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Case Report

An unusual radiological manifestation of a mandibular central giant cell granuloma

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ABSTRACT

Central giant cell granuloma (CGCG) is an uncommon benign osseous lesion of the jaw. We present the case of an aggressive CGCG in a 16-year-old boy with a 5-month history of facial swelling and nasal obstruction. He was referred for a radiology consult for multiple expanding swellings on the right hemi-face. The swellings were reported to have an insidious onset, slow progression and no association with paresthesia, nasal discharge, or systemic symptoms. Extra oral examination revealed a diffuse swelling on the right side of the face which obliterated the nasolabial fold causing facial asymmetry. A face and neck computed tomography (CT) showed expansile multi loculated lytic lesions of the right mandibular ramus which involved the condyle and hard palate. Similar lesions were also found in the skull base. Scans showed fine internal septations and internal ground glass haze. It also revealed involvement of several teeth roots and surrounding soft tissues. A pre-operative intra-oral biopsy of the lesion revealed multinucleated giant cells, comprising of both spindle-shaped and round cells. Differential diagnosis helped determine the lesions as CGCG. This case helps to demonstrate the wide variation in the clinical, radiological, and histopathological features of CGCG and the importance of thorough investigation for timely diagnosis.

Keywords: Intra-osseous lesions, CT, Histopathology, Giant cells, Dental curettage

INTRODUCTION

Central giant cell granulomas (CGCG) are characterized by the World Health Organization as intra osseous lesions that contain fibrous cellular growth and multiple hemorrhagic foci surrounded by multinucleated giant cells. They make up of only 7% of all non-malignant jaw lesions, making them an uncommon occurrence in clinical settings.[1] A common sight of development for CGCG is across the right-side midline of the mandible, ventral to the first molar.[1] They are twice as common in females as in males and are more likely to occur in individuals below the age of 30.[2]

We present the case of a 16-year-old boy with a 5-month history of facial swelling and nasal obstruction secondary to a large mandibular CGCG. Our objective is to highlight the importance of histopathology in the diagnosis of this enigmatic lesion. This case helps to demonstrate wide variation in clinical, radiological, and histopathological features of CGCG. Moreover, we aim to determine the features of CGCG of facial bones in CT and medical resonance imaging (MRI) scans.

CASE REPORT

A 16-year-old boy was referred for a radiology consult for multiple expanding swellings which had been present

for past 5 months on right side of his face. Swellings were reported to be insidious in onset and had progressed slowly from a small lesion to approximately greater than 5 cm. They were not associated with paresthesia, nasal discharge or systemic symptoms. There was h/o trauma to face in a road traffic accident 3 years back. Medical and familial histories were noncontributory and oral hygiene was good. Extra oral examination revealed a diffuse swelling on right side of face which had obliterated the nasolabial fold resulting in facial asymmetry. The swelling had no localized warmth on palpation. Overlying skin was normal with no associated lymphadenopathy.

Based on h/o and clinical examination the following differential diagnoses in addition to CGCG established. Radicular cyst is the most common type of cyst in the jaws which arises from the teeth and may be associated with trauma. It produces no symptoms unless secondarily infected. Adenomatoid odontogenic tumor (AOT) was another diagnosis to be considered. It is an uncommon tumor of odontogenic origin. It is a slow-growing and painless tumor, associated with a missing tooth. It occurs most frequently in the maxilla in the incisor-canine-premolar region. In our case, the swelling was not associated with embedded tooth. Hence extra-follicular variant of AOT was considered in the differential diagnosis. Fibrous dysplasia was also considered in the differential diagnosis. This monostotic form of fibrous dysplasia has a predilection for females and and commonly involves the maxilla. It causes expansion of the affected jaw and displacement of teeth.

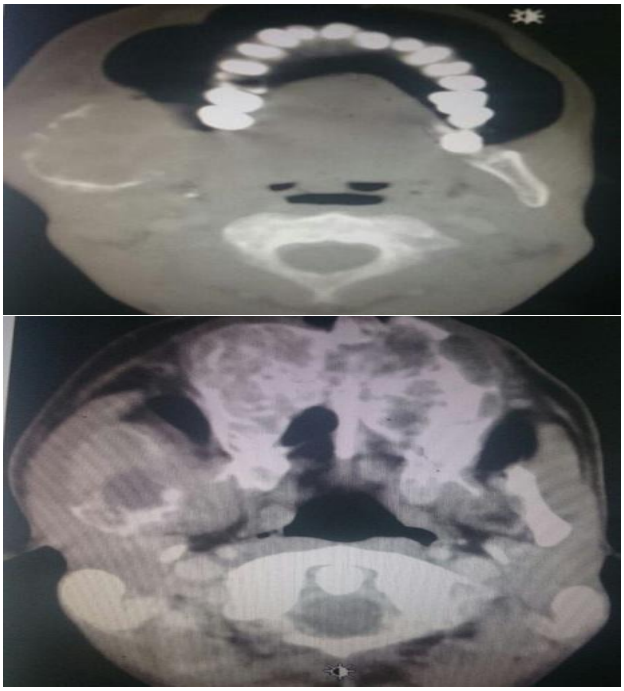

Figure 1: CGCTG; CT scan axial images shows multiloculated, expansile lytic lesions involving mandible, maxilla, hard palate with characteristics ground glass haze.

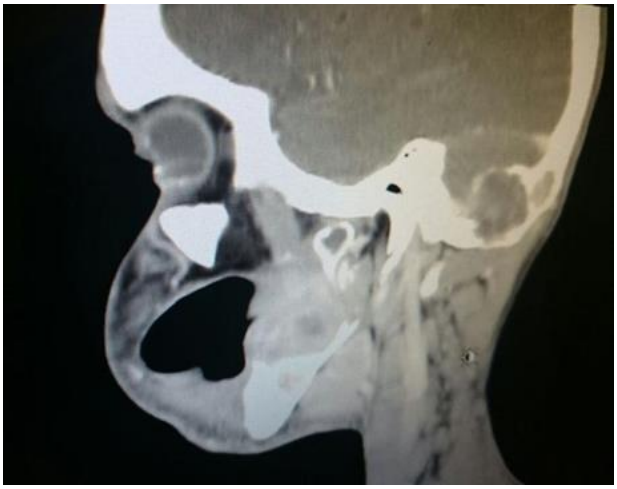

Figure 2: CGCTG; CT scan reformatted images shows multiloculated expansile lytic lesions involving mandible, maxilla, hard palate and base of skull with adjacent small soft tissue component and bony remodelling. Note the presence of significant facial deformity.

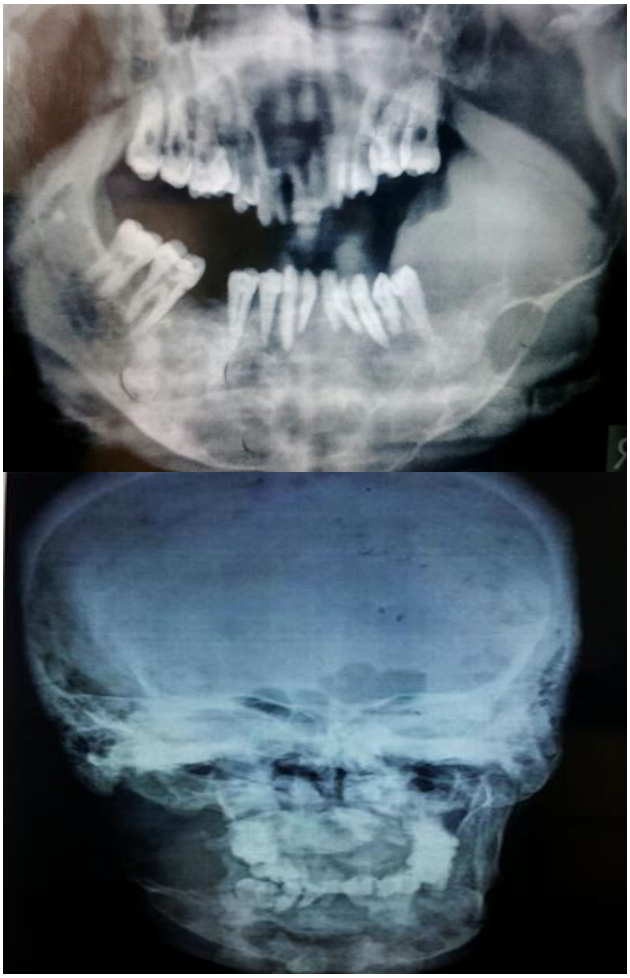

Figure 3: CGCG exhibiting a multilocular radiolucency with thin trabeculations giving honey comb pattern causing expansion of mandible, hard palate, seen to cross the midline in a 16-year-old male.

CT scans of the face and neck with contrast and reformatted images showed multiple expansile multi loculated lytic lesions involving the mandible and its ramus on right side in addition to the hard palate. Few similar lesions also involved the base of the skull. Scans showed fine internal septations and ground glass haze. It also revealed thinned, expanded, and interrupted cortical bone margins both on the buccal and lingual sides of the mandible, involving the right mandibular canal, the right mental foramen, several teeth roots and surrounding soft tissues. (Figure 1-3). A pre-operative intra-oral biopsy of the lesion revealed multinucleated giant cells, comprising of both spindle-shaped and round cells, which were found mostly in the areas of hemorrhages. These findings were consistent with diagnosis of Giant Cell Reparative Granuloma (GCRG). It is difficult to distinguish this lesion histologically from brown tumor of hyperparathyroidism. Hence serum levels of calcium, phosphorus and alkaline phosphatase were advised which were found to be in normal limits.

DISCUSSION

This case was unique in its presentation as patient was male with maxillary mass that extended into other craniofacial bones including mandible, hard palate, maxillary sinus, skull base and vault. This is rare occurrence for CGCG as it mostly commonly presents in females with most cases involving mandibular lesions. These findings bring attention to variability of CGCG which often make diagnosis through clinical and radiological findings difficult. Consequently, long-standing diagnosis is often reached solely through histopathology which shows distinguishable giant cells.

However, the presence of giant cells may be indicative of several other types of bone lesions which include Giant Cell Tumor (GCT), fibrous dysplasia, aneurysmal bone cyst, lesions caused by primary or secondary hyperparathyroidism (brown tumors), cherubism and Pagets disease, endochromatosis and Goltz syndrome.[3,4,5] These varying associations are reflective of how clinical relevance and prognosis can vary from morphological similarities and the showcases the importance of thorough diagnostic investigations.

Similarly, CGCG cases may present with a range of clinical findings as well. Radiological findings may vary depending on the aggressive nature of the lesions. Non-aggressive types present with nonspecific lytic, expansile multi loculated lesions which result is thinning and scalloping of the bony cortex. The cortex is, however, rarely breached and periosteal reaction is rarely seen. On the other hand, aggressive lesions show bone destruction often including resorption of root tips of surrounding unerupted teeth.[5] Distinguishing between these 2 types of lesions is crucial to determine the risk of future recurrence, metastasis, and malignant transformation. However, in order to determine involvement of craniofacial bones, as is case with our patient, histologic and immunohistochemical evidence is necessary.

In addition to inconsistent clinical and radiological findings, CGCG also shows highly variable patterns of disease progression.[6] It may present as a slowly developing swelling with no symptoms or it may be a fast-growing lesion that rapidly hollows out bone and displaces surrounding structures causing pain. It rarely results in neural and soft tissue infiltration as it is an encapsulated lesion, however expansion and displacement of surrounding structures is a common occurrence. Distinguishable clinical findings include an asymmetric face which is often a result of localized swelling of the maxilla.[1]

To classify a tumor as aggressive it must meet either 1 major or 3 minor criteria. Size greater than or equal to 5 cm and post curettage recurrence fall under major criteria; whereas resorption of roots, displacement of teeth, thinning of cortical bone, perforations, rapid growth and pain or paresthesia are included in minor criteria.[7] This case fulfilled 1 major and 4 minor criteria and histological findings included fusiform cells and multinucleated giant cells suspended in a fibroblastic stroma, thus confirming diagnoses of CGCG.[8]

Our patient also reported a history of trauma which is a key etiologic occurrence in the development of this lesion. Trauma may result in capillary defects leading to multicentric micro hemorrhaging which causes tissue accumulation. This has been known to cause growth of lesions and in rare cases result in multiple lesions.[6]

Radiologic findings of CGCG are slow growing, nonspecific lytic, expansile, multiloculated lesions that may thin out and scallop the bone cortex but will rarely infiltrate it. They do not show new bone deposition but aggressive lesions may cause bone destruction, and resorption of the root tips of surrounding teeth. Thinning and destruction of bone is best seen with CT and MRI scans.[5] However, a confirmed diagnosis still requires histologic and immunohistochemical evidence and is necessary to distinguish a CGCG from a GCT.[9]

Treatment of CGCG mainly includes dental curettage or excision of the lesion. Radiation therapy is not advised as it carries long-term contraindications. Supplementary to surgery, steroids or calcitonin may be prescribed to reduce osteoclastic activity. Anti-angiogenic agents such as interferon-alpha and intravenous bisphosphonates have also proven to be effective for managing aggressive forms of CGCG.[5] CGCGs have a recurrence rate of 13-49% which is reportedly higher for more aggressive lesions.[8] Recurrence is often rare if a local lesion with well-defined boundaries was excised via dental curettage.[5] Failure of treatment will often present within the first two years of therapy.[6]

CONCLUSION

These findings have highlighted the CGCG should be included in the differential diagnosis of expansile craniofacial masses because it has imaging findings similar to those of other more common lesions such as ameloblastoma, aneurysmal bone cyst, brown tumor of hyperparathyroidism and GCT. Consideration of histopathological findings is proven to be of utmost importance, especially when differentiating a CGCG from a GCT as GCT have a more severe prognosis and a higher chance of metastasis.

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