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Effect of treatment of chronic periodontitis on levels of serum markers of acute-phase inflammatory and vascular responses

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Abstract

Aims: Recent epidemiological work suggests an association between periodontal disease severity and cardiovascular disease risk. This study aimed to ascertain if circulating levels of cardiovascular and systemic inflammatory markers could be modified following treatment of periodontal disease.

Method: Adult subjects were recruited from those awaiting periodontal treatment and randomised to either immediate (test, $n = 24$) or delayed treatment (control, $n = 15$). Demographic and clinical data were collected and venous blood was taken before and either 6 weeks after completion of treatment or after an equivalent 3-month control period. Periodontal examination included probing depth, loss of attachment, plaque scores and bleeding scores. Blood was analysed to determine serum and plasma fibrinogen, C-reactive protein, sialic acid, tumour necrosis factor-α and interleukin -6 and -1β. Effects of treatment were assessed by paired tests and analysis of variance by treatment group with baseline covariates.

Results: Treatment improved plaque and bleeding scores and reduced probing depths ($p < 0.002$). However, there were no statistically significant changes in levels of any of the systemic markers.

Conclusion: Improvement in periodontal health did not influence the levels of vascular markers.

Key words: periodontitis; treatment; acute-phase proteins; assessment; human; clinical trial

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Periodontitis has been proposed as having an aetiological or modulating role in cardiovascular and cerebrovascular disease (Matilla et al. 1989, Beck et al. 1996, Morrison et al. 1999, Wu et al. 2000a), diabetes (Grossi & Genco 1998), respiratory disease (Hayes et al. 1998) and adverse pregnancy outcome (Offenbacher et al. 1996). There is still much debate regarding the nature and degree to which this may happen (Joshipura et al. 1996, 1998, Hujoel et al. 2000, 2001a,b, Matilla et al. 2000, Howell et al. 2001).

Several mechanisms have been proposed to explain or support such theories. One of these is based around the potential for the inflammatory phenomenon of periodontitis to have effects by the systemic dissemination of locally produced mediators such as C-reactive protein (CRP), interleukins -1 beta (IL-1β) and -6 (IL-6) and tumour necrosis factor alpha (TNF-α) (Gemmell et al. 1997, Kornman et al. 1997). This concept has been supported by work suggesting that elevated levels of a number of inflammatory molecules (together with sialic acid (SA)) may be accurate indicators of cardiovascular risk. In addition to profound changes following trauma or infection, there are long-term interindividual variations in concentrations of these molecules (Danesh et al. 1998, Ridker et al. 1999), at levels which until recently have been too low to be reliably determined. A series of investigations have suggested that the levels of these molecules can predict increased risk of cardiovascular disease (Lindberg et al. 1991, Liuzzo et al. 1994, Anderson et al.

1998, Ridker et al. 1998, Danesh et al. 2000, Ridker et al, 2000a,b). Furthermore, it has been proposed that patients with periodontitis may have elevated circulating levels of some of these inflammatory markers (Kornmann & Holt 1994, Offenbacher et al. 1996, Beck et al. 1998, Page 1998), although there are limited published data to support this (Collins et al. 1994, Ebersole et al. 1997, Offenbacher et al. 1998, Wu et al. 2000b, Noack et al. 2001).

The acute-phase response is a nonspecific process that may occur in the initial host response to injuries, infections, ischaemic necrosis or malignancy. It is initiated by the activation of local macrophages and other cells (including fibroblasts and endothelial cells), leading to the release of mediators such as TNF-α, IL-6 and IL-1β. These in turn cause systemic changes including hepatic release of a range of plasma proteins (including CRP), activation of complement proteins and various metabolic changes (Koj 1996, Ebersole & Capelli 2000). IL-6 also promotes induction of fibrinogen (fib), haptogloblin, α1-antitrypsin and α2-macrogloblin among others. CRP and other acute-phase molecules are usually present at relatively low levels in plasma, but may be raised dramatically within 72 h of tissue injury or with infection. CRP opsonises bacteria for complement binding and activates complement when complexed. CRP, IL-1β, IL-6 and TNF-α have been associated with the presence of various bacterial infections including periodontitis (Borden & Chin 1994, Steel & Whitehead 1994, Trautwein et al. 1994, Ebersole et al. 1997, Wagge & Steinshamn 1997). Sialic acid (*N*-acetylated neuraminic acid derivatives that form part of various glyco-proteins and glycolipids) is present in several acute-phase proteins, and so one may expect changes in circulating levels of sialic acid to mirror changes in acute-phase proteins (Sillanaukee et al. 1999).

The aim of this prospective interventional cohort study was to determine if nonsurgical treatment of moderate to advanced chronic periodontitis could influence circulating serum levels of these acute-phase proteins and inflammatory molecules. We chose to examine nonsmoking subjects to avoid the confounding effects of this important factor.

Material and Methods

Subjects

Ethical committee approval was obtained from Guy's and St. Thomas' Hospital local Ethics Committee. Subjects were recruited from nonsmoking patients aged between 30 and 60 years on the waiting list for nonsurgical treatment of moderate to advanced chronic periodontitis at GKT Dental Institute. Each subject had at least 20 standing teeth, none of which had untreated periapical lesions, and with at least five teeth with sites probing 5 mm or deeper, and radiographic evidence of alveolar bone loss. Subjects had received no previous subgingival periodontal debridement or periodontal surgery in the preceding 6 months, and had no antimicrobial therapy in the previous 6 months. Patients regularly taking any of the following drug types in the previous 6 months were also excluded: anti-inflammatories, steroids, immunosuppressants, statins, lipid-lowering drugs or anticoagulants.

Patients were not included in the study if they were suffering from any chronic inflammatory or immunological conditions, such as arthritis, gastrointestinal disorders, skin conditions, bronchitis or other chronic obstructive airway diseases, if they were diabetic, pregnant, lactating or planning a pregnancy, or if they were current smokers or had been in the previous 5 years.

Subjects were stratified for age and gender and randomly allocated to one of two groups: Group A: immediate periodontal treatment; or Group B: periodontal treatment following a 3-month nontreatment phase. Forty subjects were initially recruited to the study; however one subject was lost to follow-up, reducing the numbers involved and completing the study to 39 subjects. These were divided such that group A (immediate treatment) included 24 subjects and group B (delayed treatment) included 15 subjects.

Baseline data were collected for all subjects, comprising medical, dental and social histories by questionnaire and clinical periodontal examination. The questionnaire considered medical factors that may influence periodontal treatment, the general medical status with particular emphasis on cardiac disease and chronic inflammatory conditions, dental attendance and oral hygiene regimes. Self-reported alcohol consumption and previous tobacco usage were also determined.

Subjects provided two 10 ml venous blood samples (5 ml plain (serum) and EDTA (plasma) tubes) and underwent a standardised periodontal examination, before and 6 weeks following completion of periodontal treatment (Group A) or at the beginning and end of the 3-month nontreatment period (Group B).

Periodontal assessment was carried out at each standing tooth on four sites, namely the mesio-lingual, mesio-buccal, disto-lingual and disto-buccal sites. The variables of interest were plaque (plaque index; Silness & Löe 1964), clinical probing depth and bleeding on probing. Clinical probing depth was assessed to the nearest millimetre using a constant force periodontal probe (Yeaple design, Vine Valley Manufacturing Inc., NY, USA), with an applied load of 20 g. Probe tips were of a 0.35 mm width with a rounded working end. The presence or absence of bleeding within 30 s of probing was recorded.

Following initial assessment, nonsurgical periodontal treatment was scheduled for Group A. Subjects were treated with a course of five 90-min appointments on a weekly basis, by senior hygienist students, under the supervision of a specialist periodontist. Treatment was structured as follows:

Appointment 1: Intensive oral hygiene instruction, including plaque disclosing, tooth brushing technique (modified Bass), interdental cleaning (floss, twin-spiral and single-tufted brush as appropriate), and supragingival scaling and polishing.

Appointments 2, 3, 4 & 5: Oral hygiene was monitored and reinforced and thorough subgingival debridement was carried out under local anaesthesia on a quadrant-by-quadrant basis using ultrasonic scalers and hand instruments.

Subjects in Group A returned 6 weeks after completion of treatment for a reassessment. After baseline data had been collected, the subjects in Group B were placed in a nontreatment group. During a 3-month period they received no periodontal care. This period ended with a reassessment visit, after which they were treated in exactly the same manner as described for Group A. Consequently, Group B acted as a nontreatment control group for the duration of the study but were not denied treatment in the longer term.

At reassessment, the examiner confirmed all medical, dental and social history details, in particular that no antibiotic therapy had been administered, or that no periodontal treatment had been instigated in the control group. A full periodontal reassessment was carried out and further venous blood samples were taken. Appropriate maintenance and review appointments were organised for all subjects in both study groups.

Analysis of intraexaminer reproducibility

Reproducibility of the examiner was assessed by carrying out clinical periodontal data collection on five patients. Each subject was assessed twice in One visit, over a 1 h interval, as detailed above. The second set of recordings was carried out ''blinded'' to the first assessment. Reproducibility of the data collection was determined by calculation of the percentage of the 780 sites examined where the score was exactly repeated or repeated to an accuracy of ± 1 mm for each site. Ninety per cent of site measurements were repeated with an accuracy of ± 1 mm and 60% of sites were recorded to exactly the same value over the two visits. Assessment of the mean difference in the scores between visits indicated that there was no systematic bias in measurement between visits.

Methods: laboratory analysis

Venous blood samples were analysed to determine concentrations of various recognised cardiovascular risk markers and indicators of systemic inflammation, including CRP, fib, SA, IL-1β, IL-6 and TNF-α. All blood samples collected at initial assessment and reassessment were analysed as one batch.

The samples for serum were centrifuged for 10 min at 11,000 RPM separating the cells from the serum. The serum samples were then immediately divided into 0.2–0.5 ml aliquots and stored at -70°C, until required for analysis.

Samples were assayed for TNF-α, IL-1β and IL-6 using a commercially available quantitative enzyme immunoassay (Quantikine High Sensitivity, R&D Systems Europe Ltd, Abingdon, Oxon, UK). These assays have a lower limit of detection for TNF-α of approximately 0.1 pg/ml, for IL-1β of 0.1 pg/ml and for IL-6 of 0.09 pg/ml and a coefficient of variation between 5% and 8%. Blood specimens were analysed in pairs, with the position of the case specimen varied at random within pairs to reduce systematic bias and limit interassay variability. Laboratory personnel were unaware of case or control status. CRP was assayed using a high-sensitivity latex anti-CRP monoclonal antibody kit with immunonephelometry (Dade Behring, Milton Keynes, UK) on a Boehring nephelometer II analyser, according to the manufacturer's instructions. This method had a lower detection limit of 0.2 mg/l and an interassay coefficient of variation below 2.8%. Likewise fibrinogen was measured turbidimetrically on a Cobas Fara II analyser with an interassay coefficient of variation on below 5%. Sialic acid was assayed using a specific enzymatic linked colorimetric assay (Boehringer Mannheim, Lewes, UK) performed on a Roche Cobas Fara analyser (Welwyn Garden City, UK) as described by Crook (1993). All samples were analysed blind to treatment status.

Statistical methods

Data were analysed using Stata for Windows 7.0 (Stata Corporation, TX, USA). Frequency distributions were determined for all variables. Baseline data were compared using *t*-tests for normally distributed variables, χ^2-test for categorical variables and Mann–Whitney *U*-tests for other variables. Clinical and biochemical parameters at baseline and reassessment were compared within each group using paired *t*-test and Wilcoxon signed ranked matched pairs test for normally and non-normally distributed variables, respectively. Analysis of variance was used to determine the effect of treatment group on clinical and biochemical outcome data with baseline data as covariates for normally distributed variables, otherwise Kruskal–Wallis analysis of variance tests was employed. Finally, Spearman correlation coefficients were calculated between clinical and biochemical variables and between each of the biochemical variables.

Results

In total, 39 subjects completed the study. The characteristics of the population and of the treatment and control groups are shown in Table 1. There were no significant differences between groups at baseline in terms of age, number of teeth, reported oral hygiene and reported previous smoking habits.

Table 2 illustrates the findings for clinical variables for each group at baseline and at the reassessment stage. It can be seen that there were significant improvements in the periodontal health of the treatment group compared to control subjects, with a reduction in plaque and bleeding scores, and in probing depths. However, the circulating levels of the molecules of interest did not change (Table 3). ANOVA failed to identify changes in levels of

Table 1. Population Demographics at Baseline

Variable	Whole population $n = 39$	Group A $n = 24$	Group B $n = 15$	Group A versus Group B p
Gender				
male	23	13	10	0.181#
female	16	11	5	
Mean age: years (SD)	47.1 (7.0)	47.8 (7.5)	46.0 (6.2)	0.302**
Dental History				
Mean number of Standing teeth (SD)	27.1 (2.8)	26.8 (3.0)	27.6 (2.6)	0.522*
oral hygiene behaviour:				
mean brushing No./day (SD)	1.9 (0.5)	1.9 (0.4)	1.9 (0.7)	0.920*
Social history				
percentage previous smokers	46.2%	41.6%	53.3%	0.581**

Mean figures with standard deviations.

*Student's *t*-test.

**Mann–Whitney *U*-test.

#χ^2-Test.

Table 2. Clinical data for test and control groups at baseline and at reassessment, and analysis of variance with baseline scores as covariate to determine effect of treatment group

Variables	Group A ($n = 24$)			Group B $n = 15$			Group A versus Group B ANOVA
	baseline	reassessment	p	baseline	reassessment	p	p
Mean % sites with plaque (SD)	86.08 (20.58)	42.21 (21.04)	**<0.001***	82.74 (17.63)	81.74 (18.29)	0.768*	**<0.001**
% sites with probing depth 0–3 mm, mean (SD)	55.24 (17.98)	71.62 (15.33)	**<0.001***	55.11 (17.43)	58.78. (17.88)	**0.035***	**0.002**
4-6mm, mean (SD)	34.79 (14.47)	23.04 (11.54)	**<0.001***	31.65 (10.43)	29.09 (11.83)	0.062*	**0.041**
⩾7mm, median (interquartile range)	9.00 (4.25– 24.50)	5.50 (3.00–9.75)	**0.005#**	13.24 (6.50–24.50)	12.13 (5.50–26.50)	0.184#	**<0.005**
mean % sites bleeding (SD)	41.75 (17.44)	15.63 (10.37)	**<0.001***	45.36 (23.46)	39.45 (18.85)	0.074*	**<0.001**

*paired *t*-test.

#Wilcoxon signed ranked matched pairs test.

Table 3. Chemical data for test and control groups at baseline and at reassessment, and analysis of variance with baseline scores as covariate to determine effect of treatment group

Variables	Group A $n = 24$			Group B $n = 15$			Group A versus Group B ANOVA
	baseline	reassessment	p	baseline	reassessment	p	p
CRP (mg/l) median (interquartile range)	1.42 (0.63–2.53)	1.28 (0.39–2.45)	0.845**	2.19 (0.26–2.91)	1.91 (0.53–2.90)	0.619**	0.986 *
fibrinogen (g/l) mean (SD)	3.12 (0.70)	3.04 (0.64)	0.841#	3.19 (0.83)	3.08 (0.79)	0.787#	0.983
interleukin-1β (pg/ml) mean (SD)	0.95 (0.67)	0.89 (0.59)	0.771#	0.68 (0.54)	0.76 (0.36)	0.662#	0.540
Interleukin-6 (pg/ml) median (interquartile range)	1.56 (1.07–2.14)	1.96 (0.90–3.29)	0.553**	0.94 (0.76–1.72)	0.91 (0.70–1.81)	0.945**	0.172*
tumour necrosis factor-α (pg/ml) mean (SD)	2.21 (0.53)	2.20 (0.47)	0.915#	2.03 (0.51)	1.84 (0.46)	0.300#	0.091
sialic acid (mg/dl) mean (SD)	71.45 (5.99)	68.63 (6.63)	0.127#	70.53 (7.18)	69.33 (7.61)	0.660#	0.506

Statistical tests: *Kruskal–Wallis ANOVA. **Wilcoxon signed ranked matched pairs test. #Paired *t*-test.

any of the systemic markers following periodontal treatment.

Table 4 illustrates statistically significant correlations between molecular variables. There were moderately strong and statistically significant negative correlations between levels of fibrinogen and levels of CRP and TNF-α (both negative), and significant positive correlations between SA and fib, CRP and IL-6, between CRP and TNF-α and IL-6, and between IL-6 and TNF-α and SA.

Discussion

The baseline biochemical data reported above are similar to those reported previously for each of the molecules of interest (Crook & Tutt 1992, Liuzzo et al. 1994, Chen et al. 1996, Mendall et al. 1996, Haverkate et al. 1997, Anderson et al. 1998, Crook et al. 1998a,b, 2000, Rifai et al. 1999, Danesh et al. 2000, Ridker et al. 2000a,b, Wu et al. 2002).

The present study may be compared with that of Ebersole et al. (1997). While Ebersole et al. observed a relationship between CRP levels and the presence or absence, or severity of, adult periodontitis, it is not apparent whether their study design controlled for the effects of smoking, a confounder for both periodontitis and CRP concentration. The levels reported in their disease group as a whole were slightly lower than those seen in the present study. Ebersole et al. also failed to observe a reduction in circulating CRP following nonsurgical periodontal treatment, although there was no indication of the efficacy of this treatment. However, they did report a reduction if there was concurrent administration of an anti-inflammatory drug. The potential effect of smoking was illustrated by Fredriksson et al. (1999), who found a median CRP concentration of nonsmoking patients with periodontitis of 2 mg/l (mean age 52 years), compared to a median of 0 mg/l for nonsmoking matched controls and a median CRP of around 2 mg/l regardless of their periodontal status. More recently, Noack et al. (2001) related periodontal status in adults to circulating CRP allowing for smoking, age and obesity, and reported that there was a significant relationship between severity of periodontitis and circulating CRP. The periodontitis subjects in their study had higher CRP than those reported here, which were closer

Table 4. Correlations between biochemical variables; each cell contains Spearman's rho value for pairs of variables and probability that correlation is significant

	CRP	TNF	IL-6	IL-1β	Sialic
	− 0.45, 0.0001	− 0.36, 0.0025	*0.08, 0.510*	*0.13, 0.340*	0.245, 0.0442
CRP	—	0.46, < 0.0001	0.52, < 0.0001	*− 0.15, 0.239*	0.33, 0.0034
TNF	—	—	0.36, 0.0019	*0.02, 0.852*	*0.17, 0.136*
IL-6	—	—	—	*0.04, 0.746*	0.39, 0.0006
IL-1 β	—	—	—	—	*− 0.08, 0.540*

to those of the control subjects of Noack et al. The potential role of smoking in influencing these data is currently uncertain, and Noack's subjects did include smokers.

Slade et al. (2000) reported a relationship between CRP and severity of periodontitis, with a mean CRP of 4.5 mg/l for patients with more than 10% of sites probing 4 mm or greater, compared to 3.3 mg/l in healthy subjects. However in our untreated subjects, at least 40% sites probed 4 mm or more, yet CRP was much lower, and for those who received treatment there was a higher number of residual 4 mm or greater probing sites, but the circulating mean CRP was again lower than that reported by Slade et al. These differences could relate to variances in the populations studied in terms of age, smoking, lifestyle and diet, together with the limitations of the CRP assay employed by Slade et al, which had a detection limit of 3 mg/l. Beck et al. (2000) assessed the relationship between CRP and the periodontal status of two randomly selected quadrants in 12,949 adults. For the whole population, those with more than 10% of sites probing 4 mm or greater had a significantly higher mean CRP of 4.1 compared to 2.9 mg/l, relatively high levels compared to the data here. However, the subjects of Beck et al. included smokers, for whom the relationship between periodontal health and CRP was not as clear.

In another recent publication, Loos et al. (2000) studied 107 subjects with untreated periodontitis and 43 controls and found significant relationships between both circulating CRP and severity of periodontitis and between IL-6 and severity of periodontitis, allowing for educational status, ethnicity, body mass index (BMI), smoking, history of hypertension, plasma cholesterol and infection with cytomegalovirus, *Chlamydia pneumoniae* and *Helicobacter pylori*.

The investigation reported above failed to illustrate changes in CRP or IL-6 following treatment and improvement of periodontal health. Although we utilised a high-sensitivity CRP assay, excluded current smokers and as far as possible subjects with any other infections or inflammatory conditions, we did not assess the effects of obesity, hypertension, education or cholesterol, and it is possible that these confounders may have influenced our findings (Mendall et al. 1996, Noack et al. 2001). Likewise the data presented here are for patients following a single course of periodontal treatment, and hence some residual diseased sites remained, and there may have been insufficient time for biochemical changes to be established after disease reduction. The demographic data for our untreated subjects did not differ to a major degree from those Loos et al. classified as having generalised periodontitis (eight or more teeth with bone loss greater than one-third of root length), although those reported here were slightly older and had fewer teeth with periodontitis. Even so, the untreated subjects in this study had very similar circulating CRP levels to the generalised periodontitis subjects of Loos et al., but higher levels of IL-6. The localised periodontitis subjects of Loos et al. (less than eight teeth with bone loss greater than one-third of root length) had similar CRP concentrations to those who had received treatment described above, but again had lower IL-6 levels. Finally, both the CRP and IL-6 levels were much lower for the Loos et al. control subjects when compared to those who received treatment of periodontitis above. This fits with the observation that the single course of nonsurgical treatment carried out did not eliminate all diseased sites, and hence this may have had some bearing on the results obtained. Our observed significant correlation between CRP and IL-6 concentrations supports those reported by Loos et al. (2000) and Ridker et al. (2000a) (coefficient 0.52 versus 0.47 and 0.43, respectively). These results are consistent with the concept that IL-6 is a major promoter of CRP production. The results reported above are also consistent with those of Christgau et al. (1998), in terms of the effect of treatment on clinical and molecular data.

Other workers have reported considerably lower CRP concentrations than those reported here or in other papers investigating potential relationships with periodontal status (Shine et al. 1981: median of 0.8 mg/l in 483 subjects, 90% of which had CRP levels below 3mg/l, and 99% below 10 mg/l; Roberts et al. 2001: median of 0.9 mg/l in 388 subjects, 75% below 2 mg/l and 95% below 8.4 mg/l; and Wu et al. 2002: 73% of 5342 subjects below 3 mg/l and 94% below 9 mg/l) in healthy populations. In this study, we attempted to eliminate as many other potential confounding conditions as possible at the subject recruitment stage. However, it is certainly feasible that some subjects did have conditions that may have been causing alterations of acute-phase protein levels during the study and which would not have responded to periodontal treatment, but which would, in conjunction with other infections and chronic inflammatory conditions, have had the potential to elevate CRP levels (Armitage 2000). Furthermore, it is possible that the baseline levels of disease seen in this population, and the corresponding reduction in infective and inflammatory ''load'' may not have been sufficient to result in significant changes in circulating molecules. An alternative explanation is that the levels of circulating CRP and cytokines are induced not solely by periodontal diseases but by other ongoing, possibly occult, atherosclerotic or other disease (Haverkate et al. 1997, Armitage 2000, Yasojima et al. 2001) such that reducing the severity of periodontitis will not influence the circulating levels of these cytokines.

Therefore, within the limitations of this study, it appears that a course of nonsurgical treatment of moderate to advanced chronic periodontitis in nonsmoking subjects did not influence circulating serum levels of CRP, fib, IL-1β, IL-6, TNF-α or SA. These findings may suggest that simple periodontal treatment alone may be insufficient to reduce cardiovascular disease risk. However, this was only a short-term study. It would be interesting to determine if there are differences in the

circulating levels of these molecules following more extensive periodontal treatment including surgery (and possibly other adjuncts) and between maintenance patients who are achieving varying degrees of periodontal disease control.

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Zusammenfassaung

Auswirkung der Therapie chronischer Parodontitis auf die Spiegel der Serummarker für entzündliche und vaskuläre Akute-Phase-Reaktionen

Zielsetzung: Neuere epidemiologische Studien legen eine Verbindung zwischen dem Schweregrad von Parodontitis (CPD) und dem Risiko für Herz-Kreislauferkrankungen (CVD) nahe. Diese Studie sollte klären, in wie weit die Serumspiegel kardiovaskulärer und systemischer Entzündungsmarker nach Parodontitistherapie verändert werden.

Methoden: Erwachsene Patienten wurden aus der Gruppe der Personen ausgewählt, die auf eine systematische Parodontitistherapie warteten, und randomisiert entweder einer sofortigen (Test, n = 24) oder einer verzögerten Behandlung (Kontrolle, n = 15) zugewiesen. Demographische sowie klinische Daten wurden erhoben und venöses Blut vor und 6 Wochen nach Abschluss der Therapie bzw. nach einer äquivalenten 3monatigen Kontrollperiode entnommen. Die parodontologischen Untersuchungen umfassten die Erhebung von Sondierungstiefen, Attachmentverlust, Plaque- und Blutungsindizes. Das Blut wurde auf Serum- sowie Plasmafibrinogen, C-reaktives Protein, Sialinsäure, Tumornekrosefaktor-α und die Interleukine–6 sowie -1β untersucht. Therapieeffekte wurden mittels paariger Tests und Varianzanalyse nach Behandlungsgruppe mit Ausgangswerten als Kovariaten analysiert.

Ergebnisse: Die Behandlung reduzierte die Plaque- und Blutungsindizes sowie die Sondierungstiefen ($p < 0{,}002$). Allerdings wurde keine statistisch signifikante Veränderung der Spiegel der systemischen Marker beobachtet.

Schlussfolgerung: Die Verbesserung der parodontalen Gesundheit beeinflusste die Spiegel der Gefäßmarker nicht.

Résumé

L'effet du traitement de la parodontite chronique sur les niveaux des marqueurs sériques des réponses vasculaires et de la phase aigue inflammatoire

Buts: Un récent travail épidémiologique suggère qu'il existe une association entre la sévérité de la maladie parodontale (CPD) et un risque de maladie cardiovasculaire (CVD). Cette étude avait pour but de vérifier si les niveaux circulatoires des marqueurs inflammatoires systémiques et cardiovasculaires pourraient être modifiés par un traitement parodontal.

Méthode: Des sujets adultes dans l'attente d'un traitement parodontal furent recrutés et répartis au hasard pour un traitement immédiat (test, n = 24) ou tardif (control, n = 15). Les données démographiques et cliniques furent récoltées et du sang veineux fut prélevé avant et soit 6 semaines après la fin du traitement ou après une période équivalente de
De contrôle à 3 mois. L'examen parodontal comprenait la profondeur de poche, la perte d'attache et les scores de plaque et de saignement. Le sang fut analysé pour déterminer le fibrinogène, la protéine C-réactive, l'acide sialique, le Tumor Necrosis Factor-a et les interleukines –6 et -1β, plasmatiques et sériques. Les effets du traitement furent mis en évidence par des tests appariés et l'analyse de la variance par groupe de traitement avec covariants initiaux.

Résultats: le traitemant améliorait les scores de plaque et de saignement et réduisait les profondeurs de poche ($p < 0.002$). Cependant, il n'y avait pas de modifications statistiquement significatives des niveaux des marqueurs systémiques.

Conclusion: L'amélioration de la santé parodontale n'influence pas les niveaux des marqueurs vasculaires.

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