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Rheumatoid Arthritis & Periodontitis: A connection!

Rheumatoid Arthritis (RA) and chronic and aggressive periodontitis are chronic inflammatory disorders characterized by deregulation of the host inflammatory response. Increased secretion of pro-inflammatory mediators results in soft and hard tissue destruction of the synovium and periodontium respectively. Both diseases share risk factors and have pathological pathways in common, resulting in loss of function and disability as a final clinical outcome. The prevalence of periodontal disease has increased two-fold among patients with rheumatoid arthritis (RA) compared to the general population. This increased prevalence is unrelated to secondary Sjögren's syndrome but instead reflects shared pathogenic mechanisms, including an increased prevalence of the shared epitope HLA-DRB1-04; exacerbated T-cell responsiveness with high tissue levels of IL-17; exaggerated B-cell responses, with plasma cells being the predominant cell type found within gingival tissue affected with periodontitis and B cells being twice as numerous as T cells; RANK overexpression; and an increase in the ratio of RANK-L over osteoprotegerin with a high level of RANK-L expression on gingival B cells, most notably those capable of recognizing Porphyromonas gingivalis. Other factors conducive to periodontitis include smoking and infection with the Epstein-Barr virus or cytomegalovirus, which act by promoting the growth of organisms such as P. gingivalis, whose DNA is often found in synovial tissue from RA patients. P. gingivalis produces the enzyme peptidylarginine deiminase that induces citrullination of various autoantigens, and levels of anti-CCP antibodies are considerably higher in RA patients with than without periodontal disease, suggesting that periodontitis may contribute to the pathogenesis of RA. Further support for this hypothesis comes from evidence that other antigens involved in RA, such as HCgp39,are also present in gingival tissue. P.gingivalis may be able to induce apoptosis of some lymphocytes and to alter the T-cell response via the expression of superantigens. Furthermore, P. gingivalis DNA is often found in synovial samples from patients with RA. Data suggests that P. gingivalis may play a central role in inducing or perpetuating RA in some patients. data suggest that migration of DNA from oral bacteria to the synovial membrane may be a common event, despite the presence of an antibody response. However, migration was also found in patients with osteoarthritis, although migration of some organisms such as P. gingivalis and P. intermedia seemed more specific of RA.