ISSN Print: 2394-7489
ISSN Online: 2394-7497
IJADS 2019; 5(2): 80-82

www.oraljournal.com
Received: 04-02-2019
Accepted: 06-03-2019

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Effect of standard periodontal therapy (SPT) on serum inflammatory marker c reactive protein: A pilot study

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Abstract
Aim: The specific aim of this intervention trial was to test the short-term effects of periodontal therapy on the systemic inflammatory status of medically healthy individuals suffering from severe, generalized periodontitis.
Materials and Methods: 30 subjects suffering from severe periodontitis were recruited and received standard periodontal therapy. Blood samples were collected at baseline and 30 days after treatment and processed for biomarker CRP by high-sensitivity assay.
Results: One month after treatment, SPT resulted in significant reductions in levels of serum CRP.
Conclusion: Analysis of the data indicates that periodontitis causes moderate systemic inflammation in systemically healthy subjects and SPT can result in the reduction of the inflammatory markers.

Keywords: Periodontitis, inflammation, C-reactive protein

1. Introduction
Over the last 50 years, the prevailing view among dentists and physicians was that periodontal infections were localized only to the marginal periodontium and they rarely had systemic implications in healthy individuals. However, more recent evidence has indicated that patients with periodontitis present with increased systemic inflammation, as indicated by raised serum levels of various inflammatory markers when compared with those in unaffected control populations (Kweider *et al.*, 1993; Ebersole *et al.*, 1997; Loos *et al.*, 2000; Noack *et al.*, 2001; Buhlin *et al.*, 2002) [1-5]. Chronic low-grade inflammation, measured as elevated C-reactive protein (CRP) serum levels, has been directly associated with the onset and progression of cardiovascular diseases (CVD) (Libby *et al.*, 2002; Pearson *et al.*, 2003) [6-7]. A recent joint consensus conference of the American Heart Association (AHA) and the Center for Diseases Control (CDC) has focused on the clinical utility of these markers in the management of CVD risk (9). In addition to the classical risk estimation based on well-defined markers (such as smoking, obesity, hyperlipidaemia, hypertension, diabetes, age, gender), and due to the available evidence on the role of inflammation in the pathogenesis of atherosclerosis, the consensus conference identified three different risk categories based on serum CRP levels. Pooled epidemiological data from 40,000 subjects have shown that different levels of serum CRP predict future cardiovascular events in otherwise healthy individuals. Based on these observations, subjects with CRP concentrations less than 1 mg/l are considered to be at low risk, whereas those with concentrations in the 1–3 mg/l range are assigned a medium risk level and those with more than 3 mg/l in serum CRP are considered to be at high risk for future cardiovascular disease and events. CRP hepatic production is usually elicited by an inflammatory stimulus and mediated through a complex network of cytokines (mainly IL- 6); nonetheless, several systemic co-factors can influence its concentration (Kluft and de Maat, 2001) [8].
The aim of this pilot intervention trial was to assess whether the degree of individual response to periodontal treatment was associated with changes in serological markers of systemic inflammation, i.e., CRP in otherwise healthy individuals.

2. Materials and Methods
30 subjects presenting with severe (probing pocket depths greater than6 mm and marginal

alveolar bone loss greater than 30%), generalized (at least 50% of teeth affected) periodontitis were invited to participate in the study. Exclusion criteria included: (i) known systemic diseases, (ii) history and/or presence of other infections, (iii) systemic antibiotic treatment in the preceding 3 mos, (iv) any concomitant medical therapy, (v) pregnancy or lactation in females. All patients gave written informed consent for the trial.

At a baseline visit, a blind examiner collected a complete medical history, standard clinical periodontal parameters, and blood samples. All the subjects received a standard regimen of periodontal therapy (scaling and root planing). Periodontal and inflammatory outcomes were reassessed 1 month following completion of periodontal therapy. Blood samples were obtained by a clean venipuncture from the antecubital fossa before and 30 days after periodontal therapy.

2.1 Clinical parameters

Clinical parameters recorded during the course of the study were gingival index [9], pocket depth (PD) and relative attachment level (RAL).[10] Customized acrylic stents were made for the standardization of the clinical parameters.

2.2 Inflammatory Marker Measurements

C- reactive protein was measured by immunoturbidometre at baseline and 1 month after standard regimen of periodontal therapy. Cobas e601 analyser was used. The Roche CRP assay is based on the principle of particle enhanced immunological agglutination.

Test principle [11, 12] Particle enhanced turbidimetric assay.

Humans CRP agglutinates with latex particles coated with monoclonal anti-CRP antibodies. The precipitate is determined turbidimetrically at 552 nm.

Reagents - working solutions

R1 = TRIS buffer with bovine serum albumin and immunoglobulins (mouse); preservative; stabilizers.

R2 = SR Latex particles coated with anti-CRP (mouse) in glycine buffer; preservative; stabilizers.

2.3 Statistical analysis

The data obtained was tabulated and analyzed statistically. The intragroup comparisons were made using paired using IBM SPSS software. A p-value of less than 0.05 was considered statistically significant.

3. Results

With the exception of the presence of severe, generalized periodontitis, all subjects presented with no reported medical conditions. During the study period, patients did not report changes in lifestyle issues, including exercise, diet, smoking, and medications.

3.1 Clinical parameters

Table 3.1 shows the change in periodontal parameters at baseline and 1 month. Statistically significant improvement in periodontal parameters was seen at the end of 1 month

Table 3.1: Intra-group comparison of periodontal parameters at baseline and 1month.

Periodontal parameters.		Mean ± SD	P value
Gingival Index	Baseline	1.7±.424	
	1 month	1.35±.337	.032*
Pocket Depth	Baseline	6.08 ± 0.95	
	1month	4.15 ± 0.89	<.001*
Relative attachment level	Baseline	10.20 ±.875	
	1 month	8.90 ±.968	<.05*

*Statistically significant *P* value ≤0.05

3.2 Inflammatory parameter

Table 3.2 shows change in the levels of C- reactive protein, statistically significant reduction was seen in the levels of CRP at the end of 1 month following standard periodontal therapy

Table 3.2: Intra-group comparison of C- reactive protein at baseline and 1 month.

		Mean ± SD	P value
C-reactive protein	Baseline	2.0 ± 1.1	
	1 month	1.6±0.9	<.001*

*Statistically significant *P* value ≤0.05

4. Discussion

In present study periodontal therapy (standard) resulted in an reduction in serum CRP of at least 0.5 mg/L. A intervention study by D'Aiuto *et al.*, 2004[13] has showed how standard dental treatment led to a reduction in serum CRP and IL-6 associated with the level of dental clinical response, as determined by periodontal parameters. This suggested a potential dose-response effect between the extent of resolution of the local periodontal infection and the level of reduction in systemic inflammation. The subjects included in this study had baseline CRP concentrations in the upper quartiles of normality (mean of 2.0 ± 1.1 mg/L). During the study, no important changes in lifestyle, habits, medical health, or medications were detected. This indicates that severe, generalized periodontitis in these otherwise healthy individuals contributed to their systemic inflammatory burden. Proposed mechanistic explanations include: (i) the local, infection-driven production of inflammatory mediators (IL-1, IL-6) 'dumped' into the systemic circulation (Offenbacher *et al.*, 1981; Graves, 1999) [14, 15]; (ii) the ability of periodontal pathogens and/or their toxins to disseminate and thus induce a distant inflammatory response (Herzberg and Weyer, 1998; Haraszthy *et al.*, 2000); and (iii) a combination of the above. These results, however, do not allow for generalization to periodontal patients suffering from less severe and/or more localized forms of disease. Data will have to be confirmed and expanded in larger trials if we are to better understand what proportion of the 10-15% of subjects suffering from severe periodontitis have increased systemic inflammation as a result of this chronic infection (Papapanou, 1996) [16].

Following successful treatment, bacterial load is significantly reduced, while antibody titers and avidity to the specific pathogens are improved. As a result of these changes, local inflammation significantly decreases, and there is a significant improvement of the clinical parameters In this study, decreases in serum CRP were significantly associated with

better response to periodontal treatment in terms of decreases in the infection burden and the associated periodontal inflammation as assessed by clinical parameters. As indicated, this study was designed to detect changes in CRP concentrations only. A serious limitation of these preliminary results lies in the small number of subjects included. Further investigations are needed for further exploration of the relationship among periodontitis, periodontal therapy, and systemic inflammatory markers.

5. Summary and conclusion

In summary, periodontitis seems to contribute to systemic inflammation. The potential significance of the reported findings relates to the magnitude of the observed decreases in CRP and the fact that periodontitis can be treated. Results of this investigation should be taken into account in the design and implementation of a definitive trial.

6. References

1. Kweider M, Lowe GD, Murray GD, Kinane DF, McGowan DA. Dental disease, fibrinogen and white cell count; links with myocardial infarction? Scott Med J. 1993; 38(3):73-74.
2. Ebersole JL, Machen RL, Steffen MJ, Willmann DE. Systemic acute-phase reactants, C-reactive protein and Haptoglobin, in adult periodontitis. Clin Exp Immunol. 1997; 107:347-352.
3. Loos BG, Craandijk J, Hoek FJ, Wertheim-van Dillen PM, van der Velden V, Elevation of systemic markers related to cardiovascular diseases in the peripheral blood of periodontitis patients. J Periodontol. 2000; 71:1528-1534.
4. Noack B, Genco RJ, Trevisan M, Grossi S, Zambon JJ, De Nardin E. Periodontal infections contribute to elevated systemic Creactive protein level. J Periodontol. 2001; 72:1221-1227.
5. Buhlin K, Gustafsson A, Hakansson J, Klinge B. Oral health and cardiovascular disease in Sweden. J Clin Periodontol. 2002; 29:254-259.
6. Libby P, Ridker PM, Maseri A. Inflammation and atherosclerosis. Circulation. 2002; 105:1135-1143.
7. Pearson TA, Mensah GA, Alexander RW, Anderson JL, Cannon RO III, Criqui M *et al.* Markers of inflammation and cardiovascular disease: application to clinical and public health practice: a statement for healthcare professionals from the Centers for Disease Control and Prevention and the American Heart Association. Circulation. 2003; 107:499-511.
8. Kluft C, de Maat MP. Determination of the habitual low blood level of C-reactive protein in individuals. Ital Heart J. 2001; 2:172-180.
9. Löe H. The gingival index, the plaque index and the retention index systems. Journal of periodontology. 1967; 38:610-6.
10. Isidor F, Karring T, Attström R. Reproducibility of pocket depth and attachment level measurements when using a flexible splint. Journal of Clinical Periodontology. 1984; 11(10):662-8.
11. Eda S, Kaufmann J, Roos W, Pohl S. Development of a new microparticle-enhanced turbidimetric assay for C-reactive protein with superior features in analytical sensitivity and dynamic range. Journal of clinical laboratory analysis. 1998; 12(3):137-44.
12. Price CP, Trull AK, Berry D, Gorman EG. Development and validation of a particle-enhanced Turbidimetric immunoassay for C-reactive protein. Journal of immunological methods. 1987; 99(2):205-11.
13. D'Aiuto F, Parkar M, Andreou G, Suvan J, Brett PM, Ready D *et al.* Periodontitis and systemic inflammation: control of the local infection is associated with a reduction in serum inflammatory markers. J Dent Res. 2004; 83:156-160.
14. Offenbacher S, Farr DH, Goodson JM. Measurement of prostaglandin E in crevicular fluid. J Clin Periodontol. 1981; 8:359-367.
15. Graves DT. The potential role of chemokines and inflammatory cytokines in periodontal disease progression. Clin Infect Dis. 1999; 28:482-490.
16. Papapanou PN. Periodontal diseases: epidemiology. Ann Periodontol. 1996; 1:1-36.