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Clinicoradiologic Characteristics of Temporal Bone Meningioma: Multicenter Retrospective Analysis

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Objectives/Hypothesis: Meningioma is a neoplasm arising from cells related to the arachnoid villi. The aim of the present study was to explore the clinical and radiological characteristics of temporal bone meningioma (TBM) in a multicenter cohort.

Study Design: Retrospective cohort study.

Methods: Thirteen patients diagnosed with TBM at eight medical institutes between 1998 and 2018 were retrospectively enrolled. The clinical procedures, symptoms, signs, and images that led to the diagnosis of TBM were investigated for all patients.

Results: The most common symptom at the initial visit was hearing loss (n = 12/13, 92.3%). All patients exhibited unilateral TBMs with varied symptom durations (1–60 months). Four patients presented masses occupying the external auditory canal; the tympanic membrane (TM) could not be evaluated. The other nine patients did not show TM perforation despite the presence of inflammatory signs. The majority of patients exhibited unilateral conductive or mixed hearing loss. A retrospective review of temporal bone computed tomography (TBCT) images revealed findings suggestive of a tumor in all patients. However, three patients had been misdiagnosed with chronic otitis media and were subjected to tympanomastoidectomy (n = 3/7, 42.9%). TBCT findings that suggested TBM included diffuse trabecular hyperostosis in the middle and posterior cranial fossae and widening and destruction of the temporal bone in the jugular bulb area.

Conclusions: TBM should be suspected if patients exhibit persistent inflammatory symptoms or signs involving intact TM or unilateral conductive or mixed hearing loss with trabecular hyperostosis or destruction of the temporal bone on computed tomography images.

Key Words: Meningioma, temporal bone, computed tomography, hearing loss, multicenter study..

Level of Evidence: 4

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INTRODUCTION

Meningioma is a neoplasm arising from the arachnoid cap cells and dural border cells related to the arachnoid villi. It is the second most common primary intracranial neoplasm, accounting for 13% to 25% of all cases.[1] Common locations include the falx cerebri (parasagittal/falcine meningioma, 25%), brain surface (convexity meningioma, 19%), and sphenoid ridge (sphenoid wing meningioma, 17%), followed by the skull base near the pituitary gland and optic nerve (suprasellar meningioma, 9%), olfactory groove (olfactory grove meningioma, 8%), and posterior fossa (posterior fossa/petrous meningioma, 8%). A secondary extracranial extension or primary ectopic occurrence has also been reported. The reported prevalence of extracranial extensions is 6% to 20%,[2,3] with the most common site being the orbit, followed by the paranasal sinus, temporal bone, and other head and neck regions accessed through several foramina in the skull base. Primary ectopic meningioma, defined as extracranial meningioma with no evidence of an intracranial mass, is extremely rare (0.4%) and can involve the orbit, eyelid, paranasal sinuses, parotid gland, temporal bone, and zygoma.[1,4–7]

Temporal bone meningioma (TBM) can ectopically originate in the temporal bone or develop as a contiguous extension of an intracranial mass.[1] It is an extremely rare disease entity, with a limited number of case reports or series.[2,8–16] Most TBMs develop as extensions of primary intracranial tumors, with common pathways for extension including the tegmen tympani, posterior petrous ridge, jugular bulb, internal auditory canal, and the sulci of the greater and lesser superficial petrosal nerves.[17,18]

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TABLE I.
Clinical Features of Temporal Bone Meningiomas.

Patient No.	Age, yr	Sex	Ear Symptoms	Headache	Duration of Symptoms, mo	Findings of Tympanic Membrane	Initial Impression	Diagnosis	Treatment	Tympanomastoidectomy*	Outcomes
TBM-1	55	F	T		1	Gr	Tumor	Imaging	Wait and see		
TBM-2	40	F	HL/Or/EF/D	+	4	Dc/dull/Gr	COM	Path	Cranio-orbitozygomatic approach removal	+	
TBM-3	66	F	HL/T/D		12	Hyperemic	Tumor	Imaging	GKS		NC
TBM-4	45	F	HL/EF/T		6	Dull/bulging	Tumor	Path	GKS		NC
TBM-5	65	F	HL/Or/EF		1	Gr	Tumor	Path	Suboccipital approach removal	+	
TBM-6	40	M	HL/EF/T		12	Hyperemic	Tumor	Imaging	GKS	+	NC
TBM-7	41	F	HL/Or/T/Ot		6	Mass	Tumor	Imaging	Wait and see		
TBM-8	66	F	HL/T	+	4	Mass	Tumor	Imaging	Treatment refused		
TBM-9	47	F	HL		36	Bulging	COM	Path	Wait and see	+	
TBM-10	83	F	Or		Unknown	Mass	Tumor	Path	Treatment refused		
TBM-11	48	F	HL/T/Ot/D	+	48	Dull	COM	Path	GKS	+	Recurrence and irradiation
TBM-12	50	F	HL/EF/T		1	Bulging	Tumor	Imaging	GKS	+	Marginal increase
TBM-13	58	F	HL/T		12	Mass	Tumor	Path	Wait and see		

Blank areas indicate that results were not described. + means that patient has the symptom or undergo the treatment.

*Tympanomastoidectomy was performed in patients who were initially misdiagnosed as COM or whose mass was extended farther into the tympanic cavity and mastoid.

COM = chronic otitis media; D = dizziness; Dc = discharge; Dull = dullness; EF = ear fullness; GKS = gamma knife surgery; Gr = granulation; HL = hearing loss; NC = no change; Or = otorrhea; Ot = otalgia; Path = pathological confirmation; T = tinnitus; TBM = temporal bone meningioma. TM = tympanic membrane.

Several studies about the natural history of intracranial meningiomas reveal that the growth rate is minimal, and most patients remain asymptomatic for several years.[1,19,20] Approximately 68% to 78% patients with asymptomatic meningiomas were found to show no change in the tumor size and symptoms for 3 years.[19,20] In other studies, the mean growth rate was 2.4 to 4 mm/year in 22% to 37% patients.[20,21] Unlike intracranial meningiomas, TBMs cause various symptoms due to the mass effect or local inflammation; the most common symptoms are hearing loss and otorrhea.[15] Headache, otalgia, facial nerve palsy, dizziness, and tinnitus have also been reported.[18] Despite the presence of symptoms, most patients remain undiagnosed for relatively long durations, with an average symptom duration of 2 to 10 years.[15] Reasons for delayed diagnosis are as follows: lack of clinical attention due to its rarity, absence of initial neurological symptoms or signs in patients with typical meningiomas, and misdiagnosis as inflammatory diseases due to nonspecific symptoms. Therefore, the aim of the present study was to illuminate the clinical and radiological characteristics of TBM and determine the findings that should lead to a suspicion of TBM during routine otological examinations.

MATERIALS AND METHODS

We retrospectively reviewed the clinical data of 13 patients diagnosed with TBM at eight tertiary referral centers between 1998 and 2018. Data pertaining to patient demographics and symptoms, including hearing loss, tinnitus, ear fullness, otorrhea, otalgia, dizziness, headache, and other neurological symptoms, were reviewed in addition to the duration of symptoms (from onset to the first clinical visit). Included patients were those who visited the department of otorhinolaryngology for initial workup. Patients whose disease was limited to the petrous face and transverse sigmoid sinus, and who were diagnosed and treated in the department of neurosurgery, were excluded in this study.

Endoscopic findings for the tympanic membrane (TM) and/or external auditory canal (EAC) were evaluated and classified into eight categories: hyperemic, discharge, perforation, dullness, bulging, retraction, granulation, and EAC-occupying mass. All patients had undergone audiological evaluations using pure-tone audiometry. The pure-tone air-conduction (250–8,000 Hz) and bone-conduction (250–4000 Hz) thresholds were measured, and average thresholds were calculated with the mean value of thresholds at 0.5, 1, 2, and 4 kHz. Air-bone gap was defined as a difference between air- and bone-conduction thresholds. Average thresholds were shown as mean ± standard deviation. Radiological evaluations involved temporal bone computed tomography (TBCT) and brain magnetic resonance imaging (MRI). All TBCT images were retrospectively reviewed, and findings suggestive of meningioma were summarized.

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional review board (IRB) of Seoul National University Boramae Medical Center (IRB No. 26–2017-12), Pusan National University Hospital (IRB No. H-1910-013-084), Chonbuk National University Hospital (IRB No. CUH 2019–10-022), Seoul National University Bundang Hospital (IRB No. B-1910-572-402), Hallym University Medical center (IRB No. 2019–10-011), Catholic Medical Center (IRB No. PC19RISI0118), and Chungnam National University Hospital (IRB No. CNUH 2019–10-103) and the 1964 Helsinki declaration and its later amendments or comparable ethical standards. All data generated or analyzed during this study are included in this article.

RESULTS

Clinical Characteristics of TBM

The majority of patients were women (n = 12/13, 92.3%), and the age at diagnosis ranged from 40 to 83 (median = 52.5) years. All of the patients had intracranial meningiomas at cerebellopontine angle area, and there was no primary ectopic meningioma of temporal bone. The duration between the initial onset of symptoms and the first clinical visit ranged from 1 to 60 months (mean = 11.9 ± 14.9 months). The final diagnosis of meningioma was established on the basis of biopsies from EAC masses (n = 4/13, 30.8%) or tissue biopsies obtained from the mastoid cavity during mastoidectomy in patients initially misdiagnosed with chronic otitis media (COM; n = 3/13, 23.1%). Six patients were diagnosed on the basis of characteristic MRI findings (n = 6/13, 46.2%) because surgical biopsy was refused by the patient or seemed unnecessary.

Hearing loss was the most common symptom (conductive hearing loss with an air-bone gap of 31.2 ± 13.5 dB hearing level [HL]) (n = 11/13, 83.6%), followed by tinnitus (n = 9/13, 69.2%), ear fullness (n = 5/13, 38.5%), otorrhea (n = 4/13, 30.8%), dizziness (n = 3/13, 23.1%), headache (n = 3/13, 23.1%), and otalgia (n = 2/13, 15.4%; Table I). Although nine patients exhibited patent EACs and no TM perforation, their average air-bone gap was 29.8 ± 15.4 dB HL. None of the patients exhibited symptoms or signs of lower cranial nerve paralysis (cranial nerve IX, X, XI, and XII).

Endoscopy revealed EAC-occupying masses in four patients (30.8%), so TM could not be evaluated in these cases (Table I). From the remaining nine patients with patent EACs, five and three exhibited dullness or bulging of TM (55.6%) and granulation around the TM and EAC (33%), respectively. Inflammatory signs in the TM, such as discharge (n = 3/9, 33.3%) and hyperemia (n = 2/9, 22.2%), were also observed. No patient exhibited TM perforation. All patients showed a moderate degree of mixed hearing loss on the affected side, and the average air-bone gap in pure-tone audiometry was 31.2 ± 13.5 dB HL. The average bone-conduction thresholds on the affected and contralateral sides were 35.5 ± 14.2 and 20.0 ± 14.2 dB HL, respectively (Fig. 1).

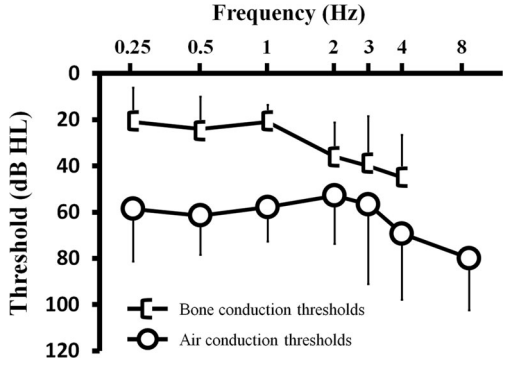

Fig. 1. Pure-tone audiometry of patients with temporal bone meningioma. The average air-conduction and bone-conduction thresholds of the subjects with temporal bone meningioma are illustrated. Error bars depict standard deviation.

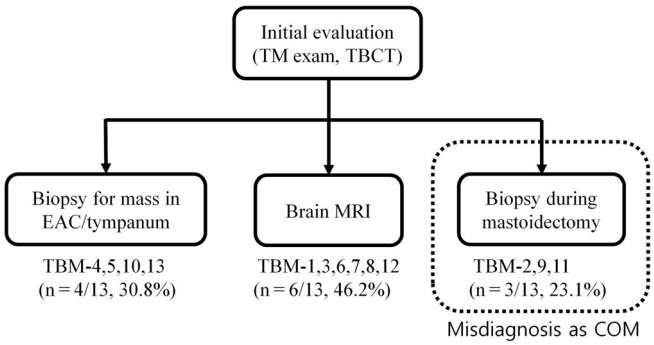

Fig. 2. The diagnostic process from initial evaluation to diagnosis of temporal bone meningioma (TBM). With the initial evaluation using otoscopic examination and temporal bone computed tomography (TBCT), a tumor in the middle ear, external auditory canal, or mastoid cavity was suspected in 10 patients. With a biopsy for the mass in the external auditory canal (EAC) or tympanum (n = 4/13, 30.8%), or brain magnetic resonance imaging (MRI) (n = 6/13, 46.2%), TBM was diagnosed. The remaining three patients (n = 3/13, 23.1%) were misdiagnosed as chronic otitis media (COM) with initial evaluation and underwent tympanomastoidectomy. TM = tympanic membrane.

Computed Tomography Findings

Clinicians and radiologists failed to suspect a tumor after initial computed tomography (CT) imaging in three cases (cases 2, 9, and 11; n = 3/7, 42.9%; Fig. 2), who were misdiagnosed with COM on the basis of preoperative clinical assessments, physical examinations, and TBCT.

Eight patients (cases 2, 4, 6, 7, 9, 10, 11, and 13; 61.5%) showed diffuse trabecular hyperostosis in the middle and posterior cranial fossae on TBCT images (Fig. 3A). The trabecular hyperostosis accompanied bone thickening without destruction of the trabecular structures and irregular margins of the inner surface, where the intracranial mass was located (Fig. 3B). Another common finding was widening and/or destruction of the temporal bone in the jugular bulb area (cases 1, 3, 5, 8, 10, 11, 12, and 13; 61.5%; Fig. 4A); these patients exhibited a large enhancing mass at the cerebellopontine angle that extended into the jugular foramen (Fig. 4B). Otologic symptoms, such as tinnitus, hearing loss, ear fullness, dizziness, and otalgia were not associated with CT findings (Supporting Table 1).

Treatment Outcomes

The surgical treatment, extent of surgery, follow-up period, recurrence, and tumor size change were reviewed for all patients. Six patients did not receive further treatment after diagnosis. From these, four patients (cases 1, 7, 9, and 13) were followed up for 17, 6, 6, and 48 months, respectively, as per a wait and watch policy. Two patients (cases 8 and 10) refused any treatment or examination for TBM because of advanced age (66 and 83 years, respectively).

Gamma knife radiosurgery was performed for five patients (cases 3, 4, 6, 11, and 12). Cases 3, 4, and 6 showed no change in the tumor size, whereas case 11 exhibited regrowth at 5 years after gamma knife surgery and received re-irradiation. Case 12 showed a marginal increase of tumor volume about 10% in the tumor size and was observed without further treatment.

Two patients underwent surgical removal of the intracranial tumor via a cranio-orbitozygomatic approach (case 2) or a suboccipital approach (case 5). Partial tumor removal, with the residual mass extending to the foramen ovale and posterior cranial fossa, was performed for case 2. Case 5 showed a large tumor in the posterior cranial fossa that spread to the jugular foramen. The tumor was

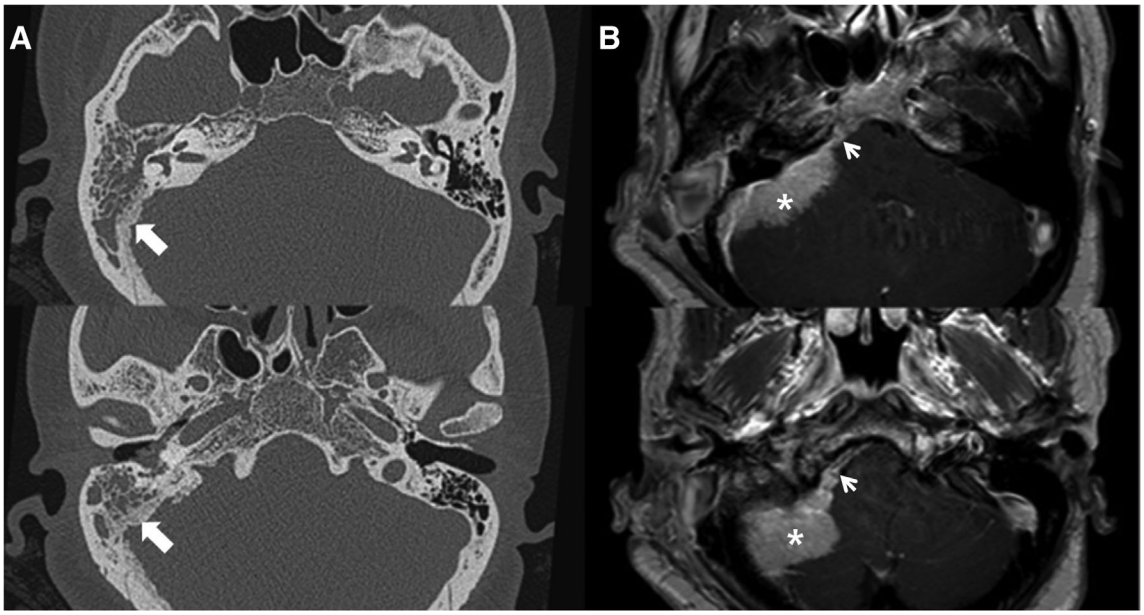

Fig. 3. Features of computed tomography imaging in the middle and posterior cranial fossa suggesting the presence of temporal bone meningioma. (A) Diffuse trabecular hyperostosis with irregular margin of the inner surface in middle and posterior cranial fossa (white arrow). (B) The intracranial enhancing mass (white asterisk) with dura tail sign (white arrow head) is identified with magnetic resonance imaging at the matched location of trabecular hyperostosis.

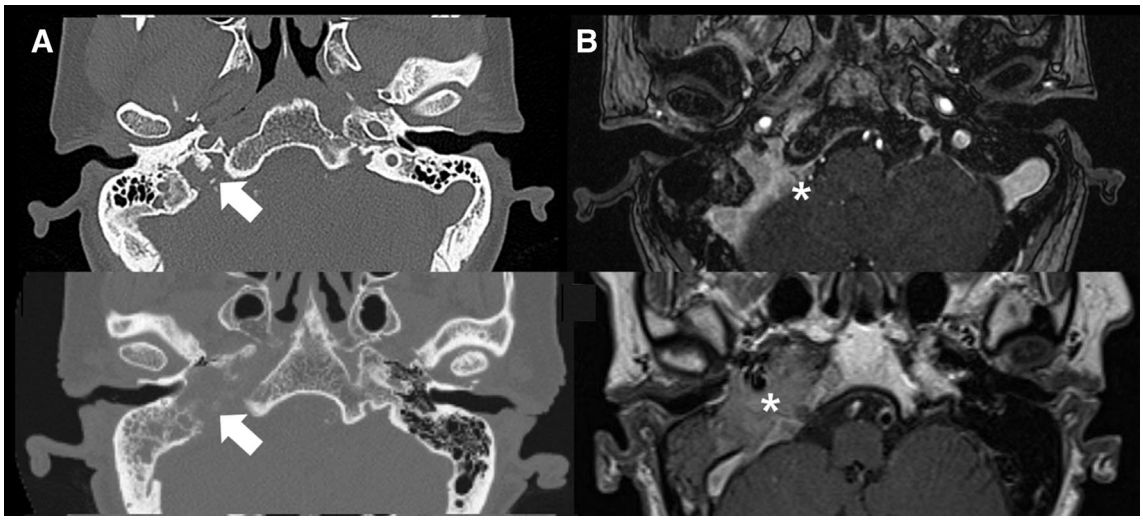

Fig. 4. Features of computed tomography imaging in the jugular bulb area suggesting the presence of a temporal bone meningioma. (A) Widening and destruction of the jugular foramen are identified with temporal bone computed tomography images (white arrow). (B) Enhanced magnetic resonance imaging demonstrates the intracranial enhancing mass extending to jugular foramen and inducing the structural change of temporal bone in the jugular bulb area (white asterisk).

completely removed without complications, and there were no complications during an 18-month follow-up period.

Tympanomastoidectomy for the removal of tumors in the tympanic cavity, mastoid, and/or EAC was performed for six patients (cases 2, 5, 6, 9, 11, and 12). In cases 5 and 6, the incus and malleus head were removed for complete removal of the middle ear tumor, followed by ossiculoplasty. Case 6 showed an improvement in the air-bone gap from 35 dB before surgery to 10 dB at 5 months after surgery. However, the ossiculoplasty in case 5 failed, with an air-bone gap of 45 dB after surgery. In case 2, the middle ear tumor was carefully removed without disruption of the ossicular chain; the air-bone gap was 0 dB at 12 months after surgery.

DISCUSSION

The present study investigated the clinical characteristics of TBM observed during routine otological examinations such as endoscopy and TBCT. Although previous studies about TBM have described the clinical features of this tumor,[15,17,18,22,23] they did not demonstrate the radiological findings suggestive of TBM. A retrospective review of TBCT images in the present study revealed abnormal radiological findings that should have led to a suspicion of TBM. This suggests that both clinical TM evaluations and TBCT are important for accurate diagnosis of TBM.

The air-bone gap caused by the loss of sound conduction was similar to that observed in children with persistent middle ear effusion[24] and smaller than that observed in ossicular interruption with an intact TM.[25] TBM may interrupt sound conduction in the ossicular chain without disruption by soft tissue or effusion in the tympanic cavity. Interestingly, our patients showed not only conductive hearing loss but also sensorineural hearing loss (SNHL). When compared with the hearing level on the unaffected side, the sensorineural component of hearing loss on the lesion side could not be fully attributed to presbycusis, and we believe that the patients may have developed SNHL because of direct or indirect compression of the auditory nerve compartment by the tumor. Although MRI data can be helpful to support this assertion, direct tumor invasion or destruction of cochlear or vestibular structures was not observed in this study. Another possible explanation for the SNHL is labyrinthitis or impairment of the endolymphatic sac. The patients exhibited recurrent and persistent local inflammation or infection caused by local extension of the mass into the middle ear. Therefore, labyrinthitis,[26] which resulted from otitis media, could be a factor for the SNHL. Headache is also a symptom of TBM and generally not associated with COM. When headache is accompanied by COM, complications such as mastoid abscess, labyrinthitis, facial nerve palsy, and malignant otitis externa should be considered.[27] If a patient with vague COM symptoms or signs exhibits persistent headache, clinicians should consider the possibility of a tumor, such as TBM, or a complication of COM.

Despite the presence of CT findings suggestive of TBM, three patients (23.1%) were misdiagnosed with COM and subjected to tympanomastoidectomy. Meningiomas present slight hyperdensity (60%) or isodensity (40%) relative to the brain parenchyma on nonenhanced CT images,[28] and the degree of attenuation is similar to that of inflammatory soft tissue or other tumors such as paraganglioma.[28] In the present study, a retrospective review of TBCT images revealed two abnormalities in the bony tissue surrounding the temporal bone in all patients: diffuse trabecular hyperostosis in the middle and posterior cranial fossae (Fig. 2A) and widening and destruction in the jugular bulb area (Fig. 3A). These bony changes are associated with the extension pathways of intracranial meningiomas,[18] which include the tegmen tympani, posterior fossa plate, internal auditory canal, and jugular foramen.[17] When the tumor extends to the temporal bone via the tegmen tympani or posterior fossa plate, diffuse trabecular hyperostosis, bone thickening,

and irregular inner surface margins may be found in the middle cranial fossa over the mastoid or tympanic cavity, or the posterior cranial fossa around the sigmoid sinus.[18] When the tumor spreads along the internal auditory canal or jugular vein, destruction or widening of the jugular foramen or carotid canal in the petrous bone may be observed. Trabecular hyperostosis is a common imaging finding presenting in approximately 25% cases of meningiomas,[1,18,29] and it is caused by tumor invasion of the bone.[30] Therefore, the spread of meningiomas via the middle cranial fossa might induce reactive changes in the bone, resulting in trabecular hyperostosis. In addition, sphenoid wing and temporal floor meningiomas can cause eustachian tube dysfunction with secondary mastoid effusion or penetrate the external auditory canal or middle ear.

Differential diagnoses for tumors in the middle ear, including TBM, can be established on the basis of the tumor location and changes in the surrounding bony structures on CT images. Glomus tympanicum paraganglioma, a common middle ear tumor originating from the cochlear promontory, usually manifests as a small discrete mass confined to the tympanic cavity.[31,32] It does not generally cause destruction or hyperostotic changes in the surrounding bony structures. Glomus jugulare paraganglioma is a destructive lesion in the jugular foramen spreading to the hypotympanum, internal jugular vein, and infratemporal fossa.[33] Differentiation of this tumor from TBM without MRI findings could be difficult because both tumors can present as a mass in the tympanic cavity or destructive lesion in the jugular bulb area on CT images.

CONCLUSION

To the best of our knowledge, this is the first study to gather data for the clinical and radiological characteristics of TBM from a multicenter cohort. However, the findings are limited by the retrospective study design. When persistent inflammatory symptoms or signs without TM perforation and unilateral mixed hearing loss with trabecular hyperostosis or destruction of the temporal bone on CT are observed, TBM should be suspected, and biopsy or MRI should be considered.

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