Giant cell granuloma - A short review

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ABSTRACT-

Giant cell granuloma lesions are benign, non-odontogenic, moderately rare tumors of the oral cavity. They develop peripherally (within gingiva) or centrally (in bone)¹. The peripheral and central giant cell lesions (PGCL and CGCL) are a group of pathological entities with similar histopathological features and whose origin has not been fully elucidated. The former is reactive and the latter exhibits a nonneoplastic proliferative behavior.

Key words: Giant cell epulis, reactive lesions, giant cell hyperplasia

INTRODUCTION

The peripheral giant cell granuloma (PGCG) is a reactive, extraosseous and exophytic lesion occurring in the gingiva and alveolar ridge, also known as a giant-cell epulis, giant-cell reparative granuloma, osteoclastoma, or giant-cell hyperplasia occurring in the gingival or alveolar bone. Central giant cell lesions (CGCL) are intraosseousnonproliferative lesions whose etiology is unknown. It is less common than PGCL and occur exclusively in maxillary bones. It has variable clinical manifestations and may present slow asymptomatic growth with no recurrence or rapid painful growth with recurrence

PERIPHERAL GIANT CELL GRANULOMA

Peripheral giant cell granuloma (PGCG) is a benign hyperplastic reactive lesion caused by local irritation or chronic trauma. The lesion can develop at any age, though it is more common between the fifth and sixthdecades of life, and shows a slight female predilection ^{4,5,6,7}. It originates from the periodontal ligament or mucoperiosteum. PGCG arise interdentally or from the gingival margin, occur most frequently on the labial surface, and may be sessile or pedunculated. The surface is smooth, regularly outlined masses to irregularly shaped, multilobulated protuberances with surface indentations ⁶. It is commonly noticed in anterior to molars. The etiologic factors are unknown, but it may be caused due to an irritant or aggressive factor such as trauma, tooth extraction, badly finished fillings, unstable dental prosthesis, plaque, calculus, chronic infections, or impacted food^{2,3}The microscopic examination shows numerous foci of multinuclear giant cells and hemosiderineparticles in a connective tissue stroma. Giant cells have various shapes and sizes, typically containing 8 to 15 nuclei. The areas of chronic inflammation are scattered throughout the lesion, with acute involvement occurring at the surface. The overlying epithelium is usually hyperplastic, with ulceration ⁶. The differential diagnosis of peripheral giant cell granuloma includes lesions with very similar clinical and histopathological features such as CGCL, pyogenic granuloma, peripheral ossifying fibroma, fibrous hyperplasia, inflamed irritation fibroma, hemangioma, lymphangioma, amelanotic melanoma and metastatic tumors. The treatment involves local surgical excision of the lesion. Relapses can occur often due to inadequate surgical technique where the curettage is not done effectively thereby promoting recurrences.

CENTRAL GIANT CELL GRANULOMA

Jaffe initially described the term "reparative GCCG" to describe lesions, and thought to be a response to an intraosseous traumatic hemorrhage of the jaw 8. GCCG occurs mainly in children or young adults, with approximately 75% cases reported before age 30 years 9. Women are affected much more often than men^{10,11,12}. Mandible is most commonly affected whereas less than 30% occur in the maxilla, with a predilection for the anterior region ¹³. CGCL display variable clinical behavior, slowasymptomatic growth without recurrence and fast painful growth with perforation of the cortical bone plate and ulceration of themucosal surface. Radiographically, the image evidences unilocular or multilocularwell-defined radiolucent bone defects of variable size, depending on the aggressiveness of the lesion. Moreover, displacement of teeth, root resorption and perforation of the cortical bone may be observed. Histologically, the lesion is characterized by dense proliferation of oval or spindle-shaped mesenchymal cells with varyingnumber of multinucleated giant cells containing 4 to 20 nuclei. Few round macrophages, deposition of hemosiderin, extravasatederythrocytes, foci of osteoid material (bone trabeculae), dystrophic calcification and predominantly mononuclear inflammatory infiltrate can also noticed. Based on clinical and radiographic features, CGCL is divided in two categories: non-aggressive and aggressive. The former lesions account for most cases. The latter lesions cause pain and exhibit rapid growth, usually larger than five centimeters, producing expansion and perforation of the cortical bone, displacement of teeth and root resorption. Besides, there is a high recurrence rate, which generally ranges between 37.5% and 70%. The treatment is usually associated with its clinical behaviour. In milder cases simple surgical excision is recommended with thorough curettage. In aggressive lesion, curettage is followed by cryosurgery, peripheral osteotomy or enbloc resection. Certain treatments involves daily local application of calcitonin, corticosteroids and subcutaneous injection of interferon 2 alpha.

CONCLUSION

The PGCG is the most common giant cell lesion attaining a large size and may follow an aggressive course so early. The diagnosis of the lesion is based on the history, clinical examination, radiographic evaluation and histopathological examination by oral and maxillofacial surgeon allowing conservative management with minimal risk to adjacent soft and hard tissue.

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