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Denervation of the Eustachian Tube and Hearing Loss Following Trigeminal Schwannoma Resection

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Abstract

Keywords

► eustachian tube dysfunction
► trigeminal schwannoma
► hearing loss
► trigeminal nerve
► patulous ET

Objectives To discuss eustachian tube dysfunction (ETD) as a cause of hearing loss and to discuss its pathogenesis following resection of trigeminal schwannomas.

Methods Presented herein are two cases of trigeminal schwannoma that were resected surgically with sacrifice of the motor branch of the trigeminal nerve. Neither of the cases had evidence of extracranial extension nor preoperative ETD. Both patients developed ETD and have been followed without evidence of schwannoma recurrence.

Conclusions Trigeminal schwannomas are rare tumors that typically require surgical resection. Hearing loss is a potential postsurgical deficit and warrants evaluation by an otolaryngologist with consideration given to a preoperative audiogram. ETD as a result of trigeminal motor branch sacrifice should be included in the differential diagnosis of postoperative hearing loss in this patient subset as it may be reversed with placement of a tympanostomy tube.

Introduction

Trigeminal schwannomas are benign nerve sheath tumors with very few reported cases of malignancy.[1,2] Many postoperative deficits cited in the literature relate to damage to cranial nerves.[3–6] Advances in surgical technique and approach have improved surgical morbidity[3–9]; however, trigeminal deficits are expectedly commonplace, given the nature of the tumor.[3,10] Dysfunction of the trigeminal nerve may be either sensory or motor and is described as either paresthesias within the sensory distribution or weakness of the muscles of mastication with or without atrophy.

Evaluating and documenting hearing loss as part of the preoperative process and during the postoperative period are not universally performed. Discussion on differentiating neural and conductive hearing loss and their potential causes is scarce. There are no reports of hearing loss caused by eustachian tube dysfunction (ETD) as a result of damage to the motor root of the trigeminal nerve, which innervates the peritubular musculature responsible for the patency of the ET. ETD can lead to a conductive hearing loss and may have other longterm sequelae, including predisposition to cholesteatoma formation.[11] The aim of this manuscript is to review two cases of trigeminal schwannoma, that were surgically resected and to review the pertinent anatomy and physiology of the ET to provide a framework for understanding the pathophysiology of its dysfunction in the setting of trigeminal schwannoma resection.

Methods

Two patients were identified between the years 2008 and 2014 at a tertiary care hospital with histologically confirmed trigeminal schwannoma. These charts were retrospectively reviewed. Data regarding presentation, diagnostic work up, management, and clinical follow up were gathered. This series was exempt from Internal Review Board approval.

Case 1

The first case is that of a 50-year-old man who underwent magnetic resonance imaging (MRI) for bilateral hand tingling. A mass was found in the right cerebellopontine angle and involved both the middle and posterior cranial fossae. A middle fossa approach was utilized with anterior petrousectomy for extirpation of the tumor. Postoperatively, the patient experienced diplopia, right facial numbness, and right mandibular weakness with rightward deviation of the mandible. The diplopia resolved within 7 months; however, after 7 years of follow up his facial numbness, absent ipsilateral corneal reflex and jaw weakness had persisted. Over time, he developed temporal wasting with intermittent right-sided hearing loss and aural pressure. His symptoms were triggered during moments of stress, particularly during public speaking. Otoscopy revealed fluttering of the tympanic membrane with the respiratory cycle. All repeat imaging showed no evidence of tumor recurrence. Repeated audiographic evaluations showed only a mild sensorineural hearing loss with normal type A tympanograms. He was thought to be suffering from patulous ETD. He opted for observation with no intervention.

Case 2

The second case was that of a 62-year-old man who initially presented with right-sided facial numbness and was diagnosed with a cerebellopontine angle tumor found on MRI. He underwent stereotactic radiation due to significant medical comorbidities. The patient developed progression of right facial numbness at 3 year followup and the repeat MRI showed enlargement of the mass. Audiogram at that time showed a mild sensorineural hearing loss with normal tympanograms and no suggestion of active ETD. Extirpation was achieved through a right temporal craniotomy. Postoperatively, he experienced diplopia and continued right facial numbness. Diplopia resolved at 8 months, but he continued to have paresthesias of the right hemi face. He also developed right aural pressure and hearing loss. Audiogram showed stable sensorineural hearing loss with a mild conductive component and a tympanogram showing evidence of ETD. The patient deferred placement of a tympanostomy tube and elected for observation but continued to have intermittent symptoms of aural pressure and muffled hearing loss.

Anatomy and Physiology of the Eustachian Tube

The ET, sometimes referred to as the "auditory tube," joins the middle ear with the nasopharynx and functions to ventilate and drain the middle ear. It also serves to protect the middle ear from reflux of nasopharyngeal secretions. The ET is between 31 to 38 mm long.[12] It descends from the skull base 45 degrees medially from the sagittal plane and 30 to 40 degrees inferiorly from the axial plane.[13] It is composed of osseous and cartilaginous segments. The osseous segment is located within the petrous portion of the temporal bone and makes up the posterior–superior one third of the ET, measuring approximately 12 mm in length. The cartilaginous segment makes up the inferomedial two thirds of the ET, measures 24 mm in length, and opens into the nasopharynx.

The shape of cartilage of the ET resembles an inverted "J." The tube is closed at rest and opens during swallowing and yawning by action of the tensor veli palatini muscle[14,15] with some assistance from the levator veli palatini and salpingopharyngeus muscles.[11,16,17] Closure of the tube at rest is thought to be due to the elastic properties of the ET cartilage, compression by surrounding soft tissue, and surface tension of the ET luminal surface. Surrounding soft tissue components include Ostmann's fat pad—a collection of fat tissue just inferior to the ET lumen—as well as bulk from the medial pterygoid muscle.[18–21]

The mucosal lining of the cartilaginous ET is pseudostratified, ciliated, columnar epithelium identical to the epithelium lining the upper respiratory tract. This region of the ET is particularly dense in goblet cells that produce and secrete mucous which contributes to the surface tension between the opposing walls of the ET lumen. The flow rate and composition of ET secretions impact the degree of surface tension and are under the control of the parasympathetic nervous system.[22] Increased parasympathetic tone would cause an increase in secretions and surface tension, resulting in impairment of the ET opening. Greater sympathetic tone would have the opposite effect and is theorized to contribute to an overly patulous ET.[23] Parasympathetic innervation of the ET is thought to be derived from the glossopharyngeal and caroticotympanic nerves with postsynaptic fibers originating from the sphenopalatine and otic ganglia. Sympathetic innervation is likely through the caroticotympanic and vidian nerves from the superior cervical ganglion.[24–26]

The tensor veli palatini is composed of two muscle fiber bundles. The lateral bundle, the tensor veli palatini proper, originates from the scaphoid fossa and the greater wing of the sphenoid bone and converges with the medial muscle fiber bundle to form the tendon that attaches to the hard palate and palatine aponeurosis of the soft palate. The medial bundle is called the dilator tubae muscle and attaches to the lateral wall of the cartilaginous portion of the ET. The tensor veli palatini muscle is innervated by the trigeminal motor nucleus through the mandibular division of the trigeminal nerve.

The levator veli palatini muscle originates from the inferior aspect of the petrous apex with some attachments to the medial lamina of the ET cartilage and descends to insert on the dorsal surface of the soft palate. Its contraction and relaxation are thought to promote clearance of middle ear and ET.[27] It is innervated by the nucleus ambiguus through the vagus nerve.

The salpingopharyngeus inserts on the cartilaginous end of the ET in the nasopharynx. It attaches inferiorly to the palatopharyngeus muscle. The muscle belly contracts during

deglutition and assists in elevating the pharynx and opening of the ET. The salpingopharyngeus is also innervated by the vagus nerve.

Pathophysiology of Eustachian Tube Dysfunction

The primary role of the ET is pressure equalization between the middle ear and the surrounding environment. It serves as a barrier against nasopharyngeal reflux into the middle ear and is also vital for allowing clearance of the middle ear.[11] The ET is closed at rest and opens briefly and intermittently when acted upon by the peritubular musculature. A patient with an ET that is abnormally open or obstructed and difficult to open will develop symptoms of ETD. Presenting symptoms of obstructive ETD include aural pressure and decreased or "muffled" hearing. These symptoms usually resolve when patients equalize pressure in their middle ear with the Valsalva maneuver.[11] Tympanic membrane retraction and a middle ear effusion may be seen on otoscopic evaluation. ETD is caused by either a mechanical or functional obstruction. A mechanical obstruction causes ET obstruction in an otherwise normally-functioning ET, such as a nasopharyngeal mass impinging on the opening of the ET into the nasopharynx. An extracranial schwannoma involving V3 may result in obstructive ETD given the close anatomic relationship of the nerve to the ET, as can be appreciated in ►Figs. 1 to 3. A functional obstruction is caused by dysfunction of the ET itself. Adults with allergic rhinitis may present with ETD caused by edema and inflammation of sinonasal mucosa.
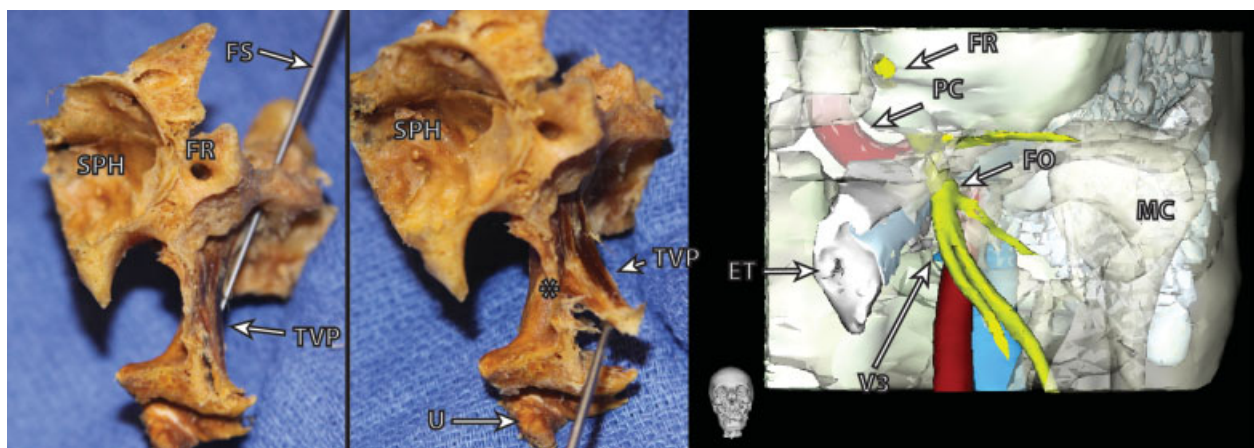

Fig. 1 Anterior view of the eustachian tube (ET). *Left:* The left sphenoid sinus (SPH) and foramen rotundum (FR) are seen. The probe is traversing the foramen spinosum (FS). The tensor veli palatini (TVP) is seen descending from the skull base just lateral to the ET where it inserts onto the palate inferiorly. *Middle:* The TVP is retracted off of the ET (*) laterally with the probe. The close relationship with the ET and TVP to the soft palate and uvula (U) is appreciated. *Right:* Digital reconstruction of the infratemporal fossa. The relationship of the ET to the V3 branch of the trigeminal branch and foramen ovale is seen. MC = mandibular condyle, PC = petrous carotid. (Used with permission from Wang H, Northrop C, Burgess B, et al. Three-Dimensional Virtual Model of the Human Temporal Bone: A Stand-Alone, Downloadable Teaching Tool. Otol Neurotol. 2006;27(4):452–457).
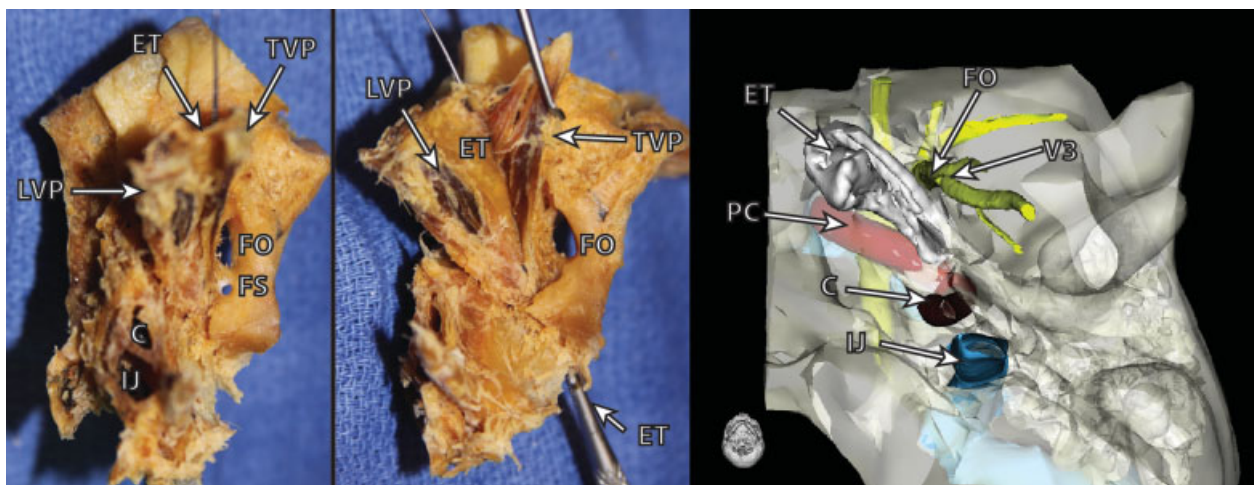

Fig. 2 Inferior view of the skull base and ET. *Left:* The proximity of the foramen ovale (FO) and foramen spinosum (FS) to the ET is appreciated. *Middle:* The TVP is retracted off of the ET to expose the cartilaginous portion. There is a probe placed in the ET from the middle ear through to the nasopharyngeal opening. *Right:* Digital reconstruction of the skull base. V3 is seen exiting FO just anterior and lateral to the ET. C, carotid canal; IJ, internal jugular vein as it enters the jugular foramen; TVP, tensor veli palatini; LVP, levator veli palatini; PC, petrous carotid. (Used with permission from Wang H, Northrop C, Burgess B, et al. Three-Dimensional Virtual Model of the Human Temporal Bone: A Stand-Alone, Downloadable Teaching Tool. Otol Neurotol. 2006;27(4):452–457).

Fig. 3 Lateral view of the infratemporal fossa and ET. *Left*: TVP is retracted superoanteriorly off of the ET by the probe. LVP is seen posterior and inferior to the ET. *Middle*: TVP is in anatomic position lateral to the ET. The yellow probe is traversing foramen ovale (FO). The unmarked probe is traversing the ET. *Right*: Digital reconstruction. V3 is seen exiting FO just lateral to the ET. MC, mandibular condyle; PC, petrous caroitd; S, styloid process. (Used with permission from Wang H, Northrop C, Burgess B, et al. Three-Dimensional Virtual Model of the Human Temporal Bone: A Stand-Alone, Downloadable Teaching Tool. Otol Neurotol. 2006;27(4):452–457).

Similar mechanisms are also responsible for ETD following radiation therapy when the field of radiation involves the ET.[28,29] Patients who have undergone resection of the oral palate may demonstrate a functional obstruction of the ET if a significant portion of the tensor veli palatini muscle is resected, essentially eliminating the anchor necessary for it to enact its dilatating effects on the ET.[30] A similar mechanism is also found in patients with cleft palates whose palatal musculature is anomalous. These patients almost universally require tympanostomy tubes to allow pressure equalization. It would seem reasonable to extrapolate that a similar situation would arise in the event of ET denervation.

The middle ear and mastoid air cells are connected by the aditus ad antrum and are lined with a mucosal membrane that actively participates in gas exchange.[31] The mucosa will absorb gas to develop negative pressure within the middle ear and mastoid, which, when paired with ETD, will result in atelectasis of the tympanic membrane. Over time, the persistent negative middle ear pressure causes a transudative effusion to accumulate. A retracted tympanic membrane or a middle ear with an effusion will have reduced compliance. This dampens the vibratory ability of the tympanic membrane and results in a conductive hearing loss. ETD can lead to recurrent episodes of acute otitis media and may also predispose to chronic otitis media, tympanic membrane perforation, and cholesteatoma formation.[11]

In addition to obstructive ETD, dysfunction can also occur if the tube is abnormally open, or patulous. When closed, the ET not only protects the middle ear from nasopharyngeal secretions but also from vocal resonance. Patulous ETD frequently presents with autophony, an abnormally loud perception of one's own voice. Symptoms are classically associated with recent weight loss and worsen during periods of stress. This may be due to decreased mucus production and surface tension within the lumen of the ET. Presentation may be similar to classic obstructive ETD and can be difficult to differentiate. These patients will have no evidence of negative middle ear pressure on tympanometry and results of tuning fork examination are normal. Movement of the tympanic membrane on otoscopic evaluation that correlates with the respiratory cycle is suggestive of patulous ETD. Symptoms typically improve in the supine position, as the peritubal venous plexus becomes engorged.[32]

Otologic Evaluation

In the absence of other otologic pathology, a patient with severe ETD may be observed to have a Weber test lateralizing to the affected ear and a Rinne test with bone conduction greater than air conduction on the ipsilateral side. Audiogram may show an air-bone gap of varying severity. Tympanometry is also performed and measures tympanic membrane compliance as a function of negative pressure introduced to the external ear canal. The tympanic membrane will reach maximum compliance when the pressures of the external auditory canal and the middle ear across the tympanic membrane are equal. Therefore, patients with ET dysfunction will require an increased amount of negative pressure in the external auditory canal to balance out the negative pressure within the middle ear to obtain maximal compliance. This is referred to as a type C tympanogram. Alternatively, ETD may present with a type B, or "flat," tympanogram, meaning that the tympanic membrane maintains a poor compliance across pressures and suggests more severe ETD; however, tympanic perforations will also have a type B but can be differentiated on physical exam as well as by the presence of a large external auditory canal volume on tympanometry. One caveat to the use of tympanometry to evaluate for milder cases of ETD is that patients who have recently autoinsufflated their ears may have a normal audiogram and tympanogram. A patulous ET generally does not cause changes in an audiogram.

Discussion

Trigeminal schwannomas were first described by Dixon in the year 1846.[3,33] They are benign nerve sheath tumors with very few reported cases of malignancy.[1,2] They represent 0.07 to 0.36% of all intracranial tumors and 0.8 to 8% of all intracranial

schwannomas.[34] Typical presentation is within the middle decades of life with a reported age range from 5 to 77 years.[8] They do not appear to have a sex predilection.[33]

Onset is typically insidious and frequently results in delayed diagnosis. A majority of patients will present with trigeminal sensory paresthesias. Trigeminal motor involvement presents with masticatory muscle weakness with possible atrophy and mandible deviation to the side of the lesion as the contralateral lateral pterygoid causes anterior displacement of the contralateral mandibular condyle while the ipsilateral mandibular condyle remains stationary.[33] Expansion of the tumor in the cavernous sinus or into the orbit may result in visual disturbances if the occulomotor, trochlear, abducens, or optic nerves become involved. Posterior fossa extension is suggested by the presence of cerebellar symptoms or evidence of lower cranial nerve palsies. Late manifestations include papilledema and headaches. Schwannomas can form along any portion of the trigeminal nerve and their location directly correlates with patient symptoms.[8,33] For example, tumors extending through the superior orbital fissure with V1 may cause changes in vision and proptosis. Trigeminal schwannomas can present as part of von Recklinghausen's syndrome.

A trigeminal schwannoma can present with symptoms identical to trigeminal neuralgia, and so a high index of suspicion is needed to make a timely diagnosis in these cases. Meningiomas are found more frequently in Meckel's cave and remain at the top of the differential diagnosis. Other masses found in this region, albeit rarely, include chondromas, sarcomas, metastases, vascular aneurysms and malformations, vascular neoplasms, granulomatous lesions, and neural tumors (including neuromas originating from other cranial nerves).

Jefferson was the first to classify trigeminal schwannomas based on location; however, a modified classification scheme has been proposed by Samii, et al.[35,36] Surgical approach is dictated by the location of the tumor.[3,4,6,36] Extirpation of these tumors had classically been difficult with high morbidity and mortality.[3] Outcomes have improved with advances in technology and skull base approaches with microsurgical dissection techniques.[3–9] Intensity-modulated radiotherapy and stereotactic radiotherapy have shown some success in treatment and is particularly useful for patients with medical comorbidities who are deemed poor surgical candidates. Prior authors recommended complete microsurgical resection as the mainstay of treatment,[3,37] although there are recent proponents for subtotal resection if the tumor is adherent to the cavernous sinus or neural structures. These patients are then followed by observation or undergo postsurgical radiation.[6]

Postoperative cranial nerve deficits, except those of the trigeminal nerve, have mostly been reported to improve within 6 months.[3,7,8] Aside from their trigeminal deficits, the two patients in this series both experienced diplopia that resolved within 8 months.

Management of hearing loss in this patient population would be determined by identifying the cause and its severity. Given the risk of hearing loss postoperatively, a preoperative audiogram should be considered to obtain a baseline.[30]

Hearing loss can be classified in three different ways. Sensorineural hearing loss implies a deficit in the neural structures that encode and interpret sound. Damage to the vestibulocochlear nerve, or neural elements proximal to it, would result in a sensorineural hearing loss. A conductive hearing loss presents as an air-bone gap on audiogram and signifies pathology affecting the structures that transmit sound waves to the cochlea. If the etiology is increased negative middle ear pressure with dampening of tympanic membrane vibration, as is observed in obstructive ETD, then it is possible to treat the hearing loss with placement of a pressure equalization tube. One report has promoted the use of balloon dilation to treat ETD,[38] although this is still controversial. There are myriad causes of conductive hearing loss, each with their own management algorithm. Discussion here will be limited to ETD as it relates to trigeminal schwannomas and their surgical resection. Mixed hearing loss is defined as containing components of both neural and conductive hearing loss.

The first patient in our series had clear signs of trigeminal motor deficit postoperatively with subjective symptoms of ETD. His audiogram suggested normal function of the ET. Otoscopy revealed a tympanic membrane that moved with patient respirations and he was diagnosed with patulous ET. The patient's progressive symptoms can be explained by denervation of the peritubal musculature, namely the medial pterygoid, tensor veli palatini, and levator veli palatini. During his 7 year surveillance period, denervation appears to have led to atrophy of the muscles, as exemplified by temporal wasting in this patient, making the ET more susceptible to abnormal opening as a result of decreased peritubal soft tissue bulk. Exacerbation of symptoms during public speaking in this patient represents the patulous effects of the sympathetic nervous system on the ET, as described earlier.

The second patient had audiologic evidence of obstructive ETD with a type C tympanogram. Prior treatment with stereotactic radiation likely was not the sole cause of ETD as the patient had a normal audiogram preoperatively and developed symptoms only after surgery, although its contribution cannot be excluded. Further studies are necessary to delineate the time frame for development and severity of ETD in patients who have undergone trigeminal schwannoma resection. It may be that patients experience obstructive ETD in the more immediate postoperative period after sacrifice of the trigeminal nerve motor branch, but go on to develop a patulous ET as peritubal musculature atrophies. Longer follow up and larger sample sizes may clarify this theory.

Conclusion

Trigeminal schwannomas are rare tumors that typically require surgical resection. Hearing loss is a potential postsurgical deficit and warrants evaluation by an otolaryngologist. Consideration should be given for a preoperative audiogram to obtain baseline functioning. Eustachian tube dysfunction should be included in the differential diagnosis of postoperative hearing loss in this patient subset, as it may improve with placement of a tympanostomy tube.

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