PERI-IMPLANTITIS: IS IT THE SURFACE OR THE ALLOY THAT’S IMPORTANT?

The increasing lifetime of the population on a worldwide scale over the last decades has led to a significant growth in the use of titanium oral implants to replace missing teeth. Longitudinal studies have reported high survival rates of the implants in function, ranging from 90% to 95% over a period up to 20 years.[1,2] This also includes the increase in revision rate of implant prostheses after the failure due to various factors. The most prominent and insidious complication around dental implants, emerging at a later stage is peri-implantitis.[3,12] A recent meta-analysis revealed a weighted mean prevalence of 22% for peri-implantitis.[4] Peri-implantitis is one of the main causes of failure in implant dentistry that affects 14%-30% of the implants.[4,5] The 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions stated that peri-implantitis is an inflammatory process from a microbial origin.[6] By definition, it is a complex multifactorial infectious disease and the inflammatory lesion in the surrounding tissues develops as a result of accumulation of bacteria on implant surfaces resulting in a biofilm.[7] Peri-mucositis and Peri-implantitis analogous to gingivitis and periodontitis respectively. They have similar pathological events but differ in extent and rapidity of tissue destruction. Experimental peri-implantitis induced by sub marginal placement of ligatures using animal models such as dogs and monkeys showed inflammatory lesions, bone loss and implant failures.[8,9] Though the microbial taxa identified in peri-implantitis is predominantly similar to periodontitis, several studies have indicated the presence of additional strains like *Staphylococcus aureus, Staphylococcus epidermidis and Candida spp.[3,10,11]* Microorganisms colonize implant surfaces and contribute to the biofilm associated infections in a manner similar to teeth.[3,12]

The physio-chemical characteristics of specific material surfaces are known to significantly influence the bacterial adhesion process. In general, surface modification of Ti implant surfaces are done to enhance osseointegration. These surface modifications lead to increased roughness, changes in surface free energy that influence the adhesion of microbes on exposed transmucosal surface. Berglundh et al. (2007),[13] studied the tissue reaction to custom-made implants with polished (Sa = 0.35 um) or roughened sandblasted, large-grit, acid-etched (SLA, Sa = 2.29 um) surfaces. The results showed spontaneous progression of peri-implantitis, more pronounced on rough surface compared to smooth surface. In a series of papers published by Albouy et al. (2008, 2009, 2012),[14,16] commercially available implant systems turned (Biomet 3i), TiOblast (Astra Tech AB), SLA (Straumann AG) and TiUnite (Nobel Biocare AB) was used. The surface roughness (Sa), varied between 1.0 and 2.0 mm for the implants of TiOblast, SLA and TiUnite, and between 0.5 and 1.0 mm for the turned surface. Though there was minimal difference in surface roughness between the four implant types, spontaneous progression of experimental peri-implantitis was most pronounced at TiUnite compared to SLA and TiOblast. The reason for this difference is not fully understood, but it may be related to other characteristics of the implant surface modifications than presented in Sa values. In addition, most published studies report the influence of modified Titanium (Ti) implant surfaces rather than newer alloys that are commercially available as an alternative. Studies to evaluate the presence of new elements in alloy composition with or without surface modification and their affinity for microbial colonization will be worth the exploration.

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