Conclusions

It was concluded that zirconia and titanium implant abutments elicit a similar soft tissue response when judged clinically, microbiologically, and histologically in people, but the parameters do not correlate. This gave rise to concerns as to the sensitivity and specificity of clinical and microbiologic parameters as indicators of the peri-implant soft tissue status in relatively healthy conditions in vivo.

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Literature Abstract

Alzheimer's disease and periodontitis—An elusive link

Alzheimer's disease (AD) is the most prevalent form of dementia, which poses a health problem worldwide. However, the etiology and pathophysiology of this complex neurodegenerative disorder has not been identified. The objective of this review thus aimed to clarify the possible role of periodontitis in exacerbating AD. A PubMed search incorporating relevant systematic reviews, meta-analyses, and original articles in English from 1994 to 2012 was used. Both human and animal studies were considered. A distinct hallmark of AD is the formation of extracellular amyloid β-peptide (AβP) plaques from the proteolytic cleavage of the amyloid precursor protein (APP) and intraneuronal neurofibrillary tangles (NFTs) of hyper-phosphorylated tau protein. The interplay of the above encourages release of pro-inflammatory mediators within the cerebral microvasculature leading to impaired degradation and clearance and subsequent loss of synaptic dysfunction and neurodegeneration. Neural damage can be induced via the exaggerated inflammation found in AD. Periodontitis, even though primarily a local disease, is able to sustain a low-grade systemic inflammation through the release of pro-inflammatory cytokines. Two plausible links are found between AD and periodontitis through periodontitis preceding systemic inflammation or infection, as well as a bacterial and viral influence. This systemic infection or inflammation is postulated to bypass a compromised blood-brain barrier and via peripheral nerves leading to microglial activation, tau protein phosphorylation, increased APP and AβP, alongside platelet aggregation and atherogenesis. This contributes to oxidative damage and inflammation, which are important features of AD. The author proposed that inflammation may be the central operating mechanism and considers that periodontitis may be a potential risk factor for the development of AD. Given the limitations of evidence available, the question of causality remained unanswered and could be clarified only by further research.

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