Treatment of glomus tympanicum tumors by preoperative embolization and total surgical resection

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Purpose: The purpose of this study was to evaluate the effectiveness on function preservation and tumor control of the treatment of glomus tympanicum tumors with pre-operative embolization followed by total surgical resection.

Material and methods: We describe a series of 6 patients with a glomus tympanicum tumor who were treated in our hospital using the same technique: the day before surgery selective tumor embolization due to denaturation with 96% ethanol. Following parameters were considered: tumor classification, tumor control, clinical and audiological outcome, effectiveness of embolization, percentage of tumor necrosis and treatment complications.

Results: There were no severe complications due to embolization or surgery. Tumor blush disappeared completely in 5 patients on DSA post embolization and histologic evaluation of the resected tissue showed a median of 69.2% of tumor necrosis. Pulsatile tinnitus disappeared in all patients and 3 patients had no symptoms at all. Hearing ameliorated in 4 patients, 1 patient without hearing loss pre-treatment still had normal hearing after treatment and 1 patient's hearing was worse after treatment. Average follow-up was 21.3 months.

Conclusions: Treatment of glomus tympanicum tumors by pre-operative embolization with ethanol and surgical resection has not been described before. Our results show that it is a safe procedure with a good long term tumor control, good clinical and audiological outcome.

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Abbreviations: ABG, Air Bone gap; AC, Air Conduction; BC, Bone Conduction; DSA, Digital subtraction angiography; HRCT, High-resolution CT images; PTA, pure tone average; SRS, stereotactic radiosurgery.
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1. Introduction

Paragangliomas, also named glomus tumors or chemodectoma, are rare highly vascular neuroendocrine neoplasms arising from neuroectodermally derived paraganglionic cells located in the walls of blood vessels or associated with specific nerves [1]. They have an estimated incidence of 1 in 1.3 million people [2]. Head and neck paragangliomas comprise 3% of all paragangliomas [3,4] and their most common site is the carotid body, followed respectively by the tympanic plexus, jugular bulb and vagal nerve [5]. Jugulotympanic paragangliomas represent the most common primary neoplasm of the middle ear [5–7]. They are usually benign, slowly growing, painless tumors with the potential to remain stable over years [7,8]. Their growth pattern is multidirectional along the tracts of least resistance. They grow primarily in the air filled spaces of the temporal bone. Vascular lumina, neurovascular foramina, and the Eustachian tube provide routes of extra-temporal extension, into the infratemporal fossa or along the skull base [9]. Classifications according to Fisch (Table 1) or Glasscock-Jackson are most used [10–12].

1.1. Diagnosis

In tympanic paragangliomas symptoms are produced at an early stage, i.e. pulsatile tinnitus and conductive hearing loss [5]. In larger tumors, sensorineural hearing loss, ear pain, ear discharge, bleeding and dizziness often occur [13]. They commonly present as a vascular middle ear mass. They may transgress the tympanic membrane and appear as an inflammatory polyp [6].

Clinical diagnosis is confirmed by imaging (Fig. 1). HRCT images with bone window display are very sensitive for delineating the extent of bone destruction and are therefore essential for classification and surgical planning [6]. Magnetic resonance imaging is sensitive for defining the soft tissue involvement. T1- and T2-weighted images after gadolinium enhancement and/or fat-suppression sequences are used. On T1-weighted images, paragangliomas have a typical ‘salt-and-pepper’ appearance [14]. MR angiographic images can be created, with identification of the tumor’s feeding vessels (most commonly the inferior tympanic branch of the ascending pharyngeal artery [1,9,15]) and the venous flow. DSA [16–19] provides an arterial ‘map’ and flow dynamics of the blood supply and it is a sensitive diagnostic study for detecting multiple paraganglioma [19–23]. Because of its invasive nature, it is only performed if followed by tumor embolization [5,20] or if there is need to assess the contralateral arterial supply.

1.2. Management

The attitude toward therapy goals has changed and more and more a function preserving treatment (maintenance of cranial nerve function, hearing preservation or amelioration) is emphasized, next to a high level of tumor control. To date, surgery remains the only curative treatment option and preoperative embolization has been introduced to facilitate surgical removal [13,24–26]. Absolute ethanol can be used for embolization as it is a sclerosing agent. It denudes the endothelial cell from the vascular wall, induces a spastic vasoconstriction with disruption of the vessel wall at the internal elastic lamina and as a result a blood clot is formed. This results in complete and permanent obliteration of the vessel lumen [27,28]. Ethanol is toxic to the system and the maximal given dose is 1 mL/kg body weight/day [29].

The role of preoperative embolization is still a matter of debate [1,26,30–34]. In this paper we will describe a group of patients with tympanic paranganglioma who were treated with preoperative embolization and surgical resection and we will examine tumor control rate, function preservation and safety.

2. Material and methods

Between 2006 and 2014, a total of 6 patients with a tympanic paranganglioma were treated with preoperative embolization with ethanol and total surgical resection with a function preserving intent at the ENT Department of the Ghent University Hospital in collaboration with the Department of Interventional Radiology. Approval of the UZ Ghent ethical committee was obtained for a retrospective review of the patient’s medical and imaging records. There were one male (age 66 years) and 5 females (mean age 58.8 years; range 54–69 years). The tympanic parangangliomas were categorized in accordance with the Fisch classification; 5 were class A. 1 patient had a class B tumor and was first treated with embolization alone but because of tumor growth, she was treated 4 years later with preoperative embolization and surgical resection.

Micro-otoscopic examination and evaluation of the cranial nerves were performed before and after treatment. Preoperative and postoperative audiograms were obtained.

### Table 1 – Fisch classification *

<table>
<thead>
<tr>
<th>Class</th>
<th>Description</th>
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</thead>
<tbody>
<tr>
<td>A</td>
<td>(glomus tympanicum) tumors arise along the tympanic plexus on the promontory of the middle ear. The blood supply is from the tympanic artery, a branch of the ascending pharyngeal artery. Class A tumors may produce minimal erosion of the promontory.</td>
</tr>
<tr>
<td>B</td>
<td>(glomus hypotympanicum) tumors originate in the canalis tympanicus of the hypotympanum and invade the middle ear and mastoid. The carotid foramen and canal are intact. By definition, class B tumors invade the bone of the hypotympanum, but the cortical bone over the jugular bulb is intact.</td>
</tr>
<tr>
<td>C</td>
<td>(glomus jugulare) tumors originate in the dome of the jugular bulb and destroy the overlying cortical bone. They may spread in the following directions: inferior, along the jugular vein and cranial nerves IX, X, XI, XII; posterior, into the sigmoid sinus; superior, toward the internal auditory canal and otic capsule; lateral, to the hypotympanum and middle ear; and medial, to the jugular foramen and cerebellopontine angle. Further classification is based on the degree of erosion of the carotid canal.</td>
</tr>
<tr>
<td>C1</td>
<td>tumors erode the carotid foramen, but do not invade the carotid artery.</td>
</tr>
<tr>
<td>C2</td>
<td>tumors destroy the vertical carotid canal between the carotid foramen to the carotid bend.</td>
</tr>
<tr>
<td>C3</td>
<td>tumors grow along the horizontal portion of the carotid artery but do not reach the foramen lacerum.</td>
</tr>
<tr>
<td>C4</td>
<td>tumors grow to the foramen lacerum and along the carotid artery to the cavernous sinus.</td>
</tr>
<tr>
<td>D</td>
<td>(most commonly the inferior tympanic branch of the ascending pharyngeal artery [1,9,15]) and the venous flow. DSA [16–19] provides an arterial ‘map’ and flow dynamics of the blood supply and it is a sensitive diagnostic study for detecting multiple paraganglioma [19–23]. Because of its invasive nature, it is only performed if followed by tumor embolization [5,20] or if there is need to assess the contralateral arterial supply.</td>
</tr>
</tbody>
</table>

Pre- and postoperative Air Conduction (AC) and Bone Conduction (BC) thresholds (at frequencies 0.5, 1, 2 and 4 kHz) were recorded for each case and the pure tone average was calculated (PTA). Subtracting the individual pre- and postoperative average BC threshold from the pre- and postoperative average AC thresholds provided the values of the average Air Bone gap (ABG) at pre- and postoperative evaluations. Preoperative audiograms were compared with the postoperative audiograms for differences in AC, BC thresholds and ABG.

HRCT of the petrous bone and MRI of the inner ear and posterior fossa were carried out in all patients. One day preoperatively, DSA was carried out in all patients with subsequent embolization of the feeding vessel(s) when possible. The follow-up period was defined as that period of the time from surgery to the most recent office visit. Criteria for successful tumor control included no recurrence of symptoms, a normal microscopic evaluation of the middle ear and normal imaging when available.

2.1. Embolization procedure

Preoperative embolization started with a diagnostic DSA, performed under general anesthesia via a transfemoral Seldinger approach. After selective probing of the common, external and internal carotid artery with a 5F guiding catheter, biplanar angiography of the skull base and petrous bone was achieved using non-ionic contrast medium (Iodixanol 270, GE Healthcare). The tympanic paraganglioma was delineated by a typical homogenous hypervascular blush. After vascular mapping, a microcatheter (Marathon, EV3; Covidien) was introduced through the guiding catheter into the tumor feeder and advanced up to the petrous bone. After confirming the tumor blush on superselective contrast injection, embolization started with pure ethanol. The microcatheter was repeatedly loaded with 0.05–0.1 cm³ of pure alcohol and subsequently slowly flushed with saline by a pump infusion at a rate of 3–6 cm³/h. After each injection, superselective control angiography demonstrated the gradual disappearance of the tumor blush. Once denudation of the tumor artery was achieved, the embolization was stopped. Catheter systems were then retrieved and the femoral artery sealed with an AngioSeal closure device. During the procedure, the patient was kept under heparin. The technical success of the embolization procedure was determined by the residual percentage of parenchymal staining of the tumor on post-embolization DSA.

2.2. Surgical technique

A retro-auricular tympanic access route with canaloplasty was used. Depending on the location and size of the glomus tumor, an endaural approach to the middle ear with additional mastoidectomy and myringoplasty was performed. Ossicular reconstruction was done if needed.

2.3. Procedure of histological preparation

The resected specimen was immersed into 4% buffered formalin, routinely processed and embedded in paraffin. 4 μ sections were made, stained with HE and histologically analyzed. Additional immunohistochemistry was performed, applying antibodies against S100-protein, chromogranin and synaptophysin. S100-protein demonstrates the sustentacular cells around the groups of cells displaying neuroendocrine activity as demonstrated with the antibodies against chromogranin and synaptophysin. Histologically, the resected tumors revealed a spectrum from severe congestion up to (partial or complete) necrosis. The positivity of the immunohistochemical staining was determined by the viability of the tumor cells.

3. Results

The most frequently reported symptoms were pulsatile tinnitus and hearing loss (Table 2). None of the patients had a pattern of familiar occurrence. A reddish bulging mass in the middle ear was seen in all patients on micro-otoscopy, the mass was invading in the external ear canal in 1 patient and the mass was...
clearly pulsating in another patient. Cranial nerve evaluation showed normal function in all patients. Evaluation of hearing pre treatment showed normal hearing in 2 patients, conductive hearing loss in 3 patients and mixed predominant sensorineural hearing loss in 1 patient. Pre treatment audiometric AC and BC thresholds are shown in Fig. 2.

In all patients HRCT showed soft tissue masses in the middle ear which appeared to be hypervascular on MRI with typical features matching tympanic paragangliomas. The tumors were divided according the Fisch classification; 5 had a class A tumor. One patient had a class B tumor: there was complete obliteration of the tympanic cavity and the mastoid but there was no bony erosion. The patient was first treated with embolization alone but there was tumor growth of the residual tumor. Four years later, she presented with a class B tumor which completely obliterated the tympanic cavity with lateral extension into the external ear canal, bony erosion of the antero-inferior wall with transgression of the temporomandibular joint, increased invasion of the Eustachian tube and increased extension into the antrum.

Angiography revealed that the ascending pharyngeal artery or one of its branches was the feeding vessel of the tumor in 5 patients (Table 2). In 3 patients this was the only feeding vessel; in 2 patients there was also blood supply from the middle meningeal artery. Subsequent super selective embolization of the ascending pharyngeal artery, the stylomastoid artery or posterior auricular artery with skeletonizing of the vessels was successful. Tympanic feeders from the middle meningeal arteries (involved in 2 patients) were not touched to avoid facial nerve damage. Tumor blush disappeared completely in 5 patients (Fig. 3). In the patient who was treated twice, blood supply from the middle meningeal artery was recognized but not

### Table 2 - Patient characteristics.

<table>
<thead>
<tr>
<th>patient</th>
<th>gender</th>
<th>age</th>
<th>Fisch</th>
<th>pulsatile tinnitus</th>
<th>hearing loss</th>
<th>ear pressure</th>
<th>otalg</th>
<th>otorrhea</th>
<th>itching ear</th>
<th>vertigo</th>
<th>tinnitus</th>
</tr>
</thead>
<tbody>
<tr>
<td>A 2006</td>
<td>F</td>
<td>55</td>
<td>B</td>
<td>+</td>
<td>+</td>
<td>−</td>
<td>+</td>
<td>−</td>
<td>−−</td>
<td>−</td>
<td>−</td>
</tr>
<tr>
<td>A 2010</td>
<td>B</td>
<td>66</td>
<td>A</td>
<td>+</td>
<td>+</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>+−</td>
<td>−</td>
<td>−</td>
</tr>
<tr>
<td>C</td>
<td>F</td>
<td>54</td>
<td>A</td>
<td>+</td>
<td>−</td>
<td>+</td>
<td>−</td>
<td>−</td>
<td>−−</td>
<td>+</td>
<td>−</td>
</tr>
<tr>
<td>D</td>
<td>F</td>
<td>58</td>
<td>A</td>
<td>+</td>
<td>+</td>
<td>−</td>
<td>+</td>
<td>−</td>
<td>−−</td>
<td>−</td>
<td>−</td>
</tr>
<tr>
<td>E</td>
<td>F</td>
<td>69</td>
<td>A</td>
<td>−</td>
<td>+</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>−−</td>
<td>−</td>
<td>−</td>
</tr>
<tr>
<td>F</td>
<td>F</td>
<td>58</td>
<td>A</td>
<td>+</td>
<td>+</td>
<td>−</td>
<td>+</td>
<td>−</td>
<td>−−</td>
<td>−</td>
<td>−</td>
</tr>
</tbody>
</table>

**Fig. 2** - Pre and post treatment audiometric AC and BC thresholds of each patient (A→F). Black lines are pre treatment thresholds, gray lines are post treatment thresholds; solid lines are AC thresholds, dashed lines BC thresholds. If AC thresholds coincide with BC, only the AC is shown.
 obliterated. Devascularization in this patient was each time incomplete (80%). No serious complications occurred during or after the intervention. One patient developed a hematoma at the groin at the puncture site, which resolved spontaneously. In 5 patients the pulsatile tinnitus disappeared immediately after embolization and in 1 patient there was no change in tinnitus.

Surgery was performed within 24 h after embolization (Table 3). A retro-auricular tympanic access route was chosen. To obtain sufficient tumor access, an additional canaloplasty was performed in all patients and a mastoidectomy was necessary in 2 patients (canal wall up procedure in 1 patient and a canal wall down (CWD) procedure in the 1 patient with class B tumor because of extensive erosion of the posterior canal wall). Invasion of the ear drum made a myringoplasty necessary in 3 patients. Reconstruction of hearing was performed in the 1 patient with CWD procedure; in the other patients the ossicular chain was kept intact during tumor removal. An easy resection of the tumor was achieved with preservation of middle ear mucosa and almost no blood loss during the operation in 5 of the procedures. In 1 patient, the feeding vessels of the tumor at the level of the hypotympanum were visible and clearly occluded. In the patient with Fisch class B, the procedure was more complex and bloody. A blind resection at the level of the Eustachian tube area was performed with suspicion of residual disease.

Histopathologic and immunohistochemical evaluation (positive staining for S100 protein, chromogranin and synaptophysin) of the resected tissue confirmed diagnosis of paraganglioma in 5 patients. In 1 patient the remaining resected specimen demonstrated nearly complete necrosis. Percentages of tumor

Table 3 – Treatment characteristics.

<table>
<thead>
<tr>
<th>Patient</th>
<th>Arterial supply</th>
<th>Embolized feeder</th>
<th>mL of ethanol</th>
<th>Technical result</th>
<th>Surgical procedure</th>
<th>% tumor necrosis</th>
<th>Tumor dissection</th>
<th>Symptoms post treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>A 2006</td>
<td>aPA mA</td>
<td>aPA</td>
<td>0.42</td>
<td>20%</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>recurrence pulsatile tinnitus (after 4 years) pulsatile tinnitus, otorrhea, sensory disorder facial half myringitis</td>
</tr>
<tr>
<td>A 2010</td>
<td>post Auricular A</td>
<td>Post Auricular A</td>
<td>0.3</td>
<td>20%</td>
<td>CWD + type III tympanoplasty</td>
<td>15</td>
<td>partial blind dissection, bloody sharp, total, bloodless sharp, total, bloodless</td>
<td></td>
</tr>
<tr>
<td>B aPA</td>
<td>aPA</td>
<td>aPA</td>
<td>0.25</td>
<td>none</td>
<td>CWU + canaloplasty + myringoplasty canaloplasty</td>
<td>30</td>
<td>–</td>
<td></td>
</tr>
<tr>
<td>C stylomastoideal A</td>
<td>Stylomast. A</td>
<td>0.5</td>
<td>none</td>
<td>canaloplasty + myringoplasty canaloplasty</td>
<td>95</td>
<td>100</td>
<td>silent tinnitus (non pulsatile)</td>
<td></td>
</tr>
<tr>
<td>D aPA</td>
<td>aPA</td>
<td>aPA</td>
<td>1.0</td>
<td>none</td>
<td>canaloplasty + myringoplasty canaloplasty</td>
<td>80</td>
<td>100</td>
<td>silent tinnitus (non pulsatile)</td>
</tr>
<tr>
<td>E aPA</td>
<td>aPA</td>
<td>aPA</td>
<td>0.3</td>
<td>none</td>
<td>canaloplasty + myringoplasty canaloplasty</td>
<td>95</td>
<td>–</td>
<td>silent tinnitus (non pulsatile)</td>
</tr>
<tr>
<td>F aPA</td>
<td>mA</td>
<td>aPA</td>
<td>0.5</td>
<td>none</td>
<td>canaloplasty + myringoplasty canaloplasty</td>
<td>95</td>
<td>–</td>
<td>silent tinnitus (non pulsatile)</td>
</tr>
</tbody>
</table>

aPA = ascending pharyngeal artery; mA = middle meningeal artery (petrosal branch); CWD = canal wall down mastoidectomy; CWU = canal wall up mastoidectomy.
necrosis on the resected specimen were estimated by the pathologist, and a median of 69.2% of necrosis (range 15%–100%) was reported (Fig. 4, Table 3).

The average length of follow-up post surgery was 21.3 months (range 1–53 months). Three patients were free of symptoms. One patient had a silent non-pulsatile tinnitus. In 4 patients, otoscopy was normal at clinical evaluation within 2 months postoperation. One patient developed a myringitis but there were no signs of recurrence of the tumor on otoscopic evaluation during follow-up. The patient who underwent the CWD procedure showed a cumbersome healing of the cavity. Eventually, after one year a stable and dry mastoid cavity was obtained. She still complains of hemifacial sensory disorders and pulsating tinnitus with further progression of sensorineural hearing loss. On micro-otoscopy residual tumor is visible at the level of the oval window. On MRI, there is residual disease at the oval window and at the level of the Eustachian tube. No tumor growth was seen during the 4 years of follow up of this patient (clinical examination and MRI evaluation are performed every year).

Post treatment audiometric AC and BC thresholds are shown in Fig. 2. The postoperative audiometry was performed after a mean interval of 4.8 months (range 1–23 months). Hearing ameliorated in 2 patients with median AC threshold differences (pre-op vs post-op PTA) of 26.25 dB and 10.00 dB and reduced median ABG (pre-op vs post-op) of respectively 27.50 dB and 3.25 dB. There was no change in hearing in 1 patient without hearing loss pre treatment, 1 patient with limited conductive hearing loss pre treatment and 1 patient with mixed predominant conductive hearing loss pre treatment (pre-op minus post-op AC PTA of respectively −3.75 dB; 1.25 dB; −2.50 dB). One patient with normal hearing pre treatment had a predominant conductive hearing loss after treatment with AC PTA differences of 26.25 dB and median increase of ABG of 18.75 dB. Follow-up audiometry of this patient however showed amelioration with at 16 months post treatment a hearing loss of 13.75 dB (AC PTA differences) and median increase of ABG of 7.5 dB compared with the pre operative measurement.

The tumor control rate following primary preoperative embolization and surgery was 100% (n = 5). The 1 patient with primary embolization alone and secondary treatment with preoperative embolization and surgery 4 years later still had residual disease post surgery, which stayed stable until the present (4 years post treatment).

4. Discussion and conclusion

In the treatment strategy for a number of medical conditions, there is an increasing concern for function preservation and quality of life. Paragangliomas are mostly benign, slow growing tumors and patients desire symptom control and prevention of neurologic compromise. Tympanic paragangliomas are symptomatic at an early stage presenting with very disturbing pulsatile tinnitus and progressive hearing loss that require treatment. Surgery remains the only curative treatment option, aiming at eradicating the tumor. To facilitate surgery by reducing operative blood loss and tumor size [13,24–26], preoperative embolization has been introduced and was first reported in 1973 for paragangliomas [24]. Until the present, there is no consensus about the indications for preoperative embolization of head and neck paragangliomas. In general, previous literature concludes that it is not advocated in tympanic paragangliomas that are confined to the middle ear cavity (Fisch class A) because the risk of potential complications of this invasive procedure are not outweighing the advantages [1,13,31–34]. The risk of severe complications (possible severe side effects include skin necrosis, blindness, cranial nerve palsies, stroke and death) is however generally low, ranging from 0% to 13% [1,13,33,35–41].

In our patient group with tympanic paragangliomas, preoperative embolization with 96% ethanol clearly did facilitate surgery and thus made complete resection with healthy margins more certain. Also, with the goal of function preservation kept in mind, the reduced tumor size resulted in less manipulation of surrounding structures [1], creating good chances to preserve hearing. In 4 of the 6 patients, preservation or improvement of hearing could be achieved.

Evaluation of pre- and postoperative hearing results for tympanic paraganglioma treated by surgery alone, was performed by Gjuric et al. [42], Papaspyrou et al. [43] and O’Leary et al. [44]. The first treated a group of 10 patients: 8 had a stable hearing and 2 patients had an increase of hearing loss. Papaspyrou et al. [43] treated 17 patients and the AC hearing thresholds improved, on average, by 3.6 dB at 500 Hz, 4.7 dB at 1000 Hz, 5.9 dB at 2000 Hz and 8.5 dB at 3000 Hz. O’Leary et al. [44] reported on 64 patients and had a reduction of mean AB-gap from 10 dB preoperation to 4 dB postoperation. However, this improvement in conductive hearing was accompanied by a...
The effectiveness of embolization hinges upon occlusion of the glomus feeding tumoral vessels. The catheterization technique should be superselective, aiming only at the feeding vessel of the paraganglioma. The most common feeding artery is the ascending pharyngeal artery [32,37,45], in particular it’s inferior side-branch which ascends in the middle ear cavity. However, with tumor growth, supply is recruited from the petrosal branch of the middle meningeal, the retroauricular artery or the stylomastoideal artery. Fastidious awareness of potential anastomoses with the internal carotid arteries and vertebral artery is mandatory to avoid paradoxical cerebral embolization [32,46–48]. Neurological complications might occur by opening of such anastomosis after the tumor bed has been occluded. This is a well-known risk, especially after embolization with particles. Therefore, in such small tumors, we preferred the use of 96% ethanol as a liquid embolic agent. By the slow injection technique we applied, the devascularization occurs inside the capillary bed of the tumor. This technique has several advantages. Firstly, by embolizing on tumor level, other feeders from the above mentioned supplementary supply have no change to maintain blood supply. In case of particle embolization, the size of the particles should be under 50 μ in order to enter the capillaries (and avoid proximal occlusion). However, such small particles can shunt into the anastomoses and subsequently block cerebral capillaries and cause neurological deficits. Secondly, there is no proximal occlusion of the feeding artery with possible reflux to the dangerous anastomoses. Also distal migration into the cerebral systemic circulation is avoided, and if it would happen the minimal amount of ethanol will be diluted fast. By intermediate angiographic control, the opening of the risky anastomosis will be detected very early. Thirdly, we avoid compromising cranial nerve supply (facial nerve, glossopharyngeal, hypoglossal nerve etc.), which is caused by overflow of particles in side-branches or anastomosis to the territory of the stylomastoid artery and the petrosal branch of the middle meningeal artery [49]. So embolization with 96% ethanol is a safe adjuvant to surgical resection.

The efficacy of the embolization procedure is based on the angiographic disappearance of the tumor blush, the absence of complications, the evolution of the symptoms, and in our case also on the completeness and safety of surgical resection. In all our cases with a single supply, tumor blush was eradicated angiographically. In 2 cases with a risky supplementary supply, no attempt to embolize these vessels was undertaken to avoid neurological complications. An 80% to 90% reduction in tumor vascularity is often obtained [13,50]. Also a reduction in tumor size as much as 25% has been described [1]. In our evaluation, we saw necrosis to as much as 100% of the glomus tumor on the histopathologic evaluation of the resected specimen.

Several papers have dealt with management of paragangliomas, but very few have provided insight into the longevity of successful outcomes [42] and there currently is a lack of Class I data reflecting recurrence risk to direct appropriate management [51–53]. No data were found concerning long-term follow-up after preoperative embolization and surgery in jugulotympanic paragangliomas. However long-term follow-up after surgical resection was evaluated by Jackson et al. [52] for jugulotympanic paragangliomas and by Forest et al. [6] for tympanic paragangliomas. The first reported recurrence in 5% of patients and 78% of these occurred within the first 10 postoperative years. The latter showed a long term tumor control rate of 92.5% and recurrence occurred 14 years after surgery. Both conclude that long term postsurgical surveillance is recommended but imaging is not routinely advised.

Tumor recurrence seems to correlate with tumor size, location and previous treatment [11,54]. In our series, none of the 5 preoperatively embolized and totally resected glomus tympanicum reoccurred during an average follow-up of 21.3 months. One patient who presented with a more advanced Fisch type B paraganglioma at diagnosis showed continuing growth after an initial treatment with embolization alone with non-curative intent. For small residual tumors with difficult surgical access, stereotactic radiosurgery (SRS) might be a useful treatment option with good tumor control. In the majority of cases treated with SRS, the tumor remains stable or continues to shrink radiographically. In the meta-analysis of Ivan et al., SRS appeared to provide better tumor control than surgery (total resection), with less risk, at least over the published follow-up periods (follow-up of 88 ± 5 months for gross total resection, follow-up of 71 ± 4.9 months for SRS) [51].

In this paper, we describe a group of patients with tympanic paraganglioma who were treated with preoperative embolization with absolute ethanol and surgical resection. The combined procedure was safe and resulted in long-term complete control of local symptoms, preservation of cranial nerve function and hearing amelioration or stabilization in 4 out of 6 patients.

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REFERENCES


