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Periodontitis as a potential risk factor for peri-implantitis

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Abstract

Objectives: To review the literature regarding the possible association between a previous history of periodontitis and peri-implantitis.

Material and Methods: A search of MEDLINE as well as a manual search of articles were conducted. Publications and articles accepted for publication up to January 2008 were included.

Results: Out of 951 papers retrieved, a total of three papers were selected for the review. Thus, the available evidence for an association between periodontitis and peri-implantitis is scarce.

Conclusions: Based on three studies with a limited number of patients and considerable variations in study design, different definitions of periodontitis, and confounding variables like smoking that not been accounted for, this systematic review indicates that subjects with a history of periodontitis may be at greater risk for peri-implant infections. It should, however, be stressed that the data to support this conclusion are not very robust.

Key words: peri-implantitis; periodontitis; risk factor

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During the last 20 years, dental implants and implant-supported supra-structures have become a commonly used alternative to conventional removable partial dentures and fixed conventional bridges. The high clinical survival rate reported in several 10-year follow-up studies has led to a widespread acceptance and use of dental implants. Although the general impression of implant therapy is that the success rate is high, infections defined as peri-implant mucositis and peri-implantitis are a common feature around implants. Peri-implant mucositis and peri-implantitis are recognized as infectious diseases. Peri-implant mucositis describes an inflammatory lesion that resides in the mucosa, while peri-implantitis also affects the supporting bone.

In a systematic review of prospective studies with a follow-up period of $\geqslant 5$ years, a relatively low prevalence rate of peri-implantitis was reported (Berglundh et al. 2002). The authors, however, concluded that the incidence of biological complications may be underestimated and should be interpreted with caution, as biological complications were considered in only 40–60% of the studies available for assessment. Recent data from longitudinal studies have demonstrated bone loss adjacent to implants and the presence of peri-implantitis in higher percentages. In a study assessing the prevalence of subjects with progressive bone loss at implants with a function time of at least 5 years, 185 (28%) of the 662 included subjects had one or more implants with progressive bone loss (Fransson et al. 2005). In another long-term study, peri-implant mucositis was present in 76.6% and peri-implantitis in 16% of the patients (Roos-Jansåker et al. 2006). These rates are comparable if not higher than those reported for gingivitis (Abrahamsson et al. 2006, Monten et al. 2006) and periodontitis (Borrell et al. 2005). As the number of patients treated with dental implants increases, it is inevitable that the incidence of peri-implant infections will increase, posing a significant future health care problem.

Bacterial colonization at newly inserted implants occurs rapidly (van Winkelhoff et al. 2000, Quirynen et al. 2006, Fürst et al. 2007, Salvi et al. 2008). The concept that microorganisms are essential for the development of infections around dental implants is well supported in the literature (Pontoriero et al. 1994, Augthun & Conrads 1997, Salcetti et al. 1997, Mombelli & Lang 1998, Leonhardt et al. 1999, Quirynen et al. 2002, 2006). The microbiota associated with peri-implantitis have been reported to be similar to the microbiota associated with periodontitis and it has been suggested that periodontal pockets of teeth may act as a reservoir

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for microorganisms to colonize the newly inserted implants (for a review, see Mombelli 2002, Quirynen et al. 2002). Peri-implant diseases have been associated with a predominantly Gram-negative anaerobic microflora. Recently, the presence of *Staphylococcus aureus* in peri-implantitis lesions has also been reported (Leonhardt et al. 1999, Renvert et al. 2007). This is not surprising as foreign bodies often are colonized by *S. aureus*. It has been reported that titanium favours colonization by *S. aureus* (Harris & Richards 2004, Antoci et al. 2007) and causes *S.aureus* complication of hip joint titanium implants (Stoodley et al. 2005).

Although the microorganisms may be considered as the initiating factor in periodontal disease, periodontitis is considered to be a multifactorial disease (Page et al. 1997). One factor of importance is genetic traits (Michalowicz et al. 2000). Patients susceptible to periodontitis may react differently to infectious agents, resulting in more advanced tissue breakdown. Thus, it has been perceived that patients with a history of periodontitis are at a higher risk of developing peri-implantitis compared with patients without a history of periodontitis.

The purpose of the present review was to assess whether individuals with a history of periodontitis are more likely to develop peri-implantitis compared with patients without such a history.

Material and Methods

The National Library of Medicine, Washington DC (Medline-PubMed), was searched for publications. In addition, a land search of relevant journals was performed. A broad search directed towards studies on implant treatment in patients with a known history of periodontal disease was performed. The primary outcome variable was peri-implantitis.

Search strategy

The database was searched up till 31 January 2008 using the following terms for the search strategy:

(''Dental Implants, Single Tooth'' [MeSH Terms] OR Implants [Text Word] OR peri implantitis [Text Word]) AND (''Periodontitis'' [MeSH Terms] OR Periodontitis [Text Word].

In addition, the reference lists of relevant review papers were hand searched.

Eligibility criteria

(a) Studies reporting on outcomes of peri-implantitis in patients with a history of periodontitis compared with non-periodontitis patients.
(b) Retrospective or prospective studies.
(c) Studies of at least 5 years of follow-up.

The following factors were recorded to be able to investigate heterogeneity of outcome across studies:

(a) Evaluation period.
(b) The definition of ''history of periodontitis''/''periodontal patient''.
(c) General health.
(d) Number of subjects.
(e) Mean age and age range of subjects.
(f) Smoking habits (defined as smokers, former smokers and non-smokers).
(g) Implant system used.

Screening and selection

The titles and abstracts of the papers were screened by two independent reviewers (S. R. and G. R. P.).

The search criteria used to include the papers for full-text screening were:

(a) Implant treatment.
(b) Periodontally compromised patients.
(c) Studies with a follow-up period of at least 5 years.
(d) Peri-implantitis and/or peri-implant bone loss as the outcome variable.

When an abstract included the above-mentioned criteria, or if there was doubt regarding one or more of the search criteria, the paper was selected for full reading. If any of these criteria was not fulfilled, the paper was disregarded. Titles without abstracts that appeared to investigate the success rate of implants were selected for full-text reading. Only papers written in the English language were selected. Case reports, letters and reviews were excluded. Disagreement regarding inclusion was resolved by discussion between the reviewers.

Search results

The search resulted in 951 titles for review. After screening the titles and abstracts, 17 full papers were selected for full-text reading. These papers were read by the reviewers, which finally yeilded three articles that fulfilled the selection criteria.

Ten papers were excluded because no comparisons were performed with a control group and/or the evaluation period was <5 years (Ellegaard et al. 1997a, b, 2006, Watson et al. 1999, Mengel et al. 2001, Yi et al. 2001, Leonhardt et al. 2002, Baelum & Ellegaard 2004, Wennström et al. 2004, Mengel & Flores-de-Jacoby 2005). In addition, three papers were excluded as bone loss and/or peri-implantitis was not the outcome variable or the evaluation period <5 years (Brocard et al. 2000, Evian et al. 2004, Rosenberg et al. 2004). The paper by Roos-Jansåker et al. (2006), linking a history of periodontitis to peri-implantitis and of sufficient length, was excluded because subgroup analysis had not been performed.

Results

Three papers compared loss of bone adjacent to the implant or presence of peri-implantitis in periodontitis and non-periodontal patients over a period of at least 5 years (Hardt et al. 2002, Karoussis et al. 2003, Mengel et al. 2007). Information from these three identified publications is summarized in Tables 1 and 2.

The study by Hardt et al. (2002) included a cohort of 147 subjects with or without a past history of periodontitis. The report is based on a chart review. Specifically, only 50 subjects were included in the analysis, representing those in the highest and the lowest percentile with evidence of alveolar bone loss around remaining teeth. A formula allowing an adjustment of subject age was used in defining subjects belonging to the different groups. Among a total of 346 implants identified, alveolar bone loss was studied in 192 implants, corresponding to 55% of the implants originally identified. The mean extent of alveolar bone loss around implants in the non-periodontitis and the periodontitis groups was 1.7 mm ($\pm$ 0.8 SD) and 2.2 mm ($\pm$ 0.8 SD). The statistical analysis failed to identify whether this difference was significant. In terms of implant loss, the data suggested that the risk of losing implants in the periodontitis group was approximately 2:1 (calculated from data presented in the article but not reported by the authors). According to the authors, the retrospective nature of the

Table 1. Selected studies, follow-up time and patient characteristics

Study	Participants	Follow-up years	Supportive care	Smoking habits	General health	Definition of periodontitis
Hardt et al. (2002)	Periodontitis, $n = 25$ (♀ = 13, ♂ = 12) Age: 53.5 years Non-periodontitis, $n = 25$ (♀ = 16, ♂ = 9) Age: 57.3 years	5	Not reported	Not reported	No systemic diseases	Periodontitis defined as an age-related bone loss score. Percentage of teeth with bone level $<50\%$ at baseline: Periodontitis = 25.7% Non-periodontitis = 1.1%
Karoussis et al. (2003)	Periodontitis, $n = 8$ Non-periodontitis, $n = 45$	10	3–6 months recall schedule at the university or in private practices	Periodontitis = 47.6% implants in smokers Non-periodontitis = 19.8% implants in smokers	Not reported	History of periodontitis
Mengel et al. (2007)	Periodontitis, $n = 5$ (♀ = 5, ♂ = 0) Age: 31–44 years Non-periodontitis, $n = 5$ (♀ = 3, ♂ = 2) Age: 20–51 years	10	3 months recall schedule	Not reported	Not reported	General aggressive periodontitis

study did not allow clinical assessments of the conditions.

The study by Karoussis et al. (2003) included a total of eight subjects with a preceding history of periodontitis and 45 subjects without a history of periodontitis. All subjects received routine supportive care. The overall implant survival rate for the group with a past history of chronic periodontitis was 90.5%, and 96.5% in the group with no past history of periodontitis. Depending on the clinical parameters and the definition of success, the difference in the success rate between the two groups varied between 20% and 27%. The success rate was always lower in those with a past history of chronic periodontitis. If the failure was defined as probing pocket depth $\geqslant 5$ mm, bleeding on probing and bone loss >0.2 mm annually, the group with a preceding history of chronic periodontitis had a success rate at year 10 of 52.4% whereas those subjects with no history of periodontitis had a success rate of 79.1%. Consistent with other aspects of the report, subjects with a history of periodontitis had a significantly higher implant complication rate (peri-implantitis). Statistical analysis failed to demonstrate that the implant complication rate in smokers *versus* nonsmokers in either group differed by periodontal status. In summary, statistical analysis failed to demonstrate that the overall survival rate or clinical success differed between subjects with or without a preceding history of periodontitis

The study by Mengel et al. (2007) assessed dental implant survival through a 10-year period among five subjects with an original diagnosis of rapidly progressive periodontitis and in five subjects diagnosed as periodontally healthy subjects. In the healthy control group and after 10 years, all implants remained in function. In the group with severe periodontitis, two implants were lost (one before the suprastructure was placed and one 7 months after placement of the suprastructure) and two were put to sleep before completion of the suprastructure. Thus, not acounting for the loss of implants during the first period, there was no difference in implant loss between the two groups. It is of interest that during the 10-year period, a large number of teeth were lost in the group with rapidly progressive periodontitis, whereas no teeth were lost in the periodontally healthy control groups. At implant sites, the probing pocket depths remained <5 mm in both group. In both groups, a rather large variation in alveolar bone levels was noticed. The mean amount of bone loss after year 1 was 1.3 m in the periodontitis group but only 0.1 mm in the periodontally healthy control group. The microbiota appeared to be similar at teeth and implant sites although subjects with rapidly progressive periodontitis had more motile rods around their implants as assessed by dark-field microscopy.

Discussion

The long-term success of dental titanium implants placed in patients with a history of periodontitis has been addressed in a number of recent reviews (Van der Weijden et al. 2005, Schou et al. 2006, Karoussis et al. 2007, Quirynen et al. 2007). In some of these reviews, short-term data <5 years have been included (Karoussis et al. 2007, Quirynen et al. 2007). In three of the reviews (Van der Weijden et al. 2005, Karoussis et al. 2007, Quirynen et al. 2007), data from studies without a control group had been included. By doing so and also by including studies with either survival rates, success rates or so-called failures as the outcome variables, it is difficult to obtain a clear picture of whether peri-implantitis is more prevalent in individuals with a history of periodontitis than in individuals without a history of periodontitis.

Data on the prevalence of peri-implantitis and periodontitis must be considered with great caution. That subjects with a history, or present periodontitis with dental implants, are at a greater risk may seem logical but would also assume that the infectious aetiology and host immune response predisposing to disease are the same. There are insufficient data on the microbiota in subjects developing peri-implantitis. It appears from a study including 213 subjects that the microbiota at implant sites did not differ whether subjects were dentate or not or by implant status (Renvert et al. 2007).

In the review by Quirynen et al. (2007), the authors concluded that the heterogeneity of data available did not allow a meta-analysis. Nevertheless, the authors of the review concluded that periodontally compromised patients in

Table 2. Selected studies; results regarding dental implants

Study	Implants at baseline	Implant system	Gingival health	Plaque	Prevalence of peri-implantitis	Bone loss (mm)	Loss of attachment	Implant loss Survival rate
Hardt et al. (2002)	Periodontitis ($n = 100$) Non-periodontitis ($n = 92$)	Brånemark (Nobel Biocare Holding AG, Zürich, Switzerland)	Not reported	Not reported	Not reported	Periodontitis = 2.2 ± 0.8 Non-periodontitis = 1.7 ± 0.8	Not reported	Periodontitis ($n = 8$) Periodontitis ($n = 92\%$) Non-periodontitis ($n = 3$) Non-periodontitis = 97%
Karoussis et al. (2003)	Periodontitis ($n = 21$) Non-periodontitis ($n = 91$)	ITI (Institut Staumann AG, Basel, Switzerland) hollow screw	Not reported	Not reported	29% 6%	Periodontitis $m = 1 \pm 1.4$ $d = 0.9 \pm 0.7$ Non-periodontitis $m = 0.5 \pm 1.1$ $d = 0.5 \pm 1.1$	Not reported	Periodontitis ($n = 2$*) Periodontitis = 91% Non-periodontitis ($n = 3$*) Non-periodontitis = 97%
Mengel et al. (2007)	Periodontitis ($n = 36$)	Not reported	Löe Silness 1 year 0.2 5 years 0.3 10 years 0.4	Silness Löe 1 year 0.8 5 years 0.7 10 years 0.1	Not reported	Periodontitis = 3.4 mm	Periodontitis = 2.4 mm	Periodontitis ($n = 2$) Periodontitis ($n = 94\%$) One at second stage surgery One 1 month after suprastructure
	Non-periodontitis ($n = 7$)	Brånemark	1 year 0.3 5 years 0.0 10 years 0.0	1 year 0.6 5 years 0.1 10 years 0.6	Not reported	Non-periodontitis = 1.3 mm	Non-periodontitis = no change	Non-periodontitis ($n = 0$) Periodontitis ($n = 100\%$)

*Calculated by author.

the presence of supportive periodontal therapy can be successfully treated with minimally/moderately rough implants. The review by Karoussis et al. (2007) also identified considerable discrepancies between studies. No statistically significant differences in either short- or long-term implant survival between patients with a history of chronic periodontitis and periodontally healthy individuals were found. Patients with a history of chronic periodontitis may, however, exhibit significantly greater peri-implant bone loss and incidence of peri-implantitis. In the review by van der Weijden et al. (2005), the authors concluded that data suggested that the implant treatment outcomes differed in subjects with periodontitis and were not being as successful as in subjects without a history of periodontitis.

In the review by Schou et al. (2006), the authors also identified that small sample sizes and differences in the methodological quality assessment in studies require that caution must be exercised in the interpretation of conclusions. Nevertheless, Schou et al. (2006) concluded that the survival of the suprastructures and the implants does not depend on periodontal status although a significantly increased incidence of peri-implantitis may occur in subjects with periodontitis-associated tooth loss.

Using somewhat different search strategies in the present review, it became obvious that once again differences in the study design, diagnosis of periodontitis, follow-up time, and differences in the methods used to include subjects in studies assessing the role of a previous history of periodontitis would not allow any meta-analysis. Two of the three reviews included by Schou et al. (2006) were also included in the present review (Hardt et al. 2002, Karoussis et al. 2003). We concur with the conclusions by Schou et al. (2006) in that statistical analysis failed to distinguish differences in the implant survival rates in subjects with or without a preceding history of periodontitis. The second conclusion by Schou et al. (2006) must be considered with great caution in that one of the papers presented by Karoussis et al. (2003) only included eight subjects with 21 implants and a preceding history of periodontitis whereas they included 45 subjects with no history of periodontitis and with 91 implants. The statistical analysis of such data must be considered with the highest caution and the report on proportions of complica-

tions must be viewed as questionable. Furthermore, Karoussis et al. (2003) did not include information on the original severity of periodontitis, or whether the subjects with periodontitis had experienced periodontitis recurrence during the study period, or what the criteria were for periodontitis diagnosis at any time-point. Although gender was reported, no information on systemic health or subject age in the different study groups was identified. In the paper by Hardt et al. (2002), the authors had identified subjects by periodontitis severity with regard to alveolar bone loss adjusting for subject age. The major shortcoming of the study by Hardt et al. (2002) is it was a restrospective study.

The study by Mengel et al. (2007) included a very limited number of individuals (five with a history of aggressive periodontitis and five without a history of periodontitis), seriously limiting the possibility to draw any definite conclusions. The absence of a clear definition of periodontitis further complicates the conclusions from the above-cited studies.

Host immune responses to infection may play a role in alveolar bone loss (Tolstunov 2007). Others have suggested that subjects with the interleukin 1 gene polymorphism (IL-1A −889 and IL-1B +3954 genotype) may only have a marginally elevated risk for peri-implantitis. (Feloutzis et al. 2003, Laine et al. 2006, Lachmann et al. 2007). Thus, assuming that this gene polymorphism constitutes a risk for periodontitis, this effect may not be carried over to the risk for peri-implantitis. It has also been shown that cytokines with a potential to activate osteoclasts were found in both peri-implantitis and chronic periodontitis. The cytokine profiles differed in that IL-1-α was the most prevalent cytokine in peri-implantitis while TNF-α was the most common cytokine in chronic periodontitis (Konttinen et al. 2006). On the other hand, several studies have suggested similarities in host cell presence at implants with peri-imlantiits and teeth with periodontitis (Berglundh et al. 2004, Berglundh & Donati 2005, Pongnarisorn et al. 2007). A large B-cell infiltrate was demonstrated both in peri-implantitis and in periodontitis lesions. In a study by Gualini & Berglundh (2003), the authors demonstrated that elastase-producing cells were common in peri-implantitis lesions, possibly indicating a more acute type of infection than in periodontitis.

Until patterns of infection and host responses are well known in subjects with peri-implantitis with either a pre-existing diagnosis or current periodontitis, it is important to acquire data on the prevalence of peri-implantitis in subjects with or without periodontitis based on well-defined criteria and control of confounding factors. New studies on the prevalence of peri-implantitis in relation to the periodontal diagnosis must include larger and balanced study populations than currently available.

In conclusion and based on three studies with a limited number of subjects and considerable variations in study design, different definitions of periodontitis, and confounding variables like smoking not accounted for, this systematic review summarizes that subjects with a history of periodontitis may be at a greater risk for peri-implant infections and complications. It should, however, be stressed that the data to support this conclusion are not very robust.

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Clinical Relevance

Scientific rationale for the study: Periodontitis patients may be at greater risk of developing peri-implantitis.

Principal findings: Based on three studies this systematic review indicates that periodontitis patients may be at greater risk for peri-implant infections.

Practical implications: Periodontitis patients with implants should be regarded as risk individuals for peri-implantitis.