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Review

Microbiological and immuno-pathological aspects of peri-implant diseases

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ABSTRACT

Peri-implant diseases are a cluster of "contemporary" oral infections in humans that have emerged as a result of the routine application of osseointegrated dental implants in clinical practice. They are characterized by the inflammatory destruction of the implant-supporting tissues, as a result of biofilm formation on the implant surface. Peri-implant mucositis and peri-implantitis are analogous to gingivitis and periodontitis that affect natural teeth. The aim of this comprehensive review was to provide insights into the infectious aetiology and immuno-pathology of peri-implant diseases, and to identify similarities and differences with periodontal diseases. The microbial composition of peri-implantitis-associated biofilms is mixed, non-specific and very similar to that of periodontitis. A considerable exception is the frequent presence of high numbers of staphylococci and enteric bacteria in peri-implantitis. The sequence of immuno-pathological events and the qualitative composition of the immune cells in peri-implant infections are similar to that of periodontal infections. The lesions are characterized predominantly by neutrophils, macrophages, T- and B-cells. Nevertheless, compared to periodontitis, peri-implantitis is marked by a more extensive inflammatory infiltrate and innate immune response, a greater severity of tissue destruction and a faster progression rate. This could well account for the structural differences between the two tissue types, predominantly the lack of periodontal ligament and Sharpey's fibres around implants. In order to support the early diagnosis and prevention of peri-implantitis, it is crucial to explain its fast progression rate by elucidating the underlying molecular mechanisms. This could be achieved, for instance, by utilizing the non-invasive collection and analysis of peri-implant crevicular fluid.

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1. Oral ecology and biofilm formation

The oral cavity is a dynamic ecosystem continuously colonized by microorganisms which are collectively defined as the oral microbial flora. These have evolved along with the host, and their growth is dependent on the available nutrients and their capacity to withstand local immune defenses. Bacteria grow on natural (tooth, mucosa) or artificial (prostheses, implants) surfaces as biofilms, which are highly organized and structured microbial communities, embedded in polymeric matrices. As part of a biofilm community, bacteria become more virulent than their planktonic forms and less penetrable by elements of the immune system, such as neutrophils and antibodies, or antimicrobial factors.[1] The contemporary notion on how oral biofilms are causing oral diseases, such as caries, periodontitis, or peri-implantitis is well summarized by the ''ecological plaque hypothesis''.[2] According to this hypothesis, it is the interrelationship between the bacteria and the host response that defines health or disease. Changes in the local microenvironment may shift the composition of the biofilm microflora. Under the newly established conditions, the predominant microbial species may display enhanced virulence and act as opportunistic pathogens, causing disease to susceptible hosts. Thus, oral infections are considered to be endogenous infections.

2. Clinical characteristics of peri-implant diseases

Osseointegrated dental implants are metallic devices made predominantly of titanium that are surgically implanted into the jaw bone, substituting one or more missing teeth. A prosthetic restoration is then fit on a transmucosal abutment structure, aiming to restore the functional and aesthetic needs of that site of the oral cavity. Nevertheless, the artificial manufactured surfaces of dental implants are also prone to microbial colonization and biofilm formation, eventually causing infection of the implant-supporting (peri-implant) tissues.

Failures of dental implant function can be classified either as early, or as late ones.[3,4] Early implant failures are the ones that occur due to incomplete osseointegration, before or after the functional loading of the implant. Such failures include early loading, surgical contamination, poor compatibility of the implanted material, or inefficient healing. On the other hand, late failures involve disruption of the function of an already osseointegrated implant, mainly due to chronic infection of the peri-implant tissues. In peri-implant mucositis, the biofilm-induced inflammation is localized on the soft peri-implant mucosa, with no evidence of destruction of the supporting bone. In peri-implantitis, the inflammation expands deeper into the bone tissue, leading to its gradual destruction, and eventually to implant loss. These two forms of peri-implant disease are analogous to gingivitis and periodontitis of natural teeth.[5]

The diagnostic criteria for peri-implant diseases are mainly clinical and radiographic.[6] Peri-implant mucositis is characterized by inflamed or erythematous mucosa and bleeding during the examination. Peri-implantitis is further characterized by the formation of a peri-implant pocket greater than 4 mm, bleeding or suppuration on probing, and, radiographically, a characteristic symmetrical ''saucer-shaped'' bone destruction (or ''crater'') around the implant. Mobility can occur at progressed stages and is associated with poor prognosis of the implant. The increased probing depth, the positive bleeding on probing and the presence of suppuration in particular are important diagnostic indicators of peri-implant diseases.[7]

The consensus risk factors for peri-implantitis are poor oral hygiene, smoking, systemic conditions (e.g. diabetes mellitus), genetic susceptibility, potentially alcohol consumption, and prior history of periodontitis.[7] The first four are shared in common with periodontitis, whereas the last one denotes an increased susceptibility to local oral infection. Hence, there appears to be a parallel trend between periodontal and peri-implant diseases.

3. Aetiology and pathogenesis of peri-implant diseases

There are two crucial steps in understanding the infectious aetiology and pathogenesis of peri-implant diseases: understanding of (a) the aetiological factors and pathogenic mechanisms that govern periodontal diseases, and (b) the structural and immuno-pathological differences between periodontal and peri-implant tissues. In other terms, the already established knowledge on periodontal diseases should be a starting point for deciphering in peri-implant diseases, keeping well in view that any identified differences between the two could yield independent research questions.

3.1. *Differences between periodontal and peri-implant tissues*

Although there are in principle clinical and histopathological similarities between the periodontal and peri-implant mucosa, there are also some fundamental differences.[5] The main one is the absence of Sharpey's fibres inserting perpendicularly to the implant surface, as opposed to the cementum of natural teeth. Instead, the collagen fibres of the submucosal connective tissue are arranged parallel to implant surface. This results in the peri-implant crevice being deeper than the gingival crevice, eventually allowing the deeper penetration of bacteria. In terms of the interface with the bone, implants are directly osseointegrated into the bone. On the contrary, natural teeth are socketed into it via the periodontal ligament and the associated Sharpey's fibres at its extremities. The lack of the periodontal ligament poses a number of biological ''disadvantages'' for the implant, compared to natural teeth.