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Case Report

Facial nerve schwannoma: Case report and brief review of the literature ☆,☆☆

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ABSTRACT

Schwannomas are rare nerve sheath tumors that can occur throughout the body, and are symptomatic based on location, size, and impingement on adjacent structures. These tumors are often benign lesions and occur sporadically or from genetic conditions such as neurofibromatosis. Schwannomas may arise from peripheral nerves, gastrointestinal nerves, spinal nerve roots and cranial nerves. Facial nerve schwannomas arise from cranial nerve VII, commonly involving the geniculate ganglion, labyrinthine segment, and internal auditory canal. While small lesions are asymptomatic, larger lesions can cause facial nerve paralysis, and facial spasms. Lesions in the internal auditory canal can cause hearing loss, tinnitus, vertigo, and otalgia. High-resolution CT imaging and MRI imaging are useful for distinguishing between other pathologies that arise from the same region. High-resolution CT scans can show bony degeneration of nearby structures such as the labyrinth or ossicles. MRI imaging shows hypo intensity on T1 imaging, and hyperintensity on T2 imaging. On T1 postcontrast, enhancement can be homogenous or heterogeneous with cystic degeneration if the lesion is large. Nodular enhancement is commonly seen on facial nerve schwannomas within the internal auditory canal. Vestibular schwannomas involving CN VIII are more common, and appear similar to facial nerve schwannomas, but can be distinguished apart due to growth in the geniculate ganglion and/or the labyrinthine segment. Management of asymptomatic or mild lesions is typically conservative with follow up imaging, and surgery for larger lesions. Here, we present a case of a facial nerve schwannoma in a 57-year-old woman.

Abbreviations: FNS, facial nerve schwannoma; HB, House Brackman scale; HRCT, high-resolution CT; IAC, internal auditory canal; NF1, neurofibromatosis 1; NF2, neurofibromatosis 2.

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Introduction

Schwannomas are benign tumors that arise from Schwann cells in the peripheral nervous system, causing symptoms based on the involved nerve and surrounding structures. They are rare tumors, with a low prevalence estimated as fewer than 200,000 individuals afflicted and make up approximately 5% of all benign soft tissue tumors [1–3]. The prevalence may be underestimated, as many of these tumors are an incidental finding on imaging due to a lack of identifiable symptoms [4]. Schwannomas can affect any nerve, such as cranial nerves, spinal nerve roots, peripheral nerves, and gastrointestinal nerves. The cranial nerve that is most often affected is the vestibulocochlear nerve, and less commonly the trigeminal and facial nerves [5,6]. Genetic conditions can increase the probability of schwannoma development, such as neurofibromatosis type 2 which can present with bilateral vestibular schwannomas which has a prevalence of 1 in 210,000 and an incidence of 1 in 33,000 [7].

Facial nerve schwannomas (FNS), which involve cranial nerve VII, are extremely rare tumors, and are often benign, with malignant evolution even less [8–10]. While the prevalence of FNS is low, it may be higher than estimated due to the possibility of an asymptomatic nature [11]. Out of all intratemporal tumors, less than 1% are facial nerve schwannomas [8,12]. However, FNS are the most common primary tumor involving the cranial nerve VII [13]. When symptomatic, FNS presents as a slowly developing facial nerve palsy, or a rapid onset akin to Bell's palsy, which are caused by direct effect on the facial nerve itself, and are common presenting symptoms [13–18]. Partial or full facial spasms can also occur [16,18]. Full facial paralysis is a possible symptom late into the course of the tumor; however, it is not always present [8]. Patients can also experience cephalgia and otalgia as well [10,16]. Mass effect onto adjacent nerves can cause symptoms such as sensorineural hearing loss, tinnitus, and vertigo, akin to Meniere's disease, and less commonly conductive hearing loss [10,14,16–18].

Differential diagnosis for FNS depends heavily on the location, history, and clinical symptoms. If symptoms such as facial nerve paralysis occur, other diagnoses such as Bell's palsy or Lyme disease may be suspected; however, the paralysis in these conditions is more of an abrupt onset. Sensorineural hearing loss with an accompanying brain mass is also consistent with a vestibular schwannoma, which can have similar imaging findings. FNS can also arise in the parotid gland, where it could be construed for a salivary gland tumor, especially if no facial nerve dysfunction is present [5,19]. Many other etiologies such as lipomas and hemangiomas can occur in the same regions, so adequate imaging technique must be obtained to differentiate the pathologies [8,20].

Here, we present a case of a facial nerve schwannoma in a 57-year-old woman.

Case report

A 57-year-old female with no relevant past medical history presented to an outside facility due to recurrent dizziness over 6 months. A trial of meclizine for symptom management was unsuccessful.

Further evaluation with CT temporal bone (Fig. 1) and MRI internal auditory canal protocol (Figs. 2 and 3) was preformed, which showed a 6 × 7 mm oval-shaped enhancing lesion in the right internal auditory canal and abnormal enhancement in the right geniculate ganglion on T1 imaging, and hyperintense enhancement on T2 imaging, findings highly suggestive of a facial nerve schwannoma. The schwannoma exerted mass effect on the vestibular nerve resulting in patient's presentation. Patient opted conservative management.

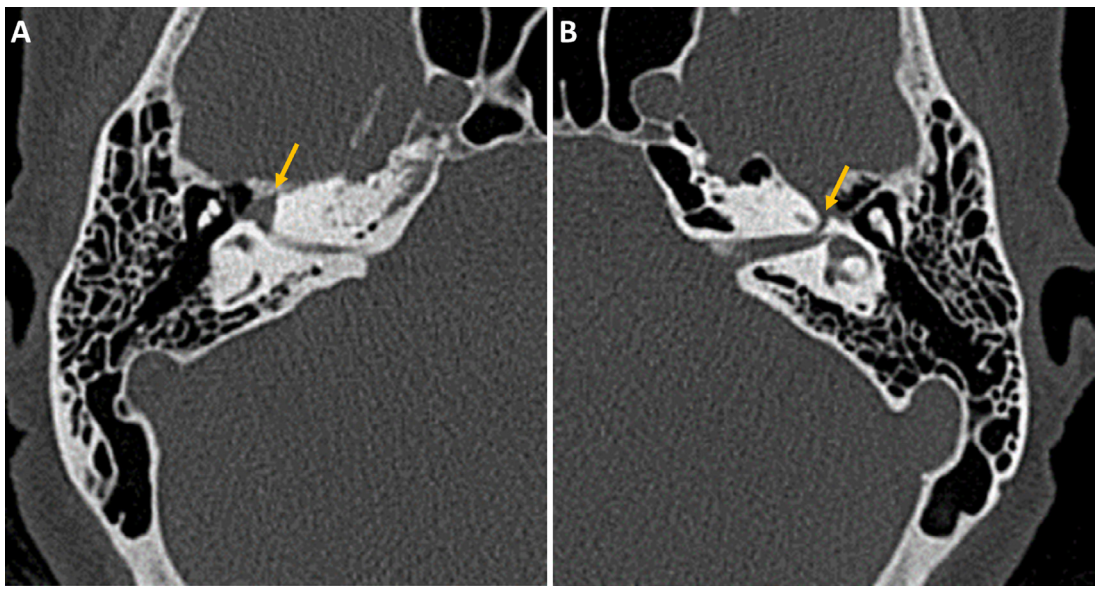

Fig. 1 – CT temporal bone, right-side (A) and normal left-side (B) in a 57-year-old woman with recurrent episodes of dizziness over 6 months. Expansion of the right geniculate ganglion (A, arrow), compared to contralateral normal left side (B, arrow).

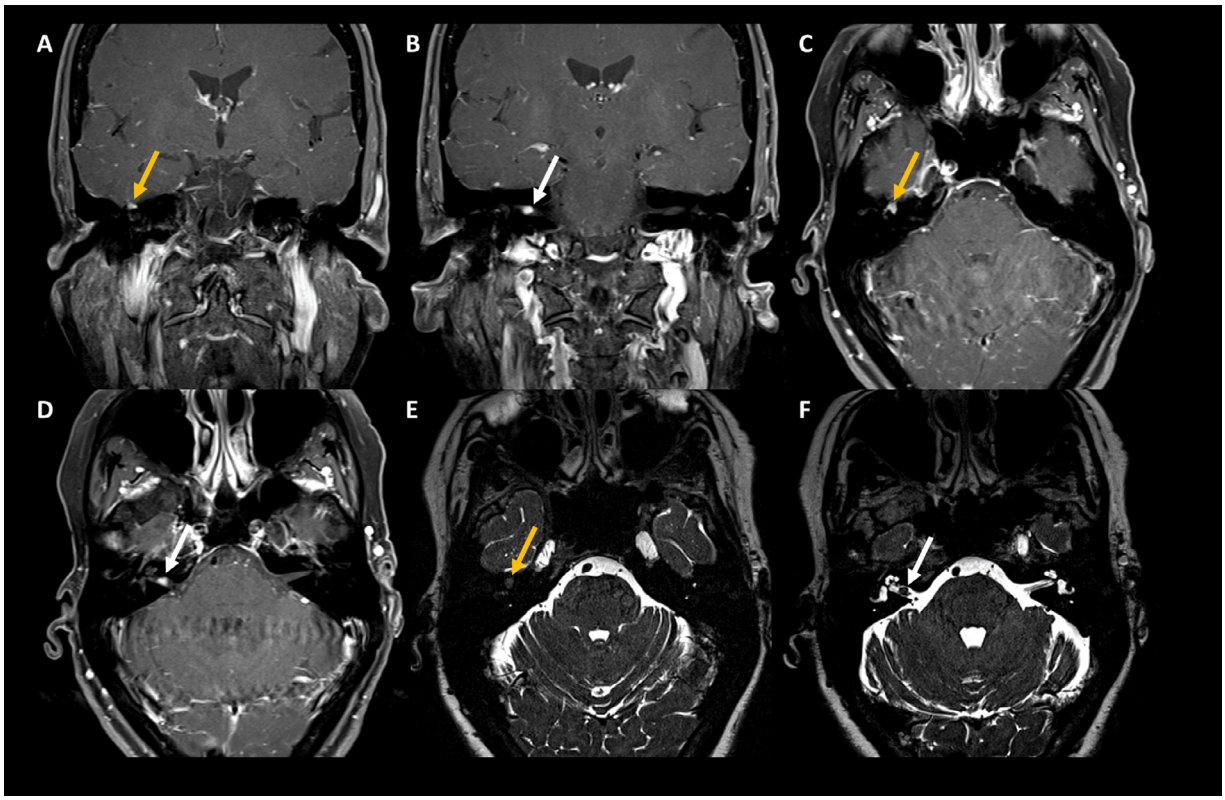

Fig. 2 – MRI internal auditory canal protocol, coronal T1 postcontrast fat-saturated images (A and B), axial T1 postcontrast fat-saturated images (C and D) and axial T2-weighted non–fat-saturated images (E and F) in a 57-year-old woman with recurrent episodes of dizziness over 6 months. Nodular oval-shaped T2-hypointense enhancing lesion measuring 6 × 7 mm was noted in the right internal auditory canal (white arrow). Furthermore, abnormal enhancement was present in the right geniculate ganglion (yellow arrow). These findings were highly suggestive of a facial nerve schwannoma.

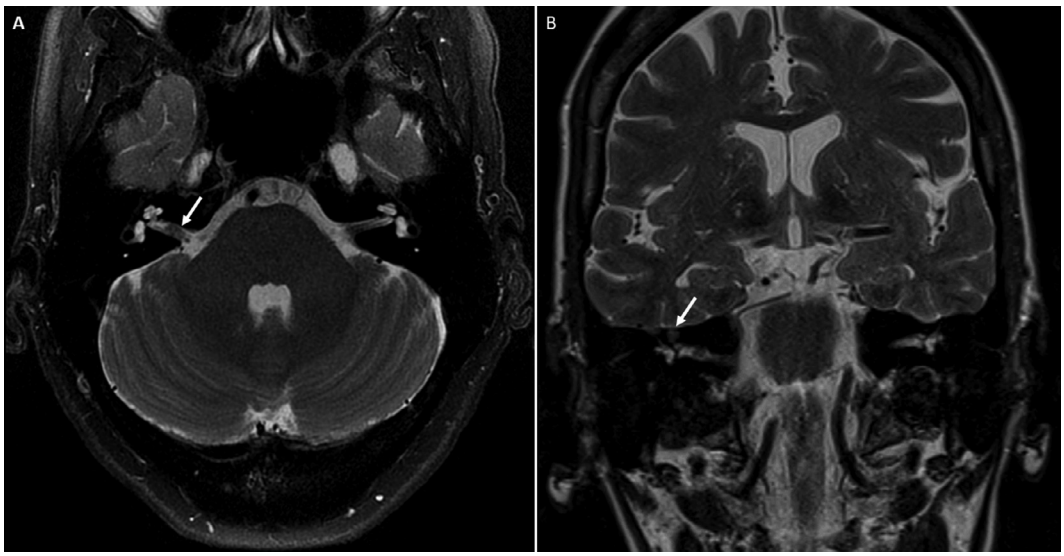

Fig. 3 – Axial (A) and coronal (B) T2-weighted images showing internal auditory canal (A, arrow) and geniculate ganglion (B, arrow) in a 57-year-old woman with recurrent episodes of dizziness over 6 months. The lesions demonstrate intrinsic T2 hyperintense signal characteristics.

Five years later, the patient presented to her primary care physician with tinnitus, ear pressure, facial numbness, and difficulty blinking on her right side. Trial of steroids for presumed Bell's palsy showed no improvement in symptoms. MRI of the internal auditory canal (IAC) showed stable abnormal enhancement in the right internal auditory canal and right geniculate ganglion, futher confirming the radiological diagnosis of facial nerve schwannoma. Considering stability, no surgical management was deemed necessary. Over the course of the year, the patient had worsening facial numbness, paresis, and intermittent spasms as well as a pulling feeling affecting her right eye, cheek, and mouth that occurred a few times a week. Trial of local Botulinum toxin injection did not help her symptoms. The patient opted for conservative management and feels that her symptoms are manageable without treatment. She would continue to follow up with neurology on an outpatient basis and get surveillance MRI.

Discussion

Peripheral nerve sheath tumors that arise from Schwann cells include schwannomas, neurofibromas, and malignant peripheral nerve sheath tumors [6]. Both schwannomas and neurofibromas are benign lesions, with rare malignant transformation [6,21–23]. Schwannomas arise in a single fascicle or bundle inside the nerve sheath and grow out eccentrically, displacing the other nerves within the nerve sheath, with possible nodular growth [1,15,21]. These tumors also have the ability to invade and erode local bony structures [23]. Neurofibromas, a similar tumor involving the nerve sheath, involve the extracellular connective tissue and endoneurium [21]. Types of neurofibromas are localized, diffuse, and plexiform, with the localized being the most prevalent [24].

Both schwannomas and neurofibromas appear in different genetic conditions such as neurofibromatosis 1 (NF1), and neurofibromatosis 2 (NF2) as well as schwannomatosis. Many types of neurofibromas can be found in NF1; however, the plexiform subtype generally only occurs in NF1 and is rarely sporadic in nature [24]. These neurofibromas can arise in many locations throughout the body such as spinal tumors, cutaneous or subcutaneous tumors, and diffuse plexiform tumors [21]. The most common presentation in neurofibromatosis 2 is bilateral vestibular schwannomas [7,17,21–23,25]. Other common tumors include spinal tumors such as meningiomas, cerebral tumors, and eye manifestations [22]. Of these intracranial tumors, it has been shown in some cases of NF2, facial nerve schwannomas can arise [18].

Anatomy of the facial nerve includes 8 parts. There are 6 divisions: cerebellopontine cistern, internal auditory canal (IAC), labyrinthine, tympanic, intra-mastoid, and extracranial [8]. The remaining 2 parts are the genu, or bends: Geniculate ganglion, and the posterior genu. [8]. The branches of the facial nerve include the greater superficial petrosal nerve, the motor nerve to the stapedius, a branch to the posterior belly of the digastric, motor innervation to the stylohyoid, the chorda tympani, the posterior auricular nerve, and 5 terminal branches (temporal, zygomatic, buccal, marginal mandibular, and cervical) [8,20]. FNS can be either intratemporal or extratemporal, the former being more common [19]. These tumors may involve multiple segments, most commonly including the geniculate ganglion, and traditionally are unilateral [8,13,17,26–28]. Other common areas for FNS to occur are the labyrinthine, tympanic, and cerebellopontine angle-internal auditory canal segments [18].

Typical imaging modalities for FNS evaluation include temporal bone high-resolution CT scan (HRCT) and MR imaging [8,20]. MRI sequences of the brain are also useful in evaluation with contrast-enhanced MRI being useful for determination of tumor origin and extension, as well as location in the cisternal or intracanalicular regions and brainstem [13,20]. HRCT of the temporal bone is useful for bony changes, as there may be involvement and possible degradation of the labyrinth or ossicles, or expansion of the facial nerve canal due to tumor growth [8,13,15]. Calcification on CT imaging has also been reported in about a third of FNS cases [29].

On T1-weighted images, FNS appear iso to hypointense, versus T2 which appears hyperintense compared to the surrounding brain tissue [5,8,14,18,19]. With gadolinium contrast on T1-weighted images, small FNS show strong homogenous enhancement but can have heterogeneous enhancement due to close compacted Antoni type A cells (hyperintense) and loosely packed Antoni type B cells (hypointense), and large FNS show cystic degeneration [5,8,14,15,18,19]. Most common presentation of schwannoma in the internal auditory canal, labyrinth, and temporal bone is a nodular enhancement. Involvement of geniculate ganglion favors facial nerve schwannoma as opposed to vestibular schwannoma [30]. It is important to note that there is physiologic enhancement of facial nerve in its labyrinthine, geniculate, tympanic, and mastoid segments. Outside the skull, the presence of cystic changes and heterogeneity mark the findings of a FNS [13].

Schwannomas share imaging characteristics with neurofibromas, as both are tumors arise from nerve sheaths. Neurofibromas also have low to iso-intense signal on T1, hyperintensity on T2, and postcontrast enhancement, which can make it difficult to discern between the two [24]. The location of the mass relative to the nerve aids in differentiation. In schwannomas, the nerve is peripheral to the mass, traveling around the nerve, differing from the nerve being central to or disrupted by the mass in neurofibromas [1,24]. Neurofibromas also have more poorly defined margins, relative to the smoother margins of a schwannoma. The presence of cystic lesions and heterogeneity also clues to the diagnosis of a schwannoma over a neurofibroma [24].

Due to shared locations of both the facial and vestibular nerves, as well as the higher prevalence of vestibular schwannomas, determining the tumor nerve of origin can be challenging. Extension into the labyrinthine segment favor vestibular schwannoma [8,13,17,18,28,31], whereas the extension into geniculate ganglion favors facial nerve schwannoma. Similar to a transmodiolar vestibular schwannoma, invasion into the cochlea can occur; however, involvement of the facial nerve canal separates the two apart [8,18]. In general, 2 key features to distinguish vestibular schwannomas from FNS is growth in the geniculate ganglion and/or the labyrinthine segment [13].

Differential diagnosis consideration with geniculate ganglion mass-lesion includes, schwannoma, meningioma, dural

metastasis, epidermoid, and cavernous hemangioma [32]. Facial nerve hemangiomas can also arise in the geniculate ganglion; however, these can be differentiated from FNS as they have irregular borders on HRCT and a "honeycomb" appearance, while FNS have a smooth well-defined border [8,18,20,33]. Bell's palsy is able to show postcontrast T1 enhancement of the geniculate ganglion and facial nerve segments, but is differentiated from a FNS due to the quicker onset and potential for spontaneous resolution of symptoms [34]. Congenital cholesteatomas can also affect the facial nerve intratemporally; however, they do not enhance with contrast on MRI, whereas FNS do [14,18,20]. Biopsy is a sure way to diagnose FNS as compared to other masses that occur in the same region; however, it carries a high risk of nerve damage and should only be done if malignancy is suspected.

Treatment of FNS depends on the degree of symptoms, commonly using the House Brackman (HB) scale which considers facial nerve palsy severity [35]. With mild hearing loss and/or normal facial nerve function with a HB score of 1 or 2, observation and serial imaging to monitor is adequate to avoid the complications of further intervention [26,36,37]. The key feature to treatment is maintaining or improving facial nerve functionality, so if there is no compression of cranial structures or worsening paralysis, it is safer to follow up and monitor [10]. If paralysis occurs upon follow-up, steroids may be the first line in treatment, with possible future decompression strategies. Gamma knife radiosurgery is another option for treatment, potentially controlling tumor burden in lesions with a HB score less than 3 [38]. One study showed after gamma knife treatment, most patients had adequate tumor control with either no change or improved facial nerve function [39]. A subset experienced decreased facial nerve function, but it was attributed to compressive damage from bony structures rather than from the gamma knife itself, as the symptoms appeared earlier than the typical radiation injury timeline [39]. Therefore, in lesions with a volume of less than 1 cm^3, early intervention with gamma knife radiosurgery may be a preferred management as opposed to observation due to the potential conservation of facial nerve functionality [38]. FNS with more severe hearing loss, impingement on the bone, and facial paralysis can be offered debulking surgery, radiotherapy, and resection with nerve grafting [37]. Multiple studies suggest that HB grade 3 may be the most adequate level for evaluation of more invasive therapy, with the recommendation increasing as HB grade increases [10,13,40]. House Brackman 3 is the highest level of functioning obtainable with surgery, so it is not recommended if the patient had normal to mild function loss prior [18]. If therapy is chosen over conservative management, an earlier diagnosis and intervention on smaller tumors improves patient outcome [18]. Even after resection, while uncommon, it is possible for intracranial schwannomas to recur [23]. Each of the treatment methods has pros and cons, in which a decision must be made with the patient to determine their goals of care.

In summary, the cases of facial nerve schwannomas generally show symptoms of facial nerve palsy, facial spasms, tinnitus, vertigo, sensorineural hearing loss, and otalgia, which are similar to our patient's presentation. Typical MRI imaging of schwannoma in the internal auditory canal and temporal bone includes nodular enhancement. Furthermore, the anatomical distribution of this pathological enhancement in the internal auditory canal can help determine the cranial nerve origin of the schwannoma.

Patient consent

We have obtained written, informed consent from the patient for the publication of their case. The patient was called initially to ask if they would be willing to participate in the report and was informed of what that would entail. We provided the patient with all relevant information regarding the purpose, nature, potential risks, and benefits associated with the publication of their case.

We ensured that the patient fully understood the implications of publication, including the possible disclosure of personal and medical details, while maintaining their confidentiality. The patient was given the opportunity to ask questions before providing their consent.

We received a signed consent form from the patient, documenting their willingness to participate in this research report.

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