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Case Report

Unusual sinonasal mass clinically mimicking malignancy

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ABSTRACT

Central giant cell lesion is a non-neoplastic proliferation, usually asymptomatic, of unknown aetiology. This case report describes the diagnosis and treatment for a maxillary central giant cell lesion. We report a case of a 30-year-old lady with a central giant cell granuloma (CGCG) who presented with a history of right-side nasal blockage, swelling and pain in the right side of the cheek, proptosis of the right eye, occasional blurred vision for one month. Images and histopathology examinations confirmed the diagnosis. The patient underwent tumour resection through the endoscopic endonasal approach successfully without any postoperative complication, there was no recurrence within a year of follow up and its histopathological analysis was consistent with a CGCG. Histopathological examination of the mass confirmed as CGCG. Complete excision of right sinonasal mass achieved by right endoscopic sinus surgery through modified Denker's approach. As CGCG is a non-neoplastic lesion of unknown aetiology, histopathology is necessary for definitive diagnosis. Treatment can vary depending on the extension of the tumour.

Keywords: Central giant cell granuloma, Multinucleated giant cells, Surgical excision, Sinonasal mass

INTRODUCTION

Jaffe first described it in 1953 in the jaws, although other craniofacial sites have been reported. Jaffee originally coined the term giant cell reparative granuloma to describe a lesion he believed represented a response to intraosseous haemorrhage from jaw trauma.[1] The maxilla and mandible are most commonly affected followed by the sphenoid and temporal bones.[2] World health organization defines CGCG as an intraosseous lesion consisting of cellular fibrous tissue containing multiple haemorrhage foci, aggregations of multinucleated giant cells and occasionally trabeculae of woven bone.[3] These lesions commonly occur in children and young adults, with a female-to-male preponderance of 2:1, 75% of GCG patients are younger than 30 years. Though defined histologically as benign lesions giant cell granuloma has the capacity for local destruction. Thus, a treatment modality that would arrest the growth is mandatory.

Here with we describe a case of CGCG affecting the maxilla and nasal cavity when a 30 year old female patient presented to us with right nasal blockage, swelling over the right cheek and proptosis.

CASE REPORT

A 30-year-old woman presented to our ENT OPD with complaints of right-side nasal blockage, swelling and pain in the right side of the cheek, proptosis of the right eye, and occasional blurred vision for one month. History revealed that the swelling was insidious in onset and gradually progressed to the present size. There was no history of loosening of teeth, difficulty in chewing, fever, or preceding trauma. Past history, medical and family history was non-contributory.

Preoperative visual acuity checked patient's vision was not affected.

External examination: solitary swelling on the right side of the face causing facial asymmetry. A swelling of size 4×3×2 cm located on the right side of the cheek was soft to firm in consistency, tender, and non-fluctuant, with no discharge, or overlying skin change. There was proptosis of the right eye seen (Figure 1). There was no associated lymphadenopathy.

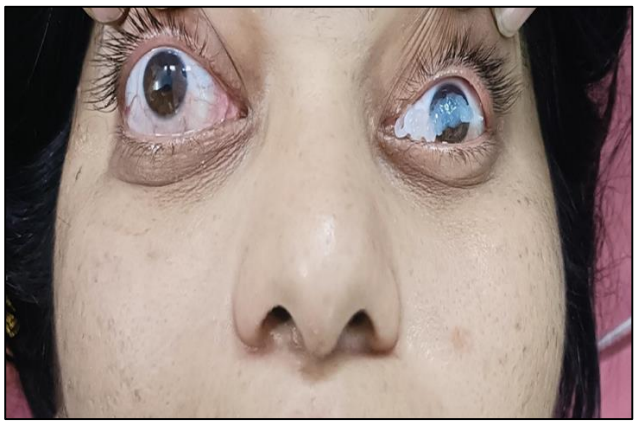

Figure 1: Upon initial examination, the patient had a facial asymmetry with right eye proptosis.

On diagnostic nasal rigid endoscopy-a polypoidal mass with the discharge was present in the right nasal cavity with a marked deviated nasal septum to left.

Intraoral examination revealed fair oral hygiene and a full complement of teeth with no extension of swelling.

She underwent preoperative laboratory tests and a punch biopsy of the right nasal mass was taken under local anaesthesia. The collected material was sent for pathological examination. The biopsy report revealed morphology consistent with a giant cell containing lesion? Aneurysmal bone cyst? CGCG. Brown tumours are identical to GCG both histologically and radiographically, but they were ruled out on the basis of normal serum levels of calcium, phosphorus, alkaline phosphatase and good renal function.

The patient underwent computed tomography (CT) paranasal sinus plain with contrast (Figure 2) that revealed a large lobulated heterogenous enhancing solid cystic mass lesion involving the right maxillary sinus and adjacent right nasal cavity. It shows destruction expansion and dehiscence of the right maxillary sinus walls, medial inferior wall of the right orbit, ethmoid air cells, osteomeatal structures. Focal bulging of disease in retro maxillary space. Mass effect and compression of the right pterygomaxillary fissure. Superior extension into right orbit with mild mass effect over adjacent extraocular muscles in right eye globe. Mass destroys the medial wall of the right maxillary sinus and shows extension into the right nasal cavity with a leftward deviation of the nasal septum. The lesion bulges posteriorly up to the posterior choana and anteriorly up to the anterior choana. Destruction in attenuation of right maxilla with mass reaching till tooth sockets of a right maxillary molar tooth.

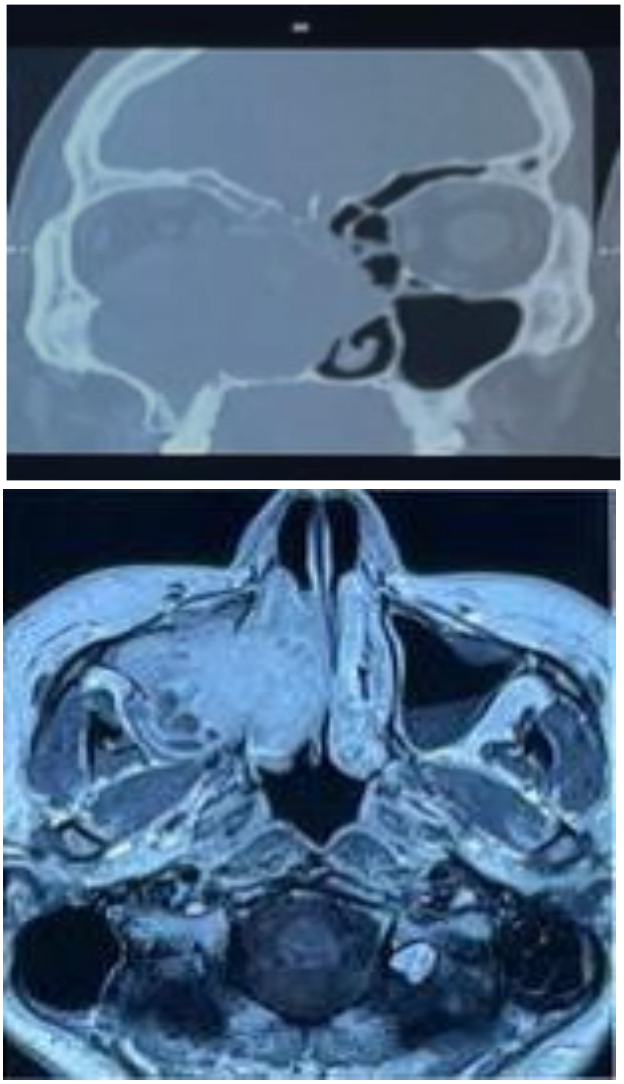

Figure 2: Computed tomography in the coronal section showing generalized opacity indicating destruction of the medial wall of the maxillary sinus.

Post obstructive right frontal, ethmoid, sphenoid polypoid sinusitis. No definite intracranial extension was seen. Left maxillary air-fluid level with minimal mucosal thickening. Left ethmoidal minimal mucosal thickening.

The patient was also subjected to MRI brain with orbit which revealed an aggressive neoplastic lesion arising from the right maxillary sinus extending into the right half of the nasal cavity.

The patient was informed about the procedure and a signed informed consent form was obtained.

The case was planned for surgery under general anaesthesia. Complete excision of right sinonasal mass achieved by right endoscopic sinus surgery through

modified Denker's approach under general anaesthesia (Figure 4). A thin and eroded part of lamina papyracea was removed for mass clearance. Mass was seen eroding the posterior wall of the maxilla, pterygopalatine fossa, and medial orbital wall. Mass was dissected all around using a microdebrider and with bipolar nasal cautery to reach its neck at the posterior maxillary wall. Drilling was done at bony attachments to prevent a recurrence. Once the bony defect was delineated, the surrounding mucosa was made raw. Nasal packing was done with merocele.

The excised biopsy was subjected to a histopathological examination.

Histopathological examination (Figure 3) revealed bony tissue with a tumour composed of spindle-shaped fibroblastic cells arranged in fascicles and whorls admixed with osteoclastic giant cells. Vascular spaces lined by giant cells are noted. Reactive osteoid is noted.

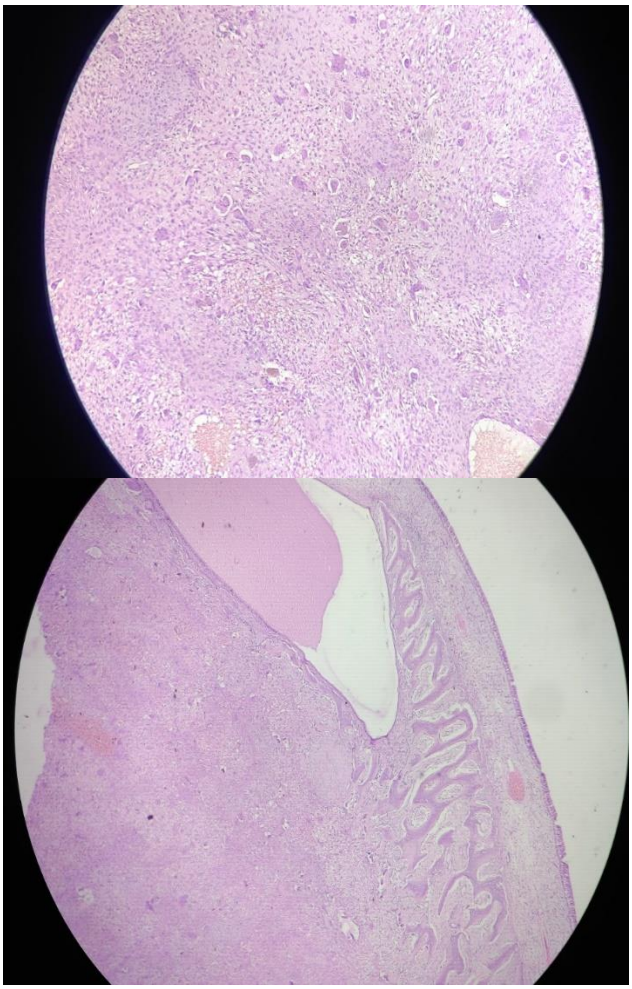

Figure 3: Histopathologic view of CGCG showing multinucleated giant cells.

A diagnosis of CGCG was made.

The nasal pack was removed after 48 hrs. On regular one-month follow-up, there was healthy nasal mucosa, with well-opened sinuses seen. No visual disturbance was seen.

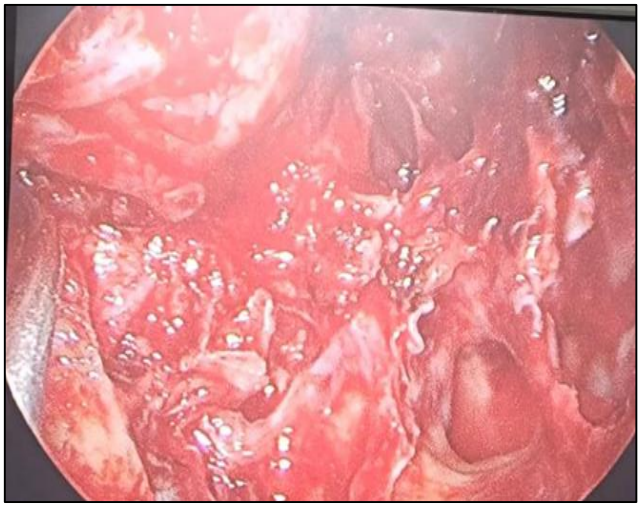

Figure 4: Intraoperative picture of right nasal cavity.

Histopathological examination of the mass confirmed as CGCG. Complete excision of right sinonasal mass achieved by right endoscopic sinus surgery through modified Denker's approach.

DISCUSSION

CGCG is a non-neoplastic proliferation of unknown aetiology. It occurs more commonly in the mandible than in Maxilla. The present case, however, involved the maxilla. Most mandibular lesions occur anterior to the first molars and often cross the midline. CGCG also occurs in other bones of the facial skeleton and cranial vault. It rarely occurs outside the craniofacial bones, but it has been described in the short tubular bones of hands and feet.[4] Giant cell granulomas of the jaw bones may be peripheral or central. Peripheral lesions present as pedunculated or sessile lesions on the gingiva while central lesions are endosteal. The peripheral type was four times more common than the central type. It strikingly occurs more commonly on the right than the left side.[5] Trauma has been considered an important etiologic factor in the initiation of this lesion. The lesions increase by an accumulation of tissue which is produced by slow, minute, continuous haemorrhages of multicentric nature due to trauma and some defect in the capillaries.[6] Non-specific pain and swelling are the most common clinical manifestations.[7]

GCG is divided into two categories according to its clinical behaviour: aggressive and non-aggressive. The non-aggressive form is more commonly seen with a characteristic slow-growth pattern and painless swelling. The aggressive form is characterized by one or more of the following features; pain, paraesthesia, root resorption,

rapid expansion, cortical resorption and high recurrence rates after surgical curettage. The aggressive form is mostly found in younger patients and cortical perforation has a high recurrence rate. There is no histological difference between aggressive and nonaggressive types. The size and number of giant cells may influence the clinical behaviour of the lesions.[8,9]

The radiological appearance of CGCG is variable. Usually, the lesion appears as a unilocular or multilocular radiolucency. It may be well-defined or ill-defined and shows variable expansion and destruction of the cortical plate. CGCG of the jaw usually presents as a painless solitary radiolucent expansion in most cases. Some lesions are more destructive with a marked tendency to recur. The radiological appearance of the lesion is not pathognomonic and may be confused with that of many other lesions of jaws. CT scan is excellent for demonstration of bone thinning or destruction. MRI is the best modality for evaluating the extent of the lesion. It has low to intermediate-intensity signals on both T1 W and T2 W images similar to GCT. Mild post-enhancement is evident both on CT and MRI. The final diagnosis eventually rests on histopathology because the clinical and radiological features are not specific.

On histological examination, CGCL is represented by multinucleated giant cells in a prominent fibrous stroma. Osteoclasts have irregular distribution and are associated with areas of haemorrhage. Structurally the proliferative cells include spindle-shaped fibroblasts, myofibroblasts, and mononuclear inflammatory cells.[10] Foci of haemorrhage with the release of hemosiderin pigment are often seen. Immunohistochemical studies in cases of CGCL have helped establish the lineage and pattern of these cells; however, they cannot predict the aggressiveness of the lesion. The differential diagnosis includes an aneurysmal bone cyst, giant cell tumour and brown tumour of hyperparathyroidism.[10]

Since the clinical and radiological signs of CGCG are not specific, histology is ultimately used to make the final diagnosis. Excision through surgery is the most effective treatment and produced superior outcomes. Depending on the type of lesion, tissue removal might range from a straightforward curettage to a thorough resection.

The management of CGCG will depend on the clinical and radiographic findings. Generally, curettage of well-defined localized lesions is associated with a low rate of recurrence. In extensive lesions, based on imaging tests, where there has been cortical drilling, more radical excision is mandatory. In such cases, even partial maxillectomy has to be done. Cryosurgery and peripheral osteotomy are the other options.[11] Five mm surgical margins that extend to healthy tissues are recommended to avoid recurrences.

The medical management of CGCG as an adjunct to surgery includes treatment with steroids or calcitonin which inhibits osteoclastic activity.[12] Interferon-alpha appears useful in the management of aggressive CGCG, presumably due to its anti-angiogenic effects.[13] Bisphosphonates have been administered intravenously in CGCG with promising results.[14] The clinical behaviour of this lesion is quite variable and difficult to predict. Hence, we suggest that CGCG should also be considered in the differential diagnosis of the swellings in the maxillary posterior area even though it has a marked propensity to occur in the mandibular posterior area.

The incidence of recurrence of CGCG after surgery is 13-22% with most treatment failures manifesting within the first two years of the therapy.[15] Rapid identification and treatment can significantly reduce morbidity and enhance long-term results. To stop a recurrence, regular radiographic surveillance and clinical assessments should be made.

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