

Giant Cell Tumor of the Temporal Bone with Secondary Aneurysmal Bone Cyst-Like Changes and Extradural Middle Cranial Fossa Extension: A Rare Combination

Akhilesh Chandra Yadav¹; Amit Kumar Tyagi¹; Tanzin Lamo^{1*}; Adithya A. G.¹; Mihir Agarwal¹

¹Department of Otorhinolaryngology and Head and Neck Surgery, All India Institute of Medical Sciences (AIIMS), Rishikesh, Uttarakhand 249203, India

Corresponding author: Tanzin Lamo^{1*}

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Abstract: Giant cell tumor (GCT) of the temporal bone is a rare, locally aggressive neoplasm. Its co-occurrence with secondary aneurysmal bone cyst (ABC)-like changes and extensive extradural extension into the middle cranial fossa (MCF) is exceptionally rare. A 24-year-old female presented with progressive left-sided conductive hearing loss and a slowly enlarging mass in the external auditory canal. High-resolution computed tomography (HRCT) revealed an expansile, erosive lesion involving the left temporal bone with extension into the external auditory canal, middle ear cavity, and mastoid air cells. Magnetic resonance imaging demonstrated a 5 × 3.2 × 4.7 cm heterogeneously enhancing solid-cystic lesion with extradural extension into the middle cranial fossa, causing mass effect on the temporal lobe. The patient underwent complete excision of the tumor with subtotal petrosectomy. Histopathology confirmed giant cell tumor of bone with secondary aneurysmal bone cyst-like changes. Postoperative recovery was uneventful, and follow-up imaging at 2 years showed no residual or recurrent disease. The combination of temporal bone GCT with secondary ABC-like changes and MCF extension represents a rare and aggressive clinical entity. This case justifies a high index of suspicion and a multidisciplinary surgical approach aiming for complete oncological clearance. Long-term surveillance is paramount for management.

Keywords: Giant Cell Tumor, Temporal Bone, Middle Cranial Fossa, Skull Base Tumor, Petrosectomy, Aneurysmal Bone Cyst, External Auditory Canal

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I. INTRODUCTION

Giant cell tumor (GCT) of bone is a benign but locally aggressive osteolytic neoplasm accounting for 4-9.5% of all bone tumors and nearly 20% of benign bone tumors.^[1] It predominantly affects individuals between 20 and 45 years of age, with a slight female predominance.^[2] GCT characteristically arises in the epiphysis of long bones; cranial involvement is rare, representing only 2% of all GCT cases. Among cranial sites, the temporal bone is relatively more susceptible because of its enchondral ossification pattern similar to long bones. Secondary ABC in GCT comprises 14% of all GCTs, but is very rare in the skull base.^[3] We report a rare case of temporal bone GCT with secondary ABC-like changes and extradural extension into the middle

cranial fossa, highlighting important diagnostic and therapeutic considerations. To the best of our knowledge, only two cases of coexisting GCT with ABC have been reported in the temporal bone; we report the third case.

II. CASE PRESENTATION

A 24-year-old female presented with a 6-month history of progressive left-sided hearing impairment, mild otalgia, and a gradually enlarging mass in the left external auditory canal. Examination revealed a reddish, firm, friable, mobile, non-pulsatile mass that filled the left external auditory canal, obscuring the tympanic membrane. Cranial nerve examination, including facial nerve function (House-Brackmann grade I), was normal bilaterally. There was no

cervical lymphadenopathy or other systemic abnormalities. Audiometry showed 50 dB moderate conductive hearing loss on the left. Speech discrimination scores were normal bilaterally. Laboratory results were normal except for severe anemia (Hb: 6.8g/dL), which was optimized preoperatively. HRCT temporal bone showed an ill-defined soft tissue lesion involving the left external auditory canal, middle ear cavity, and mastoid air cells, with extensive bone erosion involving scutum, tegmen tympani, facial nerve canal, and squamous temporal bone. Tegmen tympani erosion created a direct

communication between the middle ear and the middle cranial fossa. Inner ear structures were preserved [Figure 1]. MRI revealed a well-defined, heterogeneously enhancing extra-axial solid-cystic multilocular lesion ($\sim 5 \times 3.2 \times 4.7$ cm), showing T1/T2 hyperintensity with heterogeneous enhancement post contrast. It extended through the eroded tegmen tympani into the extradural space of the middle cranial fossa, causing mass effect on the left temporal lobe without parenchymal invasion [Figure 2].

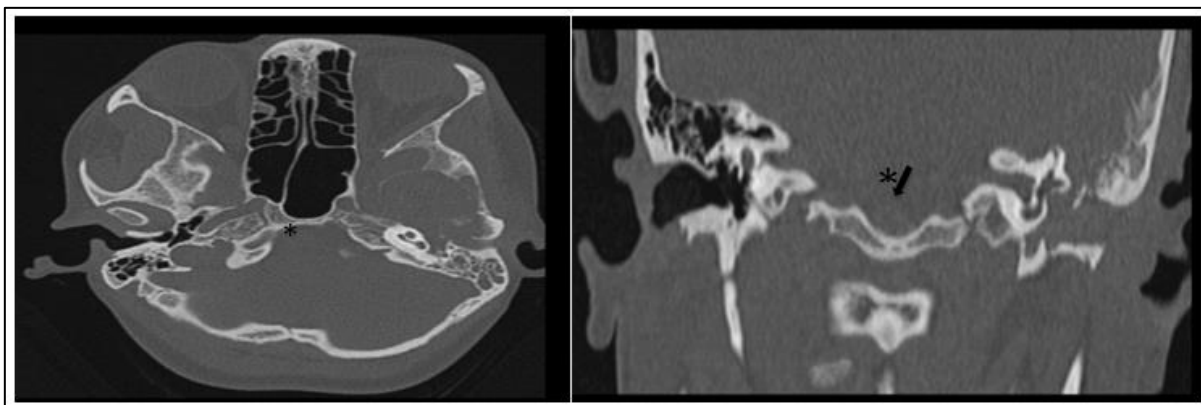


Fig 1 Multiplanar high-resolution computed tomography (HRCT) of the temporal bone showing a solitary, expansile, lucent, osteolytic lesion centered in the left external auditory canal, middle ear cavity, and left mastoid air cells with extensive bone erosion and cortical thinning. Note the destruction of the tegmen tympani (*), scutum (arrow), facial nerve canal, and squamous part of the left temporal bone, creating a pathway for intracranial extension.

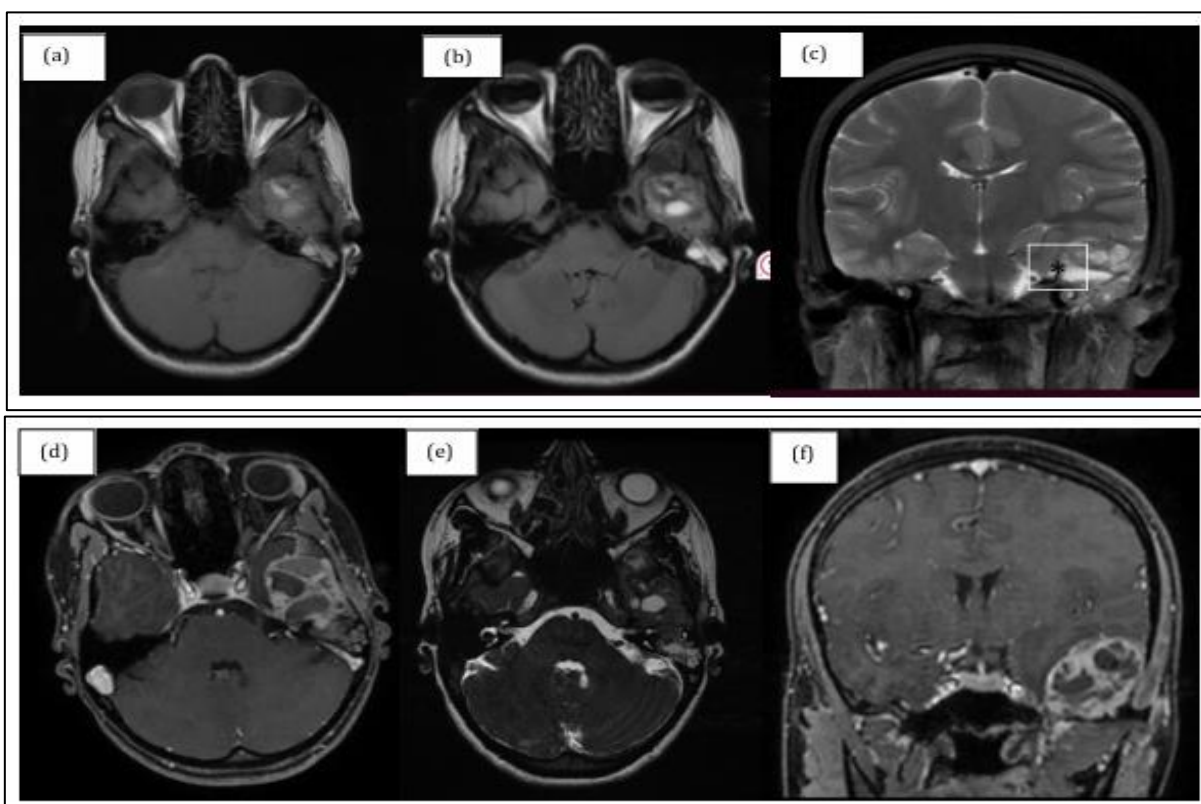


Fig 2 Magnetic resonance images demonstrating a well-defined osteolytic expansive mass in left middle cranial fossa with iso to hypointensity on T1WI (a), with intratumoral hemorrhage as patchy hyperintensity on T1WI (b) and mixed hyper- and hypointensity on T2WI (cystic changes) (e); and fluid-fluid levels (*) within the tumor (c); heterogeneously enhancing on post contrast T1WI (d,f), extra-axial solid-cystic multilocular lesion in the left external auditory canal, middle ear cavity, and extending to middle cranial fossa.

An incisional biopsy via the external auditory canal suggested giant cell granuloma. The patient underwent complete surgical excision of the tumor with subtotal petrosectomy. Intraoperatively, a soft to firm, gray-brown, highly vascular mass with areas of cystic degeneration was found, involving the external auditory canal, middle ear, mastoid air cells, and extended intracranially through the eroded tegmen tympani into the extradural space of the middle cranial fossa.

Histopathology showed sheets of mononuclear stromal cells with numerous uniformly distributed osteoclast-like

multinucleated giant cells containing 20-50 nuclei per cell. The mononuclear cells were plump, ovoid to spindle-shaped, with vesicular nuclei and inconspicuous nucleoli. Mitotic figures were present but not atypical. Areas resembling secondary aneurysmal bone cyst with blood-filled cystic spaces lined by fibrous tissue and containing giant cells were also identified. No evidence of malignant transformation, such as sarcomatous change, was observed [Figure 4]. The diagnosis of giant cell tumor of bone with secondary aneurysmal bone cyst-like changes was made.

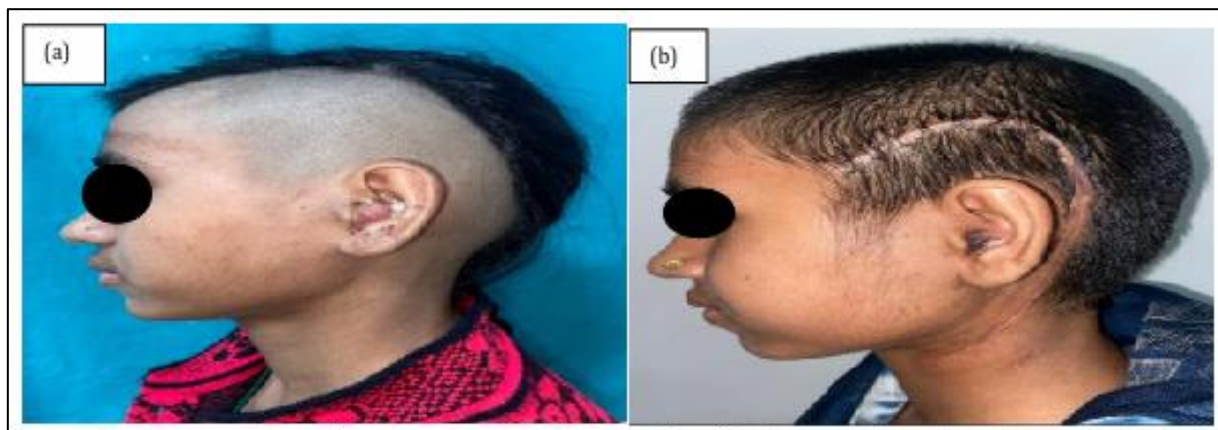


Fig 3 Preoperative and Postoperative Photographs. (A) Preoperative View Showing the Tumor Protruding from the Left External Auditory Canal. (B) Postoperative View after Complete Excision Via the Infratemporal Fossa Approach

➤ Histopathological Findings

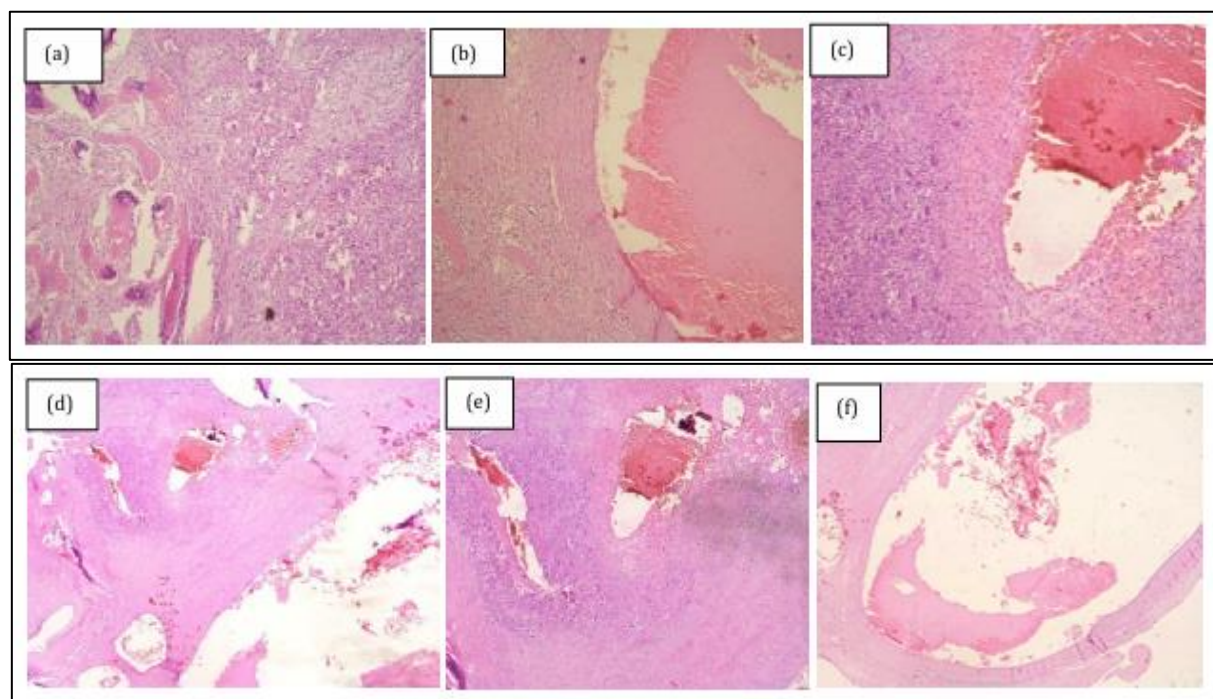


Fig 4 Histopathological features of the temporal bone giant cell tumor. (A) Low power view (×100) showing GCT areas on the left and aneurysmal bone cyst (ABC)-like areas on the right. (B) Low power view (×100) showing classic GCT areas with uniformly distributed giant cells. (C) Low power view (×100) showing ABC-like areas with blood-filled cystic spaces. (D) High power view (×200) showing GCT areas on the left and ABC-like areas on the right. (E) High power view (×200) showing ABC-like areas. (F) High power view (×200) showing osteoclast-like multinucleated giant cells with mononuclear stromal cells eroding bone

III. DISCUSSION

GCT was first described by Sir Astley Cooper in 1818, and temporal bone involvement was later reported by Doderlein in 1913.[4] In the skull, the sphenoid bone is the most commonly affected site, followed by the non-squamous portion of the temporal bone. A review of the literature reports a total of 110 cases of Giant Cell Tumor (GCT); among these, 37 from the temporal bone, 20 from the sphenoid bone, six from the occipital bone, and two from the frontal bone. Notably, none of these 110 cases showed secondary aneurysmal bone cyst (ABC) changes.[1] Nearly 14% of GCTs are associated with secondary ABC formation.[5] Common symptoms include localized pain, headache, preauricular or temporal swelling, conductive or sensorineural hearing loss, aural fullness, and, less commonly, facial nerve dysfunction.[2] CT demonstrates an expansile, lytic lesion with cortical thinning and destruction, with “soap bubble” appearance sometimes seen in GCTs. MRI provides superior soft tissue characterization and is essential for detecting intracranial extension. GCTs exhibit an isointense-to-hypointense signal on T1- and T2-weighted sequences, with heterogeneous enhancement on post-contrast sequences. Fluid–fluid levels are believed to be due to the formation of secondary aneurysmal bone cysts (ABCs).[6] Histopathological examination remains the gold standard for confirming the diagnosis. The differential diagnosis of a giant cell–rich lesion of the temporal bone includes giant cell reparative granuloma (GCRG), aneurysmal bone cyst (ABC), brown tumor of hyperparathyroidism, chondroblastoma, and metastatic giant cell–containing lesions. GCRG closely mimics GCT both clinically and radiologically; however, GCRG typically shows preservation of the cortical bone, smaller giant cells with fewer nuclei, and a more fibrous stroma. In contrast, GCT demonstrates cortical destruction and uniformly distributed large multinucleated giant cells containing numerous nuclei (typically 20–50 per cell). Histopathology of GCT is characterized by gray-to-yellow brown with cystic areas macroscopically and microscopically, it includes multinucleated giant cells, round monocyte-like cells, and spindle-shaped fibroblast-like stromal cells.[1,7] Complete surgical resection is the primary therapeutic goal to minimize the risk of recurrence. However, the complex anatomy of the skull base, involving the internal carotid artery, facial nerve, and jugular bulb, often makes wide margins unattainable. Reported recurrence rates range from 40% to 60%, highlighting the locally aggressive nature of the disease.[8] Radiotherapy and chemotherapy have limited roles in GCT management and are generally reserved for unresectable or recurrent disease. Irradiation-induced malignant transformation is the described complication.[9] GCTs exhibit RANKL overexpression, which is responsible for the recruitment of osteoclast precursors to form characteristic multinucleated giant cells. Denosumab, a RANKL inhibitor, has emerged as a promising adjuvant therapy for unresectable or recurrent GCTs and may be considered in cases where complete excision is not feasible.[5,10] Its role in temporal bone GCTs with secondary ABC is yet to be established. Intracranial extension represents a locally aggressive and invasive nature, although our patient showed no evidence of recurrence at 2-year follow-up, the

long-term prognosis for temporal bone GCTs with secondary aneurysmal bone cyst (ABC)-like changes remains uncertain, necessitating prolonged follow-up. Pulmonary metastasis is rare, occurring in approximately 1% of cases.[8]

IV. CONCLUSION

Temporal bone giant cell tumor (GCT) with secondary aneurysmal bone cyst (ABC)-like changes and intracranial extension is an exceptionally rare entity, posing significant diagnostic and therapeutic challenges. Histopathological evaluation remains the cornerstone for definitive diagnosis and for differentiating GCT from other giant cell–rich lesions. This case highlights the importance of a high index of suspicion. Complete surgical excision remains the mainstay of treatment.

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