REVISTA BRASILEIRA DE REUMATOLOGIA

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SOCIEDADE BRASILEIRA DE REUMATOLOGIA

Review article

Periodontitis and systemic lupus erythematosus☆

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ARTICLE INFO

Article history:
Received 5 January 2015
Accepted 3 July 2015
Available online 21 November 2015

Keywords:
Periodontitis
Systemic lupus erythematosus
Immunology

ABSTRACT

A large number of studies have shown a potential association between periodontal and autoimmune diseases, such as rheumatoid arthritis and systemic lupus erythematosus (SLE). Similar mechanisms of tissue destruction concerning periodontitis and other autoimmune diseases have stimulated the study of a possible relationship between these conditions. This study aims to review the literature about this potential association and their different pathogenic mechanisms. Considering that periodontal disease is a disease characterized by inflammation influenced by infectious factors, such as SLE, it is plausible to suggest that SLE would influence periodontal disease and vice versa. However, this issue is not yet fully elucidated and several mechanisms have been proposed to explain this association, as deregulation mainly in innate immune system, with action of phagocytic cells and proinflammatory cytokines such as IL-1β and IL-18 in both conditions' pathogenesis, leading to tissue destruction. However, studies assessing the relationship between these diseases are scarce, and more studies focused on common immunological mechanisms should be conducted to further understanding.

Doença periodontal e lúpus eritematoso sistêmico

Palavras-chave:
Periodontite
Lúpus eritematoso sistêmico
Imunologia

RESUMO

Um grande número de estudos tem mostrado uma potencial associação entre doenças periodontais e doenças autoimunes, como artrite reumatoide e lúpus eritematoso sistêmico (LES). Os mecanismos de destruição tecidual semelhantes entre a periodontite e as demais doenças autoimunes têm estimulado o estudo de possíveis relações entre essas condições.

☆ This work is a partnership between the Department of Dentistry, Universidade do Estado do Rio de Janeiro (UERJ), and the Sector of Rheumatology, Núcleo de Estudos da Saúde do Adolescente (NESA), Universidade do Estado do Rio de Janeiro (UERJ), Rio de Janeiro, RJ, Brazil.

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http://dx.doi.org/10.1016/j.rbre.2015.09.001

O presente estudo tem como objetivo revisar a literatura acerca dessa potencial associação e dos seus diferentes mecanismos patogênicos. Considerando-se a doença periodontal uma doença de caráter inflamatório que sofre influência de fatores infecciosos, assim como o LES, é plausível sugerir que o LES influenciaria sua progressão, assim como a periodontite influenciaria a progressão do LES. Entretanto, essa questão ainda não é totalmente elucidada e vários mecanismos têm sido propostos para explicar tal associação, como desregulações, principalmente no sistema imune inato, com ações de células fagocíticas e de citocinas pró-inflamatórias, como IL-1β e IL-18, na patogênese de ambas as condições, o que contribui para a destruição tecidual. Existem, contudo, poucos estudos na literatura que avaliam a relação entre essas doenças e mais trabalhos focados nos mecanismos imunológicos comuns a ambas as condições devem ser feitos para um maior entendimento.

Introduction

Periodontitis is a chronic destructive inflammation that leads to the loss of supporting tissues of teeth and eventually even to tooth loss. The periodontal ligament and bone tissue are destroyed by an immune and inflammatory response to the presence of bacteria, particularly gram-negative ones, in the gingival sulcus. The severity of inflammation varies between individuals, irrespective of the degree of bacterial infection, suggesting that a dysregulation of the host inflammatory response may contribute to its existence.[1]

On the other hand, systemic lupus erythematosus (SLE) is an autoimmune disease of unknown origin, which affects the connective tissue and thus various organs in the body. The clinical manifestations of SLE vary with the severity of the disease, and its course may exhibit periods of exacerbation and remission.[2] SLE is characterized by immune responses against a large number of autoantigens, affecting more often women in the second and third decades of life.[3]

A large number of studies have shown a potential association between chronic periodontitis and autoimmune disease, especially rheumatoid arthritis,[4] as well as inflammatory bowel disease and glomerulonephritis.[5] A high prevalence of periodontitis has also been detected in patients with SLE.[6,7]

The similar mechanisms of tissue destruction for periodontitis and other autoimmune diseases have stimulated the study of potential associations between these conditions. In spite of presenting different etiologies, the existence of similar destructive mechanisms could explain an eventual association between periodontitis and SLE.[8] These potential mechanisms in common may involve deregulation, especially in the innate immune system, with action of phagocyte cells and of proinflammatory cytokines, such as IL-1β and IL-18, in the pathogenesis of both conditions, contributing to tissue destruction.[6,9]

The literature describing oral conditions of patients with lupus is scarce, and pertinent information is conflicting. Considering the possibility of SLE as a modifying condition of the periodontal health-disease process and the lack of information to clarify this interrelationship, our aim is to review the literature on SLE and a potential relationship with periodontal disease. Despite its high prevalence in rheumatoid arthritis, only a limited number of studies have examined oral conditions, in particular periodontal disease in SLE patients.[10,11]

Literature review and discussion

Definition

Periodontal disease is defined as any hereditary or acquired disorder of tooth surrounding and supporting tissues (periodontium). Periodontal disease has neoplastic, developmental, inflammatory, traumatic, metabolic or genetic origin. However, the term periodontal disease generally refers to those common inflammatory disorders of gingivitis and periodontitis, which are caused by pathogenic microorganisms in a biofilm or plaque that forms adjacent to the teeth. Gingivitis, the mildest form of periodontal disease, is highly prevalent and readily reversible with an effective oral hygiene. On the other hand, the inflammation that extends deep into tissues and causes loss of supporting connective tissue and alveolar bone is known as periodontitis. It results in the formation of a soft tissue pocket between the gum and root of the teeth and can result in tooth loss.[12]

On the other hand, systemic lupus erythematosus is an autoimmune disease that affects the connective tissue and may extend to various organs of the body. The clinical manifestations vary greatly between organs and systems and the course of the disease goes through periods of exacerbation and remission.[2,10]

Etiology

Periodontitis has its onset and is perpetuated by a group of bacteria, predominantly gram-negative and anaerobes, that colonize the subgingival area. Today, it is already clear that these bacteria cause indirect tissue destruction, activating various mechanisms of host immunity.[13]

It is believed that in SLE, a disease of unknown origin, there is an accumulation of disorders. Potential complications may be associated with hormonal imbalances, viral infections, impaired function of suppressor T cells, defective genetic control of immune responses, abnormal function of macrophages, B cell intrinsic defects, poor host response to an infectious agent, or a combination of such elements.[14]