SURGICAL MANAGEMENT OF AGGRESSIVE SYNCHRONOUS JAW CENTRAL GIANT CELL GRANULOMA: CASE REPORT
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SUMMARY
Central giant cell granuloma (CGCG) appears to be a lesion that is unique to the jaws. It is difficult to predict its aggressiveness. A rare case of synchronous CGCG of the mandible and maxilla in a 14-year-old boy is presented.

INTRODUCTION
Central giant cell granuloma (CGCG) appears to be a lesion that is unique to the jaws, although the so-called giant cell reaction of the hands and feet shares many features. There is currently no way to predict, either clinically or microscopically, which lesions will behave aggressively. A case is presented of a 14-year-old boy who manifested synchronous CGCG of the right mandible and left maxilla.

CGCG lesions in the jaws appear to follow a fairly benign course, but more aggressive lesions have been noted, particularly in the long bones. Though rare, aggressive giant cell lesions are locally invasive, destructive, and vascular with a high recurrence rate. Their management poses an enormous challenge. A comprehensive history, thorough physical examination and biochemical and radiographic investigations are essential so that appropriate treatment is instituted. For multiple lesions that are resulting from endocrine causes, surgery may not be necessary. Surgical resection and not enucleation is indicated for aggressive lesions. Surgical enucleation is associated with high rates of recurrence in aggressive lesions. CGCG was formerly regarded as a reparative process and was accordingly called central giant cell reparative granuloma. Although the lesion is not a granulomatous inflammatory process, the term granuloma has persisted. Many investigators believe that CGCG should be classified as a reactive lesion. Numerous documented aggressive and recurrent cases suggest that it may behave as a neoplasm. Recurrences are more likely to be seen in children than adults. Surgery has been the treatment of choice, although alternative medical therapy with calcitonin injections has shown some promising results in reducing lesions.

CASE REPORT
A 14-year-old boy presented to the maxillofacial department complaining of a left-sided maxillary and a right-sided mandibular swellings for a period of three months (Figure 1). The swellings were painless and poorly demarcated from the surrounding tissues. Intra orally the swellings were soft and fleshy in consistency. There was buccolingual expansion of the mandibular cortex with a perforation buccally. At the time of review the patient was found to have been severely anaemic with haemoglobin concentration of 6g/dl. The anaemia was iron deficiency type. The rest of the medical and dental history was non-remarkable.

Radiographic examination revealed a multilocular lesion involving the entire body of the right mandible. The maxillary lesion manifested a radio-opacity with saucerisation, almost resembling an invasive lesion with floating teeth (Figure 2). Incisional biopsy was performed on both lesions under local anaesthesia. Histopathological analysis revealed epithelium with normal connective tissue thickness and morphology overlying a connective tissue stroma, that had poorly defined area of transition into a lesion with a highly vascular and cellular matrix (Figure 3). These cells were fibroblasts with moderate mitoses and little or no pleomorphism, interspersed by Giant cells. The features were suggestive of a giant cell granuloma.

The patient was scheduled for surgical excision of the mandibular and maxillary lesions after transfusions and haematinic therapy, followed by regular reviews. No further recurrence has been reported in the last two years and radiographic evaluation shows complete resolution of the lesion.
DISCUSSION

CGCGs occur typically in the second and third decades, with a mean age of 25 years. Females are more frequently affected than males. CGCG has a predilection for the mandible, especially the body and anterior portions of the jaw. The lesion is radiolucent and usually multilocular (1,3-5). In this case the mandibular lesion was multilocular whereas the maxillary lesion was radiopaque. Resorption and or movement of teeth may be seen, and penetration of the jaw cortex may occur in aggressive lesions (3-5). In this case there was perforation of the mandibular buccal cortical plate and there was mobility of the mandibular and maxillary teeth that were eventually lost in the affected quadrants. Most patients are asymptomatic, although pain or paresthesia may be a presenting complaint, particularly in aggressive lesions (1). Hemorrhage is usually found (1, 4, 5). Cells of fibroblastic origin are the dominant stromal cells. Mitotic figures may be frequently seen and may be numerous. Although they are of no prognostic significance, they may be confused for a malignancy (3,10). In this case the lesion had been confused for a lymphoma at the initial presentation.

There are other lesions that occur in the jaws that contain giant cells within them. These include the aneurysmal bone cyst, giant cell tumor, hyperparathyroidism and cherubism (3, 10). Their relationship to each other, however, is ill-defined. Histologically all of the giant cell lesions appear identical and usually cannot be distinguished on light microscopy alone (3,10). This perhaps highlights the importance of a good clinical history and investigations including histology and immunohistochemistry in deciding the choice of treatment when handling these lesions. Genetic markers may have to be used to differentiate some of the lesions, even though none of the histologic or immunohistologic features yet described have allowed separation of aggressive from nonaggressive lesions (2, 3-6,9). Hyperparathyroidism may be excluded from the diagnosis by serum calcium, phosphate and parathormone and parathormone-related protein assays in all but the single small and more benign lesions (2,4-6,7).

Aggressive, recurrent giant cell lesions, usually known as giant cell tumor (GCT) are common in the long bones, but not in the jaws. CGCG and GCT have overlapping histopathology features and, therefore, separation of these two lesions can be difficult. (1, 2, 4). The mainstay of treatment is usually surgery. This consists of local curettage, which is usually curative. There is a recurrence rate of 15-20% 1, 4. For lesions that are large, conservative curettage may result in the loss of many teeth or the inferior alveolar nerve in the mandible. For the aggressive variants, more aggressive surgery has been suggested.
including mandibular resection and appropriate reconstruction (1). Complete surgical excision is a therapy of choice in GCT, however, others believe in curettage of the affected area. Excision is preferred over simple curettage because of the higher risk of recurrence without complete excision (4). A number of nonsurgical treatments have been tried (1, 6, 7) Intralesional triamcinolone injected into the lesion once per week for six weeks has been tried with some success (6,7). Their actual mode of action is unknown but they may work by suppressing the inflammatory component of the lesion. They are best used for smaller lesions that can be more easily treated by intralesional injections. Calcitonin given by subcutaneous injection has also been tried with some success though evidence is theoretical (2, 5, 8, 9, 11). Interferon has also been utilised and acts by suppressing the angiogenic component of this lesion allowing healing to occur. Surgery is still required after the interferon treatment but it may be less radical surgery so that chances of recurrence are diminished. There are suggestions that the CGCG may, in fact, be a self healing lesion, with the natural healing process stimulated by the nonsurgical therapy employed (12). Multilocular lesions that are a result of the brown’s tumour of hyperparathyroidism are reported to resolve once the parathyroid adenoma is surgically removed without necessarily having to treat the jaw lesion (13, 14).

REFERENCES