Researchers have known for quite some time that elevated C-reactive protein (CRP) levels increase the risk for cardiovascular disease. A recent study published in The New England Journal of Medicine identified elevated CRP levels as a stronger predictor of heart attacks than elevated cholesterol levels, and recommended CRP and cholesterol screening for accurate risk assessment of cardiovascular disease.

However, many clinicians were unclear of the cause of elevated CRP levels. A study published earlier this year in the Journal of Periodontology reported that inflammatory effects from periodontal disease, a chronic bacterial infection of the gums, cause oral bacterial byproducts to enter the bloodstream and trigger the liver to make proteins such as CRP that inflame arteries and promote blood clot formation

Abstract

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Periodontal Infections Contribute to Elevated Systemic C-Reactive Protein Level
Barbara Noack

Department of Oral Biology, University at Buffalo, Buffalo, NY.

Robert J. Genco

Department of Oral Biology, University at Buffalo, Buffalo, NY.

Maurizio Trevisan

Department of Social and Preventive Medicine.

Sara Grossi

Department of Oral Biology, University at Buffalo, Buffalo, NY.

Joseph J. Zambon

Department of Oral Biology, University at Buffalo, Buffalo, NY.

Dr. Ernesto De Nardin

Department of Oral Biology, University at Buffalo, Buffalo, NY.

Department of Microbiology.

Background: Periodontitis is a local inflammatory process mediating destruction of periodontal tissues triggered by bacterial insult. However, this disease is also characterized by systemic inflammatory host responses that may contribute, in part,

to the recently reported higher risk for cardiovascular disease (CVD) among patients with periodontitis. Moderate elevation of C-reactive protein (CRP) has been found to be a predictor of increased risk for CVD. Elevated CRP levels in periodontal patients have been reported by several groups. In this study, we examined whether CRP plasma levels are increased in periodontitis and if there is a relation to severity of periodontal disease and to the periodontal microflora.

Methods: CRP serum levels were assessed using radial immunodiffusion assay in 174 subjects, 59 with moderate mean clinical attachment loss (AL) $(2.39 \pm 0.29 \, \text{mm})$ and 50 with high AL $(3.79 \pm 0.86 \, \text{mm})$ as compared to 65 periodontally healthy controls (AL, $1.74 \pm 0.18 \, \text{mm})$. Clinical attachment loss, probing depths, and percentage of periodontal pocket sites $\geq 5 \, \text{mm}$ were measured. The presence of periodontal pathogens *Porphyromonas gingivalis* (*P.g.*), *Prevotella intermedia* (*P.i.*), *Campylobacter recta* (*C.r.*), and *Bacteroides forsythus* (*B.f.*) in subgingival plaque samples was measured by immunofluorescence microscopy.

Results: Statistically significant increases in CRP levels were observed in subjects with periodontal disease when compared to healthy controls (P = 0.036). Subjects with high levels of mean clinical attachment loss had significantly higher mean CRP levels ($4.06 \pm 5.55 \text{ mg/I}$) than controls ($1.70 \pm 1.91 \text{ mg/I}$), P = 0.011. The CRP levels were adjusted for factors known to be associated with elevated CRP, including age, smoking, body mass index (BMI), triglycerides, and cholesterol. Age and BMI were found to be significant covariates. The reported range for CRP as a risk factor for CVD, peripheral vascular diseases, or stroke is 1.34 mg/I to 6.45 mg/I and the mean of this range is 3 mg/I. The percentage of subjects with elevated levels of CRP \geq 3 mm was significantly higher in the high clinical AL group (38%; 95% CI: 26.7%, 49.3%) when compared to the control group (16.9%; 95% CI: 9.25%, 24.5%), P = 0.011. The presence of periodontal pathogens P.g., P.i., C.r., and B.f. in subgingival samples was positively associated with elevated CRP levels (P = 0.029).

Conclusions: The extent of increase in CRP levels in periodontitis patients depends on the severity of the disease after adjusting for age, smoking, body mass index, triglycerides, and cholesterol. Also, there are elevated levels of CRP associated with infection with subgingival organisms often associated with periodontal disease, including *P.g., P.i., C.r.,* and *B.f.* Recent investigations emphasized the role of moderate elevated CRP plasma levels as a risk factor for CVD. The positive correlation between CRP and periodontal disease might be a possible underlying pathway in the association between periodontal disease and the observed higher risk for CVD in these patients. *J Periodontol 2001;72:1221-1227.*