The Microbiological Diagnosis Of The Peri-Implant Disease: Can It Really Have A Practical Significance?

Opinion

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The peri-implant disease, like periodontal disease, is supported by a bacterial load that is at the base of both its onset and, above all, of its progression. For a long time, we have been wondering about the similarity of the peri-implant mucositis with gingivitis and peri-implantitis with periodontitis. If clear and obvious are the anatomical and histological differences, rather less obvious it is the similarity on a microbiological level [1,2].

The most credited theory until recent times, and in many ways even today, is that the microflora that supports the peri-implant disease is exactly the same as that responsible for periodontal disease. To give strength to this theory, several studies conducted between the years 90 and 2000 have revealed the presence, in the peri-implant sulci of human subjects with peri-implatitis and animal models with induced peri-implantitis, of species normally present in the diseased periodontal sulcus: Porphyromonas gingivalis, Fusobacterium nucleatum, Tannerella forsythia, Treponema denticola and various species of prevotellaceae [3-13]. In addition to these findings, many studies analyzing the microflora of peri-implant crevice in fully edentulous subjects noted the almost total, or total, absence of those bacteria commonly found in the periodontal crevice, thus reinforcing the theory of the close similarity of the two environments and the bacterial reservoir role of natural teeth [14-16].

From a conceptual point of view this theory is useful to explain the importance of a correct periodontal diagnosis before starting any implant rehabilitation plan, is well known, in fact, that the presence or a history of periodontitis are indicators of risk for the onset of peri-implantitis. However, the same theory is at odds with the evidence that the peri-implant and periodontal crevices are two completely different environments and, as such, should favor or impede the development of different bacterial species or at least they should have more differentiated bacterial compositions. In fact, it is known that the promoters of bacterial adhesion, namely the formation of biofilms, are, in the first instance, the so-called van der Waals forces, repulsive or attractive forces between molecules, weaker than the bonds that form in more advanced biofilms but fundamental in the adhesion of pioneers or early colonizers. These adhesion forces are strictly dependent on the surface free energy (SFE) of the substrate on which are formed. Microorganisms with high free energy will adhere to surfaces with high SFE and vice versa. Enamel and titanium, in this case, have different SFE, 0.088 ± 0.009 J m-2 and 0.051 ± 0.001 J m-2, respectively, so they should elicit the adhesion of different bacterial populations [17,18].

In this regard, a study by Leonhardt et al. [5], analyzing the bacterial composition of periodontal and peri-implant sulci in disease and in health, had found that, if in healthy conditions the two different environments had an overlapping microflora, in sulci around implants affected by peri-implantitis was found a presence of staphylococci, enterics and yeasts with almost the same frequency of periodontopathogens, indicating a substantial difference in the compositions of the two bacterial crevices [5]. What is the difference between Leonhardt et al. study and the majority of modern studies that indicate, instead, a complete similarity between the microbial composition around teeth and implants? The study by Leonhardt et al. [5] uses an old-fashioned technique, bacterial culture, which, however, has the advantage of being "open-ended". However, the biggest drawbacks of culturing methods are the long time required and the uncultivability of numerous species.

The most modern techniques of PCR and DNA-DNA hybridization have the considerable advantage of requiring very short time, however, they are "targeted" techniques, that is, the bacteria that will be analyzed are pre-selected by choosing a certain number of DNA probes. This is the reason why all of the studies using these techniques have a strong bias due to the fact that knowledge of the crevices in terms of bacterial composition derives from the periodontal literature, therefore the analysis of peri-implant sulcus is, a priori, directed towards the search of bacteria commonly found around the teeth.

In very recent years new bacterial analysis techniques have been developed, known as metagenomics, which uses 16S rRNA sequencing, and that overcome the limits of both the cultivation and molecular methods. The metagenomic analysis is in fact both fast and "open ended". With this advancement in the microbiological field we arrived at the concept of "microbiome" intended as all the species that inhabit a certain environment in their genetic diversity and, as a result of this discovery, the human oral microbiome database (HOMD) project has been developed, with the discovery of 619 taxa arranged in 13 phyla, 54% of which has been officially named, 14% are unnamed but cultivated, the remaining 32% are categorized only as uncultivated phylotypes [19].

The, so far few, studies using these modern metagenomics methods seem to now direct towards the affirmation of a theory of bacterial diversity of the peri-implant sulcus from the periodontal one [20-23]. However, can these informations be useful in practical terms in the everyday clinical practice? Can the awareness that the bacteria around implants are different from those around natural teeth change something in our therapeutic strategies?

Surely the time is not yet ripe to answer these question with certainty, more and more detailed and complex studies may someday lead us to find a microbiological trend that is most frequently associated with the onset of peri-implant disease, thus guaranteeing an early diagnosis and more effective preventive measures. For now, we remain of the view that, although convinced that the microflora of implants and natural teeth are distinct, the host response, as in periodontal disease, certainly plays a very important role in peri-implantitis.23 Patients with a history of aggressive periodontitis, can develop forms of severe peri-implantitis, not much for the bacterial population that characterizes their oral microbiome, as for the genetic characteristics that characterize these patients (phagocyte abnormalities, hyperresponsive macrophage phenotype, elevated levels of PGE2 and IL-1, tissue destruction inconsistent with microbial deposit, etc.) [24,25]. Finally, whatever the bacterial flora, the prevention of peri-implant disease cannot be separated from the treatment of the pre-existing periodontal disease, and from a correct maintenance program after the implant rehabilitation [26,27].

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