

Central giant cell granuloma mimicking an adenomatoid odontogenic tumor

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Abstract

Central giant cell granulomas are non-neoplastic lesions of unknown etiology. They affect females more than males with the mandibular anterior region being the most common site of occurrence. Clinically central giant cell granulomas present as asymptomatic, expansile swellings causing deviation of associated teeth. Radiologically they usually presents as multilocular lesions causing expansion or perforation of cortical bone. Central giant cell granulomas are usually confused as other lesions both clinically and radiologically, and a definitive diagnosis can be made only histologically. We report here a rare case of central giant cell granuloma in association with congenitally missing tooth which was misdiagnosed to be an adenomatoid odontogenic tumor both clinically and radiologically. This case report also highlights yet another unique presentation of central giant cell granulomas that is in association with a congenitally missing maxillary lateral incisor.

Keywords: Adenomatoid odontogenic tumor, central giant cell granuloma

Introduction

Giant cell granuloma is described as a benign lesion affecting the mandible and maxilla that consists of a massive fibrohistiocytic proliferation with numerous heavily hemosidrin laden multinucleated giant cells.^[1] Jaffe in 1953 described central giant cell granulomas (CGCG) as an idiopathic non-neoplastic proliferative lesion. CGCG is known to occur in individuals less than 30 years of age with definite female predilection (2:1). It accounts for approximately 7% of all benign tumors of the jaws.^[2] Many times it may be totally asymptomatic and a coincidental finding during a routine radiographic examination.^[3] They are more prevalent in the mandible, particularly the anterior region than the maxilla.^[4] Apart from jaw bones, CGCG has also been reported to occur in small bones of hands and feet.^[5] Radiographically it presents usually as multilocular, expansile radiolucency displacing the teeth, sometimes causing root resorption and cortical perforation.^[6]

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The treatment of choice for CGCG is excision and currattage followed by bone grafting.^[7] The conservative treatment approach includes intralesional calcitonin, intralesional steroids, antiangiogenic therapy, and radiotherapy.^[8]

We report an unusual case of CGCG in the maxillary anterior region associated with a congenitally missing tooth mimicking an adenomatoid odontogenic tumor.

Case Report

A 15-year-old girl came to the department of Oral Medicine and Radiology with the chief complaint of a swelling on the left upper anterior teeth region. She gave a history of noticing a peanut sized, assymptomatic swelling 2 months back which was gradually growing. She also gave history of noticing mobility and dull pain in the teeth associated with the swelling. The patient consulted a dentist with the above complaint following which root canal treatment was done in two teeth related to the swelling. But when patient noticed further growth of swelling she reported to our department. There was no history of trauma or discharge from the swelling. Patient gave history of congenitally missing lateral incisors.

There was no significant past medical history, family history, or personal history. On general examination she was moderately built and nourished, alert, responsive, and cooperative. Vital signs were all within the normal range with no signs of pallor, cyanosis, icterus, clubbing, and edema.

Extraorally, a solitary ill-defined swelling was noticed unilaterally on the left side of the face in the region of ala of the nose. Swelling was roughly oval in shape, measuring approximately 2.5 cm in diameter, extending superoinferiorly from 0.5 cm above the ala of nose to the vermilion border of upper lip, mediolaterally 0.5 cm lateral to corner of mouth to the nasolabial fold, causing mild asymmetry of face. The

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skin over the swelling appeared normal. Swelling had caused the mild uplifting of the left ala of nose.

On palpation the swelling was bony hard, nontender, not compressible, and not reducible. Skin over swelling was pinchable and normal.

Intraorally, a well-defined solitary swelling was noticed in the left anterior maxilla, measuring 3 cm in its greatest dimensions, involving both the labial and palatal aspect. Swelling was completely obliterating the labial vestibule [Figure 1] with respect to 21, 22, 23, 24, extending palatally to involve the midpalatine raphe [Figure 2]. The mucosa over the swelling appeared normal.

On palpation the swelling over the labial vestibule was bony hard and not mobile. No egg shell crackling was noticed and there were no areas of fluctuance. Palatally, the swelling was soft in consistency, nontender, nonmobile, noncompressible, nonreducible with no areas of eggshell crackling, or fluctuance.

Examination of teeth revealed missing maxillary lateral incisors bilaterally. A total of 21 and 23 were mobile, mildly tender, and endodontically treated.

On summarizing the clinical findings, a young patient with the swelling in the maxillary anterior region associated with congenitally missing lateral incisors causing the bicortical expansion, mobility of associated teeth, lead us to give a provisional diagnosis of dentigerous cyst. Differential diagnosis considered were radicular cyst, calcifying epithelial odontogenic cyst, adenomatoid odontogenic tumor, fibrous dysplasia, desmoplastic ameloblastoma, and central giant cell granuloma.

Radicular cyst was considered as the associated teeth were endodontically treated. Considering the occurrence of this swelling associated with a missing tooth in a young female patient, the adenomatoid odontogenic tumor was included

in the differential diagnosis.

Monostotic fibrous dysplasia was considered as it is known to have a definite female predilection and presents as an asymptomatic bony swelling commonly affecting maxilla, causing expansion, and deviation of associated teeth.

Calcifying the epithelial odontogenic tumor was also considered in the differential diagnosis as it has wide age distribution and occurs equally in both the jaws usually anterior to 1st molar. It usually causes expansion, cortical plate perforation, and displacement of associated teeth.

Vitality test revealed that all the teeth were vital except 21 and 23. Fine needle aspiration was done by using 20-gauge needle which gave negative aspiration indicating the absence of fluid within the swelling. Negative aspiration ruled out all the cystic lesions.

Radiographic investigations included panoramic radiograph, occlusal radiograph, and intraoral periapical radiograph which revealed an inverted pear shaped radiolucency with ill-defined and irregular borders in the maxillary anterior region on the left side measuring approximately 3cm×2 cm. It involved the entire alveolar bone in between 21 and 23 (22 was missing), extending from the medial aspect of 23 to the mid-palatine suture [Figure 3]. Internal appearance of the lesion was hazy with no evidence of calcification [Figure 4]. The associated teeth, i.e., 21 and 23, were endodontically treated, tilted toward each other occlusally with no evidence of external root resorption. Maxillary sinus was intact. These radiological features ruled out the possibility of fibrous dysplasia, calcifying epithelial odontogenic tumor, and desmoplastic ameloblastoma.

Further investigations planned for the case were a complete hemogram, serum calcium, phosphate, and alkaline phosphatase which were found to be within normal limits.



Figure 1: Swelling obliterating the labial vestibule



Figure 2: Swelling extends palatally till the midline

Based on history, clinical features, radiological features, and other investigations we considered a diagnosis of the adenomatoid odontogenic tumor.

An excisional biopsy was done along with the extraction of 21 and 23 under local anesthesia. The lesion was surgically excised [Figure 5]. An irregular firm mass of tissue was obtained which was reddish brown in color. Cystic fluid was not encountered during surgery. The patient was prescribed antibiotics and analgesics and recalled after 7 days for suture removal.

Histopathological examination of the specimen revealed the presence of numerous multinucleated giant cells. Giant cells were irregular in shape, contained 4–20 nuclei approximately, and were dispersed throughout the lesional tissue. Some of these nuclei were compact whereas others were vesicular. Spindle-shaped cells and cells with vesicular nucleus were also seen surrounding the giant cells [Figure 6]. The microscopic picture was suggestive of central giant cell granuloma. Histopathologically it is difficult to distinguish between CGCG and brown tumor of hyperparathyroidism. Normal levels of serum calcium, phosphorous, and alkaline phosphatase in this case ruled out hyperparathyroidism. Hence we gave the final diagnosis of central giant cell

granuloma.

Sutures were removed after 7 days. There were no signs of infection and surgical site were healing appropriately. Antibiotics and analgesics were discontinued. A follow-up of the case was done every 2 months for 1 year. No signs of recurrence of the lesion were noticed clinically or radiologically even after 1 year [Figure 7]. A removable partial denture was given after 2 months of surgery. After 1 year of follow-up a fixed partial denture was fabricated with 11 and 24 as abutment teeth.

Discussion

Giant cell granulomas are mysterious lesions of unknown etiology. CGCG is diagnosis of exclusion and can be confirmed only histologically as we saw in this case. What made our case very peculiar was occurrence of CGCG in maxillary anterior region which is the less common site in association with a missing tooth. The association of two endodontically treated teeth with the lesion made it all the more interesting.



Figure 3: Panoramic radiograph showing the extensions of the lesion

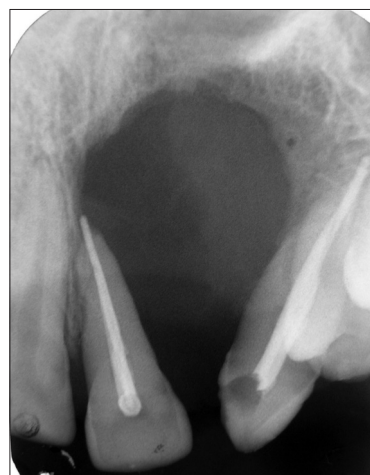


Figure 4: Intraoral periapical radiograph revealing inverted pear-shaped radiolucency



Figure 5: Excisional biopsy of the lesion

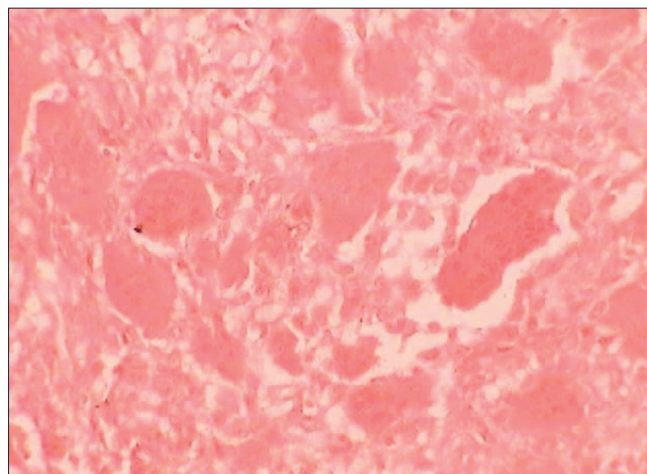


Figure 6: Histology of the specimen revealing numerous irregular giant cells dispersed throughout the lesional tissue



Figure 7: Postoperative follow-up panoramic radiograph after 10 months

A thorough literature review does not provide any information regarding the relationship between CGCG and congenitally missing tooth. Farrier *et al.* conducted a 10-year review of occurrence and treatment of CGCG and reported various presentations of the lesion such as pain, swelling, paresthesia, asymmetry, as periapical pathology, malocclusion, mobility of teeth, and as asymptomatic lesion but did not mention about concurrent occurrence of CGCG with congenitally missing tooth.^[8] Site predilection for CGCG in the mandibular anterior lesion mostly occurring on the right side whereas in our case it occurred on the left maxillary anterior region. Trauma is considered an important etiological factor whereas in our case there was no such history.

The unusual case of CGCG in the anterior maxilla associated with the congenitally missing tooth shows us yet another

unique way in which these lesions may present in oral cavity. Therefore, it also suggests that CGCG should be considered in the differential diagnosis of swellings of jaws. Further a possible etiology of CGCG as odontogenic in origin can also be suggested and further research can be planned in this direction.

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