Editorial From Early Physiological Marginal Bone Loss to Peri-Implant Disease: On the Unknown Local Contributing Factors

Peri-implant bone loss or so-called peri-implantitis has recently become the focus of many research projects and a very serious concern of many noted clinicians. Its prevalence is around 30%,[1] although estimates of this figure range from 1% to 47%.[1–3] Currently no consensus exists with regard to a suitable definition of peri-implantitis based on clinical and radiographic signs and symptoms or the best way to manage this emerging challenge. A curious aspect of this problem is that despite strict compliance with the prescribed maintenance interval, peri-implantitis can still occur.[4] It is because of this that many clinicians have now wisely turned their primary attention away from treatment and focused instead on methods of prevention. So far, known risk factors/indicators include a history of periodontitis, smoking, poor plaque control, genetic predisposition, diabetes, and occlusal trauma. These risk factors coincide to some extent with those associated with periodontitis; however, little is known regarding the influence of local contributing factors on the development of the peri-implantitis. Nature has provided Homo sapiens with a dentition well adapted to our needs, which, although it is not always ideally positioned, generally offers adequate function.[5] It has been speculated that teeth with an improper prosthetically induced occlusal overload may accelerate the progression of periodontitis.[6] Studies on peri-implantitis, however, rarely evaluate this factor. This is perhaps due to the fact that the overload is borne solely by the bone-implant interface where, in contrast to natural teeth, there is no intermediary ligamentous structure in the majority of currently available implant systems with the exception of micro-etched laser groove.[7] Future studies of how this specific design relates to peri-implant disease are needed. Likewise, many other factors that cause periodontal breakdown are not regarded as etiologic or contributing factors for peri-implantitis.

Pathogenic bacteria and/or fungi can indirectly cause peri-implant pathology through an inflammatory process that is analogous to the mechanisms identified in chronic periodontitis. Although recent studies have highlighted that both processes share few species of bacteria, similar inflammatory processes can occur that transform the microbiota and produce a dysbiosis in both entities.[8] In fact, it has been demonstrated that some microorganisms have a particular affinity for titanium alloys and that compared to healthy sites, a high level of protease activity from some gram-negative anaerobic rods dominates the sulci.[9] Along these lines, it must also be presented that this disease, although it shares some features with periodontitis, exhibits a significantly different mRNA profile.[10] Also, implant microgrooves/microthreads can serve as plaque-retentive areas where bacteria can attach, grow, and be easily harvested for study. Delicate forming bone in the early remodeling stage, which can be the result of a foreign body reaction,[11] is at high risk for pathologic destruction since there is no protective connective tissue seal, as seen in the natural dentition, to prevent threads from becoming exposed to the oral environment and accumulating plaque.[12] Furthermore, recent evidence may further unfold the etiology on peri-implantitis and whether the titanium alloy particles detached from the implant at insertion due to friction or corrosion might play a role on its induction.[13] In addition to those mentioned, current evidence suggests that other factors may contribute to peri-implant marginal bone loss. These factors include but are not limited to mucosal tissue thickness, width of keratinized tissue, implant-abutment connection type, bone density, implant positioning, and amount of loading. Unfortunately, there is limited documentation about the effect of

surgical trauma and implant malpositioning in any of the three planes of the space. These particular factors may trigger more rapid attachment breakdown, thereby producing an environment that will harbor many putative pathogens.[14] They may or may not contribute to greater physiologic bone loss caused by the reestablishment of biologic width or due to a foreign body reaction. Irrespective of the implant placement protocol, three-dimensional errors can occur. If the implant is placed too buccal, there may be excessive buccal bone resorption that could jeopardize esthetic harmony and, ultimately, implant stability. This is due not to the total amount of supporting bone loss but to thread exposure, bacterial colonization, and an ensuing inflammatory cascade that will ultimately be diagnosed as peri-implantitis (or late implant bone loss). It is speculated that this might be further aggravated by the presence of occlusal overload, caused in part by improper buccolingual implant position, which due to the lack of a periodontal ligament will not be protected by an adaptive response that will help avoid peri-implant tissue damage. Nonetheless, the link between occlusion and peri-implant bone destruction remains controversial due to difficulty in conducting human clinical trials.

In many scenarios, peri-implantitis might be the ultimate result of a combination of modifiable factors that can be triggered or prevented in the surgical setting. As with any pathology in the human body where the etiology is not removed, only a temporary fix is provided. Thus, the first step in avoiding the development of peri-implantitis is prevention in the form of excellent treatment planning and surgical execution. Currently this may be the most predictable approach. However, if an implant placement error occurs the surgeon should not hesitate to back up and start over. Otherwise, the same pathology might endlessly recur. There is a significant need to improve the study of peri-implant disease by increasing research on how local factors contribute to the development of peri-implant bone loss. Reducing the number of peri-implant inflammatory situations by decreasing the number of surgical, restorative, or material complications may lower the incidence of peri-implantitis to a more realistic figure and may suggest different and more appropriate treatment approaches.

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