CLINICAL ORAL IMPLANTS RESEARCH

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Does residual cement around implant-supported restorations cause peri-implant disease? A retrospective case analysis

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Key words: cement excess, cement-retained implant restorations, peri-implantitis, periodontally compromised patients, subgingival margins

Abstract

Objectives: The purpose of this study was to determine the relationship between patients with a history of periodontitis and development of cement-related peri-implant disease.

Materials and methods: Seventy-seven patients with 129 implants for this retrospective analysis were selected from completed implant cases that were scheduled for regular maintenance or had experienced mechanical or biological complications between years 2006 and 2011 in private practice. Implants with extracoronal residual cement and implants without cement remnants were analyzed. The selected cases were further divided into two groups – implants in patients with history of periodontitis (1) and implants in periodontitis-free individuals (2). The selection of these groups was made on the basis of treatment history and orthopantomograph. As a control group, a set of 238 screw-retained implant restorations, delivered to 66 patients during the same period of time was examined. The incidence of peri-implant disease among implants in all groups was calculated.

Results: Peri-implant disease was evident in 62 of 73 implants with cement remnants (85%). All implants in group 1 developed peri-implantitis – 4 early and 35 delayed disease cases. In the periodontally healthy group, 20 of 31 implants were diagnosed with peri-implant mucositis, 3 implants had early peri-implantitis, and 11 implants with cement remnants did not develop biological complications. In the group of implants without cement remnants, peri-implant disease was diagnosed in 17 of 56 cases (30%). In contrast, only two occurrences of peri-implant disease were registered in the control group of screw-retained restorations (1.08%).

Conclusions: Implants with cement remnants in patients with history of periodontitis may be more likely to develop peri-implantitis, compared with patients without history of periodontal infection.

Peri-implantitis-associated bone loss occurring after delivery of implant restorations remains one of the most common biological complications. Systematic literature reviews have shown that 8.6–14.4% of restored implants are prone to develop peri-implantitis within a period of 5 years (Berglundh et al. 2002; Pjetursson et al. 2004). A recent study by Fransson and co-workers showed that in certain populations, almost 40% of implants suffer from peri-implant infection-related bone loss (Fransson et al. 2009). Furthermore, it seems that despite meticulous treatment efforts, peri-implantitis may still lead to implant loss (Schou et al. 2004; Heitz-Mayfield and Lang 2004).

There is strong evidence that bacteria are the key etiological factor in peri-implantitis development (Heitz-Mayfield and Lang 2010). However, predisposing risk factors, such as a history of periodontal infection (Karoussis et al. 2003), smoking, genetics (Gruica et al. 2004), poor oral hygiene (Serino & Strom 2009) are broadly discussed in literature, but there is no consensus as to which factor is more significant. A recent report by Wilson has shown that residual cement may act as one of the predisposing factor for delayed peri-implantitis development (Wilson 2009). The study suggested that about 81% of the implants restored with cement-retained restorations with clinical and radiological signs of peri-implantitis had extracoronal residual cement. Another study by Linkevicius et al. showed that the use of cement-retained implant restorations often results in leaving

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cement in peri-implant tissues (Linkevicius et al. 2011). The most intriguing finding of Wilson study was that the peri-implant tissues showed varied reaction to extracoronal residual cement. The manifestation of the disease occurred from as early as 4 months to 9 years after delivery. It has been showed that cement-related bone loss may occur very quickly; in some cases, it is delayed, but a number of patients may be completely resistant to peri-implantitis development. The reasons for these differences are unknown and are still to be identified.

The aim of this retrospective study was to determine the relationship between patients with a history of periodontitis and the development of cement-related peri-implant disease.

Materials and methods

This study selected individuals from a private practice who had cement-retained implant restorations and were scheduled for regular implant maintenance or were consulted because of a complication. Mechanical complications included all incidents (extensive porcelain chipping, framework fracture, abutment screw loosening) that required the removal of the restoration. Biological complications included peri-implantitis and peri-implant mucositis. Peri-implantitis was diagnosed if an implant had bleeding on probing, pocket depths 6 mm or more and progressive crestal bone loss exceeding 1.5 mm after first year of service (Fig. 1a and b). Peri-implant mucositis was defined as a swelling, bleeding on probing, and increased probing depths of peri-implant tissues without evident progressive bone loss radiographically, which does not exceed acceptable norm, established by Albrektsson et al. (1986) (Fig. 2a and b).

Radiographic images were taken with RVG Windows Trophy 5.0 (Trophy Radiologie Inc, Paris, France) using a paralleling technique with Rinn-like film holder in high-resolution mode. To define the extent of crestal bone loss, control radiographic images were compared to radiographs taken at the time of complication. To confirm diagnosis, peri-implant tissues were probed with 1.0 mm marked periodontal probe (Hu-Friedy, Chicago, IL, USA) and bleeding and suppuration (if present) were recorded.

In the case of mechanical complication, the implant restoration was removed by perforating occlusal/palatinal surfaces to gain access to the abutment screw. The retrieved abutment-restoration complex and the peri-implant tissues were inspected for excess cement (Fig. 3). If a biological complication was registered, two treatment options were considered. First, the implant restoration was attempted to remove to access the peri-implant sulcus for cement remnants inspection. If removal of the restorations was impossible, the flap was raised to gain visibility of the implant site (Fig. 4). Whether extracoronal cement was present or not, the area was debrided, irrigated with 0.12% chlorhexidine solution and sutured.

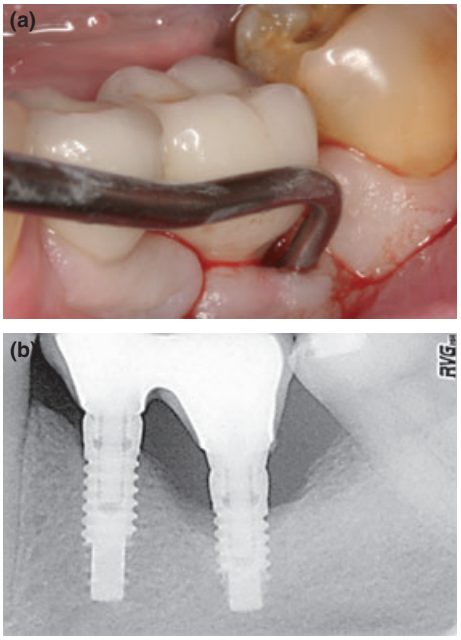

Fig. 1. (a)Probing of an implant with peri-implantitis; (b) Radiographic evidence of progressive bone loss.

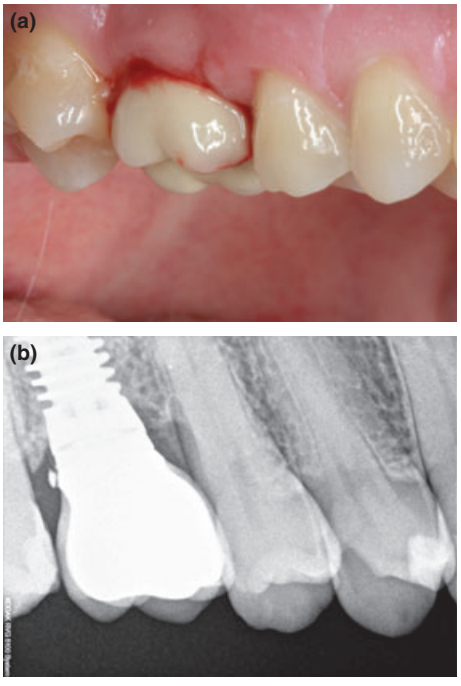

Fig. 2. (a) Bleeding and swelling of peri-implant tissues around implant restoration; (b) Radiographic image of peri-mucositis. Crestal bone loss does not exceed acceptable norms.

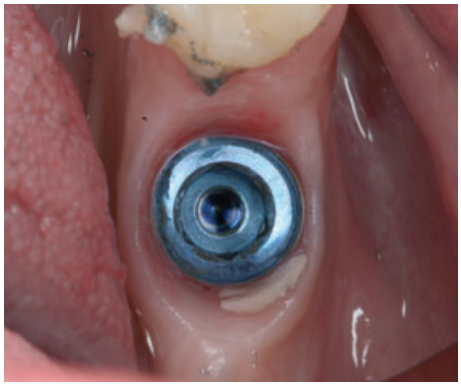

Fig. 3. Peri-implant tissues with cement remnants after removal of fractured restoration.

Patients' treatment charts were analyzed for presence or absence of periodontal disease and accordingly divided into two groups. If patient had Community Periodontal Index for Treatment Needs (CPITN) (Ainamo et al. 1982) registered TN 2 or more, generalized bone loss diagnosed from panoramic radiographs, and received surgical periodontal treatment before implant placement, the patient was regarded as periodontally compromised (with history of periodontitis) (Fig. 5); if the case history did not reveal periodontal treatment (except routine oral

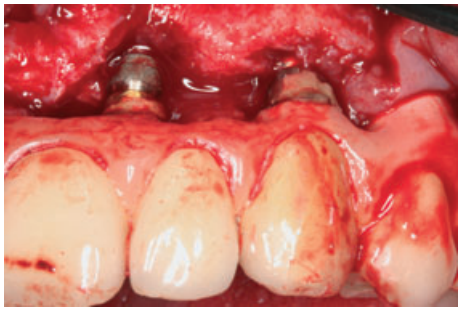

Fig. 4. Cleaning of cement from implants with delayed peri-implantitis without removing of the restoration.

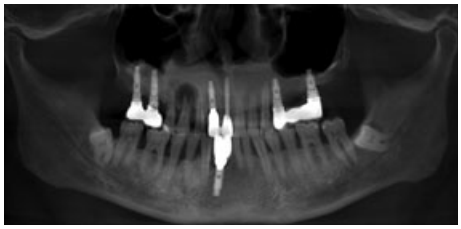

Fig. 5. Panoramic image of patient with history of periodontitis.

hygiene), CPITN TN 1 or less and pre-treatment panoramic radiographs showed crestal bone levels within the norm, the patient was considered periodontally healthy (without history of periodontal infection) (Fig. 6).

Over the 6-year period from 2006 till 2011, 77 patients (32 men and 45 women, mean age of 47.3 years) addressed the clinic with a condition allowing them to be included in the study. The sample size of 129 implants comprised of 32 implant restorations with mechanical failures (24.8%) and 97 implants affected by biological complications (75.2%). Implants were analyzed in 77 patients – 35 with history of periodontitis and 42 with no history of periodontal infection. The types of restorations are listed in Table 1. All included implants were divided into cases with cement remnants and cement-free implants. The incidence of peri-implant disease (peri-implantitis and peri-implant mucositis) in all implant groups was determined. The time from the delivery of the restorations until the diagnosis of the complication was calculated in months. If biological complication occurred within 6 months after cementation, the case was regarded as an early peri-implantitis and these implants were excluded from analysis of delayed peri-implant disease cases (Fig. 7). The definition of early peri-implant disease was detected according to available case reports in the literature (Pauletto et al. 1999; Gapski et al. 2008). The type of cement and prosthetic abutment (standard or individual) used for cementation of restorations were recorded.

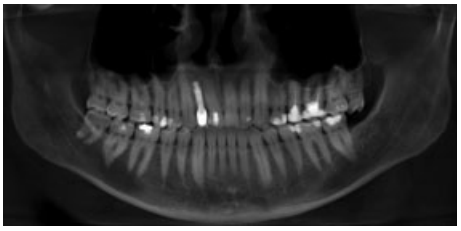

Fig. 6. Panoramic image of periodontally healthy patient.

Table 1. **Restoration type of implants included into the study**

Restoration	
Single crown	43
2 SC	13
3 SC	4
3 unit FPD	11
5 unit FPD	2
11 unit FPD	2
12 unit FPD	2
13 unit FPD	1
Total	78

FPD, fixed partial denture; SC, splinted crowns.

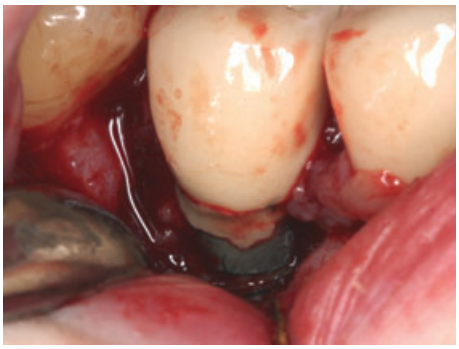

Fig. 7. Early peri-implantitis within 3 months after delivery of restoration. Note the cement on implant surface.

In addition, 238 screw-retained implant restorations in 66 patients (27 men and 39 women, mean age 54.7 years) were examined for presence of peri-implant disease, as a control group. From this amount, 53 implants were analyzed in 31 periodontally healthy patients and 185 implant restorations were evaluated in 35 individuals with history of periodontitis. All implants evaluated in the study were internally hexed (BioHorizons Internal, Birmingham, AL, USA) and had titanium plasma-sprayed surfaces with 0.5-mm polished neck.

Results

Cement remnants were found in 11 of 32 implants affected by mechanical complications and in 62 of 97 implants with biological complications, making it 73 implants of 129 in total (56%). Thirty-nine implants were analyzed in 23 patients with history of periodontitis and 34 were examined in 24 periodontally healthy individuals (Table 2). Peri-implant disease developed in 62 of 73 implants with cement remnants (85%). Early peri-implant disease occurred in seven implants (12%), whereas delayed disease was registered in 55 cases (88%). All 39 implants (100%) with extracoronal cement in the group of patients with history of periodontitis developed peri-implantitis. Eight implants (20%) in this group were lost due to extensive bone loss. All other survived implants were debrided from the cement and recovery was evident at the 1 month. In the periodontally healthy group, 20 of 31 implants were diagnosed with peri-implant mucositis, 3 implants had peri-implantitis, and 11 remaining implants from this group did not show evidence of peri-implantitis or peri-implant mucositis, although cement was present around the implants (Fig. 8a–c). The mean time of complication occurrence is presented in Table 3.

Table 2. **Distribution of implants with complications**

Complications (no. of implants) 129			
With cement remnants 73 (56.6%)		Without cement remnants 56 (43.4%)	
39 in PCP (30.2%)	34 in PHP (26.4%)	24 in PCP (18.6%)	32 in PHP (24.8%)

PCP, periodontally compromised patients; PHP, periodontally healthy patients.

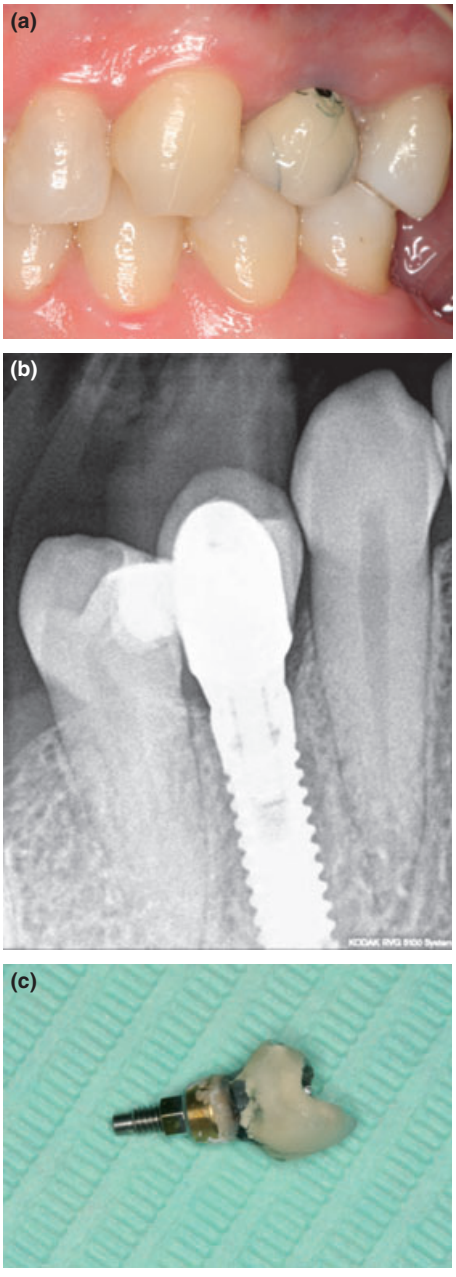

Fig. 8. (a) Mechanical complication of the implant crown; (b) Radiographic image shows no signs of peri-implantitis or cement remnants; (c) Retrieved implant restoration with undetected cement excess.

The group of implants without cement remnants consisted of 56 cases (39 mechanical and 17 biological complications) in 29 patients. Twenty-four implants in 18 patients with history of periodontitis had following outcome: 10 implants were without complications and 14 developed peri-implantitis. Thirty-two implants in 11 individuals without history of periodontitis developed three occurrences of peri-implant mucositis and 29 implants did not develop biological complications.

All restorations were cemented with glass ionomer modified with resin cement. Standard abutments were chosen to support the restorations.

The control group of screw-retained restorations had different outcome. All 53 restored implants in periodontally healthy patients did not have biological complications after mean follow-up of 42.76 ± 3.36 m (range from 18 to 74 m). There were two occurrences of peri-implant disease (1.08%) in the group of 185 implants in patients with history of periodontitis after mean follow-up of 40.52 ± 1.10 m (range from 12 to 73 m).

Discussion

This retrospective study suggests that patients with a history of periodontal disease are more prone to the development of delayed peri-implant disease when exposed to extracoronal residual cement. All 39 implants with cement remnants from the group of patients with history of periodontitis developed peri-implantitis and eight implants were lost. Eleven implants with mechanical complications in patients without history of periodontitis did not have radiographic or clinical signs of peri-implantitis, although cement was present in the tissues for about 29 months. Twenty implants in this group did develop less severe biological complications – peri-implant mucositis. It seems that the severity of cement-related peri-implant disease may depend on periodontal involvement of the patient. It is interesting to note that peri-implant disease developed within a shorter period in patients with history of periodontitis (23.4 m) than in the patients without history of periodontal infection (40.8 m). Several authors have also reported the different timing for cement-related peri-implant disease development, ranging from few weeks to 9 years after cementation (Pauletto et al. 1999; Wilson 2009). On the contrary, only two implants (1.08%) from the screw-retained control group of patients with history of periodontitis had developed peri-implant disease and implants in periodontally healthy individuals remained complication-free. This can be explained by the fact that screw-retained restorations do not require cement for their retention on implants. Similarly, implants with cemented restorations without cement remnants had lower rate of biological complications. It can be speculated that cement remnants may be important predisposing factor in pathogenesis of delayed peri-implant disease, as implants with cemented restorations showed clearly worse outcome, compared with implants with screw-retained prostheses. Conversely, there are studies reporting no adverse peri-implant tissue reaction to cement-retained implant prostheses, however, patients in these studies were not grouped whether to have history of periodontitis or not (Assenza et al. 2006; Blanes et al. 2007). Yet, it is likely that at least few factors, such as history of periodontitis and cement excess, may be required for the development of delayed peri-implant disease.

The role of cement remnants in the etiology of peri-implant disease may be compared to the function of dental calculus in the development of the periodontal disease. It has been stated that there is no etiologic association between the subgingival calculus and periodontal disease development, but the subgingival calculus may acts as a predisposing factor due to additional retention of bacteria and mechanical irrigation of the periodontal tissues (White 1997). A similar view may be applied to the peri-implant tissues, as cement remnants act like pieces of calculus. Cement has a rough surface, which has a tendency for bacterial accumulation with subsequent tissue inflammation. It has been shown that bacteria in the sulcus may cause peri-mucositis (Berglundh et al. 1992), which later can develop into peri-implantitis, with a resulting bone loss (Lindhe et al. 1992).

Another explanation of the difference in the time of peri-implant disease development may be the distance between cement remnants and crestal bone. During cementation, excess cement may not escape through the sulcus, but can be pushed further subgingivally. In contrast to teeth, the peri-implant tissues lack resistance to pressure due to the absence of an attachment to the implant surface. Connective tissue fibers do not attach to the implant and align themselves parallel along the fixture surface (Cochran et al. 1997; Hermann et al. 2000). Subsequently, the peri-implant tissues may be less resistant to pressure compared with tissues around teeth (Ericsson & Lindhe 1993). Several studies have shown that pressure ranging from 20 to 130 N can be developed during the cementation of crowns (Wang et al. 1992; Black & Amoore 1993). This would suggest that cement may be pushed deeper in the peri-implant sulcus and defy removal even after meticulous cleaning. If luting agent is located close to the bone, an acute reaction of the peri-implant tissues may occur, irrespective to whether patient has history of periodontitis or not. This can be confirmed by seven cases of early peri-implant bone loss, of which four occurred in group of periodontally susceptible patients and three in individuals without history of periodontal disease. This is the reason why early peri-implantitis was excluded from analysis of delayed disease cases. In all occurrences of early peri-implant disease, residual cement was found to be close to the bone level. This is in agreement with the case reports, which found cement having contact with bone around implant (Pauletto et al. 1999; Gapski et al. 2008). Conversely, if cement stays away from the bone in a periodontally healthy patient, peri-implant disease may not develop. Yet, another group of periodontitis patients showed no bone loss even with the heavy periodontal infection challenge (Loe et al. 1986). In current retrospective study, 11 implants in periodontally healthy patients had residual cement, but no crestal bone loss or inflammation of the tissues were recorded. It may be speculated that if cement is located in safe distance from bone in periodontally compromised individual, patient escapes early peri-implantitis; however, cement may pledge chronic process, ending in delayed peri-implantitis.

Table 3. **Analysis of implants with cement remnants**

History of periodontitis	No. of implants	Complication	Months (median)	Ranging (months)
Yes	39	35 peri-implantitis 4 early peri-implantitis*	23.46 ± 1.72 (25)	10–48
No	34	11 no complication 20 peri-mucositis 3 early peri-implantitis*	29.91 ± 4.56 (34) 40.83 ± 10.15 (38)	9–56 14–85

*Timing for early peri-implantitis was calculated in both groups – 5.75 ± 0.16 (6), range 5–6 months.

The ability of dental cement to initiate peri-implant disease is not a new issue. Clinical reports suggest that early peri-implant disease may develop within several weeks or months, if cement is not properly cleaned (Pauletto et al. 1999; Gapski et al. 2008, Wilson 2009). Thus, excess cement has been considered as a risk factor for rapid onset of inflammation or bone loss, but was not discussed as a cause for delayed peri-implant pathology. Late bone loss was attributed to the periodontal infection, overloading, or plague accumulation. However, in light of our findings in this study, cement remnant should be considered as a possible contributing factor in late bone loss around implants.

Standard abutments with subgingival cementation margins were chosen to support restorations in all patients. This might be one of the reasons for incomplete cement removal in majority of cases. Although the location of the margin was selected according to the current recommendations, it seems that cementation of crowns with subgingival margins requires re-evaluation. Linkevicius et al. (2011) have shown that it is difficult to clean all cement from implant restorations even with slightly subgingival margins. Another disadvantage of the standard abutments is that they are usually too narrow to support the entire restoration without leaving undercuts, which are very difficult to reach during cleaning. This is supported by the fact that in 73 of 129 (56%) cases, cement remnants were not completely removed from implant restorations cemented on standard abutments. Some implant systems offer standard abutments with individualized margins, which partly follow the line of peri-implant tissues. This could be helpful; however, it does not completely eliminate the undercut and the influence of this modification on amount of cement remnants is not documented. The use of individual prosthetic abutments could be advised to control the cement excess (Dumbrigue et al. 2002). It may be concluded that standard abutment with deep subgingival margins should be avoided.

Yet, another aspect influencing the detection of extracoronal cement is control radiographs. Linkevicius et al. (2012) has shown in a clinical study that dental radiographs should not be considered as a reliable method for cement excess evaluation. Results revealed that cement remnants mesially were visible in 4 cases of 53 or 7.5%, and in 6 cases of 53 distally (11.3%). In addition, only interproximal areas of implant restorations can be inspected, as paltinal/lingual and facial areas block radiographic examination and cement remnants may rest there as well. Wadhwani et al. (2010) have proved that radiographic density of implant restorative cements is poor, depends on the thickness of the specimens and smaller pieces would remain unseen.

Modified glass ionomer luting agent was the cement of choice in all cases. It is easily mixed, poses good retentive properties (Ergin & Gemalmaz 2002), and low solubility (Yanikoglu & Yesil 2007), and therefore is very popular for cementation of teeth-borne prostheses. However, Agar and co-workers have shown that cement with resin components is difficult to remove from abutment surface after cementation (Agar et al. 1997). This feature is a disadvantage, if cement is used for luting of implant-supported restorations, however, a recent survey has shown that it is the most popular permanent cement for cemented implant restorations in US dental schools (Tarica et al. 2010).

This study by its nature has limitations. Prospective design, sample size, and control of other predisposing factors are issues that should be considered in future studies. There were patients with more than one implant both in control and test groups. Consequently, the implants within a patient may not give independent observations and complicate statistical analysis, thus patient-based study with adequate number of individuals and implants would give more objective answer to the questions raised. On the other hand, cement-related peri-implantitis may be considered as an iatrogenic disease, therefore adequate number of patients and cases may be difficult to collect. Furthermore, prospective design of a study for cement-related peri-implantitis development would be problematic to perform due to obvious ethical reasons. As with the evasiveness of periodontal disease causality, it may be difficult to show a clear association of peri-implant disease correlation with periodontal disease or have strict definition of periodontally compromised patient (Karoussis et al. 2007).

Conclusions

Within the limitation of this study, it can be concluded that peri-implant disease has been shown to be associated with residual cement in particular to patients with predisposing periodontal disease. Cement remnants in patients without history of periodontitis may cause less severe peri-implant disease or may not predispose infection at all. Cement remnants should be considered as an additional predisposing factor in development of chronic peri-implant disease. Finally, screw-retained implant restorations might be considered in periodontally susceptible patients.

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