

PERIPHERAL GIANT CELL GRANULOMA ASSOCIATED WITH A DENTAL IMPLANT

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Abstract

A 56-year-old female was referred for a mandibular lesion that had been slowly increasing in size over a six-month period. Intraoral examination revealed a reddish-purple nodule involving the attached vestibular gingiva around a machined surface dental implant that had been placed two years earlier in the mandibular left second premolar region (tooth #20). Another implant was also present in the mandibular left second molar region (18). The lesion was tender and bled easily upon tooth brushing. Radiographs showed an inadequate abutment angulation. The healing caps on the 1st and 2nd implants were loose and in contact with each other, not allowing optimal oral hygiene. An excisional biopsy of the mass resulted in the diagnosis of peripheral giant cell granuloma. The implants were gently curetted and scaled followed by abrasive paste cleaning. At the last follow-up, three years later, there was no recurrence.

Key words: dental implants, peripheral giant cell granuloma, plaque control, recurrence, complications

Introduction

The peripheral giant cell granuloma (PGCG) is a tumor-like pathologic condition arising on the gingiva or alveolar mucosa in dentate or edentate patients. It probably represents a reactive lesion caused by chronic local irritants or trauma rather than a true neoplasm, with a tendency to recur.

In this report, a PGCG associated with a Brånemark system implant placed in the mandibular left premolar area is described. To the best of the authors' knowledge, the association between a PGCG and a dental implant has not been previously described in the literature. The occurrence of such a lesion may be significant in the prognosis of dental implants.

Case Report

A 56 year-old female in good general health consulted and complained of a slightly tender rapidly growing mass in the left mandibular region in relation to an implant (Fig. 1). The lesion bled profusely upon brushing leading the patient to interrupt all hygiene procedures in the region. Past dental history revealed that her dentist following the failure of a conventional fixed prosthesis in the mandibular left premolar/molar area had placed three 3.75x10mm self-tapping MkII Brånemark system implants (Nobel Biocare, Kriens LU, Switzerland) 24 months earlier. Subsequently, at second stage surgery abutments and plastic healing caps were placed. According to the dentist, there was no sign of mucosal pathology at the time of surgery.

Clinical examination revealed a broad-based reddish mass on the buccal aspect of the implant, in the position of the mandibular left second premolar measuring about two cm in diameter (Fig. 1). Radiologically, relatively similar horizontal loss of alveolar bone height was apparent at all implants (Fig 2). Even though there was correct implant spacing, the

angulation was inappropriate. The necessity for corrective angulation abutments was evident, but this was not taken into account at second stage surgery. The healing caps on the 1st and 2nd implants were partially unscrewed and in contact with each other, allowing dental plaque to accumulate with calculus formation preventing optimal oral hygiene maintenance.

A provisional clinical diagnosis of pyogenic granuloma was made. The lesion was excised under local anesthesia and submitted for histologic examination. Gentle curettage and careful scaling followed by cleaning of the associated implants was undertaken. To improve the opportunity for plaque control, the plastic healing cap on the implant in the position of the second premolar was also removed and access to the screw-hole was filled with a temporary filling material.

Microscopic examination of the lesion revealed a sessile mass covered by a parakeratinized, stratified squamous epithelium containing the proliferation of osteoclast-like multinucleated giant cells lying in a richly vascular and cellular stroma composed of elongated or plump mesenchymal cells (Fig. 3). A band of fibrous tissue delineates the giant cell proliferation from the covering epithelium. A chronic inflammatory infiltrate, as well as hemorrhage and hemosiderin deposits were present in places. Mitotic figures were not observed.

Prosthetic rehabilitation was accomplished by using commercially available corrective abutments (Fig. 4). In Fig. 5a the impression copings have been seated on the implants; note the minimal space available between the implants. As a precaution to the risk of recurrence, a provisional temporary restoration intended for long term use was fabricated (Fig. 5b).

Particular care was taken to ensure accessibility for hygiene procedures and a perfect fit, albeit the temporary nature of the rehabilitation. To maintain optimal plaque control, the patient is now on a regular maintenance program. Follow-up radiographic examination at three years showed no recurrence of the lesion and a stabilized bone level (Fig. 6). On the three year follow-up clinical photograph it is apparent that the shoulder of the implant remained slightly supragingival (Fig. 7).

Discussion

Soft tissue complications in relation to dental implants are represented by gingival hyperplasia, mucositis and eventually, peri-implantitis.¹⁻⁴ In an evaluation of different possible complications, Tolman et al.⁵ found that gingivitis and gingival hyperplasia were the most common complications accounting for 3.9% to 21 % in their series of patients. In a retrospective study, over a 6-year span, Quirynen et al. evaluated 509 implants and 146 patients, and only two patients reported having developed gingival hyperplasia resulting from prolonged inflammation resulting from plaque accumulation⁶. Treatment consisted primarily in gingivectomy. Similarly, Tolman et al. remarked that the incidence of gingival hyperplasia diminished drastically after the first 3 years⁵. In a study evaluating ITI implants, Buser et al. found that at the 2-month follow-up, 31.8% of the implants had associated gingival hyperplasia needing minor gingivectomy⁷. These figures are consistent with the findings of Engquist et al. in which 23.6 % of the patients consulted their dentist for gingival hyperplasia. In fact, more than 60% of the visits concerning soft tissue complications were the result of hyperplasia⁸. Again the observation period was less than 5 years. In a recent literature review, Goodacre et al. reported that gingival inflammation or proliferation was the most frequent complication ranging from 1% to 32%⁹. In contrast, an earlier 2-year follow-up study¹⁰ showed a relatively low incidence of gingival hyperplasia (7.5%), and Schou et al.¹¹ ,

reviewing soft tissue reactions around dental implants, concluded that the prevalence and severity of inflammation around osseointegrated implants were similar to those observed in the natural dentition. In addition, they found that marginal gingival inflammation was less pronounced in patients strictly following short interval recalls.

Some anecdotal cases of gingival hyperplasia around implants in conjunction with phenytoin medication have also been reported^{12,13}. Treatment consisted of gingivectomy and reinforced hygiene.

An unexplained gingival hyperplasia was reported by Mitchell in two patients who seemed to have developed allergy to the titanium abutments after second stage surgery¹⁴. The resulting gingival proliferation was resistant to surgery, chemotherapeutic agents and to reinforced oral hygiene. The hyperplasia only resolved when the titanium abutments were replaced by custom made gold abutments.

In this report, a case of soft tissue complication involving a PGCG associated with a dental implant has been described. Such a situation has not been described before. (Medline search 1966-2002). This was also personally confirmed by Prof B. Friberg of the Brånemark Clinic, Göteborg, Sweden.

PGCG was first described as an independent entity by Jaffe¹⁵ in 1953, who classified it as a giant cell reparative granuloma. The term “reparative” has now been abandoned because there is no evidence defending the hypothesis that it occurs in response to a healing process¹⁶. PGCG is an uncommon benign reactive lesion of the oral mucosa affecting both sexes, with a slight predilection for women¹⁶. A possible hormonal influence has also been suggested¹⁷.

The peak prevalence is in the 5th - 6th decade of life, but about 1/3 of the cases occur in the first two decades.^{16,18} There is no age or racial preponderance. The most common site of occurrence is the incisor and canine regions of the mandible. The location is invariably a bone-supported tissue; attached gingiva or alveolar mucosa. Clinically, it presents as a smooth-surfaced, reddish-blue, sessile or pedunculated mass, with a firm consistency. Notwithstanding its usual asymptomatic nature, it can become tender or more rarely painful related to trauma. Periapical radiographic examination of PGCG in a tooth-bearing area may show superficial erosion or cupping of the cortical bone, some widening of the periodontal space and, on rare occasions root resorption.¹⁹ In an edentulous patient, the cortical bone may show a concave area of resorption.

The exact etiology of the lesion remains unclear, but it seems that local irritants such as dental calculus, foreign bodies and trauma to the gingiva may favor its development. In this case, there was poor implant angulation in a region of the edentulous mandible where it is customary to find only a narrow band of keratinized mucosa in conjunction with strong and mobile muscle insertions. Furthermore, the abutment covers were not completely screwed in place, thus leading to plaque and calculus accumulation and increased inflammation causing the patient to diminish her already insufficient oral hygiene in that region of her mouth.

Histologically, the PGCG consists of a non-encapsulated proliferation of spindle-shaped or round mesenchymal cells with oval, pale nuclei and with a moderate amount of eosinophilic cytoplasm. These cells are associated with a variable amount of collagen fibers. Mitotic figures are not unusual and may even be pronounced in lesions present in young patients. Occasionally, there may be a chronic inflammatory infiltrate intermingled with the mesenchymal cells or in surrounding fibrous tissues. A narrow band of fibrous tissue, usually

containing small sinusoidal spaces, often delineates the lesion from the surface epithelium. The latter is ulcerated in about half of the cases. Mixed throughout the stroma are numerous osteoclast-like, multinucleated giant cells containing varying numbers of pale vesicular nuclei with an eosinophilic cytoplasm. Immunohistochemistry has shown the giant cells to be similar to true osteoclasts.²⁰

The origin of the multinucleated cells is still unknown, but they are assumed to arise from syncytial fusion of mononuclear preosteoclasts of bone marrow origin. Blood vessels in the lesional stroma show plump endothelial cell nuclei. Scattered extravasated red cells and hemosiderin deposits may be observed in areas of old hemorrhage. Metaplastic or osteoid formation, as well as dystrophic calcifications, may be present, usually in the lower third of the lesion. PGCG is indistinguishable from the rare extraosseous brown tumor of hyperparathyroidism. When PGCG is diagnosed, the clinician should evaluate the serum calcium and phosphorus levels to rule out hyperparathyroidism.^{21,22}

In conclusion, optimal implant placement and correct prosthetic reconstruction permitting adequate oral hygiene is important not only for prosthetic reasons but also to prevent inflammatory or reactive lesions. Careful monitoring during the healing phase is essential to detect any anomaly. Although occurrence of PGCG seems to be rather unusual, early detection and treatment is important not to jeopardize implant survival.

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Figures legend

Fig. 1 : Erythematous broad mass located on the buccal aspect of the implant in the mandibular left premolar region.

Fig. 2 : Radiograph showing horizontal bone loss associated with all implants.

Fig. 3 : Medium power view showing a well delimited focus of multinucleated cells, giant cells, stromal cells and extravasated red cells. HeE, x20

Fig. 4 : View of the corrective abutments.

Fig. 5a : Minimal space is evident between the impression copings (mirror view).

Fig. 5b : Clinical view of the temporary restorations 4 months after surgery.

Fig. 6 : Follow-up radiograph at 3 years demonstrating a stable bone level.

Fig. 7 : 3 year follow-up view of the surgical area.