Hyperbole, Clinical Dissonance, and Scratching the Surface: Complication or Disease?

Regular doses of advertised continuing education (CE) programs offer clinicians much scope for monitoring populist claims of better patient management or latest insights into new treatment territory. One announcement from a distinguished university recently piqued our interest. The teaser for the course description "Mucosal Diseases Around Implants: Not Just Gingivitis Anymore" alerted us to: "the veritable and approaching tsunami of cases coming our way, where patients who have been treated with endosseous titanium dental implants, will present with inflammatory disease around their implant supported prostheses." The hyperbole was qualified by the assertion that peri-implant mucosal inflammation (PIMI) is unlike gingivitis, which is after all relatively simple to treat. Moreover, PIMI is far less predictable insofar as treatment outcomes are concerned since the microbiota are different compared with gingivitis. The advertisement also asserts that the real puzzle lies in the fact that the tooth surface is of course very different from a titanium one, which makes for a host of different concerns to address when trying to treat PIMI. The announcement's clinical dissonance was increased by the failure to mention that teeth and implants have distinct attachment mechanisms; that the latter's genesis results from a healed surgical response, as opposed to teeth's attachment evolutionary outcome. Indeed, there are so many differences between the tooth-host and implant-host interfaces that the advertisement merely scratches the surface of a complex entity; suggesting otherwise would be an injustice to our profession's intellect.

Our own early emphasis on reported measurable bone levels around successfully osseointegrated implants demonstrated long-term stability of marginal bone in the anterior zones of edentulous patients. We proposed a range of optimal time-dependent marginal bone height measurements as a general yardstick to determine success outcomes for implant systems entering the marketplace. Nevertheless, many colleagues continue to regard specific bone height levels as an important criterion of successful individual implant outcomes, even though there is no evidence to support specific levels. This somewhat unidimensional approach (perhaps in retrospect even a naïve one) is reflected in recent published reports that propose adverse and specific bone changes around otherwise asymptomatic oral implants as a classifiable disease pattern similar to periodontal disease. We readily acknowledge our current imperfect understanding of the diverse events contributing to quantitative changes at the bone-implant interface. We also continue to be critical of the use of the term peri-implantitis since to do so implies a disease process similar to periodontitis, and we need to resist our profession's naturally anchored thinking regarding the tooth-host interface—forcing the proverbial square peg into a round hole. What we are trying to emphasize here, is that we must not fill the vacuum created by our incomplete understanding of the implant-host interface with our reasonably coherent understanding of the tooth-host interface/periodontium. We prefer instead to offer a conceptual framework that can be supported by current knowledge of bone changes around implants, one that accepts the tenet that bone loss requires a multifactorial model to explain its occurrence, and that we do not yet understand all the factors to the degree needed to complete the model. We regard the onset of marginal bone resorption around oral implants as an inevitable and variable time-dependent outcome, that it is only occasionally a treatment complication in some patients, and very rarely a serious clinical concern, eg, where secondary infections dominate the clinical picture.

We propose the notion of osseosufficiency—the state where the host and implant interface reflects the combined capacity to promote and perpetuate successful osseointegration. This should be the starting point for a discussion on how an implant actually interfaces with host bone in either optimal or suboptimal states, over variable time periods, in specific sites, and in the context of surgical judgment and protocol, and even whether its cervical location occurs in alveolar as opposed to basal bone. Moreover, scrupulous long-term documentation of clinical outcomes in implant therapy must also be reconciled with the numerous factors that may predispose to a compromised healing response, or osseoinsufficiency. Accepted contributors to osseoinsufficiency include unsuitable implant designs, inadequate imaging, and indeed selection of surgical sites, suboptimal operator skills in tissue manipulation, patients' brittle systemic health, and, of course, different degrees of foreign-body reaction.[1] Compromised osseoinsufficiency may then be regarded as manifesting itself clinically as variations on a theme of osseoseparation (OS). We have proposed this latter term to avoid an implied similarity to periodontitis and its well-established pathogenesis. Osseoseparation describes depleted marginal bone levels that occur with or without an accompanying gingivitis. It may be partial or total, is usually associated with individual implants, or may quite rarely involve all implants in either arch. Where required, its proposed management remains at the anecdotal level and not unlike heroic efforts to replace absent marginal bone around the natural dentition. We remain optimistic that the proof of principle associated with reported developments in regenerative treatment protocols will change the clinical outcome of the limited OS occurrences requiring intervention to address patient-centered concerns.

In the interim, the dental profession (prosthodontists in particular) must continue to evaluate the necessary research and clinical experiential analyses that drive this topic. The notion of osseointegration, or a controllable induction of an ankylotic-like interface, should not be usurped by application of simplistic assumptions that lead to invalid periodontitis-based therapies.

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Reference

1 Albrektsson T, Dahlin C, Jemt T, Sennerby L, Turri A, Wennerberg A. Implant marginal bone loss is initiated by a foreign body reaction combined with tissue provocations. Clin Implant Dent Relat Res (in press).

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