

The Etiology and Causative Factors in Peri-Implant Disease: Mini Review

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Abstract

Aim: To detect the high and low risk factors behind the peri-implantitis disease that will provide evidence to clinician during diagnosis stage

Material and method: An online and hand-search of the published literature was conducted to identify studies that Examined peri-implantitis. The search terms that were used, alone or in combination, were peri-implant mucositis, peri-implantitis, implant complication and crestal bone resorption.

Results: Sixty-four studies were selected for comparison and to address the details emphasized in this study. Some of the articles were not directly related to peri-implant diseases but were reviewed for better understanding histology and examination of the tissue around implant.

Conclusion: Peri-implantitis disease is a multi-factorial disease that shared between the surgical, prosthodontic, tissue health and the patient care. plaque retention around implant with weak soft tissue attachment in combined with poor hard and soft tissue thickness all work together to initiate per-implant disease.

Keywords: dental implant infection, bone resorption, bleeding on probing, gingival recession

Introduction

Dental implants are commonly used for the replacement of missing teeth in patients with partial or complete edentulism. Results of long-term follow-up studies (Toy & Uslu, 2020; Chrcanovic et al., 2020; Kim et al., 2020) have shown that dental implants can demonstrate success and survival rates of 100%. However, biological complications associated with dental implants (peri-implant diseases, namely peri-implant mucositis and peri-implantitis) may jeopardize the integrity of peri-implant mucosa and supporting alveolar bone (Klinge et al., 2018). Different methods have been used to assess peri-implant tissue health and to diagnose these disease entities. These methods include peri-implant probing, analyses of peri-implant crevicular fluid or saliva, evaluation of the peri-implant microbiota and radiographic evaluation of the peri-implant bone levels. The current consensus indicates that changes in probing depth, and the presence of bleeding on probing and suppuration, must be evaluated to assess the peri-implant tissues, whilst radiographs should be used to confirm peri-implant bone loss (Serino et al., 2013). The reported prevalence of peri-implantitis varies from less than 7% to 37% of implants (Klinge, 2012). The variation can be attributed to differences in studied populations, length of follow-up time, implant variables, and the criteria used to define peri-implantitis (Koldslund et al., 2010). Two systematic reviews concluded that peri-implantitis affected 10% of implants and 20% of patients during the 5 to 10 years after placement (Mombelli et al., 2012; Atieh et al., 2013).

Aims of the review

To focus on the role of plaque-biofilm to jeopardize the implant health.

And the importance role of the maintenance phase in prevention of implant failure, and increase the successful rate.

Materials and Methods

A search of the MEDLINE (PubMed) and Google scholar database was conducted, and the works published in the English language from 1990 until 2023 were included in the review. The search terms that were used, alone or in combination, were peri-implant mucositis, peri-implantitis, implant complication and crestal bone resorption. Titles and abstracts were screened, and a full-text analysis was performed for relevant publications. A manual search was conducted

for the following journals from 1990 until 2023: Clinical Implant Dentistry and Related Research; Clinical Oral Implants Research; International Journal of Oral & Maxillofacial Implants; Journal of Clinical Periodontology; Journal of Dental Research; International Journal of Periodontics and Restorative Dentistry; Journal of Periodontology; Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontics; British Journal of Oral and Maxillofacial Surgery; and Journal of Prosthetic Dentistry.

Inclusion criteria including studies with the following designs were included

- 1-Randomized controlled clinical trials, controlled trials, and prospective and retrospective clinical studies
- 2- invitro studies
- 3-literature review

The following publications were excluded from our data

- 1-Case reports or a case series
- 2-Studies in a language other than English or without an English language abstract

The two authors independently reviewed the included studies to assess their conformity with the inclusion criteria.

Results

The combinations of search terms resulted in a list of 140 titles (PubMed until 2021 and Google scholar). Following the screening of the titles and abstracts by applying the defined inclusion and exclusion criteria, 64 potentially relevant publications were identified in which a full-text analysis was performed. The number of articles that discuss the peri-implant disease were 19 articles. Some of the included articles were not directly related to peri-implant disease but were reviewed to better understand the process of peri-implant healthy tissue structures and implant survival and success criteria. Relevant articles discussed the local and systemic etiological factors, bacterial types, crestal bone resorption, implant occlusion and prosthetic types.

Discussion

Peri-implant health: Peri-implant health requires the absence of clinical signs of inflammation, including no bleeding on probing. Around clinically healthy implants, the mucosa forms a tight seal around the trans-mucosal component of the implant itself, the abutment or the restoration. The soft tissue height around the implant following placement determines the initial probing depth. In most cases, the probing depth associated with peri-implant health should be ≤ 5.0 mm. As part of the definition, there should be no bone loss greater than the bone level changes which occur after initial bone remodeling immediately following implant placement (Renvert et al., 2018). Healthy peri-implant tissue showed the mucosa covered by stratified squamous epithelium; in addition, a layer of vascular fibrous connective tissue was evident. A few stromal inflammatory cells and rarely some lymphoid cells in the basal layer were observed (Lucarini et al., 2019)

Peri-implant mucositis

This inflammatory response is strictly limited to the soft tissue, with no evidence of progressive bone loss subsequent to the initial remodeling after implant placement, and is known to be reversible (Ramanauskaite et al., 2016). The main clinical sign of this lesion is inflammation of the peri-implant mucosa characterized by bleeding on gentle probing (< 0.25 Ncm) (Jepsen et al., 2015). Peri-implant mucositis displayed an inflammatory infiltrate at the level of the connective tissue lateral to the barrier epithelium (Lucarini et al., 2019)

Peri-implantitis

Peri-implantitis was defined by the 2017 Proceedings of the World Workshop as “a plaque-associated pathologic condition occurring in the tissue around dental implants, characterized by inflammation in the peri-implant mucosa and subsequent progressive loss of supporting bone.” The authors stated that clinically, the inflammation around implants is manifested as erythema, edema, mucosal enlargement, bleeding on probing (67%) with or without suppuration (94%); with deeper probing depths (PD ≥ 6 mm at 59%) and bone loss radiographically with a combined supra and infra-osseous configuration progressing circumferentially around implants and faster than around teeth (Schwarz et al., 2018). In the absence of baseline radiographs and probing depths, radiographic bone level ≥ 3 mm and/or probing depths ≥ 6 mm in conjunction with profuse bleeding represents peri-implantitis (Renvert et al., 2018). In patients with poor maintenance compliance, it has been observed there is a higher risk of developing peri-implantitis, especially if there is bleeding on probing. It has been shown that for implants presenting bleeding on probing, there was a 24.1% chance of being diagnosed with peri-implantitis (Hashim et al., 2018). In peri-implantitis tissue, adjacent to an ulcerated pocket epithelium, a great inflammatory lesion showing an evident granulation tissue and a dense inflammatory infiltrate was

detected (Lucarini et al., 2019) Risk factors of peri-implantitis 1-Patient Related Risk Factors Poor Plaque Control and Peri-Implant Mucositis A patient's self-performed plaque control is one of the most important factors influencing the implant's prognosis (Schwarz et al., 2018). A high plaque index was associated with an eightfold increase in susceptibility to peri-implantitis (Kumar et al., 2018). The accumulation of bacterial biofilm on implant and abutment surfaces leads to periimplant inflammation, also known as mucositis, Peri-implantitis is always preceded by a period of mucositis. The two share several risk factors including poor oral hygiene, smoking and sub- mucosal presence of excess cement. Implants diagnosed with mucositis are at risk of developing peri-implantitis (Heitz-Mayfield & Salvi, 2018). However, not all mucositis lesions progress to peri-implantitis, even when present for extensive periods of time (Gualini & Berglundh, 2003)

Periodontal Disease and Microbiological Aspects

The diagnosis, or history, of periodontal disease is the most researched factor associated with peri-implantitis. This is partially attributed to similarities in the subgingival microbiota between the diseased teeth and implants (Ferreira et al., 2018). Current data suggests that peri-implantitis is associated with a specific microbiota resembling that of periodontal lesions, in addition to other microorganisms not commonly related to periodontitis (Faveri et al., 2015).

Nevertheless, it is well-accepted that peri-implantitis consistently presents with marked microbial diversity (Canullo et al., 2015) and that deeper peri-implant pockets exhibit significant microbial alterations and higher levels of dysbiosis (Kroger et al., 2018). Periodontal disease has been strongly associated with peri-implantitis (Saaby et al., 2016). Active periodontitis at the adjacent teeth is further considered a predictor of future peri-implantitis (Kumar et al., 2018). Periodontally compromised patients have

twice the risk of developing peri-implantitis compared with healthy individuals (Ferreira et al., 2018). Moreover, those with a history of generalised aggressive periodontitis are 5 times more prone to implant failure, and 14 times more susceptible to periimplantitis, compared with healthy (Swierkot et al., 2012). Fortunately, successful treatment of periodontal disease prior to implant placement has been shown to lower the risk of peri-implantitis and is therefore considered an essential initial part of the overall treatment plan (Renvert & Quirynen, 2015).

Lack of Maintenance phase

Has been shown to significantly lower the risk of peri-implant biological complications, and a minimum recall interval of 5–6 months has thus been recommended (Monje et al., 2016). Maintenance programs should be tailored to the individual's specific needs and susceptibility to both periodontal and peri-implant diseases. Factors used for risk assessment include the percentage of BOP, the prevalence of active residual pockets, oral hygiene level, smoking habits and the presence of systemic or genetic conditions (Lang et al., 2015). Individuals with high risk profiles require three to four annual visits (Armitage & Xenoudi, 2016), and their attendance is detrimental for prevention and early detection of peri-implantitis (Monje et al., 2017). One out of five noncompliant patients are diagnosed with peri-implantitis within 5 years (Rokn et al., 2017), On the other hand, compliance is associated with 86% fewer peri-implantitis cases. Unfortunately, those with greater needs have been known to be the least compliant. The extent and severity of periodontal disease, as well as the patient's smoking habits, affect adherence to maintenance programs (Monje et al., 2017). Therefore, it is the clinicians' duty to adequately inform their patients of the importance of regular supportive therapy for the prevention of peri-implantitis.

Smoking

The negative effects of smoking on periodontal health have long been well established. It impacts innate and adaptive immune responses, impairing the host's defence mechanisms and its response to microbial challenges (Johnson & Guthmiller, 2007). Cigarette smoking also affects wound healing, as it is therefore detrimental to periodontal treatment (Trombelli et al., 2018). Smoking further increases the oxidative stress and inflammatory burden with marked alterations in microbial flora, it significantly affects implants' colonisation with periodontal pathogens such as *Porphyromonas gingivalis* (Pg) and *Fusobacterium nucleatum* (Geisinger et al., 2017). Besides, cigarettes are not only harmful to smokers, but mere exposure to environmental smoke increases the risk of developing periodontal disease by 28% (Sutton et al., 2017). Smokers are almost twice more at risk of developing periimplantitis compared with nonsmokers (Dreyer et al., 2018). Moreover, it is associated with increased severity of peri-implantitis (Saaby et al., 2016). Nevertheless, smoking cessation has been shown to positively impact periodontal health, with favourable effects on both incidence and progression of the disease (Johannsen et al., 2014).

In addition to cigarettes commerciality, the popularity of noncigarette tobacco products has been alarmingly rising. Water pipes, also known as shisha, hookah or narjilah, have become a popular way of smoking tobacco among adolescents and adults alike (Kim et al., 2016). Their recreational use has become widely acceptable despite containing high levels of nicotine, and a multitude of carcinogens and heavy metal. Electronic cigarettes (e-cigarettes), or vaping, have lately

become an extremely widespread trend among individuals of all ages. Regardless of their nicotine content, e-cigarettes have been shown to increase oxidative/ carbonyl stress and pro-inflammatory responses, with adverse effects on endothelial cells and fibroblasts, and concomitant dysregulation in periodontal repair (Al-Aali et al., 2018).

Systemic Conditions

Diabetes mellitus is one of the most thoroughly researched conditions. The disease affects insulin's secretion, its function or both, causing disruption of glycaemic levels. This consequently results in a variety of neuropathological, retinal, microvascular and renal complications (American DA., 2010). Poor glycaemic control plays a pivotal role in the progression and severity of periodontitis (Lalla & Papapanou, 2011). This association has been explained by several vascular and cellular responses, leading to enhanced tissue destruction and impaired healing response (Knight et al., 2016). Poorly controlled diabetics are at 46% higher risk of developing peri-implantitis, with deeper peri-implant pockets and higher marginal bone loss, compared with their normoglycaemic controls (Turri et al., 2016). Interestingly, smokers and poorly controlled diabetics are considered at a similar risk for peri-implantitis. On the other hand, non-smokers with poor glycaemic control are 3.39 times at higher risk of developing peri-implantitis compared with normoglycaemic individuals (Monje et al., 2017).

Obesity is another highly prevalent condition with detrimental effects on periodontal health (Vohra et al., 2018). Obesity is also associated with a generalised and constant hyper-inflammatory state, causing an altered immune response and increased production of proinflammatory cytokines, which adversely affect periodontal tissues and alveolar bone levels (Pham et al., 2018). Clinical studies have established obesity as a risk factor for peri-implantitis (Vohra et al., 2018). When compared with individuals with normal body weight, obese patients present with significantly higher percentages of BOP, deeper peri-implant probing depths and increased marginal bone loss (Alkhudhairy et al., 2018).

Despite their prevalence, few studies have examined the association between cardiovascular diseases and peri-implantitis. Most showed a significantly higher risk of peri-implantitis and additional bone loss for patients suffering from heart disease (Ting et al., 2018).

Recently published systematic reviews have failed to confirm or refute osteoporosis, rheumatoid arthritis and Crohn's syndrome as a potential risk factor in the pathogenesis of peri-implantitis highlighting the need for randomized control trials in this regard to help evaluate this possibility (Ting et al., 2018).

Genetic Predisposition

Lee et al. in 2014 studied the role of genetic polymorphism in 6 patients with severe peri-implantitis and high rates of implant failure. They concluded that various gene sets are indirectly linked to the dysregulation of metal ion concentration like Ca^{2+} and Mn^{2+} that impair the activation of integrins and other factors that govern cell adhesion. Poor cell adhesion affects the process of osseointegration of implants and modifies the host immune response. IL-6 G174C polymorphism has been linked to periodontitis and peri-implantitis. The association between CD14-159 C/T and TNF α -308 A/G polymorphisms with peri-implantitis has been confirmed in a population of 369 Caucasian individuals (Rakic et al., 2015).

2-Prosthetic related factor Occlusal Overload and Para-Functional Habits

Occlusal overload of implant-supported prostheses is a controversial subject, and the exact mechanism in which it causes marginal bone loss is still debatable (Pellegrini et al., 2016). Yet several studies have demonstrated that overloading an implant beyond a certain threshold leads to marginal bone loss (Isidor, 1997; Miyata et al., 2002; Fu et al., 2012). The effect of overloading on peri-implant bone levels can be accentuated by sub-optimal implant positioning, poorly designed prosthetic reconstructions, inadequate bone quantity or its poor quality. Para-functional habits leading to elevated non-axial occlusal forces may also increase marginal bone loss (Fu et al., 2012). Attrition and wear of natural dentition or prosthetic reconstructions may be used

for diagnosis of occlusal overload and parafunctional habits. The presence of wear facets on implant supported prostheses is associated with a 2.4 increase in the prevalence of peri-implantitis (Dalago et al., 2017)

Implant Material and Surface Characteristics

Micro cavities are present at implant-abutment connection level in two-piece implant systems, a consequence of current manufacturing limitation that allow bacterial infiltration and inflammation around the neck of the implant (Penarrocha-Diago et al., 2017). Surface modifications creating micro-rough implant surfaces accelerate the osseointegration process of titanium implants. A systematic review and meta-analysis by Rakic et al. that the prevalence of peri-implantitis was 18.5% at the patient level and 12.8% at the implant level, as well as, implant surface characteristics could play a major role in the initiation of peri-implantitis (Rakic et al., 2018). Moreover, the review stated demonstrated a significant association between moderately rough surfaces associated with a low prevalence rate of peri-implantitis. So far, titanium has been the material of choice in implant dentistry. Nonetheless, zirconia ceramic implants have been

progressively emerging (Hashim et al., 2016). Zirconia's greatest assets lie in its biocompatibility, superior soft tissue integration (Cionca et al., 2017) low affinity to plaque (Roehling et al., 2017) and reduced inflammatory processes when compared with titanium (Cionca et al., 2017). It was hence hypothesized that zirconia implants would finally offer the solution for peri-implant disease. Unfortunately, a recent animal study had clearly demonstrated that zirconia implants can be affected by peri-implantitis (Roehling et al., 2019). Still, zirconia demonstrated significantly lower marginal bone loss compared with titanium implants with similar surface topographies. Clinical studies have also demonstrated different degrees of bone loss around zirconia implants with variable designs (Pieralli et al., 2017) but additional long-term data is still required to establish both prevalence and treatment protocols.

Residual cement

In a retrospective analysis by Linkevicius et al in 2013, it was observed that residual cement predisposes a patient to peri-implant disease; the risk is further elevated in case of individuals with a history of periodontitis (Linkevicius et al., 2013). Thus in such cases screw retained prosthesis is a better treatment option. Surgical related factor While the number of implants does not seem to influence the risk for peri-implantitis (Passoni et al., 2014), their position is critical for long-term success. Implant malpositioning represents a significant risk factor for peri-implantitis (Canullo et al., 2016). Crestal bone resorption could occur when an implant is placed too close to the natural teeth or even other implants (Lindhe et al., 2015). This could compromise access for plaque control, and thus increase the risk of peri-implant disease. Also, fixtures located outside the bony envelope or those with thin facial bone (< 1 mm) are more prone to mucosal recession, especially in patients with thin biotypes. This exposure of the fixture's rough surface increases plaque retention (Giovannoli et al., 2019), and thus the risk of peri-implantitis. Bone and/or soft tissue grafting is recommended in such cases (Lindhe et al., 2015), keeping in mind that augmentation procedures do not increase the risk of biological complications (Salvi et al., 2018). Moreover, placing an implant 6 mm or more apical to the cemento-enamel junction of the neighbouring teeth increases its risk of peri-implantitis 8.5 times. A deep submucosal position also complicates plaque control and increases the susceptibility to peri-implant inflammation (Kumar et al., 2018). with or without concomitant clinical signs of inflammation, such as redness, edema, fistula, and/or abscess formation. Endodontic evaluation of teeth adjacent to implant sites should be performed for primary prevention of periapical peri-implantitis (Sarmast et al., 2017).

Conclusions

- 1-peri-implant disease has multifactorial causes that need to address and prevent them from working together
- 2-There is a strong relationship between bacterial plaque and the development of peri-implantitis.
- 3-plaque retention around implant with weak soft tissue attachment in combined with poor hard and soft tissue thickness all work together to initiate peri-implant disease.
- 4-most of delay implants placed in compromised sites (loss of hard and soft tissues), these sites if not rehabilitated will lead into compromised relation between the prosthesis and the adjacent tissues. This will lead into difficult oral hygiene maintenance.

Abbreviations

Abbreviation	Definition
PPD	Probing pocket depth
BOP	Bleeding on probing
BL	Bone level
CBL	Crestal bone level
CHX	Chlorhexidine
CA	Citric acid
HP	Hydrogen peroxide
GCF	Gingival crevicular fluid
EDTA	Ethylidiaminetetraacetic acid
PEEK	Polyetheretherketone
AP	Air power

References

1. Al-Aali KA, Alrabiah M, ArRejaie AS, Abduljabbar T, Vohra F, Akram Z. (2018). Peri-implant parameters, tumor necrosis factor-alpha, and interleukin-1 beta levels in vaping individuals. *Clin Implant Dent Relat Res*, 20(3):410-415.
2. Alkhudhairy F, Vohra F, Al-Kheraif AA, Akram Z. (2018). Comparison of clinical and radiographic peri-implant parameters among obese and non-obese patients: a 5-year study. *Clin Implant Dent Relat Res*, 20(5):756-762.
3. American DA. (2010). Diagnosis and classification of diabetes mellitus. *Diabetes Care*, 33(1): S62-S69.
4. Armitage GC, Xenoudi P. (2016). Post-treatment supportive care for the natural dentition and dental implants. *Periodontol*, 71(1):164-84.
5. Atieh MA, Alsabeeha NH, Faggion CM Jr, et al. (2013). The frequency of peri-implant diseases: a systematic review and meta-analysis. *J Periodontol*, 84:1586-1598.
6. Berglundh T, et al. (2018). 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 Clin Periodontol*, 45(20): S286-S91.
7. Canullo L, Penarrocha-Oltra D, Covani U, Rossetti PH. (2015). Microbiologic and clinical findings of implants in healthy condition and with peri-implantitis. *Int J Oral Maxillofac Implants*, 30(4):834-842.
8. Canullo L, Tallarico M, Radovanovic S, Delibasic B, Covani U, et al. (2016). Distinguishing predictive profiles for patient-based risk assessment and diagnostics of plaque induced, surgically and prosthetically triggered peri-implantitis. *Clin Oral Implants Res*, 27:1243-1250.
9. Cionca N, Hashim D, Mombelli A. (2017). Zirconia dental implants: where are we now, and where are we heading? *Periodontol*, 73(1):241-258.
10. Dalago HR, Schuldt Filho G, Rodrigues MA, Renvert S, Bianchini MA. (2017). Risk indicators for peri-implantitis. A cross-sectional study with 916 implants. *Clin Oral Implants Res*, 28(2):144-150.
11. Dreyer H, Grischke J, Tiede C, Eberhard J, Schweitzer A, Toikkanen SE, et al. (2018). Epidemiology and risk factors of peri-implantitis: A systematic review. *J Periodontol Res*. 53(5): 657-681.
12. Faveri M, Figueiredo LC, Shibli JA, Perez-Chaparro PJ, Feres M. (2015). Microbiological diversity of peri-implantitis biofilms. *Adv Exp Med Biol*, 830:85-96.
13. Ferreira SD, Martins CC, Amaral SA, Vieira TR, Albuquerque BN, Cota LOM, et al. (2018). Periodontitis as a risk factor for peri-implantitis: systematic review and meta-analysis of observational studies. *J Dent*, 79:1-10.
14. Fu JH, Hsu YT, Wang HL. (2012). Identifying occlusal overload and how to deal with it to avoid marginal bone loss around implants. *Eur J Oral Implantol*, 5(1):S91-S103.
15. Geisinger ML, Geurs NC, Ogdon D, Reddy MS. (2017). Commentary: targeting underlying biologic mechanisms in selecting adjunctive therapies to improve periodontal treatment in smokers: a commentary. *J Periodontol*, 88(8):703-710.
16. Giovannoli JL, Rocuzzo M, Albouy JP, Duffau F, Lin GH, Serino G. (2019). Local risk indicators-consensus report of working group 2. *Int Dent J*, 69(2):7-11.
17. Gualini F, Berglundh T. (2003). Immunohistochemical characteristics of inflammatory lesions at implants. *J Clin Periodontol*, 30(1):14-18.
18. Hashim D, Cionca N, Combescure C, Mombelli A. (2018). The diagnosis of peri-implantitis: a systematic review on the predictive value of bleeding on probing. *Clin Oral Implants Res*, 29(16):276-293.
19. Hashim D, Cionca N, Courvoisier DS, Mombelli A. (2016). A systematic review of the clinical survival of zirconia implants. *Clin Oral Investig*, 20(7):1403-1417.
20. Heitz-Mayfield LJA, Salvi GE. (2018). Peri-implant mucositis. *J Periodontol*, 89(1):S257-S66.
21. Isidor F. (1997). Histological evaluation of peri-implant bone at implants subjected to occlusal overload or plaque accumulation. *Clin Oral Implants Res*, 8(1):1-9.
22. Jepsen S, Berglundh T, Genco R, Aass AM, Demirel K, Derks J, et al. (2015). Primary prevention of peri-implantitis: managing peri-implant mucositis. *J Clin Periodontol*, 42(16):S152-S157.
23. Johannsen A, Susin C, Gustafsson A. (2014). Smoking and inflammation: evidence for a synergistic role in chronic disease. *Periodontol*, 64(1):111-126.
24. Kim MJ, Yun PY, Chang NH, Kim YK. (2020). The long-term evaluation of the prognosis of implants with acid-etched surfaces sandblasted with alumina: a retrospective clinical study. *Maxillofac Plast Reconstr Surg*, 42:10.
25. Klinge B, Klinge A, Bertl K, Stavropoulos A. (2018). Peri-implant diseases. *Eur J Oral Sci*, 126(1):88-94.
26. Klinge B. (2012). Peri-implant marginal bone loss: an academic controversy or a clinical challenge? *Eur J Oral Implantol*, 5:S13-S19.

27. Knight ET, Liu J, Seymour GJ, Faggion CM Jr, Cullinan MP. (2016). Risk factors that may modify the innate and adaptive immune responses in periodontal diseases. *Periodontol*, 71(1):22-51.
28. Koldslund OC, Scheie A, Aass AM. (2010). Prevalence of periimplantitis related to severity of the disease with different degrees of bone loss. *J Periodontol*. 81:231-238.
29. Kroger A, Hulsmann C, Fickl S, Spinell T, Huttig F, Kaufmann F, et al. (2018). The severity of human peri-implantitis lesions correlates with the level of submucosal microbial dysbiosis. *J Clin Periodontol*, 45(12):1498-1509.
30. Kumar PS, Dabdoub SM, Hegde R, Ranganathan N, Mariotti A. (2018). Site-level risk predictors of peri-implantitis: a retrospective analysis. *J Clin Periodontol*, 45(5):597-604.
31. Lalla E, Papapanou PN. (2011). Diabetes mellitus and periodontitis: a tale of two common interrelated diseases. *Nat Rev Endocrinol*, 7(12):738-748.
32. Lang NP, Suvan JE, Tonetti MS. (2015). Risk factor assessment tools for the prevention of periodontitis progression: a systematic review. *J Clin Periodontol*, 42(16):S59-S70.
33. Lindhe J, Lang NP, Berglundh T, Giannobile WV, Sanz M. (2015). Clinical periodontology and implant dentistry. Sixth edition. ed. Chichester: West Sussex, John Wiley and Sons, Inc.
34. Linkevicius T, Puisys A, Vindasiute E, Linkeviciene L, Apse P (2013) Does residual cement around implant-supported restorations cause peri-implant disease? A retrospective case analysis. *Clin Oral Implants Res* 24:1179-1184.
35. Lucarini G, Zizzi A, Rubini C, Ciolino F, Aspriello SD. (2019). VEGF, Microvessel Density, and CD44 as Inflammation Markers in Periimplant Healthy Mucosa, Peri-implant Mucositis, and Periimplantitis: Impact of Age, Smoking, PPD, and Obesity. *Inflammation*, 42(2):682-689.
36. Miyata T, Kobayashi Y, Araki H, Ohto T, Shin K. (2002). The influence of controlled occlusal overload on peri-implant tissue. Part 4: a histologic study in monkeys. *Int J Oral Maxillofac Implants*, 17(3):384-390.
37. Mombelli A, Müller N, Cionca N. (2012). The epidemiology of periimplantitis. *Clin Oral Implants Res*, 23(6):67-76.
38. Monje A, Aranda L, Diaz KT, Alarcon MA, Bagramian RA, Wang HL, et al. (2016). Impact of maintenance therapy for the prevention of periimplant diseases: a systematic review and meta-analysis. *J Dent Res*, 95(4):372-379.
39. Monje A, Catena A, Borgnakke WS. (2017). Association between diabetes mellitus/hyperglycaemia and peri-implant diseases: systematic review and meta-analysis. *J Clin Periodontol*. 2017;44(6):636-648.
40. Monje A, French D, Nart J, Rakic M. (2020). Insights into the Clinical Diagnosis of Peri-implantitis: to Probe or Not to Probe. *Curr Oral Health Rep*, 7:304-331.
41. Passoni BB, Dalago HR, Schuldt Filho G, Oliveira de Souza JG, Benfatti CA, Magini RES, et al. (2014). Does the number of implants have any relation with peri-implant disease? *J Appl Oral Sci : revista FOB*, 22(5):403-408.
42. Penarrocha-Diago M, Penarrocha-Diago M, Zaragoza-Alonso R, Soto-Penalosa D, On Behalf of The Ticare Consensus M. (2017). Consensus statements and clinical recommendations on treatment indications, surgical procedures, prosthetic protocols and complications following All-On-4 standard treatment. 9th MozoGrau Ticare Conference in Quintanilla, Spain. *J Clin Exp Dent*, 9:e712-e715.
43. Pham TAV, Kieu TQ, Ngo LTQ. (2018). Risk factors of periodontal disease in Vietnamese patients. *J Investig Clin Dent*. 9(1).
44. Pieralli S, Kohal RJ, Jung RE, Vach K, Spies BC. (2017). Clinical outcomes of zirconia dental implants. *J Dent Res*, 96(1):38-46.
45. Rakic M, Galindo-Moreno P, Monje A, Radovanovic S, Wang HL, et al. (2018). How frequent does peri-implantitis occur? A systematic review and meta-analysis. *Clin Oral Investig*, 22:1805-16.
46. Rakic M, Petkovic-Curcin A, Struillou X, Matic S, Stamatovic N, et al. (2015). CD14 and TNFalpha single nucleotide polymorphisms are candidates for genetic biomarkers of peri-implantitis. *Clin Oral Investig*, 19:791-801.
47. Ramanauskaite A, Juodzbaly G. (2016). To "zu" m TF. Apical/retrograde periimplantitis/ implant periapical lesion: etiology, risk factors, and treatment options: a systematic review. *Implant Dent*, 25:684-697.
48. Ramel CF, Lüssi A, Özcan M, Jung RE, Hämmerle CH, Thoma DS. (2016). Surface roughness of dental implants and treatment time using six different implantoplasty procedures. *Clin Oral Implants Res*, 27(7):776-781.
49. Renvert S, Persson GR, Pirih FQ, Camargo PM. (2018). Peri-implant health, peri-implant mucositis, and peri-implantitis: case definitions and diagnostic considerations. *J Periodontol*. 2018;89(1): S304-S312.
50. Renvert S, Polyzois I. (2018). Treatment of pathologic peri-implant pockets. *Periodontol* 2000. 76(1):180-190.
51. Renvert S, Quirynen M. (2015). Risk indicators for peri-implantitis. A narrative review. *Clin Oral Implants Res*, 26(11):15-44.

52. Renvert, S.; Roos-Jansåker, A.M.; Persson, G.R. (2018). Surgical treatment of peri-implantitis lesions with or without the use of a bone substitute-a randomized clinical trial. *J. Clin. Periodontol*, 45:1266-1274.
53. Roehling S, Astasov-Frauenhoffer M, Hauser-Gerspach I, Braissant O, Woelfler H, Waltimo T, et al. (2017). In vitro biofilm formation on titanium and zirconia implant surfaces. *J Periodontol*. 2017;88(3):298-307.
54. Roehling S, Gahlert M, Janner S, Meng B, Woelfler H, Cochran DL. (2019). Ligature-induced peri-implant bone loss around loaded zirconia and titanium implants. *Int J Oral Maxillofac Implants*, 34(2):357-365.
55. Rokn A, Aslroosta H, Akbari S, Najafi H, Zayeri F, Hashemi K. (2017). Prevalence of peri-implantitis in patients not participating in well-designed supportive periodontal treatments: a cross-sectional study. *Clin Oral Implants Res*, 28(3):314-319.
56. Saaby M, Karring E, Schou S, Isidor F. (2016). Factors influencing severity of peri-implantitis. *Clin Oral Implants Res*. 2016;27(1):7-12.
57. Schwarz F, Derks J, Monje A, Wang HL. (2018). Peri-implantitis. *J Periodontol*, 89(1):S267-S90.
58. Serino G, Turri A, Lang NP. (2013). Probing at implants with periimplantitis and its relation to clinical peri-implant bone loss. *Clin Oral Implants Res*, 24:91-95.
59. Sutton JD, Salas Martinez ML, Gerkovich MM. (2009). Environmental tobacco smoke and periodontitis in United States non-smokers, 2009 to 2012. *J Periodontol*. 2017;88(6):565-574.
60. Swierkot K, Lottholz P, Flores-de-Jacoby L, Mengel R. (2012). Mucositis, peri- implantitis, implant success, and survival of implants in patients with treated generalized aggressive periodontitis: 3- to 16- year results of a prospective long-term cohort study. *J Periodontol*. 2012;83(10):1213-1222.
61. Ting M, Craig J, Balkin BE, Suzuki JB. (2018). Peri-implantitis: a comprehensive overview of systematic reviews. *J Oral Implantol*, 44(3):225-247.
62. Toy VE, Uslu MO. (2020). Evaluation of long-term dental implant success and marginal bone loss in postmenopausal women. *Niger J Clin Pract*, 23:147-153.
63. Turri A, Rossetti PH, Canullo L, Grusovin MG, Dahlin C. Prevalence of peri-implantitis in medically compromised patients and smokers: a systematic review. *Int J Oral Maxillofac Implants*. 2016;31(1):111-118.
64. Vohra F, Alkudhairy F, Al-Kheraif AA, Akram Z, Javed F. (2018). Periimplant parameters and C-reactive protein levels among patients with different obesity levels. *Clin Implant Dent Relat Res*, 20(2):130-136.

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