

Commentary

Peri-implantitis Through the Looking Glass

Introduction

The states of health and disease are inseparable, as the existence of one relies on the presence of the other. The definitions of health and disease in medicine and dentistry have evolved over the years from ones that are patient-centred to a complex set of terminology that has resulted in communication ambiguity. An example of such are “peri-implant diseases,” a lingering topic in the current literature.

Biological complications associated with dental implants have been recognised since the inception of implant therapy. The term *peri-implantitis* was initially described in French literature by Levignac¹ before it appeared in English literature in a landmark study by Mombelli et al.² They clearly demonstrated the site-specific characteristics of peri-implantitis with an abundance of motile rods, fusiform bacteria, and spirochetes compared with healthy implant sites. The segregation of peri-implant disease into “peri-implant mucositis” and “peri-implantitis” was endorsed in the first European Workshop on Periodontology (EWP) in 1993.³ The same definitions were later adopted in the sixth and seventh EWP in 2008 and 2011, respectively,^{4,5} and by the Glossary of Periodontal Terms of the American Academy of Periodontology in 2012. Peri-implant mucositis and peri-implantitis were both defined as a destructive chronic inflammatory process affecting the tissues around a functionally osseointegrated dental implant. The loss of supporting bone was arbitrarily used to differentiate between the 2 conditions. These same definitions were again adopted by the 2017 World Workshop classification of periodontal and peri-implant diseases and conditions,⁶ but with additional subjective thresholds for probing pocket depths and peri-implant marginal bone loss for peri-implantitis in an attempt to standardise reporting of prevalence and management of peri-implant diseases.

Other peri-implant pathologic conditions of low prevalence, and hence receiving less attention in the literature, include retrograde peri-implantitis and peri-implant abscess. Retrograde peri-implantitis is characterised by apical radiolucency, with some cases being associated with pain, tenderness, swelling, and the presence of a fistulous tract.^{7,8} Peri-implant abscess, on the other hand, is characterised by a more acute purulent inflammation that is associated with pain, swelling and, often, low-grade elevated temperature and localised lymphadenopathy.^{9,10}

Despite the widespread agreement on the aetiopathology of peri-implant diseases, the term peri-implantitis and even the existence of the condition as a disease entity has been questioned. The term peri-implantitis was conceived as loose label for a marginal bone loss that succumbed into disrepair at the altars of the “healing adaptation” hypothesis and “foreign body” reaction.^{11–14} Nevertheless, the argument on whether “peri-implantitis” exists as a separate entity lies in

the fact that not every incident of bone loss is a peri-implant disease. This argument is further complicated by the question of whether we are using the correct terminology in describing peri-implant diseases.

Facts and figures

Dental biofilm is the aetiologic factor for pathologic inflammatory conditions that affect the peri-implant tissues. The progression of these inflammatory conditions depends on patient susceptibility and other environmental factors. In the sixth and seventh EWP^{4,5} and 2017 World Workshop,⁶ the cause-and-effect relationship between supramucosal biofilm formation on implants and the development of peri-implant diseases has been highlighted. The bacterial infection was regarded as the cause of peri-implant bone loss after initial remodelling when classic signs of inflammation are evident. The marked apical extension of the lesion and the abundance of plasma cells and lymphocytes differentiate peri-implant diseases from gingivitis and periodontitis. Only in the absence of any signs of inflammation, the adaptive healing hypothesis and foreign body reaction can be used to explain the marginal bone loss and remodelling.

What is currently evident in the literature is that the prevalence of peri-implant diseases is relatively high. In fact, peri-implant diseases have been labelled as the “tsunami” of implant therapy,^{15,16} and regardless of how the levels of peri-implant marginal bone loss were defined in published reports, the fact remains that approximately 1 in every 5 patients with dental implants is at risk for inflammatory peri-implantitis.¹⁷

Gaps and shortcomings

One shortcoming in defining peri-implant diseases is the lack of clear diagnostic criteria on the amount of marginal bone loss that sets the difference between disease and physiologic “adaptation.” Historically, success criteria accepted a mean marginal bone loss of 1.0 to 1.5 mm as “adaptive” loss around machined surfaced implants in the first year of function followed by an annual average bone loss of 0.2 mm.^{3,18–20} The third EWP in 1999 called for more rigid success criteria, with a marginal bone loss of <2 mm in the first 5 years postloading.²¹ The lack of consensus on how much marginal bone loss is required to define peri-implantitis is evident. This ambiguity has resulted in a broad range of prevalence estimates that ranged from 2% to 44% depending on the unit of analysis and the case definitions adopted by the authors.^{15,17}

It is the authors’ opinion that the continued focus on establishing distinct thresholds for marginal bone loss

associated with inflammatory conditions has only led to confusion and distraction amongst clinicians. Let us consider an inflammatory peri-implant condition with bleeding, suppuration, probing pocket depths of 5.5 mm, and marginal bone loss of 2.5 mm. That condition cannot be defined as peri-implantitis per the 2017 World Workshop criteria, which require probing pocket depths of at least 6 mm and marginal bone loss of at least 3 mm to label the condition as peri-implantitis. In our opinion, the main determinants in defining the severity as well as the continuity of the peri-implant inflammatory conditions should be based on the primary signs of inflammation and bacterial infection, namely bleeding and suppuration, and additionally describing the marginal bone loss as “progressive osteitis.” In fact, the

environment of peri-implant inflammation has been recently described as one resembling a chronic nonhealing wound that affects bone homeostasis.²² In this context, the chronicity and continuity of the peri-implant inflammatory condition and its cumulative cellular damage over time might partly explain the reported challenges in managing peri-implantitis and its high recurrence rate.²²

Another gap in defining peri-implant diseases is the emphasis on the implant being the diseased organ and not the tissues surrounding it. The use of the terms *peri-implantitis* and *retrograde peri-implant disease* has added more confusion. The Greek suffix “-itis” indicates an inflammation of an organ. The association of “-itis” with a metallic device may not be appropriate, as the disease itself is an inflammatory response of the tissues

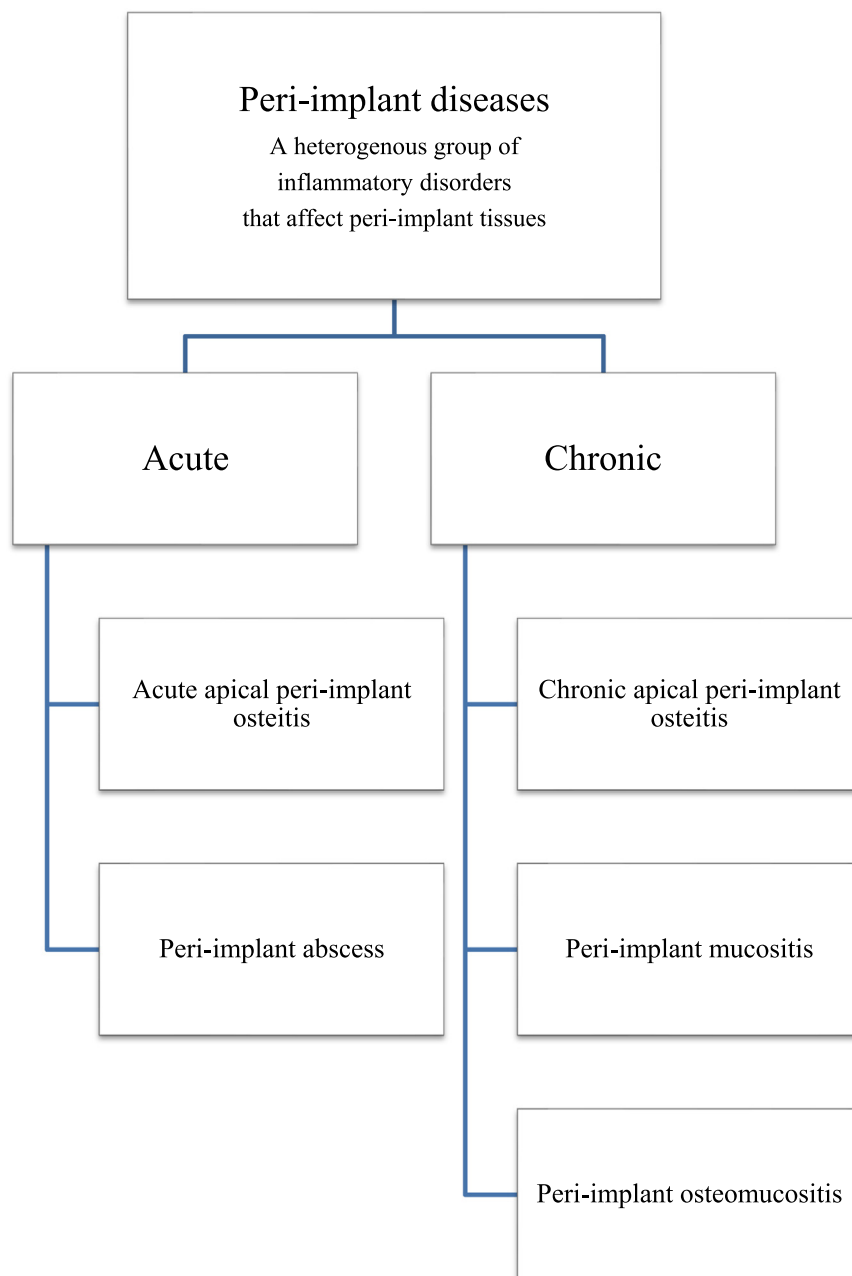


Fig – Classification of peri-implant diseases.

around the implant rather than the implant itself. Likewise, the term *retrograde* implies that the apical radiolucency, which indicates “infection,” will eventually migrate in opposite “coronal” direction. The apical radiolucency, whilst it necessitates intervention in the presence of acute symptoms (ie, pain, swelling, fistula), is often asymptomatic with a healthy osseointegration at the coronal implant interface.

The “change” before the “change”

Over the last 2 decades, peri-implant disease has been the buzz of implant dentistry. Peri-implant mucositis and peri-implantitis have been implicated as main causes of late implant failure. The well-documented high survival rate of osseointegrated dental implants is now jeopardised by peri-implant inflammatory conditions, although they are chronic in nature. Our current understanding of peri-implant disease and its consequences remains limited despite the extensive scholarly work published over the last 2 decades on its aetiology, pathogenesis, and management protocols.

To achieve a better understanding of any pathologic conditions, we should first apply the correct terminology that accurately describes it. The authors call for a classification system that accurately describes peri-implant diseases. This classification should be primarily based on whether the condition is acute or chronic and whether the bone loss involves the coronal or the apical part of the implant–bone interface. Symptomatic conditions with pain, swelling, and suppuration with or without the presence of an abscess indicate an acute condition. On the other hand, asymptomatic lesions with bleeding and deep probing pocket depths with or without bone loss are often chronic, ongoing conditions.

The authors recommend the use of the terms *peri-implant osteomucositis* instead of *peri-implantitis* and *apical peri-implant osteitis* instead of *retrograde peri-implantitis* to have a more clear and unambiguous reference to the nature of the pathologic entity (Figure). It is our conviction that proposing a new or revised terminology is very challenging, as it requires open-minded conversation and in-depth discussion amongst international periodontology and implant dentistry associations on the need to propose new diagnostic and treatment codes before full implementation can be considered. Nevertheless, the authors believe that the scientific community should be more focussed on understanding the underlying process that promotes disease onset and progression and not only on defining therapeutic protocols to treat clinical and radiographic signs of the disease. The proposed changes would have an impact on daily clinical practice, as clinicians would be able to use terms that allow them to pay more attention to the clinical intuition of the disease rather than being hindered by a set of thresholds and cutoff values. Moreover, the revised terms could potentially improve surveillance of peri-implant diseases in epidemiologic and clinical research.

Summary

The proposed terminology of peri-implant diseases would allow a more scientific description of the pathologic changes

around dental implants, would facilitate communication between researchers and clinicians, and may further set the ground for establishing a consensus on treatment guidelines focusing on inflammatory parameters rather than being lost in defining thresholds of probing pocket depths and marginal bone loss. The buzz around peri-implant diseases is here to stay, and management of implant inflammatory conditions will remain one of the fastest-growing implant research sectors over the coming years.

Conflict of interest

None disclosed.

REFERENCES

1. Levigac J. L'osteolyse periimplantaire, periimplantose. *Rev French Odontostomatol* 1965;12:1251–60.
2. Mombelli A, van Oosten MA, Schurch Jr E, Land NP. The microbiota associated with successful or failing osseointegrated titanium implants. *Oral Microbiol Immun* 1987;2:145–51.
3. Albrektsson T, Isidor F. Consensus report of session IV. In: Lang NP, Karring T, editors. *In: Proceedings of the 1st European Workshop on Periodontology*. London, PA. Quintessence; 1994. p. 365–9.
4. Lindhe J, Meyle J, Group D of European Workshop on Periodontology. Peri-implant diseases: Consensus Report of the Sixth European Workshop on Periodontology. *J Clin Periodontol* 2008;35:282–5.
5. Lang NP, Berglundh T, Working Group 4 of Seventh European Workshop on Periodontology. Periimplant diseases: where are we now?—Consensus of the Seventh European Workshop on Periodontology. *J Clin Periodontol* 2011;38(Suppl 11):178–81.
6. Berglundh T, Armitage G, Araujo MG, et al. Peri-implant diseases and conditions: Consensus report of Workgroup 4 of the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions. *J Periodontol* 2018;89(Suppl 1):S313–8.
7. Quirynen M, Vogels R, Alsaadi G, Naert I, Jacobs R, van Steenberghe D. Predisposing conditions for retrograde peri-implantitis, and treatment suggestions. *Clin Oral Implants Res* 2005;16:599–608.
8. Di Murro B, Canullo L, Pompa G, Di Murro C, Papi P. Prevalence and treatment of retrograde peri-implantitis: a retrospective cohort study covering a 20-year period. *Clin Oral Investig* 2021;25:4553–61.
9. Piattelli A, Scarano A, Piattelli M. Abscess formation around the apex of a maxillary root form implant: clinical and microscopical aspects. A case report. *J Periodontol* 1995;66:899–903.
10. Balshi TJ, Pappas CE, Wolfinger GJ, Hernandez RE. Management of an abscess around the apex of a mandibular root form implant: clinical report. *Implant Dent* 1994;3:81–5.
11. Albrektsson T, Buser D, Sennerby L. On crestal/marginal bone loss around dental implants. *Int J Oral Maxillofac Implants* 2012;27:736–8.
12. Albrektsson T, Buser D, Sennerby L. On crestal/marginal bone loss around dental implants. *Int J Prosthodont* 2012;25:320–2.
13. Albrektsson T, Buser D, Chen ST, et al. Statements from the Estepona consensus meeting on peri-implantitis, February 2–4, 2012. *Clin Implant Dent Relat Res* 2012;14:781–2.
14. Koka S, Zarb G. On osseointegration: the healing adaptation principle in the context of osseosufficiency, osseoseparation, and dental implant failure. *Int J Prosthodont* 2012;25:48–52.

15. Huynh-Ba G. Thematic abstract review: peri-implantitis: "tsunami" or marginal problem? *Int J Oral Maxillofac Implants* 2013;28:333–7.
16. Fu JH, Wang HL. Breaking the wave of peri-implantitis. *Periodontol* 2000 2020;84:145–60.
17. Atieh MA, Alsabeeha NH, Faggion Jr CM, Duncan WJ. The frequency of peri-implant diseases: a systematic review and meta-analysis. *J Periodontol* 2013;84:1586–98.
18. Adell R, Lekholm U, Rockler B, Branemark PI. A 15-year study of osseointegrated implants in the treatment of the edentulous jaw. *Int J Oral Surg* 1981;10:387–416.
19. Adell R, Lekholm U, Rockler B, et al. Marginal tissue reactions at osseointegrated titanium fixtures (I). A 3-year longitudinal prospective study. *Int J Oral Maxillofac Surg* 1986;15:39–52.
20. Albrektsson T, Zarb G, Worthington P, Eriksson AR. The long-term efficacy of currently used dental implants: a review and proposed criteria of success. *Int J Oral Maxillofac Implants* 1986;1:11–25.
21. Wennstrom J, Palmer R. Consensus report of session 3: clinical trials. In: Land NP, Karring T, Lindhe J, editors. *In: Proceedings of the 3rd European workshop on periodontology implant dentistry*. Berlin. Quintessence; 1999. p. 255–9.
22. Ganesan SM, Dabdoub SM, Nagaraja HN, Mariotti AJ, Ludden CW, Kumar PS. Biome-microbiome interactions in peri-implantitis: a pilot investigation. *J Periodontol* 2022;93:814–23.

Momen A. Atieh*

Mohammed Bin Rashid University of Medicine and Health Sciences,
Hamdan Bin Mohammed College of Dental Medicine, Dubai
Healthcare City, Dubai, United Arab Emirates
Sir John Walsh Research Institute, Faculty of Dentistry, University
of Otago, Dunedin, New Zealand

Nabeel H.M. Alsabeeha

Department of Dental Services, Emirates Health Services, Dubai,
United Arab Emirates

*Corresponding author. Mohammed Bin Rashid University of
Medicine and Health Sciences, Hamdan Bin Mohammed
College of Dental Medicine, Dubai Healthcare City, Dubai,
United Arab Emirates.

E-mail address: maatieh@gmail.com (M.A. Atieh).

0020-6539/© 2023 The Authors. Published by Elsevier Inc. on
behalf of FDI World Dental Federation. This is an open access
article under the CC BY-NC-ND license

(<http://creativecommons.org/licenses/by-nc-nd/4.0/>)

<https://doi.org/10.1016/j.identj.2023.09.001>