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Review Article

Prevalence and risk indicators for peri-implant diseases: A literature review

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ABSTRACT

Peri-implant diseases are known as undesirable conditions that can occur after implant therapy. Although several risk indicators are becoming clear, the causes of peri-implant diseases have not been completely investigated. The purpose of this review was to summarize the prevalence and risk indicators for peri-implant diseases by referring to current papers from various angles. Many studies have reported the varied prevalence of peri-implant mucositis (23.9%–88.0% at the patient level and 9.7%–81.0% at the implant level) and peri-implantitis (8.9%–45% at the patient level and 4.8%–23.0% at the implant level). Additionally, several studies concluded that poor oral hygiene and lack of regular maintenance were strongly correlated with the development of both peri-implant mucositis and peri-implantitis. Diabetes and a history of periodontitis were revealed as risk indicators for peri-implantitis. However, there was no definitive conclusion about the correlations between peri-implant diseases and other factors such as smoking, the shape of the implant superstructure, and the condition of the keratinized mucosa. Further studies useful for evidence-based decision-making are needed for predictable implant therapy in the long term.

1. Introduction

Dental implants are now an indispensable part of prosthetic treatment, but they also pose the risk of peri-implant mucositis and peri-implantitis, together known as peri-implant diseases. Although peri-implant diseases are thought to be caused mainly by plaque accumulation on the implant/abutment surface, various other risk factors have been reported. This paper aims to review and discuss the risk factors for peri-implant diseases.

2. Diagnosis

2.1. Peri-implant mucositis

Peri-implant mucositis is a reversible inflammatory condition confined to the soft tissue around the implant. It is characterized by redness, swelling and bleeding on probing (BoP) without bone loss around the implant.

2.2. Peri-implantitis

Peri-implantitis is characterized by inflammation in the peri-implant tissue and progressive bone loss. Radiological bone loss, increased probing depth and bleeding and/or suppuration on probing around the implant are used for diagnosis. However, the cut-off value of these parameters greatly differs in each study. In their systematic review, Renvert et al. found that cut-off values for bone loss in peri-implantitis ranged from 0.5 mm to 5.0 mm [1]. These differences suggest that the actual condition of peri-implantitis may not be correctly evaluated in all studies. Moreover, it is difficult to evaluate whether the bone resorption is progressing based on an X-ray evaluation at one time point. Therefore, a recent consensus report recommended that the presence of bleeding and/or suppuration on gentle probing, and a change in bone loss and probing depth in a longitudinal examination, are required for the diagnosis of peri-implantitis.

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3. Peri-implant mucositis

3.1. Prevalence

Many studies have reported the prevalence of peri-implant mucositis, and its range is relatively wide (Table 1). Wada et al. reported that the prevalence of peri-implant mucositis at the subject level was 23.9% in the Japanese population [2]. Ogata et al. also analyzed peri-implant mucositis among the Japanese population, and reported that its prevalence was 33.3% at the subject level [3]. These rates are similar to some previous studies [4,5], but other studies reported a relatively high prevalence [6,7], including Ferreira et al., who reported a prevalence of almost 70% [8]. As mentioned above, peri-implant mucositis is diagnosed only by the presence of BoP, which is affected by oral hygiene. Therefore, the prevalence generally depends on the study population. In fact, the prevalence tends to be low when the study population has attended the dentist for regular maintenance or has been treated by periodontists. Regardless of the recorded prevalence, peri-implant mucositis has already become a common disease.

3.2. Etiology

Dental plaque, comprising 700–1000 bacteria, causes periodontal disease [9]. The causality has been clearly shown in human studies. Löe et al. asked participants to stop brushing for 3 weeks and then to resume brushing [10]. They found gingivitis in all subjects after 3 weeks of plaque accumulation, and the gingivitis disappeared almost completely after toothbrushing had resumed for several days. It has been shown that the soft tissue around implants can be similarly inflamed as a result of the accumulation of plaque [11]. Additionally, the inflamed soft tissue can generally be restored to health by appropriate self-performed toothbrushing and professional care. Thus, a cause–effect relationship has been shown in humans [12]. The composition and size of inflammatory cells in peri-implant mucositis are similar to those found in gingivitis [11].

3.3. Risk indicators

Many studies have concluded that poor oral hygiene is strongly correlated with peri-implant mucositis [2,13–16]. It is clear that poor oral hygiene is a risk indicator for peri-implant mucositis development, considering that the pathology and progress of peri-implant mucositis is similar to gingivitis. In other words, plaque accumulation around the peri-implant tissue causes peri-implant mucositis. Another factor, directly or indirectly in conjunction with plaque accumulation, is keratinized mucosa. Lin et al. concluded in their systematic review that the presence of keratinized mucosa might be crucial for decreasing the plaque score, the modified gingival index score, mucosal recession, and the loss of clinical attachment [17]. However, they also noted that keratinized mucosa did not affect BoP or probing pocket depth. Therefore, additional prospective and randomized studies are required to determine whether keratinized mucosa is necessary for preventing peri-implant mucositis.

Heitz-Mayfield and Salvi identified in their review that smoking and radiation are risk indicators for peri-implant mucositis in addition to plaque accumulation [18]. Renvert and Polyzois also concluded that smoking is a risk indicator in their literature review [19]. They also reported in their review of 15 studies that the correlations between peri-implant mucositis and surface roughness, residual cement, and duration of function of the implant were weak. Other factors, such as systemic diseases including diabetes, periodontal status, location, and prosthetic design have also been proposed as risk indicators in some studies, but many of these studies were not based on multilevel analysis including all the potential factors [5,16,20–22].

3.4. Management/treatment

Because the inflammatory lesion of peri-implant mucositis is limited to the peri-implant mucosa, non-surgical therapy by mechanical or ultrasonic debridement was found to be the most effective management [23]. Other treatment therapies such as air-abrasives, lasers, and combinations of these with antibiotics or oral rinses, were also proposed, and soft tissue clinical parameters improved with these therapies in some controlled clinical studies [24]. However, a literature review by Renvert et al. reported that the additional use of antimicrobials was less beneficial in combination with mechanical debridement, while antiseptic oral rinse in combination with mechanical debridement improved the treatment outcome [25,26]. Jepsen et al. also concluded in their systematic review that antiseptics, local and systemic antibiotics, and air abrasives did not improve the efficacy of professional plaque removal in reducing clinical signs of inflammation [27]. Some studies reported that when used as an adjunct to non-surgical treatment, laser therapies might reduce BoP, but were not efficacious in periodontal pocket reduction, clinical attachment level gain, or plaque index reduction [28].

Because peri-implant mucositis is a reversible disease caused by the accumulation of plaque, it is important to maintain effective oral hygiene practices in each patient. Therefore, continuous regular maintenance recall is crucial, and if needed, the shape of the superstructure should be modified for easy maintenance.

4. Peri-implantitis

4.1. Prevalence

Table 2 shows the varied prevalence of peri-implantitis (4.7%–45% at the patient level and 3.6%–22.1% at the implant level) [2,20,29–34]. Unlike mucositis, each report embraces different diagnostic criteria. Wada et al. reported that the prevalence of peri-implantitis (BoP/suppuration and bone loss > 1 mm) at the subject level and the implant level was 15.8% and 9.2% respectively in implants with at least 3 years of function [2]. In a 9-year observational study, Derks et al. observed 45.0% of patients with peri-implantitis (BoP/suppuration and bone loss > 0.5 mm), including 14.5% with moderate/severe peri-implantitis (BoP/suppuration and bone loss > 2 mm) [29]. They also reported that the prevalence of peri-implantitis and moderate/severe peri-implantitis at the implant level was 22.1% and 7.1%, respectively. Renvert et al. reported that the prevalence of peri-implantitis (BoP/suppuration and bone loss > 3 threads) at the implant level was 4.8% at 9–14 years follow-up and 10.8% at 20–26 years follow-up [34]. Although a correct estimation of the prevalence of peri-implantitis requires evaluation with unified criteria, the reports summarized here indicate that peri-implantitis is not a rare condition.

4.2. Etiology

It is most likely that peri-implantitis occurs following peri-implant mucositis, which is caused by plaque accumulation around the implant [35]. The inflammatory reaction of peri-implantitis has been observed to be more widespread than that of periodontitis in animal and human studies [36,37]. Additionally, the composition of the inflammatory cells in peri-implantitis differs from that in periodontitis. In contrast with the chronic inflammatory features of periodontitis, peri-implantitis generally displays a more acute inflammatory status. However, some researchers have questioned whether dental plaque is the only etiologic factor in peri-implantitis

Table 1
Studies characteristics for prevalence of peri-implant mucositis.

Study	Study design	Number of patients/implants	Prevalence of peri-implant mucositis	Diagnostic criteria
Wada et al. [2]	Cross-sectional mean: 5.8 years	543/1613	PL: 23.9% IL: 27.4%	BoP with no bone loss (≤1 mm)
Ogata et al. [3]	Cross-sectional mean: 6.4 years	267/267	PL: 33.3% IL: 9.7%	BoP (<0.25 N) without changes in the level of the crestal bone
Marrone et al. [5]	Cross-sectional mean: 8.5 years	103/266	PL: 31% IL: 38%	PD > 5 mm, BoP, and bone loss ≤2 mm
Casado et al. [4]	Cross-sectional range: 1–5 years	103	PL: 19.4%	BoP, red mucosa, and swelling with no bone loss (no described criteria)
Máximo et al. [6]	Cross-sectional mean: 3.4 years	113/347	PL: 36.3% IL: 32.0%	BoP and bone loss <3 threads
Ferreira et al. [8]	Cross-sectional mean: 3.5 years	212/578	PL: 64.6% IL: 62.6%	BoP with no bone loss (no described criteria)

PL; patient level, IL; implant level.

Table 2
Studies characteristics for prevalence of peri-implantitis.

Study	Study design	Number of patients/implants	Prevalence of peri-implantitis	Diagnostic criteria
Renvert et al. [1]	Cohort 20–26 years	86/351	PL: 15.1% at 9–14 years follow-up IL: 4.8% at 9–14 years follow-up 10.8% at 20–26 years follow-up	≥3 threads exposed, and BoP/Sup
Wada et al. [2]	Cross-sectional mean: 5.8 years	543/1613	PL: 15.8% IL: 9.2%	BoP/Sup and bone loss >1 mm after 1 year of function
Derks et al. [29]	Cross-sectional mean: 8.9 years	427/1578	PL: 45.0 % (moderate/severe: 14.5%) IL: 24.9% (moderate/severe: 8.0%)	BoP/Sup and bone loss >0.5 mm (moderate/severe: BoP/Sup and bone loss >2 mm)
Aguirre-Zorzano et al. [20]	Cross-sectional mean: 5.3 years	239/786	PL: 15.1% IL: 9.8%	BoP and bone loss>1.5 mm
Dalago et al. [30]	Cross-sectional mean: 5.6 years	183/938	PL: 16.4% IL: 7.3%	PD > 5 mm, BoP/Sup, and bone loss>2 mm
Rokn et al. [31]	Cross-sectional mean: 4.4 years	134/478	PL: 20.1% IL: 8.8%	BoP/Sup and bone loss>2 mm
Schwarz et al. [32]	Cross-sectional mean: 2.2 years	238/512	PL: 13.9% IL: 7.6%	BoP/Sup and bone loss compared to baseline
French et al. [33]	Cohort 5–10 years	2060/4591	PL: 11.7% (strict), 7.8% (relaxed) IL: 7.7% (strict), 5.9% (relaxed)	strict: single-point BoP and bone loss≥1.0 mm at least 1 year after installation relaxed: multi-point BoP and bone loss≥1.0 mm at least 1 year after installation

PL; patient level, IL; implant level.

[38]. They claim that the foreign body reaction to the implant is sustained in the body and is affected by several factors such as implant hardware, patient characteristics, and surgical and/or prosthetic errors that may cause significant marginal bone resorption. As a biofilm-related complication, peri-implantitis may occur following pathological marginal bone resorption.

4.3. Risk indicators

Several systematic reviews have identified that diabetes, poor oral hygiene, a history of periodontitis and supportive peri-implant therapies are strongly associated with the development of peri-implantitis. Other studies have reported potential risk indicators with limited evidence, including smoking, lack of keratinized tissue, and cement residue.

4.3.1. Systemic factors

4.3.1.1. Smoking. Cigarette smoking is thought to reduce resistance to inflammation and immune reactions by inhibiting blood flow in the soft and hard tissue around the implant, making bone resorption more likely to progress. Various reports have shown the adverse effects of smoking on peri-implant tissue. Clementini et al. reported a higher level of peri-implant bone loss in smokers compared with non-smokers in their systematic review [39]. Furthermore, Casado et al. reported that smoking increased the likelihood of peri-implantitis occurring [40]. This tendency was also observed in relation to water smoking [41]. However, many reports showed that there was no clear association between smoking and peri-implant inflammation [20,30,31,42,43]. It has also been reported that smoking may be a modifying factor for other risk indicators [44,45]. Thus, there is currently limited evidence for an association between smoking and peri-implantitis.

4.3.1.2. Diabetes. Diabetes mellitus is a representative lifestyle disease, and is also known as a risk factor for periodontitis. A recent systematic review reported that hyperglycemia has been shown to increase the risk of peri-implantitis independently of smoking [46]. Similarly, Naujokat et al. reported that patients with diabetes have similar peri-implant health as long as good glycemic control is achieved [47]. Additionally, they also mentioned the importance of controlling the HbA1c level in diabetic patients to avoid serious peri-implant infection.

4.3.1.3. Osteoporosis. Osteoporosis, a metabolic bone disorder, is also considered to be a possible risk factor for peri-implant bone resorption because it degrades bone quality. However, although slightly higher levels of bone resorption have been reported in patients with osteoporosis, there is no clear evidence of peri-implantitis or implant loss [48,49].

4.3.2. Oral-related factors

4.3.2.1. Plaque control skills. It is widely known that inflammation caused by plaque is a major cause of peri-implantitis [35]. There is no doubt that the patient's self-cleaning skills are indispensable for maintaining health around the implant. However, other factors also affect the local cleaning status, such as the shape and position of

the implant superstructure and the presence of keratinized mucosa around the implants. Clinicians need to understand these factors and construct a good environment for easy cleaning [50]. Additionally, supportive peri-implant therapies are crucial for maintaining good oral hygiene and patient motivation [51].

4.3.2.2. History of periodontitis. Many studies have shown an association between peri-implantitis and periodontitis. Chrcanovic et al. reported that periodontally compromised patients have greater susceptibility to peri-implantitis than periodontally healthy patients [52]. Ferreira et al. also clarified the evidence that peri-implantitis was associated with individuals with a history or presence of periodontitis [53]. Additionally, Pandolfi et al. reported that patients with a history of periodontal disease had an increased incidence of peri-implantitis after 5 years of function [54]. Periodontal disease is one of the most noteworthy factors for predicting the development of peri-implantitis.

4.3.2.3. Keratinized mucosa. A consensus report from the 2017 world workshop on the classification of periodontal and peri-implant diseases and conditions indicated that there was limited evidence of the need for keratinized mucosa to maintain peri-implant health [35]. However, many recent reports stress the importance of keratinized mucosa for peri-implant health. Perussolo et al. reported that implant sites with less than 2 mm width of keratinized mucosa tended to experience more bone loss, brushing discomfort, plaque accumulation and inflammation of peri-implant soft tissue than those sites with enough keratinized mucosa [55]. Mameno et al. reported that the presence of enough keratinized mucosa was necessary to prevent marginal bone resorption [56], while Suárez-López Del Amo et al. reported that implants with thick keratinized mucosa have less bone loss [57]. A systematic review also showed that soft tissue preparation was beneficial for maintaining the marginal bone around the implant [58].

4.3.2.4. Occlusal overload. Few clinical papers have shown a correlation between overload and peri-implant inflammation, and no direct link has been found in animal studies [59]. However, excessive occlusal force may cause implant fracture [60], resulting in rapid bone resorption around the implant. Additionally, repeated lateral occlusal force on the implant superstructure may cause loosening of the abutment screw [61], resulting in a sub-marginal gap. Therefore, it could be considered an indirect factor.

4.3.3. Implant-related factors

4.3.3.1. Implant surface. The surface properties of implants have long been a subject for discussion. Implants with a rough surface are known to improve early osseointegration and are currently available from various implant brands. De Bruyn et al. concluded that minimally rough (S_a values of 0.5–1.0 μm) and moderately rough (S_a values of 1.0–2.0 μm) surfaced titanium implants result in less bone loss around the implant than rough (S_a values of more than 2.0 μm) surfaced implants in their review which evaluated long-term studies [62]. However, there is a concern that plaque accumulation on the rough surface may cause more peri-implant inflammation than implants with a turned surface. Meanwhile, Saulacic and Schaller reported that implants with rough surfaces have similar clinical parameters and prevalence of peri-implantitis to turned implants [63]. Further studies are needed to clarify this issue.

4.3.3.2. Position and design of the implant-abutment junction. Tissue level implants and platform switching are used based on the concept of a fixed biological width around the implant. The vertical and horizontal offsets between the implant–abutment junction and the bone surface are considered to minimize marginal bone resorption by separating microgaps where bacteria adhere from the bone–implant interface. However, there is no clear evidence so far that they significantly affect the development of peri-implantitis. Vouros et al. found no significant difference in short-term bone loss between bone-level and tissue-level implants in their systematic review [64]. Meloni et al. reported that there was no significant difference between the marginal bone loss of platform switching implants and platform matching implants in their 5-year randomized controlled trial [65]. Lemos et al. also indicated that the type of implant–abutment connection did not affect the prevalence of peri-implantitis or the survival rate, although internal connections had less marginal bone loss than external connections [66]. Further studies should consider the effect on peri-implantitis of the position and design of the implant–abutment junction.

4.3.3.3. Surgical procedure. Gheisari et al. reported that there was no significant difference in marginal bone loss between one- and two-stage surgical techniques, although the implants inserted with a one-stage surgical technique produced better esthetic and functional results than those with a two-stage technique [67]. Jung et al. reported that guided bone regeneration procedures were predictable technique in their study investigated the stability of the marginal bone around implants in a long-term evaluation [68]. Hopp et al. reported that tilted implants in the maxilla had similar bone loss as axial implants after 5 years of follow-up [69]. As described above, no studies mentioned the surgical procedure as a risk factor for peri-implantitis, although various factors about the surgical method were discussed. However, subcrestal placement in platform-switching implants has been reported to reduce marginal bone resorption [70].

4.3.3.4. Fixation type. There is currently no consensus on whether screw- or cement-retained implants are more beneficial for preventing peri-implantitis. One systematic review showed that a higher incidence rate of biological complications including peri-implantitis was seen with cement-retained prostheses [71]. In contrast, Lemos et al. reported that screw-retained prostheses demonstrated slightly more marginal bone loss in their meta-analysis [72]. There is evidence that excess cement exists frequently in cement-retained superstructures [73], and has been implicated as a possible risk indicator for peri-implantitis by Staubli et al. [22]. Therefore, it is necessary to pay close attention to excess cement when using cement-retained prostheses.

4.4. Management/treatment

Many protocols for peri-implantitis treatment have been proposed, including non-surgical or surgical approaches combined with antibiotics or various devices and materials.

4.4.1. Non-surgical approach

The aim of the non-surgical approach is control of the infection in the peri-implant soft tissue and debridement of the implant surface. For this purpose, several protocols have been reported involving mechanical debridement with curettes, ultrasonic devices, air-abrasives, and lasers including photodynamic therapy, and adjunctive use of antibiotics or antiseptics. Figuero et al. reviewed these protocols and concluded that many of these therapies had measurable efficacy on BoP in the short term, but a limited effect on probing depth [24]. Therefore, they recommended that surgical interventions are needed if non-surgical therapies are not able to improve the clinical parameters. Renvert and Polyzois also concluded that the effect of non-surgical therapy is limited, especially in advanced cases [74]. Therefore, it seems to be difficult to manage peri-implantitis using a non-surgical approach. How-

ever, there are some merits, such as the effect of reducing BoP, so non-surgical therapy may be helpful before surgical intervention.

4.4.2. Surgical approach by resective therapy

A surgical approach is considered to be effective in terms of the debridement and decontamination of the implant surface which has geometrical threads and various surface modifications. Berglund et al. reported in their retrospective study that the removal of inflamed tissue and cleaning of the implant surface with gauze soaked in saline during a surgical intervention, followed by oral hygiene instruction and professional supra-mucosal instrumentation, are effective in the long term [75]. Various methods for the decontamination of the implant surface besides gauze soaked with saline have been proposed, including hand curettes, ultrasonic devices, rotary devices, lasers and chemical decontamination [76–79]. Many of these methods have resulted in significant or limited improvement in clinical parameters compared to access flap surgery alone, but few studies have followed subjects over the long term. Implants with severe bone resorption (over 7 mm) seem to be less responsive to surgical therapy [80]. In addition to decontamination while maintaining the geometrical implant threads, a method of mechanical removal of the threads (implant-plasty) has recently been proposed, and demonstrated a significant improvement in probing depth and clinical attachment levels compared to apically positioned flap surgery alone [81]. However, at present, it is difficult to conclude which methods are superior in the long term. Additionally, these resective therapies are only of merit in non-esthetic areas, because the soft tissue will recede after the surgical intervention.

4.4.3. Surgical approach with regenerative therapy

Several studies proposed regenerative surgical techniques for bone loss/defects of peri-implantitis with the expectation of avoiding soft tissue recession. Generally, these techniques used grafting materials including autogenous bone with or without barrier membranes [82–84]. Chan et al. reported in their systematic review and meta-analysis that the application of grafting materials and barrier membranes results in greater pocket depth reduction and radiographic bone fill [85]. However, these techniques also involve some risks, such as membrane exposure and infection of the grafting materials as a result of biofilm remnants on the implant surface. Roccuzzo et al. reported that the survival rate for regenerative therapy of sandblasted and acid-etched implants (83.3%) was higher than that for titanium plasma-sprayed implants (58.3%), and concluded that the decision about whether implants affected with peri-implantitis should be treated or removed should be based on the implant surface characteristics [86]. The morphology of the bony defect around an infected implant also affects the success of regenerative therapy. Generally, regenerative therapy is more successful in cases with circumferential or intrabony defects [87].

5. Conclusion

A varied prevalence of peri-implant diseases has been reported from many studies, which together confirm that peri-implant diseases commonly occur. This review revealed that good plaque control skills and continual regular maintenance recalls are crucial factors for preventing both peri-implant mucositis and peri-implantitis. Additionally, diabetes and a history of periodontitis are strongly associated with the development of peri-implantitis. Although various studies have been conducted on other factors such as smoking, the shape of the implant superstructure, and the condition of the keratinized mucosa, definitive conclusions have not yet been reached. Further studies useful for evidence-based decision-making are needed for predictable implant therapy in the long term.

Conflict of interest

The authors declare that they have no competing interests.

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