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Facial Nerve Schwannoma- A Case Report

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Abstract:
Facial nerve schwannomas are rare, benign nerve sheath tumors originating from Schwann cells, comprising less than 1% of all temporal bone tumors. Their slow growth and nonspecific symptoms often result in delayed or incorrect diagnosis, with common initial presentations including hearing loss and ear fullness, frequently misattributed to middle ear pathology. They have distinct histopathological variants, each with unique morphological characteristics. Diagnosis is typically established through histopathology and confirmed by diffuse S100 and SOX10 positivity. They are also associated with genetic syndromes such as Neurofibromatosis Type 2. While these tumors are generally non-recurrent following gross total resection, cellular and plexiform variants may require subtotal removal due to their growth patterns. Malignant transformation remains exceedingly rare.

Keywords: *Facial nerve, Schwannoma, Nerve sheath tumor, Intratemporal tumor, Verrocay body.*

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I. Introduction:

Schwannoma is a benign nerve sheath tumour of differentiated Schwann cells[1]. They can occur at any age and are commonly found in peripheral nerves of the head and neck.

Facial nerve schwannomas (FNSs) are uncommon, slow-growing tumours, comprising less than 1% [3] of all temporal bone tumours. They are generally solitary, unilateral, and sporadic, though they can occur bilaterally as part of the neurofibromatosis type 2 spectrum. In rare cases, multiple schwannomas may affect peripheral branches of the facial nerve. The age at which they present can range from 5 to 84 years.They arise along the facial nerve, which originates in the pons, travels through the internal acoustic meatus, and enters the Z-shaped facial nerve canal , which consists of three segments: labyrinthine, tympanic, and mastoid.

Facial nerve schwannomas often present with peripheral facial neuropathy and various otologic symptoms, including sensorineural and conductive hearing loss. Facial paralysis may appear later in the course of the condition or might be entirely absent. This absence is thought to be due to neuronal tolerance caused by the tumour's extremely slow growth, rich vascularity, and the frequent dehiscence of surrounding bone.

II. Case Presentation:

A 35-year-old female patient presented with a 5-year history of hearing loss, which was insidious in onset and gradually progressive in nature, along with complaints of fullness in right ear for the last 2 months. Upon examination, right ear showed a fleshy polypoid mass in the external auditory canal, originating from the posterior wall and completely obstructing the canal. The tympanic membrane could not be visualised, and probe tests indicated obstruction. Hearing tests revealed a negative Rinne's test and lateralization to the right ear on the Weber test, with a mixed hearing loss of 81 dB identified on pure tone audiometry. The vestibulocochlear nerve function was affected, but other cranial nerves were normal, and there were no cerebellar signs. A perforation in the tympanic membrane was noted, with erosion of the incus, while the malleus and stapes remained intact.

Computed Tomography revealed Chronic mastoiditis with a mass size Acquired Cholesteatoma, extending into the bony and membranous part of external auditory canal with erosions of bony wall and

ossicular chains. a diagnosis of Acquired Cholesteatoma *(Figure1)*.

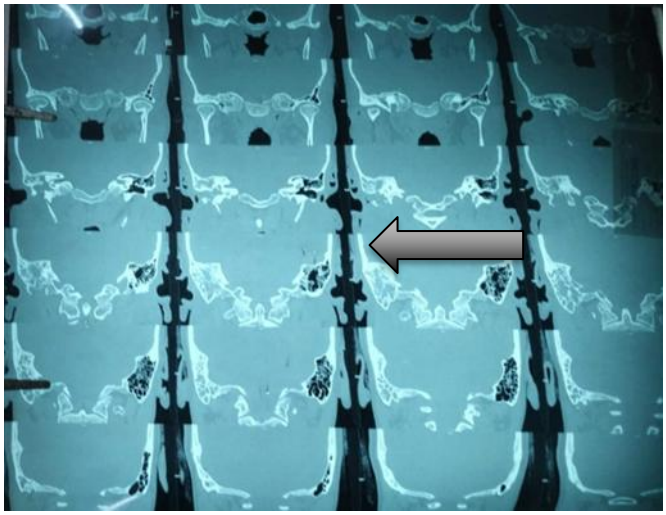

Figure 1: CT image showing dehiscence of the facial canal

A modified radical mastoidectomy was performed, during which the malleus was removed, reshaped, and repositioned over the stapes, and meatoplasty was completed.
The excised polypoid mass was sent for histopathological examination. *(Figure 2)*

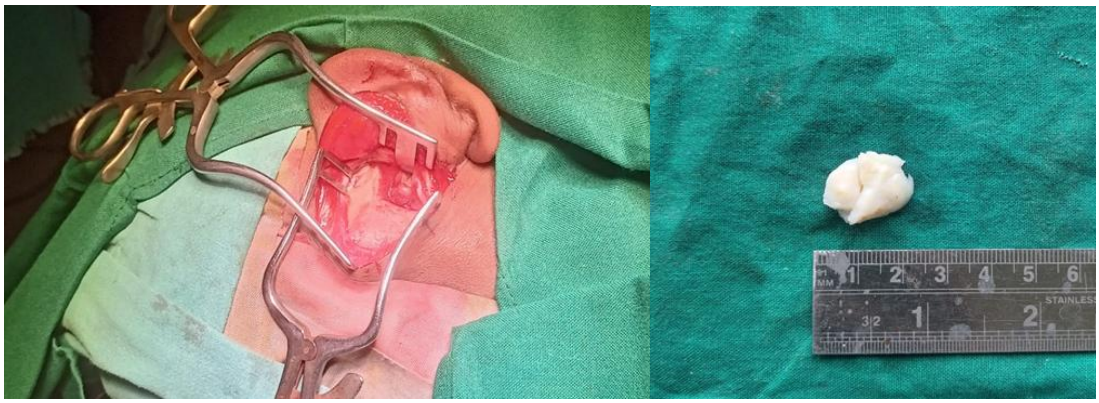

Figure 2: Intra-operative picture of polyp
Figure 3: Gross image (B-939/2023) showing single grey-white soft tissue bit

III. Pathological Findings:

Gross Findings:

Received single grey white soft tissue bit measuring 2x2x0.5 cm. Cut section shows smooth surface, grey-white in colour. *(Figure 3)*

Microscopic Findings:

Sections studied show capsulated tumour with proliferation of bland spindle shaped cells arranged in fascicles and sheets*(Figure 6)*. The individual cells show buckling of nuclei along with focal hypercellular Antoni A and hypocellular Antoni B areas*(Figures 4,5&7)*. Many foci show palisading of nuclei with central nuclear zone indicating verrocay bodies*(Figure 8)*. There is no evidence of nuclear atypia or mitotic activity.

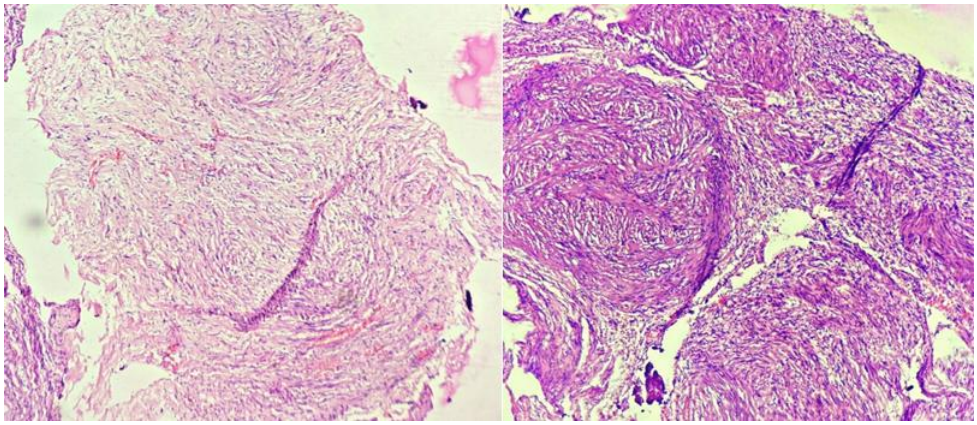

Figure 4: H&E-4x magnification showing Antoni B areas
Figure 5: H&E-4x magnification showing Antoni A areas

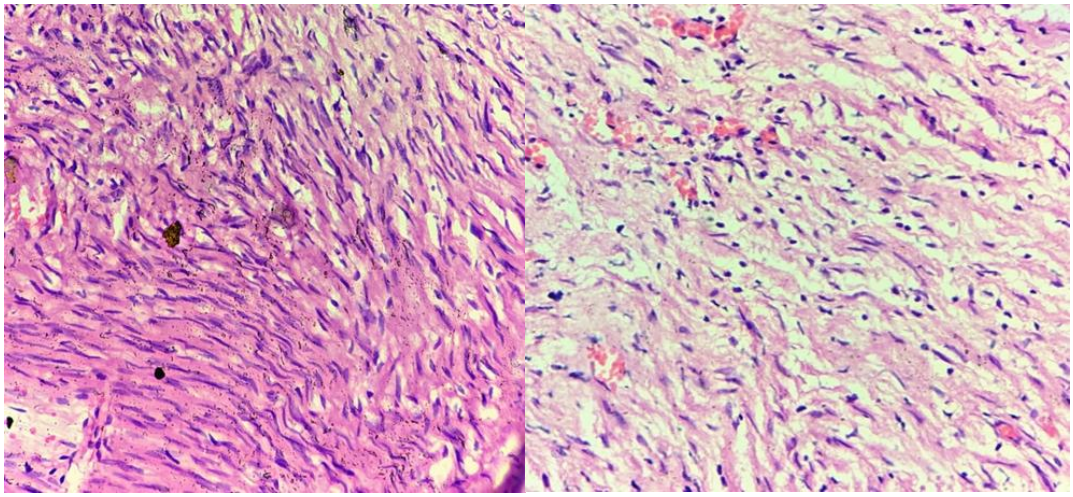

Figure 6: H&E-40x magnification showing spindle shaped Schwann cells
Figure 7: H&E-40x magnification showing Antoni B areas

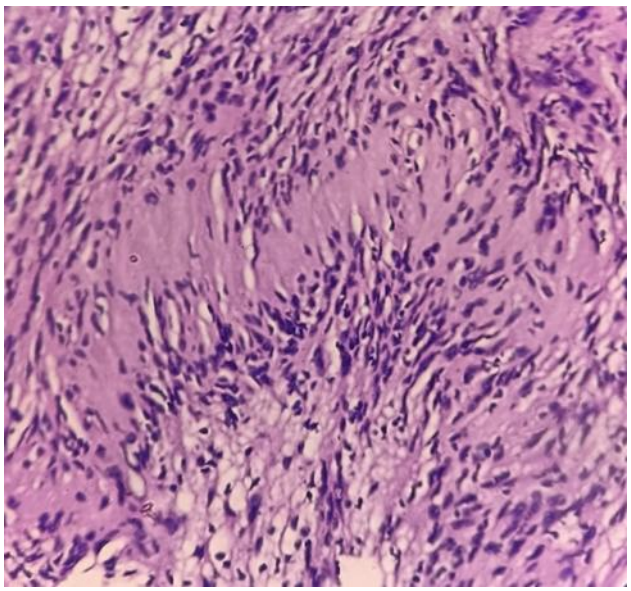

Figure 8: H&E-40x magnification showing Verrocay bodies

Differential diagnosis for this case include Neurofibroma, Leiomyoma and Fibroma.

IV. Discussion:

Facial nerve schwannomas constitute about 0.15% to 0.8% [1] of intratemporal tumors. These tumours often involve multiple segments, primarily located at the labyrinthine segment and the geniculate ganglion[1]. Facial weakness is the most common symptom, followed by hearing loss. However, some patients may maintain normal facial nerve function due to the slow growth of the tumour, allowing for tolerance of nerve compression.

Sunderland et al. reported that 27.3% of patients with FNS have normal facial nerve function [1]. In very rare instances where the mass erodes through the posterior bony wall of the external auditory canal, 27% to 50% of these patients may also exhibit normal facial function [1]; unfortunately, they are often misdiagnosed with middle ear diseases.

In this case, the misdiagnosis occurred because the patient only presented with suppurative otitis media and hearing loss, without any facial nerve paralysis or weakness. Preoperative CT scans only suggesting suppurative otitis media. This case highlights the importance of performing preoperative MRI, which could aid in diagnosis as they show heterogeneous enhancement on MRI due to close compacted Antoni type A cells and loosely packed Antoni type B cells [5,6].

Schwannomas are encapsulated lesions with the capsule continuing directly with the epinerve[3],and may exhibit cystic degeneration and haemorrhage[2].There are a few morphologically distinct variants of schwannomas, like Cellular schwannomas which have a higher cellularity and mitotic rate than conventional schwannomas, Plexiform schwannomas have an intraneural nodular pattern of growth with predominantly Antoni A-type tissue and sometimes Verrocay body formation [4] .Ancient schwannoma differs from the conventional schwannoma only by its presence of scattered atypical to bizarre-appearing nuclei, a feature that is often considered degenerative. Epithelioid schwannomas are sporadic, show multilobulated growth of epithelioid cells, arranged singly or in nests within a myxoid and/or hyalinized stroma. Microcystic/reticular schwannoma are characterized by a microcyst-rich network of interconnected bland spindle cells with eosinophilic cytoplasm, associated with a myxoid, fibrillary, and/or hyalinized collagenous stroma

A diagnosis of schwannoma is readily made on histopathology alone, which is supported by extensive S100[3,8] and SOX10 expression. Genetic Syndromes Associated with Schwannoma is Neurofibromatosis 2[2], a dominantly inherited syndrome, characterized by the formation of multiple schwannomas. Peripheral schwannomas occur in about 70% of NF2 patients[4,8].

Schwannomas do not usually recur if treated by gross total resection. Cellular and plexiform examples are least amenable to total removal and sometimes can only be debulked. Malignant transformation of conventional schwannoma is exceptionally rare [2].

Facial nerve schwannomas, though rare, present a significant diagnostic challenge due to their variable clinical manifestations and potential for mimicking more common middle ear conditions. The absence of facial nerve dysfunction in a substantial subset of patients can lead to misdiagnosis and delays in appropriate treatment. This underscores the critical role of comprehensive imaging,particularly the combined use of CT and MRI, in accurately identifying these lesions preoperatively. Histopathological evaluation remains the definitive diagnostic tool, with immunohistochemical markers providing strong diagnostic support. Recognition of the diverse histological variants is essential for guiding surgical management, especially given that some subtypes may not be amenable to complete excision. Ultimately, a multidisciplinary approach involving otolaryngologists, radiologists, and pathologists is vital to ensure timely diagnosis, appropriate intervention, and favorable patient outcomes.

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