

Peripheral Giant Cell Granuloma Associated with a Dental Implant

Mark Bischof, Dr Med Dent¹/Rabah Nedir, Dr Med Dent¹/Tommaso Lombardi, MD, Dr Med Dent, PD²

A 56-year-old woman was referred for the treatment of a mandibular lesion that had been slowly increasing in size over a 6-month period. Intraoral examination revealed a reddish-purple nodule involving the attached vestibular gingiva around a machined-surface dental implant that had been placed 2 years earlier in the mandibular left second premolar region. Another implant had been placed in the mandibular left second molar region. The lesion was tender and bled easily upon tooth brushing. Radiographs showed inadequate abutment angulation. The healing caps on these 2 implants were loose and in contact with each other, preventing optimal oral hygiene. An excisional biopsy of the mass resulted in the diagnosis of peripheral giant cell granuloma. After the implants were gently curetted and scaled, they were cleaned using abrasive paste. At the last follow-up, 3 years later, there was no recurrence. INT J ORAL MAXILLOFAC IMPLANTS 2004;19:295–299

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

The peripheral giant cell granuloma (PGCG) is a tumorlike pathologic condition arising on the gingiva or alveolar mucosa of either dentate or edentulous jaws. Considering its tendency to recur, it is probably a reactive lesion caused by chronic local irritants or trauma rather than a true neoplasm.

In this report, a PGCG associated with a Brånemark System implant (Nobel Biocare, Göteborg, Sweden) 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 determining the prognosis of dental implants.

¹Assistant Professor, Division of Stomatology and Oral Surgery, School of Dental Medicine, University of Geneva, Switzerland; Clinical Director, Clinique Dentaire de Chauderon, Lausanne, Switzerland

²Senior Lecturer, Histopathology Laboratory, Division of Stomatology and Oral Surgery, School of Dental Medicine, University of Geneva, Switzerland

Correspondence to: Dr Mark Bischof, Division of Stomatology and Oral Surgery, 19, Rue Barthélemy-Menn, CH-1211 Genève 4, Switzerland. Fax: +41 22 382 9499. E-mail: markbischof@bluewin.ch

CASE REPORT

A 56-year-old woman in good general health presented with 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. Her dental history revealed that, following the failure of a conventional fixed prosthesis, her dental practitioner had placed three 3.75 × 18-mm self-tapping Mk II Brånemark System implants in the mandibular left premolar/ molar area 24 months earlier. Abutments and plastic healing caps were placed at second-stage surgery. According to the practitioner, 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 approximately 2 cm in diameter (Fig 1). Radiologically, horizontal loss of alveolar bone height appeared relatively similar at all implants (Fig 2). Although the implants had been spaced correctly, the angulation was inappropriate. The necessity for corrective angulated abutments, although evident, had not been taken into account at second-stage surgery.



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.

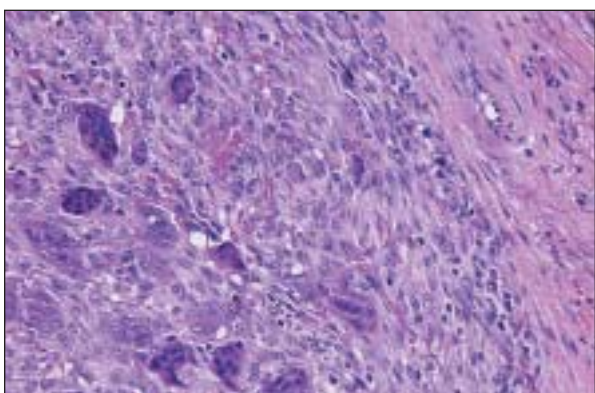


Fig 3 Medium-power view showing a well delimited focus of multinucleated giant cells, stromal cells, and extravasated red blood cells (hematoxylin and eosin; $\times 20$).



Fig 4 View of the corrective abutments.

The healing caps on 2 of the implants were partially unscrewed and in contact with each other, allowing dental plaque to accumulate. Calculus formed, 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 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 a proliferation of osteoclastlike 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 delineated the giant cell pro-

liferation from the covering epithelium. A chronic inflammatory infiltrate, hemorrhage, and hemosiderin deposits were present in places. Mitotic figures were not observed.

Prosthetic rehabilitation was accomplished using commercially available corrective abutments (Fig 4). The impression copings were seated on the implants, and the minimal space available between the implants was noted (Fig 5a). As a precaution against 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 satisfactory clinical fit, despite the temporary nature of restoration. To maintain optimal plaque control, the patient was placed on a regular maintenance program. Follow-up radiographic examination at 3 years showed a stabilized bone level and no recurrence of the lesion (Fig 6a). The shoulder of the implant remained slightly supragingival (Fig 6b). There was no evidence of recurrence of the PGCG.



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



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

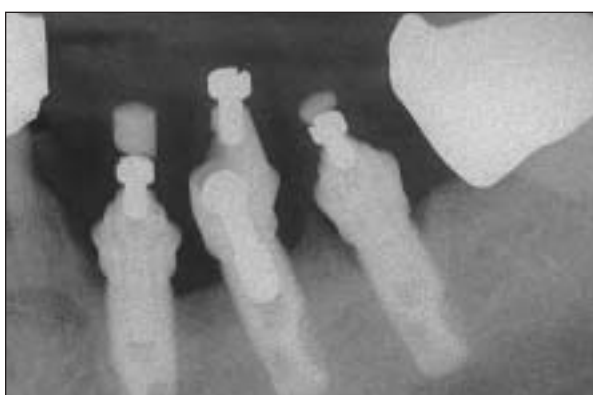


Fig 6a Follow-up radiograph at 3 years demonstrating a stable bone level.



Fig 6b Clinical view at 3-year follow-up.

DISCUSSION

Soft tissue complications related to dental implants include gingival hyperplasia, mucositis, and eventually, peri-implantitis.¹⁻⁴ In an evaluation of different possible complications, Tolman and Laney⁵ found that gingivitis and gingival hyperplasia were the most common complications, experienced by 3.9% to 21% of the patients in their study population. However, they noted that the incidence of gingival hyperplasia diminished drastically after the first 3 years. In a retrospective study, Quirynen and colleagues evaluated 509 implants and 146 patients over a 6-year period.⁶ Only 2 patients developed gingival hyperplasia resulting from prolonged inflammation resulting from plaque accumulation. Treatment consisted primarily of gingivectomy. In a study evaluating ITI implants, Buser and coworkers found that 31.8% of the implants had associated gingival hyperplasia needing minor gingivectomy at the 2-month follow-up.⁷ These figures are consis-

istent with the finding of Engquist and associates that 23.6% of patients consulted their dental practitioner for gingival hyperplasia.⁸ In their study, more than 60% of the visits concerning soft tissue complications were the result of hyperplasia. Again, the observation period was less than 5 years. In a recent literature review, Goodacre and coworkers 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 and associates,¹¹ reviewing soft tissue reactions around dental implants, concluded that the prevalence and severity of inflammation around osseointegrated implants were similar to those observed around 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 and colleagues in 2 patients who seemed to have developed an allergy to the titanium abutments after second-stage surgery.¹⁴ The resulting gingival proliferation was resistant to surgery, chemotherapeutic agents, and reinforced oral hygiene. The hyperplasia ceased only 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 process.¹⁶ PGCG is an uncommon benign reactive lesion of the oral mucosa affecting both sexes, with a slight predilection for women.¹⁶ It has been suggested that certain hormones (ie, estrogen and progesterone) influence the development of PGCG. This hormonal influence may account for the predilection for women.¹⁷ Prevalence peaks in the fifth or sixth decade of life, but about a third of the cases occur in the first 2 decades.^{16,18} There is no age or racial preponderance. The most common sites of occurrence are the incisor and canine regions of the mandible. The location is invariably a bone-supported tissue such as 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, pain-sensitive 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 PGCG remains unclear, but it seems that local irritants such as dental calculus, foreign bodies, and trauma to the gingiva may increase the likelihood of 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 and causing the patient to diminish an already insufficient oral hygiene in that region of her mouth.

Histologically, a PGCG consists of a nonencapsulated proliferation of spindle-shaped or round mesenchymal cells with pale, oval 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 osteoclastlike, multinucleated giant cells containing varying numbers of pale vesicular nuclei and 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 the syncytial fusion of mononuclear preosteoclasts originating in the bone marrow. Blood vessels in the lesional stroma show plump endothelial cell nuclei. Scattered extravasated red blood cells and hemosiderin deposits may be observed in areas of old hemorrhage. Metaplastic or osteoid formation, as well as dystrophic calcification, may occur, usually in the lower third of the lesion. PGCG is indistinguishable from the rare, extrasosseous brown tumor of hyperparathyroidism. When PGCG is diagnosed, the clinician should evaluate the patient's serum calcium and phosphorus levels to rule out hyperparathyroidism.^{21,22}

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 are important for implant survival.

ACKNOWLEDGMENTS

The authors wish thank Prof U. Belser for prosthetic technical advice, Dr J. P. Bernard, PD, for his guidance in training, and Prof J. Samson for encouragement during the preparation of the manuscript.

REFERENCES

- Lang NP, Wilson TG, Corbet EF. Biological complications with dental implants: Their prevention, diagnosis and treatment. *Clin Oral Implants Res* 2000;11:146–155.
- Adell R, Lekholm U, Rockler B, Brånemark P-I. A 15-year study of osseointegrated implants in the treatment of the edentulous jaw. *Int J Oral Surg* 1981;10:387–416.
- Worthington P, Bolender CL, Taylor TD. The Swedish system of osseointegrated implants: Problems and complications encountered during a 4-year period. *Int J Oral Maxillofac Implants* 1987;2:77–84.
- Zarb GA, Schmitt A. The longitudinal clinical effectiveness of osseointegrated dental implants: The Toronto study. Part III: Problems and complications encountered. *J Prosthet Dent* 1990;64:185–194.
- Tolman DE, Laney WR. Tissue-integrated prosthesis complications. *Int J Oral Maxillofac Implants* 1992;7:477–484.
- Quirynen M, Naert I, van Steenberghe D, Dekeyser C, Calens A. Periodontal aspects of osseointegrated fixtures supporting a partial bridge. An up to 6-years retrospective study. *J Clin Periodontol* 1992;19:118–126.
- Buser D, Weber HP, Brägger U. The treatment of partially edentulous patients with ITI hollow screw implants: Presurgical evaluation and surgical procedures. *Int J Oral Maxillofac Implants* 1990;5:165–174.
- Engquist B, Bergendahl T, Kallus T, Linden U. A retrospective multicenter evaluation of osseointegrated implants supporting overdentures. *Int J Oral Maxillofac Implants* 1988;3:129–134.
- Goodacre CJ, Kan JYK, Rungcharassaeng K. Clinical complications of osseointegrated implants. *J Prosthet Dent* 1999;81:537–552.
- Gotfredsen K, Holm B, Sewerin I, et al. Marginal tissue response adjacent to Astra dental implants supporting overdentures in the mandible. A 2-year follow-up study. *Clin Oral Implants Res* 1993;4:83–89.
- Schou S, Holmstrup P, Hjørting-Hansen E, Lang NP. Plaque-induced marginal tissue reactions of osseointegrated oral implants: A review of the literature. *Clin Oral Implants Res* 1992;3:149–161.
- Chee WL, Jansen CE. Phenytoin hyperplasia occurring in relation to titanium implants: A clinical report. *Int J Oral Maxillofac Implants* 1994;9:107–109.
- Bredfeldt GW. Phenytoin-induced hyperplasia found in edentulous patients. *J Am Dent Assoc* 1992;123:61–64.
- Mitchell DL, Synnott SA, Vandercreek JA. Tissue reaction involving an intraoral skin graft and CP titanium abutments: A clinical report. *Int J Oral Maxillofac Implants* 1990;5:79–84.
- Jaffe HL. Giant-cell reparative granuloma. *J Oral Surg* 1953;6:159–175.
- Katsikeris N, Kakarantza-Angelopoulou E, Angelopoulos AP. Peripheral giant cell granuloma: Clinicopathologic study of 224 new cases and review of 956 reported cases. *Int J Oral Maxillofac Surg* 1988;27:787–791.
- Whitaker SB, Bouquot JE. Identification and semi-quantification of estrogen and progesterone receptors in peripheral giant cell lesions of the jaws. *J Periodontol* 1994;65:280–283.
- Giansanti JS, Waldron CA. Peripheral giant cell granuloma: Review of 720 cases. *J Oral Surg* 1969;27:787–791.
- Nedir R, Lombardi T, Samson J. Recurrent peripheral giant cell granuloma associated with cervical resorption. *J Periodontol* 1997;68:381–384.
- Bonetti F, Pelosi G, Martignoni G, et al. Peripheral giant cell granuloma: Evidence for osteoclastic differentiation. *Oral Surg Oral Med Oral Pathol* 1990;79:471–475.
- Smith BR, Fowler CB, Svane TJ. Primary hyperparathyroidism presenting as a peripheral giant cell granuloma. *J Oral Maxillofac Surg* 1988;46:65–69.
- Burkes EJ, White RP. A peripheral giant cell granuloma manifestation of primary hyperparathyroidism: Report of a case. *J Am Dent Assoc* 1989;118:62–64.