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LETTER TO THE EDITOR

Giant cell tumor of the temporal bone

Temporal kemiğin dev hücreli tümörü

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To the Editor,

Giant cell tumor (GCT) arises from undifferentiated mesenchymal cells of the bone marrow[1]. GCT, a benign and locally aggressive tumor, is more commonly seen in the metaphysis of long bones[2]. It may cause local bone destruction, recurrence, and pulmonary metastases (~1%)[3]. They are rarely found in the skull and temporal bone and present with different symptoms depending on location. GCT of the temporal bone may cause otalgia, ear fullness, conductive or sensorineural hearing loss, tinnitus, localized swelling in the temporal or preauricular region, temporomandibular joint dysfunction, and facial paralysis[4]. This article presents a case of temporal bone GCT in light of the literature.

A 15-year-old male patient was admitted with complaints of swelling on the right face, a headache that has persisted for one year and progressively worsened in the last three months, and hearing loss. There were no features in his self-history and family history. On physical examination, immobile, poorly circumscribed 4x5 cm swelling in the right temporal region was not sensitive. The otoscopic review of the patient was normal. However, on audiometric examination, conductive hearing loss was observed on the right side. Computed tomography (CT) imaging depicted a mass lesion arising from the squamous part of the temporal bone, caused erosion and scalloping in the greater wing of the sphenoid bone and extends under the skin by eroding the temporal bone, including hyperdense levels and calcified septas (Figure 1).

Magnetic resonance imaging (MRI) revealed a lobulated contoured, 52x50 mm sized mass that originates from the right temporal bone squamous part, arches the adjacent dura, extends to the middle temporal fossa, subcutaneous fat tissue, temporal muscle, and the temporomandibular joint. The lesion was hyperintense in T1 WI and hypointense in T2 WI, revealing fluid-fluid levels, diffusion restriction (Figure 2), and heterogeneous peripheral and septal enhancement (Figure 3).As a result of the imaging findings, surgical excision was recommended as a treatment option. Craniotomy and total tumor excision were performed. It was observed that the tumor invaded the infratemporal fossa and extended to the anterior of the right temporomandibular joint and the inferior of the greater wing of the sphenoid bone. Histopathological examination revealed a giant cell tumor of the bone. Radiological follow-up was recommended at close intervals in the postoperative period. A voluntary consent form was obtained from the patient.

GCT originates from non-osteogenic stromal cells of the bone marrow[5]. It constitutes approximately 3-7% of all primary bone tumors[6]. It is common in the fourth or fifth decades and in women[4,7]. It usually occurs in long bones such as the distal radius and femur, proximal tibia, and fibula[6]. It is infrequent in the skull, accounting for 1–2% of the GCTs of the bone. It occurs most frequently in the sphenoid and the temporal bone due to their endochondral ossification, like long bones[8,9].

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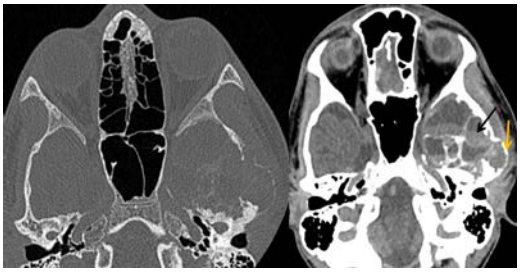

Figure 1. On cranial CT examination is revealed an expansile mass lesion, in the right temporal fossa, on the sphenoid bone greater wing, and the squamous-tympanic part of the temporal bone extends to under the skin by eroding the temporal bone (yellow arrow). In addition, it contains peripheral-septal calcifications and hyperdense fluid levels (yellow arrow).

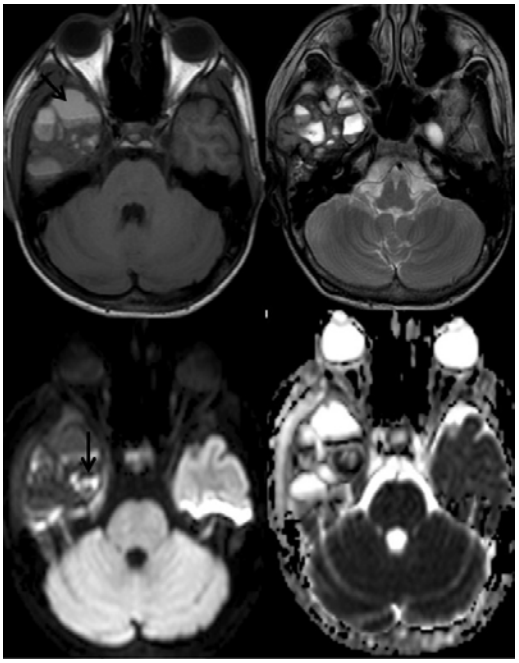

Figure 2. On cranial magnetic resonance imaging, a lobulated mass in the right temporal region, with the destruction of the temporal bone, extention under the skin, and fluid levels that hyperintense (black arrow) on T1 WI, hypointense on T2 WI, and showing diffusion restriction (black arrow) is observed.

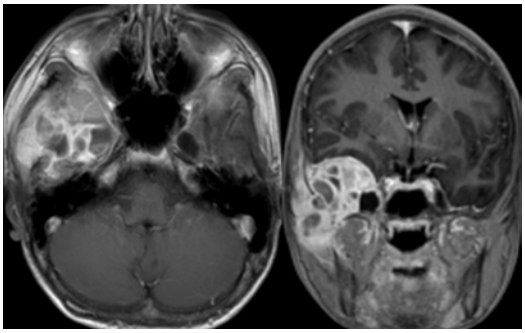

Figure 3. After contrast injection, the mass reveals heterogeneous peripheral and septal enhancement.

Signs and symptoms GCT of the temporal bone often does not occur until it reaches large sizes before clinical symptoms, and diagnosis is often delayed[2]. The patient may present with conductive or sensorineural hearing loss, vertigo and tinnitus, a feeling of fullness in the ear, a mass in the external auditory canal, ear pain, facial weakness, obstruction of the Eustachian tube, and even facial paralysis[8,9]. GCT is observed as an expanding lytic and destructive lesion on temporal bone CT examination, which is enhanced due to its vascular structure. It can sometimes extend to adjacent soft tissues, dura mater, or intracranial area and sometimes shows a soap bubble appearance[2,7]. MR images show intermediate signal intensity on T1-weighted images, hypointense on both T2 and diffusion-weighted images, and heterogeneous enhancement after intravenous contrast administration[3].

Histopathological examination reveals the predominance of large multinuclear giant cells scattered in spindle-shaped stromal cells[9]. GCT is a benign tumor with a good prognosis after complete resection[3]. In the case of partial resection, recurrence rates increase to 40-60%[7]. GCT may act locally aggressively and show malign transformation and metastasis[9]. GCT may show malignant transformation to fibrosarcoma, osteosarcoma, or malignant fibrous histiocytoma[6,9]. Malignancies can be primary or secondary. Primary malignant GCT of the bone is very rare and evident in the initial biopsy and occurs in approximately 3% of cases[2]. Secondary malignant GCT of bone occurs in the previously treated GCT region and is more common[9]. They metastasize to the lung by a hematogenous spread in approximately 5-10% of malignant cases[2].

The histopathological differential diagnosis of GCT includes giant cell granuloma, aneurysmal bone cyst, non-ossifying fibroma, osteogenic sarcoma, benign fibrous histiocytoma, and Brown tumor associated with hyperparathyroidism[9]. In addition, it should be considered in the differential diagnosis of cholesteatomas, paragangliomas, and Langerhans cell histiocytosis in middle ear lesions[8].

Complete and radical resection is the preferred treatment option[9]. However, when there is an intracranial extension, it is often challenging to perform complete excision of the temporal bone due to its close association with vital structures and morbidity[9]. The recurrence rate is proportional to the adequacy of surgical excision. Up to 50% of GCTs recur within three years after surgical curettage[10]. Adjuvant radiotherapy can be applied in such cases, but it is a controversial treatment option because it can trigger malignant transformation in residual tumor tissue[6]. In unresectable cases or with severe morbidity of surgical resection, chemotherapy can be given in possible cases that will result in complications[2]. Bisphosphonates can prevent local recurrence and reduce bone destruction following surgery[2].

GCT of the skull is a rare lesion, especially in the temporal bone. The clinical features are nonspecific and depend on the localization and extent of the tumor. Therefore, a definitive diagnosis can only be made histopathologically. Complete surgical excision with negative margins is the preferred treatment method.

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REFERENCES

1. Akyigit A, Karlidag T, Sakallioglu Ö, Polat C, Keles E. Giant cell tumor of bone involving the temporomandibular joint and temporal bone. J Craniofac Surg 2014;25:1397-9.
2. Sepulveda I, Alzerreca J, Villalobos P, Ulloa JP. Giant cell tumor of the temporal bone with skull base and middle ear extension. J Med Cases. 2021;12:149-51.
3. Iizuka T, Furukawa M, Ishii H, Kasai M, Hayashi C, Arai H et al. Giant cell tumor of the temporal bone with direct invasion into the middle ear and skull base: a case report. Case Rep Otolaryngol. 2012:2012; 690148
4. Kaya İ, Benzer M, Turhal G, Sercan G, Bilgen C, Kirazlı T. Giant Cell Tumor of the Temporal Bone and Skull Base: A Case Report. J Int Adv Otol. 2018;14:151-4.
5. Jain S, Sam A, Yohannan DI, Kumar S, Joshi D, Kumar A. Giant cell tumor of the temporal bone—an unusual presentation. Clin Neurol Neurosurg. 2013;115:646-8.
6. Borges BB, Fornazieri MA, Bezerra AP, Martins LA, Pinna Fde R, Voegels RL. Giant cell bone lesions in the craniofacial region: a diagnostic and therapeutic challenge. Int Forum Allergy Rhinol. 2012;2:501-6.
7. Koo T, Danner C. A rare case of giant cell tumor of the temporal bone. Otolaryngology Case Reports. 2020;15:100160.
8. Miller MA, Kesarwani P, Crane BT. Autophony in a patient with giant cell tumor of the temporal bone. Otol Neurotol. 2016;37:e238-9.
9. Jain A, Singh I, Shankar R, Varshney D. Giant cell tumour of temporal bone and infratemporal fossa: a rare case. Head Neck Pathol. 2020;14:503-6.
10. Czerniak B. Giant cell lesions. In Dorfman and Czerniak'sBone Tumors (Eds HD Dorfman, B Czerniak):692-759.. Philadelphia, Elsevier Saunders, 2016.