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Original Article

C-reactive protein in patients with aggressive periodontitis

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KEYWORDS
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Abstract *Background/purpose:* The aim of this study was to evaluate and compare the systemic levels of C-reactive protein (CRP) in peripheral blood samples of patients with aggressive periodontitis during the first twelve months of periodontal treatment, at exactly six month interval measurements, and compare them with clinical periodontal parameters.
Materials and methods: All patients (N = 45) were examined prior to the initiation of periodontal treatment. Patients were divided into two groups GAgP (Generalised form of aggressive periodontitis, N = 23) and group LAgP (Localised form of aggressive periodontitis, N = 22). Control group (CON) included 60 individuals with healthy periodontium. The levels of CRP were determined in both groups GAgP and LAgP three times in 6 month intervals during the periodontal treatment.
Results: CRP is a plasma protein that reflects the extent of the acute phase response to inflammation and is one of the markers of choice for monitoring this response. In our study, CRP levels decreased in course of periodontal treatment in both groups (GAgP and LAgP) in a similar way as bleeding on probing (BOP) and probing pocket depth (PPD) indices.

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Conclusion: Our study results showed that CRP levels, as well as bleeding on probing (BOP) and probing pocket depth (PPD), indices decreased in course of periodontal treatment in patients with generalised and localised aggressive periodontitis. Therefore this marker might be exploitable as a means to evaluate periodontal health in patients with aggressive periodontitis.

Introduction

Periodontal disease is an inflammatory disease that affects the soft and hard structures that support the teeth.[1] In gingivitis, inflammation localised to the supracrestal region of the periodontium leads to ulceration of the junctional epithelium. Although this is technically a loss of clinical attachment (because in healthy tissue the epithelium attaches to the surface of the tooth), clinical attachment loss (CAL) is used almost exclusively to refer to connective tissue attachment loss. Sites with periodontitis exhibit clinical signs of gingival inflammation and loss of connective tissue attachment. Connective tissue attachment loss refers to the pathological detachment of collagen fibres from the cemental surface with the concomitant apical migration of the junctional or pocket epithelium on the root surface.[2] Diagnosis of aggressive periodontitis is made on clinical, radiographic and historical findings which show rapid attachment loss and bone destruction, and possible familial aggregation of disease. The disease often occurs in people under 35 years of age, but it may also affect older patients. Except for periodontal disease, patients are systemically healthy. Other features that may be present are periodontal tissue destruction that is greater than would be expected given the level of local factors, elevated levels of *Aggregatibacter actinomycetemcomitans* or *Porphyromonas gingivalis*, phagocyte abnormalities and increased production of prostaglandin E_2 and interleukin-1b.[3] Early onset (aggressive) forms of periodontal diseases are detectable in all age and ethnic groups.[4] This disease is divided into localised and generalised form by classification of periodontal diseases from 1999. It can be further classified on the basis of extent and severity. Aggressive periodontitis is subcategorised into localised ($\leq$30% of the teeth are affected) and generalised form ($>$30% of the teeth are affected). Severity is based on the amount of clinical attachment loss (CAL) and is designated as slight (1—2 mm CAL), moderate (3—4 mm CAL) or severe ($>$5 mm CAL).[3] Bacterial aetiology of aggressive forms of periodontitis is confirmed by the studies which have demonstrated the presence of a layer of bacterial deposits on the root surface of advanced aggressive periodontitis lesions.[5] A rapid rate of destruction of the periodontium is a major criterion for the diagnosis of aggressive periodontitis.[6] Diagnostic difficulty is related to the fact that periodontal destruction is often diagnosed when the attachment loss is already fairly advanced. In general, distinct alterations in the morphology of the periodontium and substantial tissue damage are necessary for establishing a clear diagnosis. CRP (C-reactive protein) is produced in response to many forms of injury other than periodontitis, such as other infections, trauma and hypoxia, and it is regulated by diverse cytokines. CRP levels have an association with smoking, obesity, triglycerides, diabetes, and periodontal disease.[7] Changes in peripheral blood cellular and molecular components can be found in patients with periodontitis due to inflammatory changes of the periodontal tissues.[8] Positive correlation between CRP and periodontal disease severity was proved by many studies,[9—11] and levels of CRP decrease after nonsurgical periodontal therapy,[12] but most studies have focused on CRP levels in chronic periodontitis, and very few are conducted on patients with aggressive periodontitis.[13,14] The link between inflammation and systemic diseases, e.g. cardiovascular disease (CVD), appears to be firmly established.[15] Epidemiological associations between periodontitis and CVD have been reported.[16,17] Among the several biomarkers that have been proposed for cardiovascular risk stratification, high-sensitivity C-reactive protein (hs-CRP) appears to contribute to the identification of people at risk of developing CVD[18]; however, the evaluation of hs-CRP has not yet been widely recommended in guidelines.[19] Wohlfeil et al. compared systemic inflammatory mediators in patients with untreated aggressive and chronic periodontitis and in periodontally healthy controls. Patients with aggressive periodontitis have statistically significant elevations in serum CRP levels compared to subjects with healthy periodontium.[20,21]

Thus, the aim of this study was to evaluate and compare the systemic levels of CRP in peripheral blood samples of 45 patients with aggressive periodontitis in course of periodontal treatment and of 60 healthy controls and their correlation with periodontal clinical parameters.

Materials and methods

Study population

All patients (N = 45) were recruited from the patient pool of the Department of Periodontology, Institute of Dental Medicine, First Faculty of Medicine and General University Hospital, Charles University, Prague, Czech Republic, from 2014 to 2016, and all patients were examined prior to the initiation of periodontal treatment. In our study, the patients were treated by conservative treatment — deep scaling and root planing, surgical techniques were not used. Deep scaling and root planing were performed three months after the first collection of CRP samples (appointment I). We examined the periodontal parameters and measured the CRP levels in each patient every six months

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