obtained (21). After this first scanning session, 2 ml/kg of 62.24g iopromid of contrast was injected intravenously (IV) and contiguous views were obtained.

Figure 4: CT and MRI images of concomitant flexor enthesopathy in a 3-year-and-9-month old Bernese Mountain Dog. A, B) Transverse CT images (bone algorithm) showing a large fragment of the medial coronoid process (broad black arrow), an irregularly outlined medial humeral epicondyle with a sclerotic and thickened cortex (broad white arrow) and a large calcified body within the flexor muscles (white arrowhead). Severe incongruency is visible (small black arrow). C) Transverse CT image (soft tissue algorithm), just distal to the level of the calcified body within the flexor muscles, after IV injection of contrast showing enhancement at the level of the flexor muscles (white arrow). D, E) Corresponding T1-weighted transverse MRI images, before (D) and after (E) IV injection of contrast, showing clear enhancement of the flexor carpi ulnaris muscle (white circle). F, G) T1-weighted sagittal MRI images before (F) and after (G) IV injection of contrast demonstrating clear enhancement (white arrows) of the flexor carpi ulnaris muscle (white asterisk). H) T2-weighted sagittal MRI image showing fluid between the flexor muscles (white arrow). (Lhc: lateral part of the humeral condyle, mhc: medial part of the humeral condyle)
Magnetic resonance imaging of the elbow was performed under general anaesthesia in transverse, sagittal and dorsal planes with a 0.2 Tesla, permanent magnet using T1-weighted, T2-weighted and STIR sequences (22). Afterwards 0.3 ml/kg of 0.5 mmol/ml Gadopentetate Dimeglumine of contrast medium was injected intravenously and T1-weighted sequences were repeated.

Figure 5: CT and MRI images of primary flexor enthesopathy in a 3,5-year-old male Rottweiler. A, B) Transverse CT images (bone algorithm) showing a normal medial coronoid process with an osteophyte (small white arrow), an irregularly outlined medial humeral epicondyle with a thickened and sclerotic cortex (broad white arrow) and a large calcified body within the flexor muscles (white arrowhead). C) Transverse CT image (soft tissue algorithm) after IV injection of contrast, showing clear enhancement within the flexor muscles (white circle). The calcified body within the flexor muscles can be noticed (white arrowhead). D, E) Corresponding transverse T1-weighted MRI images pre- (D) and post- (E) IV injection of contrast showing clear enhancement (white arrow) of the flexor carpi ulnaris muscle and around the hypointense calcified body (white arrowhead) within the flexor muscles. F, G) T1-weighted sagittal MRI images pre- (F) and post- (G) IV injection of contrast showing clear enhancement within the different flexor muscles (white arrows): Pronator teres muscle (1), flexor carpi radialis muscle (2) and superficial digital flexor muscle (3). H) T2-weighted sagittal MRI image showing fluid between the flexor muscles (white arrows). (Lhc: lateral part of the humeral condyle, mhc: medial part of the humeral condyle)
Arthroscopy was performed with a 2.4 mm arthroscope via a standard medial approach (23).

![Arthroscopic images of primary flexor enthesopathy (A-C) and concomitant flexor enthesopathy (D-F). A-C) A male, 5-year-old Bernese Mountain Dog with a normal appearance of the medial coronoid process (black asterisk), erosion (white arrow) and fibrillation (black arrow) at the attachment site of the flexor muscles. D-F) A male, 3-year-and-9-month old Bernese Mountain Dog with a fragmentation of the medial coronoid process (black asterisk), ruptured insertion of the flexor muscles (white arrowhead) and a thickened aspect of the flexor muscles (white arrow).](image)

A technique was considered positive for flexor enthesopathy when one or more pathologic changes were recorded. The pathologic changes for each technique are listed below.

**Radiography:** Presence of an irregular bony outline of the medial humeral epicondyle, spur formation, calcified body or a combination of these signs (Figure 1).

**Ultrasonography:** Presence of an abnormal fiber structure, abnormal tendon attachment to the medial humeral epicondyle, outward bowing of the flexor muscles,
calcified body, irregular outline of the medial humeral epicondyle, or a combination of these (Figure 2).

**Scintigraphy:** Focal increased bone tracer uptake in the region of the medial humeral epicondyle (Figure 3).

**CT:** Presence of an irregular, sclerotic or thickened cortex of the medial humeral epicondyle, thickened flexor muscles, contrast enhancement of flexor muscles, or a calcified body (Figure 4 and 5).

**MRI:** Presence of an irregular or thickened cortex of the medial humeral epicondyle, thickened flexor muscles, a hyperintense signal within the flexor muscles, contrast enhancement of the flexor muscles, or calcified body (Figure 4 and 5).

**Arthroscopy:** Ruptured or fibrillated insertion of the flexor muscles, degenerated tendinous tissue, local synovitis or erosion at the attachment site of the flexor muscles to the medial humeral epicondyle (Figure 6).

Furthermore the presence and absence of other elbow disorders, mainly medial coronoid disease and osteochondritis dissecans in this study, were evaluated by plain radiography, scintigraphy, CT and arthroscopy. Radiographic signs of osteoarthritis were determined according to the guidelines of the International Elbow Working Group (24).

To exclude doubtful cases, we decided to consider an elbow joint positive for flexor enthesisopathy when signs of flexor pathology were apparent with at least three techniques. All elbow joints with a final diagnosis of flexor enthesisopathy were selected for this descriptive study. A distinction was made between primary and concomitant forms of flexor enthesisopathy based on the absence or presence of other elbow disorders. Primary flexor enthesisopathy was diagnosed when flexor pathology was found with the exclusion of other elbow disorders based on the combination of the different imaging techniques. Concomitant flexor enthesisopathy was diagnosed when flexor lesions were found in the presence of other elbow disorders.
Results

Forty-three joints (26 dogs) were classified with flexor enthesopathy based on the clinical examination and diagnostic imaging techniques showing lesions indicative for flexor enthesopathy.

<table>
<thead>
<tr>
<th>Technique</th>
<th>Primary flexor enthesopathy</th>
<th>Concomitant flexor enthesopathy</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Clinical (17)</td>
<td>Subclinical (6)</td>
</tr>
<tr>
<td>Radiography</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Flexor pathology</td>
<td>15</td>
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</tr>
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<td>2</td>
</tr>
<tr>
<td>OCD</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>OCD + coronoid pathology</td>
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<td>0</td>
</tr>
<tr>
<td>Ultrasound</td>
<td></td>
<td></td>
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<tr>
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</tr>
<tr>
<td>Coronoid pathology</td>
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<td>3</td>
</tr>
<tr>
<td>OCD</td>
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<td>0</td>
</tr>
<tr>
<td>OCD+coronoid pathology</td>
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<td>0</td>
</tr>
<tr>
<td>Scintigraphy</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Flexor pathology</td>
<td>17</td>
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</tr>
<tr>
<td>Coronoid pathology</td>
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<td>0</td>
</tr>
<tr>
<td>OCD</td>
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<td>0</td>
</tr>
<tr>
<td>OCD+coronoid pathology</td>
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<td></td>
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<tr>
<td>Flexor pathology</td>
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<tr>
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<td>0</td>
</tr>
<tr>
<td>OCD + coronoid pathology</td>
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<td>0</td>
</tr>
<tr>
<td>MRI</td>
<td></td>
<td></td>
</tr>
<tr>
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</tr>
<tr>
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</tr>
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<td>OCD + coronoid pathology</td>
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</tr>
<tr>
<td>Arthroscopy</td>
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</tr>
<tr>
<td>Flexor pathology</td>
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<td>6</td>
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<tr>
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<tr>
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<td>0</td>
</tr>
<tr>
<td>OCD + coronoid pathology</td>
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<td>0</td>
</tr>
</tbody>
</table>

Table 1: Number of elbows affected clinically and subclinically by primary flexor enthesopathy and concomitant flexor enthesopathy showing flexor pathology for each technique as well as information about the medial coronoid process and the medial aspect of the humeral condyle. (OCD: Osteochondritis dissecans, values in parentheses indicate the number of elbow joints)
Primary flexor enthesopathy was diagnosed in 23 elbows, of which 6 elbows were considered subclinically affected since no signs of lameness and pain could be detected. Of these six elbows, four were the contralateral limb of dogs affected bilaterally by primary flexor enthesopathy and 2 joints were the contralateral limb of dogs affected by concomitant flexor enthesopathy on the lame limb. Concomitant flexor enthesopathy was diagnosed in the other 20 elbows. Fifteen joints had medial coronoid disease, of which five had been treated before. Five of the 20 joints were affected by osteochondritis dissecans, of which three in combination with medial coronoid disease. Three of the 20 concomitant elbows were the subclinical side of bilaterally affected dogs. Nine dogs were bilaterally affected by primary flexor enthesopathy and seven dogs were bilaterally affected by concomitant flexor enthesopathy.

The complete series of diagnostic imaging techniques was applied in all elbow joints. Table 1 illustrates the number of elbow joints showing flexor pathology for each technique and the diagnosis of medial coronoid pathology and osteochondritis dissecans detected on radiography, scintigraphy, CT and arthroscopy.

The distribution of osteoarthritis grades in the presence of primary and concomitant flexor enthesopathy is illustrated in table 2.

<table>
<thead>
<tr>
<th>Osteoarthritis (IEWG)</th>
<th>Primary flexor enthesopathy n=23</th>
<th>Concomitant flexor enthesopathy n=20</th>
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</thead>
<tbody>
<tr>
<td>Grade 0</td>
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<td>3</td>
</tr>
<tr>
<td>Grade 1</td>
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</tr>
<tr>
<td>Grade 3</td>
<td>0</td>
<td>8</td>
</tr>
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</table>

Table 2: Distribution of osteoarthritis grade in the presence of primary flexor enthesopathy and concomitant flexor enthesopathy. (n= number of elbow joints, IEWG: International Elbow Working Group)
The mean age of dogs with primary flexor enthesopathy was 60 months (7-92 months) and for concomitant flexor enthesopathy 57 months (7-104 months). In both groups only large breed dogs were represented (Table 3). Male dogs were more frequently affected than female dogs (Table 3).

<table>
<thead>
<tr>
<th>Breed (joints)</th>
<th>Primary flexor enthesopathy</th>
<th>Concomitant flexor enthesopathy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Great Swiss Mountain Dog</td>
<td>5 (2)</td>
<td>0</td>
</tr>
<tr>
<td>Labrador Retriever</td>
<td>3</td>
<td>5 (1)</td>
</tr>
<tr>
<td>Golden Retriever</td>
<td>2 (1)</td>
<td>1</td>
</tr>
<tr>
<td>Rottweiler</td>
<td>4 (2)</td>
<td>4 (2)</td>
</tr>
<tr>
<td>Newfoundlander</td>
<td>3 (1)</td>
<td>3 (1)</td>
</tr>
<tr>
<td>Border Collie</td>
<td>2 (1)</td>
<td>0</td>
</tr>
<tr>
<td>Bernese Mountain Dog</td>
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<td>2 (1)</td>
</tr>
<tr>
<td>Bouvier</td>
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<td>2 (1)</td>
</tr>
<tr>
<td>Swiss Shepherd Dog</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Dutch Partridge Dog</td>
<td>2 (1)</td>
<td>0</td>
</tr>
<tr>
<td>Mixed Breed</td>
<td>2 (1)</td>
<td>2 (1)</td>
</tr>
<tr>
<td>Gender (dogs)</td>
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</tr>
<tr>
<td>Male</td>
<td>10</td>
<td>7</td>
</tr>
<tr>
<td>Female</td>
<td>4</td>
<td>5</td>
</tr>
</tbody>
</table>

Table 3: Detailed information about breed and gender distribution for both flexor enthesopathy groups. (Values in parentheses represent the number of bilaterally affected dogs)

In 43% of the elbows diagnosed with primary flexor enthesopathy and in 75% of elbows diagnosed with concomitant flexor enthesopathy all 6 techniques showed pathology in the region of the medial humeral epicondyle and the attaching flexor muscles (Table 4). Five techniques demonstrated the presence of flexor pathology in 35% and 15% of elbows with primary and concomitant flexor enthesopathy (Table 4). The minority of joints with primary and concomitant flexor enthesopathy showed 4 or 3 positive techniques (Table 4). The minimal number of positive techniques was four out of six for primary flexor enthesopathy and three out of six for concomitant flexor enthesopathy (Table 4). The minimal number of three techniques positive for concomitant flexor
enthesopathy was found in two elbow joints, which were both subclinically affected. In both joints arthroscopy demonstrated clear flexor enthesisopathy lesions combined with pathology demonstrated by either ultrasonography, CT or scintigraphy. The minimal number of four out of six techniques positive for primary flexor enthesisopathy was found in 4 elbow joints, which were all subclinically affected. In those 4 joints a combination of scintigraphy, CT, MRI and arthroscopy demonstrated flexor pathology.

<table>
<thead>
<tr>
<th>6 techniques positive</th>
<th>5 techniques positive</th>
<th>4 techniques positive</th>
<th>3 techniques positive</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clin</td>
<td>Subclin</td>
<td>Clin</td>
<td>Subclin</td>
</tr>
<tr>
<td>Primary flexor enthesisopathy (n=23)</td>
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<td>6</td>
</tr>
<tr>
<td>Concomitant flexor enthesisopathy (n=20)</td>
<td>15</td>
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<td>2</td>
</tr>
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</table>

Table 4: Number of elbows affected clinically and subclinically by both forms of flexor enthesisopathy on which 6 or fewer techniques demonstrated flexor pathology. (n= number of elbow joints, Clin= clinically affected joints, Subclin= subclinically affected joints)
Discussion

This study describes two forms of flexor enthesopathy, which were diagnosed in dogs with elbow lameness. In the author’s opinion, the treatment of primary and concomitant flexor enthesopathy is different and therefore the distinction between both forms is important. It may be difficult to recognize either form, but diagnosis of primary flexor enthesopathy can be considered as the most critical one, compared to the diagnosis of concomitant flexor enthesopathy, since it may be confused with medial coronoid disease, leading to an incorrect treatment. Therefore this study was performed to enable the identification of both forms of the disorder. It was not the purpose of this study to compare all imaging techniques in detail for the distinction between primary and concomitant flexor enthesopathy or to recommend a diagnostic protocol.

Although primary and concomitant flexor enthesopathy may be considered as two different forms, age and gender distribution of both groups were similar. Dogs diagnosed with primary flexor enthesopathy had an age range of 7 months to 7.7 years, which is comparable to previous reports on medial epicondylar lesions, mentioning a range from 5 months to 7 years (7-13, 25). Dogs diagnosed with concomitant flexor enthesopathy had a mean age of 4.8 years (7 months to 8.7 years). The mean age is higher than would be expected for medial coronoid disease, which was the main primary disorder diagnosed in the concomitant group (1, 26). In this group however, a number of dogs was presented because of recurrent lameness several years after the initial treatment of the medial coronoid process lesion. Dogs with primary and concomitant flexor enthesopathy in our study as well as dogs affected by elbow dysplasia were mostly male dogs (65%), which indicates a gender predilection (1, 11, 27) (Table 3). Presumably the higher activity or higher weight of male dogs can explain this predilection. Primary and concomitant flexor enthesopathy were most frequently seen in the popular medium and large breed dogs, which was also mentioned in previous reports (11). However, it was remarkable that the Great Swiss Mountain Dog, a less common breed, was the most frequently represented breed within the primary flexor enthesopathy group (Table 3). The Labrador Retriever was the main breed affected by concomitant flexor enthesopathy (Table 3). This is not surprising, knowing
that the Labrador Retriever is a very popular breed and one of the most frequently affected by medial coronoid disease (26).

Primary flexor enthesopathy was diagnosed in 23 joints, since these joints showed flexor pathology and other primary elbow disorders were excluded based on the combination of the different imaging techniques.

In the group of concomitant flexor enthesopathy, flexor pathology was diagnosed in the presence of other elbow pathology, which was in this study medial coronoid disease except for two cases with osteochondritis dissecans and three cases with medial coronoid disease in combination with osteochondritis dissecans. The cause and clinical significance of these concomitant pathologic changes are not known. Five of the joints with concomitant flexor enthesopathy had previously been treated arthroscopically for medial coronoid disease. Similar to the findings in a previous study, these joints showed no signs of flexor enthesopathy before the initial treatment (15). Apparently, joints affected by medial coronoid disease can develop concomitant flexor enthesopathy after the arthroscopic treatment. Trauma caused by the arthroscopic intervention or increased inflammation induced by the lesions or the arthroscopic treatment may have caused the development of enthesopathy and local myopathy. However, in the authors’ experience this is not routinely observed. An ongoing study on the long-term clinical and radiographic follow-up with special attention to this condition should clarify this evolution. In cases of recurrent lameness after initial treatment of medial coronoid disease it is unclear whether the relapse can be explained by the medial coronoid problem or by the development of flexor enthesopathy.

The majority of dogs diagnosed with primary and concomitant flexor enthesopathy was bilaterally affected with flexor enthesopathy (respectively 65% and 58%). This suggests the influence of conformation, genetic predisposition or activity level of the dog. A comparable influence of occupational risk factors such as forceful activities, high force combined with high repetition and awkward postures on the prevalence of human medial epicondylitis has been described in several reports (28, 29).
Some joints showed signs of flexor enthesopathy without evidence of pain or lameness. Data on these subclinically affected joints were obtained during the prospective diagnostic study, which included both elbows of each selected dog. Therefore, changes at the origin of the flexor muscle at the medial epicondyle do not necessarily cause lameness. This corresponds with the general belief that calcified bodies near the medial humeral epicondyle are of no clinical importance (4, 5, 10, 13, 15). Further follow-up of these cases might clarify the meaning of these findings.

The motivation to perform arthroscopy in subclinically affected joints can be questioned. However, arthroscopy is a minimally invasive technique and when performed correctly there are no clinical consequences for the dog (30, 31). Moreover, the arthroscopic findings of subclinically affected joints can contribute to the understanding of the findings in clinically affected joints.

In order to distinguish both groups of elbows, six imaging techniques were applied: radiography, scintigraphy, ultrasonography, CT, MRI and arthroscopy. In literature, the radiographic findings of flexor enthesopathy lesions are mainly described as a calcified body and less frequently as spur formation (7-9, 11-13, 15, 25). A recent study described flexor enthesopathy in the presence of minimal radiographic changes (4). In the present study, an irregular outline of the medial humeral epicondyle, a calcified body and/or a spur were identified as the radiographic characteristics of both primary and concomitant flexor enthesopathy (Figure 1). Only in 16 joints of the 23 with primary flexor enthesopathy and 17 joints of the 20 with concomitant flexor enthesopathy there was radiographic evidence of flexor enthesopathy (Table 1). This means that radiography can be considered as a first screening method for the detection of flexor enthesopathy, but a relatively large number of cases may be missed. Additionally, radiography is often insufficient to diagnose discrete forms of elbow dysplasia and therefore the distinction between both forms of flexor enthesopathy based on the presence or absence of other elbow diseases –mainly medial coronoid disease- cannot be made (32, 33).
Bone scintigraphy may be used for the diagnosis of lameness when clinical or radiographic findings are inconclusive. The HiSPECT system is a refined scintigraphy technique, which enables a more detailed anatomical localization of pathology within the elbow joint (20). In the present study, all elbows with primary and concomitant flexor enthesopathy showed an increased uptake at the medial humeral epicondyle, suggesting that the HiSPECT bone scan is a very sensitive technique to diagnose flexor pathology (Table 1). In 7 of the 23 elbows diagnosed with primary flexor enthesopathy, activity was noted in the medial coronoid process region. It remains difficult to compare structural and functional imaging, since functional alterations precede structural changes. It is uncertain whether increased functional activity combined with normal structural imaging data reflects true pathological remodelling or whether it is merely a reflection of subclinical remodelling. Only follow-up investigations may provide an answer to this issue.

Ultrasonography showed a range of pathologic changes of the flexor muscles and their attachment comparable to the findings reported for medial epicondylitis in man (34) (Figure 2). Ultrasonography was positive for 14 out of 23 joints with primary flexor enthesopathy and for 18 out of 20 joints with concomitant lesions (Table 1). This means that pathology cannot always be visualized, but it is known that ultrasonography depends strongly on the experience of the user (22). In the present study, the examinations were performed by observers with a different level of experience. Therefore ultrasonography can be used to confirm a suspected flexor lesion, but absence of changes cannot exclude it and care should be taken to interpret the findings of less experienced imagers.

Magnetic resonance imaging is successfully used for the diagnosis of medial epicondylitis in man (35, 36). In our study, MRI demonstrated changes of the flexor muscles in 22 of the 23 elbows affected by primary flexor enthesopathy and in 18 of the 20 elbows with concomitant flexor enthesopathy, suggesting that MRI is indeed a sensitive technique for detecting flexor enthesopathy (Table 1). The false negative cases can be explained by the low resolution and detail obtained with the low field MRI system used in this study (37).
Computed tomography of the elbow is frequently used for the diagnostic work-up of medial coronoid disease (26). The results of our study suggest that CT is a sensitive technique for the detection of flexor pathology. Changes of the medial humeral epicondyle and of the flexor muscles were detected in 21 of the 23 joints with primary flexor enthesopathy and 18 of the 20 joints with concomitant lesions (Table 1).

Arthroscopy is routinely used to diagnose and treat medial coronoid disease (30, 38-40). A recent study demonstrated the use of arthroscopy in the evaluation of the attachment of the flexor muscles to the medial humeral epicondyle (4). When the enthesis is damaged the covering synovial membrane is consequently disrupted, allowing the arthroscopic visualization of the lesions. In all joints with primary and concomitant flexor enthesopathy, signs of a damaged enthesis were noted (Table 1). Arthroscopy can be considered a reliable technique for detecting flexor pathology.

The presence of elbow dysplasia was evaluated on plain radiographs, CT and arthroscopy. Radiographic signs of medial coronoid disease were present in 52% of the primary group and absent in 10% of the concomitant group (Table 1). It is known that evaluation of the medial coronoid process is difficult on plain radiographs because of superimposition (26). Furthermore, the radiographic absence of medial coronoid disease in the concomitant group illustrates the difficult differential diagnosis in the presence of minimal lesions (32, 33, 41, 42). CT demonstrated a normal medial coronoid process in all elbows with primary flexor enthesopathy and the presence of a medial coronoid lesion in all elbows, except for one joint diagnosed with concomitant flexor enthesopathy (Table 1). In this elbow joint CT did not demonstrate a fragment, which was seen during arthroscopy. Six joints affected by primary flexor enthesopathy showed a mild irregular aspect of the medial coronoid process arthroscopically, while CT did not demonstrate any subchondral lesions. However, the arthroscopic findings of the medial coronoid process were not typical for medial coronoid disease and the pathology of the flexor muscles seen with the different imaging modalities was more prominent compared to the findings of the medial coronoid process. Therefore these joints were diagnosed as primary flexor enthesopathy instead of concomitant flexor enthesopathy. Since this study used a single slice helical CT scan, which has lower resolution capabilities compared to multi-slice scans, some fissures might have been missed. This
limitation was counteracted by the subsequent arthroscopic inspection. However, even with the combination of both techniques some subtle medial coronoid process lesions may be difficult to diagnose.

The majority of elbows diagnosed with primary flexor enthesopathy and concomitant flexor enthesopathy showed lesions in the area of the medial humeral epicondyle and the attaching flexor muscles with 6 or 5 diagnostic techniques. In only 17% of the elbows with primary flexor enthesopathy and in 10% of the elbows with concomitant flexor enthesopathy four or less techniques were positive (Table 4). This can be explained by the presence of minimal lesions or operator dependent failure. The number of diagnostic techniques that showed positive for flexor enthesopathy in subclinical cases was lower when compared to the clinically affected joints, but no gross pathologic differences were observed (Table 1 and 4). Remarkable is that only scintigraphy and arthroscopy were able to detect all subclinical cases (Table 1). A possible explanation is that both techniques can demonstrate either early or subtle lesions.

In 56% of the joints with primary flexor enthesopathy, osteoarthritis was diagnosed. The presence of osteoarthritis in these joints can be considered either as a cause or a consequence of primary flexor enthesopathy. Therefore it may be questioned whether the flexor pathology in these joints is indeed primary. However, all other primary elbow disorders were excluded and none of these joints showed severe osteoarthritis or cartilage erosions, suggesting a primary degenerative joint disease. The presence of a spur at the medial humeral epicondyle is often described as a sign of osteoarthritis and is in that case not considered as a sign of primary flexor enthesopathy (4, 5, 15, 23). However, when a joint is affected by osteoarthritis, new bone formation can be found at several locations within the joint (4). In six joints with primary flexor enthesopathy from this study, spur formation was diagnosed without the presence of osteoarthritis. This confirms that a spur can be a solitary periosteal reaction caused by the flexor enthesopathy and should then not be seen as a sign of osteoarthritis. Severe osteoarthritis was diagnosed more frequently in joints affected by concomitant flexor enthesopathy. However, the difference was not significant, so the degree of osteoarthritis has only a limited value for the differentiation between primary and concomitant flexor enthesopathy (Table 2).
All changes suggesting flexor pathology shown with the different imaging techniques were similar for primary and concomitant flexor enthesopathy. In this study only the presence of pathology for each diagnostic technique was taken into account. The further elaboration of the specific findings can only be performed in more detailed studies, comparing the changes of each technique separately within joints affected by flexor enthesopathy, normal joints and joints affected with other pathologic conditions. In this study, the distinction between primary and concomitant flexor enthesopathy was based on the absence or presence of any other elbow disorder, mainly a lesion of the medial coronoid process, using radiography as a first screening method and scintigraphy, CT and arthroscopy to confirm the lesions. Further detailed studies may reveal specific pathologic changes of the flexor muscles and their attachment as other parameters to distinguish both forms.
Footnote

a Moderin 20 mg/ml; Pfizer A.H: Brussels, Belgium
b Eklin medical systems: Santa Clara, California, USA
c MyLab 30; Esaote: Firenze, Italy
d Triad; Trionix, USA
e GE prospeed: Milwaukee, Wisconsin, USA
f Ultravist 300; Bayer Schering Pharma AG: Berlin, Germany
g Airis Mate; Hitachi: Tokyo, Japan
h Magnevist; Bayer: Wayne, New York, USA
i Richard Wolf: Knittlingen, Germany
References


Chapter 3

RADIOGRAPHIC FEATURES OF PRIMARY AND CONCOMITANT FLEXOR ENTHESOPATHY IN THE CANINE ELBOW
RADIOGRAPHIC FEATURES OF PRIMARY AND CONCOMITANT FLEXOR ENTHESOPATHY IN THE CANINE ELBOW

Summary

Primary flexor enthesopathy is a recently recognized elbow disorder and should be considered in the differential diagnosis of elbow lameness. For treatment planning purposes, it is important to make a distinction between primary and concomitant forms of the disease. The purpose of this prospective study was to compare radiographic findings for dogs with primary flexor enthesopathy (n=17), concomitant flexor enthesopathy (n=24), elbow dysplasia (n=13) and normal dogs (n=7).

All dogs underwent a complete radiographic examination and each radiographic image was evaluated for the presence or absence of the following characteristics: irregular medial humeral epicondyle, spur and calcified body. Additionally, the presence or absence of other elbow disorders (medial coronoid disease, osteochondritis dissecans, ununited anconeal process, incongruity, subtrochlear sclerosis and osteoarthritis) was recorded. Radiographic characteristics of flexor enthesopathy were found in 86% of painful joints in the primary flexor enthesopathy group and in 100% of painful joints in the concomitant flexor enthesopathy group. Radiographic characteristics of flexor enthesopathy were not found in normal elbow and elbow dysplasia groups. Frequencies and details of individual radiographic characteristics did not differ between primary and concomitant flexor enthesopathy groups.

Findings support the use of radiography as a first screening method for the detection of flexor enthesopathy, but not as a technique for distinguishing primary versus concomitant forms.
Introduction

It is important to correctly identify the cause of elbow pain in affected dogs so that correct treatment decisions can be made. Flexor enthesopathy has been recently recognized as a cause of elbow pain in medium and large breed dogs and has been characterized radiographically as irregular margination of the medial humeral epicondyle with adjacent calcified bodies or a spur (1-3). These radiographic changes have also been described in some reports as coincidental or clinically unimportant findings (1-3). The disease has been described as primary when other causes of pathology in the elbow have been excluded (2, 3). Primary flexor enthesopathy has been identified in dogs with minimal radiographic changes (3). Radiographic changes of the medial humeral epicondyle have also been identified in dogs with medial coronoid disease and elbow incongruity (2-5). A recent study on radiographic changes of the medial humeral epicondyle in 200 elbows demonstrated a prevalence of 6% for primary flexor enthesopathy and 34% for concomitant flexor enthesopathy (2) (Section III, Chapter 1). This indicates the importance of medial humeral epicondylar lesions, but demonstrates as well that these lesions are often not the primary elbow problem and a distinction between both forms of medial humeral epicondylar lesions is necessary. A combination of different diagnostic techniques was described for the detection of flexor enthesopathy including radiography, ultrasonography, scintigraphy, computed tomography (CT), magnetic resonance imaging (MRI) and arthroscopy (1-3). Each imaging technique showed specific changes in case of flexor enthesopathy (3, 6). In a recent study a description of primary and concomitant flexor enthesopathy was given based on the presence of flexor pathology and presence or absence of other elbow disorders. However, a detailed analysis of the specific changes of each technique was not performed (6) (Section III, Chapter 2).

Radiography is the most common screening method used for suspected elbow disease in dogs (1). On the flexed mediolateral and 15° oblique craniolateral-caudomedial projections an irregular margination of the medial humeral epicondyle, a calcified body and spur formation can be evaluated (1, 2, 6). However, radiography is unable to detect soft tissue pathology of the flexor muscles (7). Furthermore, it is known that radiography is often insufficient to provide a definitive diagnosis of elbow dysplasia, and more particularly medial coronoid disease, the most important elbow disorder (8-10).
The purpose of this chapter was to compare radiographic characteristics in dogs with primary flexor enthesopathy, concomitant flexor enthesopathy, joints affected by other forms of elbow dysplasia and normal elbow joints. We hypothesized that 1) radiography would detect flexor enthesopathy in the majority of affected dogs; and 2) radiography would differentiate between primary and concomitant forms of flexor enthesopathy.
Materials and methods

Dogs

Fifty dogs (n=50) were prospectively recruited for the study. All dogs underwent a complete radiographic examination and nearly all dogs received additional ultrasonographic (n=48), scintigraphic (n=45), CT (n=50), MRI (n=49) and arthroscopic (n=50) examinations. The prospective study was conducted in accordance with the guidelines of the Animal Care Committee of the University of Ghent. Dogs were divided in four groups based on the following criteria.

**Group 1 (Primary flexor enthesopathy)** consisted of 17 client-owned dogs (29 elbow joints), aged between 7 and 92 months old (median 4.7 years). Eleven dogs were male, 6 were female. Dogs were included in this group when at least three of five other imaging modalities (ultrasonography, scintigraphy, CT, MRI, arthroscopy) demonstrated lesions consistent with flexor enthesopathy (3, 6). Ultrasonographic criteria included abnormal fiber structure of the flexor tendons, abnormal attachment and outward bowing of the flexor muscles, irregular margination of the medial humeral epicondyle, and/or focal acoustic shadowing within flexor muscles consistent with a calcified body. The scintigraphic (HiSPECT) criterion was increased radiopharmaceutical uptake in the area of the medial humeral epicondyle. Computed tomographic criteria included an irregular, sclerotic, thickened medial humeral epicondyle, thickened flexor muscles with contrast enhancement, and/or a focal area of increased attenuation within flexor muscles consistent with a calcified body. Magnetic resonance imaging criteria included an irregular, sclerotic medial humeral epicondyle, thickened flexor muscles with contrast uptake, and/or a focal area of low signal intensity within the muscle consistent with a calcified body. Arthroscopic criteria included a ruptured or fibrillated insertion of the flexor muscles, thickened remnants, fibrillation, local synovitis and/or local erosion near the insertion site. Dogs included in Group 1 also had no evidence of medial coronoid disease, osteochondritis dissecans, ununited anconeal process and incongruity based on CT and arthroscopy. Seven joints of this group were not painful clinically.

**Group 2 (Concomitant flexor enthesopathy)** consisted of 24 client-owned dogs (36 elbow joints), aged between 7 months and 8.7 years old (median 4.2 years). Seventeen dogs were male and 7 female. Dogs were included in this group when flexor enthesopathy lesions were identified in at least three other imaging modalities (based on criteria
described above) and the additional presence of medial coronoid disease (n=29), osteochondritis dissecans (n=3) and medial coronoid disease + osteochondritis dissecans (n=4) was identified with CT and arthroscopy (3). Eight joints had been treated arthroscopically for medial coronoid disease several years (1-6 years) before. Six joints of this group were not painful clinically.

<table>
<thead>
<tr>
<th>Breed</th>
<th>Primary flexor enthesopathy</th>
<th>Concomitant flexor enthesopathy</th>
<th>Elbow dysplasia</th>
<th>Normal joints</th>
</tr>
</thead>
<tbody>
<tr>
<td>Labrador Retriever</td>
<td>3 (1)</td>
<td>8 (3)</td>
<td>6 (2)</td>
<td>1 (0)</td>
</tr>
<tr>
<td>Great Swiss Mountain Dog</td>
<td>4 (3)</td>
<td>1 (0)</td>
<td>0</td>
<td>1 (0)</td>
</tr>
<tr>
<td>Bernese Mountain Dog</td>
<td>0</td>
<td>2 (2)</td>
<td>1 (0)</td>
<td>0</td>
</tr>
<tr>
<td>Rottweiler</td>
<td>3 (2)</td>
<td>2 (2)</td>
<td>0</td>
<td>1 (0)</td>
</tr>
<tr>
<td>Golden Retriever</td>
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<td>Mixed Breed</td>
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</tr>
<tr>
<td>Swiss Shepherd Dog</td>
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<td>1 (0)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Border Collie</td>
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<td>0</td>
<td>0</td>
</tr>
<tr>
<td>French Bull Dog</td>
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<td>0</td>
<td>1 (1)</td>
</tr>
<tr>
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<td>3 (2)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Saint Bernard Dog</td>
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<td>1 (0)</td>
<td>1 (0)</td>
<td>0</td>
</tr>
<tr>
<td>Dutch Partridge Dog</td>
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<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Bouvier</td>
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<td>1 (1)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Bullmastiff</td>
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<td>1 (1)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Shepherd Dog</td>
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<td>1 (1)</td>
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</tr>
<tr>
<td>Appenzeller</td>
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<td>0</td>
<td>1 (1)</td>
<td>0</td>
</tr>
<tr>
<td>English Cocker Spaniel</td>
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<td>0</td>
<td>1 (1)</td>
<td>0</td>
</tr>
<tr>
<td>Fox Hound</td>
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<td>0</td>
<td>0</td>
<td>2 (2)</td>
</tr>
</tbody>
</table>

Table 1: Breed distribution of 50 dogs with primary flexor enthesopathy, concomitant flexor enthesopathy, elbow dysplasia and normal joints. (n= total number of dogs, values in parentheses indicate number of bilaterally affected dogs)
Group 3 (Elbow dysplasia) consisted of 13 clinically affected client-owned dogs (18 elbow joints), aged between 10 months and 10.5 years old (median 2.9 years). Eight dogs were male and 5 were female. In all dogs flexor enthesopathy was excluded based on the previously described criteria and five imaging methods, and the presence of medial coronoid disease (n=18) was confirmed based on arthroscopy and at least one of four other imaging modalities (ultrasonography, scintigraphy, CT and MRI).

Group 4 (Control, normal joints) consisted of 2 laboratory-owned and 5 client-owned dogs with no clinical signs of elbow pain. The age was between 19 months and 126 months (median 5.4 years). This group consisted of 5 male dogs and 2 female dogs. For this group, 11 elbow joints were included in the analysis based on absence of elbow lesions using ultrasonography, scintigraphy, CT, MRI or arthroscopy.

The breed distribution for the 4 groups is summarized in table 1.

Radiographic examination and measurements

Radiography was performed under sedation, using acepromazine (0.01 mg/kg)\textsuperscript{a} with methadone (0.1 mg/kg)\textsuperscript{b} or medetomidine (28 μg/kg)\textsuperscript{c} intravenously. Digital images\textsuperscript{d} of three projections were taken of both elbow joints: a mediolateral projection in flexion and extension, and a 15° oblique cranialateral-caudomedial projection. Radiographic findings were recorded by consensus by the first author (EdB), a Board-certified ECVDI diplomat (HvB) and an experienced orthopaedic surgeon (BVR), who were all unaware of the group status. For all elbows and all projections, the presence or absence of the following radiographic characteristics of flexor enthesopathy were recorded:

- Irregular margination of the medial humeral epicondyle
- Spur formation on the medial humeral epicondyle
  - Size: small: <2 mm, medium: 2-4 mm, large: >4 mm
  - Shape: round or elongated
- Calcified body adjacent to the medial humeral epicondyle
  - Size: small: <3 mm, medium: 3-6 mm, large: >6 mm
  - Shape: round or elongated
  - Distance to the medial humeral epicondyle: close (<5 mm) or remote (≥5 mm)
Chapter 3: Radiographic findings of primary and concomitant flexor enthesisopathy

Radiographic characteristics of the medial coronoid process (normal, unclear delineation, abnormal shape, fragment), presence or absence of subtrochlear sclerosis, and presence or absence of osteoarthritis were also recorded. Severity of osteoarthritis was scored using 4 grades previously defined by the International Elbow Working Group (8, 9) (Table 2). On the 15° oblique craniolateral-caudomedial projection, the presence or absence of humeral condyle lesions (osteochondritis dissecans or irregularities) were recorded.

<table>
<thead>
<tr>
<th>Grade of osteoarthritis</th>
<th>Radiographic finding</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grade 0</td>
<td>No osteoarthritis visible</td>
</tr>
</tbody>
</table>
| Grade 1 (mild)          | Osteophytes <2 mm at one or more of the following sites:  
                          - on the proximal aspect of the anconeal process  
                          - on the cranioproximal edge of the radius  
                          - on the proximal edge of the medial coronoid process  
                          - on the proximal edge of the lateral epicondylar ridge  
                          - sclerosis in the area caudal to the distal end of the ulnar trochlear notch and to the proximal part of the radius |
| Grade 2 (moderate)      | Osteophytes 2-5 mm high at one or more locations as described for grade 1 |
| Grade 3 (severe)        | Osteophytes >5 mm high at one or more locations as described for grade 1 |

Table 2: Grading scale used for osteoarthritis, based on criteria defined by the International Elbow Working Group.

Statistical analysis

Statistical analysis was selected and performed by a statistical consultant and the first author (EdB). Fisher’s exact test was used to compare radiographic characteristics among the 4 groups of dogs. Statistical significance was based on a value of p<0.05.
Results

No statistically significant differences in frequencies and details of individual radiographic characteristics for flexor enthesopathy were identified among the 4 groups. Radiographic lesions of the medial humeral epicondyle were found in 86% of the painful elbow joints with primary flexor enthesopathy and in 100% of the painful elbow joints with concomitant flexor enthesopathy (Table 3). Radiography demonstrated flexor enthesopathy lesions in 2 of the 7 subclinically affected joints with primary flexor enthesopathy and none of the subclinically affected joints with concomitant flexor enthesopathy (Table 3). Radiographic characteristics of flexor enthesopathy were not found in normal elbows or those affected by elbow dysplasia.

Irregular margination of the medial humeral epicondyle

An irregular margination of the medial humeral epicondyle was found in 34% of joints with primary flexor enthesopathy and in 33% of joints with concomitant flexor enthesopathy (Table 3) (Figure 1A, 1B).

![Figure 1: Medio-lateral flexed radiographic projections of primary flexor enthesopathy (A) and concomitant flexor enthesopathy (B). A) An irregular margination of the medial humeral epicondyle (black arrow), a small spur (white arrow) and a large-sized, elongated calcified body (black arrowhead) with a moderate degree of subtrochlear sclerosis (white arrowhead). B) An irregular margination of the medial humeral epicondyle (black arrow) and a small-sized, rounded spur (white arrow). The medial coronoid process is not clearly delineated (small black arrow) and a moderate degree of subtrochlear sclerosis (white arrowhead) is visible.](image-url)
One subclinically affected joint of the primary flexor enthesopathy group and none of the subclinically affected joints of the concomitant flexor enthesopathy group had an irregular margination of the medial humeral epicondyle (Table 3). In the majority of joints with concomitant flexor enthesopathy (67%), the irregular margination was found in combination with severe (grade 3) osteoarthritis.

<table>
<thead>
<tr>
<th>Radiographic lesion</th>
<th>Primary flexor enthesopathy</th>
<th>Concomitant flexor enthesopathy</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Clinical n=22</td>
<td>Subclinical n=7</td>
</tr>
<tr>
<td>Epicondyle</td>
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<td></td>
</tr>
<tr>
<td>Irregular margination</td>
<td>9</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>18</td>
<td>1</td>
</tr>
<tr>
<td>Size</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;2 mm (small)</td>
<td>6</td>
<td>1</td>
</tr>
<tr>
<td>2 mm - 4 mm (medium)</td>
<td>8</td>
<td>0</td>
</tr>
<tr>
<td>&gt;4 mm (large)</td>
<td>4</td>
<td>0</td>
</tr>
<tr>
<td>Spur</td>
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<td></td>
</tr>
<tr>
<td>Total</td>
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<td>2</td>
</tr>
<tr>
<td>Size</td>
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<td></td>
</tr>
<tr>
<td>&lt;3 mm (small)</td>
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<td>0</td>
</tr>
<tr>
<td>3 mm - 6 mm (medium)</td>
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<td>1</td>
</tr>
<tr>
<td>&gt;6 mm (large)</td>
<td>6</td>
<td>1</td>
</tr>
<tr>
<td>Shape</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Round</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Elongated</td>
<td>8</td>
<td>2</td>
</tr>
<tr>
<td>Calcified body</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>6</td>
<td>2</td>
</tr>
<tr>
<td>Size</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Close (&lt;5 mm)</td>
<td>6</td>
<td>2</td>
</tr>
<tr>
<td>Remote (≥5 mm)</td>
<td>4</td>
<td>0</td>
</tr>
<tr>
<td>Shape</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Round</td>
<td>8</td>
<td>3</td>
</tr>
<tr>
<td>Elongated</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>Grade 0</td>
<td>8</td>
<td>1</td>
</tr>
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<td>Grade 1</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Grade 3</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 3: Number of elbows with medial humeral epicondylar lesions and grade of osteoarthritis in primary flexor enthesopathy and concomitant flexor enthesopathy groups, by clinical status. (n= total number of elbow joints)
Spur

Spur formation was detected in 19 elbows (66%) with primary flexor enthesopathy, of which 1 joint was subclinically affected, and in 24 elbows (67%) with concomitant flexor enthesopathy (Table 3). Most spurs were described as small or medium in both flexor enthesopathy groups (Figure 2). The shape of the spur was more commonly round in joints with concomitant flexor enthesopathy, while both round and elongated spurs were present with similar frequencies in the primary flexor enthesopathy group (Table 3) (Figure 2). A spur was frequently accompanied by moderate to severe osteoarthritis in joints with concomitant flexor enthesopathy. In 6 joints of the primary group, a spur was demonstrated in the absence of osteoarthritis.

Figure 2: Medio-lateral flexed radiographic projections illustrating medial humeral epicondylar spurs and other elbow lesions. A) A medium-sized, elongated spur (white arrow) is visible in an elbow affected by concomitant flexor enthesopathy. The medial coronoid process is less sharply delineated (black arrowhead) with a moderate degree of subtrochlear sclerosis (white arrowhead) and a moderate degree of osteoarthritis (black arrows). B) A large-sized, elongated spur (white arrow) is visible in an elbow affected by primary flexor enthesopathy. The medial coronoid process is normal with a moderate degree of osteoarthritis (black arrow). C) A medium-sized, rounded spur (white arrow) is seen in an elbow affected by concomitant flexor enthesopathy. The medial coronoid process is unclearly delineated (black arrowhead), with a moderate degree of osteoarthritis (black arrow) and a mild degree of subtrochlear sclerosis (white arrowhead).
Calcified body
A calcified body was observed in 12 elbows (41%) with primary flexor enthesopathy, of which 2 joints were subclinically affected, and in 12 elbows (33%) with concomitant flexor enthesopathy (Table 3). The calcified body was visible on mediolateral flexed, mediolateral extended and 15° craniolateral-caudomedial oblique projections in 3 joints of the primary flexor enthesopathy group and 2 joints of the concomitant flexor enthesopathy group. Most calcified bodies of the primary flexor enthesopathy group were visible on the mediolateral flexed and extended projections, while in the concomitant flexor enthesopathy group most calcified bodies were demonstrated on the 15° oblique craniolateral-caudomedial projection (Figure 3).

![Figure 3: Radiographs illustrating the appearance of a calcified body on the 15° oblique craniolateral-caudomedial projection. A) Elbow joint affected by elbow dysplasia with concomitant flexor enthesopathy with a medium-sized, rounded and large-sized, elongated calcified body located medially to the joint space (white arrows). B) Elbow affected by primary flexor enthesopathy with a large-sized, elongated calcified body visible disto-medial to the joint space (white arrow).]
Most calcified bodies in both flexor enthesopathy groups were large (>4 mm) with elongated shape (Table 3) (Figure 4). The calcified body was remote from the medial humeral epicondyle (≥5 mm) in 4 joints with primary flexor enthesopathy and in 1 joint with concomitant flexor enthesopathy. The majority of calcified bodies in joints with concomitant flexor enthesopathy were related to severe osteoarthritis, while most calcified bodies in primary flexor enthesopathy were seen in combination with moderate osteoarthritis. One joint with concomitant flexor enthesopathy and two joints with primary flexor enthesopathy had grade 0 osteoarthritis (Figure 4). Nearly all calcified bodies of both flexor enthesopathy groups occurred with spur formation (Figure 4).

Figure 4: Medio-lateral flexed radiographic projections illustrating calcified bodies and other elbow lesions. A) Elbow with primary flexor enthesopathy demonstrating a large-sized, elongated calcified body located adjacent to the medial humeral epicondyle (white arrow) and a small-sized, rounded spur (black arrow) without osteoarthritis. B) Elbow with concomitant flexor enthesopathy demonstrating one large-sized, elongated calcified body located adjacent to the medial humeral epicondyle, one small-sized, elongated calcified body located further away from the medial epicondyle (white arrows) and a medium-sized, rounded spur (black arrow). The medial coronoid process is unclearly delineated (black arrowhead) with a moderate degree of subtrochlear sclerosis (white arrowhead) and osteoarthritis (small black arrow). C) Elbow with concomitant flexor enthesopathy showing a medium-sized, rounded spur (black arrow) in combination with a large-sized, elongated calcified body located adjacent to the medial humeral epicondyle (white arrow). The medial coronoid process is less sharply delineated (black arrowhead) with a moderate degree of subtrochlear sclerosis (white arrowhead) and no osteoarthritis.
Chapter 3: Radiographic findings of primary and concomitant flexor enthesopathy

Radiographic signs of medial coronoid disease

Subtrochlear sclerosis was frequently observed in groups 1, 2 and 3 (Table 4). In 62% of the elbows with primary flexor enthesopathy, in 70% of the joints with concomitant flexor enthesopathy and in 61% of the dysplastic elbows, the medial coronoid process was described as having an unclear delineation or abnormal shape. For all normal elbow joints, a normal appearance of the medial coronoid process was recorded (Table 4). Osteochondritis dissecans was present in 25% of the joints affected by concomitant flexor enthesopathy. An irregular outline of the medial part of the humeral condyle was seen in 24% of the joints with primary flexor enthesopathy, in 39% of the elbow joints affected by concomitant flexor enthesopathy and in 11% of the joints diagnosed with elbow dysplasia (Table 4).

<table>
<thead>
<tr>
<th>Radiographic lesion</th>
<th>Primary flexor enthesopathy (29)</th>
<th>Concomitant flexor enthesopathy (36)</th>
<th>Elbow dysplasia (18)</th>
<th>Normal elbows (11)</th>
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<tbody>
<tr>
<td>Subtrochlear sclerosis</td>
<td></td>
<td></td>
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<td></td>
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<tr>
<td>Yes</td>
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<td>21</td>
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<tr>
<td>Suspicious</td>
<td>7</td>
<td>10</td>
<td>6</td>
<td>0</td>
</tr>
<tr>
<td>Medial coronoid process</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unclear delineation</td>
<td>15</td>
<td>14</td>
<td>8</td>
<td>0</td>
</tr>
<tr>
<td>Abnormal shape</td>
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<td>16</td>
<td>3</td>
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</tr>
<tr>
<td>Fragment</td>
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<td>0</td>
</tr>
<tr>
<td>Medial part humeral condyle</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Irregular outline</td>
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<td>14</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>OCD</td>
<td>0</td>
<td>9</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

Table 4: Number of elbows with other radiographic lesions in primary flexor enthesopathy, concomitant flexor enthesopathy, elbow dysplasia and normal elbows. (OCD: Osteochondritis dissecans, values in parentheses indicate total number of elbow joints)
Discussion

This study identified strengths and limitations of radiography for detecting flexor enthesopathy and making a distinction between primary versus concomitant forms of flexor enthesopathy in dogs. Since an irregular margination of the medial humeral epicondyle, a calcified body and/or a spur were present in both flexor enthesopathy groups and absent in normal elbows and elbows with dysplasia, these features can be regarded as the radiographic indications for both primary and concomitant flexor enthesopathy.

In 19 of the 22 (86%) clinically affected joints of the primary flexor enthesopathy group and in all clinically affected joints of the concomitant flexor enthesopathy group, radiography demonstrated medial humeral epicondylar changes. Thus radiography can be considered a sensitive technique to detect flexor enthesopathy. On the other hand, 3 clinically affected joints of the primary flexor enthesopathy group did not show radiographic changes. This finding indicates that clinically affected joints with primary flexor enthesopathy may not always be detected on radiography, and that the use of additional imaging techniques may be needed.

Radiographic signs of flexor enthesopathy were observed in 2 of the 7 subclinically affected joints with primary flexor enthesopathy and in none of the subclinically affected joints with concomitant flexor enthesopathy. This indicates that subclinical flexor pathology is likely to be missed on radiography. In these cases, the diagnosis of flexor enthesopathy was based on ultrasonographic, scintigraphic, CT, MRI and arthroscopic findings. The absence of radiographic changes can be explained by the fact that in the early stage only the soft tissues are affected. Our observation of primary flexor enthesopathy in 7 subclinically affected elbows supported a previous study indicating that flexor pathology in some dogs may be clinically insignificant (1).

Spur formation was the most frequent radiographic finding in our study and was diagnosed in two thirds of the joints in both flexor enthesopathy groups. This finding is not consistent with previous reports that mention a calcified body as the most important finding (4, 5, 11-16). Spur formation has been described as a sign of osteoarthritis instead of a sign of flexor enthesopathy, but joints of the primary flexor
enthesopathy group in our study showed a spur in combination with mild or no osteoarthritis (8). Moderate and severe degrees of osteoarthritis were observed most frequently in elbows affected by concomitant flexor enthesopathy. It is possible that severe and chronic forms of medial coronoid process disease are predisposing factors for the development of concomitant flexor enthesopathy. We identified no significant differences in the size and shape of spurs for primary flexor enthesopathy and concomitant flexor enthesopathy groups. However, our findings indicated that presence of a spur in the absence of osteoarthritis and medial coronoid process lesions justifies the use of further imaging to confirm primary flexor enthesopathy.

A calcified body is the most frequently described radiographic sign of flexor enthesopathy in previous reports (4, 5, 11-16). In our study this lesion was identified in 41% of the joints with primary flexor enthesopathy and 33% of the joints with concomitant flexor enthesopathy. A calcified body in concomitant flexor enthesopathy elbows was frequently observed with severe osteoarthritis. In primary flexor enthesopathy elbows, a calcified body was mostly observed with moderate osteoarthritis. Based on size, shape or localization of the calcified body, no significant differences between primary flexor enthesopathy and concomitant flexor enthesopathy were found. The combination of a spur and calcified body occurred frequently for both groups of flexor enthesopathy dogs.

An irregular margination of the medial humeral epicondyle was found in one third of the joints in both groups of flexor enthesopathy dogs and was frequently combined with other flexor enthesopathy lesions. Irregular margination of the medial humeral epicondyle has also been reported as a radiographic sign of osteoarthritis and other elbow disorders (1-3). However, when a joint is affected by osteoarthritis, osteophytes should also be present at other locations (3). In our study, some joints of both flexor enthesopathy groups showed an irregular medial humeral epicondyle without other signs of osteoarthritis. Therefore this characteristic in the absence of other signs of osteoarthritis should be regarded as a specific sign of flexor enthesopathy. In most joints of the concomitant flexor enthesopathy group, irregular margination of the medial humeral epicondyle was found in combination with severe osteoarthritis, while most joints with primary flexor enthesopathy had an irregular medial humeral epicondyle.
with mild osteoarthritis. The frequency of irregular margination of the medial humeral epicondyle was similar for both groups of flexor enthesopathy. Therefore this feature cannot be used to distinguish between forms of flexor enthesopathy.

The majority of elbows with concomitant flexor enthesopathy (81%) had a diagnosis of medial coronoid disease. Also in 62% of the joints affected by primary flexor enthesopathy, radiographic characteristics of medial coronoid disease (subtrochlear sclerosis and an unclear delineation of the medial coronoid process) were recorded. However, additional CT and arthroscopic examination excluded medial coronoid disease in these joints with primary flexor enthesopathy. Previous studies have also reported that subtle radiographic changes are not reliable for diagnosing medial coronoid disease (17, 18).

An irregular outline of the medial part of the humeral condyle was found in 24% of joints in the primary flexor enthesopathy group. These irregularities were minimal in all but one dog and corresponding CT and arthroscopic findings were also minimal. Since the enthesis (the attachment site of the flexor muscles at the medial humeral epicondyle) is damaged in joints affected by flexor enthesopathy, the covering synovial membrane may be consequently disrupted and irregularities of the medial part of the humeral condyle may occur (3).

In conclusion, results of this study support our first hypothesis that radiography is a good first screening method for the detection of flexor enthesopathy in clinically affected elbows. However, some cases of primary flexor enthesopathy also occurred with minimal or absent radiographic signs. Study results rejected our second hypothesis that radiography would be able to differentiate between primary flexor enthesopathy and the concomitant form. Therefore use of additional imaging modalities is justified to confirm the diagnosis of flexor flexor enthesopathy and make a distinction between the two forms of flexor enthesopathy in dogs with elbow pain.
Acknowledgments

The authors thank Prof. Dr. J. DeWulf for his help with the statistical analyses.

Footnote

a Placivet; Codifar: Wommelgem, Belgium
b Mephenon; Denolin: Brussels, Belgium
c Domitor; Pfizer Animal Health: Brussels, Belgium
d DDR; Eklin: Santa Cruz, California, USA
e SPSS statistics; IBM: Armonk, New York, USA
References


Chapter 3: Radiographic findings of primary and concomitant flexor enthesopathy


Chapter 4

ULTRASONOGRAPHIC FINDINGS OF PRIMARY AND CONCOMITANT FLEXOR ENTHESOPATHY IN DOGS
ULTRASONOGRAPHIC FINDINGS OF PRIMARY AND CONCOMITANT FLEXOR ENTHESOPATHY IN DOGS

Adapted from: de Bakker E, Saunders JH, Beaumlin Y, Van der Vekens E, Gielen I, Van Ryssen B. Ultrasonographic findings of primary and concomitant flexor enthesisopathy in dogs. Veterinary Radiology and Ultrasound 2012; in revision.
Chapter 4: Ultrasonographic findings of primary and concomitant flexor enthesopathy

Summary

Flexor enthesopathy is a disorder of the canine elbow and can occur as a primary or concomitant lesion. Clinical signs are often unspecific and with radiography 15% of the cases may be missed. Additionally radiography is unable to differentiate primary from concomitant flexor enthesopathy, which is necessary for the correct treatment. Therefore the possibility of ultrasonography to diagnose flexor enthesopathy and to distinguish primary from concomitant forms of flexor enthesopathy was examined. Dogs with primary flexor enthesopathy (n=17), concomitant flexor enthesopathy (n=24), elbow dysplasia (n=12) and normal dogs (n=6) were prospectively studied. On all dogs, an ultrasonographic examination was performed of the medial part of the elbow joint and for each joint the presence or absence of pathologic changes consistent with flexor enthesopathy was studied.

Ultrasonographic signs of flexor pathology were found in 82% of the clinically affected joints with primary flexor enthesopathy and in 87% of the clinically affected joints with concomitant flexor enthesopathy, but also in 25% of the joints with elbow dysplasia. An abnormal attachment and irregular medial humeral epicondyle were the most frequent findings in both flexor enthesopathy groups, illustrating the problem at the enthesis. The ultrasonographic findings were not significantly different for both forms. Flexor pathology was not found in normal elbows.

Although ultrasonography demonstrated specific lesions of the flexor muscles and their attachment in joints of both flexor enthesopathy groups, the diagnosis of flexor enthesopathy was missed in 15% of the clinical cases. Moreover, some flexor enthesopathy characteristics were also observed in joints without flexor enthesopathy. Since the lesions were similar in both groups of flexor enthesopathy, a distinction between the primary and the concomitant form could not be made. These conclusions illustrate the need for multiple diagnostic techniques to obtain a definitive diagnosis.
Introduction

The elbow joint is a frequent localization for thoracic limb lameness in medium and large breed dogs. The most important disorder affecting the canine elbow joint is elbow dysplasia, which includes medial coronoid disease, ununited anconeal process, osteochondritis dissecans of the humeral condyle and incongruity (1-3). Recently flexor enthesopathy, a disorder which is radiographically characterized by a calcified body or spur, is reported as another cause of elbow lameness (4-11). A recent study of 200 elbows demonstrated the presence of medial humeral epicondylar lesions in a considerable number of affected elbows (40%) (4) (Section III, Chapter 1). Although these lesions often occur in the presence of elbow dysplasia and may therefore be considered as a sign of osteoarthritis, they were the only abnormal findings in a small percentage of cases (6%) and thus considered as primary lesions causing elbow lameness (4). Concomitant flexor enthesopathy was diagnosed in 34% of the cases, in which medial coronoid disease was the most frequent primary joint problem (4). Primary flexor enthesopathy most likely represents an overuse problem, similar to medial epicondylitis (Golfer’s elbow) in man, while concomitant flexor enthesopathy is mostly seen in cases of a severe or chronic pathologic process in the joint (4, 5, 12-14). The identification of both forms of flexor enthesopathy is necessary because of a different treatment approach: in case of primary flexor enthesopathy an intra-articular injection with 0.5-2 mg/kg bodyweight Methylprednisolonacetate\(^a\) can be given or the flexor muscles can be surgically transected (5, 15). The authors’ current treatment approach of concomitant flexor enthesopathy is the surgical removal of the fragment and/or flap related to the elbow dysplasia without treatment of the flexor muscles (4, 8, 11, 13).

The first screening method for flexor enthesopathy is the radiographic appearance of the medial humeral epicondyle and the presence of a calcified body (4-6, 13). However, a recent study demonstrated that 15% of cases might be missed because of absence of radiographic changes at the medial humeral epicondyle and the attaching flexor muscles (16) (Section III, Chapter 3). Furthermore, radiography is unable to differentiate between primary and concomitant flexor enthesopathy, which necessitates the use of other diagnostic modalities (13, 16).
Ultrasonography is considered a reliable, noninvasive, widely available and inexpensive imaging technique and can provide both an anatomic and functional assessment of the flexor muscles and their attachment (17). In man, ultrasonography is, as well as magnetic resonance imaging, part of the diagnostic work-up of medial and lateral epicondylitis (6, 17, 18). Ultrasonography is used for diagnosing medial epicondylitis in the majority of the patients, allowing MRI to be reserved for patients with symptoms whose ultrasonographic findings are normal (19). A previous study on flexor enthesopathy in dogs demonstrated that ultrasonography could be used to confirm a suspected flexor lesion although the absence of lesions could not exclude it. However, a detailed analysis of the specific ultrasonographic features was not performed (13) (Section III, Chapter 2).

The aim of this chapter was to describe the ultrasonographic features of primary and concomitant forms of flexor enthesopathy in dogs. We hypothesized that 1) ultrasonography would detect specific characteristics for flexor enthesopathy similar to medial epicondylitis in man; and 2) ultrasonography would reveal more obvious or different lesions in primary flexor enthesopathy versus the concomitant form.
Materials and methods

Forty-eight dogs (n=48; 90 elbow joints) were prospectively studied, in accordance with the guidelines of the Animal Care Committee of the Ghent University. All dogs, except for the normal control dogs, were presented with thoracic limb lameness at the Ghent University Veterinary Clinic. All dogs underwent a complete ultrasonographic examination and received additional radiographic (n=48), scintigraphic (HiSPECT) (n=45), computed tomographic (n=48), magnetic resonance imaging (n=48) and arthroscopic (n=48) examinations for diagnostic purposes, as well as to obtain the criteria to characterize the dogs (13, 16).

Group 1 (Primary flexor enthesopathy) consisted of 17 client-owned dogs (29 elbow joints) aged between 7 months and 7.7 years old (median 4.7 years). Eleven dogs were male, 6 were female. Twenty-two elbow joints were clinically affected, 7 were clinically not apparent, since no signs of elbow pain or lameness were found. Therefore these 7 joints were considered subclinically affected. Dogs were included in this group when at least three of the five imaging modalities demonstrated lesions consistent with flexor enthesopathy and excluded medial coronoid disease, osteochondritis dissecans, ununited anconeal process and incongruity based on CT and arthroscopy (5, 13, 16).

Group 2 (Concomitant flexor enthesopathy) contained 24 client-owned dogs (36 elbows). The age of this group was between 7 months and 8.7 years (median 4.2 years). Seventeen dogs were male and 7 dogs were female. Thirty joints were clinically affected, 6 joints of this group were subclinically affected. Dogs were included in this group when flexor enthesopathy lesions were identified with at least 3 imaging modalities and the additional presence of medial coronoid disease (n=29), osteochondritis dissecans (n=3) and medial coronoid disease + osteochondritis dissecans (n=4) was confirmed with CT and arthroscopy (5, 13, 16).

Group 3 (Elbow dysplasia) consisted of 12 client-owned dogs (16 elbow joints), all clinically affected. The age was between 10 months and 10.5 years (median 2.4 years). Eight dogs were male and 4 were female. In all dogs flexor enthesopathy was excluded based on five imaging methods, and the presence of other elbow disorders was confirmed based on arthroscopy and at least one of 4 other imaging modalities.

Group 4 (control, normal joints) consisted of 2 laboratory-owned and 4 client-owned dogs. The age of this group was between 60 months and 126 months (median 6.5 years).
This group consisted of 4 male dogs and 2 female dogs. For this group, 9 elbow joints were included in analysis based on absence of elbow lesions using radiography, scintigraphy, CT, MRI or arthroscopy.

The breed distribution for the 4 groups is illustrated in table 1.

<table>
<thead>
<tr>
<th>Breed</th>
<th>Primary flexor enthesisopathy n=17</th>
<th>Concomitant flexor enthesisopathy n=24</th>
<th>Elbow dysplasia n=12</th>
<th>Normal joints n=6</th>
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<tbody>
<tr>
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<td>1 (0)</td>
<td>0</td>
</tr>
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<td>1 (0)</td>
</tr>
<tr>
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<td>2 (0)</td>
<td>1 (1)</td>
</tr>
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<td>0</td>
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<td>0</td>
</tr>
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<td>0</td>
</tr>
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<td>0</td>
</tr>
<tr>
<td>Dutch Partridge Dog</td>
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<td>0</td>
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<td>Bouvier des Flandres</td>
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</tr>
<tr>
<td>Bullmastiff</td>
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</tr>
<tr>
<td>Fox Hound</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>2 (2)</td>
</tr>
</tbody>
</table>

Table 1: Breed distribution for primary flexor enthesisopathy, concomitant flexor enthesisopathy, elbow dysplasia and normal joints. (n= total number of dogs, values in parentheses indicate the number of bilaterally affected dogs).
For the ultrasonographic examination, the dogs were sedated using acepromazine (0.01 mg/kg)\textsuperscript{b} and methadone (0.1 mg/kg)\textsuperscript{c} or medetomidine (28 μg/kg)\textsuperscript{d} intravenously. They were positioned in lateral recumbency with the examined elbow close to the table and extended. The elbow joint was approached medially with a linear 10–15 MHz transducer\textsuperscript{e} following a standardized protocol. In order to avoid anisotropy, the transducer was placed parallel to the tendons and muscles in the longitudinal plane (17). On longitudinal scanning the superficial digital flexor muscle, the deep digital flexor muscle and the flexor carpi ulnaris muscle were visualized from cranial to caudal (Figure 1). Images of these flexor muscles were acquired from the proximal insertion on the medial humeral epicondyle to the musculotendinous junction. The medial humeral epicondyle appeared as an intensely hyperechoic linear echo with distal acoustic shadowing (20) (Figure 1).

Figure 1: Longitudinal ultrasonographic image illustrating a normal appearance of the superficial digital flexor muscle (1), deep digital flexor muscle (2) and flexor carpi ulnaris muscle (3). The medial humeral epicondyle shows a normal, smooth outline (grey arrows). The superficial digital flexor muscle (1) shows a normal muscle belly (white asterisk) with a short tendinous attachment to the medial humeral epicondyle. The superficial digital flexor muscle (2) and flexor carpi ulnaris muscle (3) show a straight proximodistal path with a normal fiber pattern and a normal attachment to the medial humeral epicondyle.
Bilateral examinations were performed in the same manner. The ultrasonographic examination was performed by three different operators. The static images of each elbow joint were evaluated on a medical imaging computer program by a Board-certified ECVDI diplomate (YB). Each image was examined for the presence or absence of following abnormalities of the flexor muscles and their attachment to the medial humeral epicondyle: loss of fiber structure (<25% (mild), 25-50% (moderate), >50% (severe)), abnormal attachment to the medial humeral epicondyle (anechoic/hypoechoic, heterogenous), outward bowing (mild, moderate, severe), presence of a calcified body (<10 mm (small), >10 mm (large)) and irregularity of the medial humeral epicondyle (mild, moderate, severe). Outward bowing was considered when the flexor muscles had lost their straight proximodistal path. Irregular outline of the medial humeral epicondyle was defined as loss of the normal smooth hyperechoic bony surface of the medial humeral epicondyle. A calcified body appeared as a focal acoustic shadowing within the flexor muscles.

Statistical analysis was selected and performed by a statistical consultant and the first author (EdB). Fisher’s exact test was used to compare the ultrasonographic characteristics among the four groups of dogs. Statistical significance was established at a value of p<0.05.
Section III: Results

Results

In 82% of the clinically affected elbow joints with primary flexor enthesopathy, in 87% of the clinically affected elbow joints with concomitant flexor enthesopathy and in 25% of the joints with elbow dysplasia, ultrasonographic abnormalities of the flexor muscles and their attachment to the medial humeral epicondyle were found (Table 2). Flexor muscle pathology was seen in 2 of the 7 subclinically affected joints with primary flexor enthesopathy and in 2 of the 6 subclinically affected joints with concomitant flexor enthesopathy (Table 2). Flexor pathology was not found in normal elbows. The distribution of the ultrasonographic flexor lesions for both groups of flexor enthesopathy is illustrated in figure 2.

![Ultrasonographic abnormalities of the flexor muscles and their attachment to the medial humeral epicondyle compared between joints affected by primary flexor enthesopathy and joints affected by concomitant flexor enthesopathy.](image)

In 20 elbow joints affected by primary flexor enthesopathy, in 28 elbow joints affected by concomitant flexor enthesopathy and in 4 elbow joints with elbow dysplasia the superficial digital flexor muscle, the deep digital flexor muscle and/or the flexor carpi ulnaris muscle were affected (Table 3). In 75% of the joints with concomitant flexor enthesopathy, the flexor carpi ulnaris muscle was affected compared to 52% of the joints with primary flexor enthesopathy (Table 3).
### Ultrasonographic findings of primary and concomitant flexor enthesopathy

#### Chapter 4: Ultrasonographic findings of primary and concomitant flexor enthesopathy

<table>
<thead>
<tr>
<th>Ultrasonographic lesion</th>
<th>Primary flexor enthesopathy</th>
<th>Concomitant flexor enthesopathy</th>
<th>Elbow dysplasia</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Clinical ( n=22 )</td>
<td>Subclinical ( n=7 )</td>
<td>Clinical ( n=30 )</td>
</tr>
<tr>
<td>Loss of fiber structure</td>
<td>&lt;25% (mild)</td>
<td>5</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>25%-50% (moderate)</td>
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<td>0</td>
</tr>
<tr>
<td></td>
<td>&gt;50% (severe)</td>
<td>8</td>
<td>0</td>
</tr>
<tr>
<td>Attachment</td>
<td>Anechoic/hypoechoic</td>
<td>6</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Heterogenous</td>
<td>11</td>
<td>2</td>
</tr>
<tr>
<td>Outward bowing</td>
<td>Mild</td>
<td>5</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Moderate</td>
<td>10</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Severe</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Calcified body</td>
<td>&lt;10 mm</td>
<td>5</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>&gt;10 mm</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Medial humeral epicondyle</td>
<td>Mild irregular</td>
<td>9</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Moderate irregular</td>
<td>5</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Severe irregular</td>
<td>2</td>
<td>0</td>
</tr>
</tbody>
</table>

Table 2: Number of elbows with ultrasonographic characteristics of flexor enthesopathy for primary flexor enthesopathy, concomitant flexor enthesopathy and elbow dysplasia, by clinical status. (n= total number of elbow joints)

<table>
<thead>
<tr>
<th>Primary flexor enthesopathy</th>
<th>Concomitant flexor enthesopathy</th>
<th>Elbow dysplasia</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Clinical (22)</td>
<td>Subclinical (7)</td>
</tr>
<tr>
<td>Superficial digital flexor muscle</td>
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<td>2</td>
</tr>
<tr>
<td>Deep digital flexor muscle</td>
<td>16</td>
<td>2</td>
</tr>
<tr>
<td>Flexor carpi ulnaris muscle</td>
<td>14</td>
<td>1</td>
</tr>
<tr>
<td>All 3 muscles</td>
<td>6</td>
<td>1</td>
</tr>
</tbody>
</table>

Table 3: Number of elbow joints with one or more affected flexor muscles for primary flexor enthesopathy, concomitant flexor enthesopathy and elbow dysplasia. (Values in parentheses represent total number of elbow joints)
**Loss of fiber structure**

Loss of fiber structure was observed in 18 elbows with primary flexor enthesopathy (62%) and in 24 elbows with concomitant flexor enthesopathy (67%). Mild and severe loss of fiber structure (<25%, >50%) were mostly seen in joints with primary flexor enthesopathy, while mild and moderate loss of fiber structure (<50%) were mostly seen in joints with concomitant flexor enthesopathy (Figure 3) (Table 2). However, no statistically significant differences were found. Mild and moderate loss of fiber structure was found in 3 elbows with elbow dysplasia (19%) (Table 2).

![Figure 3: Loss of fiber structure seen ultrasonographically in an elbow with primary flexor enthesopathy (A) and in an elbow with concomitant flexor enthesopathy (B) with corresponding radiographic images. A) More than 50% (severe) loss of fiber structure is seen in the deep digital flexor muscle (white arrow) and in the flexor carpi ulnaris muscle (white arrowhead). On the mediolateral flexed projection a medium-sized, rounded spur is visible (small white arrow). B) Loss of fiber structure between 25% and 50% (moderate) is seen in the flexor carpi ulnaris muscle (white arrow). The mediolateral flexed projection shows a small-sized, rounded spur (small white arrow), a small calcified body located adjacent to the medial humeral epicondyle (small black arrow), a moderate degree of subtrochlear sclerosis (black arrowhead) and osteoarthritis (white arrowheads).]
Abnormal attachment

An abnormal attachment was found in 19 elbows with primary flexor enthesopathy (66%) and in 27 elbows with concomitant flexor enthesopathy (75%). A heterogenous attachment of the flexor muscles to the medial humeral epicondyle was a frequent finding in elbows with primary flexor enthesopathy. In a significantly higher number of elbow joints of the concomitant flexor enthesopathy group, an anechogenic/hypoechogenic attachment was demonstrated (Figure 4). In 3 joints with elbow dysplasia (19%) an anechogenic/hypoechogenic attachment was found (Table 2).

Figure 4: Abnormal attachment of the flexor muscles to the medial humeral epicondyle seen ultrasonographically with corresponding radiographs. A) Heterogenous attachment of the flexor carpi ulnaris muscle (3) to the medial humeral epicondyle (broad white arrow) in an elbow affected by primary flexor enthesopathy. The superficial digital flexor muscle (1) shows a normal appearance and the deep digital flexor muscle (2) shows loss of fiber tissue (small white arrow). The mediolateral flexed radiographic image shows a medium-sized, elongated spur (small white arrow) with an irregularly outlined medial humeral epicondyle (broad white arrow). B) The flexor carpi ulnaris muscle (3) shows an anechogenic/hypoechogenic attachment (white arrow) in an elbow joint affected by concomitant flexor enthesopathy. The deep digital flexor muscle (2) and the superficial digital flexor muscles (1) show a normal appearance. The mediolateral flexed radiographic projection demonstrates a very small spur (small white arrow) with an abnormally shaped medial coronoid process (black arrowhead) and severe osteoarthritis (black arrows).
Outward bowing
Outward bowing was found in 18 elbows with primary flexor enthesopathy (62%) and in 25 elbows with concomitant flexor enthesopathy (69%). A moderate degree of outward bowing was a frequent finding in both flexor enthesopathy groups (Figure 5) (Table 2). No significant differences were found between both groups of flexor enthesopathy. A mild degree of outward bowing was observed in 2 elbows with elbow dysplasia (13%) (Table 2).

Figure 5: Ultrasonographic image illustrating outward bowing with the corresponding radiographic image in an elbow affected by concomitant flexor enthesopathy. A severe degree of outward bowing of the deep digital flexor muscle (2) is visible (white arrow). On the mediolateral flexed radiographic projection a small-sized, elongated spur (small black arrow) with a large-sized, elongated calcified body (small white arrow) is visible. The medial coronoid process is ill defined (black arrowhead) with a severe degree of subtrochlear sclerosis (white arrowhead) and osteoarthritis (broad white arrows).
Calcified body

A calcified body was present in 5 elbows of the primary flexor enthesopathy group (17%) and in 15 elbows of the concomitant flexor enthesopathy group (42%) (Table 2). Small-sized calcified bodies were more frequently seen in joints affected by concomitant flexor enthesopathy compared to joints of the primary flexor enthesopathy group (respectively 36% versus 17%), although not significantly different (Figure 6) (Table 2).

Figure 6: A large (>10 mm) (A) and small (<10 mm) (B) calcified body visible on ultrasonography with the corresponding radiographic images. A) A large-sized calcified body (white arrow) within the superficial digital flexor muscle in an elbow affected by primary flexor enthesopathy. The mediolateral flexed radiographic projection shows a small-sized, rounded spur (black arrow) with a large-sized, elongated calcified body (white arrow). B) A small-sized calcified body (white arrow, 1) within the superficial digital flexor muscle (1) in an elbow affected by concomitant flexor enthesopathy. The deep digital flexor muscle (2) and the flexor carpi ulnaris muscle (3) are also visible. On the mediolateral flexed radiographic projection a small-sized, rounded spur (small black arrow) and a small-sized, elongated calcified body (small white arrow) are visible. A moderate degree of subtrochlear sclerosis (white arrowhead) with osteoarthritis (broad white arrows) can be seen.
Irregular outline of the medial humeral epicondyle

An irregular delineation of the medial humeral epicondyle was demonstrated in 18 elbows with primary flexor enthesopathy (62%) and in 28 elbows with concomitant flexor enthesopathy (78%). A mild irregularity was often seen in elbows with primary flexor enthesopathy, while a moderate irregular medial humeral epicondyle was a frequent finding for elbows with concomitant flexor enthesopathy (Figure 7). No significant differences were noticed between both groups of flexor enthesopathy. A mild irregular medial humeral epicondyle was found in 2 elbows with elbow dysplasia (13%) (Table 2).

Figure 7: Ultrasonographic image of an irregular outline of the medial humeral epicondyle with the corresponding radiographic image of an elbow affected by concomitant flexor enthesopathy. Severely irregular outline of the medial humeral epicondyle (white arrows) is seen along the attachment of the superficial digital flexor muscle (1), deep digital flexor muscle (2) and flexor carpi ulnaris muscle (3). The corresponding mediolateral flexed radiographic image demonstrates a small-sized, rounded spur (black arrow), an unclearly delineated medial coronoid process (black arrowhead) and a mild degree of subtrochlear sclerosis (white arrowhead) and osteoarthritis (white arrow).
Discussion

The ultrasonographic findings of flexor enthesopathy in dogs have not been described previously. To be certain that the changes of the medial humeral epicondyle and the attaching flexor muscles found ultrasonographically in this study were related to flexor enthesopathy, normal joints and joints affected by elbow dysplasia were included as control groups. An irregular outline of the medial humeral epicondyle, loss of fiber structure, abnormal attachment, outward bowing and calcified body were clearly detected in joints of both flexor enthesopathy groups. With the exception of calcified bodies, these features were also found in joints with elbow dysplasia, although less pronounced. Therefore, ultrasonographic changes of the flexor muscles and their attachment to the medial humeral epicondyle need to be interpreted carefully and should be correlated to the results of other imaging modalities.

Ultrasonography in the diagnosis of human medial epicondylitis has been described as a valuable diagnostic technique, with a detection rate of 95.2% (17). However, in our study ultrasonography detected flexor muscle abnormalities in only 82% of the clinically affected joints with primary flexor enthesopathy and in 87% of the clinically affected joints with concomitant flexor enthesopathy. A possible explanation is that some lesions are discrete and therefore difficult to visualize on ultrasonography. In addition, ultrasonography demonstrated changes of the flexor muscles and their attachment to the medial humeral epicondyle in 25% of the joints with elbow dysplasia, which makes it a less specific technique as well. The lower sensitivity and specificity can also be explained by the fact that ultrasonography is a dynamic diagnostic method with operator dependency (17). Several operators with different levels of experience performed the ultrasonographic examinations in our study. From the dynamic ultrasonographic images in the proposed study, the most informative static sonograms were selected and reviewed by an experienced ultrasonographer (YB; Board-certified ECVDI diplomate). For this person it was difficult to evaluate the ultrasonographic findings using static images instead of real-time imaging. The real time aspect of ultrasonography is a limitation in a clinical study in contrast to the other imaging techniques, which easily allow the evaluation of the images at a different moment. This
Section III: Results

is considered as a limitation of this study, although it reflects the same situation when the technique would be applied in practice.

When the ultrasonographic findings of flexor enthesopathy in dogs are compared to those reported in ultrasonographic studies of medial epicondylitis in man similarities and differences can be noted. Medial epicondylitis in man has been described as a primary condition and to the authors’ knowledge a concomitant form has never been reported (15, 21, 22). In contrast, this study and previous reports demonstrated that flexor enthesopathy in dogs is more frequently seen in the concomitant form than in the primary form (4, 13). The flexor carpi radialis and the pronator teres are the most frequently affected muscles in human medial epicondylitis, while in dogs the superficial digital flexor muscle and the flexor carpi ulnaris muscle are mostly affected. This can be explained by the different motion of the human elbow mostly throwing and swinging (15). A focal echogenic abnormality is the most common ultrasonographic finding in human medial epicondylitis (17). This corresponds well with the loss of fiber structure seen frequently in our dogs with flexor enthesopathy. This loss of fiber structure can be explained, similar to the focal echogenic abnormalities in human medial epicondylitis, as an area of collagen degeneration and intrasubstance tendon rupture, which can fill up with reparative granulation tissue (23). Cortical irregularities are also frequently seen in both human medial epicondylitis and flexor enthesopathy in dogs (17). The presence of these irregularities can be explained as bony outgrowths that extend from the skeleton into the soft tissue of a tendon (22). These enthesophytes represent a skeletal response to high tensile forces within a tendon (22). Intratendinous calcifications are a less frequent finding in both medial epicondylitis in man and flexor enthesopathy in dogs (17). The presence of a calcified body can be considered part of the tendon degeneration process. In man, several stages of medial epicondylitis have been described (15). In the early stages of medial epicondylitis, inflammatory or synovitic characteristics may be visible. In later stages, results of microtearing can be seen characterized by tendon degeneration often accompanied by the presence of calcified bodies (15).
In order to investigate the possibility of ultrasonography to differentiate between both forms of flexor enthesopathy, the ultrasonographic findings were scored in detail using different gradations of severity and size (Table 2). Some differences in flexor pathology between both groups of flexor enthesopathy could be noted, but only one was statistically significant. This anechoic/hypoechoic attachment was seen in a significantly higher number of joints with concomitant flexor enthesopathy. However joints affected by concomitant flexor enthesopathy often demonstrated the combination of an anechoic attachment with severe outward bowing. The presence of severe outward bowing can lead to the inability to scan the tendon perpendicularly, which results in an anechoic appearance of the tendon. This artefact is called anisotropism (17). Therefore the value of an anechoic/hypoechoic attachment for the distinction between both forms of flexor enthesopathy can be questioned. Despite the observed minor differences in severity and size of each type of flexor lesion it was not possible to formulate a method to distinguish primary and concomitant forms of flexor enthesopathy with ultrasonography.

Seven joints affected by primary flexor enthesopathy and six joints affected by concomitant flexor enthesopathy were subclinically affected and were the contralateral side in bilaterally affected elbow joints with unilateral lameness. Data of these subclinically affected joints were obtained during the prospective diagnostic study, which included both elbows of each selected dog. The number of subclinically affected joints showing flexor pathology on ultrasonography was low for both groups of flexor enthesopathy (Table 2). Hence most subclinical flexor pathology was missed on ultrasonography. A possible explanation is that subclinically affected joints show less obvious changes of the flexor muscles, and are therefore not visible on ultrasonography. In these cases the diagnosis of flexor enthesopathy was based on additional radiographic, scintigraphic, CT, MRI and arthroscopic examinations. The detection of pathology found with other diagnostic techniques does not necessarily have a clinical meaning, since this was mainly seen in subclinically affected joints. In contrast, ultrasonography detected flexor pathology in 4 subclinically affected joints: 2 with primary flexor enthesopathy and 2 with concomitant flexor enthesopathy. Some flexor enthesopathy lesions may be present before the onset of lameness or without the development of a clinical problem, which is consistent with literature describing medial humeral epicondylar lesions as coincidental findings (6, 24).
The overall conclusion of this study is that different types of lesions indicating flexor enthesopathy can be demonstrated with ultrasonography, which supports our first hypothesis. However, not all findings are specific for flexor enthesopathy since they were also observed in joints without flexor enthesopathy. Minor differences between primary and concomitant flexor enthesopathy were observed, but the number, extent and severity of the changes were not significantly different. Therefore we reject our second hypothesis. Similar as radiography, ultrasonography can be considered a screening method to detect flexor enthesopathy in the majority of the cases. The considerable number of false negative and false positive diagnoses added to the inability of ultrasonography to distinguish both forms of flexor pathology necessitates the use of other imaging techniques to detect and differentiate primary flexor enthesopathy.
Chapter 4: Ultrasonographic findings of primary and concomitant flexor enthesopathy

Acknowledgments

The authors thank Prof. Dr. J. DeWulf for his help with the statistical analyses.

Footnotes

a Moderin 20 mg/ml; Pfizer A.H.: Louvain La Neuve, Belgium
b Placivet; Codifar: Wommelgem, Belgium
c Mephenon; Denolin: Brussels, Belgium
d Domitor; Pfizer Animal Health: Brussels, Belgium
e Mylab 30; Esaote: Firenze, Italy
f OsiriX Imaging software; Pixmeo: Geneva, Switzerland
g SPSS statistics; IBM: Armonk, New York, USA
References


Chapter 5

THE USE OF PLANAR BONE SCINTIGRAPHY AND HISPECT FOR PRIMARY AND CONCOMITANT FLEXOR ENTHESOPATHY IN THE CANINE ELBOW
THE USE OF PLANAR BONE SCINTIGRAPHY AND HISPECT FOR PRIMARY AND CONCOMITANT FLEXOR ENTHESOPATHY IN THE CANINE ELBOW

**Summary**

The aim of this study was to investigate the possibilities and limitations of planar bone scintigraphy and HiSPECT to diagnose flexor enthesopathy and to distinguish primary flexor enthesopathy from the concomitant form. A prospective study of 46 dogs with primary flexor enthesopathy, concomitant flexor enthesopathy, elbow dysplasia and normal elbows was performed. All dogs underwent a planar bone scan and a HiSPECT scan. The obtained images were visually scored for increased tracer uptake in the medial humeral epicondylar region and medial coronoid process area using a score from 1-3.

Planar bone scan demonstrated increased tracer uptake in all diseased elbow joints, except for one. HiSPECT demonstrated increased tracer uptake of the medial humeral epicondyle in nearly all clinically affected joints with primary flexor enthesopathy and concomitant flexor enthesopathy. Additional uptake of the medial coronoid process was recorded in all clinically affected joints with concomitant flexor enthesopathy and in 6/18 clinically affected joints with primary flexor enthesopathy. No significant difference in intensity of the uptake was noticed.

Planar bone scan allows attributing lameness to the elbow joint in cases of primary flexor enthesopathy with minimal or even absent radiographic changes. The more detailed HiSPECT enables to localize pathology within the elbow joint and is a sensitive technique to detect flexor enthesopathy. However HiSPECT is insufficient to distinguish primary from concomitant flexor enthesopathy, which necessitates the use of multiple diagnostic modalities.
Introduction

Front leg lameness in medium and large breed dogs is often localized in the elbow joint. The most common cause is elbow dysplasia, which includes medial coronoid disease, osteochondritis dissecans of the medial aspect of the humeral condyle, ununited anconeal process and incongruity (1-3). Recently, flexor enthesopathy has been added to the list of elbow disorders (4-6). Most flexor enthesopathy lesions occur concomitant with elbow dysplasia, while a small percentage represents the only primary lesion within the joint (5). Clinical signs of primary flexor enthesopathy are mostly unspecific: elbow lameness, distension of the elbow joint, limited range of motion and elbow pain (4, 5, 7). In some cases a firm well-defined swelling in the caudodistal region of the medial humeral epicondyle can be palpated (4). Radiographic signs of flexor enthesopathy are spur formation on the medial humeral epicondyle and soft tissue calcified body in the area of the medial humeral epicondyle (5-12). A recent study has demonstrated the presence of obscure forms of primary flexor enthesopathy with minimal or even absent radiographic changes (4) (Section I, Part II). Therefore, diagnosis may be missed or confusion with discrete forms of medial coronoid disease may occur (4). Furthermore, other recent studies demonstrated that it is difficult to distinguish primary from concomitant flexor enthesopathy based on the radiographic findings (6, 12) (Section III, Chapter 2 and 3). Therefore, diagnosis of flexor enthesopathy is often challenging but is necessary for a correct treatment: in cases of primary flexor enthesopathy the elbow joint and surrounding flexor muscles are infiltrated with 0.5-2 mg/kg bodyweight methylprednisolonacetate or the affected flexor muscle is surgically transected. Joints with concomitant flexor enthesopathy are treated by removal of the fragment or flap related to the elbow dysplasia (4, 6).

Functional gamma camera imaging or bone scintigraphy has been classically used in cases of unlocalized lameness or inconclusive radiographic findings (13-15). It is a highly sensitive tool for the detection of early skeletal remodelling and to evaluate the activity of lesions found on structural imaging (13-16). The technique is based on the IV injection of technetium-labelled diphosphonates, which incorporate into bone tissue proportional to blood perfusion and bone activity (13, 15). Increased bone remodelling is reflected as “hot-spots” on planar static images two to four hours after administration of the tracer. Therefore the localization of the problem can be readily indicated.
However besides the disadvantage of the use of radioactive material, planar gamma camera imaging with conventional collimators suffers from limited resolution, which makes detailed topographic localization within a joint usually not possible (15). In order to improve the resolution, micro-Single Photon Emission Computed Tomography (μ-SPECT) systems have been introduced, specifically designed for laboratory animals (15). With this system, a three-dimensional magnified image is created which improves the ability to detect and localize lesions (15). However, the limited gantry opening in the conventional μ-SPECT systems precludes their use for dogs and cats (15). Therefore the High resolution Single Photon Emission Computed Tomography (HiSPECT) system, adapted for use on conventional gamma cameras, was developed (15). This system allows with its larger gantry opening a detailed three-dimensional examination of the distal extremities of larger species, such as dogs and cats.

The goal of this chapter was to investigate the ability of planar bone scintigraphy and HiSPECT to detect flexor enthesopathy and the possibilities and limitations of HiSPECT to distinguish primary flexor enthesopathy from the concomitant form. It was hypothesized that 1) HiSPECT would be a sensitive technique to detect flexor enthesopathy; and 2) HiSPECT would differentiate between the primary and the concomitant form of flexor enthesopathy.
Materials and methods

Fourty-six dogs were included in this prospective study, carried out according to the guidelines of the Animal Care Committee of the Ghent University. All dogs, except for the normal control dogs, were presented with thoracic limb lameness at the Ghent University Veterinary Clinic. All dogs underwent a planar bone scan and a HISPECT scan of one or both elbows. Furthermore, they received radiographic, ultrasonographic, computed tomographic (CT), magnetic resonance imaging (MRI) and arthroscopic examinations for diagnostic purposes, as well as to obtain the criteria to characterize the dogs.

Group 1 (Primary flexor enthesopathy) consisted of 24 elbow joints of 14 client-owned dogs. The mean age was 4.6 years (range 7 months - 7.7 years). Eighteen elbow joints were clinically affected, 6 elbow joints were clinically not apparent, since no signs of elbow pain or lameness were found. Therefore these 6 joints were considered subclinically affected. Dogs were included in this group when at least three of the five imaging modalities (radiography, ultrasonography, CT, MRI, arthroscopy) demonstrated lesions consistent with flexor enthesopathy and excluded medial coronoid disease, osteochondritis dissecans, ununited anconeal process and incongruity (4, 6, 12).

![Figure 1: Distribution of male and female for the 4 groups of dogs.](image-url)
Group 2 (Concomitant flexor enthesopathy) contained 24 elbow joints of 15 client-owned dogs. The mean age was 4.4 years (range 7 months - 8.7 years). Twenty-one joints were clinically affected, 3 joints were considered subclinically affected. Dogs were included in this group when flexor enthesopathy lesions were identified with at least three imaging modalities (as described for group 1) and the additional presence of medial coronoid disease was confirmed with CT and arthroscopy (4, 6, 12).

Group 3 (Elbow dysplasia) consisted of 15 elbow joints of 10 client-owned dogs, all clinically affected. The mean age was 3 years (range 10 months - 10.5 years). In all dogs flexor enthesopathy was excluded with five imaging methods, and the presence of medial coronoid disease was confirmed based on arthroscopy and at least one of four other imaging modalities (radiography, ultrasonography, CT and MRI) (4, 6, 12).

Group 4 (Control, normal joints) consisted of 2 laboratory-owned and 3 client-owned dogs. The mean age was 5 years (range 19 months - 10.5 years). For this group, 9 elbow joints were included in the study based on absence of elbow lesions using radiography, ultrasonography, CT, MRI or arthroscopy.

The distribution of breed and gender for the 4 groups is illustrated in figure 1 and 2.

![Figure 2: Distribution of breed within the four groups of dogs.](image-url)
Intravenous injection of $^{99m}$Tc-disodium oxidronate (HDP) (injected activity 639-1184 MBq (mean/kg: 24.4 MBq/kg)) was performed two to four hours prior to image acquisition. All dogs were sedated using acepromazine (0.01 mg/kg)$^b$ and methadone (0.1 mg/kg)$^c$ or medetomidine (28 μg/kg)$^d$ intravenously and then anaesthetized with propofol (6 mg/kg, IV). After intubation, anaesthesia was maintained with isoflurane in oxygen.

Planar ventral flexed and extended, right and left lateral projections were obtained with a Toshiba GCA 7200 A/DI gamma camera (500000 counts, 128x128 matrix). The obtained images were visually evaluated for presence or absence of increased tracer accumulation.

The HiSPECT study was performed using a conventional triple head gamma camera$^e$ adapted with 3 multi-pinhole collimators (6 multi focussed holes, 3 mm diameter, resolution 2.3 mm) (Bioscan). The radius of rotation was set at 22 cm to allow positioning of the elbow between the camera heads. The dogs were positioned in lateral recumbency on the scanning bed with the elbow joint extended cranially, parallel to the bed. The dog's head was pulled back to the lateral side to fit the elbows in the gantry. Data were acquired in step-and-shoot mode (10 steps, 36° angular step, 120 seconds/step). Images were reconstructed using a dedicated ordered subset-expectation maximisation algorithm$^f$ (OSEM, 9 iterations, 6 subsets) and a Butterworth filter was applied (order 5, cut-off frequency 2.5 cycles/cm) (15). The obtained HiSPECT data were visually analyzed by a Board-certified ECVDI diplomate (KP) using a grading system from one to three (1: mildly increased, 2: moderately increased (1 and 2 were graded on the three planes of the reconstructed slices), 3: strongly increased activity on the 3-dimensional reconstruction movie). All images were examined for the presence of increased activity in the region of the medial humeral epicondyle and in the region of the medial coronoid process.

Statistical analysis was selected and performed by a statistical consultant and the first author (EdB). The difference in intensity of tracer uptake, as seen on the HiSPECT scans, in the medial humeral epicondyle and the medial coronoid process area between joints with primary flexor enthesopathy and concomitant flexor enthesopathy was evaluated by Fisher's exact test$^g$. Statistical significance was established at a level of $p<0.05$. 

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Chapter 5: HiSPECT of primary and concomitant flexor enthesopathy

Results

Planar scintigraphic images

Increased tracer uptake in the region of the elbow joint was reported in all but one (subclinically affected) joint with primary flexor enthesopathy, in all joints with concomitant flexor enthesopathy and in all joints with elbow dysplasia. In none of the elbows an unequivocal distinction between primary flexor enthesopathy, concomitant flexor enthesopathy and elbow dysplasia could be made.

Figure 3: Planar static bone scan (A), HiSPECT images (B, C) and corresponding radiographic images (D, E) of a 3.5-year-old female Rottweiler affected by bilateral primary flexor enthesopathy. A) Ventral view with increased tracer uptake in the region of both elbow joints (black arrows). B and C) Latero-medial HiSPECT images with focal uptake in the medial humeral epicondylar region (white arrow). D) Medio-lateral flexed projection of the left elbow showing a spur (broad white arrow), normal medial coronoid process (white arrowhead), mild osteoarthritis (small white arrow) and no subtrochlear sclerosis (black arrow). E) Medio-lateral flexed projection of the right elbow showing a small spur (broad white arrow), a calcified body adjacent to the medial humeral epicondyle (small white arrow), normal medial coronoid process (white arrowhead), moderate osteoarthritis (small black arrow) and no subtrochlear sclerosis (broad black arrow). (R: Right, L: Left, H: Humerus, U: Ulna, R: Radius)
Increased uptake in both elbow regions was seen in 9/14 dogs of the primary flexor enthesopathy group, in 9/15 dogs of the concomitant flexor enthesopathy group and in 5/10 dogs of the elbow dysplasia group (Figure 3 and 4). This bilateral pathology was confirmed in all cases with the structural imaging techniques. No increased tracer uptake of the elbow joint was observed in the normal elbow group.

**Figure 4:** Planar static and HiSPECT images (A, B) with corresponding radiographic (C), CT (D, E) and arthroscopic (F, G) images of a 5.3-year-old male Great Swiss Mountain Dog with bilateral primary flexor enthesopathy. A) Ventral planar bone scan showing increased tracer uptake in both elbows (black arrows) (Left elbow> Right elbow). B) Latero-medial sagittal HiSPECT image of the left elbow showing clear tracer uptake in the medial humeral epicondylar region (white arrow). No tracer uptake is found at the medial coronoid process. C) Medio-lateral flexed projection of the left elbow demonstrating a normal medial humeral epicondyle (white arrow), normal medial coronoid process (white arrowhead) with no subtrochlear sclerosis and osteoarthritis. D, E) Transverse CT images in bone algorithm at the level of the medial coronoid process (D) and at the level of the humeral epicondyles (E) showing a normal medial coronoid process (white arrowhead) with mild irregular margination of the medial humeral epicondyle (white arrow). F, G) Arthroscopic images showing a normal medial coronoid process (white arrowhead) and obvious lesions of the flexor muscles at their attachment site to the medial humeral epicondyle: an erosion (small white arrow), local synovitis and fibrillations (broad white arrow). (R: Right, L: Left)
HisSPECT

Medial humeral epicondylar activity was present in all clinically affected joints with primary flexor enthesopathy (18/18) and in 18/21 of the clinically affected joints with concomitant flexor enthesopathy (Figure 3-5).

Figure 5: Planar static and HisSPECT images (A, B) with corresponding radiographic (C), CT (D) and arthroscopic (E, F) images of an 8.7-year-old female Labrador Retriever diagnosed with medial coronoid disease and concomitant flexor enthesopathy. A) Planar static bone scan (ventral left (L) and right (R) view) showing increased uptake of the right elbow joint (black arrow). B) Lateromedial HisSPECT image demonstrating clear focal uptake of the medial coronoid process region (black arrow) and the medial humeral epicondylar region (white arrow). C) Medio-lateral flexed projection showing a clear medial humeral epicondylar spur (black arrow), an unclearly delineated medial coronoid process (white arrowhead) and mild subtrochlear sclerosis (white arrow). D) Transverse CT image in bone algorithm at the level of the medial coronoid process, showing a large displaced fragment (white arrowhead). A large calcified body is visible within the flexor muscles (white arrow). E) Arthroscopic image at the level of the medial coronoid process showing a large displaced fragment (white arrowhead) with severe erosions of the medial part of the humeral condyle and the medial coronoid process (black asterisks). F) Arthroscopic image at the level of the flexor muscles showing a fibrillated aspect (white arrowhead). (R: Right, L: Left)
The intensity of the tracer uptake was grade 3 in most clinically affected joints of the primary and concomitant flexor enthesopathy groups (Figure 6). No significant difference in intensity of tracer uptake was found between primary and concomitant forms of flexor enthesopathy.

![Figure 6: Grading scale for the intensity of tracer uptake in the medial humeral epicondylar region for clinically affected joints with primary flexor enthesopathy and clinically affected joints with concomitant flexor enthesopathy. (Values in parentheses indicate total number of clinically affected joints)](image)

Medial humeral epicondylar activity was registered in 5 of the 6 subclinically affected joints with primary flexor enthesopathy and in all subclinically affected joints of the concomitant group (3/3). No significant difference in uptake intensity was found between the primary and concomitant forms of flexor enthesopathy (Figure 7).

![Figure 7: Grading scale for the intensity of tracer uptake for subclinically affected joints with primary flexor enthesopathy and subclinically affected joints with concomitant flexor enthesopathy. (Values in parentheses indicate total number of subclinically affected joints)](image)
Chapter 5: HiSPECT of primary and concomitant flexor enthesopathy

In the concomitant group, increased activity was noted in the medial coronoid process area in all clinically affected joints (21/21), which was confirmed by medial coronoid lesions seen with CT and arthroscopy (Figure 5).

![Figure 8: HiSPECT images with corresponding radiographic (Row A), CT (Row B) and arthroscopic images (Row C) of a 4.5-year-old female Rottweiler diagnosed with bilateral primary flexor enthesopathy. Row A) Sagittal HiSPECT images (left and middle image) showing clear focal uptake at the medial humeral epicondyle (white arrow) and mild focal uptake (intensity 1) at the medial coronoid process (black arrow). On the medio-lateral flexed projection (right image) a spur is visible (white arrow) with an unclearly delineated medial coronoid process (white arrowhead), mild osteoarthritis (black arrow) and no subtrochlear sclerosis. Row B) Transverse CT images in bone algorithm showing an osteophyte at the level of the medial coronoid process (white arrowhead) and an irregular margination of the medial humeral epicondyle with osteophytosis (small white arrow) and a small calcified body within the flexor muscles (broad white arrow). Row C) Arthroscopic images demonstrating a mild irregular aspect of the medial coronoid process (white arrowhead) in combination with an obvious thickened and fibrillated aspect of the flexor muscle attachment (white arrows).]
In the primary group, increased activity was also reported in the medial coronoid process area in 6 clinically affected elbow joints. In this group no obvious medial coronoid process lesions were found with the other structural imaging techniques (Figure 8).

No significant differences were found in the intensity grade of tracer uptake in the medial coronoid process area between both groups of flexor enthesopathy (Figure 9).

![Figure 9: Grading scale for the intensity of tracer uptake of the medial coronoid process for joints clinically affected by primary flexor enthesopathy and joints clinically affected by concomitant flexor enthesopathy. (Values in parentheses indicate total number of clinically affected joints)](image)

Increased activity in the medial coronoid process area was observed in all subclinically affected joints of the concomitant group (3/3), with grade 3 intensity in 1 joint and grade 1 intensity in the other 2 joints. One of the 6 subclinically affected elbow joints of the primary flexor enthesopathy group also showed increased tracer uptake of the medial coronoid process with grade 2 intensity.

All elbow joints of the elbow dysplasia group (15/15) showed increased activity in the medial coronoid region. In addition, increased medial humeral epicondylar activity was noticed in 5 of the 15 elbows.
Discussion

This study explored the possibilities and limitations of the planar bone scan and HiSPECT to detect flexor enthesopathy and to make the distinction between the primary and concomitant form. Primary flexor enthesopathy can occur with obvious clinical signs of elbow lameness and clear radiographic lesions such as calcified bodies or spur formation at the medial humeral epicondyle. However some cases of flexor enthesopathy can be a diagnostic challenge when clinical and radiographic examination are inconclusive (4, 17). In these cases bone scintigraphy can be of help to localize the origin of pain or to evaluate relevance of structural imaging findings (13, 14) (Figure 4).

The results of the planar bone scan in the present study demonstrated that it is a valuable tool to get a survey of the remodelling status of bone in cases of elbow pathology (13). All clinically apparent joints with primary flexor enthesopathy and concomitant flexor enthesopathy and all joints with elbow dysplasia showed increased tracer uptake in the region of the elbow joint. However one joint with primary flexor enthesopathy, which was clinically not apparent and therefore considered subclinically affected, did not show tracer uptake while all other applied imaging modalities demonstrated flexor pathology. Possibly the lesions in this joint, visualized with other diagnostic methods, were not localized within the bony structures but in the soft tissues and did therefore not cause any increased bone activity. On the other hand, 8 subclinically affected joints -5 joints with primary flexor enthesopathy and 3 joints with concomitant flexor enthesopathy- were detected with the conventional planar bone scan. This can be explained by the fact that scintigraphy is an extremely sensitive technique and that it detects early bone remodelling before the onset of lameness or without the development of a clinical problem (13).

In the present study the detailed HiSPECT system was able to identify different pathological areas within the elbow joint. Increased tracer uptake in the region of the medial humeral epicondyle, indicative for flexor pathology, was detected in all clinically affected joints with primary flexor enthesopathy and in 18 of the 21 clinically affected joints with concomitant flexor enthesopathy. Therefore the HiSPECT scan can be considered a sensitive technique to detect flexor enthesopathy, either as a single
pathology or as a part of a more complex elbow disease. The intensity of tracer uptake in the medial humeral epicondylar region was evaluated as a possible feature to differentiate primary flexor enthesopathy from the concomitant form. Most joints of the primary and concomitant flexor enthesopathy groups showed high intensity of tracer uptake in the medial humeral epicondylar area. Consequently no significant difference was found between both groups of flexor enthesopathy. In 5 of the 6 subclinically affected joints with primary flexor enthesopathy and in all subclinically affected joints with concomitant flexor enthesopathy (3/3) tracer uptake of the medial humeral epicondyle was also increased. This finding is consistent with literature describing medial humeral epicondylar lesions as coincidental findings (7). In addition, increased uptake of the medial humeral epicondyle in subclinically affected joints can also be explained as adaptive remodelling, without exceeding the pathological limit. On the other hand, similar to the planar scintigram, one subclinically affected joint with primary flexor enthesopathy did not show increased tracer uptake in this region.

In 5/15 joints with elbow dysplasia, increased uptake was also found in the area of the medial humeral epicondyle. The presence of tracer uptake in this region, while all other imaging modalities ruled out flexor pathology, is difficult to explain. It remains uncertain whether increased functional activity combined with normal structural imaging data reflects true pathological (maladaptive) remodelling or whether it is merely a reflection of subclinical (adaptive) remodelling (6). The answer can only be provided with follow-up studies.

Increased tracer uptake in the region of the medial coronoid process was seen in all joints of the concomitant group and in all joints of the elbow dysplasia group. The presence of medial coronoid pathology in these joints was confirmed with CT and arthroscopy. Therefore HiSPECT can be considered a sensitive technique for the detection of medial coronoid process pathology. Nevertheless, in 7 joints with primary flexor enthesopathy (6 of the 18 clinically affected and 1 of the 6 subclinically affected) additional uptake in the medial coronoid region was found, while CT and arthroscopy excluded typical primary medial coronoid pathology. The increased tracer uptake in these joints may represent asymptomatic or temporary bone remodelling (6). Indeed, minor cartilage lesions and osteophytosis were seen with other diagnostic techniques.
(Figure 8). The intensity of tracer uptake in the medial coronoid process area in the primary affected joints was not significantly different from the concomitantly affected joints. Therefore, the intensity of tracer uptake in the medial coronoid region cannot be used as a feature to differentiate between primary and concomitant flexor enthesopathy.

In conclusion, the conventional planar bone scan can be useful in attributing the origin of lameness caused by primary flexor enthesopathy in elbows with equivocal clinical findings and minimal or even absent radiographic changes. The HiSPECT system on the other hand can clearly identify regional increased uptake within the elbow joint and can therefore be used in the diagnostic work-up of an elbow problem in general. The presence of increased activity in the medial humeral epicondylar area is strongly indicative for flexor enthesopathy since it is found in nearly all clinically affected joints of both flexor enthesopathy groups, which supports our first hypothesis. However, increased activity in the medial humeral epicondylar area was also found in joints without flexor enthesopathy. Based on the intensity of tracer uptake in this area no distinction can be made between primary flexor enthesopathy and concomitant flexor enthesopathy. Furthermore the presence of increased uptake in the medial coronoid process area cannot be used to differentiate between primary flexor enthesopathy and the concomitant form as some elbows diagnosed with primary flexor enthesopathy show increased uptake in the medial coronoid process region as well. Therefore our second hypothesis is rejected. Hence the use of multiple imaging modalities remains necessary to make the distinction between primary flexor enthesopathy and concomitant flexor enthesopathy.
Acknowledgments

The authors thank Prof. Dr. J. DeWulf for his help with the statistical analysis.

Footnote:

a Moderin 20 mg/ml; Pfizer A.H.: Louvain La Neuve, Belgium
b Placivet; Codifar: Wommelgem, Belgium
c Mephenon; Denolin: Brussels, Belgium
d Domitor; Pfizer Animal Health: Brussels, Belgium
e Triad; Trionix: Twinsburg, Ohio, USA
f Scivis: Göttingen, Germany
g SPSS statistics; IBM: Armonk, New York, USA
Chapter 5: HiSPECT of primary and concomitant flexor enthesopathy

References


Chapter 6

COMPUTED TOMOGRAPHY OF CANINE ELBOW JOINTS AFFECTED BY PRIMARY AND CONCOMITANT FLEXOR ENTHESOPATHY
COMPUTED TOMOGRAPHY OF CANINE ELBOW JOINTS AFFECTED BY PRIMARY AND CONCOMITANT FLEXOR ENTHESOPATHY

Adapted from: de Bakker E, Gielen I, Van Caelenberg A, van Bree H, Van Ryssen B. Computed tomography of canine elbow joints affected by primary and concomitant flexor enthesisopathy. Veterinary Radiology and Ultrasound 2012; in revision.
Summary

Flexor enthesopathy is a recently used term to describe lesions of the medial humeral epicondyle and is an important differential diagnosis for elbow lameness. It can be considered a primary cause of elbow lameness, but also occurs concomitantly with other elbow pathology. Since treatment of both forms is different it is necessary to make a distinction between the primary and the concomitant form.

A computed tomographic (CT) examination (before and after IV injection of contrast) was performed in 17 dogs with primary flexor enthesopathy, 24 dogs with concomitant flexor enthesopathy, 13 dogs with elbow dysplasia and 7 normal dogs in a prospective study. The final diagnosis was based on the absence or presence of flexor pathology and other elbow disorders obtained with several imaging modalities. CT findings of the medial humeral epicondyle and the attaching flexor muscles consistent with flexor pathology were evaluated and compared. Additionally, the presence of other elbow disorders and general osteoarthritis were noted.

CT signs of flexor enthesopathy were found in 100% of the clinically affected joints with primary flexor enthesopathy and in 97% of the clinically affected joints with concomitant flexor enthesopathy. Those signs were not found in sound elbows or joints affected by elbow dysplasia. Flexor lesions diagnosed in primary flexor enthesopathy were not significantly different from those in the concomitant form.

In conclusion, CT can be applied to detect flexor enthesopathy, but a detailed analysis of the flexor lesions could not reveal significant differences between both forms. Since discrete medial coronoid process lesions may be difficult to diagnose with CT, an indirect distinction between the primary and concomitant form is not always possible. Therefore multiple diagnostic techniques are necessary to obtain a definitive diagnosis.
Introduction

Flexor enthesopathy is defined as an abnormality of the flexor muscles and their attachment to the medial humeral epicondyle (1-4). It has been fairly neglected until recently and should be considered in the differential diagnosis for elbow lameness (1-4). Flexor enthesopathy can be considered a primary cause of elbow lameness when underlying pathology of the elbow joint is absent. However, flexor enthesopathy has been described in the presence of other elbow pathology, mainly medial coronoid disease and incongruity (2, 3, 5, 6). To what extent these concomitant flexor lesions contribute to the lameness of the dog is not yet determined (4). The distribution of both forms of flexor enthesopathy was demonstrated in a recent study describing a prevalence of 6% for primary flexor enthesopathy and 34% for concomitant flexor enthesopathy in a group of lame dogs (2) (Section III, Chapter I). Both forms of flexor enthesopathy require a different treatment: in case of primary flexor enthesopathy the elbow joint and surrounding flexor muscles are infiltrated with 0.5-2 mg/kg bodyweight methylprednisoloneacetate or the affected flexor muscle is surgically transected (1, 3, 7). The current treatment approach for joints affected by concomitant flexor enthesopathy is limited to the surgical removal of the fragment and/or flap caused by the primary elbow dysplasia, without treatment of the flexor muscles (2). In order to perform a correct treatment, diagnosis of flexor enthesopathy and distinguishing primary from concomitant forms of flexor enthesopathy are essential. Since the clinical signs are rather unspecific, imaging of the lesions is necessary to obtain a correct diagnosis (1-3). Radiography and ultrasonography can be considered good screening methods for the detection of flexor enthesopathy demonstrating specific pathology (4, 8, 9). However, in 15% of the cases diagnosed with flexor enthesopathy both radiography and ultrasonography were unable to demonstrate flexor pathology, suggesting that a considerable number of flexor lesions may be missed (8, 9). Furthermore, both techniques were unable to distinguish primary flexor enthesopathy from the concomitant form (8, 9).

CT is a non-invasive imaging technique, which creates sectional images of anatomic structures (10). It is a widely used imaging technique with a high diagnostic accuracy and sensitivity for the detection of bony lesions of elbow joints (11-14). Since CT produces cross-sectional images of the elbow joint it eliminates the problems of
superimposition associated with conventional radiology and therefore it is a sensitive technique to diagnose elbow dysplasia (12). According to the results of a recent clinical study, CT proved to be a valuable technique in the diagnosis of flexor enthesopathy in dogs by demonstrating specific changes (4) (Section III, Chapter 2). However, in that study a detailed analysis of the specific CT findings for flexor enthesopathy was not performed (4). Since CT visualizes both bony and soft tissue structures using bone and soft tissue algorithms, the medial humeral epicondylar changes as well as the flexor muscle lesions can be evaluated. Additionally, IV contrast-enhanced CT could be used to evaluate flexor enthesopathy lesions in dogs, since tendon injury and repair represent new vessel formation and increased vascular permeability (15, 16). The main disadvantages of CT are exposure to ionizing radiation and the need for general anaesthesia (17).

The aims of this chapter are to assess the CT findings of the flexor muscles and their attachment to the medial humeral epicondyle in elbows diagnosed with flexor enthesopathy, and to evaluate the ability of CT and IV contrast-enhanced CT to distinguish primary flexor enthesopathy from concomitant flexor enthesopathy. It was hypothesized that 1) CT would be a sensitive technique to detect flexor enthesopathy; and 2) CT would demonstrate clear differences in details of flexor enthesopathy lesions between both forms of flexor enthesopathy.
Materials and methods

Fifty dogs (n=50) were prospectively investigated. All dogs underwent a complete CT examination and received additional radiographic (n=50), ultrasonographic (n=48), scintigraphic (HiSPECT) (n=45), magnetic resonance imaging (n=49) and arthroscopic (n=50) examinations. The prospective study was conducted in accordance with the guidelines of the Animal Care Committee of the Ghent University. The elbow joints of the 50 dogs were divided in four groups based on the final diagnosis obtained with several imaging modalities (3, 4, 8). The joints of eleven dogs were assigned to different groups (Table 1).

The first group (Primary flexor enthesopathy) consisted of 17 client-owned dogs (29 elbow joints) with a mean age of 4.7 years (range 7 months to 7.7 years). Eleven dogs were male, 6 were female. Dogs were included in this group when at least 3 of the 5 imaging modalities demonstrated lesions consistent with flexor enthesopathy (3, 4, 8). Dogs included in group 1 also had no evidence of other elbow disorders based on CT and arthroscopy. In 7 joints no signs of elbow pain or lameness were found and therefore these joints were considered subclinically affected.

The second group (Concomitant flexor enthesopathy) consisted of 24 client-owned dogs (36 elbow joints) with a mean age of 4.2 years (range 7 months to 8.7 years). Seventeen dogs were male and 7 dogs were female. Dogs were included in this group when flexor enthesopathy lesions were identified with at least 3 imaging modalities and the additional presence of medial coronoid disease (n=29), osteochondritis dissecans (n=3) and medial coronoid disease + osteochondritis dissecans (n=4) was confirmed with CT and arthroscopy (3, 4, 8). In 6 joints no signs of elbow pain or lameness were found and therefore these joints were considered subclinically affected.

The third group (Elbow dysplasia) consisted of 13 client-owned dogs (18 elbow joints) with a mean age of 2.9 years (range 10 months to 10.5 years). Eight dogs were male and 5 were female. In all dogs flexor enthesopathy was excluded based on five imaging methods, and the presence of other elbow disorders was confirmed based on arthroscopy and at least one of the 4 other imaging modalities (3, 4, 8).
The fourth group (Control, normal joints) consisted of 2 laboratory-owned and 5 client-owned dogs. The mean age was 5.4 years (range 19 months to 10.5 years). This group consisted of 5 male dogs and 2 female dogs. For this group, 11 elbow joints were included in analysis based on absence of elbow lesions using radiography, ultrasonography, scintigraphy, MRI or arthroscopy.

The breed distribution for the 4 groups is illustrated in table 1.

<table>
<thead>
<tr>
<th>Breed</th>
<th>Total of dogs</th>
<th>Primary flexor enthesopathy (Joints)</th>
<th>Concomitant flexor enthesopathy (Joints)</th>
<th>Elbow dysplasia (Joints)</th>
<th>Normal joints (Joints)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Labrador Retriever</td>
<td>12</td>
<td>4</td>
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<td>8</td>
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<tr>
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<td>7</td>
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<td>1</td>
</tr>
<tr>
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<td>4</td>
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<td>0</td>
</tr>
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<td>4</td>
<td>2</td>
<td>2</td>
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<tr>
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<td>0</td>
</tr>
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<td>0</td>
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<td>0</td>
<td>0</td>
<td>2</td>
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<tr>
<td>English Cocker Spaniel</td>
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<td>0</td>
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<td>0</td>
</tr>
<tr>
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<td>0</td>
<td>0</td>
<td>4</td>
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<tr>
<td><strong>Total</strong></td>
<td><strong>50</strong></td>
<td><strong>29</strong></td>
<td><strong>36</strong></td>
<td><strong>18</strong></td>
<td><strong>11</strong></td>
</tr>
</tbody>
</table>

Table 1: Breed distribution within the four groups of elbow joints.
A multi-slice helical CT device was used. Prior to CT, dogs were sedated using acepromazine (0.01 mg/kg, IV) with medetomidine (28 μg/kg, IV) and then anaesthetized with propofol (6 mg/kg, IV). After intubation, anaesthesia was maintained with isoflurane in oxygen. Dogs were positioned on the scanning table in left lateral recumbency with both elbow joints parallel and extended cranially in order to scan both elbow joints simultaneously (14). The head of the dogs was pulled back to the lateral side to avoid artefacts (14). In order to obtain perfect symmetry and to be able to compare both elbow joints at the same level a wedge was positioned in between both elbow joints. A lateral survey view was performed to confirm correct positioning. Acquisition variables were 120 kV and 140 mA and a matrix size of 512x512 was used. Transverse CT slices, in bone and soft tissue algorithm, of 1.25 mm thickness with an overlap of 0.6 mm were obtained from the proximal part of the ulna to 3 cm distal to the radial head, parallel to the humero-radial joint space. After this first scanning session, 2 ml/kg of 62.24 g iopromide of contrast was injected intravenously and contiguous slices in soft tissue algorithm were repeated. The DICOM studies were retrieved and analyzed on the eFilm Workstation PACS software. Images of all elbow joints were evaluated in bone and soft tissue algorithm and 3-dimensional (3D) multiplanar reconstructed images in a sagittal and dorsal plane were made. Each image was examined by consensus by a board-certified ECVDI diplomate (HvB) and the head of the CT/MRI unit (IG). Both assessors were blinded to the knowledge of the findings of other modalities and the final diagnosis. Following bony and soft tissue parameters were assessed: appearance of the medial humeral epicondyle (irregular delineation, sclerotic cortex, thickened cortex), presence of a calcified body (size: length (<3 mm, 3 mm - 5 mm, >5 mm) and width (<1 mm, 1 mm - 3mm, >3 mm), distance to medial humeral epicondyle: close (<5 mm) and remote (≥5 mm)), thickening of the flexor muscles and contrast enhancement of the flexor muscles. Thickening and contrast enhancement of the flexor muscles were evaluated in comparison to normal elbow joints. Thickening of the flexor muscles was characterized by enlargement of the muscle belly and loss of fat density surrounding the flexor muscles. Contrast enhancement was positive when the muscle belly showed clear contrast uptake.
Chapter 6: CT findings of primary and concomitant flexor enthesopathy

Additionally abnormalities of the medial coronoid process, including a fragment, fissure, sclerosis, osteophytosis and demineralized tip were noted. Furthermore, the medial aspect of the humeral condyle was inspected for the presence of osteochondritis dissecans lesions or an irregular delineation. Finally the presence of osteoarthritis was determined following a 4-point ordinal grading scheme (Table 2) (12).

<table>
<thead>
<tr>
<th>CT osteophyte score</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 (absent)</td>
<td>No osteophytes present</td>
</tr>
<tr>
<td>1 (mild)</td>
<td>Osteophytes &lt;2 mm present</td>
</tr>
<tr>
<td>2 (moderate)</td>
<td>Osteophytes 2 – 5 mm present</td>
</tr>
<tr>
<td>3 (severe)</td>
<td>Osteophytes &gt;5 mm present</td>
</tr>
</tbody>
</table>

Table 2: Osteophyte grading scheme for CT (12).

Statistical analysis was selected and performed by a statistical consultant and the first author (EdB). Fisher's exact test was used to compare computed tomographic characteristics between dogs affected by primary flexor enthesopathy and dogs affected by concomitant flexor enthesopathy. Significance was established at a value of p<0.05.
Results

CT abnormalities of the medial humeral epicondyle and the attaching flexor muscles were found in 100% of the clinically affected joints with primary flexor enthesopathy and in 97% of the clinically affected joints with concomitant flexor enthesopathy. CT demonstrated flexor pathology in all subclinically affected joints of the primary group and in 3 of the 6 subclinically affected joints of the concomitant group. Abnormalities of the flexor muscles and their attachment to the medial humeral epicondyle were not found in normal elbow joints or those affected by elbow dysplasia.

<table>
<thead>
<tr>
<th>Computed tomographic lesion</th>
<th>Primary flexor enthesopathy</th>
<th>Concomitant flexor enthesopathy</th>
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</thead>
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<tr>
<td></td>
<td>Clinical (22)</td>
<td>Subclinical (7)</td>
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<tr>
<td>Medial humeral Epicondyle</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Irregular outline</td>
<td>18</td>
<td>2</td>
</tr>
<tr>
<td>Sclerotic cortex</td>
<td>20</td>
<td>5</td>
</tr>
<tr>
<td>Thickened cortex</td>
<td>20</td>
<td>5</td>
</tr>
<tr>
<td>Flexor muscles</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thickened</td>
<td>21</td>
<td>6</td>
</tr>
<tr>
<td>Contrast enhancement</td>
<td>22</td>
<td>5</td>
</tr>
<tr>
<td>Calcified body</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total Length</td>
<td>17</td>
<td>2</td>
</tr>
<tr>
<td>&lt;3 mm</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>&lt;3 mm</td>
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<tr>
<td>&gt;3 mm</td>
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<td>Close</td>
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<td>0</td>
</tr>
<tr>
<td>Remote</td>
<td>12</td>
<td>2</td>
</tr>
</tbody>
</table>

Table 3: Number of elbows with CT lesions of the medial humeral epicondyle and the attaching flexor muscles in primary flexor enthesopathy and concomitant flexor enthesopathy, by clinical status. (Values in parentheses indicate total number of elbow joints)
Appearance of the medial humeral epicondyle
Abnormalities of the medial humeral epicondyle were a frequent finding in both groups of flexor enthesopathy with CT, although not significantly different. An irregular outline of the medial humeral epicondyle was seen in 69% of the joints with primary and in 75% of the joints with concomitant flexor enthesopathy (Table 3) (Figure 1-3). Sclerosis of the medial humeral epicondyle was seen in the majority of elbow joints of both flexor enthesopathy groups (Table 3) (Figure 1-3). A thickened cortex was noticed in 86% of the joints with primary and in 78% of the joints with concomitant flexor enthesopathy (Table 3) (Figure 1-3). A combination of all three abnormalities of the medial humeral epicondyle was seen in 69% of the joints with primary and in 67% of the joints with concomitant flexor enthesopathy (Figure 1-3).

Thickening of the flexor muscles
Thickening of the flexor muscles was seen in the majority of elbow joints of both flexor enthesopathy groups with CT: 93% of the joints with primary flexor enthesopathy and 81% of the joints with concomitant flexor enthesopathy (Table 3) (Figure 1 and 3). No statistically significant difference was found between both groups.

Presence of a calcified body
More than half of the joints of both flexor enthesopathy groups showed a calcified body with CT: 65.5% of the joints with primary and in 55.5% of the joints with concomitant flexor enthesopathy (Figure 1-3). A calcified body was not found in the subclinically affected joints from the concomitant group (Table 3). No clear differences were found in length of the calcified body between both groups of flexor enthesopathy, while the width was mostly between 1 mm and 3 mm in both groups. A significantly higher number of joints with primary flexor enthesopathy demonstrated a calcified body remotely to the medial humeral epicondyle (Table 3). Multiple calcified bodies were noticed in the minority of joints of both flexor enthesopathy groups.
Figure 1: Images of a left elbow joint of a 4.5-year-old female Rottweiler diagnosed with primary flexor enthesopathy. A-C) Lateromedial extended (A), flexed (B) and 15° oblique craniolateral-caudomedial (C) radiographic projections showing a clear spur (black arrow), mild osteoarthritis (small white arrow), an irregularly outlined medial coronoid process (broad white arrow) and a mild, patchy sclerotic aspect (white arrowhead). On the transverse CT images in bone algorithm (D-F), an irregularly delineated medial humeral epicondyle with a sclerotic and thickened cortex is visible (broad white arrow) with a small-sized calcified body located closely to the medial humeral epicondyle within a thickened flexor carpi ulnaris muscle (small white arrow). F) Medial coronoid process showing hypodense new bone formation at the tip, representing an osteophyte (white arrowhead). Transverse CT images in soft tissue algorithm before (G) and after (H) IV-injection of contrast demonstrating a thickened flexor carpi ulnaris muscle (white arrow, G) and heterogeneous contrast enhancement of this flexor muscle (broad white arrow, H). The calcified body is also visible (small white arrow, H). Reconstructed image in dorsal plane after IV-injection of contrast (I) shows prominent thickening (white arrowhead) and clear contrast enhancement of the flexor carpi ulnaris muscle (white arrow).
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Figure 2: Images of a right elbow joint of a 5.2-year-old male Dutch Partridge Dog with primary flexor enthesopathy. Radiographic images (lateromedial extended (A), flexed (B) and 15° oblique craniolateral-caudomedial (C) projections) showing a spur (small white arrow) and two calcified bodies near the medial humeral epicondyle (small black arrows). Unclearly delineated medial coronoid process (broad black arrow) with a mild amount of sclerosis (black arrowhead) and moderate osteoarthritis (broad white arrow). Transverse CT image in bone algorithm at the level of the epicondyles (D) showing moderate osteoarthritis (small white arrows), an irregularly delineated medial humeral epicondyle with a sclerotic and thickened cortex (broad black arrow) and a large-sized and small-sized calcified body located closely to the medial humeral epicondyle (broad white arrows). The joint space between humerus and ulna is widened, suggesting incongruency (small black arrow). Transverse CT image in bone algorithm at the level of the radius and ulna (E) demonstrating an osteophyte at the medial coronoid process (black arrow). Transverse CT image in soft tissue algorithm after IV-injection of contrast at the level of the humeral epicondyles (F) showing clear contrast enhancement of the flexor carpi ulnaris muscle and around the calcified body (black circle). The 3D reconstructed dorsal image (G) demonstrates the large calcified body just below the medial humeral epicondyle (white arrow). The dorsal reconstruction after IV-injection of contrast (H) demonstrates clear contrast enhancement of the flexor muscles (small white arrow). The calcified body is also visible (white arrow).
Figure 3: Images of a left elbow joint of a 3.7-year-old male Bernese Mountain Dog diagnosed with concomitant flexor enthesopathy. Row A: Radiographic images (lateromedial extended, flexed and 15° oblique cranial-lateral-caudomedial projections) demonstrating calcified bodies near the medial epicondyle (black arrows), fragmented medial coronoid process (broad white arrow), severe osteoarthritis (small white arrow) and sclerosis (white arrowhead). Row B-D: Transverse CT images in bone algorithm (left), in soft tissue algorithm (middle) and in soft tissue algorithm after IV contrast (right) at different levels of the elbow joint. Row B is at the level of the humeral epicondyles, showing new bone formation (left, black arrow) and an irregular delineation with a thickened and sclerotic cortex (left, white arrow). A calcified body is visible within the flexor carpi ulnaris muscle (left, black arrowhead). Dense joint capsule (middle, white arrows) and clearly visible calcified body within the flexor carpi ulnaris muscle (middle, black circle). Contrast enhancement of the joint capsule (right, white arrow) and flexor carpi ulnaris muscle (right, black circle). Row C is at the level of the distal humeral condyle with new bone formation (left, white arrow). Denser joint capsule (middle, white arrow) and thickened, more dense (presumably due to fibrosis or small mineralizations) flexor carpi ulnaris muscle (middle, black circle). Clear contrast enhancement of this flexor muscle (right, black circle). Row D is at the level of the medial coronoid process showing a large, displaced fragment (left, black arrow) and osteophytes (left, white arrow). Thickened, denser appearance of the flexor carpi ulnaris muscle (middle, black circle) with clear contrast enhancement (right, black circle). (ME: medial epicondyle, LE: lateral epicondyle, MC: medial part humeral condyle, LC: lateral part humeral condyle, R: radius, U: ulna)
**Contrast enhancement of the flexor muscles**

Ninety-three percent of the joints with primary flexor enthesopathy and 81% of the joints with concomitant flexor enthesopathy showed an increased uptake of iodine (Table 3). No significant differences were found between both groups of flexor enthesopathy. This increased uptake was also a frequent finding in subclinically affected joints from the primary flexor enthesopathy group (Table 3). Enhancement of contrast was visible within and around the flexor muscles (Figure 1-3). Thickening without contrast enhancement of the flexor muscles was seen in one subclinically affected joint with primary flexor enthesopathy and in one clinically and one subclinically affected joint with concomitant flexor enthesopathy.

**Association between flexor abnormalities and gradation of osteoarthritis**

A substantial number of calcified bodies in joints of both flexor enthesopathy groups were seen in combination with severe (grade 3) osteoarthritis (Table 4). Abnormalities of the medial humeral epicondyle and abnormalities of the flexor muscles were also demonstrated in combination with lower grades of osteoarthritis in joints of both flexor enthesopathy groups, which was even more evident in the primary group. However, the association between these flexor abnormalities and gradation of osteoarthritis was not significantly different between both groups of flexor enthesopathy (Table 4).

<table>
<thead>
<tr>
<th></th>
<th>Irregular outline, thickened and sclerotic cortex medial humeral epicondyle</th>
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<tr>
<td></td>
<td>PFE (25)</td>
<td>CFE (28)</td>
<td>PFE (27)</td>
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<tr>
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<tr>
<td>Grade 3</td>
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</table>

Table 4: Correlation between osteoarthritis grade (according to the grading scheme, table 2) and the different CT lesions consistent with flexor enthesopathy for joints affected by primary flexor enthesopathy (PFE) and joints with concomitant flexor enthesopathy (CFE). (Values in parentheses indicate number of elbow joints showing specific flexor pathology)
Presence of other elbow disorders

Abnormalities of the medial coronoid process were seen in all joints with elbow dysplasia and in all but one of the joints affected by concomitant flexor enthesopathy (Table 5) (Figure 3). In 55% of the joints with primary flexor enthesopathy, osteophytes at the medial coronoid process were found (Figure 1 and 2). Osteochondritis dissecans without medial coronoid disease was seen in three joints affected by concomitant flexor enthesopathy (22%). In 11% of the joints affected by elbow dysplasia, in 28% of the joints with concomitant flexor enthesopathy and in 6% of the joints with primary flexor enthesopathy longitudinal sclerotic stripes of the medial part of the humeral condyle were seen (Table 5).

<table>
<thead>
<tr>
<th>Other elbow pathology</th>
<th>Primary flexor enthesopathy (29)</th>
<th>Concomitant flexor enthesopathy (36)</th>
<th>Elbow dysplasia (18)</th>
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</tr>
<tr>
<td>MCD + OCD</td>
<td></td>
<td>0</td>
<td>5</td>
</tr>
<tr>
<td>Sclerotic stripes MHC</td>
<td></td>
<td>2</td>
<td>10</td>
</tr>
</tbody>
</table>

Table 5: Distribution of osteoarthritis grade and other elbow disorders for elbows affected by primary flexor enthesopathy, concomitant flexor enthesopathy and elbow dysplasia. (OCD: Osteochondritis dissecans, MCD: Medial coronoid disease, MHC: Medial part of the humeral condyle, values in parentheses indicate total number of elbow joints)
Discussion

The present study explored the possibilities and limitations of CT to detect flexor enthesopathy and to distinguish primary flexor enthesopathy from the concomitant form. In a similar, well-described condition in man (medial epicondylitis or Golfer’s elbow) the diagnostic work up mostly includes radiography, ultrasonography and magnetic resonance imaging (18, 19). This is in contrast with recent studies on the diagnosis of flexor enthesopathy in dogs, which demonstrated the inability of both radiography and ultrasonography to detect and distinguish both forms of flexor enthesopathy (4, 8, 9). In addition, CT is rarely used as a diagnostic possibility of medial epicondylitis in man. In the present study, CT demonstrated flexor pathology in 100% of the clinically affected joints with primary flexor enthesopathy and in 97% of the clinically affected joints with concomitant flexor pathology. This suggests that CT can be considered a sensitive technique to detect flexor enthesopathy. Moreover, CT is a sensitive technique to detect bony changes, which is important to diagnose additional pathology.

Bony changes at the flexor attachment to the medial humeral epicondyle were easily diagnosed with CT (Table 3). A frequent sign of flexor enthesopathy is a sclerotic aspect and thickened cortex of the medial humeral epicondyle, seen in both flexor enthesopathy groups. An irregular outline of the medial humeral epicondyle was less frequently seen. No significant differences in bony changes were found between both groups. These changes at the medial humeral epicondyle can be explained as a skeletal response to high tensile forces within a tendon, as is described in medial epicondylitis in man (20). However, an irregular outline of the medial humeral epicondyle has been described as a sign of osteoarthritis and is considered a logical finding in any joint affected by an elbow disorder, similar to the radiographic epicondylar changes (1-3, 8). Indeed most elbows of both flexor enthesopathy groups in our study with an irregular outline of the medial humeral epicondyle showed a moderate to severe grade of osteoarthritis (Table 4). Moreover, an irregular outline was absent in joints with elbow dysplasia of which most joints had no osteoarthritis. However, some joints of the primary and concomitant flexor enthesopathy groups showed an irregular outline
without general osteoarthritis. Therefore it should not be exclusively considered as an early sign of osteoarthritis, but also regarded as a specific sign of flexor enthesopathy.

Although CT is a technique most often used to visualize bony changes, pathology within the flexor muscles was the most frequent finding in both flexor enthesopathy groups. In contrast to radiography, CT is able to visualize the soft tissue pathology using a soft tissue window (14, 18). Additionally, IV contrast-enhancement enables the visualization of active processes within the soft tissues. Both thickening of the flexor muscles and contrast enhancement were found in 93% of the joints with primary flexor enthesopathy and in 81% of the joints with concomitant flexor enthesopathy. However, the changes were similar for both groups. Thickening of the flexor muscles can be explained by the presence of fibrous or granulation tissue between or within the flexor muscles, formed as a reparative response to microtrauma exerted on the flexor muscles (21). A comparable finding was seen ultrasonographically (9). Contrast enhancement in this study was found within the superficial digital flexor muscle, the deep digital flexor muscle and the flexor carpi ulnaris muscle. An increase in iodine concentration within the flexor muscles can be explained by the increase of blood flow and vascular permeability of the tendon tissue, which is part of the tendon injury and repair mechanism (16). A similar increase in iodine concentration was described in inflammatory and neoplastic tissue caused by an increase in perfusion and vascular permeability (15). Since the additional costs for contrast-enhanced CT are quite low and it only requires a few additional slices, we recommend this additional examination for the diagnostic work-up of any elbow suspected for lesions other than elbow dysplasia.

A calcified body was found in the majority of joints of both flexor enthesopathy groups, which is consistent with literature describing a calcified body as the most frequent sign of flexor enthesopathy (5, 6, 22-27). The presence of a calcified body can be considered part of the tendon degeneration process in tendinopathies (7). In man, several stages of medial epicondylitis have been described. In the early stages inflammatory or synovitic characteristics may be visible. In later stages results of microtearing can be seen, characterized by tendon degeneration, often accompanied by the presence of calcified bodies (7). Although the length of the calcified bodies in our study was mostly larger in the primary form this was not a significant difference. Also the width of the calcified
body was not different between both groups. The only significant difference was the localization of the calcified body: in a significantly higher number of joints affected by primary flexor enthesopathy the calcified body was located further away from the medial humeral epicondyle than in concomitant flexor enthesopathy. However, this finding solely was insufficient to make a distinction between both forms of flexor enthesopathy. In nearly all cases of flexor enthesopathy a calcified body was seen in combination with a moderate to severe grade of osteoarthritis. Similar to medial epicondylitis in man, the presence of a calcified body presumably represents a later stage of the disorder, involving degenerative changes of the elbow joint (7).

In this study not only the differences between both forms of flexor enthesopathy, but also the differences between clinically and subclinically affected joints of both flexor enthesopathy groups were studied in detail. All 7 subclinically affected joints of the primary flexor enthesopathy group and 3 of the 6 subclinically affected joints of the concomitant flexor enthesopathy group demonstrated clear CT changes consistent with flexor enthesopathy. The subclinically affected joints did not show gross pathologic differences when compared to the clinically affected joints (Table 3). This high number of subclinically affected joints detected with CT is consistent with the study of 200 elbows described in the previous chapter (Section III, Chapter 1), which reports medial humeral epicondylar changes as coincidental findings (1). However, 3 subclinically affected joints with concomitant flexor enthesopathy were not detected on either CT or radiography. These joints were diagnosed with concomitant flexor enthesopathy based on additional ultrasonographic, scintigraphic, MRI and arthroscopic examination. The absence of flexor pathology on both radiography and CT can be explained by the fact that in the early stage only subtle lesions are present. The detection of pathology found with other diagnostic techniques does not necessarily have a clinical meaning, since this was mainly seen in subclinically affected joints.

CT and arthroscopic findings were used as gold standards to confirm or exclude elbow dysplasia in this prospective study (12). In 55% of the joints diagnosed with primary flexor enthesopathy, CT revealed osteophytes at the medial coronoid process. Based on the CT findings these joints could have been diagnosed with concomitant flexor enthesopathy, because of suspected medial coronoid process lesions. However,
arthroscopic examination of these joints excluded an abnormal appearance of the medial coronoid process. In all but one joint of the concomitant group, CT confirmed an abnormal appearance of the medial coronoid process. In this one joint, CT showed a normal medial coronoid process while arthroscopy demonstrated chondromalacia. This can be explained by the presence of cartilaginous lesions, which are not detectable by CT (12). With CT, all cases of osteochondritis dissecans in the concomitant group were correctly diagnosed. Longitudinal sclerotic stripes of the medial part of the humeral condyle were a frequent finding in joints of the concomitant group with severe degrees of osteoarthritis. The presence of those sclerotic stripes reflects the severity of lesions in chronically affected joints with concomitant flexor enthesopathy. However, CT also demonstrated sclerotic stripes of the medial part of the humeral condyle in two joints diagnosed with primary flexor enthesopathy. These joints showed severe degenerative changes and the sclerotic stripes of the medial part of the humeral condyle were considered part of the degenerative process. Since the enthesis (the attachment site of the flexor muscles at the medial humeral epicondyle) is often damaged in joints affected by flexor enthesopathy, the underlying synovial membrane may be consequently disrupted, involving the entire joint and resulting in degenerative changes (4).

In conclusion, the results of this study support our first hypothesis that CT can be considered a sensitive technique for the detection of flexor enthesopathy in clinically affected elbow joints. However, study results reject our second hypothesis since a detailed analysis of the CT signs of flexor enthesopathy revealed only minor significant differences, which are insufficient to make the difference between the primary and the concomitant form. Furthermore, discrete lesions of the medial coronoid process may be difficult to diagnose with CT, which makes an indirect distinction between the primary and concomitant form not always possible solely based on CT findings. This illustrates the need for multiple diagnostic modalities to differentiate between primary and concomitant forms of flexor enthesopathy.
Chapter 6: CT findings of primary and concomitant flexor enthesopathy

Acknowledgments

The authors wish to thank prof. Dr. J. DeWulf for his help with the statistical analysis.

Footnote

a Modern 20 mg/ml; Pfizer A.H.: Louvain La Neuve, Belgium
b GE Lightspeed QX/I: Milwaukee, Wisconsin, USA
c Placivet; Codifar: Wommelgem, Belgium
d Domitor; Pfizer Animal Health: Brussels, Belgium
e Ultravist 300; Bayer Schering Pharma AG: Berlin, Germany
f Merge Efilm; Merge eMed: Milwaukee, Wisconsin, USA
g SPSS statistics; IBM: Armonk, New York, USA
References


Chapter 7

MAGNETIC RESONANCE IMAGING OF PRIMARY AND CONCOMITANT FLEXOR ENTHESOPATHY IN THE CANINE ELBOW
Adapted from: de Bakker E, Gielen I, Kromhout K, van Bree H, Van Ryssen B. Magnetic resonance imaging of primary and concomitant flexor enthesopathy in the canine elbow. Veterinary Radiology and Ultrasound 2012; in revision.
Chapter 7: MRI findings of primary and concomitant flexor enthesopathy

Summary

Flexor enthesopathy is a recently recognized elbow disorder and is considered an important differential diagnosis for elbow lameness. Diagnosis of flexor enthesopathy and differentiation between joints affected by the primary and concomitant form of the disease can be challenging, but is essential for a correct treatment. The goal of this prospective study was to compare MRI findings for dogs with primary flexor enthesopathy (n=17), concomitant flexor enthesopathy (n=23), elbow dysplasia (n=13) and normal elbows (n=7).

Each elbow joint underwent transverse and sagittal T1-weighted (before and after IV contrast), transverse and sagittal T2-weighted, and dorsal STIR sequences. Presence or absence of MRI characteristics of flexor enthesopathy (irregular, thickened medial humeral epicondyle with edema; thickened flexor muscles with hyperintense signal and contrast enhancement; calcified body) and other elbow disorders were recorded.

MRI characteristics of flexor pathology were found in 100% of clinically affected joints with primary flexor enthesopathy and 96% of clinically affected joints with concomitant flexor enthesopathy. Thickened flexor muscles were the most common finding, followed by hyperintense signal and contrast enhancement. Abnormal outline of the medial humeral epicondyle, edema and a calcified body were less frequently observed. MRI characteristics of flexor enthesopathy were not found in normal joints or in joints affected by elbow dysplasia. No significant differences in frequencies and details of individual flexor characteristics were noted between primary and concomitant flexor enthesopathy.

Although MRI is a very sensitive technique for the detection of flexor enthesopathy, it cannot be used to differentiate the primary from the concomitant form.
Introduction

Flexor enthesopathy is described as a pathologic condition of the flexor muscles and their attachment to the medial humeral epicondyle (1-3). It is considered a primary elbow disorder when underlying pathology of the elbow is absent. However, in many cases flexor enthesopathy is described in the presence of elbow dysplasia (2, 4, 5). In these cases of concomitant flexor enthesopathy it is unknown to what extent the concomitant flexor lesions contribute to the lameness of the dog and if additional treatment is necessary (2, 3, 6). Therefore, the current treatment is limited to removal of the fragment or flap related to the elbow dysplasia. This is in contrast to joints with primary flexor enthesopathy in which the elbow joint and surrounding flexor muscles are infiltrated with 0.5-2 mg/kg bodyweight methylprednisolonacetate or the affected flexor muscle is surgically transected (3). In different studies the authors explored the available imaging modalities for the diagnosis of flexor enthesopathy and the distinction between primary and concomitant forms of flexor enthesopathy. Radiography and ultrasonography detected flexor enthesopathy in 85% of the cases and were considered first screening methods. Both techniques were unable to distinguish primary flexor enthesopathy from concomitant flexor enthesopathy (7, 8) (Section III, Chapter 2-4). With a detection grade of more than 90%, computed tomography and HiSPECT scintigraphy were considered superior to radiology and ultrasonography to detect flexor pathology, but were equally unable to differentiate both forms (9, 10) (Section III, Chapter 2, 5, 6).

Magnetic resonance imaging (MRI) is a non-invasive imaging technique and is an excellent imaging modality to evaluate soft tissue structures in the musculoskeletal system (11, 12). It is a commonly used technique for the diagnosis of medial epicondylitis in man (Golfer’s elbow) and is specifically used when the diagnosis based on history, physical examination, radiography and ultrasonography is unclear, when there is no satisfactory response to nonsurgical treatment or for preoperative evaluation of the amount of tendon damage (13). MRI has been reported to be accurate in both detecting and characterizing clinical medial epicondylitis in man (14, 15). In contrast, MRI has not yet been routinely performed to diagnose orthopaedic conditions in small animals in general and to evaluate the elbow joint of dogs in particular (16). Since MRI allows imaging in multiple planes without repositioning the patient and it
uses multiple sequences, which allow better delineation of subchondral bone and cartilage, it can be a useful technique for the diagnosis of elbow dysplasia (17-19). The use of intra-articular contrast medium in the elbow has been reported for the diagnosis of medial coronoid disease but not for the soft tissue structures of the elbow (12).

The purpose of this chapter was to examine the possibilities and limitations of MRI to diagnose flexor enthesopathy and to differentiate between primary and concomitant forms of flexor enthesopathy. We hypothesized that 1) MRI would demonstrate specific features of flexor enthesopathy similar to medial epicondylitis in man and 2) with MRI differences in details between both forms of flexor enthesopathy lesions would be observed.
Materials and methods

Fourty-nine dogs (n=49) were included in this prospective study, carried out according to the guidelines of the Animal Care Committee of Ghent University. All dogs, except for the normal control dogs, were presented with thoracic limb lameness at the Veterinary University Clinic of Ghent. All dogs underwent low-field magnetic resonance imaging of one or both elbow joints after a radiographic (n=49), ultrasonographic (n=49), scintigraphic (n=45), CT (n=49) and arthroscopic (n=49) examination.

*Group 1 (Primary flexor enthesopathy)* consisted of 17 client-owned dogs (28 elbow joints) aged between 7 and 92 months (median 4.7 years). Eleven dogs were male, 6 were female. In 7 joints no signs of elbow pain or lameness were found and therefore these joints were considered subclinically affected. Dogs were included in this group when at least three of the five imaging modalities demonstrated lesions consistent with flexor enthesopathy (3, 6, 7). Dogs included in Group 1 had no evidence of other elbow disorders based on CT and arthroscopy.

*Group 2 (Concomitant flexor enthesopathy)* consisted of 23 client-owned dogs (33 elbow joints) aged between 7 months and 8.7 years (median 4.4 years). Seventeen dogs were male and 6 female. Dogs were included in this group when flexor enthesopathy lesions were identified with at least three imaging modalities and the additional presence of medial coronoid disease (26 joints), osteochondritis dissecans (3 joints) and medial coronoid disease + osteochondritis dissecans (4 joints) was confirmed with CT and arthroscopy (3, 6, 7). In five joints medial coronoid disease with flexor enthesopathy was found without signs of elbow pain or lameness and therefore these joints were considered subclinically affected.

*Group 3 (Elbow dysplasia)* consisted of 13 client-owned dogs (18 elbow joints) aged between 10 months and 10.5 years (median 2.9 years). Eight dogs were male and 5 were female. In all dogs flexor enthesopathy was excluded based on five imaging methods, and the presence of elbow dysplasia was confirmed based on arthroscopy and at least one of the four other imaging modalities.

*Group 4 (Control, normal joints)* consisted of 2 laboratory-owned and 5 client-owned dogs aged between 19 months and 126 months (median 5.4 years). This group consisted of 5 male dogs and 2 female dogs. For this group, 11 elbow joints were included in the
study based on the absence of elbow lesions using radiography, ultrasonography, scintigraphy, CT and arthroscopy.

The breed distribution for the 4 groups is summarized in table 1.

<table>
<thead>
<tr>
<th>Breed</th>
<th>Primary flexor enthesopathy n=17</th>
<th>Concomitant flexor enthesopathy n=23</th>
<th>Elbow dysplasia n=13</th>
<th>Normal elbows n=7</th>
</tr>
</thead>
<tbody>
<tr>
<td>Labrador Retriever</td>
<td>3 (1)</td>
<td>8 (3)</td>
<td>6 (2)</td>
<td>1 (0)</td>
</tr>
<tr>
<td>Great Swiss Mountain Dog</td>
<td>4 (3)</td>
<td>1 (0)</td>
<td>0</td>
<td>1 (0)</td>
</tr>
<tr>
<td>Bernese Mountain Dog</td>
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<td>2 (1)</td>
<td>1 (0)</td>
<td>0</td>
</tr>
<tr>
<td>Rottweiler</td>
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<td>2 (2)</td>
<td>0</td>
<td>1 (0)</td>
</tr>
<tr>
<td>Golden Retriever</td>
<td>1 (1)</td>
<td>3 (1)</td>
<td>2 (0)</td>
<td>1 (1)</td>
</tr>
<tr>
<td>Mixed Breed</td>
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<td>1 (1)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Swiss Shepherd Dog</td>
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<td>0</td>
</tr>
<tr>
<td>Border Collie</td>
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<td>0</td>
<td>0</td>
</tr>
<tr>
<td>French Bull Dog</td>
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<td>0</td>
<td>0</td>
<td>1 (1)</td>
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<tr>
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<td>3 (2)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Saint Bernard Dog</td>
<td>0</td>
<td>1 (0)</td>
<td>1 (0)</td>
<td>0</td>
</tr>
<tr>
<td>Dutch Partridge Dog</td>
<td>1 (1)</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Bouvier</td>
<td>0</td>
<td>1 (1)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Shepherd Dog</td>
<td>0</td>
<td>0</td>
<td>1 (1)</td>
<td>0</td>
</tr>
<tr>
<td>Appenzeller</td>
<td>0</td>
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<td>1 (1)</td>
<td>0</td>
</tr>
<tr>
<td>English Cocker Spaniel</td>
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<td>0</td>
<td>1 (1)</td>
<td>0</td>
</tr>
<tr>
<td>Fox Hound</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>2 (2)</td>
</tr>
</tbody>
</table>

Table 1: Breed distribution for the 4 groups. (n= total number of dogs, values in parentheses indicate number of bilaterally affected dogs)

MRI was performed using a permanent 0.2 Tesla magnet. Dogs were sedated using acepromazine (0.01 mg/kg, IV) with medetomidine (28 μg/kg, IV) and then anaesthetized with propofol (6 mg/kg, IV). After intubation, anaesthesia was maintained with isoflurane in oxygen. Dogs were positioned in lateral recumbency with the examined elbow joint close to the table and extended cranially while the other elbow was pulled back dorsally. A human wrist coil was used, based on the volume of the
elbow joint (16). Both elbow joints were scanned one after another in transverse plane (trs) with slice thickness 4 mm and 3 mm in sagittal (sag) sequences with no interslice gap in all sequences. Both T1-weighted (repetition time (TR)= 580 ms, echo time (TE)= 17 ms) and T2-weighted (TR= 5200 ms, TE= 120 ms) spin echo sequences (T1W SE, T2W SE), extending from the mid part of the humerus to 10 cm distal to the elbow joint, were performed in both planes (16). Short inversion time inversion recovery (STIR) (TR= 4500 ms and TE= 30 ms), slice thickness 3 mm, was obtained in a dorsal (dors) plane. Acquisition matrix was 256x256. After this first scanning, 0.3 ml/kg of 0.5 mmol/ml Gadopentetate Dimeglumine of contrast medium was injected intravenously and T1-weighted sequences were repeated. The DICOM studies were retrieved and analyzed on the eFilm Workstation PACS software. Each MRI image was examined by consensus by a board-certified ECVDI diplomate (HvB) and the head of the CT/MRI unit (IG) for following bony and soft tissue parameters: Appearance of the medial humeral epicondyle (irregular outline, thickened cortex, subcortical edema seen on STIR and T2W SEtrs), changes of the flexor muscles (thickening seen on T1W SE sag, T1W SE trs, T2W SE sag, T2W SE trs; hyperintense signal seen on T2W SE sag, T2W SE trs, STIR; and contrast enhancement seen on T1W SE) and presence of a calcified body seen on T1W SE sag, T1W SE trs, T2W SE sag, T2W SE trs. Changes of the flexor muscles were evaluated in comparison to normal elbow joints. A calcified body was diagnosed when a focal area of low signal intensity within the flexor muscle was visible. MRI characteristics of the medial coronoid process (abnormal shape, fragment, fissure) and abnormalities of the medial aspect of the humeral condyle (hyperintense signal in the subchondral bone on STIR and T2W SE) were also recorded.

Statistical analysis was selected and performed by a statistical consultant and the first author (EdB). Fisher’s exact test was used to compare MRI characteristics of flexor enthesopathy between joints with primary flexor enthesopathy and joints with concomitant flexor enthesopathy. Statistical significance was established at a value of p<0.05.
Chapter 7: MRI findings of primary and concomitant flexor enthesopathy

Results

MRI demonstrated abnormalities of the medial humeral epicondyle and the attaching flexor muscles in 100% of the clinically affected elbow joints with primary flexor enthesopathy and in 96% of the clinically affected elbow joints with concomitant flexor enthesopathy (Table 2). Flexor pathology was found in all (7 of the 7) subclinically affected joints with primary flexor enthesopathy and in 2 of the 5 subclinically affected joints with concomitant flexor enthesopathy (Table 2). MRI characteristics of flexor enthesopathy were not found in normal elbow joints or those affected by elbow dysplasia.

Appearance of the medial humeral epicondyle
An irregular outline of the medial humeral epicondyle was seen in the majority of the joints of both flexor enthesopathy groups (Table 2) (Figure 1). Additionally, a thickened cortex was seen in two thirds of the clinically affected joints of both flexor enthesopathy groups. These findings were less frequently observed in the subclinically affected joints. Subcortical edema, only seen on dorsal STIR and transverse T2-weighted sequences, was found in the minority of joints of both flexor enthesopathy groups (Figure 1). No significant differences in appearance of the medial humeral epicondyle were found between both groups of flexor enthesopathy.

Thickened flexor muscles
Thickening of the flexor muscles was found in 100% of the clinically and subclinically affected joints of the primary flexor enthesopathy group and in respectively 96% and 60% of the clinically and subclinically affected joints with concomitant flexor enthesopathy (Table 2) (Figure 1 and 2). Most of the thickened flexor muscles were seen on the sagittal T2-weighted sequence in both groups of flexor enthesopathy, which was not significantly different. In 64% of the joints of the primary flexor enthesopathy group and in 72% of the joints of the concomitant flexor enthesopathy group the thickening was seen on all four MRI sequences (Sagittal and transverse T1-weighted sequences, and sagittal and transverse T2-weighted sequences).
Hyperintense signal of the flexor muscles

A hyperintense signal was found in 100% of the clinically and subclinically affected joints with primary flexor enthesopathy and in respectively 89% and 60% of the clinically and subclinically affected joints with concomitant flexor enthesopathy (Table 2). Most joints of both flexor enthesopathy groups showed a hyperintense signal on transverse T2-weighted and STIR sequences (Figure 1 and 2). No significant differences were found between both groups of flexor enthesopathy.
Chapter 7: MRI findings of primary and concomitant flexor enthesopathy

Figure 2: Transverse pre- (A, C) and postcontrast (B) T1-weighted sequences at the level of the humeral epicondyles (A, B) and medial coronoid process (C), dorsal STIR (D), sagittal postcontrast T1-weighted (E) and T2-weighted (F) sequences at the level of the attachment of the flexor muscles, and T2-weighted sequence at the level of the medial coronoid process (G) of a 3.7-year-old male Bernese Mountain Dog diagnosed with concomitant flexor enthesopathy. A) A large, irregular calcified body (broad white arrow) is visible within the humeral head of the flexor carpi ulnaris muscle, which is thickened (small white arrow). The superficial digital flexor muscle also has a thickened aspect (white arrowhead). B) The humeral head of the flexor carpi ulnaris muscle (small white arrow) and the superficial digital flexor muscle (white arrowhead) show clear heterogeneous contrast enhancement. The large, irregular calcified body (broad white arrow) is visible within the humeral head of the flexor carpi ulnaris muscle. C) Large, displaced fragment of the medial coronoid process (white arrow). D) A clear hyperintense signal is visible within the flexor muscles (white arrowhead). E) A hyperintense signal within the flexor muscles at the level of the attachment site to the medial humeral epicondyle (broad white arrow) with the large irregular calcified body (small white arrow) is demonstrated. F) At the same level as E, the large irregular calcified body (small white arrow) with a hyperintense signal at the level of the attachment site of the flexor muscles (broad white arrow) can be seen. G) Displaced fragment of the medial coronoid process (small white arrow). A hyperintense signal at the attachment site of the flexor muscles to the medial humeral epicondyle can be observed (broad white arrow).
**Contrast enhancement**

Ninety percent of the clinically affected elbow joints of the primary flexor enthesopathy group and 86% of the clinically affected joints of the concomitant flexor enthesopathy group showed contrast enhancement, which was not significantly different (Table 2) (Figure 1 and 2). In 86% of the subclinically affected joints with primary flexor enthesopathy, increased uptake of contrast was found, while this was a less frequent finding in the subclinically affected joints with concomitant flexor enthesopathy.

### Table 2: Number of elbows with bony and soft tissue lesions of the medial humeral epicondyle and the attaching flexor muscles affected by primary flexor enthesopathy and concomitant flexor enthesopathy, by clinical status. (Values in parentheses represent total number of elbow joints)

<table>
<thead>
<tr>
<th>Magnetic resonance imaging lesion</th>
<th>Primary flexor enthesopathy</th>
<th>Concomitant flexor enthesopathy</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Clinical (21)</td>
<td>Subclinical (7)</td>
</tr>
<tr>
<td><strong>Medial humeral epicondyle</strong></td>
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<td></td>
</tr>
<tr>
<td>Irregular outline</td>
<td>14</td>
<td>3</td>
</tr>
<tr>
<td>Thickened cortex</td>
<td>14</td>
<td>4</td>
</tr>
<tr>
<td>Subcortical edema</td>
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<tr>
<td><strong>Flexor muscle</strong></td>
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<td></td>
</tr>
<tr>
<td>Thickening</td>
<td>21</td>
<td>7</td>
</tr>
<tr>
<td>T1W SE sag</td>
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<td>6</td>
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<tr>
<td>T1W SE trs</td>
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<td>4</td>
</tr>
<tr>
<td>T2W SE sag</td>
<td>19</td>
<td>7</td>
</tr>
<tr>
<td>T2W SE trs</td>
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<td>5</td>
</tr>
<tr>
<td>Hyperintense signal</td>
<td>21</td>
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</tr>
<tr>
<td>T2W SE sag</td>
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<td>6</td>
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<td>T2W SE trs</td>
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<tr>
<td>STIR dors</td>
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<tr>
<td>Contrast enhancement</td>
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<td>T1W SE sag</td>
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</table>
Calcified body
A calcified body within or between the flexor muscles was observed in 33% of the clinically affected joints with primary flexor enthesopathy and in 32% of the clinically affected joints with concomitant flexor enthesopathy (Table 2) (Figure 1 and 2). A calcified body was a less frequent finding in subclinically affected joints of the primary flexor enthesopathy group and was not found in subclinically affected joints of the concomitant flexor enthesopathy group. Most calcified bodies were seen on the sagittal T1-weighted sequence in the primary flexor enthesopathy group and on the transverse T1-weighted sequence in the concomitant flexor enthesopathy group, although not significantly different (Table 2). A calcified body was not visible on the transverse T2-weighted sequence in the primary flexor enthesopathy group, while a calcified body was not visible on the sagittal T2-weighted image in the concomitant flexor enthesopathy group.

Presence of other elbow disorders
An abnormal appearance of the medial coronoid process was found in 18% of the joints affected by concomitant flexor enthesopathy and in 21% of the joints with elbow dysplasia (Table 3) (Figure 2). Abnormalities of the medial coronoid process were not found in joints with primary flexor enthesopathy and in normal joints.
A hyperintense signal on STIR and T2-weighted sequences in the subchondral bone of the medial part of the humeral condyle was found in 7 joints of the concomitant group.

<table>
<thead>
<tr>
<th></th>
<th>Primary flexor enthesopathy (29)</th>
<th>Concomitant flexor enthesopathy (33)</th>
<th>Elbow Dysplasia (28)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Medial coronoid process</td>
<td>Abnormal shape 0 2 0</td>
<td>Fragment 0 4 5</td>
<td>Fissure 0 0 1</td>
</tr>
</tbody>
</table>

Table 3: Number of elbows with abnormalities of the medial coronoid process and the medial part of the humeral condyle affected by primary flexor enthesopathy, concomitant flexor enthesopathy and elbow dysplasia. (Values in parentheses indicate total number of elbow joints)
Discussion

This study on four groups of elbow joints showed several typical MRI changes caused by flexor enthesopathy, which were not observed in joints affected by elbow dysplasia and normal joints.

MRI demonstrated flexor pathology in 100% of the clinically affected joints with primary flexor enthesopathy and in 96% of the clinically affected joints with concomitant flexor enthesopathy. Hence MRI can be considered a sensitive technique to detect flexor enthesopathy. The few cases with concomitant flexor enthesopathy, which were not detected with MRI, can be explained by the fact that the flexor tendons may not be visible on all images in the different planes because of their very short size and the relatively thick slice thickness, which was used in this study (16). All subclinically affected joints with primary flexor enthesopathy and 3 of the 5 subclinically affected joints with concomitant flexor enthesopathy demonstrated clear MRI changes consistent with flexor enthesopathy. Data of these subclinically affected joints were obtained during the prospective diagnostic study, which included both elbows of each selected dog. No gross pathologic differences were noticed between the subclinical and clinical joints. This high number of subclinically affected joints detected with MRI is consistent with literature, which describes medial humeral epicondylar changes as coincidental findings (1).

With MRI, lesions of the flexor muscles were more often found compared to abnormalities of the medial humeral epicondyle.

Thickened flexor muscles were found in all joints of the primary flexor enthesopathy group (both clinical and subclinical) and in nearly all of the clinically and subclinically affected joints of the concomitant flexor enthesopathy group. Flexor tendon and muscle thickening were also one of the most specific MRI findings of medial epicondylitis in man (14). Thickening of the flexor muscles, caused by ingrowth of fibrous tissue and the presence of fluid, was detected with the sagittal T2-weighted sequence in our study. This can be explained since T2-weighted sequences specifically detect fluid based lesions (16).

A hyperintense signal of the flexor muscles was seen in nearly all joints of both flexor enthesopathy groups, without any significant difference between both forms. The
transverse T2-weighted and dorsal STIR sequences most frequently demonstrated this hyperintense signal. A similar high T2-weighted intratendon signal intensity was also the most common abnormality in human medial epicondylitis (14). The presence of a hyperintense signal can be explained by disruption of collagen bundles, vascular and fibroblast proliferation and focal hyaline degeneration (14, 20, 21). Additionally the presence of an intense fluid-like intratendon T2-weighted signal intensity could represent tears, similar to medial epicondylitis in man (21).

The presence of an irregular outline of the medial humeral epicondyle and a thickened cortex was seen in two thirds of joints of both flexor enthesopathy groups. Again no significant difference between both forms was noticed. The bony reactions of the medial humeral epicondyle can be considered a logical finding in any joint affected by an elbow disorder and were described as a sign of osteoarthritis in previous reports (1-3). However, irregularities of the medial humeral epicondyle have also been reported as bony outgrowths that extend from the skeleton into the soft tissue of a tendon (22). These enthesophytes represent a skeletal response to high tensile forces within a tendon, which has been described in human enthesopathies (22). The irregularity and thickened cortex found in our study can represent a similar skeletal response.

Subcortical edema of the medial humeral epicondyle was found in the minority of joints of both flexor enthesopathy groups. It has been previously reported that MRI is a sensitive technique for the detection of subtle changes in bone architecture including bone marrow lesions (18, 19). Subcortical edema was also described in medial epicondylitis in man to a limited extent (14). It was explained as a sequel of a microscopic or macroscopic avulsion of the common flexor tendon, which is consistent with the findings of our study (14).

A calcified body was infrequently seen in joints of both flexor enthesopathy groups, while it is the most frequently described radiographic sign in literature (2, 4, 5, 23-25). Several calcified bodies were not visualized because of the use of low field MRI in this study, which produces a higher slice thickness. The calcified bodies were mostly seen on T1-weighted transverse and sagittal sequences, which provide the best anatomical
Section III: Results

details (16). No significant differences in sequences and details were found between both groups of flexor enthesopathy.

Contrast enhancement was seen in the majority of joints of both flexor enthesopathy groups. Unfortunately, no obvious differences in severity of contrast uptake were noticed between both forms of flexor enthesopathy and only minor differences between clinical and subclinical lesions. The concentration of the contrast medium applied in this study was based on reports for the use in man (26). Similar to contrast enhancement in CT, an increase in contrast medium within the flexor muscles can be explained by the increased blood flow and vascular permeability of the tendon tissue, which is part of the tendon and enthesis injury and repair mechanism (27). A comparable increase in contrast concentration was described in inflammatory and neoplastic tissue caused by an increase in perfusion and vascular permeability (28). Since the additional costs for contrast enhanced MRI are quite low and it only requires a few additional sequences, we recommend this additional examination for the diagnostic work-up of any elbow suspected for lesions other than elbow dysplasia.

Our study demonstrated abnormalities of the medial coronoid process in 18% of the joints with concomitant flexor enthesopathy and 21% of the joints with elbow dysplasia. These findings are in contradiction with the findings of the other imaging techniques applied in this study. Based on the MRI findings, joints of the concomitant group could have been assigned to the primary group. This finding confirms our experience that MRI is insufficient for the diagnosis of medial coronoid process lesions, which is in contrast with older reports (17, 29, 30). The lower detection of medial coronoid process lesions in our study can partially be explained by the low field MRI system, which has a decreased resolution and detail compared to the high field MRI systems used in the previously mentioned studies (29-31). Low field MRI also uses thicker slices in order to get signal, which may contribute in missing small lesions (31). Therefore, the use of a low field MRI in our study can be considered a limitation.
The results of our study support our first hypothesis that MRI can be considered an excellent technique for the evaluation of the flexor muscles in the canine elbow joint and can be used for the detection of flexor pathology. However, our study results reject our second hypothesis, since a detailed analysis of the MRI signs of flexor enthesopathy did not reveal any significant differences between primary and concomitant forms of flexor enthesopathy. Furthermore, MRI detected the presence of other elbow disorders in only one third of the joints affected by elbow dysplasia with concomitant flexor enthesopathy, which makes the indirect distinction between both forms of flexor enthesopathy difficult. These conclusions illustrate the need for multiple diagnostic techniques to obtain a definitive diagnosis and to distinguish primary from concomitant flexor enthesopathy.
Acknowledgments

The authors thank Prof. Dr. Dewulf for his help with the statistical analysis.

Footnote

a Moderin 20 mg/ml; Pfizer A.H.: Louvain La Neuve, Belgium
b Airismate; Hitachi: Tokyo, Japan
c Placivet; Codifar: Wommelgem, Belgium
d Domitor; Pfizer Animal Health: Brussels, Belgium
e Magnevist; Bayer: Wayne, New York, USA
f Merge Efilm; Merge eMed: Milwaukee, Wisconsin, USA
g SPSS statistics, IBM: Armonk, New York, USA
Chapter 7: MRI findings of primary and concomitant flexor enthesopathy

References


Chapter 8

ARTHROSCOPIC FEATURES OF PRIMARY AND CONCOMITANT FLEXOR ENTHESOPATHY IN THE CANINE ELBOW
ARTROSCOPIC FEATURES OF PRIMARY AND CONCOMITANT FLEXOR ENTHESOPATHY IN THE CANINE ELBOW

Adapted from: de Bakker E, Samoy Y, Coppieters E, Mosselmans L, Van Ryssen B. Arthroscopic features of primary and concomitant flexor enthesopathy in the canine elbow. Veterinary and Comparative Orthopaedics and Traumatology 2012; in revision.
Summary

The goal of this study is to describe the arthroscopic features of flexor enthesopathy in dogs and to investigate possibilities and limitations of arthroscopy to detect flexor enthesopathy and to make a distinction between the primary and concomitant form. Fifty dogs (n=94 elbow joints) were prospectively studied: dogs with primary flexor enthesopathy (n=29), concomitant flexor enthesopathy (n=36), elbow dysplasia (n=18) and normal elbow joints (n=11). All dogs underwent an arthroscopic examination of one or both elbow joints. Presence or absence of arthroscopic characteristics of flexor enthesopathy as well as presence or absence of other elbow disorders were recorded.

With arthroscopy several pathologic changes of the enthesis were observed in 100% of the joints of both flexor enthesopathy groups, but also in 72% of the joints with elbow dysplasia and in 25% of the normal joints. No clear differences were seen between both flexor enthesopathy groups.

Arthroscopy allows a sensitive detection of flexor enthesopathy characteristics, although not very specific since they may also be found in joints without flexor enthesopathy. The similar aspect of both forms of flexor enthesopathy and the presence of mild irregularities at the medial coronoid process in joints with primary flexor enthesopathy impedes the arthroscopic differentiation between primary and concomitant forms, requiring additional diagnostic techniques to ensure a correct diagnosis.
Introduction

Thoracic limb lameness in medium and large breed dogs is often localized in the elbow joint. The most important cause is elbow dysplasia, which is a collective term for medial coronoid disease, osteochondritis dissecans of the humeral condyle, ununited anconal process and joint incongruity (1-5). Flexor enthesopathy is a recently recognized elbow disorder and is considered an important differential diagnosis for elbow dysplasia (6-8). It is defined as an abnormality of the medial humeral epicondyle and the attaching flexor muscles, radiographically seen as a calcified body or a spur (6-13). In the past, these radiographical changes were often considered as coincidental or clinically unimportant findings (6, 7). However, a recent study has demonstrated the relatively frequent occurrence of medial humeral epicondylar changes (8) (Section III, Chapter 1). Most cases of flexor enthesopathy are described concomitant with other elbow disorders, mainly medial coronoid disease (7-9, 11). The challenge in these cases is to define the cause of the elbow pain in order to perform the correct treatment. In a small percentage of cases, flexor enthesopathy occurs as the only finding and is therefore considered as the primary cause of elbow lameness (8). Primary flexor enthesopathy can occur with clear radiographic changes, but a recent study demonstrated the presence of obscure forms of primary flexor enthesopathy with minimal or even absent radiographic changes (7) (Section I, Part II). Therefore radiography can be used as a first screening method for the detection of flexor enthesopathy, but diagnosis may be missed or confusion with discrete forms of medial coronoid disease may occur. Additional more sophisticated imaging modalities, such as computed tomography and magnetic resonance imaging both with IV contrast, and scintigraphy are sensitive techniques to detect flexor enthesopathy. However, these techniques are unable to differentiate between primary flexor enthesopathy and the concomitant form (14-18).

Arthroscopy of the canine elbow joint can be used as a diagnostic and therapeutic tool and is a widely accepted diagnostic and treatment method for medial coronoid disease (19-22). Because arthroscopy allows the direct visualization of the articular surface it can provide information that is not available with radiography or clinical examination, both most frequently used diagnostic techniques in veterinary medicine (22, 23). The results of a preliminary study show that arthroscopy can also be used for the diagnosis of flexor enthesopathy in dogs (14) (Section III, Chapter 2). In that same study however
a detailed analysis of the specific arthroscopic findings consistent with flexor enthesopathy was not performed (14).

The aim of this chapter is to examine the possibilities and limitations of arthroscopy to detect flexor enthesopathy and to distinguish primary flexor enthesopathy from the concomitant form. It was hypothesized that 1) arthroscopy would be a sensitive technique to detect flexor enthesopathy; and 2) arthroscopy would reveal clear differences in details of flexor enthesopathy characteristics between both forms.
Materials and methods

A prospective study was performed on 50 dogs (n=50) according to the guidelines of the Animal Care Committee of the Ghent University. All dogs, except for the normal control dogs, were presented with thoracic limb lameness at the Veterinary University Clinic of Ghent. All dogs underwent an arthroscopic examination and received additional radiographic (n=50), ultrasonographic (n=48), scintigraphic (HiSPECT) (n=45), computed tomographic (n=50) and magnetic resonance imaging (n=49) examinations for diagnostic purposes as well as to obtain the criteria to characterize the dogs.

Group 1 (Primary flexor enthesopathy) consisted of 17 client-owned dogs (29 elbow joints) between 7 months and 92 months old (median 4.7 years). Eleven dogs were male, 6 were female. Twenty-two elbow joints were clinically affected, 7 elbow joints were clinically not apparent, since no signs of elbow pain or lameness were found. Therefore these 7 joints were considered subclinically affected. Dogs were included in this group when at least three of the five imaging modalities demonstrated lesions consistent with flexor enthesopathy (7, 14, 15). Dogs included in group 1 also had no evidence of other elbow disorders based on CT and arthroscopy.

Group 2 (Concomitant flexor enthesopathy) consisted of 24 client-owned dogs (36 elbow joints) between 7 months and 8.7 years old (median 4.2 years). Seventeen dogs were male and 7 dogs were female. Thirty joints were clinically affected, 6 joints were considered subclinically affected. Dogs were included in this group when flexor enthesopathy lesions were identified with at least 3 imaging modalities and the additional presence of medial coronoid disease (n=29), osteochondritis dissecans (n=3) and medial coronoid disease + osteochondritis dissecans (n=4) was confirmed with CT and arthroscopy (7, 14, 15).

Group 3 (Elbow dysplasia) consisted of 13 client-owned dogs (18 elbow joints), all clinically affected. The age was between 10 months and 10.5 years (median 2.9 years). Eight dogs were male and 5 were female. In all dogs flexor enthesopathy was excluded based on five imaging methods, and the presence of elbow dysplasia was confirmed based on arthroscopy and at least one of the 4 other imaging modalities (7, 14, 15).

Group 4 (Control, normal joints) consisted of 2 laboratory-owned and 3 client-owned dogs, aged between 19 months and 126 months (median 5.4 years). This group consisted of 3 male dogs and 2 female dogs. For this group, 8 elbow joints were included.
in analysis based on absence of elbow lesions using radiography, ultrasonography, scintigraphy, CT or MRI.

The breed distribution for the 4 groups of dogs is illustrated in table 1.

<table>
<thead>
<tr>
<th>Breed</th>
<th>Primary flexor enthesopathy n=17</th>
<th>Concomitant flexor enthesopathy n=24</th>
<th>Elbow dysplasia n=13</th>
<th>Normal joints n=5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Labrador Retriever</td>
<td>3 (1)</td>
<td>8 (3)</td>
<td>6 (2)</td>
<td>0</td>
</tr>
<tr>
<td>Great Swiss Mountain Dog</td>
<td>4 (3)</td>
<td>1 (0)</td>
<td>0</td>
<td>1 (0)</td>
</tr>
<tr>
<td>Bernese Mountain Dog</td>
<td>0</td>
<td>2 (2)</td>
<td>1 (0)</td>
<td>0</td>
</tr>
<tr>
<td>Rottweiler</td>
<td>3 (2)</td>
<td>2 (2)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Golden Retriever</td>
<td>1 (1)</td>
<td>3 (1)</td>
<td>2 (0)</td>
<td>1 (1)</td>
</tr>
<tr>
<td>Mixed Breed</td>
<td>2 (2)</td>
<td>1 (1)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Swiss Shepherd Dog</td>
<td>0</td>
<td>1 (0)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Border Collie</td>
<td>1 (1)</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>French Bull Dog</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1 (1)</td>
</tr>
<tr>
<td>Newfoundlander</td>
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<td>3 (2)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Saint Bernard Dog</td>
<td>0</td>
<td>1 (0)</td>
<td>1 (0)</td>
<td>0</td>
</tr>
<tr>
<td>Dutch Partridge Dog</td>
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<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Bouvier</td>
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<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Bullmastiff</td>
<td>0</td>
<td>1 (1)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Shepherd Dog</td>
<td>0</td>
<td>0</td>
<td>1 (1)</td>
<td>0</td>
</tr>
<tr>
<td>Appenzeller</td>
<td>0</td>
<td>0</td>
<td>1 (1)</td>
<td>0</td>
</tr>
<tr>
<td>English Cocker Spaniel</td>
<td>0</td>
<td>0</td>
<td>1 (1)</td>
<td>0</td>
</tr>
<tr>
<td>Fox Hound</td>
<td>0</td>
<td>0</td>
<td>2 (2)</td>
<td></td>
</tr>
</tbody>
</table>

Table 1: Breed distribution for the 4 groups of elbow joints. (n= total number of dogs, values in parentheses indicate number of bilaterally affected dogs)

Arthroscopy was performed with a 2.4 mm, 25° fore-oblique arthroscope. Prior to arthroscopy, dogs were sedated using acepromazine (0.01 mg/kg, IV) with medetomidine (28 μg/kg, IV) and then anaesthetized with propofol (6 mg/kg, IV). After intubation, anaesthesia was maintained with isoflurane in oxygen. Dogs were positioned in lateral recumbency with the examined elbow close to the operating table and extended. The elbow joint was arthroscopically visualized via a medial approach.
The intra-articular structures were inspected and specific regions within the elbow joint were visually assessed. By moving the arthroscope towards the ulnar trochlear notch and rotating the viewing angle in the direction of the medial humeral epicondyle (viewing angle directed upwards), the flexor muscles and their entheses were visualized (Figure 1).

**Figure 1**: Arthroscopic images illustrating the approach of the flexor muscles and their attachment to the medial humeral epicondyle. Following the incisura trochealis of the ulna (1) with viewing angle upwards in the direction of the medial humeral epicondyle (black arrowhead), the attaching flexor muscles can be visualized (2). 3: Medial part of the humeral condyle, 4: Anconeal process.

Digital still and video images of the arthroscopic procedure in all elbows were recorded. Each arthroscopic image was evaluated by consensus by the first author (EdB) and an experienced orthopaedic surgeon specialized in arthroscopy (BVR). The presence or absence of the following arthroscopic characteristics of flexor enthesopathy were recorded: fibrillated or ruptured insertion of the flexor muscles, local synovitis and an erosion near the insertion site, and a thickened and yellow discoloured appearance of the flexor muscles. A fibrillated insertion was characterized by the presence of loose, shiny, undulating fibers, while in a ruptured insertion pieces of the flexor muscle were visible or the flexor muscle looked cleaved (Figure 2). Thickening of the flexor muscles was visualized as white and swollen tissue (Figure 3).
Arthroscopic characteristics of the medial coronoid process (Table 2), appearance of the medial part of the humeral condyle (OCD, cartilage lesions scored according to the modified outerbridge classification system (Table 3)) and presence or absence of incongruity were also noted (25).

<table>
<thead>
<tr>
<th>Medial coronoid process diagnosis</th>
<th>Detailed arthroscopic findings of the medial coronoid process</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chondromalacia</td>
<td>Irregular, soft or fibrillated cartilage. No fissure.</td>
</tr>
<tr>
<td>Fissure</td>
<td>Cartilage fissure or irregular, soft or fibrillated cartilage. No mobile fragment when probing.</td>
</tr>
<tr>
<td>Non-displaced fragment</td>
<td>Complete fissure. Fragment located at its original position and mobile when probing.</td>
</tr>
<tr>
<td>Displaced fragment</td>
<td>Fragment cranially displaced.</td>
</tr>
<tr>
<td>Medial compartment erosions</td>
<td>Erosions of the medial coronoid process. No fragmentation, except cartilaginous mini-fragments smaller than 2 mm.</td>
</tr>
</tbody>
</table>

Table 2: Detailed description of the arthroscopic findings of different types of medial coronoid process lesions (25).

All dogs received an intravenous injection of Carprofen 50 mg/ml during the arthroscopic procedure except for dogs with primary flexor enthesopathy, which were treated with an intra-articular injection of 0.5-2 mg/kg bodyweight methylprednisolonacetate. All treated dogs of the elbow dysplasia and concomitant flexor enthesopathy groups, and all surgically treated dogs of the primary flexor enthesopathy group received additional intra-articular injection of Mepivacaine Hydrochloride at the end of the procedure. A light pressure bandage was applied on the elbow. All dogs except the ones with primary flexor enthesopathy, which received an intra-articular injection of 0.5-2 mg/kg bodyweight methylprednisolonacetate, were treated with Carprofen 50 mg/ml for three weeks postoperatively. For all dogs, restricted exercise with leash walks was advised.
Modified outerbridge score | Arthroscopic description of the medial part of the humeral condyle cartilage condition
---|---
0 | Normal cartilage
1 | Chondromalacia (cartilage with softening and swelling)
2 | Partial thickness fibrillation. Superficial erosions with pitting or a 'cobblestone' appearance. Lesions that do not reach the subchondral bone.
3 | Deep ulceration that does not reach the subchondral bone
4 | Full thickness cartilage loss with exposure of the subchondral bone
5 | Eburnated bone

Table 3: Modified outerbridge scoring system, used for the arthroscopic evaluation of the cartilage condition of the medial part of the humeral condyle (25).

Statistical analysis was selected and performed by the first author (EdB) and a statistical consultant. Fisher’s exact test was used to compare arthroscopic characteristics of flexor enthesopathy between dogs affected by primary flexor enthesopathy and dogs affected by concomitant flexor enthesopathy. Significance level was set at $p<0.05$. 
Chapter 8: Arthroscopic findings of primary and concomitant flexor enthesisopathy

Results

Arthroscopic abnormalities of the flexor muscles and their attachment to the medial humeral epicondyle were found in 100% of the clinically and subclinically affected joints with primary flexor enthesisopathy, in 100% of the clinically and subclinically affected joints with concomitant flexor enthesisopathy and in 72% of the joints affected by elbow dysplasia. Arthroscopic characteristics of flexor enthesisopathy were also found in 25% of the normal elbow joints.

A fibrillated insertion of the flexor muscles was observed in 10 elbow joints of the primary flexor enthesisopathy group and in 12 elbow joints of the concomitant flexor enthesisopathy group, while a ruptured insertion was demonstrated in 5 elbow joints of the primary flexor enthesisopathy group and in 11 elbow joints of the concomitant flexor enthesisopathy group (Table 4) (Figure 2).

Figure 2: Arthroscopic images illustrating a fibrillated (A-C) and ruptured (D-F) insertion of the flexor muscles to the medial humeral epicondyle. A-C) Minimal (A) to clear (B, C) fibrillation visible, characterized by loose, shiny (black arrow), undulated fibers (white arrowhead). D-F) Ruptured insertion characterized by a cleaved appearance of the flexor muscles (D) and ruptured pieces of flexor muscles (E, F) (black arrow).
The combination of a fibrillated and ruptured insertion was found in 12 joints with primary flexor enthesopathy and in 5 joints of the concomitant flexor enthesopathy group. No significant differences in appearance of the insertion site were found between both groups of flexor enthesopathy. In the minority of joints with elbow dysplasia, a fibrillated (6/18) or ruptured (2/18) insertion was observed, and some joints showed an inhomogenous aspect of the attachment site. One sound elbow joint showed a ruptured insertion.

Thickening of the flexor muscles was a frequent finding in both flexor enthesopathy groups and in the elbow dysplasia group (Table 4) (Figure 3). No significant differences were found between both forms of flexor enthesopathy. In 2 sound elbow joints thickened flexor muscles were observed. One of these 2 sound joints also had a ruptured insertion site.

<table>
<thead>
<tr>
<th>Arthroscopic lesion</th>
<th>Primary flexor enthesopathy</th>
<th>Concomitant flexor enthesopathy</th>
<th>Elbow dysplasia</th>
<th>Normal joints</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Clinical (22)</td>
<td>Subclinical (7)</td>
<td>Clinical (30)</td>
<td>Subclinical (6)</td>
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<tr>
<td>Insertion site</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fibrillated</td>
<td>7</td>
<td>3</td>
<td>10</td>
<td>2</td>
</tr>
<tr>
<td>Ruptured</td>
<td>2</td>
<td>3</td>
<td>8</td>
<td>3</td>
</tr>
<tr>
<td>Local synovitis</td>
<td>16</td>
<td>4</td>
<td>6</td>
<td>3</td>
</tr>
<tr>
<td>Flexor muscles</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Local erosion</td>
<td>5</td>
<td>2</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Thickened</td>
<td>17</td>
<td>6</td>
<td>25</td>
<td>5</td>
</tr>
<tr>
<td>Yellow tissue</td>
<td>10</td>
<td>1</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>Trochlear notch ulna</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Irregular surface</td>
<td>8</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

Table 4: Number of elbow joints with arthroscopic characteristics of flexor enthesopathy for primary flexor enthesopathy, concomitant flexor enthesopathy, elbow dysplasia and normal joints. (Values in parentheses represent total number of elbow joints)
Chapter 8: Arthroscopic findings of primary and concomitant flexor enthesopathy

Local synovitis was found in a significantly higher number of joints with primary flexor enthesopathy compared to joints of the concomitant flexor enthesopathy group (Table 4) (Figure 3). Local erosion was a less frequent finding for both groups of flexor enthesopathy and mostly seen in joints with primary flexor enthesopathy, although not significantly different (Table 4) (Figure 3).

A yellow discoloration of the flexor muscles was a typical finding for joints with flexor enthesopathy (Table 4) (Figure 3). It was observed in a significantly higher number of joints with primary flexor enthesopathy compared to concomitant flexor enthesopathy.

An irregular surface of the mid portion of the incisura trochlearis of the ulna was only found in joints affected by primary flexor enthesopathy (Table 4) (Figure 3).

![Arthroscopic images illustrating different findings of the flexor muscles and their entheses](image)

**Figure 3:** Arthroscopic images illustrating different findings of the flexor muscles and their entheses. A) Normal flexor muscle attachment (white arrow) to the medial humeral epicondyle (black asterisk). B) Local synovitis (white arrowhead) and thickening of the flexor muscles (white arrow). C) Thickened flexor muscle (white arrow). D) Thickening and yellow discoloration of the flexor muscles (black arrow). E) Local erosion near the insertion site of the flexor muscles to the medial humeral epicondyle (white arrowhead). F) Irregular surface of the mid portion of the incisura trochlearis of the ulna (black arrow).
In 94% of the elbows with concomitant flexor enthesopathy and in 100% of the elbows with dysplasia, lesions of the medial coronoid process were found (Figure 4) (Table 5).

Figure 4: Radiographic (Row A), CT (Row B) and arthroscopic (Row C) images of a 3.2-year-old male Newfoundlander with concomitant flexor enthesopathy. Row A) Mediolateral extended (left), flexed (middle) and 15° oblique cranialateral-caudomedial (right) projections revealing a small-sized, rounded spur (small black arrow), a large-sized, elongated calcified body (small white arrow), unclearly delineated medial coronoid process (black arrowhead), moderate sclerosis (broad black arrow) and osteoarthritis (broad white arrow). Row B) Transverse CT images in bone algorithm at the level of the medial coronoid process (left) and humeral epicondyles (middle). Displaced fragments of the medial coronoid process (black arrowhead), irregular outline of the medial humeral epicondyle with a sclerotic and thickened cortex (black arrow) and a large-sized, elongated calcified body (white arrow) are visible. Transverse CT image in soft tissue algorithm after IV contrast (right) demonstrating thickening of the flexor carpi ulnaris muscle with clear enhancement of contrast (black circle). Row C) Arthroscopic images demonstrating displaced fragments of the medial coronoid process (black arrowhead, left), handburr at the treatment site of the fragmented medial coronoid process (middle) and thickened flexor muscles (white arrowhead) with fibrillation (black arrow).
In 31% of the joints with primary flexor enthesopathy, a mild irregular aspect of the medial coronoid process was observed (Figure 5) (Table 5).

Figure 5: Radiographic (Row A), CT (Row B) and arthroscopic (Row C) images of a 3.2-year-old male Great Swiss Mountain Dog diagnosed with primary flexor enthesopathy. Row A) Mediolateral extended (left), flexed (middle) and 15° oblique craniolateral-caudomedial (right) projections only revealing a small spur at the medial humeral epicondyle (white arrow) with a normal medial coronoid process (white arrowhead). Row B) Transverse CT images in bone algorithm at the level of the medial coronoid process (left) and humeral epicondyles (middle), and in soft tissue algorithm after IV injection of contrast (right). The medial coronoid process is normal (left, white arrowhead) and the medial humeral epicondyle shows a mild irregular outline with a sclerotic and thickened cortex (middle, white arrow). Thickened flexor carpi ulnaris muscle with clear enhancement of contrast (right, white arrow). Row C) Arthroscopic images demonstrating a minimal irregular aspect of the medial coronoid process (left, white arrowhead) and obvious lesions of the flexor muscles and their attachment: fibrillated insertion (middle, black arrow), thickened (right, white arrow) and yellow discoloured (right, black arrowhead) flexor muscles.
One dog with primary flexor enthesisopathy had severe erosions of the medial coronoid process and medial part of the humeral condyle bilaterally. In 72% of the joints of the primary flexor enthesisopathy and elbow dysplasia group, the medial part of the humeral condyle had a normal appearance (outerbridge 0) (Table 5). Osteochondritis dissecans was only observed in the concomitant flexor enthesisopathy group (Table 5). All normal elbow joints had a normal appearance of the medial coronoid process and the medial part of the humeral condyle. Incongruity was found in the minority of joints with concomitant flexor enthesisopathy and elbow dysplasia and was absent in joints with primary flexor enthesisopathy (Table 5).

<table>
<thead>
<tr>
<th>Medial coronoid process</th>
<th>Primary flexor enthesisopathy (29)</th>
<th>Concomitant flexor enthesisopathy (36)</th>
<th>Elbow dysplasia (18)</th>
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<tr>
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Table 5: Arthroscopic findings of the medial coronoid process, the medial part of the humeral condyle and the joint space for primary flexor enthesisopathy, concomitant flexor enthesisopathy and elbow dysplasia. (Values in parentheses indicate total number of elbow joints)
Chapter 8: Arthroscopic findings of primary and concomitant flexor enthesopathy

Discussion

This study evaluated the role of arthroscopy in the diagnosis of flexor enthesopathy. Since arthroscopy is the preferred method for the inspection of the intra-articular structures and is frequently applied in the diagnosis and treatment of elbow problems, we were interested to know to what extent arthroscopy could contribute to the under recognized problem of flexor enthesopathy (19-22). Although the flexor muscles are located extra-articularly, the arthroscopic visualization of flexor pathology is possible, because the enthesis - the tendon-to-bone organ - is damaged in those joints and the covering synovial membrane is disrupted consequently (14). However, in some cases the visualization of the enthesis can be hindered because of severe synovitis, periarticular fluid accumulation and associated induced edema developed during the arthroscopic examination. Therefore evaluation of the flexor muscles and their entheses is recommended at the beginning of the arthroscopic examination. Furthermore, the arthroscopic orientation and interpretation of the different visualized characteristics requires a correct position of the arthroscope and some experience to correctly interpret the findings. In a similar well-described condition in man (medial epicondylitis) arthroscopy is not part of the diagnostic and therapeutic management, because the arthroscopic approach of medial epicondylitis differs from classical elbow arthroscopy and injury to the medial collateral ligament, infection and risk to harm nearby neurovascular structures remain common concerns (26, 27). However, a recent study demonstrated that arthroscopic treatment of medial epicondylitis may be performed with a low risk of injury to the ulnar nerve or medial collateral ligament (26). In dogs, arthroscopy of the elbow joint and inspection of the enthesis has been reported as a safe procedure and the punctures sites are identical to the approach for medial coronoid disease (7, 28).

Arthroscopy demonstrated abnormalities of the flexor muscles and their attachment site in all joints with primary flexor enthesopathy and in all joints with concomitant flexor enthesopathy. However, the same characteristics were also found in a large percentage of the elbow dysplasia group and even in some of the normal elbows. Therefore we can conclude that arthroscopy is a very sensitive technique for the detection of flexor lesions, but also little specific. Arthroscopic characteristics of flexor enthesopathy were
also observed in all subclinically affected joints of both forms of flexor enthesopathy. Some flexor enthesopathy lesions may occur before the onset of lameness or without the development of a clinical problem, which is consistent with literature describing medial humeral epicondylar lesions as coincidental findings (6, 17). From this point of view we can also explain the lesions found in the joints of the elbow dysplasia group, in which no other technique showed abnormalities of the flexor muscles.

Arthroscopy enabled a detailed inspection of the macroscopic changes of the enthesis. The tendinous part showed various types of pathology, which may reflect different stages in the development of the problem or different grades of severity. A partially ruptured insertion was most frequently found in joints with concomitant flexor enthesopathy, even though in those joints overuse is presumably not the cause of the development of flexor enthesopathy. A more plausible explanation is the presence of a severe or chronic pathologic process affecting the flexor muscle at its enthesis and resulting in a ruptured insertion (14). In a few joints with elbow dysplasia an inhomogenous aspect of the attachment site was observed, which was not seen in joints with flexor enthesopathy. Possibly the origin or type of lesion is different or in an earlier stage.

Thickening of the flexor muscles was a frequent finding in joints of both flexor enthesopathy groups and can be explained by the presence of fluid and/or fibrous tissue within the flexor muscles, as is also seen with MRI (29). A ruptured or fibrillated insertion and thickening of the flexor muscles were also observed in both control groups, although less pronounced compared to the joints diagnosed with flexor enthesopathy. Additional imaging modalities (radiography, ultrasonography, scintigraphy, CT and MRI) excluded flexor enthesopathy lesions in both control groups. Therefore, the diagnosis of flexor enthesopathy cannot be based solely on the arthroscopic findings. Especially in cases of discrete medial coronoid lesions, arthroscopy may be misleading.

Local erosion and local synovitis near the insertion site, as well as yellow discoloration of the flexor muscles at their attachment to the medial humeral epicondyle were exclusively found in joints affected by both forms of flexor enthesopathy. The latter two features were found in a significantly higher number of joints with primary flexor
enthesopathy. The presence of these features in joints affected by flexor enthesopathy fits in the descriptive stages of medial epicondylitis in man: the early stages represent inflammatory or synovitic characteristics, while later stages demonstrate tendon degeneration, characterized by pathologic tissue alteration (27, 30).

In one third of the joints with primary flexor enthesopathy, an irregular surface of the mid portion of the incisura trochlearis of the ulna was observed. This finding is unrelated to the enthesis and has been previously described as a typical feature for incongruent joints (31). However, all joints of the primary flexor enthesopathy group in our study were diagnosed as congruent on both CT and arthroscopy. The changes may be explained by the overload, which has caused the primary flexor enthesopathy.

The diagnosis of medial coronoid disease in the elbow dysplasia group and the concomitant group was based on the combination of CT and arthroscopic findings. Minimal lesions of the medial coronoid process were also seen in 9 of the 29 joints with primary flexor enthesopathy. Additional imaging modalities, including scintigraphy and computed tomography, excluded medial coronoid disease in these joints and therefore the arthroscopic findings were regarded as degenerative lesions and not as primary lesions (Figure 5). Furthermore, the lesions of the flexor muscles were more pronounced compared to the minimal lesions of the medial coronoid process in these joints. As in many affected joints of this study, the combination of the applied imaging techniques, the severity of the flexor enthesopathy lesions but also the experience of the orthopaedic surgeon was necessary to define the final diagnosis.

In 6 joints with primary flexor enthesopathy, the medial part of the humeral condyle showed mild cartilage lesions. The damaged enthesis in these joints and the consequently disrupted synovial membrane involve the joint in the pathologic process and can result in these degenerative changes (14). One dog with primary flexor enthesopathy showed severe erosions of the complete medial compartment bilaterally. No real fragment of the medial coronoid process was found arthroscopically or on CT. Although this was not the typical appearance of a joint affected with primary enthesopathy, it was still considered as such because of the absence of medial coronoid process lesions.
In conclusion, arthroscopy can easily visualize lesions of the flexor muscle enthesis, which supports our first hypothesis. However, not all findings are specific for flexor enthesopathy, since they were often seen in joints without flexor enthesopathy. The distinction between both forms of flexor enthesopathy is difficult, especially in cases of discrete medial coronoid disease. Not only because of the similar flexor pathology in both forms but also because of the presence of mild irregularities of the medial coronoid process in joints with primary flexor enthesopathy. Therefore the second hypothesis is rejected. We thus suggest the combination with CT to confirm flexor enthesopathy and to enable the visualization of subchondral bone lesions of the medial coronoid process.
Acknowledgments

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Footnote

a Richard Wolf GmbH: Knittlingen, Germany
b Placivet; Codifar: Wommelgem, Belgium
c Domitor; Pfizer Animal Health: Brussels, Belgium
d Rimadyl; Pfizer S.A.: Louvain-La-Neuve, Belgium
e Moderin 20 mg/ml; Pfizer A.H.: Louvain La Neuve, Belgium
f Carbocaine 2%; Pfizer Animal Health: Brussels, Belgium
g SPSS statistics, IBM: Armonk, New York, USA
References


Section III: Results


SECTION IV

GENERAL DISCUSSION
GENERAL DISCUSSION

This thesis identified strengths and limitations of all available diagnostic techniques for detecting flexor enthesopathy of the canine elbow and making a distinction between primary versus concomitant forms of flexor enthesopathy. It is important to correctly identify the cause of elbow pain in lame dogs so that correct treatment decisions can be made. For several decades, elbow dysplasia has been considered as the most important cause of elbow lameness in medium and large breed dogs. This collective term includes several developmental disorders such as fragmented medial coronoid process, ununited anconal process, osteochondritis dissecans of the medial part of the humeral condyle and incongruity (1-4). Except for obvious disorders such as traumata, osteoarthritis, inflammatory and tumoral processes, no other causes of lameness were known. In the course of the last few years, several cases of elbow lameness in our university hospital have been ascribed to a poorly known problem characterized by changes of the medial humeral epicondyle and the attaching flexor muscles (5-12). In the recent veterinary literature, these lesions are not considered as a clinical elbow problem and are therefore not included in the differential diagnosis of canine elbow lameness (13-15). In our described review of 200 affected elbows, radiography revealed a surprisingly high number of joints with medial humeral epicondylar lesions (Section III, Chapter 1). Furthermore, this same study demonstrated that joints can be affected solely by flexor enthesopathy or concomitant with other elbow disorders, mainly elbow dysplasia. Therefore we introduced two terms to name flexor enthesopathy in both groups of joints: primary flexor enthesopathy and concomitant flexor enthesopathy.

The term primary was used because no clear cause of elbow pain could be diagnosed except for the lesions of the flexor muscles and their attachment (‘the enthesis’) to the medial humeral epicondyle (Section I, part II). This form of flexor enthesopathy resembles an overuse problem as described in man, more specifically Tennis elbow (lateral epicondylitis) and Golfer’s elbow (medial epicondylitis) (16, 17). The primary aetiology of medial epicondylitis is described as repetitive stress or overuse caused by chronic repetitive concentric or eccentric contractile loading of the forearm muscles leading to tendinosis with partial tearing progressing to a full-thickness tendon tear (18-20). Similarly, overuse can occur in active dogs caused by repeated microtrauma as a
result of conformation, weight or hyperactivity of the dog. Alternatively a single traumatic event such as a direct blow or a sudden, extreme eccentric contraction of the flexor muscles is a less commonly reported cause in man (19). This was also seen in the dog, likewise on a less frequent base. Another presentation of primary flexor enthesopathy in our hospital was occasionally diagnosed in immature dogs, but was not seen during the collection of cases for this PhD thesis. In those cases, acute trauma led to avulsion of a small part of the medial humeral epicondyle. This would presumably happen before the fusion of the growth centre at 10 weeks (5, 21, 22). A similar presentation is described in Little Leaguer's elbow in man, which is caused by valgus stress across the elbow, resulting in a complete avulsion of the medial humeral epicondyle (23-25). A developmental problem could also be considered as an underlying cause, since it has been sporadically mentioned in veterinary literature (2).

Concomitant flexor enthesopathy is diagnosed in the presence of other elbow disorders that are assumed to be the cause of the elbow pain, although it is not known to what extent the flexor enthesopathy lesions may contribute to the pain. A similar problem has not been reported in man (16, 19, 26). In our patients, concomitant flexor enthesopathy was mainly observed in cases of a severe or chronic pathologic process within the joint, which may have induced the development of the flexor enthesopathy lesions. Severe or chronic inflammation may easily affect the flexor muscles and their entheses since they are located adjacent to the synovial membrane. In these cases, flexor enthesopathy would be secondary to the main problem. Since we cannot prove this hypothesis, we changed the original term 'secondary' to 'concomitant'. Concomitant flexor enthesopathy was also found in elbow joints of dogs with recurrent lameness several years after arthroscopic treatment of fragmented medial coronoid process and/or osteochondritis dissecans. These joints did not show evidence of flexor enthesopathy before the initial treatment. Trauma caused by the arthroscopic intervention or increased inflammation induced by the lesions or the arthroscopic treatment may have caused the development of flexor enthesopathy in these joints. However, in our experience this is not routinely observed. Moreover, it is generally accepted that arthroscopy is a minimally invasive technique with minimal trauma and a low complication rate (27-29). It is not known whether the relapse of lameness in these joints is due to the original elbow problem and/or formation of scar tissue, or due to the
development of flexor enthesopathy. Cartilage erosions and progression of osteoarthritis may also cause lameness in these joints.

The identification of the two different forms of flexor enthesopathy is necessary because of a different treatment approach. Since a treatment protocol was not described in veterinary literature we decided to deduce the treatment from human literature. Conservative therapy is the standard treatment of medial epicondylitis in man and consists of oral nonsteroidal anti-inflammatory medication or local corticosteroid injection around the affected flexor muscle insertion (30). Successful short-term efficacy of these corticosteroid injections has been reported (31, 32). However there is a paucity of literature on long-term outcome of non-surgical treatment, and in some cases of persistent symptoms surgical intervention might be necessary (17, 33). Since nonsteroidal anti-inflammatory drugs have been reported to be insufficient in dogs - which was also our experience - joints with primary flexor enthesopathy are treated by a local injection of corticosteroids or by surgical transection of the affected flexor muscle (Section I, part II). In contrast, concomitant flexor enthesopathy is left untreated and only the primary elbow disorder is treated. Although differently reported by Meyer-Lindenberg and older reports, it was our choice to approach the joints in that way because of the unknown clinical significance of the flexor lesions (6, 9). Only a well-documented follow-up study will allow us to justify this choice of treatment or to adapt our treatment approach.

Two aspects in the diagnosis of flexor enthesopathy should be considered. Flexor enthesopathy should be detected in the first place to enable treatment. From that perspective we set our first hypothesis that each diagnostic technique shows specific signs of flexor enthesopathy. We therefore wanted to examine which features of flexor enthesopathy can be observed by each technique, and in how many cases a diagnosis of flexor enthesopathy can be missed. Secondly a distinction between joints affected by primary flexor enthesopathy and joints affected by concomitant flexor enthesopathy should be made. This distinction can either be based on the absence or presence of other elbow disorders or - according to our second hypothesis - on a different appearance of both forms of flexor enthesopathy. It was expected that not only joints with primary flexor enthesopathy could be diagnosed with certainty, but also that it could be
determined whether flexor enthesopathy found concomitant with other disorders needed treatment based on a difference in diagnostic findings, in other words to differentiate clinical from subclinical forms. In primary flexor enthesopathy this is not an issue, because in these cases the dogs show lameness and flexor enthesopathy is the only pathologic finding within the painful elbow. In the concomitant form however, lameness can be caused by the other elbow problem. In cases of medial coronoid process, it is reasonable to assume that the medial coronoid process lesion is the main cause of the problem. In cases of recurrent lameness after treatment, however, it is more difficult to identify the cause of the pain. Therefore the decision to take these dogs in the study and to assign them to the concomitant group can be questionable. However, we wanted to know which changes would be found. Certainly those joints cannot be compared to joints affected by primary flexor enthesopathy, since the latter joints were free from other disorders.

When considering the distinction between joints affected by primary flexor enthesopathy and joints affected by concomitant flexor enthesopathy based on the detection of other elbow diseases, medial coronoid disease represents the main problem because of its high prevalence in the concomitant flexor enthesopathy group. It is known that the radiographic diagnosis of discrete medial coronoid process lesions is often challenging because the primary lesion is not visible (34-36). Even with more advanced imaging techniques, the diagnosis of medial coronoid disease remains challenging, moreover because the experience of the clinician may play an important role. We also demonstrated that joints affected by primary flexor enthesopathy may resemble joints with medial coronoid disease because of the presence of minimal or no radiographic changes in the area of the medial humeral epicondyle and the attaching flexor muscles (Section I, part II). Additionally there are often discrete pathologic changes of the medial coronoid process, impeding the distinction even more (34, 37). When a dog is presented with elbow lameness and minor radiographic changes of the medial coronoid process, other imaging techniques will be applied to obtain a definitive diagnosis. An attempt can be made to distinguish primary from concomitant forms of flexor enthesopathy based on different signs or differences in severity of the lesions of flexor enthesopathy. Since flexor enthesopathy is partially a soft tissue problem, ultrasonography or magnetic resonance imaging could be proposed (30, 38-42). Alternatively, computed tomography
or arthroscopy could be applied, as both techniques are frequently used in the diagnosis of elbow problems (27, 40, 43, 44). It is therefore important to know which possibilities and limitations each imaging technique offers for that purpose.

During this study it became clear that the diagnosis of flexor enthesopathy can be very challenging. Clinical signs of primary flexor enthesopathy are mostly unspecific: elbow lameness, distension of the elbow joint, limited range of motion and elbow pain (5, 6, 8-13). Only in some cases, careful palpation of the medial side of the elbow joint, caudodistal to the medial humeral epicondyle, can reveal a firm, well-defined swelling (Section I, part II). Therefore the value of the available imaging modalities was examined, including radiography, ultrasonography, scintigraphy, computed tomography, magnetic resonance imaging and arthroscopy. For each technique pathological changes consistent with flexor enthesopathy were determined. A technique was considered positive for flexor enthesopathy when one or more of these pathological changes were present. For each elbow joint the number of positive techniques was established, and from this we concluded that at least 3 techniques should be positive to confirm the diagnosis of flexor enthesopathy. Which techniques had to be positive was not decided, since this was quite variable in our patient groups. An important limitation of this study was that the presence of minimally visible lesions, operator dependent failure and the limited knowledge of the disease could have had an influence on the detection of flexor enthesopathy and thus on the number of positive techniques. Therefore dogs may have been excluded because of false negative findings with one or more techniques. One could question the requirement of only 3 positive techniques to consider a joint affected with flexor enthesopathy, since pathology should be visible with each technique. Again, false negative conclusions may be drawn or lesions may be limited or in a subclinical stage. Anyway, in most elbow joints with flexor enthesopathy 5 or 6 techniques demonstrated flexor pathology.

The detection of a 'new' pathology can be explained by the use of these more sophisticated imaging modalities and the improved knowledge of canine orthopaedics by using these modalities. Since flexor enthesopathy was only recently recognized as a clinically significant elbow disorder, some criticism or scepticism is inevitable. Especially in joints with primary flexor enthesopathy one could question whether
lameness was really attributed to the elbow joint and whether the changes of the medial humeral epicondyle and the attaching flexor muscles were of clinical significance. We can be confident on our findings, because lameness was allocated to the elbow joint based on an accurate orthopaedic examination, a flexion test and when necessary intra-articular anaesthesia, and afterwards confirmed by scintigraphy. The specific pathologic changes in the area of the medial humeral epicondyle and the flexor entheses were confirmed by a combination of different diagnostic techniques, including radiography, ultrasonography, HiSPECT, computed tomography, magnetic resonance imaging and arthroscopy while other primary disorders were excluded.

Additional proof for the presence of pathology in the area of the medial humeral epicondyle was obtained with contrast-enhanced CT or MRI. Our study demonstrated that IV injection of contrast resulted in an increased contrast enhancement of one or more flexor muscles in nearly all dogs with flexor enthesopathy. This can be explained by the increased blood flow and vascular permeability caused by the injury or inflammation of the affected flexor muscle and the activated repair mechanism (45). Unfortunately, no obvious differences in severity of contrast uptake were noticed between both forms of flexor enthesopathy, and differences between clinical and subclinical lesions were minor. Since the additional costs for contrast-enhanced CT or MRI are quite low and it only requires a few additional slices or sequences, we recommend this additional examination for the diagnostic work-up of any elbow suspected for lesions other than elbow dysplasia and thus also in joints suspected of flexor enthesopathy.

Confusion may also arise by the presence of subclinically affected joints in our study. Because of the study design, contralateral joints without signs of elbow lameness or pain but with obvious flexor enthesopathy lesions were also included. Pathology in these joints may represent a pre-stage of the disease. We assumed that subclinically affected joints would be less severely affected or would be detected with a lower number of imaging techniques. Still, most subclinically affected joints were detected with 3 or 4 techniques, mainly a combination of scintigraphy, computed tomography, magnetic resonance imaging and arthroscopy. A possible explanation is that these techniques can demonstrate either early or subtle lesions. However, the detailed differences of flexor
enthesopathy lesions found in clinically and subclinically affected joints of both flexor enthesopathy groups were rather limited. Only further follow-up of these cases can explain the significance of these findings.

Another issue is the general belief that radiographic lesions of the medial humeral epicondyle and the attaching flexor muscles represent osteoarthritis rather than a primary problem (10). Also, according to the criteria defined by the International Elbow Working Group, the medial humeral epicondyle is considered a location for osteoarthritis (46). However, when a joint is affected by osteoarthritis, new bone formation is usually found at several locations within the joint. In some joints of our study, no osteophytes at locations other than the medial humeral epicondyle could be demonstrated. Thus we concluded that radiographic lesions of the medial humeral epicondyle and the attaching flexor muscles do not necessarily express the presence of osteoarthritis. Therefore, the guidelines of the International Elbow Working Group should be interpreted carefully: when an osteophyte at the medial humeral epicondyle is the only finding, a positive elbow dysplasia score may be falsely attributed to a joint. Similarly, joints with a calcified body near the medial humeral epicondyle are not necessarily affected by elbow dysplasia. At this moment medial humeral epicondylar lesions are not included in the elbow dysplasia complex and joints should be judged accordingly.

The presence of osteoarthritis in joints with primary flexor enthesopathy may question whether the flexor lesions in these joints are indeed primary and not a consequence of an underlying problem. Most joints with primary flexor enthesopathy in our study did not show severe grades of osteoarthritis or cartilage erosions that would suggest a primary degenerative joint disease at the base of the problem. Degenerative changes in these joints are most likely a result of the damaged enthesis and the consequently disrupted covering synovial membrane, thus involving the entire joint in the pathologic process.

In a limited group of dogs, histopathology was performed in an attempt to further unravel the aetiology of flexor enthesopathy (Section I, part II). Only biopsies of surgically treated joints with primary flexor enthesopathy were available since the
concomitant flexor lesions were left untreated. The tissues consisted for the major part of dense collagenous tissue with normal muscle fibers at the distal end and synovial villi at the proximal end. In some cases local cartilaginous metaplasia was found. Similar findings were reported in older case reports, mainly revealing bone trabeculae centrally, surrounded by degenerated cartilage at the end of the calcified body and irregular cartilage infiltrated by fibrocartilagenous tissue towards the flexor tendon (2, 5, 6, 9, 11, 12, 21). However, these histopathological findings do not seem to explain the cause of flexor enthesopathy, since the lesions are more likely to be of chronic nature hiding the early original pathologic changes. Histopathological examination of medial epicondylitis in man also demonstrated predominantly tendon degeneration and incomplete reparative processes, although it was originally reported as an inflammatory process (16, 18).

In this PhD thesis, six studies examined the limitations and possibilities of the available diagnostic techniques by describing and comparing the pathologic changes in four groups of dogs. Normal joints and joints only affected by elbow dysplasia served as a reference for the sensitivity and specificity of the pathologic findings in the area of the medial humeral epicondyle. Our results support the first hypothesis that each diagnostic technique can demonstrate changes that are specific for flexor enthesopathy. However, there were also false negative and false positive results, in other words some flexor changes were not detected in joints with flexor enthesopathy and some changes were observed in joints without flexor enthesopathy. Radiography and ultrasonography missed flexor enthesopathy lesions most frequently and in a considerable percentage of the cases (15%). Moreover ultrasonography demonstrated flexor changes in 25% of the joints with elbow dysplasia. Scintigraphy (HiSPECT), computed tomography and magnetic resonance imaging only missed 5% of the cases, but HiSPECT also demonstrated flexor changes in 33% of the elbow dysplasia group. Arthroscopy identified flexor enthesopathy lesions in 100% of the cases, but some findings were quite unspecific since they were also present in the reference groups. Therefore we can conclude that a combination of diagnostic imaging modalities is necessary to demonstrate flexor lesions in all cases.
The second hypothesis of this study was that a detailed comparison of the flexor characteristics would reveal a difference between the specific flexor lesions seen with primary or concomitant flexor enthesopathy, since both forms have a different aetiology. However, none of the available imaging modalities was able to identify significant differences that could be used as a differentiation. Therefore, the second hypothesis is rejected. This means that based on the pathologic changes of the flexor muscles and their attachment to the medial humeral epicondyle, none of the techniques can be used to further differentiate between joints affected by primary or concomitant flexor enthesopathy. In other words differentiation needs to be done by identifying other elbow problems, mainly medial coronoid disease. Therefore, ultrasonography and magnetic resonance imaging are not the first choice since these techniques are less suitable to demonstrate bone and cartilage lesions (40, 47). Furthermore the operator dependence, long learning curve and real time imaging aspect of ultrasonography on the one hand and the high cost of the equipment and need for general anaesthesia of MRI on the other hand also make both techniques less favourable (13, 38). In contrast, both techniques are considered the methods of choice in the diagnostic work-up of medial epicondylitis in man (38, 48, 49). HiSPECT scintigraphy is a sensitive technique for the detection of both medial coronoid disease and flexor enthesopathy, but the low availability and the use of radioactive products are major limitations for routine diagnosis (50). Computed tomography strengthened by IV contrast and arthroscopy are also reliable imaging modalities, and a combination of both techniques will even increase the diagnostic accuracy (28, 40, 44, 51-53). For the present, we propose the combination of computed tomography strengthened by IV contrast medium and arthroscopy as additional techniques after screening with radiography, knowing that radiography may miss flexor enthesopathy and is often insufficient to diagnose medial coronoid disease, especially when the changes are subtle (54, 55). The combination of these techniques could be considered as the ‘gold standard’. A more detailed study comparing the specific flexor characteristics of each form of flexor enthesopathy may reveal more information necessitating us to adapt the proposed diagnostic protocol.

The goal of our search for a diagnostic protocol is to enable the appropriate treatment of a diseased elbow. Since veterinary literature does not provide information on the treatment of flexor enthesopathy, further research on the proposed treatment protocols
for both forms of flexor enthesopathy should be performed. By means of a long-term follow-up study with control visits (clinical and radiographic examination) on a regular base, more information should become available about treatment results for primary flexor enthesopathy (corticosteroid injection versus surgical intervention). Furthermore, the optimal method of treatment of concomitant flexor enthesopathy needs to be explored by examining three possible treatment protocols: 1) arthroscopic fragment and/or flap removal related to the primary elbow dysplasia without treatment of the flexor problem (the current treatment approach); 2) treatment of the flexor muscles without treatment of the elbow dysplasia; 3) treatment of both disorders at the same time, as is suggested by some previous reports (6, 9).

By collecting more samples for histopathological examination, additional information for the differentiation between both forms of flexor enthesopathy may become available.

Future studies may further unravel other aspects of concomitant flexor enthesopathy. As was mentioned previously, the meaning and clinical significance of the flexor lesions in these joints is unknown. Likewise, the development of flexor enthesopathy in joints after treatment of elbow dysplasia should be further investigated. It is interesting to determine the cause of these lesions not only to prevent their development, but also to determine the optimal treatment and results.

In addition, the meaning of subclinically affected joints with flexor enthesopathy should be further explored. It is important to know whether subclinically affected joints will develop into clinically affected joints and what factors would trigger that.

Finally one could question the use of two different terms - primary and concomitant flexor enthesopathy - since both pathologies look more or less identical on each imaging modality. However, the terms primary and concomitant refer to the absence or presence of other elbow disorders and are relevant as both conditions require a different treatment. As in primary osteoarthritis, the term primary flexor enthesopathy refers to the only lesion in the joint, which requires a specific treatment.
In conclusion we can state that this study was a first step in the unravelling of an unknown elbow disorder. We gained insight about how to recognize flexor enthesisopathy and we suggested a diagnostic protocol. Based on that, further studies on the diagnosis, treatment, aetiology and prevention can be performed.
References


SUMMARY
SUMMARY

Over the past few years, changes of the medial humeral epicondyle have received more attention as a differential diagnosis for elbow lameness in dogs. Because the flexor muscles and their entheses are involved, the term ‘flexor enthesopathy’ was recently suggested to describe the disorder. When other causes of pathology in the elbow have been excluded, flexor enthesopathy is considered the primary lesion and treatment is addressed solely to the flexor muscles. However, flexor enthesopathy is often seen concomitant with elbow dysplasia, mostly chronic cases of medial coronoid disease. In these cases, elbow dysplasia is considered as the primary problem and treatment is aimed at fragment removal related to the elbow dysplasia, since it is not known whether the concomitant flexor enthesopathy has any clinical consequences. Detection of flexor enthesopathy and differentiation between primary and concomitant forms can be challenging, but is necessary for a correct treatment.

In Section I (part I) an overview of the existing veterinary and human literature on medial humeral epicondylar changes is given. Radiographic changes at the medial humeral epicondyle were originally reported as ununited medial epicondyle in 1966, characterized by the presence of loose ossified bodies either on the medial side of the elbow joint or distal to the medial humeral epicondyle. Ununited medial epicondyle has been reported as a rare problem and is often considered as a clinically insignificant finding. Since then several clinical papers reported similar lesions, but used different terms: dystrophic calcified body of the flexor tendon origins, traumatic avulsion of the medial humeral epicondyle, medial humeral condylar osteochondritis dissecans and development of a preformed ossification centre. Bony spur formation at the caudal edge of the medial humeral epicondyle was described as another radiographic finding, although less frequently compared to calcified body near the medial humeral epicondyle. Since the pathological changes in dogs seem to have similarities to certain enthesopathies in man, the term ‘flexor enthesopathy’ was recently suggested to describe the disorder in dogs. Clinical findings of flexor enthesopathy are non-specific, and therefore the first step in imaging flexor enthesopathy is radiography, often revealing a calcified body or a spur. However, some cases of primary flexor enthesopathy occur in the presence of minimal or even absent radiographic changes,
which is documented in **part II**. In eight described cases, lameness was attributed to the elbow joint and radiographic examination revealed minimal or absent changes. Therefore additional imaging techniques (ultrasonography, computed tomography (CT) and magnetic resonance imaging (MRI)) were performed prior to arthroscopy. The combination of the minimal radiographic changes combined with specific ultrasonographic, CT, MRI and arthroscopic findings at the medial humeral epicondyle led to the diagnosis of primary flexor enthesopathy. Since these obscure cases of primary flexor enthesopathy can be easily confused with discrete forms of medial coronoid disease, a correct diagnosis is essential in order to obtain a correct treatment decision. Current treatment options for flexor enthesopathy are either conservative or surgical, adopted from enthesopathies in man.

In **section II** the scientific aims of this PhD thesis are described. The general aim is to further unravel the aetiology, clinical significance, diagnostic aspects and treatment options of medial humeral epicondylar lesions (‘flexor enthesopathy’) in dogs. Although medial humeral epicondylar changes have mainly been considered as coincidental or clinically unimportant lesions, it is our experience that lameness caused by these lesions occur on a regular base. Therefore the first aim is to determine the frequency and radiographic aspect of medial humeral epicondylar lesions in a series of elbow radiographs. The second aim is to describe joints affected by primary and concomitant forms of flexor enthesopathy based on the presence of flexor pathology and presence or absence of other elbow disorders. Finally, the third aim is to determine whether detailed pathological changes of the flexor muscles and their entheses could be used as other parameters to detect flexor enthesopathy and to distinguish both forms of flexor enthesopathy.

**Chapter 1** assesses the frequency and radiographic aspect of medial humeral epicondylar lesions and evaluates their association with osteoarthritis. Medical records of dogs diagnosed with elbow lameness were reviewed. Inclusion criteria for this study were a complete clinical examination, a complete set of digital radiographs (mediolateral flexed and extended and 15° oblique craniolateral-caudomedial projections) and a final diagnosis based on CT or MRI and arthroscopy. Changes of the medial humeral epicondyle (irregular outline, spur formation, and/or a calcified body)
were recorded and correlated with the radiographic osteoarthritis grade and final diagnosis. In 80 of the 200 elbows, changes of the medial humeral epicondyle were observed. In 12 of these 80 elbows, changes of the medial humeral epicondyle were the only finding within the joint and these elbows were diagnosed with primary flexor enthesopathy. In the remaining 68 elbows, other elbow pathology was found. In these cases of concomitant medial humeral epicondylar changes, higher grades of osteoarthritis were recorded, while most elbows with primary flexor enthesopathy showed a lower grade of osteoarthritis. In conclusion, radiographic changes of the medial humeral epicondyle are a frequent finding in elbow lameness and are often concomitant with medial coronoid disease. However, medial humeral epicondylar changes may be the only finding and are then considered as the primary cause of lameness and not as a sign of osteoarthritis. The medial humeral epicondylar area should be evaluated carefully to detect the lesions in the first place and to interpret them correctly in order to make the right treatment decision.

Chapter 2 provides a description of joints affected by primary and concomitant forms of flexor enthesopathy. A prospective study over a period of 3 years was performed on dogs admitted for elbow lameness. Based on the radiographic findings, a selection of dogs underwent a complete series of different imaging modalities including ultrasonography, scintigraphy, CT, MRI and arthroscopy. With each technique, pathology of the medial humeral epicondyle, consistent with flexor enthesopathy, and the presence of other elbow disorders were recorded. All joints with signs of flexor enthesopathy seen with at least 3 techniques were selected. A distinction was made between joints with primary flexor enthesopathy and concomitant flexor enthesopathy based on the absence or presence of other elbow disorders. Twenty-three joints were diagnosed with primary flexor enthesopathy and 20 joints with concomitant flexor enthesopathy. In 43% of the joints with primary flexor enthesopathy and in 75% of the joints with concomitant flexor enthesopathy, pathology at the medial humeral epicondyle was demonstrated by all techniques. All joints with concomitant flexor enthesopathy had a diagnosis of medial coronoid disease and/or osteochondritis dissecans. In conclusion, pathology in the area of the medial humeral epicondyle can be demonstrated by several diagnostic techniques in both forms of flexor enthesopathy, without a detailed analysis of the findings. The distinction between primary and concomitant flexor enthesopathy
was based on the presence or absence of other elbow pathology, mainly medial coronoid disease.

In **chapter 3 to chapter 8** the possibilities and limitations of six diagnostic modalities for the detection of flexor enthesopathy and the distinction between the primary and the concomitant form based on a detailed analysis of specific flexor enthesopathy characteristics are explored. Fifty dogs were prospectively studied and underwent radiographic (n=50), ultrasonographic (n=49), scintigraphic (n=47), CT (n=50), MRI (n=49) and arthroscopic (n=50) examinations. The dogs were divided in four groups (primary and concomitant flexor enthesopathy, elbow dysplasia and normal joints) based on the presence or absence of elbow dysplasia and presence or absence of specific flexor enthesopathy criteria determined for each technique.

Radiographic criteria (**chapter 3**) included an irregular margination of the medial humeral epicondyle, spur formation and presence of a calcified body, which were found in 86% of the clinically affected joints with primary flexor enthesopathy and in 100% of the clinically affected joints with concomitant flexor enthesopathy. Flexor pathology was not found in normal elbows and those affected by elbow dysplasia. The detailed radiographic medial humeral epicondylar changes found in primary flexor enthesopathy were not significantly different from those found in concomitant flexor enthesopathy. Furthermore, radiography was unable to diagnose mild changes of the medial coronoid process. The conclusion of this study was that radiography can be used as a first screening method for the detection of flexor enthesopathy, but no significant differences between primary and concomitant forms can be found.

Ultrasonographic criteria (**Chapter 4**) included loss of fiber structure, abnormal attachment and outward bowing of the flexor muscles, irregular outline of the medial humeral epicondyle, and/or focal acoustic shadowing within the flexor muscles consistent with a calcified body. These characteristics were found in 82% of the clinically affected joints with primary flexor enthesopathy, in 87% of the clinically affected joints with concomitant flexor enthesopathy and in 25% of the joints with elbow dysplasia. An abnormal attachment and irregular medial humeral epicondyle were the most frequent findings in both flexor enthesopathy groups, illustrating the problem at the enthesis. The detailed evaluation of the ultrasonographic findings did not show significant differences for both forms of flexor enthesopathy. Flexor pathology was
not found in normal elbows. Although ultrasonography demonstrated specific lesions of the flexor muscles and their attachment, the diagnosis of flexor enthesopathy was missed in 15% of the clinical cases. Moreover, some of the findings were not specific for flexor enthesopathy, since they were also seen in joints without flexor enthesopathy. Since the lesions were similar in both groups of flexor enthesopathy, a distinction between the primary and concomitant form could not be made with this technique.

The HiSPECT criterion (chapter 5) included increased tracer uptake at the medial humeral epicondyle, and was found in nearly all clinically affected joints with primary and concomitant flexor enthesopathy. The intensity of the tracer uptake was not different for both forms of flexor enthesopathy. Furthermore, increased uptake of the medial coronoid process was evaluated and found in 33% of the clinically affected joints with primary flexor enthesopathy and in 100% of the clinically affected joints with concomitant flexor enthesopathy. Increased uptake of the medial humeral epicondyle was also found in 33% of the joints of the elbow dysplasia group. None of the normal joints showed increased uptake of the elbow joint. In conclusion, HiSPECT enables to localize pathology within the elbow joint and is a sensitive technique to detect flexor enthesopathy. However, HiSPECT is insufficient to distinguish primary from concomitant forms of flexor enthesopathy.

CT criteria (chapter 6) included an irregular, sclerotic, thickened medial humeral epicondyle, thickened flexor muscles with contrast enhancement, and/or a focal area of increased attenuation within flexor muscles consistent with a calcified body. These specific characteristics were found in 100% of the clinically affected joints with primary flexor enthesopathy and in 97% of the clinically affected joints with concomitant flexor enthesopathy. The size, shape and localization of the flexor lesions diagnosed in primary flexor enthesopathy were not significantly different from those in the concomitant form. Flexor pathology was not found in normal elbows or those affected by elbow dysplasia. In conclusion, CT is a sensitive technique for the detection of flexor enthesopathy, but a detailed analysis of the flexor lesions could not reveal significant differences between both forms. Since discrete primary lesions of the medial coronoid process may be difficult to diagnose with CT, an indirect distinction between the primary and concomitant form is not always possible.

MRI criteria (chapter 7) included an irregular, sclerotic medial humeral epicondyle, thickened flexor muscles with contrast uptake, and/or a focal area of low signal
Summary

intensity within the flexor muscle consistent with a calcified body. These characteristics were found in 100% of the clinically affected joints with primary flexor enthesopathy and in 96% of the clinically affected joints with concomitant flexor enthesopathy. Thickening of the flexor muscles was the most common finding, followed by a hyperintense signal and contrast enhancement. An abnormal outline of the medial humeral epicondyle and the presence of a calcified body were less frequently observed. No significant differences in frequency and details of flexor enthesopathy lesions were noted between primary and concomitant forms of flexor enthesopathy. Flexor enthesopathy was not found in normal joints or those affected by elbow dysplasia. Although MRI is a very sensitive technique for the detection of flexor enthesopathy, it cannot be used to differentiate the primary from the concomitant form.

Arthroscopic criteria (chapter 8) included a fibrillated or ruptured insertion of the flexor muscles, local synovitis and local erosion near the insertion site, and/or a thickened and yellow discoloured appearance of the flexor muscles. One or more of these findings were seen in 100% of the joints of both flexor enthesopathy groups, in 72% of the joints with elbow dysplasia and in 25% of the normal joints. A ruptured and/or fibrillated insertion as well as thickening of the flexor muscles were a frequent finding in joints of both flexor enthesopathy groups, but were also –though less frequently- observed in joints with elbow dysplasia and normal joints. Local erosion, local synovitis and yellow discoloration of the flexor muscles were exclusively found in joints with flexor enthesopathy, but the latter two in a significantly higher number of joints with primary flexor enthesopathy. In 31% of the joints with primary flexor enthesopathy, an irregular aspect of the ulnar trochlear notch was noted. Additionally, discrete medial coronoid process lesions were found in 31% of the joints with primary flexor enthesopathy. With arthroscopy, lesions of the flexor muscles and their entheses can easily be visualized. However, some of the findings are not specific for flexor enthesopathy, since they were also seen in joints without flexor enthesopathy. The distinction between both forms of flexor enthesopathy is difficult, especially in cases of discrete medial coronoid disease. Not only because of the similar flexor pathology in both forms but also because of the presence of mild irregularities of the medial coronoid process in joints with primary flexor enthesopathy.
The overall conclusion of this PhD thesis is that each described diagnostic technique can demonstrate changes that are specific for flexor enthesopathy. However, there are also false negative and false positive results, in other words some flexor changes are not detected in joints with flexor enthesopathy and some flexor changes are also observed in joints without flexor enthesopathy. Furthermore none of the described techniques enables a clear distinction between primary and concomitant forms of flexor enthesopathy based on specific flexor enthesopathy criteria on the one hand and presence or absence of other elbow disorders on the other hand. Therefore a combination of different imaging modalities is necessary. The presently proposed protocol includes a combination of CT with contrast and arthroscopy additional to a complete orthopaedic and radiographic examination. This protocol may be adapted according to the results of future studies.
SAMENVATTING
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De afgelopen jaren hebben pathologische veranderingen ter hoogte van de mediale epicondyl van de humerus meer aandacht gekregen als mogelijke differentiaal diagnose voor elleboogmanken bij de hond. Omdat de buigspieren en hun aanhechtingsplaats hierbij betrokken zijn, werd onlangs de term 'flexor enthesopathie' ingevoerd om deze aandoening te beschrijven. Flexor enthesopathie kan beschouwd worden als een primair letsel als alle andere oorzaken van elleboogmanken uitgesloten zijn. De uiteindelijke behandeling zal dan ook volledig gericht zijn op de aangetaste buigspier. Echter, flexor enthesopathie wordt vaak samen gezien met elleboogdysplasie, meestal bij chronische gevallen van aandoeningen van de mediale processus coronoideus. Bij deze gevallen wordt elleboogdysplasie beschouwd als het primaire probleem en bestaat de behandeling uit het verwijderen van het fragment dat verband houdt met de elleboogdysplasie, aangezien het niet bekend is of de samengaan de flexor enthesopathie enige klinische gevolgen heeft. Zowel het opsporen van flexor enthesopathie als het onderscheid tussen primaire en concomitante flexor enthesopathy vormen een uitdaging, die echter noodzakelijk is voor een correcte behandeling.

In sectie I (deel I) wordt een overzicht gegeven van de bestaande veterinaire en humane literatuur omtrent pathologische veranderingen ter hoogte van de mediale epicondyl van de humerus. Radiografische veranderingen van de mediale humerus epicondyl bij de hond werden in 1966 voor het eerst beschreven als het niet fusioneren van de mediale humerus epicondyl, gekenmerkt door de aanwezigheid van losse, verbeende fragmenten die zich ofwel aan de mediale zijde van het ellebooggewricht, ofwel distaal van de mediale humerus epicondyl bevonden. Eén niet-gefusioneerde mediale humerus epicondyl werd als een zeldzaam probleem beschreven en werd vaak beschouwd als een klinisch niet-significante bevinding. Sindsdien zijn er in een aantal klinische studies soortgelijke letsels vermeld, alhoewel deze anders werden benoemd: dystrofische calcificatie ter hoogte van de aanhechting van de flexorpees, traumatische avulsie van de mediale humerus epicondyl, osteochondritis dissecans van de mediale humerus condyl en de ontwikkeling van een voorgevormd verbeningscentrum. Beenderige spoorvorming aan de caudale rand van de mediale humerus epicondyl werd eveneens beschreven als een radiografische bevinding, hoewel
minder frequent in vergelijking met een calcificatie ter hoogte van de mediale humerusepicondyl. Aangezien de pathologische veranderingen bij de hond overeenstemmen met bepaalde enthesopathieën bij de mens werd de term ‘flexor enthesopathie’ onlangs ingevoerd om deze aandoening te beschrijven bij de hond. Vermits de klinische bevindingen bij flexor enthesopathie niet-specifiek zijn, is radiografie de eerste stap in de beeldvorming van flexor enthesopathie, waarbij in veel gevallen een calcificatie of een beenderige spoorvorming wordt gezien. Bij sommige gevallen van primaire flexor enthesopathie komen echter geen of slechts minimale radiografische veranderingen voor, hetgeen in deel II wordt gedocumenteerd. In acht gevallen werd manken toegeschreven aan de elleboog en toonde radiografie minimale of geen veranderingen. Daarom werden er voorafgaand aan de arthroscopie aanvullende beeldvormingstechnieken uitgevoerd (echografie, computer tomografie (CT) en nucleaire magnetische resonantie (NMR)). De minimale radiografische veranderingen in combinatie met de specifieke echografische, CT, NMR en artroscopische bevindingen van de mediale humerusepicondyl leidden tot de diagnose van primaire flexor enthesopathie. Aangezien deze onduidelijke gevallen van primaire flexor enthesopathie gemakkelijk verward kunnen worden met subtiele letsels van de mediale processus coronoideus is het essentieel om een juiste diagnose te stellen zodat een correcte behandeling kan worden toegepast. Net als in de humane geneeskunde kan flexor enthesopathie zowel op een conservatieve als een chirurgische manier behandeld worden.

In sectie II worden de wetenschappelijke doelstellingen van dit doctoraatsonderzoek beschreven. De algemene doelstelling is om de etiologie, klinisch belang, diagnostische mogelijkheden en behandelingsopties van letsels ter hoogte van de mediale humerusepicondyl (‘flexor enthesopathie’) bij de hond verder te ontrafelen. Hoewel pathologische veranderingen van de mediale humerusepicondyl voornamelijk beschouwd worden als toevallige of klinisch onbelangrijke letsels, leert onze ervaring dat manken regelmatig veroorzaakt wordt door deze aandoening. Daarom is de eerste specifieke doelstelling om de frequentie en het radiografische aspect van letsels ter hoogte van de mediale humerusepicondyl in een reeks van elleboogradiografieën te bepalen. De tweede specifieke doelstelling is om een beschrijving te geven van gewrichten aangetast met primaire en concomitante vormen van flexor enthesopathie,
op basis van de aanwezigheid van flexor pathology en aan- of afwezigheid van andere elleboogaandoeningen. Tenslotte is de derde specifieke doelstelling om te bepalen of gedetailleerde pathologische veranderingen van de buigspieren en hun aanhechting gebruikt kunnen worden als andere parameters om flexor enthesopathie op te sporen en beide vormen van flexor enthesopathie te onderscheiden.

**Hoofdstuk 1** beoordeelt enerzijds de frequentie en het radiografische aspect van de letsels ter hoogte van de mediale humerusepicondyl en anderzijds de associatie van deze letsels met osteoartrrose. De medische gegevens van honden gediagnosticeerd met elleboogmanken werden geanalyseerd. De inclusiecriteria voor deze studie waren een volledig klinisch onderzoek, een complete reeks van digitale radiografieën (mediolaterale opname in flexie en extensie en een 15° schuine craniolaterale-caudomediale opname) en een definitieve diagnose op basis van CT of NMR en arthroscopie. Alle afwijkingen ter hoogte van de mediale humerusepicondyl (onregelmatige aflijning, beenderige spoorvorming, en/of calcificatie) werden genoteerd en vergeleken met de radiografische gradatie van osteoartrrose en de uiteindelijke diagnose. Bij 80 van de 200 ellebogen zijn afwijkingen ter hoogte van de mediale humerusepicondyl waargenomen. Bij 12 van deze 80 ellebogen werden uitsluitend afwijkingen ter hoogte van de mediale humerusepicondyl aangetroffen. Deze gevallen werden gediagnosticeerd met primaire flexor enthesopathie. Bij de resterende 68 ellebogen zijn ook andere elleboogaandoeningen teruggevonden. Bij deze concomitante gevallen werd een hogere graad van osteoartrrose waargenomen, terwijl de meeste ellebogen met primaire flexor enthesopathie een lagere graad van osteoartrrose bleken te hebben. Hieruit kan men concluderen dat radiografische veranderingen van de mediale humerusepicondyl een frequente bevinding zijn bij elleboogmanken en vaak samen voorkomen met aandoeningen van de mediale processus coronoideus. Echter, wanneer de pathologische veranderingen ter hoogte van de mediale humerusepicondyl de enige bevinding zijn, dan worden deze beschouwd als de belangrijkste oorzaak van manken en niet als een teken van osteoartrrose. Bij het beoordelen van elleboogradiografieën moet steeds de mediale humerusepicondyl zorgvuldig geëvalueerd worden om in de eerste plaats letsels op te sporen om daarna door een correcte interpretatie ervan tot de juiste behandelingsbeslissing te komen.
Hoofdstuk 2 geeft een beschrijving van gewrichten aangetast met primaire en concomitante vormen van flexor enthesopathie. Gedurende een periode van drie jaar werden honden met elleboogmaken in een prospectieve studie opgenomen. Op basis van de radiografische bevindingen ondergingen de geselecteerde honden een complete reeks van verschillende beeldvormingstechnieken: echografie, scintigrafie, CT, NMR en arthroscopie. Met iedere techniek werden afwijkingen van de mediale humerusepicondyl, overeenstemmend met flexor enthesopathie, en de aanwezigheid van andere elleboogpathologieën geregistreerd. Alle gewrichten met tekenen van flexor enthesopathie bij minstens drie beeldvormingstechnieken werden geselecteerd. Er werd een onderscheid gemaakt tussen gewrichten met primaire en concomitante flexor enthesopathie op basis van de afwezigheid of aanwezigheid van andere elleboogaandoeningen. Drieëntwintig gewrichten werden gediagnosticeerd met primaire flexor enthesopathie en twintig gewrichten met concomitante flexor enthesopathie. Bij 43% van de gewrichten met primaire flexor enthesopathie en bij 75% van de gewrichten met concomitante flexor enthesopathie werd met alle beeldvormingstechnieken een afwijking ter hoogte van de mediale humerusepicondyl aangetoond. Alle gewrichten met concomitante flexor enthesopathie werden gediagnosticeerd met een aandoening van de mediale processus coronoideus en/of osteochondritis dissecans. De conclusie van deze studie was dat in beide vormen van flexor enthesopathie afwijkingen in het gebied van de mediale humerusepicondyl konden aangetoond worden met meerdere diagnostische technieken, zonder hierbij de afwijkingen in detail te bekijken. Het onderscheid tussen primaire en concomitante flexor enthesopathie was gebaseerd op het al dan niet aanwezig zijn van andere elleboogpathologieën, voornamelijk aandoeningen van de mediale processus coronoideus.

In hoofdstuk 3 tot hoofdstuk 8 worden de mogelijkheden en beperkingen van zes diagnostische modaliteiten nagegaan om flexor enthesopathie op te sporen en een onderscheid te maken tussen de primaire en concomitante vorm door middel van een gedetailleerde analyse van specifieke flexor enthesopathie kenmerken. Er werden 50 honden prospectief onderzocht met radiografie (n=50), echografie (n=49), scintigrafie (HiSPECT) (n=47), CT (n=50), NMR (n=49) en arthroscopie (n=50). De honden werden verdeeld in vier groepen (primaire en concomitante flexor enthesopathie,
elleboogdysplasie en normale gewrichten) gebaseerd op de aanwezigheid of afwezigheid van elleboogdysplasie en op de aanwezigheid of afwezigheid van specifieke criteria voor het bepalen van flexor enthesopathie die voor elke diagnostische techniek werden vastgesteld.

Radiografische criteria (hoofdstuk 3) bestonden uit een onregelmatige aflijning van de mediale humerusepicondyl, beenderige spoorvorming en aanwezigheid van een calcificatie. Deze werden gevonden bij 86% van de klinisch aangetaste gewrichten met primaire flexor enthesopathie en bij 100% van de klinisch aangetaste gewrichten met concomitante flexor enthesopathie. Bij normale en dysplastische ellebogen werden geen afwijkingen van de buigspieren teruggevonden. De gedetailleerde radiografische veranderingen ter hoogte van de mediale humerusepicondyl die teruggevonden werden bij primaire flexor enthesopathie waren niet significant verschillend van die bij concomitante flexor enthesopathie. Bovendien konden milde letsels van de mediale processus coronoides niet met behulp van radiografie worden waargenomen. De conclusie van deze studie was dat radiografie geschikt is als eerste screeningsmethode voor het vaststellen van flexor enthesopathie, maar dat er met deze techniek geen significante verschillen tussen primaire en concomitante flexor enthesopathie kunnen worden aangetoond.

Echografische criteria (hoofdstuk 4) bestonden uit een verlies van vezelstructuur, afwijkende aanhechting en uitpuilen van de buigpezen, een onregelmatigheid ter hoogte van de mediale humerusepicondyl, en/of een focale akoestische schaduw in de buigspieren die overeenstemt met een calcificatie. Deze kenmerken werden teruggevonden bij 82% van de klinisch aangetaste gewrichten met primaire flexor enthesopathie, bij 87% van de klinisch aangetaste gewrichten met concomitante flexor enthesopathie en bij 25% van de gewrichten met elleboog dysplasie. Een afwijkende aanhechting en een onregelmatig afgelijnde mediale humerusepicondyl waren de meest frequentte bevindingen in beide flexor enthesopathie groepen, en illustreren het probleem ter hoogte van de enthesis. De gedetailleerde evaluatie van de echografische bevindingen toonde geen significante verschillen tussen beide vormen van flexor enthesopathie. De normale ellebogen vertoonden geen echografische afwijkingen van de buigspieren en hun aanhechting. Hoewel echografie specifieke letsels van de buigspieren en de aanhechting ervan kon aantonen, werd de diagnose bij 15% van de klinische gevallen gemist. Bovendien waren deze bevindingen niet altijd specifiek voor
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flexor enthesopathie aangezien ze ook teruggevonden werden in andere gewrichten zonder flexor enthesopathie. Met deze techniek kon geen onderscheid worden gemaakt tussen de primaire en concomitante vorm van flexor enthesopathie, aangezien de bevindingen voor beide groepen van flexor enthesopathie vergelijkbaar waren.

Het HiSPECT criterium (**hoofdstuk 5**) was een verhoogde opname van het radioactief product ter hoogte van de mediale humerusepicondyl, hetgeen in bijna alle klinisch aangetaste gewrichten met primaire en concomitante flexor enthesopathie werd teruggevonden. De intensiteit van de opname was niet verschillend voor beide vormen van flexor enthesopathie. Bovendien werd verhoogde opname ter hoogte van de mediale processus coronoideus teruggevonden bij 33% van de klinisch aangetaste gewrichten met primaire flexor enthesopathie en bij 100% van de klinisch aangetaste gewrichten met concomitante flexor enthesopathie. Verhoogde opname ter hoogte van de mediale humerusepicondyl werd eveneens teruggevonden bij 33% van de ellebooggewrichten met dysplasie. Bij normale ellebogen werd geen verhoogde opname van het radioactief product ter hoogte van het ellebooggewricht gezien. De conclusie van deze studie was dat met HiSPECT de pathologie binnenin het ellebooggewricht kon worden gelokaliseerd en dat het daarbij ook een gevoelige techniek is voor het opsporen van flexor enthesopathie. HiSPECT is echter onvoldoende om een onderscheid te kunnen maken tussen de primaire en concomitante vorm van flexor enthesopathie.

CT criteria (**hoofdstuk 6**) omvatten een onregelmatige, sclerotische, verdikte mediale humerusepicondyl, verdikking van de buigspieren met contrastopname en/of een focaal gebied met verhoogde dentsiteit overeenstemmend met een calcificatie. Deze specifieke kenmerken werden gevonden bij 100% van de klinisch aangetaste gewrichten met primaire flexor enthesopathie en bij 97% van de klinisch aangetaste gewrichten met concomitante flexor enthesopathie. De grootte, vorm en locatie van de letsels waren niet significant verschillend tussen de beide vormen van flexor enthesopathie. In normale ellebogen alsook in de ellebogen aangetast met dysplasie werden geen afwijkingen ter hoogte van de buigspieren teruggevonden. Er kon geconcludeerd worden dat CT een gevoelige techniek is voor het vaststellen van flexor enthesopathie, maar dat een diepgaande analyse van de letsels geen significante verschillen tussen beide vormen van flexor enthesopathie kon onthullen. Aangezien subtiele primaire letsels van de mediale processus coronoideus soms moeilijk te diagnosticeren zijn met behulp van CT is het
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niet altijd mogelijk om een indirect onderscheid te maken tussen beide vormen van flexor enthesopathie.

NMR criteria (hoofdstuk 7) omvatten een onregelmatige, sclerotische mediale humerusepicondyl, verdikking van de buigspieren met contrast opname, en/of een focaal gebied in de buigspier met een lage signaalsterkte of intensiteit overeenstemmend met een calcificatie. Deze kenmerken werden teruggevonden bij 100% van de klinisch aangetaste gewrichten met primaire flexor enthesopathie en bij 96% van de klinisch aangetaste gewrichten met concomitante flexor enthesopathie. Verdikking van de buigspieren was de meest voorkomende bevinding, gevolgd door een hyperintens signaal en contrastopname. Een abnormale aflijning van de mediale humerusepicondyl en de aanwezigheid van een calcificatie werden minder vaak waargenomen. Er werden geen significante verschillen opgemerkt tussen de primaire en concomitante vorm van flexor enthesopathie met betrekking tot de frequentie en details van de letsels. Flexor enthesopathie werd niet teruggevonden in normale gewrichten of deze aangetast met dysplasie. Hoewel NMR een zeer gevoelige techniek is om flexor enthesopathie op te sporen, kan deze niet worden gebruikt om de primaire vorm te onderscheiden van de concomitante vorm.

Artroscopische criteria (hoofdstuk 8) bestonden uit een gefibrilleerde of gescheurde aanhechting van de buigspieren, lokale synovitis en een lokale erosie nabij de aanhechtingsplaats, en/of een verdikking en geelverkleuring van de buigspieren. Bij 100% van de gewrichten behorende tot één van beide groepen van flexor enthesopathie werden één of meerdere van deze kenmerken teruggevonden, maar ook bij 72% van de gewrichten met elleboogdysplasie en bij 25% van de normale gewrichten. Een gefibrilleerde en/of gescheurde aanhechting en een verdikking van de buigspieren werden frequent aangetroffen in de gewrichten met beide vormen van flexor enthesopathie maar werden ook, hoewel minder frequent, waargenomen in gewrichten met elleboogdysplasie en normale gewrichten. Locale erosie en lokale synovitis nabij de aanhechtingsplaats, als ook geelverkleuring van de buigspieren werden uitsluitend teruggevonden in ellebogen met flexor enthesopathy, maar de twee laatstgenoemde kenmerken werden in een significant groter aantal gewrichten met primaire flexor enthesopathie teruggevonden. In 31% van de gewrichten met primaire flexor enthesopathie werd er een onregelmatig aspect van de incisura trochlearis van de ulna opgemerkt. Bovendien werden in 31% van de gewrichten met primaire flexor
enthesopathie subtiele veranderingen ter hoogte van de mediale processus coronoideus waargenomen. Hoewel letsels van de buigspieren gemakkelijk gevisualiseerd kunnen worden met behulp van artroscopie zijn deze bevindingen niet altijd specifiek voor flexor enthesopathie aangezien ze ook teruggevonden worden in andere gewrichten zonder flexor enthesopathie. Vooral in gevallen met discrete letsels ter hoogte van de mediale processus coronoideus is het moeilijk om via artroscopie een onderscheid te maken tussen beide vormen van flexor enthesopathie, niet alleen vanwege de vergelijkbare flexor pathologie in beide vormen maar ook door de aanwezigheid van milde onregelmatigheden van de mediale processus coronoideus in gewrichten met primaire enthesopathie.

Als algemene conclusie van deze doctoraatsthesis kunnen we stellen dat elke beschreven diagnostische techniek specifieke letsels voor flexor enthesopathie kan aantonen. Desalniettemin zijn er ook vals negatieve en vals positieve resultaten, wat betekent dat sommige veranderingen van de buigspieren niet opgespoord kunnen worden in gewrichten met flexor enthesopathie en sommige veranderingen waargenomen worden in gewrichten zonder flexor enthesopathy. Bovendien kan met geen van de beschreven technieken een duidelijk onderscheid worden gemaakt tussen gewrichten aangetast met de primaire en de concomitante vorm van flexor enthesopathie op basis van specifieke criteria voor flexor enthesopathie enerzijds en de aanwezigheid of afwezigheid van andere elleboogaandoeningen anderzijds. Daarom is een combinatie van meerdere beeldvormingstechnieken noodzakelijk. Het voorlopig voorgestelde protocol is de combinatie van CT met contrast en artroscopie als aanvulling op het klinische en radiografische onderzoek. Mogelijk zal dit protocol in de toekomst aangepast worden aan de hand van verder onderzoek.
DANKWOORD
Hoe het allemaal begon.

“Wat ga jij doen?” was de centrale vraag in ons laatste jaar van de studie diergeneeskunde. En alhoewel zeker meer dan de helft van ons jaar een duidelijk antwoord op die vraag wist was het voor mij nog één groot, duister gat. Mede door het schrijven aan mijn laatstejaarsscriptie en door de inspirerende woorden van mijn beste vriendin “ik weet zeker dat jij in het onderzoek terecht komt, daar hoor jij thuis” is het balletje beginnen rollen. De eerste stap werd gezet door bij professor Van Ryssen op haar deur te kloppen en te vragen of ze misschien een geschikt onderwerp had voor een doctoraat en of ze dat eventueel met mij zag zitten. Ze had zeker een geschikt onderwerp – het was iets nieuws en onbekend in de wereld van de orthopedie en ze ging erover nadenken. Na deze “nadenk-periode” was de kogel door de kerk: ik ging een doctoraat starten met als onderwerp: “flexor enthesopathie, een niet gekend elleboogprobleem bij de hond”. De aanvragen voor beurzen werden gestart en ergens op een mooie zomeravond in augustus 2008 “op café” kreeg ik de verlossende sms: ‘WE HEBBEN DE BOF BEURS’!!!! Dat het echte werk pas dan begint kun je enkel maar achteraf beseffen. Het schrijven van een doctoraat is een echt proces, een meerjaren plan met vele ‘ups and downs’ en waar ontzettend veel mensen bij betrokken zijn.

De Hoofdpromotor

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**De “medical imagers” van radiografie&echografie**

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**De “medical imagers” van de CT en MRI unit**

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Dankwoord

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Dankwoord

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Stijn “grote huisdieren”: De laatste fase van dit doctoraat heb je redelijk van dichtbij mogen (en verplicht moeten?!?) meemaken en menig zaag/zeur/klaaguurtje is er gepasseerd op onze buro’s. Maar uiteindelijk wint de aanhouder altijd, daar zijn wij het levende bewijs van. Doe dat nog goed, binnen een maand is het aan jou!!!

David&Valentine: ik heb het altijd heel tof gevonden om met jullie samen te werken op de unief. We gaan nu echt ons best doen om snel eens af te komen voor een gezellige strandwandeling en tour van jullie huis+praktijk!!

**Recente aanwinst**

En ineens had ik er een hele nieuwe vriendengroep en familie bij!!!

Seppe&Karlien: dankzij jullie trouw heeft mijn leven een hele andere wending aangenomen. Zou ik na mijn doctoraat hoogstwaarschijnlijk terug richting Nederland zijn getrokken, nu blijft mijn leven zich ineens verder in België afspelen. En dat enkel en alleen door een beste vriend, alias getuige... Bedankt voor de toffe vriendschap, voor de vele gezellige avondjes samen, dagjes Mechelen, bbq’jes, praatjes en adviezen over het doctoraat...Succes met alles in jullie (nabije) toekomst en wie weet Karlien ben jij op dit moment ook wel aan het presteren ;-)!

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Ben&Dorien: zouden we nu eindelijk eens samen kunnen eten? Of überhaupt iets samen kunnen doen?!? Ik ga er in elk geval mijn best voor doen! Bedankt voor alle support!

Anneke&Stefan, Mien&Haroen, Bjorn en Patrick: bedankt voor telkens weer een gezellig samenzijn en hopelijk gaan we daar nog heel lang mee door!
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_Pappak&mamma_

Ik kan me echt geen betere pappie en mammie wensen!!! Welke ouders zijn zo gek om je weer eens met de auto tot ergens bij Roosendaal te brengen omdat de treinen weer eens niet wilden rijden of komen je halen op Utrecht Centraal nadat er echt geen andere mogelijkheid meer was om tot in Leiden te geraken. Van een half ingestort huisje in de Ruststraat tot een appartement in het centrum van Gent: de studie in Gent heeft toch heel wat uitzonderlijke situaties opgeleverd! Bij elke examenperiode weer waren er de nodige huilpartijen, “ik zie het niet meer zitten”, strandwandelingen om de hersentjes te ventileren en peptalks. Ik zou jullie telefoonrekening van toen niet graag weten..! Gelukkig had studeren in het verre Gent ook voordelen: gezellige weekendjes Gent, kerstmarktjes overal in België, antiekmarktjes op zondagochtend vroeg...Gaandeweg kwamen er nog de nodige hobbels (terug naar Nederland of toch doctoren) die door jullie allemaal als sneeuw voor de zon verdwenen. Ook nu nog tijdens de laatste fase van het doctoraat heb ik op jullie onvoorwaardelijke steun mogen rekenen.
In de ideale wereld zouden jullie wel veel dichterbij wonen, zodat ik jullie vaker zou kunnen zien als nu. Maar gelukkig blijven de weekendjes samen even bijzonder, in Leiden of in Lokeren. Immens en ontelbaar veel dank voor alles!

_Den grote broer_

Ro’tje, mijn grote broer, mijn trots. ‘Weinig woorden zeggen veel’ is zeker jouw motto! Ook al zien we elkaar nu veel te weinig, ik weet dat je er altijd voor me bent! Hopelijk zien we elkaar in de toekomst toch wel iets vaker, want ik mis je stiekem toch wel heel erg veel...Heel veel succes in de toekomst met Judith en mijn allerliefste, kleinste, schattigste nichtje Saar! Judith, ook heel erg bedankt voor je steun in de moeilijkere periodes maar ook in de goede periodes. Het ver weg wonen zal altijd een lastig iets blijven en ik waardeer het enorm dat jullie ook zo’n moeite blijven doen om af te spreken als we dan weer in Leiden zijn. Jullie mogen zo trots zijn op Saartje! Ik ben er verliefd op!!!! 😊
Dankwoord

Maxxje/Milsaantje/”den bruine"

Mijn dank voor jou wordt uitgesproken (of beter: uitgebeeld) op de voorkant…Voor altijd vereeuwigd op één van de belangrijkste prestaties van mijn leven..Wat een goede keuze om jou in mijn leven te laten! Vanaf het eerste moment dat we elkaar zagen zijn we maatjes door dik en dun en ben je mijn beste vriendje. Iedere dag laat je me wel ergens om lachen, iedere ochtend sta ik goedgezind op en iedere chagrijnige bui verdwijnt door jouw vrolijk karakter. Je hebt me de studie diergeneeskunde doorgeslept en niet veel later dit 4-jaar durend project. Jouw onuitputtelijk drang om te spelen en vrolijk te zijn is machtig! Ik hoop dat we nog lang van jou mogen genieten! Een hele dikke dank-je-wel knuffel!

Den Belgische prins op het witte paard

En daar was jij ineens, Tomas. Totaal onverwachts, maar des te leuker!! Wat een “best man” al niet kan doen…Vanaf het moment dat jij mijn leven bent binnen gefietst (😊) is het allemaal veel leuker, spannender, boeiender, drukker, hectischer…. Iedere dag wakker worden is een feest, en ieder weekend is een zoektocht om alles wat we willen doen in dat ene weekend te kunnen proppen..! Enorm bedankt om steeds weer al die kilometers af te leggen naar “boven de rivieren”, zodat mijn leventje in Nederland ook niet vergeten wordt. Bedankt ook om zo goed voor ons allerliefste Maxxje te zorgen, die iedere dag rond 18u uitzinnig blij is met z’n duiveltje in z’n bek als jij de deur binnenstapt! Ontelbaar veel dank-je-wel om het de laatste tijd met mij uit te houden, om alle zaagmomenten persoonlijk en via mail te ontberen, en om dit doctoraat mede tot een goed eind te brengen! Je was en bent zo ongelofelijk lief voor mij, ik hoop dat dat nooit verandert!! Ik kijk enorm uit naar onze toekomst samen, inclusief de Belgisch-Nederlandse tegenstellingen en miscommunicaties...😊 En ook al bevat ons leven samen soms wat hobbels (jij hebt nu eenmaal een Hollandse uitgekozen!) ik weet zeker dat we er iedere keer weer overheen zullen fietsen 😊!!!!
Ik zie je super graag!!!!!

Evelien

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Door het schrijven aan haar laatsteklascriptie “De artroscopische behandeling van een losse processus coronoideus in erg incongruente ellebooggewrichten: een lange termijn follow-up studie”, die bekroond werd met een prijs voor de beste scriptie in verband met een diergeneeskundig onderwerp, raakte zij zeer geïnteresseerd in de orthopedie. De keuze voor een combinatie van wetenschappelijk onderzoek en kliniek orthopedie was dan ook snel gemaakt. Na de succesvolle aanvraag van een doctoraatsmandaat startte zij haar doctoraat op 1 september 2008 onder leiding van Prof. Dr. Bernadette Van Ryssen aan de vakgroep Medische Beeldvorming van de Huisdieren en Orthopedie van de kleine Huisdieren, Universiteit Gent. Het doctoraatsmandaat werd gefinancierd door het Bijzonder Onderzoeksfonds van de Universiteit Gent. Tevens volgde zij de Doctoraatsopleiding in de Diergeneeskundige Wetenschappen en de opleiding Laboratory Animal Science.

Evelien de Bakker is auteur en mede-auteur van verschillende publicaties in wetenschappelijke nationale en internationale tijdschriften en zij heeft verschillende (poster) presentaties gegeven op diverse congressen.
PUBLICATIONS


SCIENTIFIC PRESENTATIONS


Gielen I, Van Ryssen B, **de Bakker E**, van Bree H. Magnetic resonance (MRI) features of flexor enthesopathy (FE) in the canine elbow. Proceedings of the 8th European Veterinary MR User days, May 11th-12th, 2012, Ghent, Belgium. pg 84-85. (Oral presentation)


“The only way to make your dreams come true is to wake up”

Paul Valery