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CASE REPORT

Aggressive Giant Cell Tumour of Maxilla

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ABSTRACT

Giant cell tumours (GCT) of bone are infrequently encountered in craniofacial skeleton and require careful evaluation to differentiate it from other giant cell lesions. The management of these tumours is ofter complicated, as complex anatomy of the region makes complete resection difficult and lesion has significant recurrence rate. Loco regionally advanced disease makes complete resection even more challenging. Here, a case of locoregionally advanced GCT maxilla in 37-year-old female is reported. The patient was successfully managed with complete resection and reconstruction of orbital floor using temporalis muscle flap sling.

Key Messages: Giant cell tumours of head and neck are rare. However, they require careful evaluation to differentiate it from other similar lesions and a comprehensive management, considering their recurrent nature and probable malignant transformation.

Keywords: Benign tumour, GCT, Giant cell tumour, Maxilla

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INTRODUCTION

Giant cell tumour (GCT) of head and neck region is a rare primary bone tumour, constituting 1–2% of all GCTs. Although locally aggressive, the tumour is benign in nature with arguable malignant potential. Of all GCTs, less than 10% are malignant.[1-3] Rare occurrence and similarity with other giant cell lesions makes the diagnosis challenging and critical for appropriate management. A case of locally aggressive GCT of maxilla causing orbital floor erosion is presented here, which was managed with complete resection and primary reconstruction of orbital floor using temporalis muscle flap sling.

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CASE HISTORY

A 37-year-old female, presented with swelling of left side of face for 4 months. Clinically there was a diffuse, hard and nontender swelling over left cheek measuring 4 cm in diameter. External nasal pyramid was not deformed, however there was a mass arising from lateral wall of left nasal cavity. There was a bulge in hard palate limited to left side. Rest of the oral cavity, neck, eyes and dental examination were unremarkable. Diagnostic nasal endoscopy showed a diffuse bulge on lateral wall of left nasal cavity, displacing inferior and middle tubinates medially. A biopsy was taken and histopathological examination of lesion revealed giant cell tumour. All hematological and biochemical investigations including serum calcium, serum phosphate and serum alkaline phosphatase were normal.

Computed tomography (CT) scan of nose and peripheral nervous system (PNS) showed a well-defined homogenously enhancing expansile lesion in left maxillary sinus and causing destruction of anterior maxillary wall. Medially, it was causing destruction of medial wall of maxilla, extending into left nasal cavity abuting left nasolacrimal duct and nasal septum. Superiorly, there was erosion of inferior orbital wall and tumour was extending into inferomedial part of orbit, abutting inferior rectus muscle.

In MRI, the lesion was heterogeneously hyperintense on T2W1 and FLAIR and iso to hypointense on T1W1. The mass demonstrated heterogenous contrast enhancement , with no perineural extension. The fat plane with inferior rectus were preserved and inferiorly mass was destroying alveolar process of maxilla and hard palate with intraoral extension (Fig. 1). Whole body bone scan revealed diffuse tracer uptake at left maxillary region, with no evidence of any skeletal metastasis or multifocality.

Total maxillectomy was done under general anesthesia with intraoperative frozen section guided clearance of the margins. Orbital floor was found to be eroded and required reconstructed using temporalis muscle sling with periosteum. Split skin graft was used to cover the intraoral maxillary defect. Postoperative recovery of the patient was uneventful.

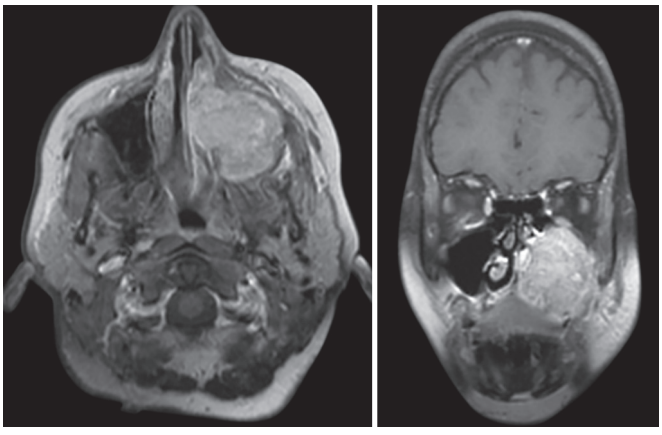

Fig. 1: Preoperative MRI

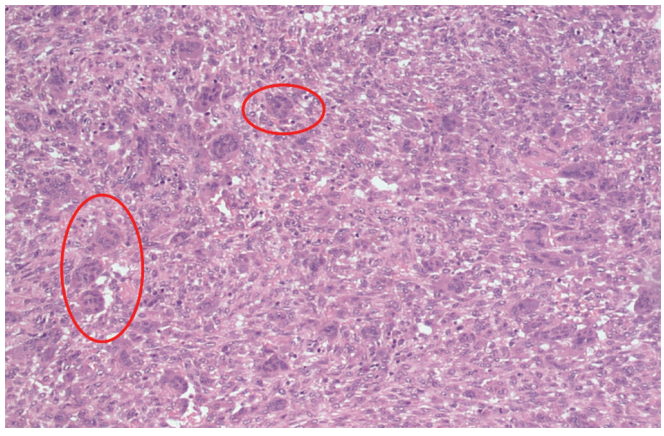

Fig. 2: Histopathological examination of lesion showing multiple giant cells (marked)

The histopathological examination of specimen was performed and it showed two populations of cells, composed of numerous osteoclasts and mononuclear cells (Fig. 2). The giant cells showed 10–15 nuclei with vesicular chromatin and conspicuous nuclei with no atypical mitosis.

The patient was followed up regularly clinically and with imaging. Follow-up MRI (Fig. 3), which was repeated after 3 months and 1 year, showed no evidence of recurrence and satisfactory reconstructive outcome.

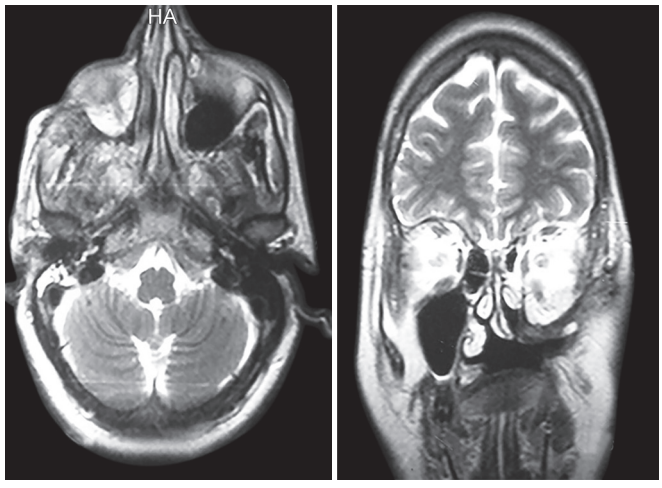

Fig. 3: Postoperative MRI

DISCUSSION

Giant cells are large multinucleated cells of varied origin and are nonspecific diagnostic value. The various neoplastic and benign lesions mimicking neoplastic conditions like central giant cell granuloma, GCT, osteosarcoma and rhabdomyosarcoma, can show presence of giant cell. Metabolic diseases like hyperparathyroidism and other conditions like cherubism, aneurysmal bone cyst, pagets disease and fibrous dysplasia can also present as giant cell lesion.[4] GCT is an important clinicopathological entity due to its obscure diagnosis, benign yet locally aggressive nature, tendency to recur and possibility of malignant transformation.[5]

The GCTs are common bone tumour constituting 4–5% of all bone tumour. Most commonly affected site is epiphyseal region of long bones, and head and neck region constituting only 2% of the cases.[6] The common sites in head and neck region are mandible maxilla, ethmoid, sphenoid, temporal bone, zygoma and temporal bone.[2,7]

It is worth noting that true neoplastic cells in GCT are mononuclear stromal cells of mesenchymal origin, and not the multinucleated giant cells. Histopathologically, benign GCTs have loose or highly vascular stroma with numerous spindle cells and many multinucleated giant cells. Malignant GCT shows marked polymorphism of nuclei, atypical mitosis and stromal dominance.[2]

True GCTs of head and neck region are not always easy to differentiate from other giant cell lesions, even histologically. Giant cell granuloma (GCG) can mimic GCT. It can be a central GCG, which is an intraosseous lesion or peripheral GCG, also known as giant cell reparative granuloma. Sometimes GCT and GCG are considered a spectrum of same disease and the differentiation between the two entities is important from management point of view. Brown tumour of hyperparathyroidism can be differentiated by laboratory investigations like serum calcium and phosphate, ALK phosphatase and PTH assay. Aneurysmal bone cyst can be differentiated from GCTs on histologically.

Although GCT is typically described as well circumscribed expansile osteolytic lesion on CT scan and heterogeneously enhancing mass of variable signal intensity on MRI, but in head and neck region, it can present with nonspecific radiological features.[8] An increased uptake by the tumour is demonstrated on bone scan.

Complete resection of tumour is most acceptable modality among all modalities available for management. Simple curettage results in high recurrence rates reaching

60% as compared to 7% by wide local resection.[6] Most recurrences appear in first 2–3 years and continuous evaluation from 5 years to indefinite period have been recommended in view of possible pulmonary metastasis.[5,9,10] Radiotherapy as adjunct therapy is utilized only for inoperable or non-radically operable cases. The risk of malignant transformation has been anticipated, however the risk is considerably low after orthovoltage radiation has been replaced by megavoltage radiation.[8]

In conclusion, giant cell tumour of maxilla in head and neck is a uncommon condition and requires high index of clinical suspicion. Even though biopsy and radiology are helpful in detection of the lesion, a battery of other investigations is often required to differentiate it from other condition mimicking GCT. The importance of distinguishing it from other conditions is due to its tendency to recur and metastatic potential. Complete wide excision is considered to be curative with low recurrence rate, however long term follow-up is suggested in literature.

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