



■ PERIODONTOLOGY

Nonsurgical and surgical management of biologic complications around dental implants: peri-implant mucositis and peri-implantitis

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Biologic complications around dental implants may be categorized into peri-implant mucositis and peri-implantitis. Peri-implant mucositis is defined as reversible inflammation in the peri-implant mucosa without any apparent bone destruction. Peri-implantitis refers to inflammatory process that resulted in destruction of alveolar bone and attachment. Potential etiologic and contributing factors to both diseases are discussed in this review. By targeting and eliminating the etiologic factors nonsurgically as well as surgically, dental implants presenting with peri-implant diseases may be rescued, and then maintained with proper long-term peri-implant support-

ive therapy. Furthermore, clinical cases and their management are presented to demonstrate the available treatment options. Implant therapy should be carefully planned and executed with consideration of potential etiologic and contributing factors to developing biologic complications. During the initial consideration, patients should be informed of the potential biologic complications in dental implant therapy. Clinicians should monitor implants for any development or recurrence of peri-implant disease to ensure timely therapeutic intervention. (*Quintessence Int* 2020;51:810–820; doi: 10.3290/j.qi.a44813)

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Peri-implant health is characterized by absence of clinical signs of erythema, edema, suppuration, and bleeding on probing.¹ Biologic complications around dental implants may be categorized into two different disease identities. Peri-implant mucositis refers to marginal inflammation in peri-implant mucosa with no apparent bone destruction.² Similar to gingivitis, peri-implant mucositis is reversible with appropriate treatment. On the other hand, peri-implantitis refers to inflammatory process that is associated with loss of marginal supporting bone and attachment around a dental implant.² Sites with peri-implantitis exhibit active bleeding and/or suppuration upon light probing, increased recession and/or deep probing depths, in conjunction with progressive radiographic bone loss compared to previous examinations.³ It is important to record the baseline radiographic marginal bone level and probing depth values in order to identify any progression of the disease. Variable factors could alter the degree of early marginal bone

level remodeling, such as the depth of the implant insertion, mucosal thickness, and abutment height.⁴ Attention must be paid to the clinical signs of inflammation and changes in radiographic bone levels to properly and timely identify peri-implantitis. The loss of the supporting bone and attachment is irreversible. In the absence of treatment, peri-implantitis progresses in a “nonlinear and accelerating pattern,” more rapid than the progression of periodontitis.³ In the absence of previous examinations for comparison, the diagnosis of peri-implantitis can be based on active bleeding and/or suppuration upon gentle probing, probing depths ≥ 6 mm, and bone loss beyond 3 mm from the most coronal intraosseous aspect of the implant.³ According to a previous meta-analysis of a total of 1,497 subjects with 6,283 dental implants, 30.7% of dental implants comprising 63.4% of subjects presented with peri-implant mucositis.⁵ Similarly, 9.6% of dental implants representing 18.8% of subjects presented with peri-implantitis.⁵

The treatment of peri-implantitis, as described in the literature, ranges from nonsurgical to surgical resective and regenerative methods with no specific “gold standard” method proven to be superior to the others.⁶ However, the treatment of peri-implantitis does not stop after the initial treatment. Peri-implant maintenance therapy (PIMT) and adequate plaque control have been well established as crucial for long-term success.⁷⁻⁹ In a recent systematic review on the success of peri-implantitis therapy followed by PIMT, implant survival rates of 81.73% to 100% at 3 years (seven studies) and 69.63% to 98.72% at 7 years (two studies) were reported.⁶

The aim of this review was to discuss potential etiologic and contributing factors to biologic complications around dental implants, ie peri-implant mucositis and peri-implantitis. Furthermore, clinical cases and their management are presented to demonstrate the available treatment options.

Etiology and risk indicators

Dental plaque

According to Ferreira et al,¹⁰ patients with poor oral hygiene exhibited approximately 15 times greater chance of developing peri-implantitis. Considering that initiation of peri-implant diseases may be triggered by the presence of dental plaque similar to that of periodontal diseases,¹¹ achieving and maintaining excellent plaque control is an important factor in prevention as well as treatment of peri-implant diseases. Based on the principles of cause-related therapy, clinicians should educate their patients on how to effectively remove the main etiologic factor, dental plaque around teeth and implants.^{12,13} Furthermore, both surgical and restorative phases of implant therapy should be carefully planned and delivered to allow easy access for patient’s home oral hygiene care. Serino and Ström¹⁴ evaluated a total of 109 implants in 23 patients and found 81 implants had no accessibility or capability for proper oral hygiene. Among these 81 implants, 53 implants had peri-implantitis. In comparison, among the 28 implants that were accessible for proper oral hygiene care, only five implants had peri-implantitis, confirming the importance of delivering a hygienic implant restoration to achieve and maintain optimal peri-implant health.¹⁴

Past and active periodontitis

According to Lee et al,¹⁵ implants in patients with residual periodontal probing depths of 6 mm or more were at approximately

5.5 times greater chance of developing peri-implantitis than implants in patients without any periodontal probing depths of 6 mm or more. In addition, the transmission of perio-pathogenic bacteria, including the so-called red complex bacteria, from the gingival crevice of the adjacent teeth with deep periodontal probing depth to the implant sulcus was reported.^{16,17}

Patients with history of periodontitis, who most likely carried these periodontal pathogens or were genetically more susceptible, exhibited up to 14 times greater risk of developing peri-implantitis.¹⁸⁻²⁰ Thus, clinicians should ensure treating and stabilizing any active periodontal disease prior to proceeding with implant therapy. Furthermore, patients with history of periodontitis should be clearly informed regarding the potential increased risk of implant failure or biologic complications around it.

Irregular peri-implant maintenance therapy

Irregular PIMT after implant placement has been proven to be a major risk indicator of developing peri-implant diseases.³ Costa et al²¹ investigated the incidence of peri-implantitis over a 5-year follow-up period in 80 subjects with peri-implant mucositis. In subjects who received PIMT, the incidence of peri-implantitis was significantly lower than that of subjects who did not receive PIMT.²¹ Similarly, in a cross-sectional study evaluating a total of 206 implants in 115 subjects, the prevalence of peri-implantitis was 4.5% in a subject group who received regular PIMT at least two times annually.²² In comparison, the prevalence of peri-implantitis was 23.9% among subjects who were either erratic compliers (PIMT less than two times a year) or noncompliers (no PIMT).²² Furthermore, the positive effect of PIMT on the long-term survival of implants was reported in patients who successfully received surgical peri-implantitis therapy.²³

Thus, clinicians should educate their patients, prior to implant placement, regarding the importance of establishing regular PIMT as well as meticulous home care habits in order to reduce the incidence or the recurrence of peri-implant disease. During PIMT, if indicated, a periapical radiograph may be prescribed to determine marginal bone level. In addition to professional mechanical debridement, home care therapy should be carefully reviewed with the patient in each PIMT appointment.^{22,23}

Smoking

Similar to periodontal diseases, dental implant patients with active smoking habits exhibited up to three to five times greater chance of developing peri-implantitis.^{19,24,25} Further-



more, according to Levin et al,¹⁹ during the approximately first 5 years, smokers exhibited a similar implant survival rate compared to nonsmokers. However, after 5 years, the cumulative implant survival rate was significantly lower in smokers than that of nonsmokers.¹⁹ Thus, the importance of smoking cessation on prevention and treatment of peri-implant diseases should be discussed with and reinforced to patients.

Lack of keratinized peri-implant mucosa

According to Schrott et al,²⁶ implants with less than 2 mm of keratinized mucosa exhibited significantly higher Bleeding Index, Plaque Index, and greater buccal peri-implant mucosal recession. Similarly, Monje and Blasi,²⁷ in their evaluation of 66 dental implants in a total of 37 patients, found that the presence of less than 2 mm of keratinized mucosa was associated with a significantly higher odds of developing peri-implant diseases, especially in patients with erratic PIMT patterns. Thus, similar to natural teeth,²⁸ the presence of at least 2 mm of keratinized mucosa may be preferable to prevent peri-implant diseases. Furthermore, the presence of an adequate width of keratinized mucosa around dental implants may lead to better soft and hard tissue stability, less plaque accumulation, limited soft tissue recession, and lower incidence of peri-implant diseases.²⁹ Therefore, clinicians should carefully examine the amount of keratinized mucosa around dental implants. This should also be part of the pre-implantation assessment and treatment considerations, especially concerning flap design. If deficient, mucogingival therapy around dental implants might be considered.

Presence of foreign body

The presence of a foreign body around dental implants was found to be associated with local inflammatory lesions, eliciting potential peri-implant diseases.³⁰ These foreign bodies include predominantly excessive cement materials,³⁰⁻³⁵ retained retraction cord,³⁶ and titanium debris.³⁰ In a retrospective analysis of 129 dental implants, 62 of 73 implants with cement remnants developed peri-implant disease.³³ In comparison, in the group of implants without cement remnants, only 17 of 56 implants were diagnosed with peri-implant diseases, suggesting a potential role of excessive cement on the development of peri-implant disease.³³ In addition, in recent systematic reviews, a potential role of residual cement material in developing peri-implant disease and crestal bone loss was demonstrated.^{34,35} Thus, clinicians may consider delivering a screw-retained restoration or placing a shallow crown-abutment margin in a cement-retained restoration to

allow easier removal of excess cement material.³⁷ If an abutment level impression technique is used, clinicians should carefully examine and ensure the absence of any residual retraction cord or impression materials.³⁶ Lastly, during the surgical placement of a dental implant, copious irrigation should be used during osteotomy in order to minimize any residual titanium debris.³⁰

Thin buccal plate

The proceedings of the 2017 World Workshop added “hard and soft tissue implant site deficiencies” to the list of peri-implant diseases and conditions.¹ Significant ridge deficiencies can develop after extraction of teeth that had endodontic infections, periodontal defects, root fractures, maxillary sinus pneumatization, poor anatomical position within the alveolar ridge, etc.¹

Implants with thin buccal plate, dehiscence, or fenestration may be at a greater risk of developing peri-implantitis and ultimately failure. According to a recent animal study, a critical buccal bony wall thickness of at least 1.5 mm appeared to be essential for maintaining the buccal bony wall integrity during physiologic and pathologic resorption.³⁸ Following the extraction of a natural tooth, clinicians should carefully examine the remaining socket and determine the potential risk of vertical and horizontal residual ridge resorption.^{39,40} If necessary, clinicians should consider performing a socket preservation procedure to ensure an adequate amount of alveolar bone is available not only for the placement of the implant but for having an adequate volume of alveolus surrounding the implant.^{41,42}

During the placement of an implant, clinicians should carefully select the diameter of the dental implant, ensuring an adequate alveolar bone support around the implant. Furthermore, clinicians should avoid placing the implant too buccally, leaving a thin remaining buccal bone wall.⁴³ If necessary, a simultaneous buccal contour augmentation or a smaller diameter implant should be considered.⁴⁴⁻⁴⁶ Furthermore, existing implants should be carefully monitored for any signs of pronounced buccal plate resorption, resulting in thin buccal plate, dehiscence, or fenestration. Guided bone regeneration therapy may be performed in order to increase the thickness of the buccal plate or to repair dehiscence or fenestration around the existing implant when indicated.⁴⁷

Excessive occlusal force

The potential negative effect of excessive occlusal loading of a dental implant on the level of the surrounding marginal bone,



Fig 1a Maxillary right central incisor implant with peri-implant mucosal recession and limited keratinized tissue.

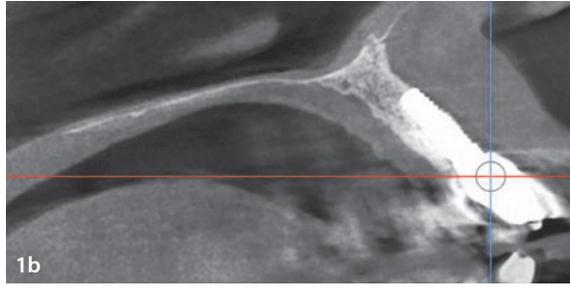


Fig 1b A CBCT scan revealed that the implant was positioned buccally, outside of the alveolar housing.



Fig 1c A periapical radiograph revealed satisfactory interproximal peri-implant bone level.



Figs 1d to 1f Gingival graft surgery (recipient site preparation, donor site preparation, and immediate postoperative view).



Fig 1g One-year follow-up.

the percentage of bone to implant contact, and the implant survival have been reported in animal studies.⁴⁸⁻⁵⁰ Furthermore, in the presence of plaque-induced peri-implant inflammation, excessive occlusal forces resulted in a greater horizontal and angular bone loss.⁴⁹

The effect of excessive occlusal force on a dental implant in humans is still unclear.^{51,52} There are two human case reports suggesting successful radiographic bone regain by reducing excessive occlusal contact alone⁵³ and with anti-infective therapy.⁵⁴ Within the limited literature, which is predominantly derived from the animal studies, clinicians should avoid excessive vertical and excursive occlusal forces on the implant.

Other factors

Patients with penicillin allergy were reported to be at three to four times greater risk for implant failure due to a higher incidence of postoperative infection compared to non-penicillin allergic patients.^{55,56} In addition, according to a recent systematic review with meta-analysis, diabetic patients exhibited approximately 50% greater chance of developing peri-implantitis than nondiabetic patients.⁵⁷ Furthermore, a restorative emergence angle of greater than 30 degrees and convex profile were associated with a greater risk for developing peri-implantitis in bone-level implants.⁵⁸



Fig 2a A mandibular left canine implant with peri-implant mucosal erythema and edema.

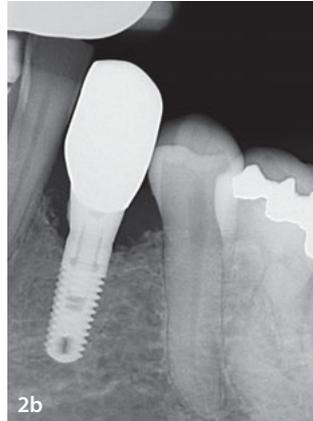


Fig 2b A periapical radiograph of the implant revealed a potential excessive cement mass on its mesial aspect.



Fig 2c Following debridement, a periapical radiograph confirmed a complete removal of the excessive cement mass.



Fig 2d A 6-week follow-up revealed significant resolution of peri-implant mucosal erythema and edema.

Case one

A 68-year-old man was referred by his general dental practitioner for an evaluation of his progressive buccal soft tissue recession around the maxillary right central incisor implant that presented also with a deep probing depth. His medical history was noncontributory and reported no known drug allergy. Clinical evaluation revealed a 3-mm peri-mucosal buccal recession on his maxillary right central incisor implant, partly exposing its abutment (Fig 1a). The peri-implant mucosa appeared thin and an 8-mm midbuccal peri-implant probing depth with bleeding was noted. The patient reported pain upon palpating the buccal aspect of the implant. He was also concerned esthetically due to progressive exposure of the metal portion of implant-abutment complex. A cone beam computed tomography (CBCT) evaluation revealed a complete bony dehiscence on the buccal aspect of the implant (Fig 1b). An adequate interproximal bone height was noted around the implant (Fig 1c). The buccal bony dehiscence appeared to be associated with the implant, which was positioned buccally outside of the alveolar housing. Since guided bone regeneration outside of the existing alveolar housing may be unpredictable, a soft tissue augmentation based on the principle of gingival unit graft was performed (Figs 1d to 1f).⁵⁹⁻⁶¹

The recipient site was prepared in a partial thickness manner. The gingival graft was harvested from the palatal aspect of

the maxillary premolar and stabilized over the recipient site. At the 1-year follow-up, a significant reduction in peri-implant mucosal recession was noted with a significant gain in the thickness of the mucosa (Fig 1g). The patient no longer experienced any pain upon buccal palpation. The peri-mucosal probing depth was reduced from 8 mm to 3 mm. The patient was recommended for every 4 months PIMT to continuously monitor the implant and determine his compliance with the suggested home care therapy on gingival line tooth brushing (ie, modified Bass technique)⁶² and flossing.

Case two

A 57-year-old man presented for a follow-up after receiving a final restoration on the mandibular left canine implant from his general dental practitioner. The clinical evaluation revealed peri-mucosal inflammation with erythema and edema, which was more pronounced on the mesial aspect (Fig 2a). A 6-mm probing depth was noted on its mesial aspect with bleeding on probing. A radiographic evaluation revealed a foreign body on the mesial aspect, which appeared to be residual excessive cement (Fig 2b). After achieving an adequate local anesthesia, a piezoelectric scaler (Piezon, Hu-Friedy) with chlorhexidine irrigation was used to remove the excessive cement. Immediately following the scaling, another periapical radiograph was obtained to confirm the complete cement removal (Fig 2c). At



Fig 3a Maxillary right lateral and left central incisor implants with deep probing depth.



Fig 3b A periapical radiograph revealed a moderate peri-implant bone loss.

Fig 3c Following scaling, the existing prosthesis was modified.



Fig 3d Passive insertion of an interdental brush was confirmed.



Fig 3e One-year follow-up revealed a significant resolution of peri-implant mucosal erythema and edema.

the 6-week follow-up, a significant resolution of the peri-implant inflammation was noted with a reduction in erythema and edema (Fig 2d). Home care therapy on gingival line tooth brushing,⁶² flossing, and interproximal rubber tipping was thoroughly reviewed with the patient. The patient was recommended a follow-up every 4 months to monitor the implant and determine his compliance with the suggested home care therapy.

Case three

An 80-year-old man was referred for an evaluation of the maxillary right lateral incisor and maxillary left central incisor implants due to evidence of bone loss. The clinical exam revealed deep probing depth of 8 to 10 mm surrounding the implants with bleeding and suppuration. The patient reported extreme difficulty of performing home care as the fixed prosthesis was in extremely close contact with the soft tissue ridge, leaving no space to insert and use a cleaning device such as

Super Floss, toothbrush, or interdental brush (Fig 3a). Radiographic examination revealed approximately 30% peri-implant bone loss around both implants (Fig 3b). After achieving an adequate local anesthesia, scaling was performed using a piezoelectric scaler (Piezon, Hu-Friedy) and Gracey curettes (Hu-Friedy; Fig 3c). Excessive vertical and lateral occlusal contact were adjusted. The “gingival” portion of the existing prosthesis was then removed and modified until an interdental brush (Oral B, Procter & Gamble) could be inserted passively (Fig 3d). The patient was then advised to perform adequate home care involving gingival line tooth brushing,⁶² interdental brushing, and interproximal rubber tipping (Sunstar). The patient received PIMT every 3 months with continuous monitoring of home care therapy. At the 1-year follow-up, a significant reduction of 4 to 6 mm in probing depth was noted. A complete resolution of peri-implant mucosal erythema and edema were also observed. No visible residual plaque was recorded, indicating his effective home care (Fig 3e).



Fig 4a Maxillary right lateral incisor implant with peri-implant mucosal edema.



Fig 4b A 12-mm probing depth was noted on the mid-buccal aspect.



Fig 4c Upon probing, suppuration and bleeding were noted.



Fig 4d A peri-apical radiograph revealed 50% to 70% alveolar bone loss around the implant.



Fig 4e Guided bone regeneration therapy was performed around the implant using Nd:YAG laser.



Fig 4f At the 5-month follow-up, an increase in alveolar height as well as density were noted, possibly indicating bone regeneration.



Fig 4g At the 5-month follow-up, a significant resolution of peri-implant mucosal edema was noted.

Case four

A 67-year-old man was referred for an evaluation of severe bone loss around the maxillary right lateral incisor implant. The clinical evaluation revealed a 9- to 12-mm probing depth around the implant with peri-implant mucosal swelling on its buccal aspect (Fig 4a). The buccal peri-implant mucosal tissue was tender upon palpation. Suppuration and bleeding were noted upon probing (Figs 4b and 4c). A radiographic examination revealed 50% to 70% alveolar bone loss around the implant, which was more pronounced on the mesial aspect. A partial two- to three-walled alveolar defect was suspected on both mesial and distal aspects (Fig 4d). Thus, peri-implant guided bone regeneration was attempted following the cause-related, nonsurgical treatment phase.^{12,13} After achieving adequate local anesthesia, Nd:YAG laser (Millennium Dental Technologies) was used to selectively remove the diseased inner sulcular epithelium while carefully avoiding the implant

surface.^{63,64} Piezoelectric scalers (Piezon, Hu-Friedy) with copious chlorhexidine irrigation were used to mechanically and chemically clean the contaminated implant surface. The osseous defect was carefully mapped and was instrumented with a fine piezoelectric scaler tip (PS tip, Hu-Friedy). Occlusion was adjusted in order to remove excessive centric and lateral excursive contacts (Fig 4e). At 5 months follow-up, a significant increase in bone density was noted on a periapical radiograph, possibly suggesting peri-implant bone regeneration (Fig 4f). Upon probing, a significant reduction in probing depth was noted to 4 to 5 mm with no evidence of suppuration or bleeding. The peri-implant mucosal swelling was resolved (Fig 4g). The patient was advised to perform adequate home care involving gingival line tooth brushing,⁶² interdental brushing, and interproximal rubber tipping, and to use chlorhexidine daily around the marginal peri-implant mucosa in order to maintain a favorable environment for continuous peri-implant tissue regeneration.



Fig 5a Maxillary left first premolar implant with moderate buccal horizontal ridge deficiency and peri-implant mucosal cyanosis.



Fig 5b Upon probing, suppuration and bleeding were noted.

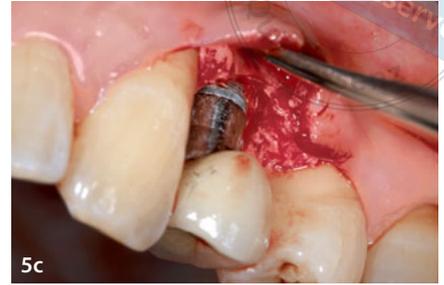


Fig 5c Upon elevating the flap, thin buccal plate with apical fenestration (not shown) and crestal dehiscence was noted.



Fig 5d Occlusal view showing the moderate buccal concavity.



Fig 5e The bony defect was repaired with FDDB and a cross-linked collagen membrane.



Fig 5f Immediate postoperative view.



Fig 5g At the 6-month follow-up, a complete resolution of the buccal ridge deficiency was noted.



Fig 5h Occlusal view confirming the resolution of the buccal ridge deficiency.



Fig 5i At the 6-month follow-up, a significant reduction in probing depth to 4 mm was noted. No suppuration or bleeding was noted.

Case five

A 55-year-old woman was referred for evaluation of bone loss around the maxillary left first premolar implant (Fig 5a). Her medical history was noncontributory and reported no known drug allergy. Clinical evaluation revealed 6- to 8-mm probing depth on the buccal aspect with suppuration and bleeding upon probing (Fig 5b). No radiographic bone loss was noted on the mesial and the distal aspect of the implant. Moderate horizontal ridge deficiency was noted on the buccal aspect,

which was more pronounced apically. Thus, peri-implantitis appeared to be associated with possible underlying hard tissue defect on the buccal aspect for which guided bone regeneration therapy was planned following the cause-related, non-surgical treatment phase. After achieving adequate local anesthesia, a buccal periosteal pocket flap was prepared as described by Steigmann et al⁶⁵ with an anticipation for horizontal guided bone regeneration therapy. After removing granulation tissue completely, extremely thin buccal plate with apical fenestration along the body of the implant in com-



bination with 2-mm crestal dehiscence was noted (Figs 5c and 5d). To repair the bony defect and increase the thickness of the buccal plate, freeze-dried bone allografts (FDBA; RegenerOss, Zimmer Biomet) were introduced into the buccal periosteal pocket and a cross-linked collagen membrane (BioMend Extend Membrane, Zimmer Biomet) was placed between the grafts and the inner aspect of the periosteal pocket (Fig 5e).⁶⁵ The flap was repositioned and sutured (Fig 5f). At 6 months follow-up, a complete resolution of buccal horizontal ridge deficiency was noted, suggesting possible thickening of the underlying buccal plate (Figs 5g and 5h). Upon probing, a significant reduction in probing depth was noted to 4 mm with no evidence of suppuration or bleeding (Fig 5i). In addition to regular PIMT every 4 months, the patient was advised to per-

form adequate home care involving gingival line tooth brushing, interdental brushing, and interproximal rubber tipping, and to use chlorhexidine daily around the marginal peri-implant mucosa. ■■

Conclusion

Implant therapy should be carefully planned and executed with consideration of potential etiologic and contributing factors to developing biologic complications. During the initial consultation, patients should be informed of the potential biologic complications in dental implant therapy. Clinicians should continuously monitor implants for any development or recurrence of peri-implant disease to ensure timely therapeutic intervention.

References

- Caton JG, Armitage G, Berglundh T, et al. A new classification scheme for periodontal and peri-implant diseases and conditions – Introduction and key changes from the 1999 classification. *J Clin Periodontol* 2018;45 (Suppl 20):S1–S8.
- Cortellini S, Favril C, De Nutte M, Teughels W, Quirynen M. Patient compliance as a risk factor for the outcome of implant treatment. *Periodontol* 2000 2019;81:209–225.
- 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–S318.
- Pico A, Martín-Lancharro P, Caneiro L, Nóvoa L, Batalla P, Blanco J. Influence of abutment height and implant depth position on interproximal peri-implant bone in sites with thin mucosa: A 1-year randomized clinical trial. *Clin Oral Implants Res* 2019;30: 595–602.
- Atieh MA, Alsabeeha NHM, Faggion CM, Duncan WJ. The frequency of peri-implant diseases: a systematic review and meta-analysis. *J Periodontol* 2013;84:1586–1598.
- Roccuzzo M, Layton DM, Roccuzzo A, Heitz-Mayfield LJ. Clinical outcomes of peri-implantitis treatment and supportive care: a systematic review. *Clin Oral Implants Res* 2018;29(Suppl 16):331–350.
- Hirschfeld L, Wasserman B. A long-term survey of tooth loss in 600 treated periodontal patients. *J Periodontol* 1978;49:225–237.
- Axelsson P, Lindhe J. The significance of maintenance care in the treatment of periodontal disease. *J Clin Periodontol* 1981;8: 281–294.
- Lindhe J, Nyman S. Long-term maintenance of patients treated for advanced periodontal disease. *J Clin Periodontol* 1984;11: 504–514.
- Ferreira SD, Silva GLM, Cortelli JR, Costa JE, Costa FO. Prevalence and risk variables for peri-implant disease in Brazilian subjects. *J Clin Periodontol* 2006;33:929–935.
- Heitz-Mayfield LJA, Lang NP. Comparative biology of chronic and aggressive periodontitis vs. peri-implantitis. *Periodontol* 2000 2010;53:167–181.
- Kwon T, Levin L. Cause-related therapy: a review and suggested guidelines. *Quintessence Int* 2014;45:585–591.
- Kwon T, Salem DM, Levin L. Nonsurgical periodontal therapy based on the principles of cause-related therapy: rationale and case series. *Quintessence Int* 2019;50:370–376.
- Serino G, Ström C. Peri-implantitis in partially edentulous patients: association with inadequate plaque control. *Clin Oral Implants Res* 2009;20:169–174.
- Cho-Yan Lee J, Mattheos N, Nixon KC, Ivanovski S. Residual periodontal pockets are a risk indicator for peri-implantitis in patients treated for periodontitis. *Clin Oral Implants Res* 2012;23:325–333.
- Aoki M, Takanashi K, Matsukubo T, et al. Transmission of periodontopathic bacteria from natural teeth to implants. *Clin Implant Dent Relat Res* 2012;14:406–411.
- Socransky SS, Haffajee AD, Cugini MA, Smith C, Kent RL. Microbial complexes in subgingival plaque. *J Clin Periodontol* 1998;25:134–144.
- Swierkot K, Lottholz P, Flores-de-Jacoby L, Mengel R. 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;83:1213–1225.
- Levin L, Ofec R, Grossmann Y, Anner R. Periodontal disease as a risk for dental implant failure over time: a long-term historical cohort study. *J Clin Periodontol* 2011;38: 732–737.
- Renvert S, Aghazadeh A, Hallström H, Persson GR. Factors related to peri-implantitis: a retrospective study. *Clin Oral Implants Res* 2014;25:522–529.
- Costa FO, Takenaka-Martinez S, Cota LOM, Ferreira SD, Silva GLM, Costa JE. Peri-implant disease in subjects with and without preventive maintenance: a 5-year follow-up. *J Clin Periodontol* 2012;39:173–181.
- Monje A, Wang H-L, Nart J. Association of preventive maintenance therapy compliance and peri-implant diseases: a cross-sectional study. *J Periodontol* 2017;88:1030–1041.
- Heitz-Mayfield LJA, Salvi GE, Mombelli A, et al. Supportive peri-implant therapy following anti-infective surgical peri-implantitis treatment: 5-year survival and success. *Clin Oral Implants Res* 2018;29:1–6.
- Strietzel FP, Reichart PA, Kale A, Kulkarni M, Wegner B, Küchler I. Smoking interferes with the prognosis of dental implant treatment: a systematic review and meta-analysis. *J Clin Periodontol* 2007;34:523–544.
- Alsaadi G, Quirynen M, Komárek A, van Steenberghe D. Impact of local and systemic factors on the incidence of oral implant failures, up to abutment connection. *J Clin Periodontol* 2007;34:610–617.

26. Schrott AR, Jimenez M, Hwang J-W, Fiorellini J, Weber H-P. Five-year evaluation of the influence of keratinized mucosa on peri-implant soft-tissue health and stability around implants supporting full-arch mandibular fixed prostheses. *Clin Oral Implants Res* 2009;20:1170–1177.
27. Monje A, Blasi G. Significance of keratinized mucosa/gingiva on peri-implant and adjacent periodontal conditions in erratic maintenance compliers. *J Periodontol* 2019;90:445–453.
28. Lang NP, L oe H. The relationship between the width of keratinized gingiva and gingival health. *J Periodontol* 1972;43:623–627.
29. Chackartchi T, Romanos GE, Sculean A. Soft tissue-related complications and management around dental implants. *Periodontol* 2000 2019;81:124–138.
30. Wilson TG, Valderrama P, Burbano M, et al. Foreign bodies associated with peri-implantitis human biopsies. *J Periodontol* 2015;86:9–15.
31. Korsch M, Walther W. Peri-implantitis associated with type of cement: a retrospective analysis of different types of cement and their clinical correlation to the peri-implant tissue. *Clin Implant Dent Relat Res* 2015;17(Suppl 2):e434–e443.
32. Kotsakis GA, Zhang L, Gaillard P, Raedel M, Walter MH, Konstantinidis IK. Investigation of the association between cement retention and prevalent peri-implant diseases: a cross-sectional study. *J Periodontol* 2016;87:212–220.
33. Linkevicius T, Puisys A, Vindasiute E, Linkeviciene L, Apse P. Does residual cement around implant-supported restorations cause peri-implant disease? A retrospective case analysis. *Clin Oral Implants Res* 2013;24:1179–1184.
34. Quaranta A, Lim ZW, Tang J, Perrotti V, Leichter J. The impact of residual subgingival cement on biological complications around dental implants: a systematic review. *Implant Dent* 2017;26:465–474.
35. Staubli N, Walter C, Schmidt JC, Weiger R, Zitzmann NU. Excess cement and the risk of peri-implant disease - a systematic review. *Clin Oral Implants Res* 2017;28:1278–1290.
36. Salem D, Alshihri A, Levin L. Peri-implantitis induced by a retained retraction cord. *Quintessence Int* 2014;45:141–143.
37. Linkevicius T, Vindasiute E, Puisys A, Linkeviciene L, Maslova N, Puriene A. The influence of the cementation margin position on the amount of undetected cement. A prospective clinical study. *Clin Oral Implants Res* 2013;24:71–76.
38. Monje A, Chappuis V, Monje F, et al. The critical peri-implant buccal bone wall thickness revisited: an experimental study in the beagle dog. *Int J Oral Maxillofac Implants* 2019;34:1328–1336.
39. Ara ujo MG, Lindhe J. Dimensional ridge alterations following tooth extraction. An experimental study in the dog. *J Clin Periodontol* 2005;32:212–218.
40. Chappuis V, Ara ujo MG, Buser D. Clinical relevance of dimensional bone and soft tissue alterations post-extraction in esthetic sites. *Periodontol* 2000 2017;73:73–83.
41. Bassir SH, Alhareky M, Wangsrimongkol B, Jia Y, Karimbux N. Systematic review and meta-analysis of hard tissue outcomes of alveolar ridge preservation. *Int J Oral Maxillofac Implants* 2018;33:979–994.
42. Juodzbalyg G, Stumbras A, Goyushov S, Duruel O, T oz um TF. Morphological classification of extraction sockets and clinical decision tree for socket preservation/augmentation after tooth extraction: a systematic review. *J Oral Maxillofac Res* 2019;10:e3.
43. Monje A, Galindo-Moreno P, T oz um TF, Su arez-L opez del Amo F, Wang H-L. Into the paradigm of local factors as contributors for peri-implant disease: short communication. *Int J Oral Maxillofac Implants* 2016;31:288–292.
44. Buser D, Chappuis V, Bornstein MM, Wittneben J-G, Frei M, Belser UC. Long-term stability of contour augmentation with early implant placement following single tooth extraction in the esthetic zone: a prospective, cross-sectional study in 41 patients with a 5- to 9-year follow-up. *J Periodontol* 2013;84:1517–1527.
45. Jensen SS, Bosshardt DD, Gruber R, Buser D. Long-term stability of contour augmentation in the esthetic zone: histologic and histomorphometric evaluation of 12 human biopsies 14 to 80 months after augmentation. *J Periodontol* 2014;85:1549–1556.
46. Chappuis V, Rahman L, Buser R, Janner SFM, Belser UC, Buser D. Effectiveness of contour augmentation with guided bone regeneration: 10-year results. *J Dent Res* 2018;97:266–274.
47. Merli M, Merli I, Raffaelli E, Pagliaro U, Nastri L, Nieri M. Bone augmentation at implant dehiscences and fenestrations. A systematic review of randomised controlled trials. *Eur J Oral Implantol* 2016;9:11–32.
48. Isidor F. Histological evaluation of peri-implant bone at implants subjected to occlusal overload or plaque accumulation. *Clin Oral Implants Res* 1997;8:1–9.
49. Kozlovsky A, Tal H, Laufer B-Z, et al. Impact of implant overloading on the peri-implant bone in inflamed and non-inflamed peri-implant mucosa. *Clin Oral Implants Res* 2007;18:601–610.
50. Nagasawa M, Takano R, Maeda T, Uoshima K. Observation of the bone surrounding an overloaded implant in a novel rat model. *Int J Oral Maxillofac Implants* 2013;28:109–116.
51. Chambrone L, Chambrone LA, Lima LA. Effects of occlusal overload on peri-implant tissue health: a systematic review of animal-model studies. *J Periodontol* 2010;81:1367–1378.
52. Kim Y, Oh T-J, Misch CE, Wang H-L. Occlusal considerations in implant therapy: clinical guidelines with biomechanical rationale. *Clin Oral Implants Res* 2005;16:26–35.
53. Merin RL. Repair of peri-implant bone loss after occlusal adjustment: a case report. *J Am Dent Assoc* 2014;145:1058–1062.
54. Passanezi E, Sant’Ana ACP, Damante CA. Occlusal trauma and mucositis or peri-implantitis? *J Am Dent Assoc* 2017;148:106–112.
55. French D, Noroozi M, Shariati B, Larjava H. Clinical retrospective study of self-reported penicillin allergy on dental implant failures and infections. *Quintessence Int* 2016;47:861–870.
56. Salom o-Coll O, Lozano-Carrascal N, L azaro-Abdulkarim A, Hern andez-Alfaro F, Gargallo-Albiol J, Satorres-Nieto M. Do penicillin-allergic patients present a higher rate of implant failure? *Int J Oral Maxillofac Implants* 2018;33:1390–1395.
57. Monje A, Catena A, Borgnakke WS. Association between diabetes mellitus/hyperglycaemia and peri-implant diseases: Systematic review and meta-analysis. *J Clin Periodontol* 2017;44:636–648.
58. Katafuchi M, Weinstein BF, Leroux BG, Chen Y-W, Daubert DM. Restoration contour is a risk indicator for peri-implantitis: a cross-sectional radiographic analysis. *J Clin Periodontol* 2018;45:225–232.
59. Kuru B, Yildirim S. Treatment of localized gingival recessions using gingival unit grafts: a randomized controlled clinical trial. *J Periodontol* 2013;84:41–50.
60. Yildirim S, Kuru B. Gingival unit transfer using in the Miller III recession defect treatment. *World J Clin Cases* 2015;3:199–203.
61. Jenabian N, Bahabadi MY, Bijani A, Rad MR. Gingival unit graft versus free gingival graft for treatment of gingival recession: a randomized controlled clinical trial. *J Dent (Tehran)* 2016;13:184–192.
62. Poyato-Ferrera M, Segura-Egea JJ, Bull on-Fern andez P. Comparison of modified Bass technique with normal toothbrushing practices for efficacy in supragingival plaque removal. *Int J Dent Hyg* 2003;1:110–114.
63. Romanos GE, Everts H, Nentwig GH. Effects of diode and Nd:YAG laser irradiation on titanium discs: a scanning electron microscope examination. *J Periodontol* 2000;71:810–815.
64. Suzuki JB. Salvaging implants with an Nd:YAG Laser: a novel approach to a growing problem. *Compend Contin Educ Dent* 2015;36:756–761.
65. Steigmann M, Salama M, Wang H-L. Periosteal pocket flap for horizontal bone regeneration: a case series. *Int J Periodontics Restorative Dent* 2012;32:311–320.



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