Central Giant Cell Granuloma: A Case Report

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ABSTRACT

Central giant cell granuloma (CGCG) is an intra-osseous lesion consisting of cellular fibrosis tissue containing multiple foci of hemorrhage, multinucleated giant cells and trabecules of woven bone. This lesion accounts for <7% of all benign jaw tumors. It has been reported that this lesion is diagnosed during the first two decades of life in approximately 48% of cases, and 60% of cases are evident before the age of 30. It is considerably more common in the mandible than in the maxilla. The aim of this report is to describe an unusual presentation of CGCG involving the mandibular body, ramus, condylar and coronoid processes, and to discuss the differentiated diagnosis, the radiographic presentation and the management of this lesion.

Keywords: Giant cells, Histopathological examination, Mandibular angle

INTRODUCTION

Central giant cell granuloma (CGCG) was first described by Jaffe in 1953. It is an uncommon, benign and proliferative non-neoplastic process. Jaffe considered it as a locally reparative reaction of bone, which can be possibly due to either an inflammatory response, hemorrhage or local trauma. Females are affected more frequently than males. Most lesions occur in the molar and premolar area, some of these extending up to the ascending ramus. The presence of giant cell granuloma in the mandibular body area, the entire ramus, condyle and coronoid represents a therapeutic challenge for the oral and maxillofacial surgeons. It occurs over a wide age range. The term central giant cell lesion has been proposed as the microscopic features are not those of a true granulomatous process.

The purpose of this case report is to understand the diagnostic challenge that CGCG presents in the dental clinic as well as the surgical challenge in the treatment of CGCG.

CASE REPORT

A 22-year-old woman from the remote village of Uttar Pradesh state reported to our Department of Oral and Maxillofacial Surgery with a chief complaint of painless swelling on the right back jaw region since 6 months. On examination, a swelling on the right side was revealed on posterior mandibular region, which was firm and painless. The patient had difficulty in speech and chewing, as the cheek was crushed in the interocclusal space on the right side.

Clinical examination revealed large swelling, focal, non-tender, with ill-defined margins, non-fluctuant and non-compressible, restricting the mandibular movements. On extra-oral examination, a single, focal, swelling was seen on the right side of the mandible (Figure 1).

The swelling measured about 4 cm × 4 cm. The surface of the swelling was smooth and extended from tragus of the ear superiorly and inferiorly to the angle of the mandible. The swelling was firm in consistency, showed no secondary changes and was slightly tender on palpation.

Intra orally expansile localized swelling was seen in the third molar region extending posteriorly to the posterior border of mandible in the bucco-lingual direction.

Radiographically (i.e., Orthopantomogram (OPG) and postero-anterior view of mandible) (Figures 2 and 3) the lesion was seen as a well-defined, expansile, unilocular radiolucency with varying degrees of expansion of the cortical plates occupying the ramus, angle and coronoid process region. Radiographic appearance of the lesion...
is not pathognomic and may be confused with that of many other lesions of the jaws.

Based on the clinical and radiological examination, a provisional diagnosis of ameloblastoma, dentigerous cyst, pindborg’s tumor, true giant cell lesion, odontogenic keratocyst cyst was made. Fine-needle aspiration cytology was performed which came out to be negative to confirm the diagnosis, an incisional biopsy was planned and performed under left atrium for H/E examination.

Biopsied specimen revealed connective tissue made up of mature collagen fibres, fibroblasts and showing numerous multinucleate giant cells with foci of osseous structures (Figure 4). On the basis of histopathological and radiological findings, a diagnosis of aggressive CGCG was established.

Pre-operative evaluation was performed before approaching the patient under general anesthesia. Routine hemogram and urine examination were done, which turned out to be normal. The serum calcium, phosphorous, parathyroid hormone were also normal, thereby excluding the possibility of hyperthyroidism.

The patient was put under general anesthesia and prepared for surgical curettage of the lesion. The mandible was approached by deep vestibular incision intraorally. The lesion was exposed buccally (Figure 5).

Enucleation with curettage was done with the removal of a small amount of bone surrounding the lesion peripherally. Extraction of 48 was done followed with primary closure of the surgical site with the help of 3-0 silk suture. Post-operative radiograph OPG was taken after 3 months (Figure 6) No signs of recurrence were found in follow-up period of 2 years.

**DISCUSSION**

The CGCG appears as a painless expansile mass. The clinical behaviour of the CGCG ranges from a slowly growing asymptomatic swelling to an aggressive lesion causing pain, local bone destruction, root resorption or displacement of the tooth. The lesion has been reported as confined to the tooth bearing area of the jaws, being more common in the anterior portion of the mandibular body.4

Aggressive central giant cell lesions have been described as painful, rapidly growing and producing cortical perforation, root resorption.3 Giant cells are the most prominent histopathological feature of CGCG and many
investigative studies have been directed to the role of the original mononuclear cells in its pathogenesis. The mononuclear cells can form osteoclasts like giant cells in vitro by the development of osteolytic lesions. Besides osteoclasts, the mononuclear cells differentiate themselves in macrophages that play a critical in connective tissue during inflammatory and reparative process.

The most widely accepted method of surgical treatment of CGCG is aggressive curettage. Curettage of the tumor mass, followed by the removal of the peripheral bony margins results in a low recurrence rate and good prognosis.

Another conservative treatment of CGCG is intralesional injection of corticosteroids, calcitonin and bisphosphonates. It remains somewhat controversial because some surgeons have not been able to duplicate the original success of this method. The use of exogenous calcitonin may have some merit in the treatment of aggressive lesions; function of giant cells is inhibited by calcitonin. It can be administered in two different modes, i.e., 100 IV calcitonin subcutaneously daily or 50 IV calcitonin subcutaneously and 200 IV nasal spray daily. Some investigators have reported successful treatment, using intra-lesional injections of corticosteroids. As corticosteroids inhibit osteoclasts in marrow cultures and under conditions of bone absorption by increased apoptosis, their use for giant cell granuloma has been advocated. Bisphosphonates have been used to treat giant cell lesions in children, because of their inhibiting action of osteoclastic bone resorption.

CONCLUSION

For the present case, treatment protocol was surgical and we have not used conservative treatment. Surgery is the traditional and accepted form of treatment for CGCG which ranges from curettage to en bloc resection of the lesions. The present case highlights the difficulty in diagnosing and management of the CGCGs.

REFERENCES


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