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Risk profile development in subjects during supportive periodontal therapy

Sviluppo di un profilo di rischio in pazienti in terapia parodontale di sostegno

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Summary

We determined if a risk profile (Spider web) at the subject level could predict future progression of periodontal disease. Data from 110 subjects participating in a recall program for > 8 years were studied, IL-1 gene, and smoking status, medical history, and the extent of bleeding failed to predict tooth loss in a 3 –year period of periodontal maintenance.

There is a need for future development of more accurate periodontal risk assessment tools.

Riassunto

Abbiamo cercato in questo lavoro di determinare se un profilo di rischio (Spider web) a livello individuale sia in grado di predire la futura progressione della malattia parodontale. Sono stati studiati i dati provenienti da 110 pazienti che partecipavano ad un programma di richiami per un periodo superiore a 8 anni e sia il gene IL-1 che i dati relativi al fumo, l'anamnesi medica e la quantità di sanguinamento al sondaggio non si sono dimostrati in grado di predire la perdita di denti durante un periodo di 3 anni di mantenimento parodontale.

È quindi necessario lo sviluppo di strumenti di accertamento di rischio parodontale più accurati.

Introduction

Periodontal risk assessment is the foundation for periodontal clinical decision-making and successful periodontal therapy. The current information on periodontal conditions for the purpose of risk assessment is complicated. The prevailing number of studies has used the periodontal site as the unit of observation. Many risk factors for periodontitis are subject specific. It is therefore important to study subject-based risk for periodontitis. There are several reviews on periodontal risk management and risk factors (i.e Persson 2008). Thus, repeated bleeding on probing at several different time points have been suggested to predict future tooth loss (Schätzle et al. 2004). There appears to be insufficient evidence to establish if a positive IL-1 genotype status contributes to periodontitis and/or treatment outcomes (Huynh-Ba et al. 2007). Further, increasing evidence suggests a strong causal link between smoking and periodontitis (Hujoel et al. 2003). Systematic reviews have identified that diabetes mellitus and cardio-vascular diseases are associated with an elevated risk for periodontitis (Salvi et al. 2008, Persson & Persson 2008). Analysis of full-mouth radiographs has shown that reduced marginal bone level is associated with a higher risk for tooth loss in a 5-year period (Bahrami et al. 2008).

The aim of the present study was to determine whether or not the risk profile (Spider web) at the subject level could predict a future progression of periodontal disease.

Material and Methods

The Ethical Committee of the Canton of Bern, Switzerland, approved the study. The study was conducted between March 2007 and February 2008. These subjects had previously been treated for chronic periodontitis as defined by the American Academy of Periodontology (1999) and received an individualized periodontal recall at least since 1999 (Lang et al. 2000, Agerbaek et al. 2006). The present study cohort included 110 subjects of the original 323 subjects who consented to participate, and who were still available in the supportive periodontal therapy (SPT) program.

Clinical study procedures

Probing pocket depth (PPD) measurements were performed using the Michigan probe (-3-6-8-11-, Deppeler S.A. Rolle, Switzerland) at six sites per tooth. Immediately thereafter, bleeding on probing (BOP) was recorded as present or absent at four sites per tooth. The proportional distribution of sites with BOP was calculated. Comparing clinical data between 2005 and the current examination (2008), the number of teeth lost and the number of SPT visits was accounted for. Information on smoking habits, (former smokers were identified as non-smokers) and medical history was also collected.

Statistical Data Analysis

Descriptive and analytical statistics were used. Independent *t* tests (equal variance not assumed) were used for parametric data, and Mann-Whitney U test was used for nonparametric data. Paired T-tests were used to assess within subject changes. Odds ratios were calculated using the Mantel-Haenszel common odds ratio statistics. Stepwise linear regression analysis was used to identify what factors were explanatory to tooth loss due to periodontitis between 2005 and the current examination. Significance was declared at the $p<0.05$ level.

Results

Subject characteristics are listed (Table 1). Statistical analysis demonstrated gender differences only in regards to heart conditions where men had a higher prevalence rate ($p<0.05$). Furthermore, the number of PPDs > 4.0mm was significantly higher among men ($p<0.05$), and with a similar trend for PPDs ≥ 6.0mm ($p=0.06$). No gender differences were found for Il-1 gene status, smoking status, the number of remaining teeth, the number of teeth lost, BOP, or the number of SPT visits (equal variance not assumed). Subjects with reported osteoporosis were significantly older ($p<0.01$) Statistical analysis failed to demonstrate that other medical, or any of the dental conditions differed by age. The distribution of systemic conditions by gender is reported (Table 2).

Since 1999, no tooth losses were found in 65.5% of the subjects participating in the present study. During the latest 3-year period no tooth losses were found in 79.1%, and two or more teeth lost were found in 11.8% of the subjects. Related to the number of SPT visits, subjects tended to loose more teeth when they had more SPT visits. Only 2 subjects had lost more than 10 teeth. On average (all subjects included), the subjects had lost 0.1 tooth per year (S.D. ± 0.4).

Table 1. Subject characteristics

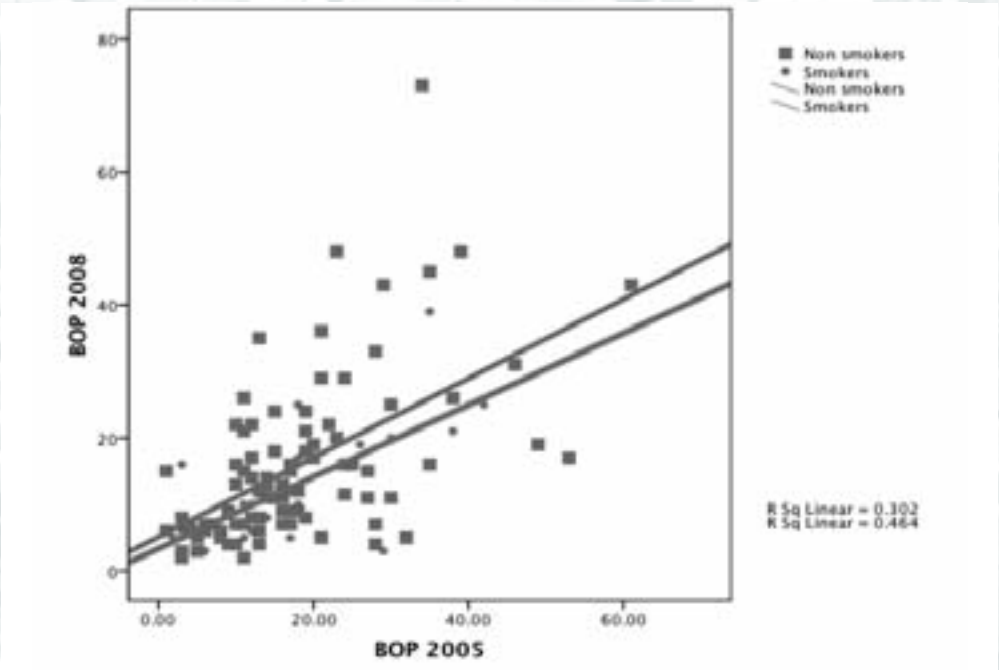
Age-distribution	<50 years	50-65 years	>65 years
(%)	33.6	28.2	28.2
Gender	Female	Male	
Number (%)	70(63.6)	40(36.4)	
Number of teeth	<20 teeth	20-27 teeth	28 teeth
(%)	31.8	64.6	3.6
Smoking status	Smokers	Non-smokers	
2005 (%)	34.5	65.5	
2008 (%)	20.0	80.0	

Table 2. Systemic condition by genderi 2008

Gender	IDDM	Blood pressure	Stroke	Heart sound	Heart diseases	Osteoarthritis	Osteoporosis	Depression
Women	1.4	22.9	2.9	4.3	5.7	8.6	14.3	8.6
Men	5.0	40.0	2.5	10.0	20.0	5.0	5.0	2.5
Sign	NS	NS	NS	NS	0.05	NS	NS	NS

During the last three years, 10.9% (12 subjects) had more than 10 SPT visits (mean: 7.0, S.D ± 3.0). BOP (> 20% of sites) was found in 26 subjects (23.6%), PPDs ≥ 4mm in 90 subjects (81.8%), and PPD ≥ 6mm in 32 subjects (29.1%). The relationship between BOP scores in 2005 and 2008 are presented in a scatter-plot diagram (Figure 1). The likelihood ratio that subjects with a BOP ≤ 20% in 2005 remained ≤ 20% in 2008 was 7.4 (95%CI: 2.8 – 19.6, p<0.001) (sensitivity: 50%, specificity: 88.2). Pair-wise t-test failed to demonstrate differences in the proportions of sites with PPD ≥ 6mm between the two time points (p=0.47). In 2005, 34.5% of the subjects were smokers and this proportion was reduced to 20.0% in 2008. This change was statistically significant (p<0.001). The proportion

Figure 1. Scatterplot diagram illsutrating the realtionship between BOP (%) in 2005 and 2008.



of sites with PPD ≥ 6mm was significantly higher in 2008, among subjects who in 2005 were smokers (mean diff: 3.8%, 95%CI: 0.7-6.9, p<0.05).

The impact of BOP on tooth loss due to periodontitis

Statistical analysis failed to demonstrate that BOP > 20% had an impact on tooth loss between 2005 and 2008 (OR: 0.7:1 95%CI: 0.3-1.6, p=0.38).

The impact of PPD ≥ 6mm on tooth loss due to periodontitis
Statistical analysis failed to demonstrate that PPD ≥ 6mm was predictive of tooth loss between 2005 and 2008 (OR: 2.6:1, 95%CI: 0.9-8.4, p=0.09).

The impact of IL-1 gene status on BOP, PPD and tooth loss due to periodontitis
Using the BOP ≤ 20% as the cutoff value, the relationship between BOP and IL-1 gene status was neither significant in 2005 (OR: 0.7, 95%CI: 0.3-1.6, p= 0.37) nor in 2008 (OR: 1.5, 95%CI: 0.6-3.6,p=0.41). Statistical analysis also failed to demonstrate that IL-1 gene status was predictive of having PPD ≥ 6mm in 2005 (OR: 0.7:1, 95%CI: 0.3-1.7,P=0.46) or in 2008 (OR: 0.9, 95%CI: 0.4-2.2, p=0.88). Further analysis also failed to demonstrate that IL-gene status was predictive of future tooth loss (OR: 1.0, 95%CI: 0.3-3.3, p<0.98).

The impact of smoking on BOP, PPD, and tooth loss
Analysis by Mantel-Haenszel odds ratio calculations failed to demonstrate that smoking status in 2005 was predictive of BOP > 20% (OR: 0.8:1, 95%CI: 0.3-2.1,p=0.64), or the presence or absence of PPD ≥ 6 mm (1.2, 95%CI: 0.5-2.8,p=0.67), or tooth loss (OR: 2.1, 95%CI: 0.7-6.5, p=0.20).

The accuracy of the "spider web" risk assessment model
In 2005, 89 subjects were classified either in a high-risk (group 1 = 32 subjects), or a low-risk group (group 2 = 57 subjects) in accordance with the risk factors of the spider web (Persson et al. 2003). These subjects were also classified as outcome-risk subjects based on the absence of tooth loss within the last three years, having no periodontal sites with PPD ≥ 6mm and < 21% of periodontal sites with BOP (Table 3). The specificity and sensitivity for these risk categories from 2005 was 41.2% resp. 67.9% (Table 4).

Table 3. 2005 groups were classified according to the spiderweb, 2008 group 2 was classified no PPd≥6 mm, BOP<21% and no tooth loss between 2005 and 2008

Risk	**Risk-category 2005**	**Outcome-risk 2008**
High (number of subjects)	Group 1 (32)	Group 1 (53)
Low (number of subjects)	Group 2 (57)	Group 2 (36)

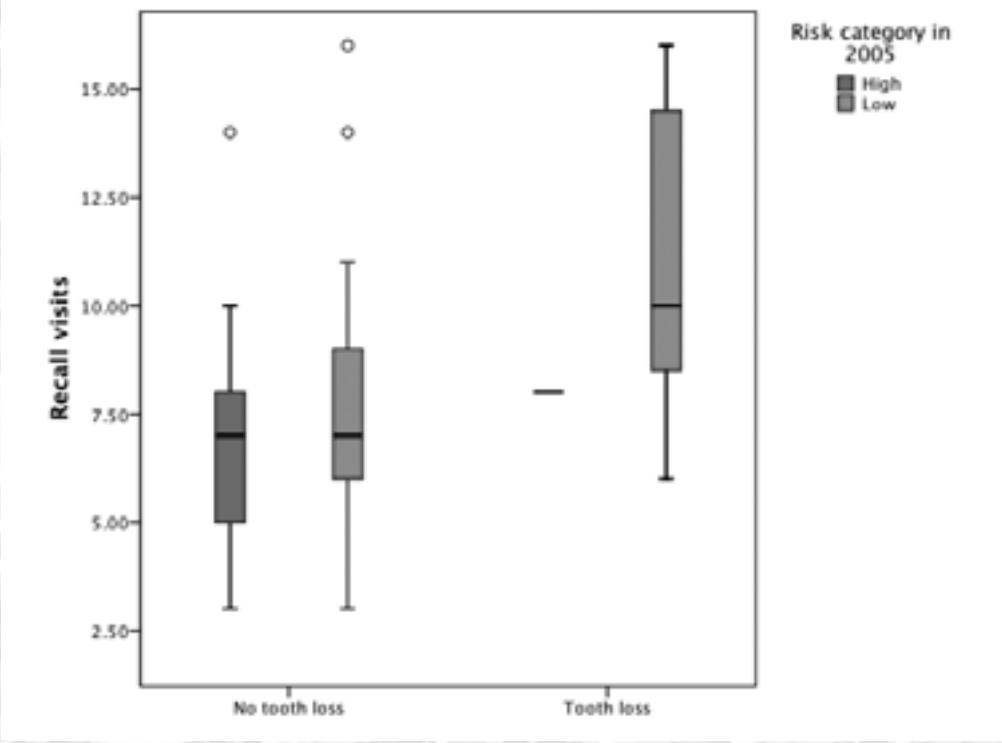
Table 4. Calculation for specificity and sensitivity for the risk-caregories from 2005

	Risk categories in 2005		Total
	Low risk	**High risk**	
Low risk category in 2008	21	15	36
High risk category in 2008	36	17	53
Total	57	32	89

The decrease in the proportion of sites with BOP > 20% in the low-risk group was 3.3% and 1.1% in the high-risk group. This reduction was significant in the low-risk group ($p < 0.05$) but not in the high-risk group. On average, the number of sites with a PPD ≥ 6mm decreased in the low-risk group by, on average, 0.2 sites (S.D. ± 0.2), but increased by, on average, 0.6 sites in the high-risk group (S.D. ± 0.3) ($p < 0.05$).

Statistical analysis failed to demonstrate that the number of SPT visits differed by the periodontal risk categories defined in 2005. Subjects in the low-risk group had, however, lost more teeth due to periodontitis (mean: 0.3, S.D.±0.7) than subjects in the high-risk group (mean: 0.1, S.D.±0.4)($p=0.03$). The distribution of the number of SPT visits and tooth loss due to periodontitis by risk groups is presented (Figure 2).

Figure 2. Box-plot diagram illustrating the distribution of SPT visits in relation to tooth loss due to periodontitis and risk group assignments.



Analysis by linear stepwise regression demonstrated that, when all subjects were included, only the number of SPT visits within the study period was explanatory to the number of teeth lost due to periodontitis ($p < 0.01$).

Discussion

The present study population included 34,5% of the original 1999 study cohort

and 72.8% of the remaining subjects in 2005. Thus, a rather large proportion of subjects were lost to follow-up. This is consistent with many reports. In spite of an individualized SPT program approximately 1/3 of the subjects had lost teeth. Thus, the risk assessment tool might have identified the high-risk subjects, but the SPT failed to prevent further tooth loss. It should be recognized that only a few subjects lost more than 10 teeth and that in fact, the average tooth loss was approximately 0.1 per year and consistent with many other reports (i.e. Löe et al.1986).

The data demonstrated that the extent of BOP remained stable between 2005 and 2008 but also that the identified low-risk group improved oral hygiene as reflected by a further reduction in BOP, which did not occur in the high-risk group. Although the focus of intervention was on subjects and sites with PPD ≥ 6mm in the SPT program, this did not result in a reduction of PPD ≥ 6mm during this recall period. The reduction in the number of subjects who remained smokers in 2008 reflects that participation in the SPT program motivated subjects to a health behavioral change. In the present study, the impact of smoking was evident by the higher proportion of sites with PPD ≥ 6mm in 2008 among those who reported a smoking habit in 2005.

The present study demonstrated that a one-time assessment of BOP in subjects on supportive periodontal therapy could not predict future tooth loss. Others have reported that repeated BOP is a predictor of future tooth loss (Schätzle et al 2004). Although it may require several assessments of BOP over time to predict future tooth loss, the present study demonstrated that the subjects in the low-risk group despite a reduction in BOP still had more tooth loss due to periodontitis than the high-risk group.

The fact that PPD ≥ 6mm was not a predictor for future tooth loss may be explained by the short observation period and by the fact that teeth irrational to treat might already have been extracted. Another explanation why the PPD ≥ 6mm variable could not predict future tooth loss may be due to the fact that PPDs ≥ 6mm had remained stable over several years. The current data confirm the previous findings from 2005 that Il-1 gene status was not predictive of BOP, and PPD status or tooth loss due to periodontitis. These findings are consistent with others who have concluded that Il-1 gene status may be less significant than previously considered (Huynh-Ba et al. 2007). The association between BOP and Il-1 gene status reported from the original cohort of 323 subjects in 1999 differ from both the 2005 and the present report and may be explained by the decreasing number of subjects.

The impact of smoking on clinical conditions such as BOP and PPD ≥ 6mm appears to depend on definition of these variables. Thus, dichotomized data may not yield the same results as analysis by proportional distributions of BOP and PPD ≥ 6mm and may explain differences in opinion on the role of smoking on periodontal disease risk.

It is of interest that the number of SPT visits was the only variable that was predictive of future tooth loss. This was, however, only applicable to subjects in the

defined low-risk group from 2005 to suggest that the risk assessment tool has limitations in predicting future tooth loss. One explanation to why the risk assessment tool was not predictive could be linked to the fact that more tooth loss was found in the low-risk group who also demonstrated a reduction in BOP during the current study period. The fact that several subjects quit smoking might also have effects on future periodontal disease risk including tooth loss.

In conclusions, the periodontal risk assessment tool (the spider web) is limited in predicting future tooth loss. This may be due to the fact that periodontitis is a multi-factorial disease where all factors were not weighted in when the risk assessment tool was developed. Further, there may be currently unknown factors influencing periodontitis risk. However, it still presents a reliable tool to assess subject needs for SPT in preventing the progression of periodontitis. There is a need for future development of more accurate periodontal risk assessment tools.

References

– Agerbaek MR, Lang NP, Persson GR. Microbiological composition associated with Interleukin-1 gene polymprphism in subjects undergoing supportive periodontal therapy. J Periodontol 2006;77:1397-1402.
– Bahrami G, Vaeth M, Kirkevang LL, Wenzel A, Isidor F. Risk factors for tooth loss in an adult population: a radiographic study. J Clin Periodontol. 2008;35:1059-65.
– Hujoel PP, del Aguila MA, DeRouen TA, Bergström J. A hidden periodontitis epidemic during the 20th century? Community Dent Oral Epidemiol. 2003;31:1-6.
– Huynh-Ba G, Lang NP, Tonetti MS, Salvi GE. The association of the composite IL-1 genotype with periodontitis progression and/or treatment outcomes: a systematic review. J Clin Periodontol. 2007;34(4):305-17.
– Lang NP, Tonetti MS, Suter J, Sorrell J, Duff GW, Kornman KS. Effect of interleukin-1 gene polymorphisms on gingival inflammation assessed by bleeding on probing in a periodontal maintenance population. J Periodontal Res. 2000;35(2):102-7.
– Löe H, Anerud A, Boysen H, Morrison E. Natural history of periodontal disease in man. Rapid, moderate and no loss of attachment in Sri Lankan laborers 14 to 46 years of age. J Clin Periodontol. 1986;13(5):431-45.
– Persson GR. Perspectives on periodontal risk factors. J Int Acad Periodontol. 2008;10:71-80.
– Persson GR, Persson RE. Cardiovascular disease and periodontitis: an update on the associations and risk. J Clin Periodontol. 2008;35(8 Suppl):362-79.
– Salvi GE, Carollo-Bittel B, Lang NP. Effects of diabetes mellitus on periodontal and periimplant conditions: update on associations and risks. J Clin Periodontol. 2008;35 (8 Suppl):398-409.
– Schätzle M, Löe H, Lang NP, Bürgin W, Anerud A, Boysen H. The clinical course of chronic periodontitis J Clin Periodontol. 2004;31(12):1122-7.