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Case Report: Central Giant Cell Granuloma –A Clinical Experience

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ABSTRACT- Central giant cell granuloma (CGCG), formerly called giant cell reparative granuloma, is a non-neoplastic proliferative lesion of an unknown aetiology. It occurs most commonly in the mandible. The case reported here resembled a wide variety of conditions that led to a misdiagnosis on both clinical and radiographic examinations but it was histopathology gave diagnosed as CGCG of left maxilla. We managed this case by wide surgical resection via extra oral approach using the weber-Fergusson incision.
KEY WORDS: CGCG, Maxilla, Weber-Fergusson.

I. INTRODUCTION:

An intraosseous lesion called a central giant cell granuloma (CGCG) is made up of cellular fibrous tissue, aggregations of multinucleated giant cells, and occasionally trabeculae of woven bone[1]. Jaffe first identified CGCG as an idiopathic non-neoplastic proliferative lesion in 1953 [2] .When CGCG was first discovered, it was thought to be predominantly a local reparative reaction of bone, presumably in response to intramedullary bleeding or trauma. At one point, the name reparative giant cell granuloma was generally recognised. Now the lesion actually symbolises a destructive process, the word "reparative" has since been abandoned[3]. Despite the lack of knowledge on its origin and pathophysiology, the histology and clinical behaviour have been well investigated [4-7].

The World Health Organization recently characterised it as localised benign but occasionally aggressive osteocytic proliferation made up of fibrous tissue with bleeding and hemosiderin deposits, the presence of large cells that resemble osteoclasts, and reactive bone formation[7]. Mandible and maxilla are the two areas that are most frequently affected. It can be damaging locally even if it is innocuous. The most often used treatment for the illness is surgery.Our case who is 20 years old young female presented with a giant cell lesion that involved the left maxilla.

CASE PRESENTATION: A 20 years old female patient reported to our department of oral and maxillofacial surgery with the complaint of

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swelling in left upper jaw since ten months.

Complete case history revealed that the swelling started as small size and progressively increased to the present size over a period of ten months. In the last ten months patient had sought treatment from his family dentist who extracted her 27 and but the swelling persisted even after extraction. The nature of medication was unknown. There was no history of trauma, neurological deficit, Fever, Loss of appetite, Loss of weight, Nasal discharge or Difficulty in swallowing. Patient was systemically healthy. Medical and family histories also not reveal any abnormalities.

On examination, there was an approximately 4x4 cm^2 diffuse swelling over the left side of face causing slight obliteration of Nasolabial fold resulting in facial asymmetry. Swelling extends superiorly 1cm below the infraorbital margin. Inferiorly at the level of cheek hallow region. Medially at the nasolabial fold.Laterally 3cm infront of the ear region.The overlying skin was normal and temperature was normal. There was no associated lymphadenopathy. The swelling was firm in consistency and was slight tender on palpation. Paraesthesia present in relation to infraorbital region. Intraoral examination shows a purple expansible mass in the region of upper left 22,23,24,25,26 areas.The swelling was in labial to buccal aspect extending from 22 to 26 area obliterating the buccal vestibule. The swelling had smooth surface, firm and slightly tender on palpation. There was also slight expansion of palate to the midline of palate Medio-laterally and Antero-posteriorly from 22 till 26 region.

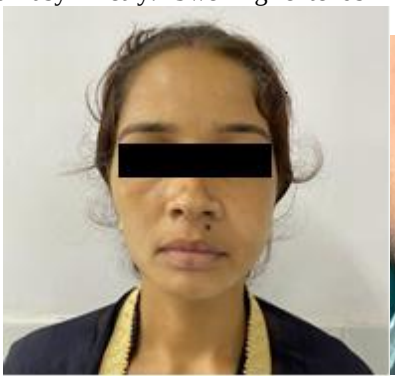

Fig.1.Swelling over the left side offace

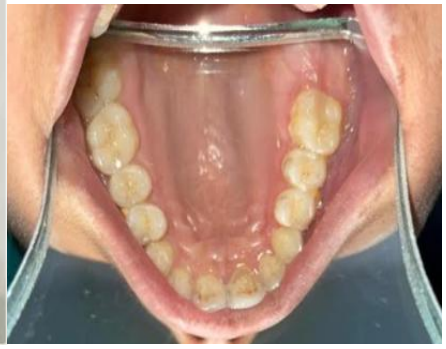

Fig.2. Palatal swelling on left side

INVESTIGATIONS:

Routine haematological were normal. Aspiration was negative which confirm solid tumour. The serum chemistry of Glucose, Urea nitrogen, Creatinine was normal.

Contrast- enhanced computed tomography scan revealed heterogeneously enhancing soft tissue density lesion with CT value of 50-60 HU with multiple linear and branching hyperdense areas within it appears to be arising from left superior alveolar arch,at the root of left second molar tooth, extending superiorly and completely filling and expanding the left maxillary sinus and posterior ethmoid sinus,extending antero-medially into the left nasal fossa with blockade and marked widening of ipsilateral maxillary osteium with remodelling of adjacent bony structures in the form of marked thinning of walls left maxillary sinus, leading to pressure erosions. Posteriorly the lesion is obliterating left Pterygopalatine fossa. Superiorly it is abutting and upwardly displacing the posterior part of left orbit. Laterally it is abutting left infra temporal fossa.

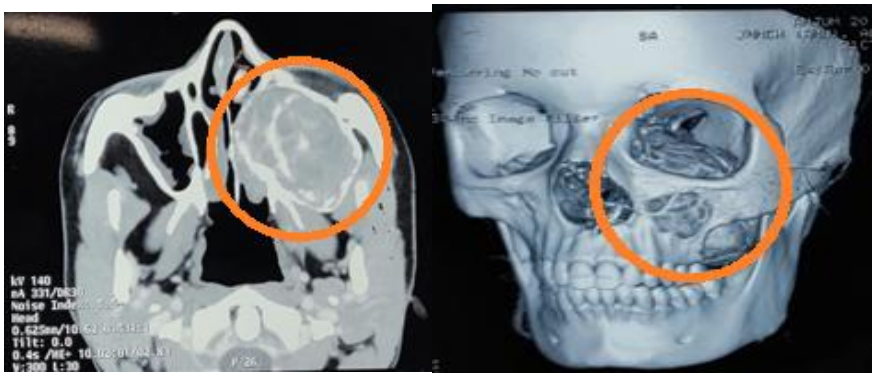

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Fig.3. Axial view shows mediolateral expansion

Fig.4.Three dimensional view shows aggressive character of tumour.

DIFFERENTIAL DIAGNOSIS:

On the basis of clinical and radiological examination a provisional differential diagnosis of Periapical cyst, Adenomatoidodontogenic tumour (AOT),calcifying epithelial odontogenic cyst (CEOC),Giant cell tumour, Ameloblastoma, Ossifying fibroma, Fibrous dysplasia and Central Giant Cell Granuloma was made.

PREOPERATIVE BIOPSY:

Biopsy was done under local anaesthesia and good amount of bleeding was encountered suggesting of high vascularity of lesion. Histopathological examination of the specimen revealed numerous osteoclastic giant cells distributed in loose vascular stroma composed of plump stromal cells and extensive red blood cell extravasation. Focal new bone formation present at the edge of lesion. It's suggestive of central giant cell granuloma.

WIDE SURGICAL EXCISION (WEBER-FERGUSON APPROACH):

The case was planned for surgery under general anaesthesia. Through the extra oral approach via weber-ferguson incision along the lower eyelid,lateral nasal and alar basal region along with philtrum of left side via the midline of upper lip. Intraorallycrevicular incision was given along 22_26 with vertical releasing incision mesial to 22. The lesion entirely freed from all the aspects and removed completely. Complete curettage was done. Bleeders were identified and cauterized using monopolar and bipolar. Finally irrigation was done with Betadine and normal saline. Excised lesion sent for confirmatory histopathology examination. One unit PRBC was administered during surgery. Closure done in layers using vicryl 4-0 and nylon 5-0 for extra orally and intraorallycrevicular incision closed with vicryl 3-0 suture. Mild nasal bleeding was present for 1-2 days which was clotted blood from the right maxillary sinus.

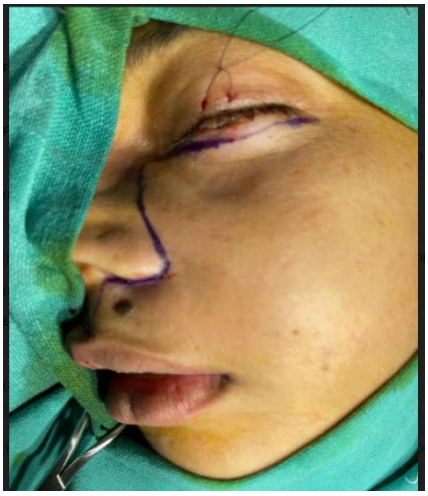

Fig.5.Weber-ferguson incision marking

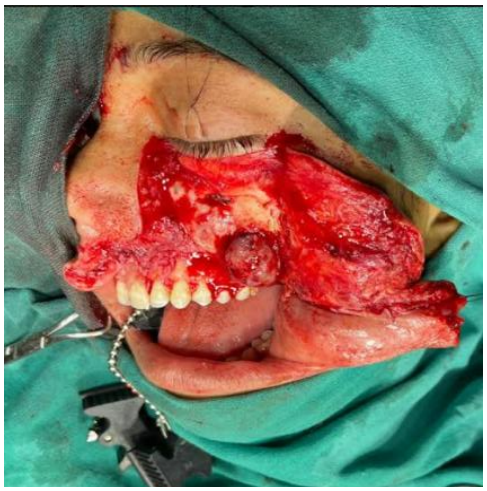

Fig.6.Exposure of the lesion

Fig.7. Resected the lesion Fig.8. closure of surgical site

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FOLLOWUP:

· The patient recovered well in the postoperative period. In the last three and six monthsfollow-up, there has not been any recurrence. There was no further expansion in operated site and patient facial profile also improved and become a normal. There is no complaint from the patient. No signs of recurrence found in 3, 6 months period of follow up.

Fig.9. follow up after 3 months.

CONFIRMATORY HISTOPATHOLOGY REPORT:

Postoperative histopathology report confirms dispersed numerous osteoclast like giant cells with foci of new bone formation. Stroma is predominantly cellular and fibrous with vascular proliferation and haemorrhage. Focal myxoid changes also present. It confirms central giant cell granuloma.

II. DISCUSSION:

Central giant cell granuloma is a non-neoplastic proliferative lesion of an unknown aetiology. It occurs most commonly in the mandible than in maxilla. Also, it is more common on the right than the left side with females having more predilection than males in the ratio 2:1.Chuong et al [8] in 1986 and Ficarraet al[9] in 1987 suggested categorizing CGCG into aggressive and nonaggressive types based on their clinical and radiographic characteristics. The more common, non-aggressive, lesions grew slowly and usually presented clinically as painless swellings, with only 20% of patients complaining of pain or parasthesia.[10,11,12] This is totally in contrast with our case, wherein left maxilla was involved in a young female. It rarely may involve bones other than those of the craniofacial region. Though trauma has been considered as an important aetiological factor in the initiation of this lesion, but the history of trauma was absent in this case. The lesion expansion by accumulation of tissue which is produced by slow, minute, continuous haemorrhages of multi centric nature because of trauma and some defect in the capillaries. The giant cell granuloma is often confused with giant cell tumour. However, the giant cell tumour occurs in the age range 25–40 years, involves long bones and is more aggressive in nature with frequent recurrence after curettage. Microscopically, the giant cells are osteoclastic giant and almost uniformly distributed, whereas in giant cell granuloma, numerous osteoclastic giant cells distributed in loose vascular stroma composed of plump stromal cells and extensive red blood cell extravasation. Focal new bone formation seen at the edge of lesion. A diagnosis of CGCG is based on histopathology. This statement is further supported by our case which presented with clinical features leading to differential diagnoses of conditions such Periapical cyst,Adenomatoid odontogenic tumour (AOT),calcifying epithelial odontogenic cyst (CEOC),giant cell tumour,Ameloblastoma,Ossifying fibroma, Fibrous dysplasia and Central Giant Cell Granuloma. The most common presenting sign of CGCG is a painless swelling with noticeable facial asymmetry. The radiological appearance may be unilocular or multilocular radiolucency, with expansion and destruction of surrounding bone. The recurrence rate is reported to be 13–22% with mostly manifesting within first 2 years postoperatively. Generally, curettage of well-defined localised lesions is associated with a low rate of recurrence but in extensive lesions with evidence of perforation of cortex, more radical excision is mandatory, which may lead to loss of teeth.In our case we went wide surgical excision via Weber-Ferguson approach because the lesion was aggressive and prevent the recurrence. In recent years, medical treatment including intralesional corticosteroid triamcinolone injection to suppress the inflammatory reaction of the lesion, injection calcitonin antagonist of bone resorption by inhibiting giant cells action and alpha interferon injection helps to suppress the Angiogenic action of lesion. Medical therapy helps the further aggressive action of lesion and acts as adjunct to surgery.

III. CONCLUSION:

Evaluation of the surgical therapy in the population shows an overall recurrence rate of 26.3%, which lies within the range of recurrence rates reported in the literature. Surgical curettage is not an effective therapy for CGCGs in young people who show aggressive signs and symptoms. Wide surgical excision prevents and reduce the recurrence rate of lesion.

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