Philip Leander Keeve

Patient-specific treatment of peri-implant inflammation

Summary: The use of dental implants in order to rehabilitate patients with fixed or removable implant-supported restorations has become widespread in recent decades. For example, according to the current German Oral Health Study (DMS V), patients were already 10 times more likely to be treated with implants in 2014 than in 1997 [41]. According to statistics from the American Dental Association, an estimated 5 million implants are placed annually in the USA alone [30]. The increasing life expectancy together with the desire for fixed restorations is expected to further strengthen this trend in the future. The steadily increasing number of implants that are placed by dentists has also been accompanied by an increase in the overall number of post-implant complications. Thus, due to the increased prevalence of biological complications, relevant patient-specific risk factors must be accounted for as part of implant planning and treatment. In this sense, a synoptic treatment concept that considers the foreseeable patient-specific risk factors for peri-implant inflammation plays an important role from the pre-implant to the post-prosthetic treatment phase. The article explores the multitude of patient-specific risk factors and the various therapeutic options available as the key to long-term implant treatment success.

Keywords: implants; peri-implant mucositis; peri-implantitis; risk factor; treatment

Weser Specialist Dental Center, Hameln, Germany: Philip Leander Keeve, DDS, MSc
Translation from German: Cristian Miron
Citation: Keeve PL: Patient-specific treatment of peri-implant inflammation. Dtsch Zahnärztl Z Int 2022; 4: 18–29
Peer reviewed article: submitted: 08.04.2021, revised version accepted: 18.09.2021
DOI.org/10.53180/dzz-int.2022.0003

Definition and diagnosis of peri-implant inflammation

When referring to peri-implant inflammation, reversible peri-implant mucositis, which is inflammation confined to the peri-implant soft tissue, must be distinguished from irreversible peri-implantitis, which also involves the progressive inflammation of the surrounding bone [4].

Due to the difficulty in diagnosing peri-implant conditions, the World Workshop on the Classification of Periodontal and Peri-implant Diseases and Conditions defined the characteristics of periodontal and peri-implant health for the first time in 2017. These include the absence of mucosal redness, bleeding on probing (BOP) as well as swelling and suppuration around implants. The definition of a critical probing depth, which is no longer associated with peri-implant health, is not readily available for implants, unlike for periodontitis. In the absence of clinical signs of inflammation, the peri-implant tissues around implants may be healthy even when increased pocket probing depths above 3 mm are present. If bleeding and/or suppuration occurs during gentle probing of the peri-implant soft tissues, this is defined as peri-implant mucositis. If there is a combination of bleeding/suppuration with an increasing probing depth compared to previous examinations, or probing depths of ≥6 mm and radiological detectable bone resorption beyond the initial bone level after implant placement, then this is referred to as peri-implantitis [11]. In the absence of initial radiological findings, bone resorption ≥3 mm apical to the intrabony part of the implant is considered indicative of peri-implantitis [91].

The prevalence of peri-implantitis has been estimated to vary between 10 and 29 % [24, 42]. The variation of these prevalence figures is primarily due to the complex definition and diagnosis of peri-implantitis as well as a high heterogeneity of study criteria [67].

The prevalence of mucositis is on average 40 % and of peri-implantitis 21.7 % (95 % CI 14–30 %) according to systematic reviews [24]. Half of the implants affected by peri-implantitis become diseased within 3 years, and overall, peri-implantitis is diagnosed considerably more often 5 years consecutive to prosthetic restoration [86, 87]. It is assumed that initial mucositis can develop into peri-implantitis and that peri-implant bone resorption accelerates over time [102].

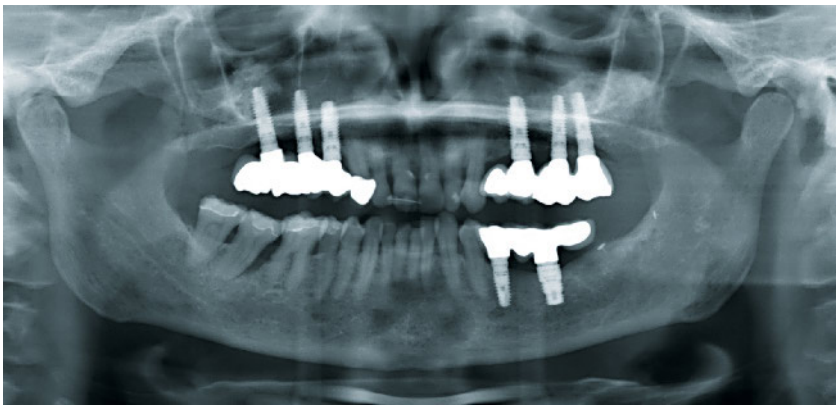

Figure 1 Orthopantomogram (initial condition).

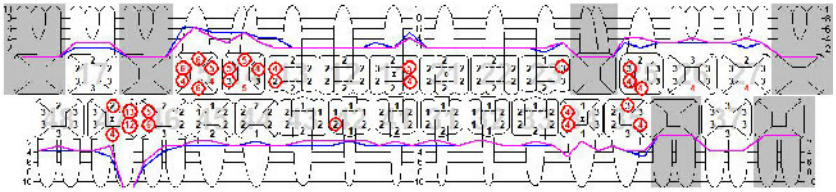

Figure 2 Clinical probing values of the entire dentition (initial condition).

For the classification of peri-implantitis and corresponding peri-implant bone lesions, a classification of different defect morphologies – especially against the background of the various therapeutic options – is recommended. In this respect, intrabony defects (Class I) are distinguished from horizontal supracrestal defects (Class II). The supracrestal portion is defined as the distance between the transition from the smooth to the machined implant portion and the peri-implant crestal alveolar bone [108].

Intrabony defects can be divided into purely vestibular or oral dehiscence defects (Class Ia), vestibular or oral dehiscence defects with additional semicircular portions (Class Ib), vestibular or oral dehiscence defects with additional circular bone resorption (class Ic), and into circular bone resorption with vestibular and oral dehiscence defects (class Id) or bilaterally preserved compact bone (class Ie).

Horizontal and intrabony defects mainly occur together. According to current data, 55.3 % of peri-implant bone defects belong to Class Ie [103].

Patient-specific risk assessment of treatment-relevant risk factors

Possible risk factors include patient age, gender, gene polymorphisms, cardiovascular disease, rheumatoid disease, osteoporosis, condition of residual dentition, implant design and surface as well as implant site and type of restoration. In the following chapter, the 5 most important treatment-relevant risk factors relating to peri-implant inflammation are discussed in detail [102].

Association of periodontitis and peri-implant diseases

The similarity in the etiopathogenesis of peri-implant and periodontal inflammatory diseases highlights that periodontitis is a risk factor for biological complications and failures of dental implant treatment [42]. The corresponding causal association between plaque formation around implants and peri-implant mucositis has been demonstrated. However, the reaction of hard and soft tissues to

the pathological biofilm around teeth and implants is only to some degree comparable. The microflora found around teeth and implants, that have been exposed to the oral environment for 6 months, is already comparable; however, it does not lead to the development and progression of peri-implant disease in every case. Periodontitis is considered a risk factor for peri-implantitis due to the possible transfer of periodontal pathogens onto the implant surfaces and the reservoir effect of existing periodontal pockets [42]. Additionally, genetic factors are strongly involved in the etiopathogenesis of periodontitis and peri-implantitis and they lead to a correspondingly high susceptibility to both diseases in the same patient group [12, 34–36]. The occurrence of peri-implant diseases clearly correlates with the predisposition and severity of existing periodontitis in the individual patient. However, due to the anatomical conditions of peri-implant tissues, inflammation-induced bone resorption often proceeds faster than at natural teeth. Therefore, patients with severe forms of periodontitis have significantly lower implant survival rates (88–98.4 %) than patients with moderate periodontitis (92.8–100 %) or periodontally healthy individuals (96–100 %) in a 5- to 10-year period after periodontal treatment and implant placement [58]. Implant success after 10 years is significantly lower in patients with generalized, severe periodontitis (83.33 %) than in periodontally healthy patients who have success rates of up to 100 % [66, 114]. Overall, periodontally treated patients with initial stage I–II periodontitis have higher implant survival rates and less bone resorption around implants than patients with more pronounced stage III–IV periodontitis [58, 86]. For more severe grade C periodontitis, much lower survival and success rates and greater marginal bone resorption [21] are observed than for grades A and B [66, 86, 114]. In particular, patients with a history of severe periodontitis accompanied by poor plaque control and irregular maintenance therapy are at significantly higher risk for the occurrence of peri-implantitis [23, 81, 86].

Currently, the strongest risk factors for peri-implantitis include remaining large pocket depths, lack of follow-up care, poor oral hygiene and severe forms of periodontitis. Even localized, remaining inflammation (PPD ≥6 mm with BOP) leads to a 5-fold higher risk of inflammatory processes around implants compared to successfully treated periodontitis [17].

Poor oral hygiene/irregular maintenance therapy

The lack of compliance during maintenance therapy is associated with tooth loss and attachment loss [6, 8, 121]. The relationship between microbial plaque and diseases such as gingivitis and periodontitis has been demonstrated in numerous studies [7, 8, 61]. Causal therapy in the sense of plaque removal showed improvements in inflammatory lesions in plaque-associated forms of etiopathogenesis [61]. A subsequent study involving mucositis patients demonstrated that efficient plaque control was critical for the prevention of peri-implantitis [18]. Thus, the incidence of peri-implantitis over a 5-year period was significantly lower in patients undergoing maintenance therapy (18 %) than in patients not undergoing maintenance therapy (44 %). A study by Roccuzzo et al [85] also found a higher prevalence of peri-implantitis over a 10-year period in the absence of maintenance therapy (41 %) than in the presence of maintenance therapy (27 %). Patients who attended maintenance therapy less than 2 times per year showed an increased risk of developing peri-implantitis (OR 4.69; 95 % CI 1.17–18.79).

Moreover, a strong association between inadequate home-based oral hygiene and peri-implantitis was shown in 4 studies with an odds ratio ranging from 5 to 14 [3, 27, 90, 101]. However, conflicting findings have also been published [53, 65, 96], despite the fact that a singular plaque index recording in these studies generally does not reflect an exhaustive means of measuring the long-term oral hygiene status. Serino and Stroem investigated the oral hygiene ability of patients who displayed peri-implantitis at implant-supported restorations [110] and were able to show that peri-implantitis was diagnosed in only 18 % of the areas accessible to oral hygiene and in 65 % of the areas not accessible to hygiene.

Smoking

Smoking is associated with chronic periodontitis, attachment loss and tooth loss [9, 116]. There is also an association between smoking and peri-implantitis [25]. In a 10-year study by Karoussis et al, smokers displayed peri-implantitis at 18 % of all implants and non-smokers at only 6 % of all implants. In addition to the incorporation of nicotine, cotinine, and their decay products into periodontal tissues, smoking produces hydroxide and peroxyl radicals which destroy host DNA, cause lipid peroxidation of the cell membrane, damage endothelial cells, and induce vascular smooth muscle growth, thus causing numerous tissue changes [117]. Reactive oxygen species (ROS) also activate the formation of proinflammatory mediators such as interleukin-6, tumor necrosis factor-alpha or interleukin-1 beta which are important in the pathogenesis of peri-implant diseases. Smoking also leads to a reduction of blood vessel density [84] and to the exacerbation of the inflammatory lesion through genetic variation in the biotransformation of N-acetyltransferase-2, cytochrome P450, CYP2E4, and gluthathione S-transferase [51, 52]. Moreover, the functional capacity and number of polymorphonuclear neutrophil granulocytes decreases in smokers [33, 72] in relation to a cytotoxic effect of nicotine on fibroblast migration [26].

Lindquist et al. showed considerably greater crestal bone resorption in smokers than in nonsmokers [60]. However, contrasting results from Aguirre-Zorzano et al. showed a peri-implantitis prevalence of 15 % in 239 patients over 5 years, with no increased risk among smokers [3, 20, 23, 76].

On the whole, smoking cannot be considered a relevant predictor of peri-implantitis development, but it should be considered a cofactor, especially when other risk factors such as periodontitis are present. Patients

with existing cofactor smoking and a periodontally compromised dentition have a 4.6-fold increased risk of peri-implantitis compared to periodontally compromised nonsmokers [113].

Future studies should survey the cumulative amount of nicotine abuse in "pack-years" and differentiate between smokers, former smokers, and nonsmokers in order to further elucidate the associations [25].

Diabetes mellitus

With a worldwide prevalence of approximately 8 % in adults [111], diabetes mellitus is considered another important risk factor for peri-implant disease [11] and periodontitis [29]. Due to the parallelisms in the pathogenesis of peri-implantitis and periodontitis, it is suspected that biological complications at implants are favored by this metabolic disease. Since there are bidirectional relationships between periodontitis, peri-implantitis and diabetes mellitus, glycemic control (HbA_{1c} value) and its re-evaluation are mandatory as part of patient-specific treatment. Hyperglycemia results in the formation of advanced glycation end products (AGE) which dock to inflammatory cells via their receptor (RAGE) and lead to an increased release of inflammatory molecules (reactive oxygen species and cytokines), a reduction in chemotaxis and the adhesion performance of inflammatory cells as well as an increase in bacterially induced inflammation of peri-implant tissues [31]. Collagen cross-linking via AGE also leads to more difficult turnover of the peri-implant connective tissue [31]. A large number of studies have found a higher risk of peri-implantitis in patients with poorly controlled diabetes mellitus. Ferreira et al. showed a peri-implantitis prevalence of 24 % in untreated diabetic patients or patients with a blood glucose level of ≥126 mg/dL compared with 7 % in the control group of non-diabetic patients, which corresponds to an odds ratio of 1.9 [27]. Patients who received their diabetes diagnosis at the time of implant placement showed a 3-fold higher risk of developing peri-implantitis at the time of the 11-year follow-up evaluation [19].

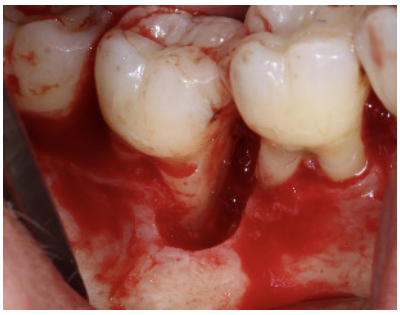

Figure 3 Illustration of the morphology of the mesial intrabony defect at 46 by means of simplified papilla preservation flap after re-evaluation of the previously performed conservative periodontal therapy.

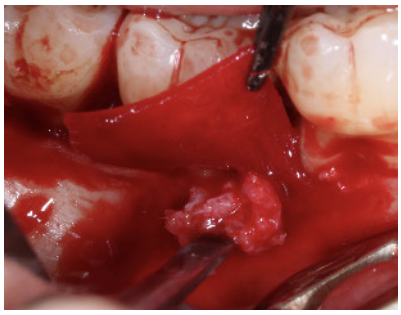

Figure 4 Debridement of the root surface with subsequent membrane positioning in the context of guided tissue regeneration and defect filling with autologous bone.

Tawil et al. studied 45 patients with diabetes mellitus over an average duration of 42 months (1–12 years); they diagnosed no peri-implantitis in patients with an HbA_{1c} ≤7 %, but in the group of patients with HbA_{1c} values between 7 and 9 %, they diagnosed peri-implantitis at 6 of 141 implants [115].

Diabetes is thus considered an important potential risk factor for peri-implantitis [76, 102]. More specifically, it has been shown that diabetics have a two-fold higher risk of peri-implantitis than non-diabetics (OR 2.5, 95 % CI 1.4–4.5) [25]. From 3 studies in which the information on diabetes mellitus was collected, not only anamnestically, but also clinically, 2 studies showed a significant effect of diabetes [27] or HbA_{1c} levels [115] on peri-implantitis.

Attached and/or keratinized mucosa

Although previous reviews [119] have shown that the lack of attached mucosa has no negative influence on peri-implant health, further meta-analyses, mainly based on cross-sectional studies, have conveyed that lower plaque accumulation, less tissue inflammation, recession, and clinical attachment loss occurs when a minimum width of 1–2 mm keratinized mucosa is present in comparison to when this minimum width is absent [59]. A lack of attached mucosa may negatively affect the ability of the patient to clean [59]. Pain-free, home-based cleaning of implant superstructures is considered an important goal in patient-specific treatment. The attached mucosa – independent of muscle movements – should not allow any microorganisms to deposit on the peri-implant transmucosal attachment due to crevice formation in the area of the implant neck [55]. Recent reviews have shown significantly less peri-implant inflammation and lower plaque and gingival indices in patients with at least 2 mm of keratinized or attached peri-implant mucosa [13, 44, 59]. Although less gingival recession and attachment loss occurred with sufficient mucosa, no significant differences could be seen with respect to probing depth values [2, 44, 96, 123]. A non-significant trend indicates increased bone resorption when there is insufficient mucosa [46, 96]. Rokn et al. demonstrated a lack of keratinized mucosa as a statistically significant risk factor for peri-implantitis (OR 3.89; 95 % CI 2.34–5.98) [90]. Moreover, Souza et al. found increased discomfort during home-based oral hygiene in areas where there is less than 2 mm of keratinized mucosa, which was accompanied by correspondingly higher plaque values and increased bleeding on probing [112].

Treatment options for peri-implant inflammation

Prevention of patient-specific risk factors

Patient-specific treatment of peri-implant inflammation comprises of a synoptic treatment concept with, on

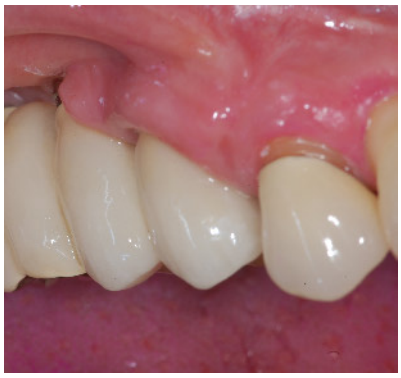

Figure 5 Peri-implantitis in region 15 and 16 (initial clinical condition).

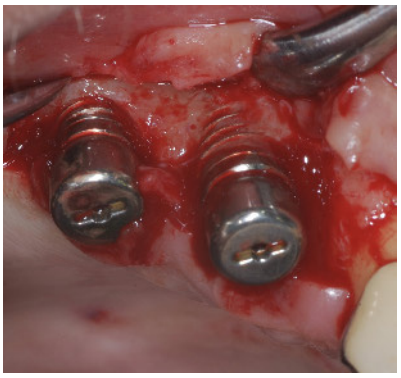

Figure 6 Surgical treatment of peri-implantitis in region 15 and 16 (horizontal bone resorption) and removal of the superstructure 8 weeks after closed scaling and decontamination of the implant surface.

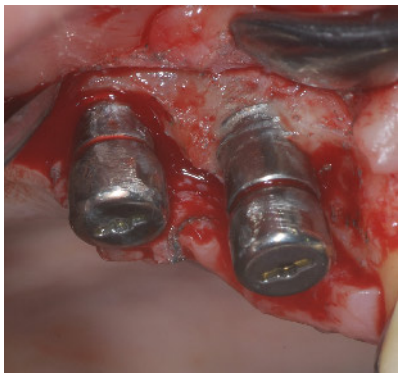

Figure 7 Implantoplasty using rotary instruments and subsequent removal of the granulation tissue and direct insertion of the restoration.

the one hand, attention to the detailed risk factors so as to prevent the development or renewed progression of peri-implant infections and, on the other hand, anti-inflammatory, if possible reconstructive treatment of peri-implant lesions.

Fundamental to the success of implant treatment is the long-term avoidance of biological, technical and esthetic complications. At the biological level, the absence of peri-implant mucositis, peri-implantitis and the establishment of stable soft tissue conditions is necessary, especially as part of maintenance therapy after the active treatment of peri-implant infections. Biological complications at implants differ in their frequency and severity in patients with and without periodontitis. The implementation of careful anti-infective periodontal therapy with the reduction of inflammatory signs and probing depth values prior to the treatment of peri-implant inflammation is thus mandatory (Fig. 3–4).

For the successful long-term treatment of peri-implant inflammation, particularly from the patient-specific point of view, it is essential to design the prosthetic restoration as close as possible to the natural appearance of the teeth, with correspondingly good hygiene characteristics and an optically and functionally satisfactory result; often, this can only be achieved by restoring the lost tissue dimensions.

Tooth loss leads to both bone and soft tissue loss, which are often exacerbated by atrophic bone remodeling processes. It is not uncommon to have partially limited bone volume at the time of the indication for implant placement. Augmentation of the alveolar ridge may be required in order to insert an implant in a physiological position, with sufficient bone quantity, and a prosthetically correct position.

The extent to which the crown-to-implant length ratio has an influence on the survival, marginal bone level or prosthetic complications in the absence of augmentation is controversially discussed. Some reviews concluded that no negative influences exist [69, 75]. In contrast, other systematic reviews observed a higher incidence of prosthetic complications such as abutment loosening or fractures, mainly in posterior jaw regions. Restoration of the near-original dimensions of the hard and soft tissues can minimize these risks in the long term [64]. Moreover, the esthetic result is significantly improved and the ability to maintain oral hygiene, thus ensuring the prevention of inflammatory processes [44].

Treatment of peri-implant mucositis

If peri-implant mucositis develops despite consideration of these recommendations and risk factors, the causal therapy of the existing risk factors needs to start with the utmost priority; this includes smoking cessation, control of diabetes mellitus and specific oral hygiene instruction. Localized plaque-induced inflammation should be eliminated by non-surgical mechanical plaque removal, optimization of oral hygiene skills, and inclusion in a regular maintenance therapy program [73]. Efficient plaque removal without damaging the implant structure is the primary goal [63]. Home-based oral hygiene can be carried out using manual or electric toothbrushes and appropriate interdental brushes [83].

In the case of isolated inflammatory sites in combination with cemented restorations, remaining cement remnants should be taken into account and gently removed by non-surgical cleaning. In cases where non-surgical cleaning is unsuccessful, the removal of the prosthetic restoration and surgical cleaning and cementation under direct view are recommended [83] because the removal of cement remnants leads to a significant improvement of peri-implant tissue health [120].

The question of whether fixed prosthetic restorations should be screw-retained or cemented is still controversially discussed in literature. In a 2016 review, no clinically relevant differences were found with regard to marginal bone loss at the implant site for screw-retained or cemented restorations [57]. Other authors found increased plaque adhesion to cement remnants in combination with increased incidence of peri-implant in-

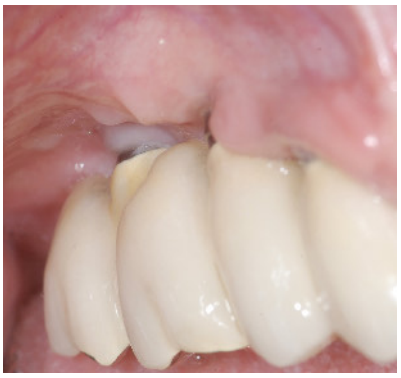

Figure 8 Condition after surgical peri-implantitis treatment with insufficient soft tissue (3 months).

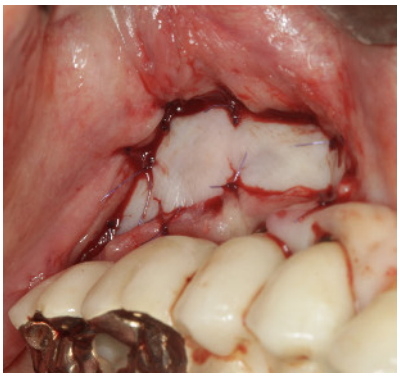

Figure 9 Harvesting of free mucosal graft (right palate) and vestibuloplasty in order to widen the keratinized mucosa.

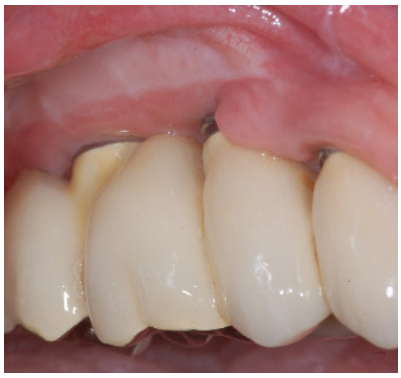

Figure 10 Stable peri-implant and inflammation-free soft tissue condition at the time of a 3-year follow-up check of region 15 and 16.

flammation when methacrylate-based cements were used [71]. In periodontitis patients, the use of screw-retained restorations appears to be desirable because it reliably excludes retention of cement remnants and makes the construction easier to remove in cases of biological or technical complications. On the other hand, technical complications such as fracturing of the veneering are more common among screw-retained restorations [99]. Thus, when choosing cemented restorations, the fabrication of customized, anatomical abutments is helpful for preventing a deep subgingival position of the cement gap and for ensuring the removal of cement remnants. In addition, the avoidance of overhanging margins or concave surfaces on crowns and bridges should be aimed for in order to facilitate ideal home-based oral hygiene.

During mechanical cleaning, titanium and carbon fiber instruments as well as plastic and teflon coated ultrasonic systems are used specifically in order to protect the implant surface [97]; this appears to be advantageous for any potential augmentative therapy approaches in the future. However, it must be noted that debridement is in this case more ineffective and remnants may be left over on the surface [122]. In a randomized controlled trial, it was shown that the use of glycine powder systems gave better results for bleeding on probing in comparison to mechanical cleaning with carbon fiber instruments [40, 98]. Nonsurgical therapy is considered a successful treatment step in reversible peri-implant mucositis and is subsequently characterized by the absence of bleeding or suppuration on probing [73].

Treatment of peri-implantitis

Peri-implantitis lesions can be differentiated into early and late infections. Early peri-implant inflammation occurs immediately or in the first weeks after implant placement and it is mostly caused by postoperative wound healing disorders. Late peri-implantitis is usually diagnosed after the implant's osteointegration has been completed and its prosthetic restoration [82].

The removal of the affected implant is usually indicated upon clinical and radiological diagnosis, as well as, very low Resonance Frequency Analysis (RFA) values or very high Damping Capacity Analysis (DCA) values, deep tapping sounds, mobility and large probing values, which check for osseointegration [73]. In all other cases, the peri-implant inflammation must be permanently reverted to a stagnation phase, beginning with a non-surgical treatment phase and the adjustment of all oral hygiene parameters.

The basis for systematic and continuous prevention and treatment of peri-implant diseases is the original CIST concept (cumulative interceptive supportive therapy or antiseptic cumulative supportive therapy) according to Mombelli and Lang [68]. The CIST concept is a step-by-step model divided into 4 treatment steps. Depending on the diagnostic course, the modular therapy guide initially includes hygiene instructions and professional dental cleanings (part A), followed by chlorhexidine rinses, gel applications (part B) and systemic antibiotic medication (part C) as well as subsequent surgical interventions with either resective or regenerative treatment approaches (part D). However, especially in the further development of patient-specific treatments, the existing risk factors must be recognized and adjusted, and the evaluation of the treatment at each step must not be made according to rigid consideration of the probing values, but according to the change in probing values over time [43].

Nonsurgical treatment of peri-implantitis can be expected to reduce bleeding on probing, but it can only result in a limited improvement in probing values [77, 118]. When adjuvant irrigation solutions or antibiotics were used, such as minocycline products and tetracycline derivatives, they proved to be effective and improved the bleeding on probing values as well as the probing depths [10, 14, 78, 79, 100]. However, the administration of systemic antibiotics should be avoided for nonsurgical procedures [77]. The adjuvant use of Nd:YAG and Er:YAG lasers in addition to mechanical therapy has also been shown to have only short-term

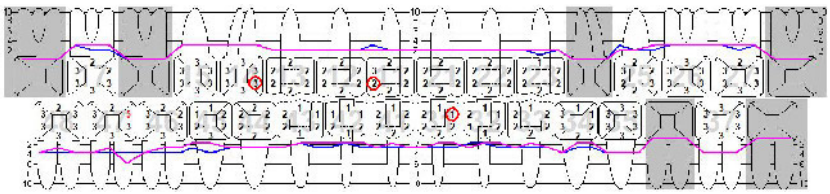

Figure 11 Clinical probing values of the entire dentition 3 years postoperatively (final findings).

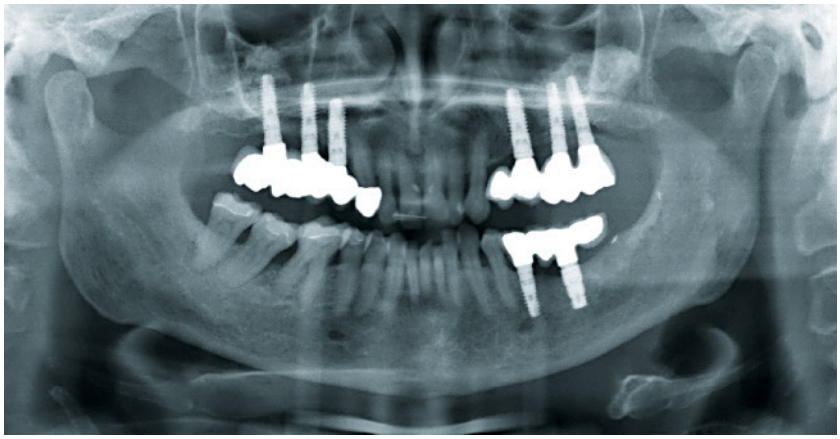

Figure 12 Orthopantomogram 3 years postoperatively (final findings).

success, which lasted a few months in terms of bleeding on probing and probing depths [1, 80].

Six weeks after the nonsurgical procedure, surgical, mechanical debridement including chemical decontamination of the implant surface should be performed. Access flaps, resective therapy approaches with or without implantoplasty, or augmentative procedures can be used during this operative intervention. In this context, the bony defect morphology and the position of the affected implant – inside or outside the esthetic area – are considered to be the decisive factors in further treatment planning. In principle, augmentative measures for intrabony components such as bowl-shaped defects (class Ie [108]) and 3- or 4-walled bone defects can achieve improved clinical and radiological therapeutic results in addition to anti-inflammatory ones. The remaining bony defect morphologies are usually treated with resective therapeutic procedures.

Surgical access flaps and resective treatment approaches are indicated for supracrestal bone defects (horizontal bone resorption) with exposed implant threads [45, 50]. Resective treatment of peri-implant inflammation can recontour the bone and reduce probing values. This can be performed together with or without smoothening of the implant surface. In the esthetic region, an access flap with a strictly intrasulcular incision can be used while preserving the soft tissue; in the posterior region, an apically displaced flap can be used [45]. In esthetic regions with moderate bone loss and shallow bone defects, the combination of surgical debridement with a free connective tissue graft is a recommended option in order to achieve significant clinical improvement while still avoiding the high risk of recession [37, 105]. In posterior areas, resective treatment together with implantoplasty lead to improved clinical and radiological results after a 3-year follow-up compared to the control group with only the resective approach without implantoplasty (STM: 1.64 ± 1.29 vs. 2.3 ± 1.45 mm) [93, 94] (Figs. 5–6). For implantoplasty, flame or ellipse shaped carbide burs (30 mm length) can be used with normal (12 cutting edges) and ultrafine (30 cutting edges) finishing grades. The smoothening of the surface is finalized with Arkansas and Greenie tips. However, the remaining titanium particles in the tissue should be reduced by means of gauze exposure and excision of the granulation tissue after implantoplasty or, depending on the indication and diagnosis, implantoplasty should be limited to the supramucosal areas before flap formation, since the effect of tissue reactions to the remaining titanium with regard to progressive peri-implant inflammation is currently unclear [45, 102]. In order to improve the course of treatment, it is recommended to remove the superstructure before the respective operative intervention, especially in the case of implantoplasty; in this way, the superstructure can be adapted with regard to its oral hygiene design before being reinserted [45]. Adjuvant systemic antibiotics in the case of resective procedures did not result in significant clinical and radiological long-term improvement [16].

Augmentative procedures are indicated for bowl-shaped bone defects (Class Ie [108]) and 3- or 4-walled bone defects where the bone contour is preferably preserved as a scaffold shape, especially in the case of moderately rough implant surfaces after considering the corresponding existing risk factors [29, 73, 88, 103, 108]. Pre-operatively, especially the implant position and design as well as the hygienic suitability of the prosthetic reconstruction should be critically evaluated [73]. For the execution of augmentative surgical interventions, the use of bone or bone substitutes in combination with or without a membrane technique for guided tissue regeneration, or in combination with biologically active agents, primarily enamel matrix protein derivatives, bone morphogenetic proteins (BMPs) or platelet-rich fibrin membranes (PRF), is available [74]. In the majority of studies, the augmentative interventions resulted in an improvement of the clinical and radiological parameters over a study period ranging from 6 months to 7–10 years [74]. Bleeding on probing reduced by an average of 25.9 % [32] to 91 % [28] over the follow-up period of up to 7 years. The probing values also decreased between 0.74 mm and

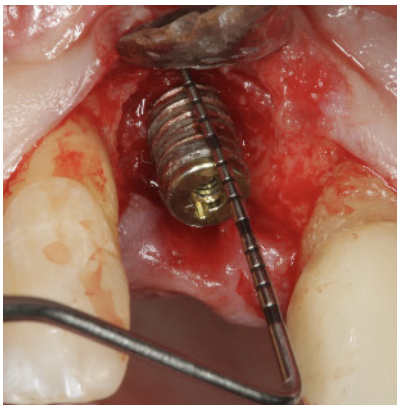

Figure 13 Combination of a supracrestal and Class Ie defect in the esthetic maxillary anterior region. After non-surgical treatment, surgical cleaning and decontamination of the implant surface is carried out.

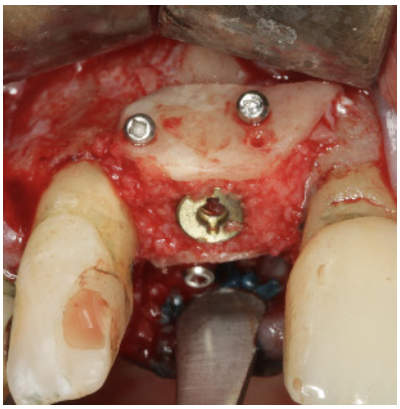

Figure 14 Augmentation of the defect using the biological 3D shell technique according to Khoury and retromolar bone harvesting with subsequent closed wound healing.

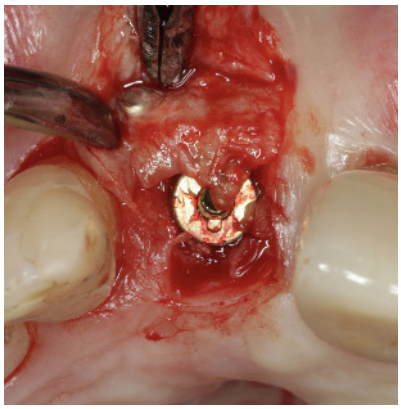

Figure 15 Re-entry at the exposed site after 3 months with complete reconstruction of the bony alveolar process and insertion of the existing prosthetic restoration.

Fig. 1–15: P. Keeve

5.4 mm [48, 104]. The type of surface decontamination had no significant effect on these parameters [22, 48, 54], and thus, cleaning with saline-soaked gauze can be considered as the standard for all surgical procedures [107]. Titanium granules as a filler did not show a positive influence on clinical parameters in augmentative procedures compared to simple access flaps [5, 39]. In 2 studies, there were no significant differences between the use of autologous bone alone and the combination with resorbable [95] and non-resorbable membranes [48]. In contrast, one study provided better clinical results when bone graft substitute was combined with a membrane [106]. Furthermore, the addition of enamel matrix protein derivatives did not improve probing depths and bleeding on probing compared to the control group with access flaps [38]. Therefore, long-term studies currently show no evidence for the clinical superiority of any particular combination in [74].

The question of whether to allow open or closed healing [92] and the benefit of adjuvant systemic antibiotics [74] also cannot be clearly answered on the basis of the current state of literature. If the superstructure permits a non-destructive removal and, in particular, the use of membrane technology where a correspondingly increased risk of exposure is considered [48], closed healing may be favored.

Stabilization and improvement of the treatment outcome

The described augmentative techniques, in contrast to the purely surgical access flaps and resective treatment approaches, aim not only to achieve an anti-inflammatory effect, but also to improve the therapeutic outcome in terms of probing depths, attachment level and defect filling. Additional options for hard and soft tissue management are described below.

Hard Tissue Management

Generally, augmentative procedures are limited to the intrabony region, so supracrestal implant surfaces should be treated with either debridement only or supracrestal limited implantoplasty, depending on the risk profile [104]. In the esthetic area, 3D restoration of the alveolar process including the supracrestal portions may be considered in the absence of risk factors – currently without scientific evidence. The author recommends the shell technique as a modification to the autogenous block augmentation for vertical bone resorption consecutive to peri-implantitis, so as to improve healing and bone stability [49] (Figs. 13–15). This concept of bone block grafting from the retromolar mandible uses a thin block graft as a biological membrane, which gives the particulate bone graft material the desired shape and dimension. Particulate bone has an increased surface area with a high regeneration potential and thus mostly improves osteoconduction. For closed healing, absolutely tension-free wound closure with periosteal slitting or adjunctive rotation/swing flaps is mandatory.

Soft Tissue Management

Before, during and after surgical peri-implantitis treatment, all risk factors (e.g. lack of attached keratinized mucosa) must be immediately checked [109]. If there is a strong muscular influence on the peri-implant soft tissue, the width of the keratinized mucosa should be increased previous to surgical augmentation therapy in order to optimize soft tissue handling, including primary wound closure. In the remaining cases, to prevent recurrence, this potential risk factor can be surgically resolved after successful treatment of the peri-implant inflammation [109]. In most cases, there is a deficit of attached keratinized mucosa after hard tissue augmentative or resective surgery. In this regard, despite limited scientific evidence, the absence or inadequate width of keratinized peri-implant mucosa is considered a source of risk for recurrent peri-implant disease. The presence of an adequate keratinized collar reduces plaque accumulation, tissue inflammation, mucosal recession, and attachment loss [44]. From a clinical perspec-

tive, a minimum width of 2 mm of keratinized, attached peri-implant mucosa is recommended in order to improve peri-implant soft tissue stability, allow the patient to adequately clean and minimize subsequent risks due to increased plaque accumulation. In the absence of this keratinized mucosa, it is imperative to utilize a free mucosal graft so as to improve the clinical situation. [15, 89, 109] (Figs. 8–10). In this regard, autologous free mucosal grafts from the palate show better results in terms of widening the keratinized mucosa compared to vestibuloplasty alone, acellular dermal matrices, or xenogeneic collagen matrices [15, 62].

Follow-up care

Follow-up care (supportive periodontal therapy) is key to the successful, long-term treatment of peri-implant inflammation [73] and it only functions when potential patient-specific risk factors are taken into consideration. During maintenance therapy, intensive, repetitive instruction, demonstration, and motivation of the patient is indispensable [47]. Moreover, the peri-implant probing depth values must gently be recorded and the re-evaluation of effective home-based as well as professional hygiene skills must be carried out. The recall interval should be selected according to the individual's risk profile [56, 70], whereby patients with previous peri-implant inflammation are generally considered to be at an increased risk [73]. For this reason, a close-meshed 3-month interval for supportive periodontal therapy should always be selected initially, which can always be adapted on a patient-specific basis according to existing risk factors.

Conclusion

Patient-specific treatment of peri-implant inflammation is based on a synoptic treatment concept with special attention to therapy-relevant risk factors. The prevention of newly recurring peri-implant infections and anti-inflammatory, if possible reconstructive, treatment of peri-implant lesions is considered to be the therapeutic goal.

With successful active periodontitis treatment, the establishment of adequate oral hygiene including prosthetic and/or soft tissue conditioning, as well as, possible nicotine reduction and the adjustment of diabetes mellitus with HbA_{1c} target value <7, significant risk factors can be eliminated and the initial conditions for the subsequent treatment of peri-implant inflammation can be created.

Peri-implant inflammation should be initially treated with non-surgical mechanical plaque removal and antimicrobial rinses. After re-evaluation, surgical mechanical debridement using access flaps, resective therapy approaches together with or without implantoplasty, or augmentative procedures may be used. In principle, resective therapy procedures together with or without implantoplasty can be used for supracrestal bone defects (horizontal bone resorption) and augmentative measures for intrabony components such as bowl-shaped defects. In the context of patient-specific treatment of peri-implant inflammation, particular importance is accorded to follow-up care and the accurate re-evaluation of risk factors.

Conflict of interest

P. L. Keeve is a lecturer among others for the companies Dentsply Sirona, Straumann, Hager & Meisinger, Stoma Dentalsysteme and Resorba. There is no direct cooperation with these companies for this article. No studies on humans or animals were conducted by the author for this article. The ethical guidelines stated in each case apply to the studies listed.

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Photo: P. Keeve

DR. PHILIP KEEVE L, MSC.
Fachzahnarzt Zentrum Weser:
Dr. Keeve + Kollegen
Süntelstraße 10–12,
31785 Hameln, Germany
E-Mail: pl.keeve@outlook.com