Central giant cell granuloma: a case report with diagnostic dilemma

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ABSTRACT

Central Giant Cell Granuloma (CGCG) was differentiated from Giant Cell Tumor of bone by Jaffe in 1953. Various authors have advocated this intraosseous lesion as a reactive lesion or developmental anomaly or a benign neoplasm. Actual etiology is not comprehensible till date and it has also been hypothesized to have a genetic etiology. Lesions are most commonly found in mandible but frequently crossing the midline. However, variable clinical features and radiological changes have been demonstrated by giant cell lesions of the jaws. In this case report, a young female patient reported with the chief complaint of swelling in right mandibular posterior tooth region since 2 wks after dental extraction from the same region. The patient underwent the treatment as surgical curettage followed by removal of the lesion and subsequent histopathological examination which confirmed the diagnosis of CGCG. There was no recurrence reported in 10 months of follow up.

Key words: Central giant cell granuloma, giant cells, granuloma, reparative granuloma

Introduction

Central giant cell granuloma (CGCG), also termed as reparative giant cell granuloma, is an uncommon benign intraosseous lesion affecting the maxilla and mandible, which consists of massive fibrohistiocytic proliferation and heavily hemosiderin laden multinucleated giant cells [1,2]. World Health Organization has defined it as "an intraosseous lesion consisting of cellular fibrous tissue with multiple hemorrhagic foci, aggregations of multinucleated giant cells, and occasionally trabeculae of woven bone" [3,4].

CGCG contributes for <7% of all benign lesions of the jaws [5]. Clinically, it is seen most commonly in patients younger than 30-year-old with a female predilection [6]. Most commonly, lesions are located in the anterior mandible in incisor, canine, and premolar regions, frequently crossing the midline. Furthermore, CGCG reveals erratic clinical features which range from slow growing asymptomatic swelling to the aggressive lesion with pain, osseous destruction, cortical plates perforation, root resorption, and recurrence. Facial asymmetry is the most common sign with swelling, which is usually painless [7].

Etiopathogenesis is still not clear, but some authors consider it as a reparative response rather than neoplastic while others consider it to have a neoplastic potential [5,6,8]. This

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presentation is to report a case of CGCG with an unusual presentation leading to the diagnostic dilemma.

Case Report

A young female patient of 25 years reported with the chief complaint of pain as well as swelling in the right lower back tooth region, since 2 weeks after she underwent extraction of the tooth from the same region. There was a small swelling initially and increased to the present size gradually. Mild, spontaneous, and intermittent pain was noted, thereafter relieved on taking medications. Past dental history was the extraction of the tooth from the same region 15 days back, as the tooth was grossly decayed with no pain and extraction of left mandibular first molar 1-year back. There was no relevant medical history of any systemic disease or any drug allergy. On extraoral examination, facial asymmetry was seen with a diffuse swelling on the right lower jaw which was extending to the canine region [Figure 1]. Skin overlying the swelling was normal. On palpation, swelling appears to be nontender and bony hard in consistency. On intraoral examination, the swelling was bony hard and present on the buccal aspect of right mandibular first molar, measuring approximately 1.5 cm × 1 cm, extending from the distal aspect of the second premolar till the mesial aspect of the second molar with normally appearing overlying mucosa [Figure 2]. The margins were well-defined with a smooth surface and there was missing permanent first molar with obliterated buccal sulcus in the involved region.

On radiological examination, orthopantomogram exhibits a well-defined radiolucency with radiopaque border extending from the distal aspect of the right second premolar till the mesial aspect of the right second molar. Right, first molar space, seen as edentulous areas, indicated missing tooth with root resorption in the right second premolar. Left mandibular

first molar is also perceived as missing edentulous area in the radiograph [Figure 3]. Differential diagnosis of the residual cyst, keratocystic odontogenic tumor, CGCG, and ameloblastoma were given on the basis of clinical and radiographic features. All the blood investigations were done for the patients and were found to be under the normal range. Incisional biopsy was done under local anesthesia and tissue was sent for histopathological examination. H and E stained section revealed loose fibrous connective tissue stroma with plump fibroblasts, multinucleated giant cells of varying size containing nuclei up to 20 in number along with scattered hemosiderin pigments and extravasated red blood cells (RBCs) [Figure 4]. The final diagnosis of CGCG was made and the complete surgical excision of the lesion was done.

Discussion

The term CGCG was first introduced to differentiate between giant cell tumor and CGCG on the basis of clinical and histopathological features. It occurs frequently in the mandible



Figure 1 Clinical picture showing facial asymmetry with diffuse swelling on the right side of lower jaw



Figure 3 Panoramic radiograph showing well-defined radiolucency in the right mandibular first molar region

than in maxilla, generally anterior to the first molar and often crosses the midline in contrast to our findings where the lesion was present in the right mandibular first molar region. According to Bender's osteogenesis, exfoliation and eruption of teeth are the steps in actively developing craniofacial skeleton and these processes cease, as the individual reaches to adulthood, therefore, leading to CGCG in young people mostly occurring in first three decades of life with females being more commonly affected than males [9], due to increased levels of estrogen hormone secretion [10,11] in a ratio of 2:1, which is so in our case.

Clinically, majority of the cases of CGCG are asymptomatic, osteolytic lesions and may be discovered accidentally on routine radiological examination. Some lesions are aggressive ones and may present with pain, swelling, root resorption, cortical perforation, and/or recurrence [12]. In a study done by Kruse-Lösler et. al., out of 26 patients, 16 were asymptomatic [13]. In the present case, the lesion showed nonaggressive behavior with no recurrence after 10 months of follow-up.



Figure 2 Photograph revealing missing 46 along with intraoral swelling in the same region

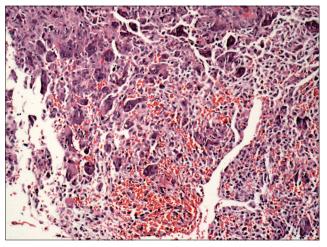


Figure 4 Pictomicrograph (×10) showing multinucleated giant cells with plump fibroblasts and scattered hemosiderin pigments

CASE REPORT

Several studies were done for the radiological evaluation of CGCG and shown unilocular radiolucency in early and small lesions while long standing lesions develop multilocular radiolucency [3,14]. In the present case report, the lesion showed unilocularity with 2 cm in diameter.

Histologically, World Health Organization has defined giant cell granuloma as "a localized benign, but sometimes aggressive osteolytic proliferation consisting of fibrous tissue with hemorrhage and hemosiderin deposits, presence of osteoclast like giant cells, and reactive bone formation." On histological examination, CGCG exhibits highly vascular and cellular granulation tissue with multinucleated giant cells scattered in the connective tissue stroma. Extravasated RBCs with hemosiderin and bone formation may be seen occasionally [15]. In the present case, plump fibroblasts were seen in the connective tissue stroma with multinucleated giant cells of varying size and containing nuclei up to 20 in number. Scattered hemosiderin pigments and extravasated RBCs were also seen.

CGCG are a mysterious lesion of unknown etiology. Many authors consider that the origin of CGCG is due to a proliferative response to aggression [5]. Some authors consider it as a benign hyperplastic reactive lesion which is caused due to many factors such as complicated dental extractions, chronic trauma, irritation, restorations in poor conditions, food impaction due to teeth malpositioning, and plaque [16,17]. Our case adds to the findings of other authors, that is, a history of dental extraction makes the case interesting. So, it can be considered as the etiological factor for the occurrence of the lesion.

Despite the benign nature of the disease, there are few cases which showed metastasis and documented in the literature [18]. Malignant transformations of CGCG to fibrosarcoma and osteosarcoma have also been reported in the literature [19]. Most accepted form of treatment for CGCG is a conservative surgical treatment involving curettage alone or with peripheral osteotomy. For the aggressive type of CGCG, radical surgical techniques of resection without continuity defect and peripheral osteotomy, or en bloc resection are also advised. There is incidence of recurrence after surgery in approximately 4–20% of cases whereas aggressive lesions show higher recurrence rate due to incomplete removal of the lesion. In our case, recurrence was not reported on 10 months follow-up.

The nonsurgical techniques for CGCG treatment include chemical cautery, electrocautery, and cryotherapy. Newer therapies in this field are calcitonin, interferon alpha, and intralesional steroids, which can be used to avoid disfigurement [20].

Conclusion

CGCG is an uncommon benign intraosseous lesion with varying etiological factors. So, to conclude, unusual sites of

presentation of the lesion can also be seen due to a proliferative response such as chronic trauma or dental extractions.

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Authors' Contributions

All authors contributed to literature review, drafting the article, sequence alignment. GS helped revise the manuscript. All authors read and approved the final version of the manuscript.

CASE REPORT

Consent

The authors certify that a written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the editor-in-chief of this journal.

Competing Interests

The authors declare that they have no competing interests.

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