EDITORIAL

An Update on the Molecular Alterations and Inflammation Levels of Peri-implant Tissues

Rosemary Abbagnale[1], Shilpa Bhandi[2], Francesco Pagnoni[3], Dario Di Nardo[4]

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In the last years, dental implant therapy became very common, due to the higher quality results and the great predictability of the treatment.[1] Often, despite the current high-level endodontic imaging techniques, in the presence of complex molar or premolars endodontic retreatments, in many cases it is preferred to resort to the implant-prosthetic solution, which is sometimes more predictable in the long term, and often with better esthetic outcomes, and shorter operating times.[2–5]

Results of recent studies suggest that soft tissues around implants may be characterized by a higher proinflammatory state compared with soft tissues around teeth, despite adequate implant-prosthetic planning, correct implant emergence, correct prosthetic profiles, and adequate esthetics.[1,6–9] Controlling and, if possible, reducing the levels of this inflammation can guarantee long-term implant success, and slow down if not completely cancel peri-implant bone loss.[6,10–12]

Comparing the crevicular fluid molecular composition around dental implants placed with one-stage protocol and surgically accessed adjacent teeth, the authors found higher levels of pro-inflammatory cytokines around implants.[1,11] Peri-implant higher inflammation levels and early peri-implant bone resorption have been mainly explained by the effect of the inflammatory cell infiltration produced by the implant-abutment interface (micro-gap), and connective tissue stabilization, whereby the circular fibers on the rehabilitation surface protect the internal ones.[13] Moreover, the presence of cement-retained implant restorations, which may have produced intrasulcular cement remnants, can also cause an increase in peri-implant inflammation levels and subsequently the onset of peri-implantitis phenomena.[14,15]

Achieving soft tissue stabilization as coronal as possible is one of the main objectives in obtaining esthetic results, and may help to diminish early peri-implant bone resorption.[16] Further evidence in the literature reports how some surfaces and surface treatments of the implant neck guarantee greater adhesion of fibroblasts and therefore better long-term tissue stability.[1,11,16] When the connective tissue is stabilized, it prevents the apical migration of the epithelium and dictates how much bone resorption occurs.[17]

The peri-implant tissues (connective tissue and epithelium) have two functions regarding the protective role. The first one is similar to periodontal sealing and adhesion on the tooth which mitigates bacterial contamination.[7,8,18,19] The second role is related to mechanical tissue stability around the implant.[18]

The supracrestal area: This part of the restoration is a key factor for maintaining the connective tissue at the supracrestal level and, in turn, for bone preservation. It is now clear the importance of the connective tissue around the implants, and of the adequate thickness of the peri-implant tissues, keratinized and not: in this regard, the evidence regarding the grafts of matrices is always greater, with increasingly better esthetic outcomes, and less biological damage for the patient.[20,21] The abutment, in theory, must fulfill this role.[12] Unfortunately, the implant abutment lacks the capability to retain the connective tissue fibers by means of periodontal fiber insertion, unless the current possibilities of also using prosthetic components that have undergone laser treatments are exploited.[11,12] This inherent deficiency forces an apical migration of the connective tissue fibers until they are retained at the level of the first thread of the implant.

It is now clear that a correct prosthetic load is essential in the early stages of implant integration, with materials that can absorb part of the occlusal load and reduce stress at the bone-implant level, increasing the predictability of the treatment.[22]

[1,3,4]Department of Oral and Maxillo-Facial Sciences, Sapienza University of Rome, Rome, Italy

[2]Department of Restorative Dental Sciences, Jazan University, Jazan, Kingdom of Saudi Arabia

Corresponding Author: Francesco Pagnoni, Department of Oral and Maxillo-Facial Sciences, Sapienza University of Rome, Rome, Italy, Phone: +393282177087, e-mail: francesco.pagnoni22@gmail.com

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