Case Report

JRAD Journal of Research and Advancement in Dentistry

J Res Adv Dent 2019;9:3:241-244.

Central Giant Cell Granuloma (CGCG): A Case Report

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ABSTRACT

Background: Central giant cell granuloma (CGCG) is a non-neoplastic lesion which exhibits a spectrum of clinical behavior ranging from non-aggressive to aggressive variants. The etiopathogenesis of CGCG is still not properly known. In the maxillofacial region, CGCG most commonly occurs in the jaw bones as an asymptomatic swelling. Radiographically, it presents as either unilocular or multilocular radiolucent lesions in the maxilla or mandible. Treatment of CGCG varies from local curettage to wide surgical excision depending upon the extent and progression of the lesion. This paper presents a case of CGCG having different clinical and radiographic presentation.

Keywords: Giant cell granuloma, immunohistochemical staining, mandible, multinucleated giant cells, non-neoplastic lesion.

INTRODUCTION

Central giant cell granuloma (CGCG) is a nonneoplastic proliferation of fibroblasts and multinucleated giant cells (MCGs). Young adults are commonly affected by CGCG. Females are more commonly affected than males.[1] CGCG accounts for less than 7% of all benign lesions which occur in the jaws.[2] In the orofacial region, mandible is affected more than maxilla in the ratio of 2:1-3:1.[3] Central giant cell granuloma is a reparative lesion as it develops in response to trauma and secondary to hemorrhage.

CASE REPORT

A 28 year old male patient reported to the Department of Oral Medicine and Radiology with a chief complaint of swelling in lower anterior region since 3 months. History of present illness revealed that patient was asymptomatic 3 months back when he noticed swelling in mandibular anterior region. Initially swelling was smaller in size but gradually increased to present size. On intra oral examination, a diffused swelling was present from 34 region to 44 region, roughly oval in shape, roughly 4 x 5 cm in diameter, colour of swelling was of normal mucosal colour. overlying surface appears to be smooth. Surrounding mucosa appears to be normal. On palpation, it was non-tender, firm in consistency. 83 was present [Figure 1]. No pain was associated with swelling. No history of trauma and pus discharge. In investigations, orthopantomogram, blood investigations and excisional biopsy were done. Blood investigation's findings were within normal limits. The OPG revealed an ill-defined radiolucency roughly oval in shape and 1 × 3 cm in diameter, extending from distal aspect of 33 to distal aspect of 47. The internal structure appeared completely radiolucent. Also, 41, 42, 83 were displaced from their normal position. 43 was unerupted and was present al the lower border of mandible i.e impacted. Sclerotic borders were present [Figure 2]. Based on the history and radiographic examination a provisional diagnosis of central giant

Received: Mar. 19, 2019**: Accepted**: May. 2, 2019
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pISSN 2278-0076
eISSN 2321-9270

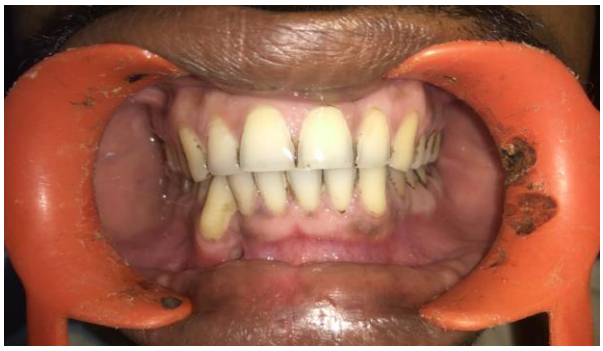

Fig 1: Clinical presentation of oral cavity.

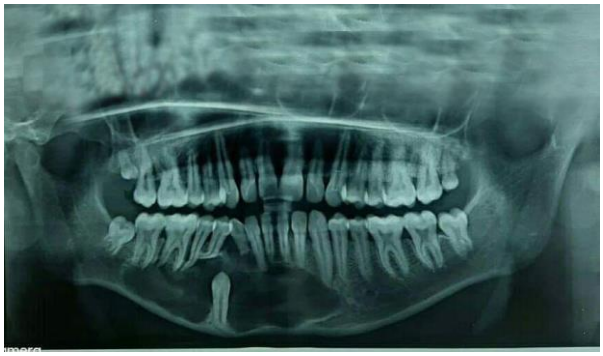

Fig 2: OPG Showing the lesion.

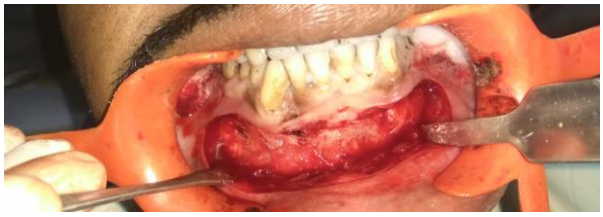

Fig 3: Full thickness mucoperiosteal flap has been raised.

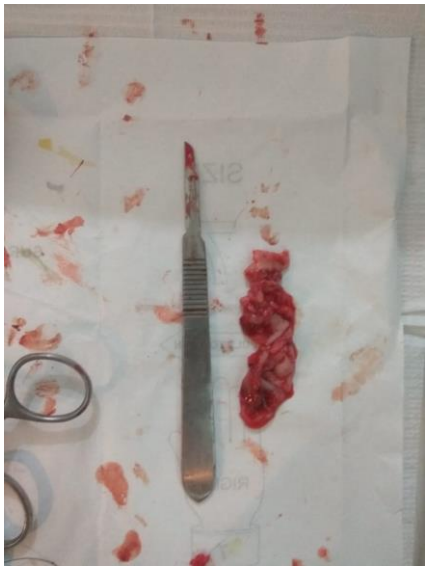

Fig 4: Excised tissue.

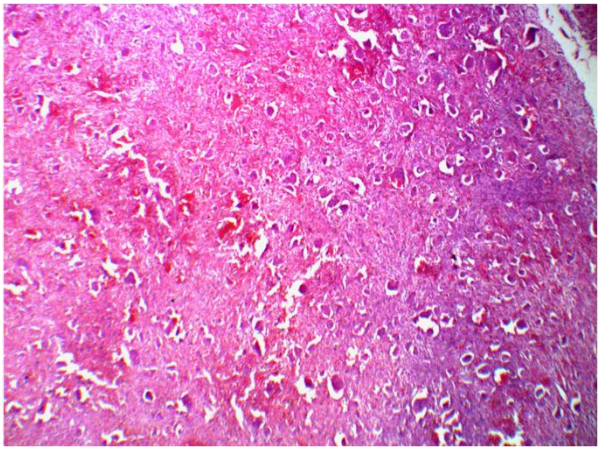

Fig 5: Histopathologic section of excised tissue.

cell granuloma was given. This case was done under local anaesthesia. After asepsis bilateral inferior alveolar nerve block, lingual nerve block and long buccal nerve block was given. Lot of infiltration given in the anterior region. vestibular degloving incision given extending from 46 region to 36 region. Full thickness mucoperiosteal flap has been raised [Figure 3]. Buccal corticotomy done with the help of straight fissure bur to expose the cystic lesion. Enucleation of cystic lesion done with the help of curettes. Impacted tooth was removed. Betadine impregnated guaze piece placed in the cystic cavity and suturing done. Patient recalled after 5 days for change of dressing and instructions given for frequent visits, so that change of dressing can be done which will promote secondary healing. The excised specimen was sent for histopathological examination [Figure 4]. Histopathologic report revealed giant cells and highly cellular stroma. Abundant giant cells with multiple nuclei were visible. Endothelial cells lining the blood vessels were seen [Figure 5]. All these features were suggestive of CGCG. So the final diagnosis of a CGCG involving the mandibular anterior region was given.

DISCUSSION

Central giant cell granuloma is defined by the World Health Organization as an intraosseous lesion consisting of cellular fibrous connective tissue that contains multiple foci of hemorrhage, aggregates of MCGs, and occasionally trabeculae of woven bone.[4] Jaffe, in the year 1953, described CGCG for the fi rst time as “Central Giant Cell Reparative Granuloma.”[5] In mandible, the anterior portion is more commonly affected, with the lesion frequently

crossing midline. Rarely, the lesions involve the posterior jaws, including the ramus and condyle, but gross soft tissue involvement is rare as it often remains limited to its effects on periosteum. Usually, as seen in both the cases, CGCG lesions are asymptomatic and do not include paresthesia, but 5-11% of these lesions are painful.[6] Based on the clinical and radiographic features, CGCG can be divided into two types: Aggressive and non-aggressive. The aggressive form of CGCG is characterized by pain and rapid growth of the lesion which leads to cortical perforation as well as resorption of root. The aggressive form has marked tendency to recur after treatment, compared to the non-aggressive form. The non-aggressive form is slow growing and exhibits no symptoms.

Radiographic appearance of CGCG varies according to the size and nature of the lesion. Small lesions usually appear on radiographs as unilocular radiolucent lesions. Large lesions appear as unilocular or multilocular radiolucent lesions. In some lesions, there are few internal wispy septa; however, crenations produce scalloping of the margins. These septae are a characteristic radiographic feature of CGCG.[7] In the cases reported here, unilocular radiolucency was seen in OPG of both cases. Whitaker and Waldron reported tooth displacement and root resorption in 36% and 43% of the cases, respectively; in the reported cases, lesion-related tooth displacement was found in both the cases. Histologically, CGCGs are characterized by the presence of numerous MCGs which are concentrated in the areas of hemorrhage and distributed in a collagenous stroma. Concentric areas of hemorrhage with liberation of hemosiderin pigment and newly formed osteoid or bone are often seen. Similar histologic features were seen in our cases.[8]

The treatment of choice is typically surgical curettage of the involved area. The recurrence rate given in various literature reports after conventional surgical curettage ranges from 11 to 49%.[9] Traditionally, surgical curettage has been relied upon as the treatment of choice for CGCGs. In some cases, curettage combined with adjunctive therapies comprising peripheral osteotomy, cryotherapy with liquid nitrogen, use of Carnoy's solution, radiotherapy, or postoperative use of interferon alpha; all provide satisfactory to excellent results. Intralesional injections of an aqueous solution of triamcinolone with either 2% lidocaine or bupivacaine, 50% mixture by volume, are used. The solution is administered with a 5-cm disposable syringe, delivering a dose of 30 mg in adults and 25 mg in children. The site of injection is gauged by clinically estimating the site where the cortical bone is more expanded and thinnest and once inside the lesion, small amounts are injected into different areas. These injections are repeated every 3 weeks and the treatment is limited when there is a significant amount of resistance caused by the bone being formed and calcified. No side effects are reported.

It has been suggested that immunohistochemical staining for glucocorticoid and calcitonin receptors on the mononuclear or multinucleated cells in giant cell lesions can help in choosing the most appropriate conservative, medical therapy. It has also been shown that osteoclastogenesis is under the influence of osteoprotegrin (inhibition of bone resorption) and its antagonist osteoprotegrin ligand (initiation of resorption) via osteoclast receptor proteins that have been found to be present in CGCG and are known as receptor activator of nuclear factor kappa-B (RANK). Osteoprotegrin as a pharmacologic agent has been proposed to inhibit bone resorption. However, the utility of osteoprotegrin is untested, and the systemic effects have not been evaluated. The future holds promise for the therapy of CGCG, as the ongoing clinical and laboratory research on the gene and protein expression of these tumors can lead to identification of therapeutic targets.

CONCLUSION

The relatively high frequency of CGCGs in the population makes it important for clinicians to understand their clinicoradiologic presentation and clinical behavior. Classifying these lesions as "aggressive" or "nonaggressive" can help in choosing the most appropriate treatment. The clinical behavior of this lesion is quite variable and difficult to predict. Hence, we suggest clinicians should consider all the possible differential diagnoses and must rule them out one by one considering all diagnostic points, so that proper treatment of lesion can be planned.

CONFLICTS OF INTEREST

The authors declare they have no potential conflict of interests regarding this article.

REFERENCES

1. Alok A, Singh ID, Singh S, Kishore M. Central giant cell granuloma: A report of 2 cases and review of literature. J Indian Acad Oral Med Radiol 2015;27:156-9.

2. Cohen MA. Management of a huge central giant cell granuloma of the maxilla. J Oral Maxillofac Surg 1998;46:509-13.

3. Whitaker SB, Waldron CA. Central giant cell lesions of the jaws. A clinical, radiologic, and histopathologic study. Oral Surg Oral Med Oral Pathol 1993;75:199-208.

4. Kaff e I, Ardekian, Taicher S, Li ner MM, Buchner A. Radiologic features of central giant cell granuloma of the jaws. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1996;81:720-6.

5. Jaff e HL. Giant-cell reparative granuloma, traumatic bone cyst, and fi brous (fi bro-oseous) dysplasia of the jawbones. Oral Surg Oral Med Oral Pathol 1953;6:159-75.

6. Spraggs PD, Roth J, Young-Ramsaran J, Goodwin WJ. Giant cell reparative granuloma of the maxilla. Ear Nose Throat J 1997;76:445-6, 449.

7. Güngörmüs M, Akgül HM. Central giant cell granuloma of the jaws: A clinical and radiologic study. J Contemp Dent Pract 2003;4:87-97.

8. Cha ha MR, Ali K, Aslam A, Afzal B, Shahzad MA. Current concepts in central giant cell granuloma. Pakistan Oral Dent 2006;26:71-8.

9. De Lange J, Van den Akker HP. Clinical and radiological features of central giant-cell lesions of the jaw. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2005;99:464-70.