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Brief communication



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ABSTRACT

Periodontal disease results from the interaction between pathogenic bacteria that colonize supragingival and subgingival biofilms and the host, triggering an inflammatory response, with systemic effects leading to immune-mediated destruction of the attachment apparatus and loss of supporting alveolar bone. Immunological pathways and predisposing genetic factors common to periodontal disease and rheumatic diseases, including systemic lupus erythematosus, have been described. Case reports have suggested greater severity of periodontal disease in patients with systemic lupus erythematosus. However, studies evaluating the influence of the treatment of one disease on the clinical and laboratory manifestations of the other have yielded conflicting results.

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Há associação entre o lúpus eritematoso sistêmico e a doença periodontal?

RESUMO

Palavras-chave: Lúpus eritematoso sistêmico Periodontite Periodontite crônica A doença periodontal resulta da interação entre bactérias patogênicas que colonizam os filmes supra e subgengival e o hospedeiro e deflagram uma resposta inflamatória local, com efeitos sistêmicos, que leva à destruição imunomediada dos tecidos de sustentação dos dentes e do osso alveolar. Vias imunológicas e fatores genéticos predisponentes comuns

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à doença periodontal e às doenças reumáticas, entre elas o lúpus eritematoso sistêmico, vêm sendo descritos. Relatos de caso sugeriram maior gravidade da doença periodontal em pacientes com lúpus eritematoso sistêmico. No entanto, estudos que avaliaram as influências do tratamento de uma sobre as manifestações da outra apresentaram resultados conflitantes.

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Introduction

Currently, the relationship between connective tissue diseases and periodontal disease (PD) has been the subject of much discussion.¹

PD is a group of infectious and inflammatory diseases that result from the interaction between periodontal pathogens present in supragingival and subgingival biofilms and the host, generating an inflammatory response of variable intensity, which can lead to immune-mediated destruction of the attachment apparatus and loss of supporting alveolar bone. Gingivitis, the most common form of periodontal disease, is an inflammatory process characterized by erythema, edema and gingival bleeding. Periodontitis is characterized by gingival inflammation accompanied by an inflammatory response by the host, which results in destruction of the attachment apparatus and loss of supporting alveolar bone and that have systemic effects. The changes found in dental evaluation of periodontitis are an increase in probing depth (probing pocket depth - PPD), which reflects the distance from the bottom of the pocket to the gingival margin, the presence of clinical attachment loss (CAL), which measures the position of the soft tissue in relation to the cemento-enamel junction, and the occurrence of gingival bleeding on probing (BOP), mobility and tooth and alveolar bone loss.²

The existence of immune pathways and of a genetic predisposition common to PD and connective tissue diseases, among these systemic lupus erythematosus (SLE), is recognized and has been described.¹

In this brief communication, we reviewed studies published in English or Portuguese, which investigated possible associations between PD and SLE, found through a systematic search in PubMed/Medline and LILACS databases, using the terms "Lupus Erythematosus, Systemic" and "Periodontitis", "Periodontitis, Chronic" or "Periodontitis, Adult". There was no restriction about the search period. Twenty-two articles from PubMed/Medline and five articles from LILACS were found. Thirteen original articles that dealt with the topic were included in this review.^{3–15} A manual search of the references of included articles was carried out, and five articles were selected.^{16–20}

Studies of association between SLE and periodontal disease

Case reports suggesting that clinical and therapeutic associations between SLE and PD have been published since the 1980s,^{3–5} reporting a greater severity of PD in patients with SLE, probably associated with immunosuppression caused by the disease, or its treatment.

The frequency of periodontitis in SLE patients varied in different studies, between 60 and 93.8% (Table 1).^{6–11} A Japanese study reported that SLE patients had a higher frequency of PD versus the general population of that country,⁹ but no study has compared the frequency of PD with a control group (healthy volunteers). The variability of the frequency of periodontitis found in different studies is probably associated with the use of different criteria for its diagnosis, or to differences in patient groups with SLE with regard to disease severity or activity. Thus, the question of frequency of periodontitis in patients with SLE remains an open one, and controlled studies are needed for setting up whether periodontitis is actually more common in SLE patients.

Several authors evaluated the severity of PD in SLE patients compared to healthy volunteers, or to patients with PD without SLE, and their results were conflicting. Periodontal parameters were found to be: similar,^{9,10,12,13} less severe,^{9,10,13-15} or more severe¹⁶⁻¹⁸ (Table 2).

These controversial data prompted some questions as: immunosuppression induced by SLE or its treatment would increase, would not affect, or would reduce the infectious and inflammatory periodontal destruction? The studies that found lower severity of periodontal parameters in SLE suggest a smaller immune-mediated periodontal destruction associated with immunosuppressive drugs.^{9,10,13–15} However, the composition bias of the control group, consisting primarily of patients referred for specialized treatment and potentially with more serious periodontitis, was not controlled. Studies were also published that suggest a greater severity of PD in patients with SLE, especially when the disease is active.¹⁶⁻¹⁸ Then, immunosuppression would increase the periodontal decay associated with chronic infection? This point also is not defined and requires further studies that include patients with active and inactive SLE, evaluating the influence of SLE activity and of the immunosuppressive treatment on periodontal parameters, with the inclusion of a control group representative of the general population, with people with and without PD.

Biological basis of the association between SLE and PD

In PD, the gum infection triggers a series of immune responses that involve the participation of immune cells and cytokines, that results in the destruction of the attachment apparatus and alveolar bone loss. A study reported an increase of serum levels of C-reactive protein (CRP) and of salivary Download English Version:

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