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Case Report

Giant cell tumor of sellar region: A rare case report and review of literature

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ABSTRACT

Primary bone neoplasms are an uncommon group of neoplasms that include benign to malignant and locally aggressive or rarely metastasizing types. Giant cell tumor is a very rare tumor of the skull, with few of those reported cases arising from the sphenoid and temporal bone. Exact localisation is essential in the formulation of differential diagnosis between the diverse pathologies that affect this region. Herein, we discuss a case of Giant cell tumor in the sellar region.

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INTRODUCTION

Primary bone neoplasms are uncommon and account for only 0.2% of human neoplasms that range from benign to malignant, with intervening locally aggressive or rarely metastasizing types.[1] Among other characteristics, each bone tumor has its own age predilection and radiological findings, which are useful from a differential diagnosis point of view.

Giant cell tumor (GCT) is one of the many primary bone neoplasms arising from the epiphysis of long bones of

young adults. It is a very rare tumor of the skull, with few of those reported cases arising from the sphenoid and temporal bone. Bone tumors arising from the skull comprise diverse groups ranging from benign entities like osteoma, fibrous dysplasia to the rare malignant ones consisting of chondrosarcoma, osteosarcoma, and metastasis. In regard to the clinicopathological and radiological characteristics, the most common location was clivus, followed by the parasellar and frontal region.

The sellar region is made of the sphenoid bone and includes sella turcica and pituitary gland together with the ventral adenohypophysis. The parasellar region encompasses the cavernous sinuses, suprasellar cistern, hypothalamus, and ventral inferior 3rd ventricle. Exact localisation is essential in the formulation of differential diagnosis between the diverse pathologies like neoplastic, inflammatory/granulomatous, infectious, and vascular disease that affect this region. The commonly encountered neoplasms include pituitary macroadenoma/ microadenoma, craniopharyngioma, meningioma, and hypothalamic chiasmatic glioma.

GCT should be distinguished from the other giant cell lesions, of which central giant cell reparative granuloma is an important one, as the latter has a lower recurrence rate and for which no cases of malignant transformation or metastasis have been reported, although both lesions may require postoperative radiotherapy to achieve local control.

CASE REPORT

A 17-year-old male patient without any previous co-morbidities presented with diplopia, ipsilateral ptosis, and frontal headache for 2 months at a tertiary care hospital. The patient was clinically examined and investigated.

The MRI revealed a well-defined extra-axial lobulated solid lesion isointense in T1 and T2 weighted sequences with few peripheral areas of GRE blooming (calcification), total lesion measuring 31x36x32 mm in the Sella turcica, causing its widening. (fig.1) It extends superiorly to the optic chiasma, displacing it. Laterally it abuts the cavernous sinus. Inferiorly towards the sellar floor there is erosion of the floor, clivus, anterior and posterior clinoid process. Serum Prolactin levels were normal and thus a provisional diagnosis of non-functioning pituitary adenoma was put forth.

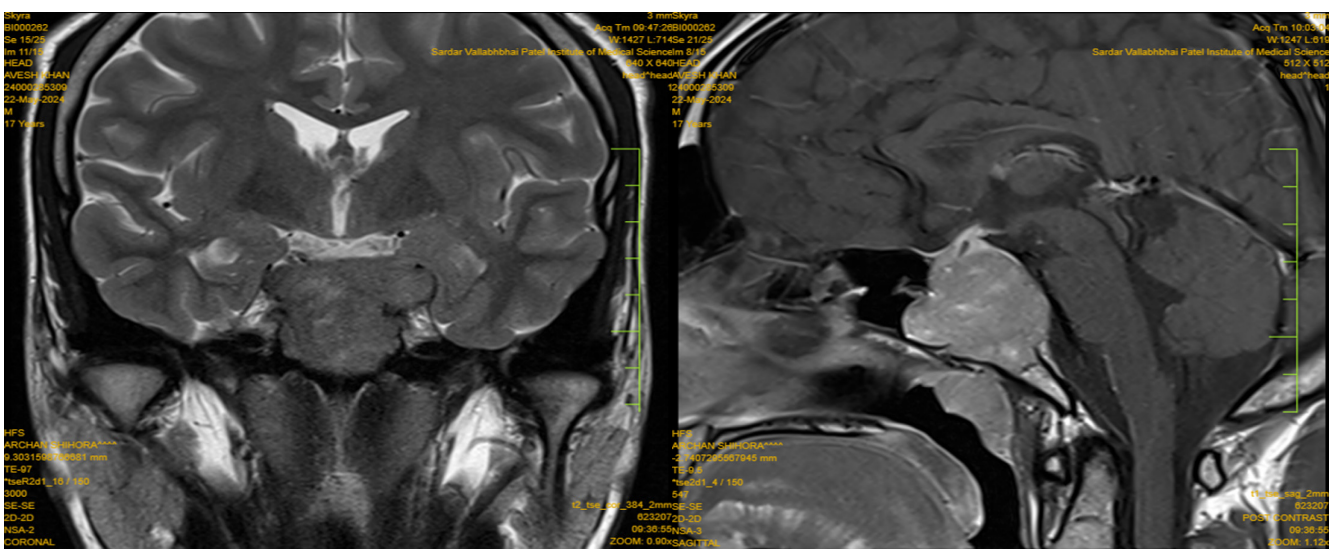

Figure 1: *MRI images revealed a well-defined extra-axial lobulated solid lesion isointense in T1 and T2 weighted sequences with few peripheral areas of GRE blooming (calcification).*

The patient underwent transsphenoidal surgery, and the resected tumor was sent for histopathological examination. Grossly, the specimen consisted of multiple reddish white soft tissue portions totaling 3.5 x 3 cm in aggregate. Microscopic examination shows cytologically benign oval to polyhedral mononuclear cells admixed with numerous evenly distributed osteoclast-like giant cells with more than 15 nuclei. (fig.2) There was evidence of woven bone formation within the tumor. (fig.2) No evidence of sarcomatous transformation, haemorrhage lakes with clustering of giant cells (as in Giant cell reparative granuloma), or pituitary tissue was identified.

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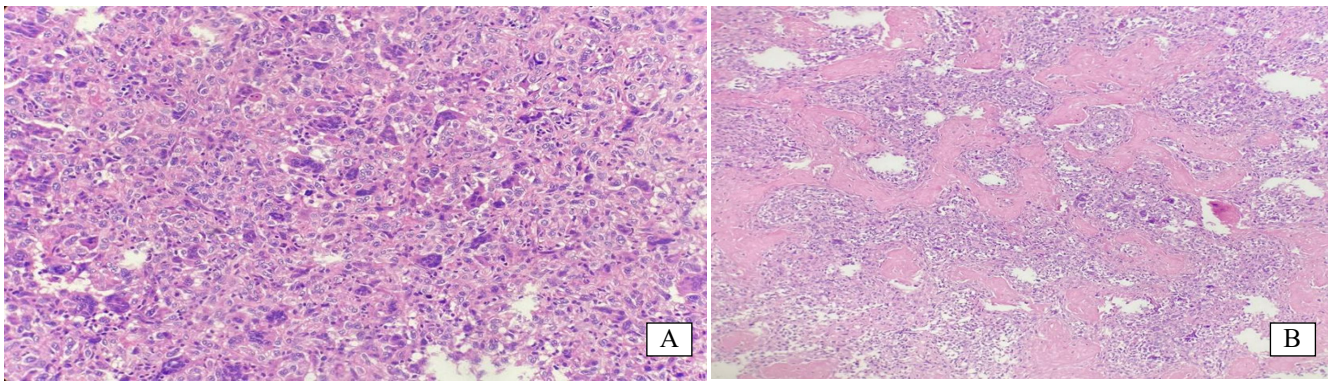

Figure 2: *A. Cytologically benign oval to polyhedral mononuclear cells, admixed with numerous evenly distributed osteoclast-like giant cells. (HE stain, X400) B. Woven bone formation between the tumor. [H&E; 40x]*

DISCUSSION

Giant cell-containing tumors of the bone comprise a wide variety of tumors and tumor-like lesions characterized by the presence of numerous osteoclasts or osteoclast-like giant cells. The differential diagnosis of these GCTs has a long list as follows: Giant Cell Tumor of bone, Chondroblastoma, Chondromyxoid fibroma, Aneurysmal Bone Cyst, Langerhans Cell Histiocytosis. Central Giant Cell Reparative granuloma, Fibrous dysplasia, Giant Cell-rich osteosarcoma, Telangiectatic osteosarcoma, simple bone cyst with fracture, Paget disease, Fracture, Osteitis fibrosa cystica.

Giant Cell tumor comprises less than 5% of benign tumors arising from the bone marrow. Just 2% of all GCTs are seen in the cranium [2,3]

The significance of the correlation of histology with clinical, radiological, and laboratory findings cannot be emphasized enough in such scenarios.

The MRI enhancement pattern may vary, although they usually show a low intensity on T2-weighted images due to associated hemosiderin and calcification. The differential diagnosis on imaging includes CGCG, Osteitis Fibrosa Cystica, Osteolytic metastasis, Plasmacytoma, and chondroblastoma. GCT and CGCG cannot be distinguished solely on imaging.

In our case, MRI showed a well-defined extra-axial lobulated solid lesion isointense in T1 and T2 weighted sequences. A possibility of a pituitary gland-based tumor was put forth. S. PRL levels were normal, which indicated either a nonfunctioning pituitary adenoma or a non-pituitary-based tumor. Histologically, there were oval to polyhedral mononuclear cells admixed with evenly distributed osteoclast-like giant cells. No pituitary tissue was identified, thus reducing our list of differential diagnoses to a bone origin tumor containing osteoclast giant cells that include Brown tumor, GCT, and CGCG.

On further prodding patient had normal Serum calcium, S.PTH, and S.ALP levels that helped us exclude brown tumor, leaving us with two main differentials of GCT of bone and CGCG.

CGCG of the jaw was first defined by Jaffe in 1953.[4] It is a benign process commonly occurring in the mandible and maxilla, which is related to trauma and intraosseous haemorrhage or infection and developmental abnormalities.[5] It presents over a wide range, particularly in the middle-aged to older adults, as a broad-based nodule that is reddish or blue and is less than 2 cm in diameter. Microscopically, it consists of a mixture of multinucleate osteoclast-like giant cells and plump, spindled to oval mononuclear cells. There are many overlapping features between GCT and CGCG. Hirsch & Katz[6] defined five major histological criteria that help distinguish between these two lesions, though borderline cases that show features of both are also encountered.

Table 1: Criteria for differentiation of giant cell reparative granuloma from giant cell tumor of bone (modified Hirschl and Katz)[6]

	Giant cell reparative granuloma of bone	Giant cell tumor of bone
Age	<20 years old	20-40 years old
Location	Mandible, Maxilla	Long bone epiphyses
Histological features	1. Giant cells in groups around hemorrhagic foci. 2. Stroma shows oval cells, many spindle-shaped fibroblastic cells with zones of abundant collagen, and relatively few giant cells. 3. Evidence of marked haemorrhage with marked hemosiderin deposits in older lesions. 4. Giant cells are smaller, irregular, and elongated, with relatively few nuclei. 5. Foci of osteoid and new bone formation are often seen in the middle of the lesion.	1. Uniformly dispersed giant cells 2. Stroma is composed of plump, round, and oval cells together with a rich vascular network. 3. Fresh haemorrhage is uncommon; hemosiderin deposits are rare and small. 4. Giant cells are larger, more rounded, with many nuclei. 5. Does not usually produce osteoid or new bone.

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Skull and facial bone giant cell lesions occur rarely, except within the mandible and maxilla. Bertoni et al[7] and Williams et al[8] have compiled the most recently published cases, and there have been only a small number of additional case reports since.[9,10]

CGCG and GCT of bone both have a female predilection. Most patients of CGCG are less than 35 years old at the time of diagnosis, while 20-33% of patients with GCT are over 50 years of age. Expression of p63 has been found by some to be useful to distinguish between GCT and other non-neoplastic giant cell lesions.

In our case, microscopic examination revealed uniformly dispersed giant cells with more than 15 nuclei with surrounding oval to polyhedral, benign-looking mononuclear cells. There was no evidence of hemorrhage or hemosiderin deposit.

GCT of Bone should be distinguished from CGCG because there is a higher recurrence rate, risk of metastasis, and malignant transformation in the former. The recurrence rate of 25% in GCT is based predominantly on reports of GCT of long bones. No such analysis was done on the rarely occurring ones in the skull.

CONCLUSION

GCT of skull bone is a rare entity, and most of them arise from the jaw bones, the sphenoid, and the temporal bones. The main differential diagnosis of Giant cell lesions of bone includes CGCG, GCT of bone, and Brown tumor. Elevated S.PTH and S. Ca help one exclude Brown tumor. CGCG and GCT are indistinguishable radiologically, and in a few cases show overlapping histological features. Histologically, our case showed features favouring GCT over CGCG. Surgical excision is the first line of treatment for both lesions. There is no consensus regarding their behaviour and prognosis. Advanced research, including molecular genetic investigations, is required to elaborate further on their treatment protocol and prognosis.

Conflict of Interest: None

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