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Peri-implantitis: Better understanding, better treatment!

Houda El Ghoulbzouri[1], Houda El Ayachi[2], OumKeltoum Ennibi[3], Amine Cherkaoui[4]

[1,2,3,4] *Department of Periodontology, Laboratory of Oral Biology and Biotechnology, Dental School, Mohammed V University, Rabat, Morocco*
Corresponding Author; Houda El Ghoulbzouri

Abstract: *Over the last decades, dental implants have been used as a standard treatment option to support dental restorations after tooth loss and the proportion of patients with dental implants is increasing. Despite these encouraging data for the use of dental implants in oral rehabilitation, clinicians consider Peri-implantitis as one of the most common biological complications that may be encountered. Peri-implantitis is defined as an inflammatory process affecting tissues around an osseointegrated implant in function. Peri-implantitis is considered the most challenging biological complication as, if untreated, it may progress and result in implant loss. In addition, treatment of peri-implantitis requires extensive resources in dentistry. Prevention of the disease is therefore a high priority in every-day clinical practice to minimize the occurrence and the severity of the problem. This overview provides a synopsis on the identification of etiology and risk factors of peri-implantitis using current data prevention and management of the disease are also described.*

Keywords:*Dental implant,osseointegration, peri-implantitis,debridement, surgical treatment, periodontal maintenance.*

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I. Introduction

Dental implants are a common treatment modality in the replacement of missing teeth in partially or fully edentulous patients.[1]Although data showing long-term success of using dental implants some complications may occur. Such problems mainly refer to inflammatory conditions associated with a bacterial challenge.[2]With an increasing number of implants placed, complications associated with implants such as peri-implant diseases have also increased, occurring with a frequency ranging from 1% to 47%.[3,4]Peri-implantitis is considered the most challenging complication, as untreated disease may progress and result in implant loss. In addition, treatment of peri-implantitis requires extensive resources in dentistry. Prevention of the disease is therefore a high priority in every-day clinical practice to minimize the occurrence and the severity of the problem.[5,6]

II. Definition of peri-implantitis

In 2017's World Workshop Classification of Periodontal and Peri-implant diseases and Conditions, characteristics together with disease definitions and case definitions were presented for peri-implant health, peri-implant mucositis and peri-implantitis. It stated thatosseointegrated peri-implantitis is a plaque-associated pathological condition occurring in tissues arounddental implants, characterized by inflammation in the peri-implant mucosa and subsequent progressive loss of supporting tissue.[8]

Peri-implant Heath

Teeth and dental implants are alike, as they constitute hard materials passing from alveolar bone through oral mucosa. While there are obvious differences between the enamel and dentin at teeth as opposed to implant materials, there are also important differences between teeth and implants regarding the interface towards the surrounding hard and soft tissues. [9,10]

The tooth is anchored to the alveolar bone and gingiva through a periodontal ligament and supra-crestal connective tissue fibers. The fibrous attachment between root cementum and alveolar bone proper is formed in conjunction with root formation. The interface between the gingiva and the tooth crown is composed of a thin junctional epithelium, which is continuous with a sulcular and oral epithelium (Fig. 1).[9,10]

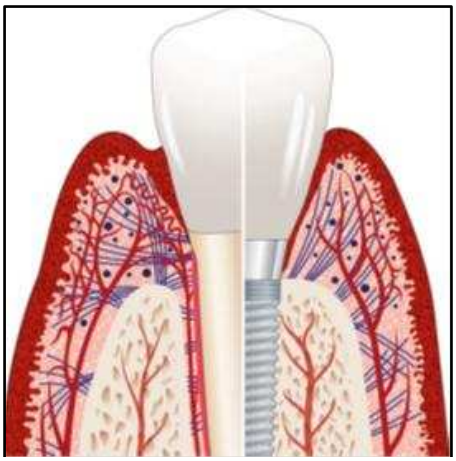

Figure 1: Schematic illustration of healthy tissues around a tooth and an implant[8]

Peri-implant hard and soft tissues, on the other hand, are formed as a result of a wound-healing process. The tissue injury elicited during the osteotomy procedure during implant installation leads to a series of reactions in bone, including degradation of the bone compartment immediately lateral to the implant after implant placement. The modeling and remodeling processes of the hard tissue interface to implants take several weeks and result in the formation of new bone in contact with the implant, i.e. osseointegration.[11]Similarly, and irrespective if a one-stage or a two-stage implant installation technique is used, the healing of the peri-implant mucosa takes several weeks and includes the formation of a junctional epithelium and an adaptation of the connective tissue towards the implant material in the compartment between the epithelium and the bone.[12]While the connective tissue-implant interface lacks a fibrous attachment, collagen fibers in this zone of the peri-implant mucosa are aligned parallel to the long axis of the implant. Furthermore, the density of blood vessels in the supra-crestal connective tissue of the peri-implant mucosa is lower than in the corresponding tissue compartment at teeth. [9,11]

Healthy peri-implant mucosa is clinically characterized by absence of visible signs of inflammation, swelling and redness and bleeding on probing.[7,10]The sealing function of the epithelial and connective tissue interface portions towards the implant device contributes to maintain the healthy status of the peri-implant mucosa. The supra-gingival/mucosal part of teeth and dental implants are constituents of the oral environment and are consistently exposed to a multitude of microorganisms. Implants and teeth do not possess the ability to shed-off microorganisms by epithelial desquamation as does the oral mucosa. Thus, the hard materials of implants and teeth are excellent niches for bacteria to attach and form a biofilm, in particular in the gingival- or peri-implant mucosal sulcus compartment. [10,11]

Two clinical varieties may be distinguished: peri-implant mucositis and peri-implantitis.

Peri-implant mucositis

It is the reversible inflammation of soft tissue without any signs of loss of supporting bone around the dental implant in function. It is a result ofthe accumulation of plaque in the implant mucosa. The symbiosis, a mutually beneficial relationship among members of the microbial community and between the microbial communities and the host with varying degrees of benefit, turns into an incipient dysbiosis. This change in the microbial communities associated with health results in a breakdown of the developing process, including the transition from healthy mucosa to peri-implant mucositis, was described recently (Fig. 2). [11,12]

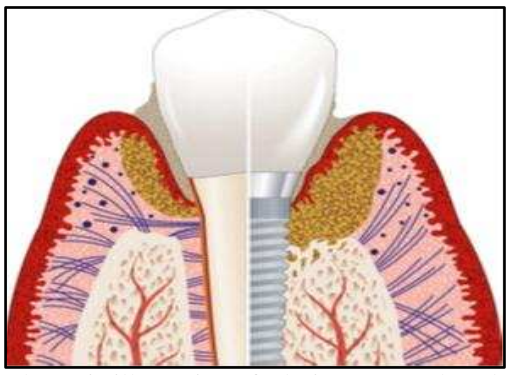

Figure 2: Schematic illustration of a lesion at a tooth and anImplant[8]

Peri-implant mucositis, if left untreated, can eventually lead to peri-implantitis. [8]

Peri-implantitis

It is an inflammation of tissues around an osseointegrated implant in function, resulting in soft tissue inflammation with loss of supporting bone around implant. Microbial differences between healthy and diseased peri-implant sites have been studied in detail.[13,14]Increases in peri-implant pocket depth have been shown to be associated with substantial changes in the submucosal microbiome and increasing levels of dysbiosis(Fig. 2). [15]
Mirroring the progression of gingivitis to periodontitis, peri-implant mucositis is assumed to precede peri-implantitis.[16]
Currently, features or conditions characterizing the conversion from peri-implant mucositis to peri-implantitis have not been identified. [16]

III. Etiology of peri-implantitis

A number of factors have been involved in the etiology of peri-implantitis over the years and although there has been some evidence for and against these factors, it is now accepted that this disease is caused by a microbial infection and represents inflammatory conditions in response to bacterial plaque. Other factors, such as history of periodontitis, may also contribute to the initiation and/or progression of peri-implantitis but remain incompletely understood.[17]

Bacterial biofilm

The presence of bacterial biofilms has been noted in numerous experimental and clinical examinations as the primary etiological factor for the development and progression of peri‑implant infections. The composition of these biofilms is similar to the subgingival bacteria of chronic periodontitis, dominated by Gram negative bacteria. Notably, studies have generally reported Porphorymonasgingivalisand other red complex bacteria at higher frequencies in peri-implantitis sites than healthy sites.[4-6] In healthy implants with stable probing depths of 5 mm or less the flora is characterized by gram-positive cocci and small number of gram-negative species.

A number of risk factors for peri-implantitis have been identified in the literature, ranging from microbial biofilm retentive elements associated with the design of the implant-supported prosthesis, to systemic predispositions and environmental exposures such as pre-existing periodontitis or cigarette smoking.[18]

Risk factors

History of periodontitis

Periodontitis is a common disease. It is ranked 6thamong the most prevalent disorders.[19]In a recent survey carried out in the United States, Eke et al. reported thatroughly 50% of the adult population (aged ≥30 years) presentedwith periodontitis. In individuals aged ≥65 years, the corresponding number was 68%.[20]
Current data shows that there is strong evidence from longitudinal and cross-sectional studies that a history of periodontitis constitutes a serious risk factor/indicator for peri-implantitis. [2,21]

Smoking

Smoking has been strongly associated with chronic periodontitis, attachment loss as well as tooth loss.[22,23]The majority of publications, however, failed to identify smoking as a risk factor/indicator for peri-implantitis. Aguirre-Zorzano et al. examined 239 implant-carrying individuals after a mean follow-up time of about 5 years and found an overall prevalence of peri-implantitis of 15%.[24]Smokers were not at higher risk. There is currently no conclusive evidence that smoking constitutes a risk factor/indicator for peri-implantitis.

Diabetes

Diabetes mellitus comprises a group of metabolic diseases where type 1 describes an autoimmune destruction of insulin producing β-cells and type 2 is characterized by insulin resistance.[25]. Available evidence is inconclusive as towhether diabetes is a risk factor/indicator for peri-implantitis.[2, 25]

Poor plaque control or lack of regular maintenancetherapy

As demonstrated in classical studies on periodontal diseases, lack of regular maintenance therapy is associated with tooth mortality and clinical attachment loss at teeth.[26]These findings have highlighted the importance of self-performed and professionally-administered infection control measures in the prevention of periodontal diseases. There is evidence that poor plaque control and lack of regular maintenance therapy constitute risk factors/ indicators for peri-implantitis.[2,27]

Implant surface

It is commonly classified into four categories depending on surface roughness values as:smooth, minimally rough, moderately rough or rough, with the majority of marketed implants having moderately rough surfaces,whichisoptimalforbonehealingresponse.Presentlyonlyafewstudiesprovideddataonhowimplantsurfacesinfluenceperi-implantdiseasewithnoevidencethatimplantsurfacecharacteristics can have a significant effect on the initiation of peri-implantitis [18,21,25]

Lack of keratinized mucosa

Whetherornotkeratinizedmucosaisneededarounddentalimplantstomaintainperiimplanthealthisacontroversialsubject. Several studies suggested that the absence of keratinized mucosa around dental implants increases thesusceptibilityoftheperiimplantregiontoplaqueinducedtissuedestruction,whileincreasedwidthofkeratinizedtissuearoundimplantsisassociatedwithlowermeanalveolarbonelossandimprovedindicesofsofttissuehealth.However,itstatedthatthewidthofkeratinizedtissuedidnotinfluencethesurvivalrateofdentalimplants,andthereisnoevidencetorecommendaspecifictechniquetopreserve/augmentkeratinized tissue;furthermore,factorsincluding bone level, keratinized tissue and implant features have not been shown to be associated with future mucosal recession around dental implants. [28,29]

A number of additional factors have been associated with peri-implantitis in case reports or preclinical researchas: implant placement, cement excess, systemic conditions (cardiovascular disease or rheumatoid arthritis),genetic factors,iatrogenic factor, occlusal overloads or titanium particles.But, at the time being, the available evidence doesnot allow an evaluation of the role of these factors in the pathogenesis of peri-implant diseases.[2,28,30]

IV. Diagnosis of peri-implantitis

The diagnosis of peri-implantitis becomes challenging under the absence of baseline information and fails to generalize these criterions to all available implant systems, this is why the majority of diagnostic methods conventionally used in periodontics have been adopted by clinicians and researchers to diagnose peri-implant diseases as well as to assess the health status of peri-implant tissues. These methods include clinical, radiographic and laboratory examinations.

Probing depth (Pd)

Periodontal probing is a common basic diagnostic tool in periodontal diagnosis around teeth. Ericsson and Lindhehad described distinct differences between teeth and implants in soft tissue composition, organization and attachment between the gingiva and the root surface on one hand and between the peri-implant mucosa and the implant surface on the other[31].

Therefore, this affects the interpretation of probing, by rigid plastic probe,depth measurements. In healthy tissue, the probe penetration is more advanced around implantsalthough this is depending on the probing force. Soft tissue around implants has also been found thicker than around teeth.[32]The soft tissue cuff that surrounded a toothvaried between 2 mm at flat surfaces and 4 mm at proximal surfaces, while at implant sites, the mucosa at proximal as well as flat surfaces was 1–1.5 mm greater. The probing depth (PD) was greater at proximal than at facial or palatal/lingual surfaces at tooth sites andfrequently also at implant sites. Furthermore, the PD and the soft tissue thickness were greater at implant than at adjacent tooth sites.[30-32]

Bleeding on probing (BOP)

Bleeding on probing is used in periodontal diagnosis. However, it has been shown that it is a poor predictor of disease progression, but the absence of BoP is a good predictor of future tissue stability.

Mobility

Implant mobility is an indication of lack of osseointegration, but it is of no use in diagnosingearly implant disease, rather it shows the final stages of de- integration. Periotest can be used to assess the stability of an implant.[30,32]

Bone loss

Although the threshold for bone loss as a diagnostic criterion for disease is not exactly specified in the previous EFP or EAO consensus meetings, there is agreement on the fact that stable crestal bone levels are most important for implant success because it is paramount for long-term survival, esthetics, as well as peri-implant health.[30]

Today, there is a general consensus that a baseline radiograph is required for the assessment of bone changes over time, andit isadvised that critical bone loss ≥2 mm from the time of placement of the prosthetic device, in combination with bleeding on probing, should be interpreted as a “red flag” for the clinician to

critically evaluate whether any intervention is indicated in the individual case and whether follow-up and reassessment are required to confirm ongoing bone loss. [33,34]

V. Management of peri-implantitis

Bacterial biofilm on the surface of implant playsan important role in the appearance of peri-implantitis. This is why the management of peri-implantitisis focused on infection and bacterial controls to stop progression ofboneloss. This treatment is based onthe evidence gained from the treatment of periodontitis.Hence, in 2004, Lang et al. gave consensus statements and recommendations for clinical procedures regarding implant survival and complications.[35]

Both surgical and non-surgical techniques have been developedand have shown promising results in stopping peri-implant bone loss and preventing implant loss. These results depend on the access to the contaminated implant surface and the effectiveness of biofilm removal from implant surfaces during treatment. However, management of peri-implant diseases still remains unpredictable for full reconstruction of lost tissues and completely stops disease progression.[35,36]

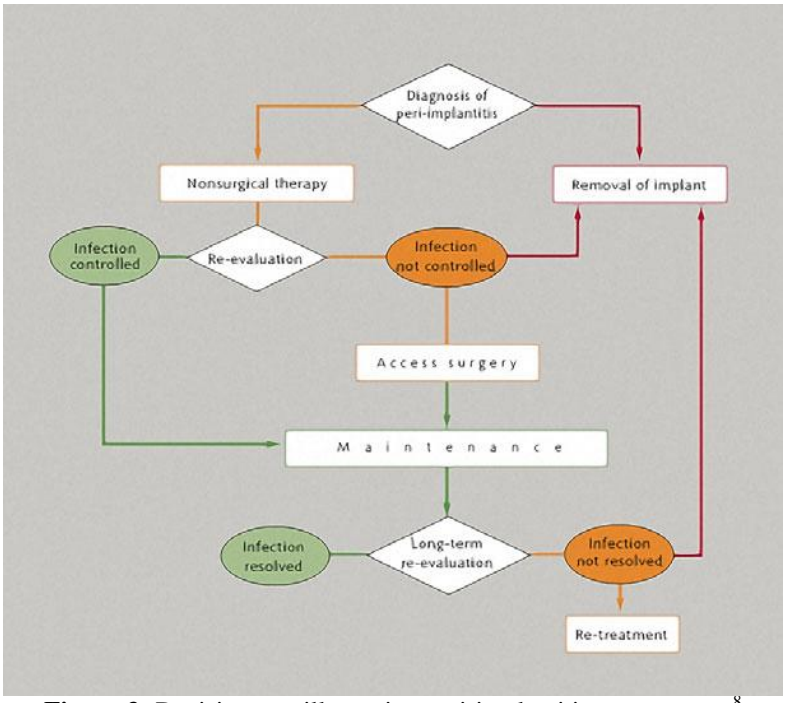

Figure 3: Decision tree illustrating peri-implantitis management[8]

Non- surgical techniques

Mechanical debridement

Non-surgical methods of biofilm removal in the supra-mucosal area around implants together with a comprehensive information and instruction on self-performed infection control procedures are fundamental in the treatment of peri-implant diseases. Numerous tools are available for supra and sub gingival biofilm removal aspolishing brushes,rubber polisher,plastic, carbon or titanium curettes;ultrasonic tips or air powder flows.[31,37]

Surface debridement constitutes the basic element for treatment of peri-implant mucositis and peri- implantitis. However, the design of the implant may hinder the mechanical treatment of the infected surface. Reduction of the bacterial load to a level allowing healing isdifficult to accomplish with mechanical technics alone. Therefore, some additional adjunctive therapies such as antibiotics and laser treatments have been proposed to improve the results.[37]

Adjunctive antimicrobials

Antiseptics

Schwarz et al. showed that addition of antiseptic therapy (chlorhexidine0.2%) to mechanical debridement does improved BOP and peri- implant probing PD.[38]However, the study by Renvertet al. showed that addition of antiseptic therapy to mechanical debridement does not provide adjunctive benefits in shallow peri- implant lesions with mean pocketPD <4 mm but seems to provide additional clinical improvements in deep peri- implant lesions with mean pocket PD >5 mm.[39]

Antibiotics

A number of different local antimicrobials have been used over the years, such as tetracycline-containing fibers, a slow-release doxycycline-containing gel or minocycline microspheres. The adjunctive useof a slow-release doxycycline-containing preparation was evaluated in a controlled study in whichthe supra-structure was removed before nonsurgical therapy, including mechanical cleaning and irrigation with 0.2% chlorhexidine. It was concluded that the local application of this antimicrobial significantlyimproved the results.[40]From a clinical perspective, this combined therapy may serve as an alternative therapy in cases where access is difficult or the patient is not suitable for a surgical intervention.[8]

Regarding systemic antimicrobial treatment, there is a lack of controlled studies evaluating theirefficiencyon peri-implantitis.Data from case series suggest clinical improvements following a combination ofmechanical and antimicrobial treatments.[41,42]However, caution should be exercised wheninterpreting these results in light of the observation that the case series include both local irrigation with antimicrobials and systemic administration of antimicrobials.[41,42]

Local or systemic antibiotics are an additional therapy option. In combination with peri-implant debridement it results in more efficient reductions of clinical peri-implantitis symptoms [37]. In no wayadministration of antibiotics should bea treatment option by itself.[37]

Photodynamic therapy

Photodynamic therapy generates bactericidal effects against aerobic and anaerobic bacteria such as Aggregatibacteractinomycetemcomitans, Porphyromonasgingivalis, Prevotella intermedia, Streptococcus mutans and Enterococcus faecalis. However,Bassetti et al.,after manual debridement by titanium curettes and glycine air powder treatment, half of the patients received adjunctive photodynamic therapy and the other half received minocycline microspheres into implant pockets. After 12 months, the number of periopathogenic bacteria and level of IL- 1s decreased significantly in both groups without significant differences between them.[43]

Laser

Laser therapy has also been suggested as a nonsurgical approach for decontaminating the implant surfaces deep in the peri-implant pocket. Various types of lasers have been used: neodymium- doped: yttrium aluminium garnet, Erbium: yttrium aluminium garnet (Er: YAG), $CO_2$and Diode laser with variable results.[8]Their use could offer an advantage over traditional mechanical treatment as they have a bactericidal effect. Muthukuruet al.evaluated the efficacy and safety of nonsurgical treatment of peri- implantitis suggested that submucosal debridement with adjunctive local delivery of antibiotics, submucosal glycine powder air polishing or Er: YAG laser treatment may reduce clinical signs of peri- implant mucosal inflammation to a greater extent relative to submucosal debridement using curettes with adjunctive irrigation with chlorhexidine.[44]Study by Mettrauxet al.ofnon-surgical mechanical therapy with adjunctive repeated application of a diode laser, yielded significant clinical improvements after an observation period of at least 2 years. [45]These results highlight the clinical benefits of laser application in conjunction with non-surgical mechanical therapy.
Non-surgical treatment could improve significantly clinical parameters but in considerable lesions (larger than 5 mm),wherebacterial pathogens are not reduced, this approach may be insufficient. In that casesurgical access therapy is required.[8]

Surgical techniques

When peri-implant probing depth and bone loss is advanced or persistant, despite the initial non-surgical treatment provided, a surgical intervention of peri-implantitis is required[17]. The major objective for this treatment is to provide access for removal of the bacterialbiofilm, granulation tissue and calcified deposits from the implant surface in order to allow healing and reduce the disease progression**.**[8]
When surgical intervention is necessary,various approaches are proposed;

Access flap surgery and implant decontamination

The main objective of this therapy is to stopthe progression of the disease, to achieve a maintainable site by the patientand to create a theoretically compatible surface for re-osseointegration.

Following removalof a full-thickness flap, mechanical decontamination to remove plaque and mineralized deposits from the implant surface should be performed and, for this task, instruments made of pure titanium are recommended.[8,46]The use of a titanium rotary brush makes this procedure easier than use of conventional curettes(Fig. 4).[8,46].Airborne-particle abrasion devices have also been recommended for the decontamination of implant surfaces during surgery but because of the risk of developing subcutaneous emphysema, care must be taken during their use.[47]

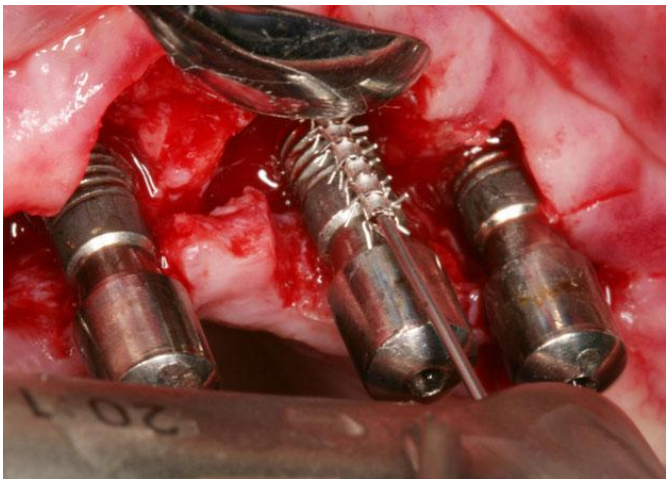

Figure 4: Use of titanium rotarybrush for implant decontamination.[8]

In general, mechanical decontamination should be followed by application of chemical agents onto the exposed surface of the affected implants. In this respect, the substances that have been recommended are hydrogen peroxide, citric acid, sodium chloride, chloramines, tetracycline hydrochloride and chlorhexidine gluconate. From the evidence available, no single method has been proven superior.[48]

Owing to its availability, efficiency and safety, hydrogen peroxide applied on the implant surface for 2 min has been the most widely used substance for chemical decontamination. However, irrespective of the agent used, the implant and the peri-implant wound area should be thoroughly rinsed with a sterile solution following decontamination.[8,48]

A number of other methods for decontamination,such as the use of lasers or abrasive devices, and implantoplasty of the exposed part of the implant, have been suggested as adjuncts to surgical approach but the clinical improvements reported when using these techniques are limited and the evidence is weak. Currently, it is difficult to conclude whatadditional benefitthese technics provide to surgical acces.[8,17,45,48]

Peri- implant resective surgery

In analogy to periodontitis, resective surgery has been shown to be effective in reduction of BOP, probing depths and clinical signs of inflammation. The basic principles include the elimination of the peri-implant osseous defect using ostectomy and osteoplasty.It is indicated in moderate to severe horizontal bone loss, moderate (<3 mm) vertical bone defects (1 and 2 wall bone defects), and to reduce the overall pocket depth and implant position in the unesthetic area where exposure of the titanium components is not a major complication.[15,49]

Additionally, cleaning and polishing of the supra-crestal implant surface (implantoplasty) may be applied.[30]

Guided bone regeneration

Regenerative approaches can be used in conjunction with surgical access, drawing upon the paradigms used in managing periodontal defects associated with teeth. Containable bony defects—walled defects associated with the implant—should be more amenable by grafting to favorable gains in bone fill and reductionsin probing depth. Debridement and implant surface decontamination are still essential prior to regenerative treatment,as the infection must be completely eliminated before application of bone graft.[8,15,50]Patient risk factors, such as smoking, poorly controlled diabetes and unsatisfactory oral hygiene may hamper the successof peri-implant defect regeneration. At present, heterogeneity of study designs andstudy quality prevent strong conclusions on the efficacy of regeneration in treatingperi-implantitis.[51]

The decision for the appropriate regenerative technique is usually based on the morphology of the defect and the degree of bone loss. In the presence of a crater-like four-wall bony defect or a three-wall defect, regenerative techniques are recommended and the use of autogenous bone or bone substitutes can be used to obtain bone fill. A resorbable membrane can also be used in combination with the above-mentioned grafting materials. For two-wall defects, regenerative procedures are usually not indicated as the morphology of the alveolar bone does not allow the grafting material to be properly maintained in the required area.[8,51]

Still, there is promise in surgical studies demonstrating the maintenance of probing depth improvements or bone fill at almost three years out.[15,52]Notably in these studies, implant maintenance was a crucial component ofperi-implantitis treatment, which presumably played a part in achieving the aforementioned results.Most recently, Chan and colleagues conducted a systematicreview and meta-analysison theoutcomeefficacy of surgical treatments ofperi-implantitis.While the use of grafting and membranes tended to

yield greater improvements in probing depth and defect fill, however they conclude that complete fill of the bony defect using guided bone regeneration (GBR) seems not to be a predictable outcome.The authors cite the need for higher quality and longer-term investigations.[8,53,54]

Explantation

When nonsurgical and surgical therapies are ineffective,the simplest and most gentle form of treatment isexplantation by unscrewing against the insertion direction.

Indications include suppurative exudate, overt BOP,severely increased peri-implant PD (≥8 mm), peri- implantradiolucency which may be extending along the outline ofthe implant (>half length) and mobility.[30]

VI. Prevention of peri-implantitis

While long-term data on surgical treatment of peri-implantitis are encouraging, there are problems remaining to be resolved. that, call for a strong focus on preventive strategies. These strategiesshould be a main priority in implant dentistry. As stated in the consensus report from the 11thEuropean Workshop on Periodontology, a continuum exists from healthy peri-implant mucosa to peri-implant mucositis and to peri-implantitis.[3,9]

Thus, prevention of peri-implantitis includes the prevention of peri-implant mucositis and treatment of existing peri-implant mucositis in order to prevent the conversion from peri-implant mucositis into peri-implantitis.[3]Elements in the prevention of peri-implant diseases are thorough information and instruction in self-performed oral hygiene measures around implants and a personalized follow-up supportive therapy program taking into account the specific needs and potential risk factors / risk indicators presented by the individual patient. There is strong evidence of an increased risk of developing peri-implantitis in patients who have a history of severe periodontitis, poor plaque control and no regular maintenance care after implant therapy. Data identifying smoking and diabetes as potential risk indicators for peri-implantitisare, however, inconclusive.[2,7]The recall visits should include a clinical examination and, when indicated, a radiological examination to detect peri-implant diseases. It is necessary to probe peri-implant tissues to assess the presence of bleeding on probing and to monitor probing depth changes and mucosal margin migration. It is recommended that the clinician obtain baseline radiographic and probing measurements following the completion of the implant-supported therapy.[2]

VII. Conclusion

The use of dental implants over past decades has increased exponentially and is considered as a viable treatment alternative for partially and completely edentulous patients given its high predictability and success.Despite of these encouraging data, Peri-implantitisis considered one of the most common biological complication that may be encountered. Unfortunately,given the complex histopathological appearance and the dramatic nature of disease progression in peri-implantitis, the treatmentofperi-implantitisisextremely challengingand difficult. Until now, no**"**ideal treatment protocol" has been shown effective;Indeed, while early attempts of treatment of peri-implantitis performed 20-25 years ago mainly focused on reconstructive procedures of the osseous defect resulting from the disease, the main goal of the treatment was often overlooked; prevention and early interception of etiology and contributing factors associated with peri-implant disease should be emphasized.

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