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Peripheral Giant Cell Granuloma Associated With Residual Cement and Periimplantitis: A Case Report

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Case Report

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ABSTRACT

Residual cement in cement retained restorations, poses a problem which cause peri-implant disease. Peripheral giant cell granuloma (PGCG) is a reactive and exophytic lesion occurring on the gingiva and alveolar ridge. Although PGCG is the most common giant cell lesion of the jaws, there is little data in the literature regarding the prevalence of reactive lesions associated with dental implants. The purpose of this paper is to report a rare case which a PGCG was found in association with excess cement around a dental implant and to discuss its etiology and management. After completion of phase 1 periodontal therapy, the lesion was excised surgically. The residual cement that were detected on the buccal aspect of the implant were removed, bony defect was debrided and platelet rich fibrin membrane placed around the implant. The fixed prosthesis was cemented by paying attention to remove residual cement. No evidence of recurrence and complications were seen over 18 months of follow up.

INTRODUCTION

Implant supported fixed restorations can be cemented or attached with screws to implants. Due to its relative simplicity, improved esthetics, easier control of occlusion and economy compared to screw-retained prosthesis, cement retained dental restorations have become popular in dental practice [1,2]. Residual cement in cement retained restorations, poses a problem, by promoting the formation of a biofilm [3], which cause periimplant disease [4]. In a previous study, it was found that 81% of the implants restored with cement-retained restorations with sign of periimplantitis had residual cements in the subgingival spaces [1].

Peripheral giant cell granuloma (PGCG), is a reactive and exophytic lesion occurring on the gingiva and alveolar ridge [5,6]. Although PGCG is the most common giant cell lesion of the jaws, there is little data in the literature regarding the prevalence of reactive lesions associated with dental implants [7]. Inadequate restorations, food impaction, complicated dental extractions, orthodontic treatment, ill-fitting dentures, plaque and calculus have been associated with PGCG [5,8,9].

The purpose of this paper is to report a case which a PGCG was found in association with excess cement around a dental implant and to discuss its etiology and management.

CASE REPORT

A 52-year-old woman referred to Baskent University-Faculty of Dentistry, Department of Periodontology with complains of bleeding and overgrowth of the gingiva at the maxillary left premolar area close to an implant restoration. She had received two dental implants 9 months ago. She was otherwise healthy, with an unremarkable medical history. Intraoral examination revealed a 12x5 mm painless lesion with a non-ulcerated surface, red to bluish color, involving the attached gingiva at the buccal aspect of

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the implant. Clinical and radiographic assessment revealed increased probing depths and crestal bone loss around the implant respectively **(Figure 1).**

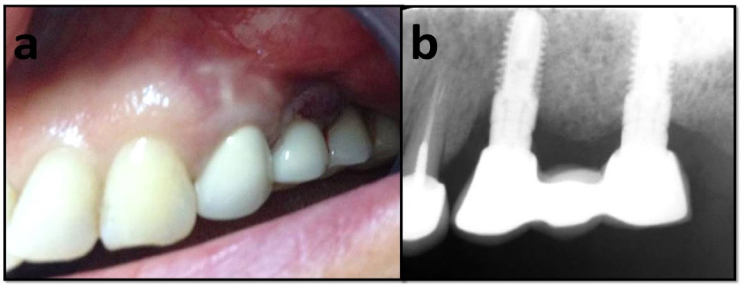

Figure 1. A. Intraoral view showing the exophytic red mass on the buccal aspect associated with the implant. **B.** Periapical radiograph of the lesion. Note the vertical crestal bone loss around the implant.

The patient was informed of all risks and benefits of the procedure and signed an informed consent. The restoration was removed to allow adequate access. Dental implants were clinically osseointegrated and stable. Four weeks after phase I periodontal therapy, although the hyperemic appearance and bleeding of the lesion were reduced, the size of the lesion did not change. Therefore the patient was scheduled for surgery. After the lesion was excised, buccal and to access to the defect both buccal and lingual incisions were made to access the defect and mucoperiosteal flaps were reflected, residual cement were detected on the buccal aspect of the implant. Residual cement was removed, the bony defect and exposed implant threads were decontaminated with tetracycline hydrochloride (HCl) solution (1 g) in 20 mL of sterile saline for 3 minutes. Following decontamination, the implant surface was washed with sterile saline to remove all remnants of the tetracycline HCl. Just prior to surgery, intravenous blood was taken to obtain Platelet Rich Fibrin membrane according to Choukroun's protocol [10] and placed around the implant. The primary closure was obtained by 4-0 silk sutures and periodontal dressing was placed over the surgical area. Sutures were removed after 7 days and healing was uneventful.

HISTOPATHOLOGIC ANALYSES

The specimen was grayish- white, irregularly shaped had firm consistency and measuring about 12x5 mm. Microscopic examination revealed the proliferation numerous giant cells admixed with endothelial lined blood capillaries, extravasated red blood cells in stroma. The connective tissue stroma was highly cellular, consisting of mesencymal cells. The giant cells showed various shapes and sizes and were containing 8-15 nuclei. The surface epithelium was hyperplastic and also seperated with a zone of connective tissue stroma from the lesion. With these histopathological findings, it was diagnosed as PGCG **(Figure 2).**

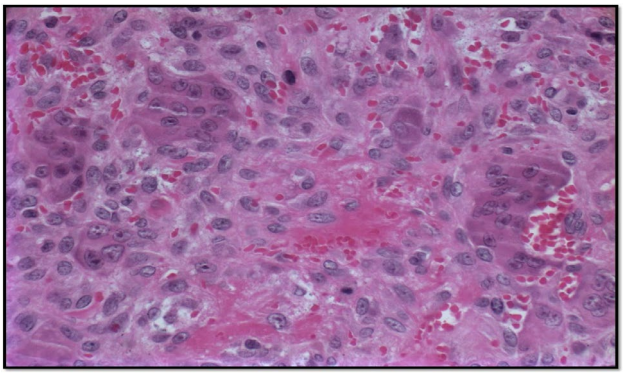

Figure 2. Histopathologic appearance of the excised specimen. Osteoclast like multinucleated giant cells admixed with uniform mesenchymal cells in a vascular background. Abundant hemorrhagic foci are evident (X400 HE stain).

Eight weeks after periodontal surgery, the fixed prosthesis was cemented using glass ionomer cement (AquaCem; Dentsply DeTrey, Konstanz, Germany) by paying attention to remove residual cement. No evidence of recurrence was seen over 18 months of follow up.

DISCUSSION

PGCG is considered to be a reactive lesion caused by local irritation or trauma. Complicated tooth extraction, ill-fitting dentures, inadequate restorations are primary causes of trauma [8,11,12].

Clinically and histologically the lesion is similar to the classical appearance of PGCG. The pathogenesis of PGCG in peri-implant soft tissue is not clear because there is only a few numbers of reported cases regarding the association between dental implants and the development of PGCG [8,13,14] **(Figure 3).**

In literature, there are some theories about the etiology of the PGCG. In one theory, trauma triggered by the placement of a dental implant cause alveolar bone resorption around the implant and the eventual exposure of the implant surface became a

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source of chronic irritation that could encourage the formation of PGCG [13]. This theory of traumatic reaction around some dental implants is not conclusive for each peri-implant bone defects and also the reason of why all dental implants do not display lesions such as PGCG cannot be clarified with this theory.

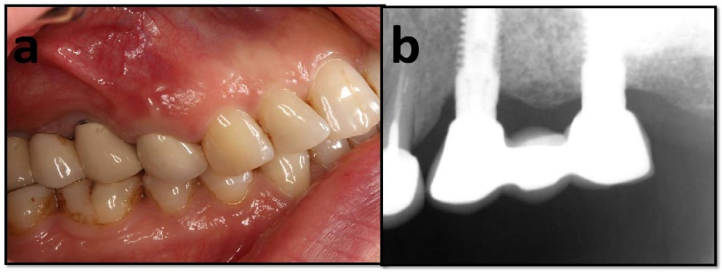

Figure 3. A. Clinical photograph taken 18 months after the surgery. **B.** Periapical radiograph 18 months after the surgery.

Another theory could be explained from a wound healing perspective. Blood protein deposition on a biomaterial surface is described as provisional matrix formation. The provisional matrix furnishes structural, biochemical, and cellular components to the processes of wound healing and foreign body reaction. The presence of mitogens, chemoattractants, cytokines, growth factors, and other bioactive agents within the provisional matrix provides for a rich milieu of activating and inhibiting substances capable of modulating macrophage activity, along with the proliferation and activation of other cell populations in the inflammatory and wound healing responses [15]. Excess cement could activate macrophage activity by this mechanism **(Figure 4).**

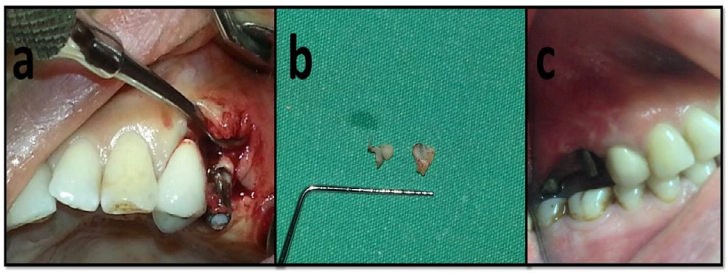

Figure 4. A. Intraoperative view showing the residual excess cement remnants on the buccal aspect of the implant. **B.** Removed cement particles from the buccal aspect of the implant. C. Clinical photograph taken 7 days after the surgery.

Removing all irritating factors and surgical excision of the lesion from entire base of bone were recommended for the treatment PGCG. In some previous reported cases [8,13] the involved implants were removed in order to provide adequate access to the lesion and to prevent the recurrence. It is concluded that the decision to remove an involved implant should be based on the judgment of the practitioner balanced with the risk of recurrence of the lesion [8,13].

The etiology of the PGCG in this case could be related to residual cement detected on the buccal aspect of the implant. Therefore after completion of periodontal therapy the fixed prosthesis was cemented by giving importance to removal of excess cement to prevent a recurrence of PGCG. The role of residual cement in the etiology of peri-implant disease is not fully understood but it is believed to be related to additional bacterial colonization of the foreign material due to its rough surface [1]. Based on these findings it is obvious that removal of residual cement from implants is indispensable for the maintenance of the periodontal health. Therefore in all cemented cases, diligence should be paid to removing residual cement at the time of placement of the fixed partial denture and screw retained fixed partial dentures may be indicated in situations where cement margins will be located under the gingiva and excess cement removal will be suspicious [1]. Furthermore, in cementing to teeth, the aim is to prevent bacterial leakage to prevent decay. In implants, the cement is used as a luting agent so excess cement should be removed before it sets. Several preventive techniques were adopted to avoid excess cement like using a radiopaque type of cement, placing rubber dam to prevent subgingival flow or fabrication of an abutment replica to try and remove cement excess [16].

Linkevicius et al. [17] and Agar et al. [18] have demonstrated that if restoration margins are placed more than 2 mm subgingivally, excess cement around implant restorations could not be fully removed. In order to avoid this situation, margin location should be carefully evaluated before cementation.

Implants and teeth have many similarities and differences; the junctional epithelium and connective tissue attachment around natural teeth insert perpendicularly, this tends to limit and compartmentalize the flow of excess cement. This is in contrast to the epithelium and connective tissue around dental implants, where the connective tissue runs parallel and does not insert into the body of the implant. As a result, the flow of cement is not restricted and easily migrates apically [16].

This case illustrates the development of peri-implant disease and PGCG and its management. In order to ensure long-term stability and predictability and reduce the incidence of peri-implant disease related to implant-retained restorations, clinicians must have a thorough understanding of the biologic differences between restorations on natural teeth and dental implants and techniques to prevent complications.

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