

Central Giant Cell Granuloma of the Mandible: A Case Report and Review of Literature

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Abstract

Background: Central giant cell granuloma is a benign intraosseous cellular lesion, containing abundant fibroblasts and multinucleated giant cells. It has a destructive course. Central giant cell granuloma commonly affects mandible followed by maxilla. Most cases occur in the first 3 decades of life. **Case presentation:** A 42-year-old woman presented to maxillofacial Department, Sohag University Hospital because of painless, slowly growing, cosmetically disfiguring mass lesion in the right side of the face. Radiological evaluation showed a multilocular osteolytic lesion occupying the right ramus of the mandible, no detected cortical bone destruction or tooth root resorption. The lesion was curetted and sent to Pathology Laboratory of Sohag University Hospital. Histopathological examination of the submitted specimen showed vascularized granulation tissue with prominent benign-featuring fibroblastic proliferation, abundant multinucleated giant cells and hemosiderin-laden macrophages. **Conclusion:** Central giant cell granuloma is a benign intraosseous cellular lesion with a destructive course; it usually affects jaw bones, especially the mandible. Central giant cell granuloma should be suspected in any giant cell-rich jaw lesions.

Keywords: Central giant cell granuloma- Cherubism- Reparative granuloma- Multinucleated giant cells- Mandible

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Introduction

Giant cell granuloma was first described by Jaffé in 1953; he described two distinct types of giant cell granulomas; central giant cell granuloma (CGCG) which has an intraosseous location and peripheral giant cell granuloma (PGCG) which develops in soft tissues as gingival soft tissues, mucoperiosteum and peri-odontal ligaments [1]. CGCG was previously called reparative giant cell granuloma as it was considered as a local reparative reaction of the bone to variable traumatic or inflammatory causes. However, the term reparative giant cell granuloma has been discontinued as the lesion is destructive [2].

World health organization (WHO) has defined CGCG as a benign intraosseous cellular, composed of vascularized granulation tissue rich in fibroblasts, multinucleated giant cells, multiple areas of hemorrhage and hemosiderin-laden macrophages [3].

CGCG occurs more common in mandible than in maxilla, about 70% of the reported cases of CGCG

are located in the mandible, anterior mandible is most frequently affected, other rare sites include sellar and temporal bones [2, 4]. CGCG is more frequently reported in women. Male to female ratio is 2:3. Most cases of CGCG occur in the first three decades of life [1].

The exact aetiology of CGCG is not fully understood; various traumatic, inflammatory or genetic factors are suspected [1, 3].

CGCG includes two distinct subtypes; aggressive and non-aggressive forms. Non-aggressive CGCG is the most common; it usually develops as a slowly growing painless nodule, expanding the cortical bone. On the other hand, CGCG is considered aggressive when it shows rapid progression, destructive course, tooth root resorption, teeth displacement and cortical perforation [1, 5].

Radiologically; CGCG appears typically as a multiloculated, osteolytic, radiolucent, expansile lesion, with wavy septations perpendicular to cortical bone. Large lesions may show peripheral scalloping and cortical

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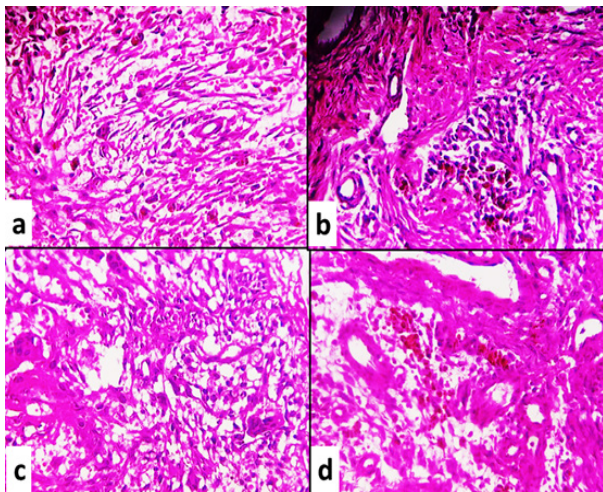


Figure 1. Photomicrographs of H&E-stained sections show granulation tissue contains abundant fibroblasts (a) hemosiderin-laden macrophages (b & d) and multinucleated giant cells (c), (X100).

destruction [6].

There are different treatment modalities for treatment of CGCG. However, there is no ideal treatment modality that can strictly prevent recurrence of CGCG [1].

Herein, we have reported right mandibular CGCG in a 42-year-old lady.

Case presentation

Patient information and clinical findings

A 42-year-old lady has been admitted to Maxillofacial Department, Sohag University Hospital, because of painless, slowly growing, cosmetically-disfiguring right facial mass lesion. Radiologically; there was a rather circumscribed, radiolucent, multilocular lesion, about 3.5x3cm, occupying right ramus of the mandible. There was no cortical perforation or teeth roots resorption.

The lesion was curetted and sent to Pathology Laboratory, Sohag University Hospital; it was fixed in 10% formalin fixative and labeled with patient's name and hospital serial number.

Pathological features

Grossly; the specimen was sent in the form of multiple, brownish firm fragments and few blood clots. Representative sections were obtained from the specimen and submitted for subsequent tissue processing and microscopic evaluation.

Microscopic examination showed vascularized granulation tissue with considerable fibroblastic proliferation with benign nuclear features. There are frequent multinucleated giant cells, extravasated red blood cells, hemosiderin-laden macrophages and occasional lymphocytes. The condition was diagnosed as central giant cell granuloma (Figure 1).

The patient was generally good, she was discharged home. No available data about serum calcium, phosphorous, parathormone hormone or alkaline phosphatase levels.

Discussion

CGCG is a rare, benign, intraosseous lesion. Its incidence is about 0.0001% of general population. Mandible is the most frequent site for development of CGCG, especially molar and premolar areas. Most cases occur during the first 3 decades of life [1-3]. CGCG includes aggressive and non-aggressive forms. Aggressive CGCG is characterized by high rate of recurrence, reaching 30-70% in some literatures [5, 6]. In rare cases, CGCG is associated with systemic conditions as neurofibromatosis type I and Noonan syndrome [6]. CGCG is a unicentric lesion; multifocal lesions may indicate underlying conditions as hyperparathyroidism or cherubism [1, 6]. There are various surgical and non-surgical treatment options for CGCG. Surgical approaches may cause cosmetic disfigurement and functional disturbances [1].

The first and most successful non-surgical treatment modality is intralesional injection of corticosteroids; corticosteroids block bone resorption and enhance osteoclasts apoptosis. It is advisable in small sized, solitary and unilocular lesions. Disadvantages of intralesional injection of corticosteroids include the long follow-up periods and systemic effects of corticosteroids [1, 7].

Another non-surgical treatment option for CGCG is subcutaneous injection of calcitonin. It is advisable in young patients with aggressive CGCG. The main disadvantages of subcutaneous injection of calcitonin are lowering serum calcium levels and increased risk for peptic ulcers [8].

Subcutaneous injection of alpha-interferon is used in aggressive CGCG. Alpha-interferon inhibits angiogenic growth factors. There are many side effects for alpha-interferon; they include drug-induced lupus erythematosus, fever, malaise and pancreatitis, all these side effects make alpha-interferon less preferable treatment option [7].

In the current case; we have reported a unicentric, right mandibular CGCG in a 42-year-old woman. Although most cases of CGCG occur during the first 3 decades of life, Bocchialini et al, [9] and Kudva et al, [10] have reported two distinct cases of CGCG in a 60-year old and a 45-year-old women, respectively.

Histological examination of the current case showed giant cell-rich fibroblastic lesion. Giant cell-rich jaw lesions include giant cell tumor, aneurysmal bone cyst, cherubism and osteitis fibrosa cystica in hyperparathyroidism. Presence of abundant bland-appearing stromal fibroblasts and absence of neoplastic stromal cells exclude giant cell tumor. Absence of endothelial-lined, wide vascular spaces excludes possibility of aneurysmal bone cyst [1].

Cherubism is a familial disease, it occurs in early childhood. Cherubism is characterized by bilateral, symmetrical enlargement of jaw bones. Spontaneous regression of cherubism usually occurs after puberty. Bilateral involvement, early onset and positive family history are the main hallmarks that distinguish cherubism from other giant cell-rich bone lesions [4].

osteitis fibrosa cystica occurs in primary hyperparathyroidism. Hyperparathyroidism enhances osteoclastic activity within the bone, creating small

tunnels that are replaced by fibrovascular tissues, these tunnels cause microfractures, secondary hemorrhage, recruitment of macrophages and fibrous tissues creating a mass called brown tumor. Cystic degeneration may occur [11]. CGCG may mimic osteitis fibrosa cystica. However, the latter is suspected when the lesion is multicentric or recurs after adequate management. Serum calcium, alkaline phosphatase and parathormone hormone levels are helpful to differentiate CGCG from osteitis fibrosa cystica definitely [6].

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Authors' contribution

Conception or design; Maisa hashem mohammed.

Acquisition, analysis or interpretation of data; Maisa hashem mohammed.

Drafting the manuscript; Maisa hashem mohammed.

Approval of the manuscript versio to be published; Maisa hashem mohammed.

Agreement to be accountable for all aspects of the work; Maisa hashem mohammed.

Conflict of interest

The author declares that she has no conflict of interest to disclose.

Data availability

Included in the manuscript.

Ethical considerations

This case report was approved by the Ethics Committee of Sohag Faculty of Medicine, Sohag, Egypt. Verbal consent was obtained from the patient as she was discharged from the hospital one day after curettage of the mandibular lesion.

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Study registration

Not applicable.

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