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A Large Vestibular Peripheral Giant Cell Granuloma On Toothless Patient Associated With Prosthetic Stomatitis : A Case Report

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ABSTRACT:
Giant cell peripheral granuloma (PGCG) is a benign oral lesion occurring on the gingiva and alveolar ridge. This exophytic lesion can occur as a result of irritants such as trauma, microorganisms, plaque, calculus, restorations and dental appliances.Clinically and histologically it is normally in the form of a purplish-red tissue nodule consisting of multinucleated giant cells on a background of mononuclear stromal cells and extravasated red blood cells. In general, the treatment consists of surgical removal and curettage of the bone reaches the walls. Recurrence is a distinct possibility.
This article reports a case of peripheral giant cell granuloma occurring in the maxillary anterior region in a 48-year-old toothless patient. The lesion was completely excised with curettage and there was no apparent residual or recurrent bone swelling or defect in the biopsy area after a 6-month follow-up period.

KEYWORDS:-Peripheralgiantcellgranuloma, prosthetic stomatitis, toothless patient.

I. INTRODUCTION

Also known as giant cell epulis, osteoclastoma, cellular granuloma repair or giant cell hyperplasia, giant cell granuloma occurs either as peripheral exophytic growth of the gingiva, or as a centrally located lesion of the jaw, skull or the bones of the face or as giant central granuloma [1]. It is an infrequent lesion, considered as a reagent, extraosseous, non-neoplastic, from the periosteum or periodontium ligament. The maximum incidence of PGCG is between 40 and 60 years old. No ethnic predilection associated with the lesion has so far been described. The origin of giant cell granuloma is not yet clearly identified. Possible causes included dental extraction, periodontal surgery, presence of local irritants, biofilm and dental calculus, over-abundant restorations, indiscriminate use of toothpicks, chronic infection, dietary impaction and fractured teeth. , poorly adapted dental prostheses [2]. Other authors [3] reported this lesion in patients with hormonal imbalance associated with hyperparathyroidism.

II. CLINICAL CASE PRESENTATION

A 48-year-old womanreported to the department of oral surgery with the complaint of a vestibular mass in the upperanteriorregionevolving for nearly 8 months. The growthwasinitiallysmall and graduallyincreased in size. Shealsoreportedepisodes of severe intermittent pain.

The patient wasdiabetic, hypertensive undertreatment (Glucophage® 1000, Staticol® 20mg, Amlor® 5mg). Palpation of the neck revealedtwofirms, tender and mobile lymphnodes.

The endobuccal examination showed that the patient is edentulous total in the upper maxillary and the presence of the teeth of the incisivo-lower canine block. Two purplish mucous proliferations are noted in the maxillary anterior region, on either side of the medial brake, of about 2 and 1.5 cm, sessile, implanting themselves on the vestibular surface of this region but one more extended as the other, covered with an inflammatory mucosa, with whitish edgings, soft on palpation, not bleeding, painless.

Their surfaces appeared lobulated, smooth and shiny and showed ulceration. (**Fig.1a**).

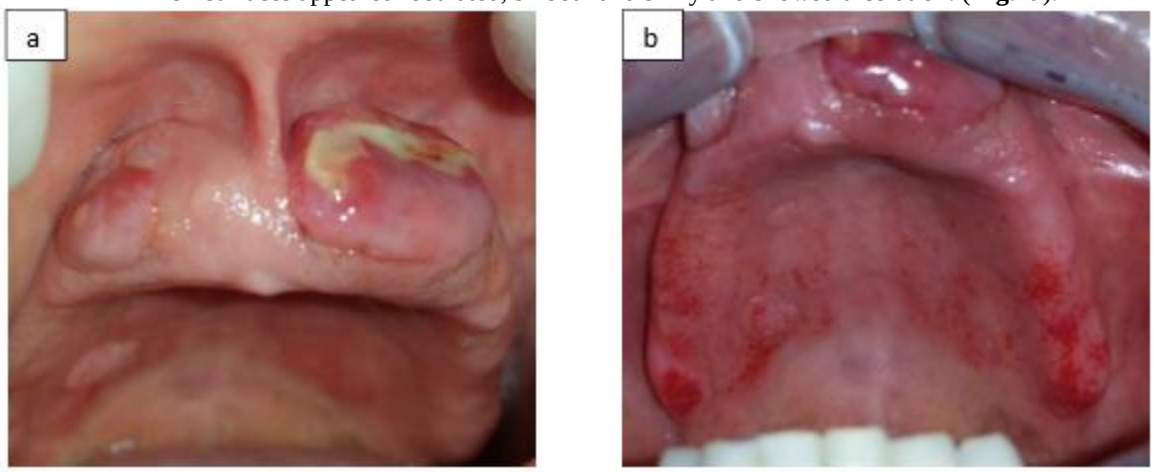

Fig 1 : a - Preoperative intraoral presentation of lesion pinkish lobulated mass in the anterior part of Vestibular gingival at the region of left and rigth incisors. b- Palatal view of the prosthetic stomatitis

There was also redness scattered on the palatal vault and on the posterior crests (**Fig.1b**) confirmed as prosthetic stomatitis. Examination of the prosthesis showed and old and poorly adapted prosthesis, old, with sharp edges. The lesion interfered with the maxillary prosthesis (**Fig.1c and 1d**).

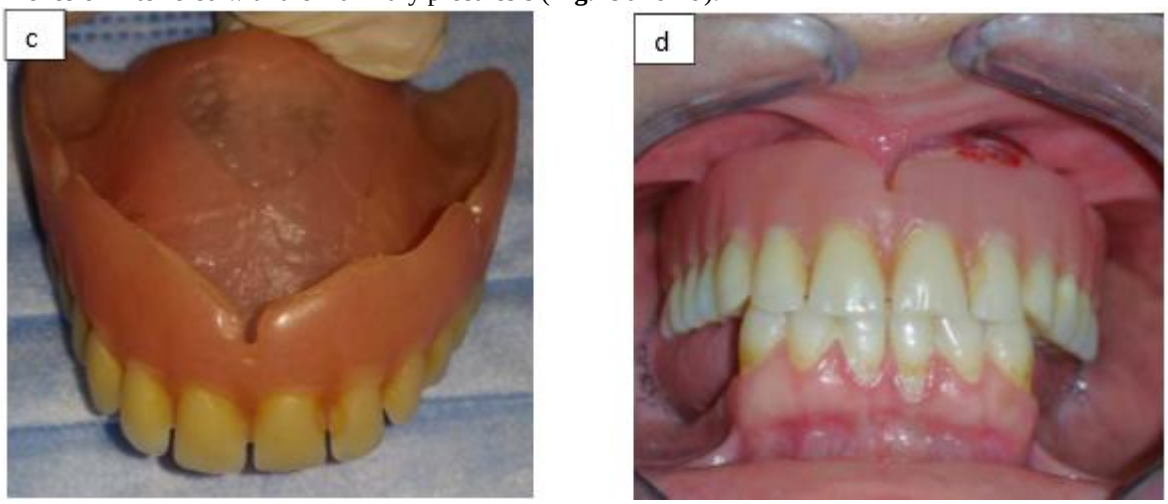

Fig 1 : c- View of maxillary prosthesis with sharp edges d-The lesion interfered with the maxillary prosthesis Panoramic X-ray showed superior alveolar resorption and probable presence of residualrootintothislesion. (**Fig 2**).

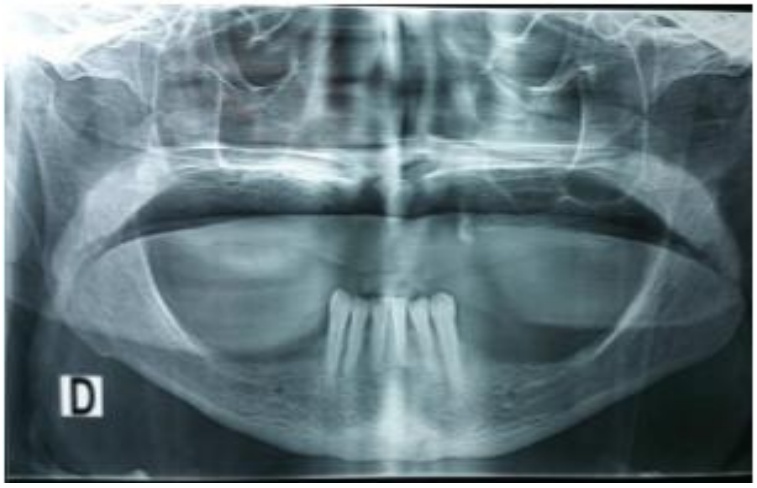

Fig 2 : Panoramic image showing maxillary radiolucent bone defect;

We had thought of other giant cell lesions as the peripheral brown tumor of hyperparathyroidism, but the parathyroid hormone dosage was normal, or the pyogenic granuloma or giant central granuloma. The

incisional biopsy of the small portion of the lesion was performed and the specimen was sent for histopathological examination. Microscopic examination of hematoxylin and eosin stained sections revealed paraperatinized, stratified, squamous epithelium with focal area of ulceration. The connective tissue showed inflammatory infiltrates, mainly lymphocytes and few plasma cells, giant multi-nucleated cells and some capillaries. In addition, few areas of reactive bone formation. All these features suggest Peripheral Giant Cell Granuloma. Note that 2 weeks after the biopsy, and before obtaining the results of the pathology, there was a recurrence of the lesion at the biopsied portion.

In view of this result, total excision of the lesion, underlying bone curettage and suture were performed (**Fig. 3**).

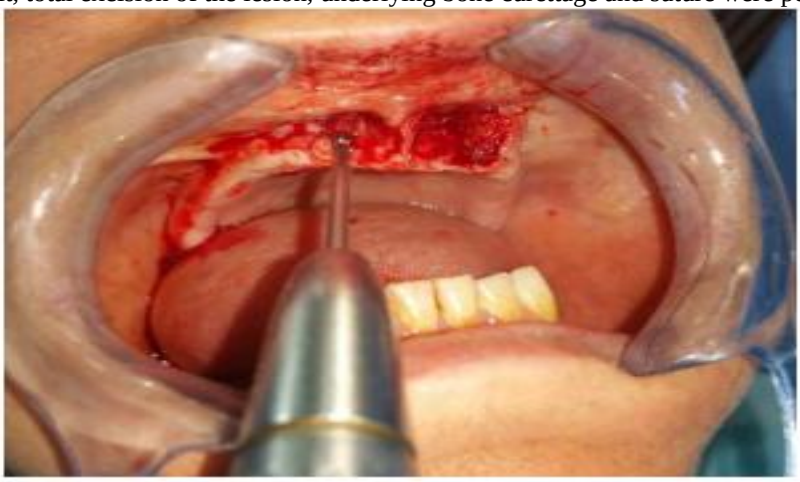

Fig 3 :Total excision of the lesion, underlying bone curettage

Histological examination confirmed that it is a giant cell peripheral granuloma (**Fig 4a et 4b**). We prescribed the patient a corticosteroid-based mouthwash (oral Betneval 0.5mg / kg / day) in use 3 times daily for 10 days and we noted the disappearance of the palatal lesion.

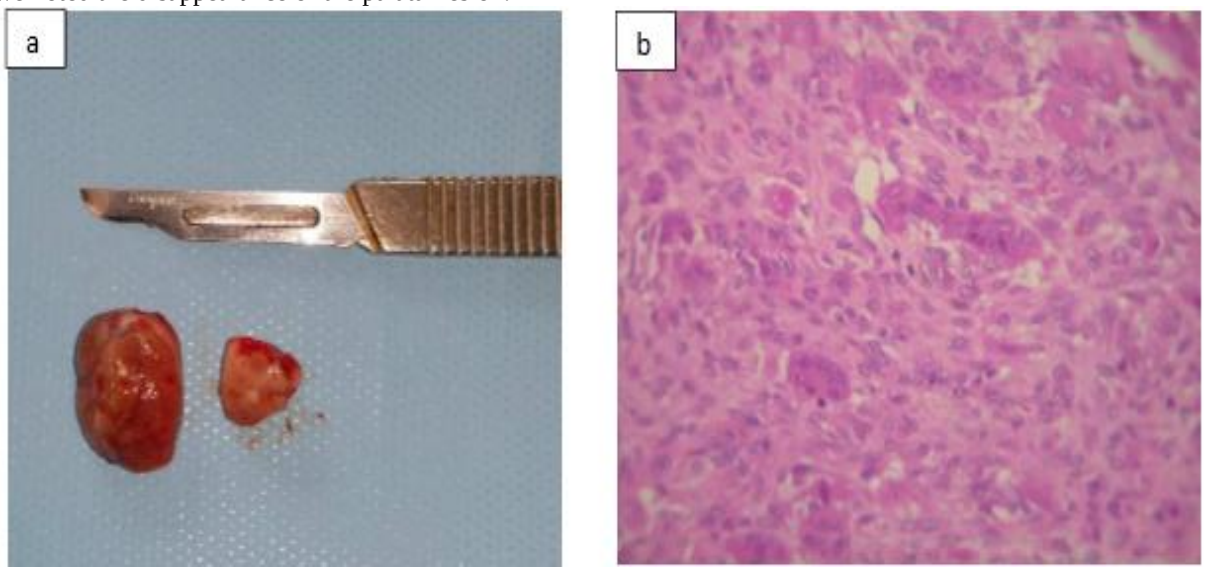

Fig 4: Macroscopic (a) and histological, (b) aspect of the lesion. Giant cell proliferation with pigmentation of hemosiderine in macrophages and fibrous stroma (HE x 250) can be observed

The patient presented for a follow-up examination1 month, 3 months, 6 monthspostoperatively. The surgical site appeared to behealingwell (**Fig. 5a, 5b, 5c, 5d**). There was no evidence of recurrence of the lesion, and the patient wasasymptomatic.

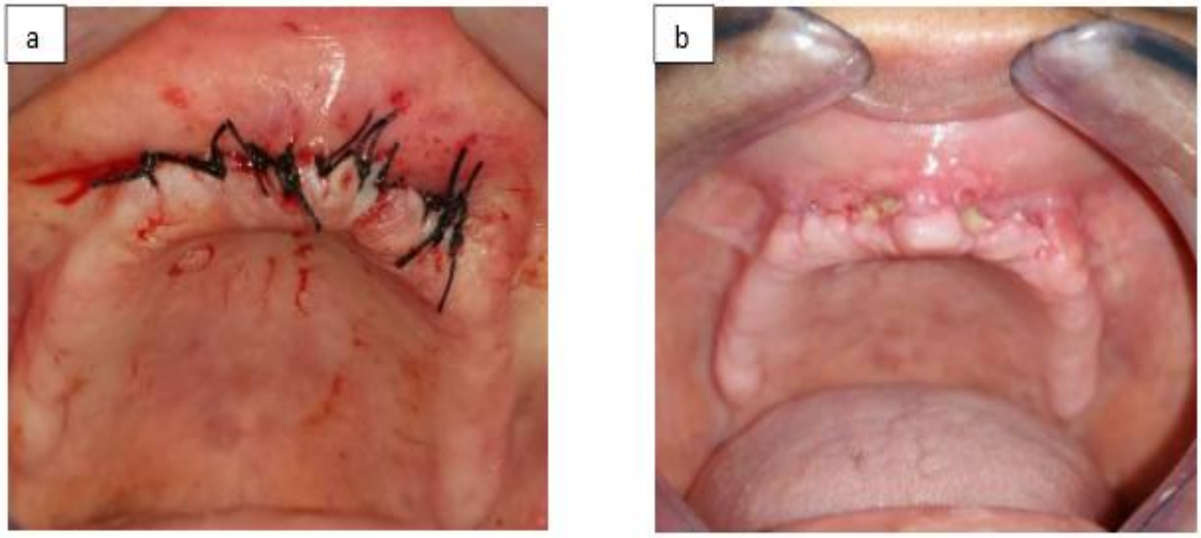

Fig 5 : a - Immediate postoperative view. b-1 months postoperative healing.

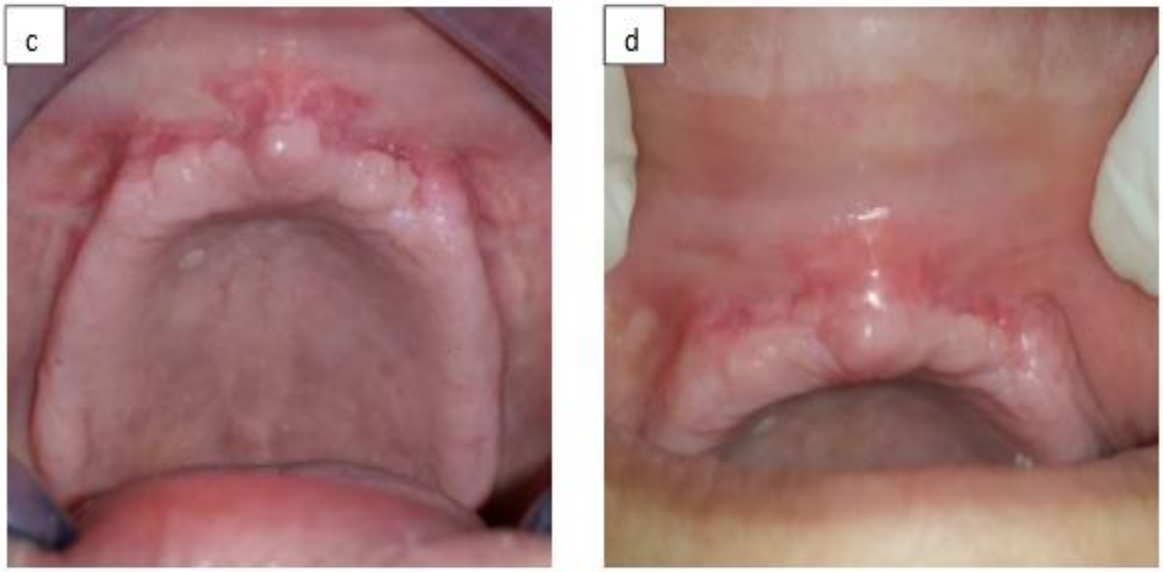

Fig 5 : c- 3 months post-operative healing and totally disappearance of the palatal lesion d- 6 months post-operatively with no evidence of recurrence

Currently, we continue to monitor the patient.

III. DISCUSSION

Giant cell peripheral granuloma (PGCG) is the most common lesion of giant oral cells. They appear as an extra-osseous re nodule consisting of multinucleated giant cells on the background of mononuclear stromal cells and extravasated red blood cells [4]. They may vary in appearance from smooth masses, regularly described to irregularly shaped, multi-lobulated protuberances with surface indentations. The consistency of the lesion ranges from mild reddish to firm pink depending on the age of the lesion, the more time passes, the more the lesion becomes fibrotic. [5]. In our case, the lesion was pink and firm without bleeding tendency.

Women over the age of 30 are the most affected. The most common seat is the gingiva and the edentulous alveolar ridge of the incisive-canine mandibular region. [6]. Nevertheless, the lesion can occur throughout life, with peaks of incidence during the mixed dental years and in the 30-40 age group. It is more common among women (60%). The mandible is slightly more often affected than the maxilla. The lesions can become important, some reaching 2cm. Although PGCG develops in the soft tissues, superficial or extensive osteolysis of the alveolar bone is observed radiologically. Sometimes it can be difficult to determine whether the mass is a peripheral lesion or a giant central granuloma that erodes through the cortical plate in soft gingival tissues [7]. There is no indicative clinical characteristic of distinguishing the giant cell granuloma lesion since both lesions share similar clinical and histological features.

In the case of our patient is near the fifties and the lesion is maxillary with a vestibular localization, and the lysis of the alveolar bone is superficial.

The etiology and the nature of PGCG (peripheral giant cells granuloma) remain unclear. In the past, several hypotheses have been proposed to explain the nature of giant multinucleated cells, including the explanation that they were osteoclasts resulting from the physiological resorption of teeth or a reaction to periosteal injury. There is strong evidence that these cells are osteoclasts because they have been shown to have receptors for calcitonin and have been able to search the bone in vitro.

In this case, our patient was wearing a prosthetic obsolete and ill adapted, not to mention its hormonal imbalance related to diabetes. This made us think of iatrogenic and hormonal causes Histologically, PGCG is composed of giant multinucleated giant cell nodules in a background of ovoid and fusiform, spindle-shaped mesenchymal cells and extravasated red blood cells. Giant cells may contain only a few nuclei or up to several dozens of them. Some of them are large vesicular nuclei; others have small pyknotic nuclei [8]. Stromal mononuclear cells (monocytes and macrophages) may participate in the formation of multinucleated giant cells through two members of the tumor necrosis factor (TNF) group: κβ nuclear factor ligand activating receptor (RANKL) and osteoprotegerin (OPG).[9]

The differential diagnosis of peripheral giant cell granuloma is made with lesions having very similar clinical and histopathological features such as CGCL (central giant cells lesions), pyogenic granuloma, fibroid peripheral ossification, fibrous hyperplasia, inflamed irritation fibroma, hemangioma, lymphangioma, amelanotic melanoma and metastatic tumors, or Kaposi's sarcoma, which may be in the form of a red or red-brown gingival tumor, sometimes associated with irregular underlying osteolysis.[6].

With respect to treatment, local surgical resection of the lesion is considered the most appropriate approach. However, relapses can occur due to inadequate surgical technique, especially when the surgeon is not effectively curating the periosteum underlying the lesion or small parts of the lesion remain in the tissue and then proliferate, which promotes recurrence. [10]

IV. CONCLUSION

PGCG is one of the many lesions encountered in oral surgery. However, this lesion is probably not presented as a true neoplasm, but its resemblance to a metastatic tumor can lead to uncertainty in the treatment plan. A careful radiographic and histopathological clinical study is necessary to differentiate these two entities. The most important is the differential diagnosis for eliminating giant cell diseases. Surgical treatment must be conducted with rigor, and a good follow-up must be planned to anticipate cases of recurrence.

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