Maxillary central giant cell granuloma: An uncommon presentation

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ABSTRACT

Central giant cell granuloma (CGCG) previously came under the category of reparative granuloma. It is an intraosseous, non-neoplastic lesion of an undetermined etiology. The mandible is the most common location for this lesion. Histopathology plays an essential role in diagnosing it. Here, we present the case of an aggressive type of CGCG of the maxilla in a young adult lactating female patient.

Keywords: Giant cells, Granuloma, Osteolytic lesion.

1. INTRODUCTION

Central giant cell granuloma (CGCG) is a benign lesion of a jaw bone. It is a rare entity accounting for less than 7% of all benign jaw lesions and has a variably aggressive nature. The CGCG was first described by Jaffe in 1953. According to WHO, CGCG is an osteolytic lesion consisting of many multinucleated giant cells in the background of intense chronic inflammatory cell reaction and hemorrhagic area (Mohan et al., 2013). Its manifestations have a wide age range; 2/3rd of the cases occur below the thirty years of age with a female to male ratio of 2:1. Both the jaws are affected but more commonly seen in the mandible as compared to the maxilla and 80% of the lesions have a preference for the anterior jaw region as compared to the posterior jaw region. Initially, CGCG is an asymptomatic lesion that later becomes expansive (Ramesh, 2020). The present case is of a CGCG in a young lactating female managed by a conservative surgical approach.

2. CASE PRESENTATION

A 22 years female visited Sharad Pawar Dental College & Hospital, Wardha, with the chief complaint of painless swelling in the upper front region of the jaw for 1-1.5 months. The swelling was associated with the upper right canine to upper left central incisor in the palatal area. She was alright 1.5 months back; then, she started experiencing swelling in the upper front region of the jaw, which was small initially and gradually increased to the current size of 3 x 2 cm (Figure 1).
On inspection, the swelling was roughly oval in shape and tender in nature with no rise in local temperature. The patient is a lactating mother of a 3 month old baby. No history of any associated systemic disease was reported. History of the root canal with 11, 12, 13 and 21 was reported three months back. History of pus discharge and loosening of associated teeth was given. The patient visited a local dentist, where her orthopantomogram was carried out. The lesion was surgically excised and report was given by oral pathologist as central giant cell granuloma (Figure 2).

Extra oral examination of the patient revealed normal and non-tender overlying skin. The face was bilaterally symmetrical and swelling was absent. No clicking sound was present on TMJ palpation. No history of trauma was reported. History of balm application & hot fomentation were not present. Bilaterally no lymph nodes were palpable.

3. DISCUSSION

CGCG is a non-neoplastic proliferative lesion of an unknown etiology. It has more predilections in the mandibular arch than the maxillary arch and is most commonly seen on the right side than the left side of the jaw (Sidhu et al., 1995; Curtis and Walker, 2005). Because of high estrogen levels during pregnancy, females have more incidences of CGCGs. Most commonly, CGCGs are discovered incidentally on routine dental radiographs. Still, sometimes patients show symptomatic findings like swelling, pain, paraesthesia or cortical bone perforation, resulting in ulceration or many times there is presence of painless expansion of the bone that is noticed by the patient (Barnes et al., 2005; Whitaker et al., 1994).

The histogenesis of CGCG in the jawbones remains controversial. One of the most important etiological factors for CGCG is trauma or it may be an active, an inflammatory and an infective oral neoplastic process in origin. Vascular hypothesis suggests that CGCG belongs to the spectrum of mesenchyme proliferative vascular primary jaw lesions. Various cytokines and growth factors like vascular endothelial growth factor and basic fibroblast growth factor are produced and released from activated monocytes and macrophages which shows a synergistic effect and thereby regulates the process of angiogenesis (Gupta et al., 2011; Shrivastava et
Another theory states that the initial CGCG occurs due to an end steal hemorrhage. In support of this, Kramer in 1962 concluded that if the formation of CGCG is concerned with the repair following hemorrhage. Trauma and some defect in capillaries lead to the formation of a multi centric hemorrhagic center. Because of these hemorrhagic centers, there is an accumulation of tissue that forms this lesion (Mohan et al., 2013).

There is confusion between the CGCG and giant cell tumor. In the case of the giant cell tumor, age range varies between twenty to forty years, which occurs in long bones. It is aggressive in nature with high recurrence rate after curettage. Histopathologically, in giant cell tumor, there is uniform distribution of giant cells which are osteoclastic and the stroma is made up of plump tumor cells, whereas in CGCG, foreign body type giant cells are seen to have irregular distribution and vacuolation and the stroma is made up of plump tumor cells (Gupta et al., 2013). CBCT 3D view of anterior maxillary region shows loss of labial cortex with 11 and 12 region (Figure 3).

Figure 3 CBCT 3D view of the patient showing bony defect in the apical region of incisors on right side

Histopathological features

Low power view (X10) showed haphazardly arranged densely to loosely packed bundles of collagen fibers with many fibroblasts, which are primary tumor cells. These fibroblasts are responsible for the production and retention of monocytes which transform into multinucleated giant cells (Gomes et al., 2020). Many small to large, round to oval irregularly shaped, secondary tumor cells like multinucleated giant cells were seen (Figure 2). Many endothelial lined blood vessels of varying shapes & sizes with intravasated and extravasated RBCs were noted along with mild to moderate chronic inflammatory cell infiltrate.

High power view (X40) showed multinucleated giant cells were seen with eosinophilic cytoplasm and 10-12 nuclei are arranged haphazardly in the center of cells (Figure 2). Nuclei was round to oval and vesicular to hyper chromatic. The inflammatory cell infiltrate, chiefly comprised lymphocytes and plasma cells.

CGCG is a destructive lesion of unknown etiology. It may occur at any age but is commonly seen concerning the second to the third decade, as in the present case. Profound research has been carried out in oral cancer, precancerous lesions, odontogenic cysts and tumors at our institution by different researchers (Patil, 2021; Sonone et al., 2021; Agarwal et al., 2014; Kadashetti et al., 2015; Gupta et al., 2019).

Treatment

The choice of treatment for CGCG is curettage which is almost curative. In the present case, peripheral osteotomy was done followed by chemical curettage and reconstruction. After that, specification with 11, 12 and 21 was done successfully. Fifteen days of follow-up was taken for the same patient. The patient recovered well in the postoperative period.

4. CONCLUSION

Rarely could we find connective tissue tumors like central giant cell granuloma at our place. The present case is unique in terms of location as it involves maxilla, as 2/3rd of these types of lesions are common in mandible. Many swellings can occur on the hard palate and they can be misdiagnosed as salivary gland neoplasms. CGCG in the oral cavity can be the first expression of hyperparathyroidism (HPT), which is rare, so such patients should be screened for (HPT). Histopathology is only the gold standard to come to the final diagnosis.
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Author Contributions
Dr. Swati Patil: Selection of case and manuscript preparation
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Dr. Madhuri Gawande: Data analysis and interpretation
Dr. Alka Hande: Manuscript editing
Dr. Archana Sonone: Selection of figures
Dr. Aayushi Pakhale: Manuscript review

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Data and materials availability
All data sets collected during this study are available upon reasonable request from the corresponding author.

REFERENCES AND NOTES