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Update peri-implantitis – peri-implant inflammation and peri-implant disease

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1 Methods

Purpose

This guideline aims to provide dental and orofacial implantologists with recommendations for recognizing potential biological complications and initiating the treatment required for the respective condition. It is an update of the 2015 guideline.

Introduction

This consensus paper covers only screw-type titanium implants, typically placed in accordance with the indications recommended by the Implantology Consensus Conference (German).
All consensus recommendations in this paper should be considered as guidelines only. The patient's specific situation is always an important consideration and may justify a deviation from the recommendations of this consensus paper.

Background to consensus development

Biological complications are observed as early or late complications and require diagnostic and therapeutic experience on the part of the treatment provider if a progression of the pathological processes is to be prevented.

Literature search

The Cochrane Library, EMBASE, DIMDI and Medline databases were used in the literature search. The search strategy included selected search terms such as

peri-implantitis, peri-implant mucositis and biological complication, **dental implant.**

The abstracts of the resulting literature were then reviewed. Literature not considered relevant was identified and eliminated at that point. The full text of all (potentially) relevant citations was obtained if necessary and reviewed.

The number of papers with the topic peri-implantitis increased each year up to 368 including 89 reviews in 2019 many of which are case reports. But still only 6 RCT were publish between 2010 and 2019. Due to the high number of available review papers, also reviews on reviews are published stating the limited clinical relevance of the reviews ***[29, 42]***.

2 Definition

Peri-implantitis or peri-implant disease is defined as an inflammatory pathological process that affects the soft and/or hard tissue surrounding osseointegrated implants.

Pathogenesis:

- Mucositis is the initial, reversible condition manifesting as inflammation of the soft tissue surrounding the implant, with reddening, hyperplasia and bleeding ***[45]***.
- Peri-implantitis is the advanced, currently irreversible condition with bone resorption, loss of osseointegrated contact area, probable pockets, suppuration and inflammation of the peri-implant connective tissue, which can lead to reduced bone-to-implant contact ***[95]***.

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- A special case is apical inflammation in patients with a history of endodontic treatment and/or periapical granuloma or burned bone syndrome, so-called retrograde peri-implantitis **[*53, 66, 67, 79*]**

Reports on the prevalence of mucositis or peri-implantitis vary widely (1% to 80%) **[*12, 35, 71, 99*]**. One meta-analysis revealed prevalence ranges of 19% to 65% for mucositis and 1% to 47% for peri-implantitis **[*23*]**. Based on the data of 29 papers a prevalence for the occurrence of peri-implantitis was calculated with 18.5% at the patient level and 12.8% implant level **[*81*]**. Patients with the history of periodontitis may have a higher risk of peri-implantitis according to the treatment and maintenance parameters **[*30, 82*]**.

It can be concluded that the initial stage of mucositis is more frequently reported.

3 Risk factors

3.1 General risk factors for the development of peri-implant disease

- Habits (especially pathological bruxism, poor oral hygiene and smoking) **[*64, 73, 97*]**
- Prevalence is higher in patients susceptible to periodontitis than in those in good periodontal health. The placement of implants in patients with untreated periodontitis is contraindicated **[*15, 30, 61, 64, 80, 82, 85, 96, 106*]**
- Systemic diseases and pharmacological interventions (e.g. diabetes mellitus, metabolic syndrome, low-vitamin D-level, high cholesterol level or bisphosphonate therapy, chemotherapy, osteoporosis, immunosuppression, radiotherapy, cardiovascular diseases) **[*56, 64, 69, 73, 77, 84, 98, 107, 108*]**
- The pathological role of human herpes viruses (HHVs) (Epstein-Barr virus [EBV], Human cytomegalovirus [CMV], and Herpes simplex virus [HSV]) in peri-implant disease is a subject of current research and may have role in development of peri-implant disease **[*1, 8*]**

Advanced biological age by itself does not increase the risk of peri-implantitis.

3.2 Local risk factors

Biological quality of the available bone **[*3, 12, 90*]**

- Non-augmented bone is associated with the best prognosis
  - Lower risk in the maxilla than in the mandible
- Bone volume (dimension of buccal plate)
- Bone quality
  - Caution is required in the event of poorly vascularized bone
- Augmentation technique increases risk in the following order, starting with lower risk technique as listed below
  - Vascularized augmentation (distraction, bone splitting, LeFort I)
  - Free autologous augmentation (lateral, vertical)
  - Allogeneic (GBR techniques)
  - Synthetic and xenogeneic (GBR techniques)

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Biological quality of the gingiva

- The presence of less than 2 mm of keratinized and/or fixed soft tissue seems to be associated with a higher risk of peri-implant disease for implants with rough surface **[*3, 9, 12, 32, 38, 72, 94*]**
- Thin biotype seems to be associated with a higher risk of peri-implant disease **[*46*]**

Implant design

There is currently no evidence to suggest that tapered implants are associated with a higher risk of peri-implantitis than cylindrical implants **[*50*]**. One RCT in private practice demonstrate that implants with micro-grooves at the collar show less bone loss **[*39*]**. Different studies on platform switching have revealed heterogeneous results; therefore, no conclusions can be drawn as regards the risk of peri-implantitis **[*11, 88, 113*]**. There is no evidence that the abutment connection has an influence on the peri-implantitis risk **[*59*]**.

Implant surface

Various articles state conflicting data that rough surfaces do increase the risk of peri-implantitis when compared with smooth surfaces **[*24, 49, 92*]** In general, there is no compelling evidence that moderately rough surfaces have an increased risk of peri-implantitis **[*113*]**.

Titanium particles

Actual reviews make the presence of titanium particles in the peri-implant tissue as a subject of discussion as cause of peri-implant disease **[*21, 34, 70, 74, 75, 78, 101*]**. It seems that implants with platform switch show less tribocorrosion **[*4*]**.

Surgical technique

The surgical implantation procedure may damage the tissue surrounding the implant and predispose the patient to peri-implantitis.

- Thermal injury to bone
- Mechanical trauma (excessive compression of healthy bone)
- Poor soft- and hard-tissue management
- Malposition of the implant (vertically, horizontally, axially)

Prosthetics

The type of prosthetic, the various associated treatment procedures and the resulting functional loading are potential risks.

- Malposition of the superstructure relative to the soft-tissue level
- Poor hygienic access
- Poor subgingival cementation technique
- Static stress due to prosthetic misfit
- Micromovement of the abutment and/or superstructure (e.g. screw loosening, cement failure)

Overloading is an additional risk factor for the development of peri-implantitis **[*36*]**. In general there is no increased risk for either screw or cement retained superstructures **[*18*]**.

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4 Prevention

Careful case selection to avoid inadequate soft and hard tissue and an excess of systemic risk factors **[*7*]**.
A patient specific recall schedule and maintenance program should be implemented since mucositis is mainly biofilm induced and the preliminary stage of peri-implantiits **[*16, 62, 87, 89*]**.

5 Microbiology

There is no evidence that the microbial environment around an implant that exhibits signs of peri-implantitis is similar or differs to that found around teeth with periodontal disease**[*86*]**. However, additional bacteria not typically connected with periodontal disease and with a high affinity for titanium surfaces, such as *Staphylococcus aureus* or anaerobic gram-negative periopathogens, including P. gingivalis and T. forsythia, can be found **[*2, 43*]**.

Peri-implant infections exhibit periodontal pathogens, and a very large number of patients have infections resistant to at least one antibiotic **[*83*]**. Tetracycline resistance seems more pronounced than resistance to beta-lactam preparations **[*58*]**.

6 Diagnosis

To assess the peri-implant bone level, radiological documentation is necessary following implant placement, osseointegration and placement of the prosthetic restoration **[*60*]**.

Patients should be informed of any potential pathological changes around the implant that they can identify themselves, such as bleeding, soft-tissue changes or swelling.
Identifying the disease requires a careful clinical examination that follows the principles of periodontology, with the risk of false positive data**[*41*]**:

- Bleeding on probing
- Careful probing of peri-implant pockets on four sides (0.2 N probing force)
- Where signs exist: radiological follow-up using dental X-ray

Due to beam-hardening artefacts, the use of high-resolution CBCT is not indicated for diagnosing peri-implant bone destruction **[*22, 47*]**. However, defects >0.5 mm have been successfully diagnosed using CBCT **[*31, 37*]**.

Analysis and identification of potential causes

Evidence of inflammatory mediators in the sulcular fluid of implants with peri-implantitis is considered as biomarker for the condition **[*5, 14, 111*]**. The detections of biomarkers may enable a differentiation between early and late stages of peri-implantitis**[*65*]**. However, no evidence of biomarker reduction has been found following successful treatment **[*110*]**.

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7 Treatment

Treatment is aimed at reducing acute symptoms and preventing progression and recurrence **[*44*]**. An actual meta-analysis shows no superiority between the currently available treatment modalities to reduce clinical signs of peri-implantitis **[*17*]**.

General recommendations in implants for maintained stability

Conservative approach for decontaminating the implant surface

- Starting treatment as early as possible is essential, ideally in the **initial** stages
- Mechanical cleaning
- Local disinfection
- Reduction of deep pockets and/or hyperplasia
- Augmentation of vertical bone defects in some cases
- Frequent patient recall, 3–4 times a year

Depending on the findings, closed conservative treatment or surgical treatment – if necessary, with defect reconstruction – is recommended. In addition to mechanical debridement, various techniques can be used to decontaminate the infected tissue and disinfect the implant surface; recontamination cannot be avoided. Various meta-analyses and RCTs have drawn different conclusions regarding the therapeutic relevance of the procedures listed below.

7.1 Peri-implant disease due to excess cement

Non surgical removal of residual methacrylate cement may lead to healing of peri-implant bone defects, if reseating is done with a resorbable zinc-oxide eugenol cement **[*19*]**.

7.2 Peri-implant mucositis

A meta-analysis lists the optimization of oral hygiene and additional disinfection with air polishing, CHX rinses, ultrasonic debridement, periodontal treatment, manual debridement using curettes, manual cleaning plus local delivery of CHX and photodynamic therapy as effective treatments for mucositis **[*28, 91, 104*]**. There is no evidence that one type of curette-material is superior to another **[*93*]**.

Photodynamic therapy is a multifactorial treatment procedure **[*102*]**. PDT has shown to be as effective as local antibiotic therapy **[*6*]**. Utilizing PDT improves the outcome of peri-implant disease parameters **[*33, 109*]**. Some system modifications available for photodynamic therapy have limited support in the literature for peri-implantitis therapy **[*25*]**.

Conflicting data it available for adjunctive laser therapy for surgical and non-surgical peri-implantitis treatment **[*26, 57, 63*]**.

Meta-analysis suggests that systemic antibiotic adjuvant therapy is not indicated **[*40, 48*]**.

In cases of lack of keratinized/fixed peri-implant mucosa, soft tissue thickening with connective tissue graft may improve peri-implant health **[*10, 103*]**.

7.3 Peri-implantitis

In advanced peri-implantitis, surgical procedures are more likely than closed procedures to improve probing depths and attachment levels **[*27, 54*]**. In surgical treatment

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approaches, the additional use of laser therapy **[*57, 68, 76, 112*]** (RCT and meta-analysis) or chlorhexidine applications has not been shown to improve long-term success **[*20*]**.

7.3.1 Non augmentative procedure

Surgical debridement, implantoloplasty, local decontamination and apical reposition flap outwith the aesthetic area has been shown to get positive results in case of horizontal bone loss.

7.3.2 Augmentative procedure

In cases of vertical peri-implant bone loss an augmentative procedure may improve clinical findings Various materials are used for defect augmentation in addition to autologous bone. The use of membranes when augmenting defects may improve results **[*13*]**. No conclusive statement on the effectiveness of the grafting materials, membranes can be made **[*17, 51, 52, 55, 100, 105*]**. Success will be improved by achieving submerged healing in combination with implant surface decontamination and defect grafting **[*55*]**.

8 Therapeutic success

It is important to identify and eliminate all possible causes in susceptible patients. Due to the reversible nature of mucositis, diagnosis and early intervention will help to reduce the occurrence of peri-implant disease.

The treatment outcome is considered less predictable in peri-implantitis than in periodontal disease, but results may be improve by plaque control, post-operative maintenance and non-smoking patients. Currently, the goal is to reduce the signs and symptoms of inflammation and to avoid progression. A frequent recall scheme is essential.

Cologne, 22 February 2020

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