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Nine- to fourteen-year follow-up of implant treatment. Part III: factors associated with peri-implant lesions

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Abstract

Objective: The aim of the present paper was to analyse, on patient and implant basis, factors related to peri-implant lesions.

Material and Methods: Two hundred and eighteen patients treated with titanium implants were examined for biological complications at existing implants 9–14 years after initial therapy. The effects of several potentially explanatory variables, both on patient and on implant levels, were analysed.

Results: On the implant level, the presence of keratinized mucosa ($p = 0.02$) and plaque ($p = 0.005$) was associated with mucositis (probing depth $\geqslant 4$ mm+bleeding on probing). The bone level at implants was associated with the presence of keratinized mucosa ($p = 0.03$) and the presence of pus ($p < 0.001$). On the patient level, smoking was associated with mucositis, bone level and peri-implantitis ($p = 0.02$, < 0.001 and 0.002, respectively). Peri-implantitis was related to a previous history of periodontitis ($p = 0.05$).

Conclusions: Individuals with a history of periodontitis and individuals who smoke are more likely to develop peri-implant lesions.

Key words: associated factors; mucositis; peri-implant bone level; peri-implant lesions; peri-implantitis

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Biological complications occur around implants (Mombelli & Lang 1998, Leonhardt et al. 1999, Berglundh et al. 2002, Quirynen et al. 2002, Roos-Jansåker et al. 2003, 2006a, b). In a previous report, 48% of the implants were found to have peri-implant mucositis [defined as probing depth $\geqslant 4$ mm and bleeding on probing (BOP)] and 13.3% of the implants had a bone level at three to four threads (3.1–3.7 mm) after 9–14 years in function (Roos-Jansåker et al. 2006b). Peri-implantitis may lead to complete disintegration and implant loss (Esposito et al. 1998a, Quirynen et al. 2002, Roos-Jansåker et al. 2006a) even if extensive treatment aiming at resolving the peri-implant infection has been performed (Leonhardt et al. 2003).

It is likely that patient-associated factors may be important for the development peri-implantitis. Patients susceptible to periodontal disease have been reported to develop more peri-implantitis (for a review, see Van der Weijden et al. 2005). Smoking is another risk factor that has been associated with peri-implant infections (Haas et al. 1996, Lindquist et al. 1996, Esposito et al. 1998b, Baelum & Ellegard 2004), and in patients treated for peri-implant infections, the outcome of treatment seemed to be negatively influenced by smoking (Leonhardt et al. 2003). The importance of oral hygiene for the development of peri-implantitis was also highlighted in a paper by Lindquist et al. (1996).

In a previous publication, the frequencies of peri-implant lesions 9–14 years after implant therapy were reported (Roos-Jansåker et al. 2006b). The aim of the present paper was to analyse, on patient and implant basis, associated factors related to peri-implant lesions.

Material and Methods

This study was approved by the Institutional Review Board, University of Lund, Sweden. All participating individuals signed an informed consent. The study reports on patients treated with titanium implants (Brånemark System®, Nobelpharma. Göteborg, Sweden) at the

Public Dental Health Service in Kristianstad, Sweden, during a period from January 1988 to December 1992. During this interval, a total of 294 patients were provided with implant-supported fixed or removable restorations at the Department of Prosthodontics. The two-step implant surgery procedures were performed either at the Department of Oral Surgery or the Department of Periodontology.

Systemic antibiotics were prescribed to all patients for 10 days, starting the day before implant installation, and twice-daily chlorhexidine rinses were recommended until the sutures were removed after 7 days. Submerged implant healing was allowed for a minimum of 3 months, after which the surgical abutments were fitted. Shortly thereafter, the suprastructure was placed. All patients were instructed in how to maintain proper oral hygiene around the implants and the remaining teeth. The patients were then referred back to their general dentist for supportive therapy and follow-up.

One and 5 years after placement of the suprastructure, the patients where examined at the Department of Prosthodontics and new sets of intra oral radiographs were obtained. Between January 2000 and December 2002, the patients were again called in for a clinical and long-cone radiographic examination. This final examination, on which the data of this paper are based, was performed 9–14 years after suprastructure placement at the dental clinic of the University of Kristianstad by one and the same examiner (C.L.). An update of the medical and dental history was made on all patients attending this examination, which included the following, for data analyses:

- *age at final examination;*
- *gender;*
- *years of education* (<12 *versus* $\geqslant 12$ years);
- *total number of dental visits* (*dentist and dental hygienist*) since placement of the suprastructure;
- *smoking habits* (current smoker, former smoker, never smoking; if current or former smoker, numbers of cigarettes/day were used to calculate pack-years);
- *medical history* (focus on diabetes, osteoporosis and coronary heart disease);
- *keratinized mucosa* (measured in mm at the buccal marginal portion of the implant mucosa);
- *probing depth* measured at four sites (mesial, buccal, distal and lingual) of each implant to the nearest mm using a plastic probe with 0.25 N force (Hawe Click-Probe®, KerrHawe SA, Bioggio, Switzerland);
- *BOP* (presence or absence following probing depth measurement) total score for both teeth and implants and at implants measured at four sites around teeth and implants and expressed as a percentage of examined sites. The bleeding scores were divided into three categories as follows: 0–20%, 21–60% and 61–100%;
- *suppuration* (if apparent following probing);
- *plaque score* (presence/absence of plaque at four sites around teeth and implants after using an disclosing dye, and expressed as a percentage of the examined sites);
- *% remaining teeth before implant placement with bone loss* $\geqslant 4$ mm (measured on approximal surfaces from the cemento enamel junction on radiographs at the time of implant installation). The extent of bone loss within the patient was then divided into two categories: 0–30% and 31–100% of teeth with bone loss $\geqslant 4$ mm; and
- *number of implant threads not supported by bone* (measured at the mesial and distal aspects of the implant on radiographs obtained 1 year after placement of the suprastructure and at final examination. The bone level was divided in two categories: <3 threads and $\geqslant 3$ threads).

Mucositis was defined as probing depth $\geqslant 4$ mm and BOP, and peri-implantitis was defined as bone loss $\geqslant 3$ threads when comparing the radiographs taken at the final examination with the radiograph taken 1 year after placement of the suprastructure, combined with BOP and/or pus.

Data analyses

Outcome data on mucositis (probing pocket depth $\geqslant 4$ mm and BOP), bone level (<3 threads or $\geqslant 3$ threads) and peri-implantitis ($\geqslant 3$ threads of bone loss between the 1 year and final radiographic examination combined with BOP), respectively, were considered. We analysed the effects of several potentially explanatory variables on each of these binary outcomes. Explanatory variables on patient level [gender, age ($\leqslant 59$, 60–74 or 75+), years in school (i.e. <12 or $\geqslant 12$ years), smoking habits (i.e. never, former or smoker), general disease (diabetes, osteoporosis and coronary heart disease *versus* no such disease), visits to dental hygienist (<1/year *versus* >1/year), visits to dentist (<1/year *versus* >1/year), bone loss at teeth at implant placement or before tooth extraction in conjunction with implant treatment (i.e. 0–30%, 31–100%)], as well as on implant level [keratinized mucosa (yes *versus* no), plaque (yes *versus* no), ''pocket depth'' (<4 mm *versus* $\geqslant 4$ mm), pus (yes *versus* no) and bleeding (yes *versus* no)] were analysed. Logistic regression with random effects was used, which should be used in any situation involving grouped binary data in which the outcome observations within individual groups are correlated (EGRET for Windows User Manual 1999); here, the outcomes for implants are correlated within each patient. We used, technically speaking, the extension of the logistic-binomial model for distinguishable clustered outcome data (EGRET for Windows User Manual 1999). First, the effect of each explanatory variable was examined in univariate analyses (i.e., based on models involving a single explanatory variable). Each explanatory variable with a p-value <0.1 obtained from the univariate analysis was forwarded into a multivariate analysis. The magnitude of the effect of an explanatory variable was estimated by an odds ratio (OR) with 95% confidence interval (CI). Logistic regressions with random effects analyses were carried out using EGRET (EGRET for Windows User Manual 1999). Other statistical computations were carried out using SPSS for Windows (release 11.5.1; SPSS Inc., Chicago, IL, USA).

Results

Out of the 294 patients receiving implants during the period of 1988–1992, 218 patients attended the 9–14-year examination. Twenty-two patients had died, and 54 patients did not wish to participate or were unable to attend because of health reasons. Out of the 1057 implants in these 218 patients, 12 implants in 10 patients were not used in the supra-construction because of different reasons. These implants were con-

Table 1. Analysis of potential explanatory variables for the outcome event mucositis

Explanatory variable	Outcome #yes/total	Univariate analyses		Multivariate analysis	
		OR (95% CI)	*p*	OR (95% CI)	*p*
On implant level					
Keratinized[†] mucosa					
No	201/473	1.0[*]	0.02	1.0[*]	0.008
Yes	275/520	1.6 (1.1–2.4)		1.6 (1.1–2.3)	
Plaque[‡]					
No	99/291	1.0[*]	0.005	1.0[*]	0.004
Yes	376/704	1.9 (1.2–2.9)		1.9 (1.2–2.9)	
On patient level					
Gender					
Male	244/508	1.0[*]	1.0	NI[**]	
Female	233/490	1.0 (0.53–1.9)			
Age (years)					
⩽59	107/182	1.0[*]	0.02	NS[††]	
60–74	245/508	0.53 (0.24–1.2)			
75+	125/308	0.29 (0.12–0.68)			
School[§](years)					
<12	406/852	1.0[*]	0.4	NI[**]	
⩾12	67/140	1.4 (0.60–3.4)			
Smoking					
Never	132/309	1.0[*]	0.009	1.0[*]	0.02
				1.0 (0.53–2.1)	
				2.8 (1.2–6.2)	
Ex-smoker	164/382	1.1 (0.57–2.3)			
Smoker	181/307	2.9 (1.4–6.0)			
General disease					
No	285/603	1.0[*]	0.8	NI[**]	
Yes	192/395	1.1 (0.55–2.0)			
Visit hygienist[¶]					
<1/year	320/694	1.0[*]	0.21	NI[**]	
⩾1/year	152/297	1.5 (0.78–3.0)			
Visit dentist[‖]					
<1/year	139/325	1.0[*]	0.11	NI[**]	
⩾1/year	333/666	1.8 (0.88–3.6)			
% Teeth with bone loss					
0–30	94/185	1.0[*]	0.07	NS[††]	
31–100	242/457	0.96 (0.45–2.1)			
No teeth	141/356	0.47 (0.20–1.1)			

[*]Reference category.
[†]Five study implants with missing information.
[‡]Three study implants with missing information.
[§]Number of years in school; one patient with six study implants had missing information.
[¶]Number of visits at hygienist per year; one patient with seven study implants had missing information.
[‖] Number of visits at dentist per year; one patient with seven study implants had missing information.
[**]Not included in the multivariate analysis.
[††]Not significant ($p>0.05$) in the multivariate analysis.
OR, odds ratio; CI, confidence interval.

sidered ''sleeping implants'' and were excluded from the analyses. In addition, 46 implants in 22 patients were lost before placement of the suprastructure or during the follow-up period, leaving 999 implants available for this study (for further details on the patient material, the reader is referred to Roos-Jansåker et al. 2006a).

In Table 1, the potential explanatory variables for the outcome event mucositis are presented. On the implant level, the presence of keratinized mucosa and plaque were explanatory in both the univariate and multivariate analysis ($p = 0.02$ and 0.005, respectively) and multivariate analysis ($p = 0.008$ and 0.004, respectively). On the patient level smoking was the only variable that was significant in the multivariate analysis ($p = 0.02$), whereas in the univariate analysis, age ($p = 0.02$) and smoking ($p = 0.009$) were significant.

The potential explanatory variables for the outcome event peri-implant bone level are presented in Table 2. On the implant level, the presence of keratinized mucosa, pocket depth and pus were explanatory in the univariate analysis ($p = 0.01$, 0.002 and <0.001, respectively). In the multivariate analysis, only keratinized mucosa and pus were explanatory ($p = 0.03$ and <0.001, respectively). On the patient level, smoking was significant in both the univariate and the multivariate analyses ($p<0.001$).

Table 3 presents the potential explanatory variables for the outcome event peri-implantitis. On the patient level, smoking and bone loss around teeth (evidence of a history of periodontitis) were the variables that were significant both in the univariate ($p<0.001$ and 0.01) and in the multivariate analyses ($p = 0.002$ and 0.05).

Discussion

As it has been reported that the implant design may be of importance regarding the incidence of peri-implantitis (Karoussis et al. 2004), it is desirable to eliminate implant design as a confounding variable when aiming to analyse factors of importance for the development of biological complications at implants. All patients in this study were treated with the Brånemark implant system, eliminating implant design as a possible confounder. Although the surgical procedure as well as the initial follow-up were standardized for all individuals, a uniform supportive periodontal treatment programme was not applied in this study. It is likely that infrequent supportive care visits will increase the risk of developing peri-implant lesions, especially among individuals who have lost their teeth because of periodontal disease. The supportive care intervals in this study were based on the risk assessments made by the referring dentists. It is possible that some patients were seen too seldom to prevent disease progression.

In multivariate modelling, the problem of colinearity between explanatory variables should be taken into consideration. Our final multivariate models are solely based on conventional statistical criteria. Nevertheless, the fact that one explanative variable was included, and another was excluded, does not necessary mean that the excluded one is not important.

Table 2. Analysis of potential explanatory variables for the outcome event Bone level (i.e. < 3 threads and ⩾3 threads

Explanatory variable	Outcome #yes/total	Univariate analyses		Multivariate analysis	
		OR (95% CI)	*p*	OR (95% CI)	*p*
On implant level					
Keratinized[†] mucosa					
No	78/468	1.0*	0.01	1.0*	0.03
Yes	127/514	2.0 (1.2–3.5)		1.8 (1.1–3.0)	
Plaque[‡]					
No	51/291	1.0*	0.5	NI[††]	
Yes	376/704	1.9 (1.2–2.9)			
Pocket depth					
0–3 mm	44/379	1.0*	0.002	NS[‡‡]	
4+	161/608	2.3 (1.4–4.0)			
Pus					
No	111/784	1.0*	<0.001	1.0*	<0.001
Yes	94/203	6.8 (3.4–13)		5.0 (2.7–9.2)	
Bleeding					
No	13/133	1.0*	0.08	NS[‡‡]	
Yes	192/854	2.2 (0.90–5.1)			
On patient level					
Gender					
Male	117/509	1.0*	0.14	NI[††]	
Female	88/478	0.52 (0.22–1.2)			
Age (years)					
⩽59	64/182	1.0*	0.009	NS[‡‡]	
60–74	104/509	0.47 (0.20–1.1)			
75+	37/296	0.19 (0.07–0.55)			
School[§](years)					
<12	174/841	1.0*	0.04	NS[‡‡]	
⩾12	27/140	0.34 (0.12–0.96)			
Smoking					
Never	29/301	1.0*	<0.001	1.0*	<0.001
Ex-smoker	49/379	1.8 (0.69–4.8)		1.6 (0.61–4.0)	
Smoker	127/307	17 (6.1–50)		10(4.1–26)	
General disease					
No	132/596	1.0*	0.09	NS[‡‡]	
Yes	73/391	0.51 (0.23–1.1)			
Visit hygienist[¶]					
<1/year	136/683	1.0*	0.4	NI[††]	
⩾1/year	68/297	1.4 (0.66–3.2)			
Visit dentist[‖]					
<1/year	54/317	1.0*	0.5	NI[††]	
⩾1/year	150/663	1.5 (0.48–4.7)			
% Teeth with bone loss[**]					
0–30	26/185	1.0*	0.004	NS[‡‡]	
31–100	128/458	4.6 (1.6–13)			

*Reference category.
†Five study implants with missing information.
‡Three study implants with missing information.
§Number of years in school; one patient with six study implants had missing information.
¶Number of visits at hygienist per year; one patient with seven study implants had missing information.
‖ Number of visits at dentist per year; one patient with seven study implants had missing information.
**Three hundred and forty-four study implants (62 patients) with missing information.
††Not included in the multivariate analysis.
‡‡Not significant in the multivariate analysis.
OR, odds ratio; CI, confidence interval.

On the implant level, the amount of keratinized mucosa was explanatory for mucositis as well as a bone level at ⩾3 threads in both the univariate and multivariate analyses. These results are contradictory to the results reported by Block et al. (1996), suggesting that the absence of keratinized mucosa around implants is correlated to soft and hard tissue health. However, other long-term studies have not been able to demonstrate that an adequate with of keratinized mucosa is essential in order to maintain a clinical healthy condition at dental implants (Wennström et al. 1994, Bengazi et al. 1996, Zitzmann et al. 2001). The finding in our study, that the presence of keratinized mucosa was explanatory for mucositis, defined as probing pocket depth ⩾4 mm and BOP, could possibly be related to the fact that recession, and thereby less pocket formation may be more common in areas without keratinized mucosa.

On the implant level, the presence of pus was explanatory for a bone level at ⩾3 threads in both the univariate and multivariate analyses. In a recent consensus report by Lang et al. (2004), the need for probing measurements and registrations of bleeding and the presence of pus were recommended. Our data support that it is essential to monitor the clinical conditions around implants using probing and registration of pus, as the presence of pus was indicatory of peri-implant bone level at ⩾3 threads.

On the patient level, smoking was significantly associated with mucositis, bone level at ⩾3 threads and peri-implantitis. These results are in concordance with the results reported previously by Lindquist et al. (1996, 1997), Carlsson et al. (2000), and Ekelund et al. (2003), who also found more advanced peri-implant bone loss around Brånemark implants among smokers than non-smokers. Smoking as a risk factor for peri-implantit disease has been highlighted in several other studies (Haas et al. 1996, Esposito et al. 1998b, Leonhardt et al. 2003, McDermott et al. 2003, Baelum & Ellegard 2004), and the data of our long-term study support the concept that smoking should be regarded as a risk factor for development of peri-implantitis.

Another factor associated with peri-implantitis was bone loss at teeth at the time of implant placement. Loss of bone around existing teeth is an obvious sign of, at least a previous presence, of periodontal disease. We think that a bone loss ⩾4 mm at ⩾30% of measurable approximal surfaces at existing teeth can be used to categorize patients into periodontitis/nonperiodontitis patients. This method of categorizing the patient group is probably more reliable than using anamnesis data and/or assuming that all patients treated at a periodontal clinic are periodontal

Table 3. Analysis of potential explanatory variables for the outcome event peri-implantitis

Explanatory variable	Outcome #yes/total	Univariate analyses		Multivariate analysis	
		OR (95% CI)	*p*	OR (95% CI)	*p*
On implant level					
Keratinized[†] gingival					
No	24/468	1.0*	0.13	NI[††]	
Yes	42/514	1.8 (0.84–3.7)			
Plaque[‡]					
No	14/291	1.0*	0.2	NI[††]	
Yes	52/693	1.7 (0.73–3.8)			
On patient level					
Gender					
Male	37/509	1.0*	0.8	NI[††]	
Female	29/478	0.89 (0.32–2.4)			
Age (years)					
⩽59	21/178	1.0*	0.8	NI[††]	
60–74	30/509	0.62 (0.12–3.2)			
75+	37/296	0.52 (0.09–3.1)			
School[§](years)					
<12	54/845	1.0*	0.7	NI[††]	
⩾12	8/136	0.79 (0.24–2.6)			
Smoking					
Never	12/301	1.0*	<0.001	1.0*	0.002
Ex-smoker	7/383	0.52 (0.15–1.9)		0.42 (0.09–12.1)	
Smoker	47/303	7.7 (2.5–24)		4.6 (1.1–19)	
General disease					
No	45/592	1.0*	0.3	NI[††]	
Yes	21/395	0.58 (0.21–1.6)			
Visit hygienist[¶]					
<1/year	39/683	1.0*	0.2	NI[††]	
⩾1/year	27/297	2.1 (0.67–6.4)			
Visit dentist[‖]					
<1/year	18/313	1.0*	0.6	NI[††]	
⩾1/year	48/667	1.3 (0.48–3.7)			
% Teeth with bone loss[**]					
0–30	4/185	1.0*	0.01	1.0*	0.05
31–100	42/458	7.7 (1.5–39)		4.7(1.0–22)	

*Reference category.

[†]Five study implants with missing information.

[‡]Three study implants with missing information.

[§]Number of years in school; one patient with six study implants had missing information.

[¶]Number of visits at hygienist per year; one patient with seven study implants had missing information.

[‖] Number of visits at dentist per year; one patient with seven study implants had missing information.

[**]Three hundred and forty-four study implants (62 patients) with missing information on bone loss were excluded from the multivariate analysis (i.e., 344 study implants were excluded). Based on the 344 study implants with missing information on bone loss, the effect of smoking was significant ($p = 0.009$) and of similar magnitude: *ex-smokers* (two PERIIMPL out of 135 implants) *versus never smokers* (four out of 118), OR = 0.56 (95% CI: 0.06–4.9); *smoker* (14 out of 87) vs. *never smokers* (four out of 118), OR = 9.4 (95% CI: 1.4–62).

[††]Not included in the multivariate analysis.

OR, odds ratio; CI, confidence interval.

patients. Using our definition of a periodontitis patient, a history of periodontitis was significantly associated with the presence of peri-implantitis after 9–14 years. This is in concordance with the results from a recent systematic review by van der Weijden et al. (2005), who concluded that the outcome of implant therapy in periodontitis patients may be different compared with individuals without such a history. Patients previously treated for periodontitis seem to run a greater risk for loss of supporting bone and implant loss, as compared with individuals without such a history. Baelum & Ellegaard (2004) reported 78% 10-year survival rates for one-stage implants placed in periodontitis patients. This figure is lower than what has been observed in many other long-term follow-up studies of implant survival, indicating that prognosis may be less favourable in patients with a history of periodontitis. Wennström et al. (2004), on the other hand, reported implants to be stable in periodontitis patients in a 5-year prospective study. It is, however, important to mention that the patients in their study had an individualized maintenance care programme. In another recent follow-up study on implants placed in periodontally healthy patients, patients with generalized aggressive and patients with generalized chronic periodontitis, the attachment loss after 3 years was greater around implants than around teeth and patients in the aggressive group demonstrated the greatest bone loss (Mengel & Flores-de-Jacoby 2005). The results from our long-term study and other cited papers, indicating that patients with a history of periodontal disease may be more prone to developing peri-implant lesions, support the concept that peri-implantitis may share risk factors with periodontal disease.

In conclusion, this long-term study demonstrated the following:

- the presence of keratinized mucosa was associated with mucositis and bone level at ⩾3 threads;
- pus was indicative of bone level at ⩾3 threads;
- smokers had more mucositis, bone level at ⩾3 threads and peri-implantitis; and
- periodontitis patients had more peri-implantitis.

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Clinical Relevance

Scientific rationale for study: Although implant treatment is a well-documented procedure with favourable long-term results, infections do occur. It is therefore important to analyse implant-specific and patient-related factors that may explain why some individuals are more likely to be affected by such infections than others.

Principal findings: The absence of keratinized mucosa, smoking and a history of periodontal disease were found to be associated factors for infections around implants.

Practical implications: Smokers and periodontitis patients should be monitored more closely in a supportive periodontal programme.