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Huge peripheral giant cell granuloma leading to bone resorption: a report of two cases

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ABSTRACT
Although peripheral giant cell granulomas (PGCG) develop mainly within soft tissue, on rare occasions they may involve underlying bone and become visible radiologically. In such cases, the diameter of the hemorrhagic lesion rarely exceeds 20 mm, and pain is uncommon. This paper describes the successful treatment of two cases of huge PGCGs that led to alveolar bone resorption.
Key words: Peripheral giant cell granuloma, bone resorption, surgery.

INTRODUCTION

Peripheral giant cell granulomas (PGCG) are relatively uncommon reactive exophytic lesions of the oral cavity.[1-4] Although PGCGs may be observed at any age, they are reportedly more common among individuals in their 50s and 60s, present slightly more frequently in the mandible than in the maxilla and have a slightly higher incidence in females than males.[1,3-5] PGCGs may develop at the interdental papilla, gum or edentulous alveolar margins. These hemorrhagic lesions rarely exceed 2 cm in diameter, may be pedunculated or sessile, are red or reddish blue in colour and reveal an elastic structure on palpation.[1-5] PGCGs tend to be asymptomatic; however, while pain is uncommon, the lesion may become ulcerated as a result of repeated trauma.[2,5] PGCG very rarely affects the underlying bone, although the latter may suffer superficial erosion.[5]

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Treatment of a PGCG consists of surgical excision with extensive clearing of the lesion base to prevent recurrence.[1-5] This paper describes the successful treatment of two cases of huge PGCGs with alveolar bone resorption.

Case 1

A 31-year-old female with no disease antecedents of interest was referred due to pain and bleeding in the left mandible during mastication. Her history suggested that the lesion had been present for four years. Extraoral examination revealed no pathological findings; however, intraoral examination revealed an exophytic pedunculated lesion originating at the distal surface of the left mandibular first premolar and extending into the retromolar region. The lesion measured 40x20 mm, had a soft consistency and was reddish blue in color with patchy ulceration. The occlusal surface of the lesion mimicked the form of the tubercles on the antagonist teeth (Figure 1). Radiographic examination showed slight bone resorption in the form of a concave depression, a residual root, widespread vertically oriented bony spicules at the base of the lesion, and a normal trabecular component in the

edentulous region of the left mandible (Figure 2). Intraoral examination revealed poor oral hygiene, with extensive plaque on the surface of all teeth and caries in Teeth 11, 12, 13, 21, 44 and 45. Tooth 34 was vital, non-painful to percussion and showed no mobility. According to the patient's history, the teeth at the site of the lesion had been extracted due to caries. Calcium, phosphorus, alkaline phosphatase and parathyroid hormone levels were all within normal ranges. Surgical treatment consisted of incisional biopsy of the lesion under local anaesthesia. Due to a preoperative diagnosis of reactive hyperplasia, a cold scalpel was used. Histological analysis indicated the lesion to be a PGCG (Figure 3). One year after resectioning, the lesion showed no sign of recurrence.

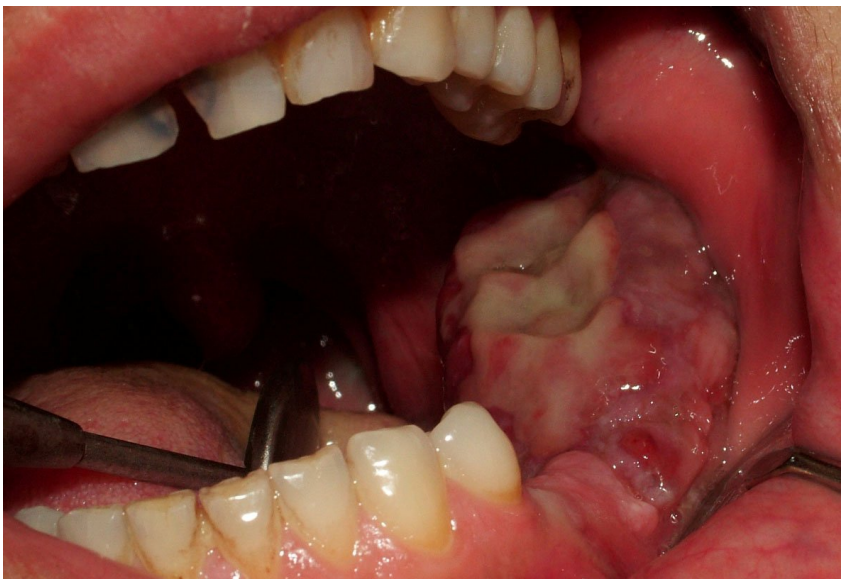

Figure 1. Intra-oral view of the lesion.

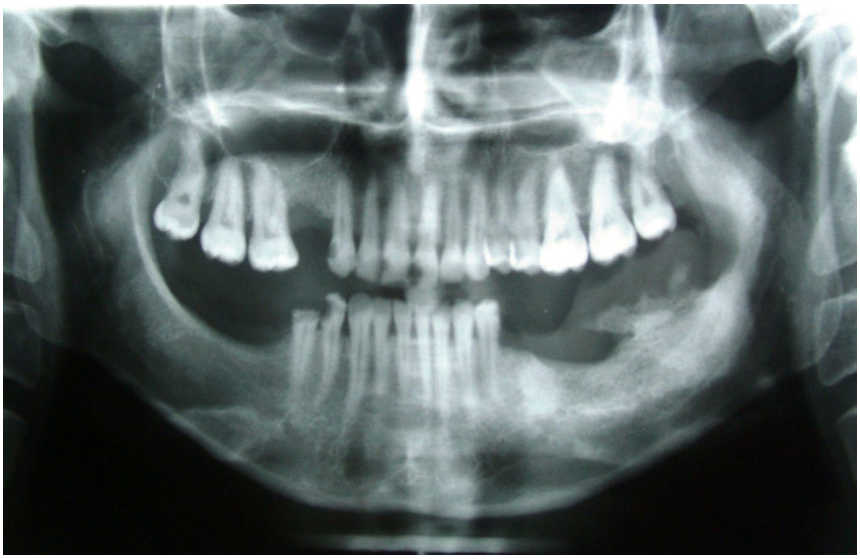

Figure 2. Panoramic view of the lesion on the left mandible.

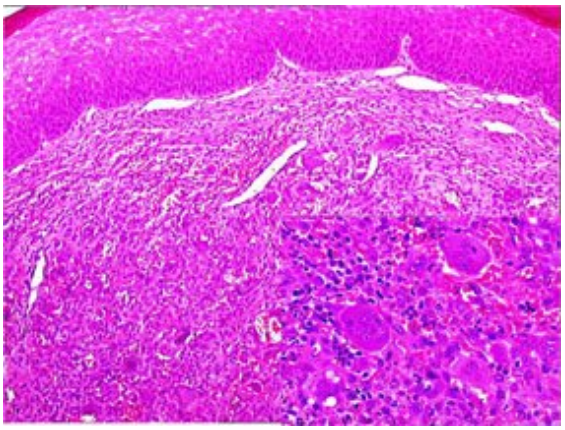

Figure 3. A multinucleated giant cell with erythrocytes in the fibrocellular base (large image magnified at H&E x200, small image at H&E x400).

Case 2

A 34-year-old male presented with pain in the left maxilla during mastication. Anamnesis established that the patient had visited a dentist approximately 1 year earlier due to pain in the left maxillary molar region while eating and occasional hemorrhage, but had not subsequently presented at the dental faculty for advanced testing and treatment as recommended. No pathology was identified at extraoral examination; however, intraoral examination revealed a reddish-blue, pedunculated lesion with a mildly ulcerated surface that completely covered the mucogingival sulcus on the buccal side and up to the mid-line on the palatal side, extending from the left maxillary second premolar to the left maxillary second molar (Figure 4). Radiographic analysis revealed concave bone resorption in the left maxillary molar region (Figure 5). An incisional biopsy of the lesion showed a PGCG. Calcium, parathyrin, alkaline phosphatase and parathyroid hormone levels were within normal ranges. The lesion was completely extracted under local anesthetic, and microscopic analysis of the soft tissue confirmed the diagnosis of PGCG (Figure 6). No sign of recurrence was observable at 10 months postoperatively.

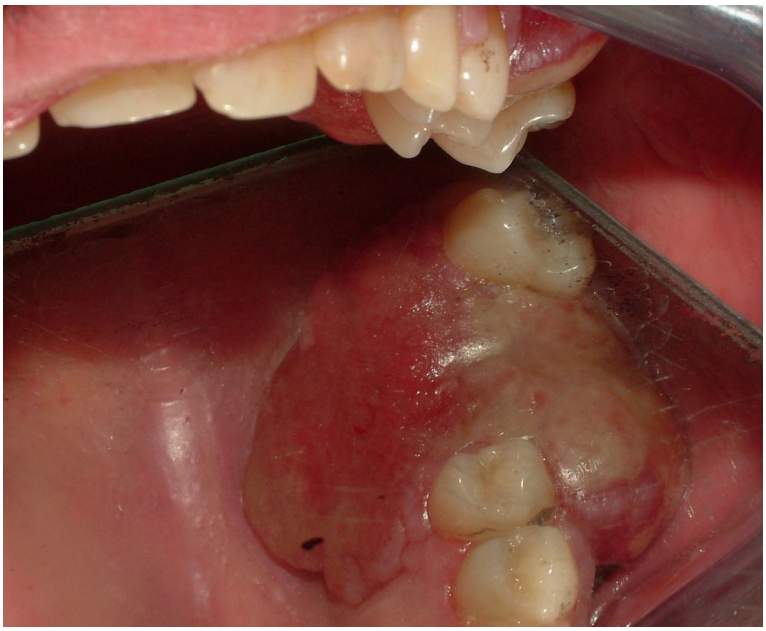

Figure 4. Intra-oral view of the lesion.

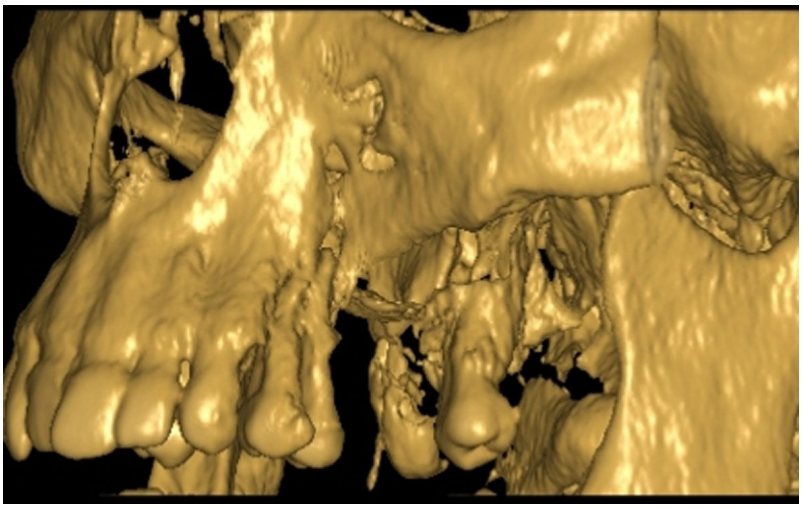

Figure 5. Case 2: CT image of the lesion.

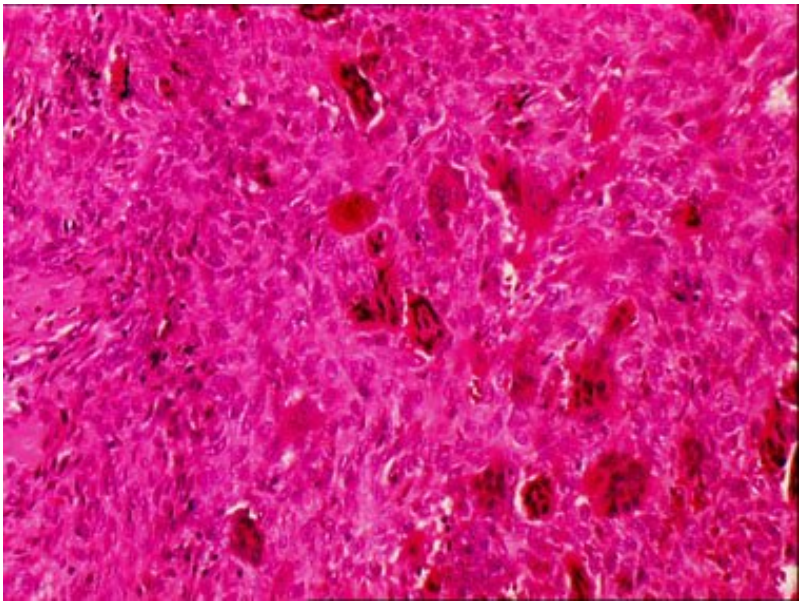

Figure 6. A fibrovascular stroma and the region under the epithelium with scattered multinucleated giant cells (H&E staining; x200).

DISCUSSION

PGCGs account for less than 10% of all hyperplastic gingival lesions. These lesions have a reported average diameter of less than 20 mm,[1] but the extent of their growth capacity is not well known.[4] The literature contains no report of a gigantic PGCG, which may be related to total growth capacity or to early treatment. Given that their highly vascularised nature makes large PGCGs susceptible to ulceration as a result of continuous occlusal trauma,[2,5] it is likely that these lesions are excised before they achieve their full growth capacity. On the other hand, considering that the gum and alveolar crest are the only places where PGCGs have been reported,[4] it is also possible that the growth capacity of these lesions is limited. Our Case 1 presented at a very late stage, suggesting that the lesion, which completely covered the edentulous alveolar area, had attained its maximum growth. Nevertheless, comprehensive scientific studies are needed to assess the maximum growth potential of PGCGs.

PGCGs usually develop in soft tissue, but they may sometimes involve the underlying bone as well and become visible on x-rays. Radiographically, the lesion may appear as superficial erosion of the involved bone in the edentulous area and as superficial destruction of the alveolar bone and widening of the periodontal ligament space in the involved teeth.[3-5] In some cases, a detailed examination of a PGCG may reveal vertically oriented bony spicules at the base of the lesion.[5] In our Case 1, occlusal trauma resulted in superficial destruction of the alveolar bone and bony spicules at the base of the lesion.

PGCG is histologically defined as a non-capsulated tissue mass that contains abundant ovoid or fusiform young connective tissue cells and multinucleated giant cells within reticular and fibrillar stroma. Hemorrhage, hemosiderin, inflammatory cells, newly formed bone and calcified material may also be seen throughout the cellular connective tissue.[4] Multinucleated giant cells are reminiscent of osteoclasts or larger than typical osteclasts, and bone resorption is rarely observed.[5] The histological findings in the cases presented here, which showed bone resorption but no osteoclasts, is in line with this data.

Clinical differentiation of PGCGs include nonossifying fibromas, which differ in consistency and coloration; CGCGs, which are expansive and destructive intraosseous lesions that can perforate the cortex, mimicking PGCGs; chondroblastomas and metastatic carcinomas, which, when localized in the gum, may provoke irregular bone destruction below the exophytic lesion; parulis, which is frequently associated with a necrotic tooth or with a periodontal disorder and occasional pain; and hemangiomas, which, unlike PGCG lesions, are pulsatile and disappear under pressure.[2] Pyogenic granulomas are difficult to differentiate from PCGC lesions on clinical grounds, but may be distinguished histopathologically. Despite the large dimensions of the PGCGs reported here, the lack of nuclear or cytological atypism indicated they were not malignant.

Traditional treatment of a PGCG consists of surgical resectioning of the lesion and elimination of the etiological factors.[1-3,5] When the periodontal membrane is affected, full resectioning may require extraction of adjacent teeth.[1,3,5] As an alternative to surgery, carbon-dioxide laser resectioning involves less intra-operative bleeding, provides wound sterilization and requires no sutures. However, laser treatment is contraindicated in cases where the lesion is oriented close to the bone and where careful curettage is required.[5]

No malignant variations of PGCGs have been reported, and recurrence rates have been reported to range from 4.41%–50%,[2,3,5] with the differences in rates possibly related to the type of surgical resectioning procedure used. Neither of the cases described here showed any sign of recurrence during approximately 1 year of follow-up.

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