

Central giant cell granuloma (cgcg) case study



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ABSTRACT:

Central giant cell granuloma (CGCG) is an intraosseous lesion which occurs as an uncommon benign condition in jaws. WHO defines this intraosseous lesions as “ a lesion that contains multiple foci of haemorrhage, consisting of cellular fibrous tissue and there is trabeculae of woven bone. It may become aggressive leading to expansion and perforation of the cortex. Mobility and displacement of the involved teeth and root resorption are often observed. Here is a case report of an 18 year old female patient who is diagnosed with an aggressive type of CGCG.

KEYWORDS:

Central giant cell granuloma, granuloma, giant cell granuloma, giant cell lesions.

Introduction

Central giant cell granuloma (CGCG) is a benign proliferation of fibroblasts and multinucleated giants cells that almost exclusively occurs within the jaw. It commonly occurs in young adults showing a female predilection. ¹ CGCG rarely occurs in areas elsewhere other than the jaws, like maxillary sinus, temporal bone, cranial vault and other bones of the craniofacial complex. ² It was thought that CGCG is a reparative lesion as it develops in response to intrabony hemorrhage and inflammation secondary to trauma. However, it can be considered as an aggressive lesion because of its aggressive behaviour as seen in the present case.

Case report

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A 18 year old female patient, presented with a painful swelling over the lower border of mandible of 3 months duration. The patient had a history of trauma 6 months back with fractured 31. On extra oral examination, gross asymmetry of face was seen with a diffuse swelling of approximately 3×4 cm size on chin. Lymph nodes were not palpable. Local examination revealed a diffuse swelling extending mentolabial sulcus, inferiorly below the lower border of mandible and antero-posteriorly extending from the midline to the level of corner of mouth both side extending 1.5 cm size. Colour over the surface appeared normal; no ulceration or discharge from the swelling was seen. Surface of the swelling was smooth, consistency was hard. The swelling was non - fluctuant; No rise in temperature, no pulsations were felt. The swelling was tender on palpation. On intraoral examination, tenderness on palpation was evident in relation to left mandibular canine and first premolar. No lingual expansion.

Based on the history given by the patient and the clinical examination, a provisional diagnosis of traumatic bone cyst in relation to anterior lower border of mandible was given. However, radiographic examination was suggested to confirm the provisional diagnosis.

Orthopantomographs demonstrated normal anatomic hard tissue structures with a diffuse radiolucency seen in the mandibular anterior region crossing the midline, measuring approximately 3 cm x 3.5 cm, extending medio-laterally from 34 to 44 and supero-inferiorly from the apex of mandibular anterior extending to 1.5 cm below the level of inferior border of mandible suggestive of expansion of inferior border of mandible with sclerotic border on superior aspect and no sclerotic border inferiorly. Based on the clinical

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and the radiographic examination, differential diagnosis of odontogenic keratocyst, ameloblastoma, osteosarcoma and central giant cell granuloma were considered.

Histopathological evaluation of the excisional biopsy specimen showed the presence of connective stroma containing numerous young fibroblasts as well as multinucleated giant cells. Trabeculae of osteoid and woven bone were also seen in the periphery. Numerous extravasated RBCs were present within the connective tissue stroma. These findings are suggestive of CGCG, but in order to differentiate this from brown tumor of hyperparathyroidism, we carried out blood investigation, to find the serum calcium, serum phosphorus and alkaline phosphatase levels, which are found within normal limits. Based on the above histological and investigational findings, a diagnosis of CGCG was given.

Discussion

Central giant cell granuloma is a benign intraosseous lesion of the jaws. Jaffe in the year 1953, described this intraosseous lesion as “ central giant cell reparative granuloma”.¹ Since there is not reparative process, the name “ reparative giant cell granuloma” was denominated. The etiology and pathogenesis of CGCG is unknown, but the granulomatous process is induced by an exacerbated reparative process due to trauma and haemorrhage.³ Giant cell granuloma is considered as a benign proliferation of fibroblasts and multinucleated giant cells that occurs almost exclusively within the jaws. It is seen in all age groups ranging from 2 to 80 years, but more than 60% of the cases occurs under the age of 30 years.⁵ Although Sex distribution

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varies in different reviews, CGCG show female predilection with a prevalence of almost twice that of males.⁴ It commonly occurs in mandible, anterior to the first molar, often crossing the midline. It occurs in the short tubular bones of hands and feet and also in the other bones of facial skeleton and cranial vault but rarely occurs in craniofacial bones.⁶ It may be peripheral or central. The peripheral lesions occur as pedunculated or sessile lesions where the central lesion is endosteal. Females, children and young adults have more predilection since the female male ratio is 2: 1.^{7, 8} The main etiological factor for this lesion is trauma. The lesion progresses by accumulation of tissue which due to slow and continuous haemorrhage of multicentric nature as a result of trauma and defect in the capillaries.⁹

Though the CGCG is a benign lesion, it occurs as aggressive and non-aggressive types. The aggressive type shows painful and rapid growth occurs in younger patients and often involves cortical perforation and root resorption and may recur. The non-aggressive type is of slow growing, asymptomatic, without any resorption or perforation of the involved teeth and it never recurs.¹⁰

The signs of CGCG are a painless swelling, which causes facial asymmetry, where the radiological investigations reveals that there is unilocular or multilocular radiolucency, which is well or ill-defined with variable expansion along with destruction of cortical plate. Since the radiological appearance of this lesion is not pathognomonic, it is usually confused with the other lesions of the jaws. But the final diagnosis is based on its histopathology, though the clinical and radiological features are not specific.¹¹

Histopathological features reveal that it is comprised of dense proliferation of oval or spindle shaped cells with varying number of multinucleated giant cells containing 20 nuclei. There is a deposition of hemosiderin, extravasted RBC's, foci of osteoid material dystrophic calcification around the periphery of the lesion. ¹²

Though multinucleated giant cells are in more in number, they cannot be considered as proliferative cells, since the macrophages, mesenchymal cells and fibroblasts are accountable for the growth of the lesion. Hence these cells release cytokines that stimulate the proliferation and recruitment of blood monocytes to become osteoclast like cells. ¹³ The multinucleated giant cells may be large or small in number and they may be irregular or round cells that contains more than twenty nuclei which are responsible for bone resorption and local progression of lesion. ¹⁴ The giant cells containing more nuclei and dese cellular stroma are found to be more aggressive and may relapse after surgical treatment. ¹³

Some studies reveal a significant difference in the number of giant cells in aggressive and non-aggressive lesions where other studies reveal only few differences in the cell size in histomorphic analysis. Some of them found that the aggressive lesions show the higher number of giant cells with more irregular shape, where the giant cells are larger. There is a an increase in the mitotic activity along with a difference in histomorphic analysis which indicates increase in the fusion of resident macrophages and recruitment of monocytes and also there is higher metabolic activity of multinucleated giant cells that shows an aggressive clinical behavior. ¹⁵ According to the

differential diagnosis of the central giant cell granuloma , based on radiological investigation, being a small unilocular lesions it may be confused with granulomas and periapical cyst and the large multilocular lesions it may be ameloblastoma or lesions the resemble PGCL , aneurysmal bone cyst, central odontogenic fibroma, brown tumor of hyperthyroidism, giant cell tumor.

The CGCG and brown tumor of hyperparathyroidism resembles each other histologically, in having an intense endogenous brownish pigmentation of hemosiderin. The additional test that help in diagnosis are serum calcium, phosphate, parathyroid hormone and alkaline phosphatase levels which are normal in CGCG, but increased in brown tumor of hyperparathyroidism.

The CGCG usually occurs in both maxilla and mandible but the giant cell tumour more commonly occurs in the epiphyses of long bones. But both the lesions appear as osteolytic defects radiographically but can be differentiated histologically. Evidence reveals that the giant cells are larger, numerous and more round in giant cell tumor in CGCG, with a higher number of nuclei and eventually dispersed. There is fewer foci of osteoid material, areas of haemorrhage and there is deposition of hemosiderin and fibrosis and the stroma contains large and oval cells.

The aneurysmal bone cyst can be differentiated from CGCG in having a network of multiple cystic cavities fill with blood within thin walls.

Depending on the clinical and radiographic findings, if there is a well-defined lesion, curettage can be done where there is low recurrence, but if it is extensive lesion with perforated cortex, the radical excision is mandatory.

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Sometimes even partial maxillectomy or mandibulectomy and jaw reconstruction plates or placement of bone grafts can be done.

Conclusion

Based on the clinical, radiological, histopathological features, it is considered as an aggressive variant of CGCG, which is rare in occurrence. More clarification is needed regarding the pathogenesis and nature of giant cell lesions.