Peri-implant inflammation: Prevention – Diagnosis – Therapy

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Guideline

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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 2008 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 AND 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. Numerous reviews, but few RCTs (randomized controlled trials) or other systematic clinical trials are available on this topic.

2. Definition
Peri-implantitis 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.
- Peri-implantitis is the advanced, currently irreversible condition with bone resorption, loss of osseointegrated contact area, probable pockets, suppuration and inflammation, which can lead to reduced bone-to-implant contact.
- 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 [30, 35].

Reports on the prevalence of mucositis or peri-implantitis vary widely (1% to 80%) [6, 32, 46]. One meta-analysis revealed prevalence ranges of 19% to 65% for mucositis and 1% to 47% for peri-implantitis [12]. Another meta-analysis showed that patients with periodontal disease are at significantly higher risk of peri-implantitis [37]. 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-implantitis
- Habits (especially bruxism, poor oral hygiene and smoking) [29, 33, 44].
- Prevalence is higher in patients susceptible to periodontitis than in those in good periodontal health. The placement of implants in patients with active periodontitis is contraindicated [8, 28, 29, 36, 37, 40, 43].
- Systemic diseases and pharmacological interventions (e.g. diabetes mellitus, bisphosphonate therapy, chemotherapy, osteoporosis, immunosuppression, radiotherapy, cardiovascular diseases) [29, 33, 39, 45].

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

Biological quality of the available bone [1, 6]
- 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
  - Vascularized augmentation (distraction, bone splitting, LeFort I)
  - Free-flap autologous augmentation (lateral, vertical)
  - Allogeneic and xenogeneic (GBR techniques)

Biological quality of the gingiva [1, 5, 6]
- Availability of keratinized gingiva
- Phenotype of gingiva

Implant design
There is currently no evidence to suggest that tapered implants are associated with a higher risk of peri-implantitis than cylindrical implants. Different studies on platform switching have revealed heterogeneous results; therefore, no conclusions can be drawn as regards the risk of peri-implantitis [41]. There is no evidence that the abutment connection has an influence on the peri-implantitis risk.

Implant surface
One study suggested that rough surfaces increase the risk of peri-implantitis when compared with smooth surfaces [13]. In general, there is no compelling evidence that moderately rough surfaces have an increased risk of peri-implantitis.

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-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 [18]. In general there is no increased risk for either screw or cement retained superstructures [9].
4. Prevention

Careful case selection to avoid inadequate soft and hard tissue and an excess of systemic risk factors[4]. Minimally traumatizing procedures and specific recall schedules.

5. Microbiology

The microbial environment around an implant that exhibits signs of peri-implantitis is similar to that found around teeth with periodontal disease. However, additional bacteria not typically in connection with periodontal disease and with a high affinity for titanium surfaces, such as *Staphylococcus aureus*, can also be found [21]. Peri-implant infections exhibit periodontal pathogens, and a very large number of patients have infections resistant to at least one antibiotic [38]. Tetracycline resistance seems more pronounced than resistance to beta-lactam preparations [26].

6. Diagnosis

To assess the peri-implant bone level, radiological documentation is recommended following implant placement, osseointegration and placement of the prosthetic restoration [27]. Evidence of inflammatory mediators in the sulcular fluid of implants with peri-implantitis is considered a biomarker for the condition [2]. However, no evidence of biomarker reduction has been found following successful treatment [48].

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 may require a careful clinical examination that follows the principles of periodontology, although precise evidence is lacking:

- 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 [11]. However, defects >0.5 mm have been successfully diagnosed using CBCT [17, 19].

- Analysis and identification of potential causes

7. Treatment

Treatment is aimed at reducing acute symptoms and preventing progression and recurrence. There is no evidence that treatment provides long-term stability or regression of the disease [22].

General recommendations for implants with maintained stability:

Conservative approach for decontaminating the implant surface

- Starting treatment as early as possible is essential, ideally in the initial stages
- Mechanical cleaning/smoothing
- 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; various meta-analyses and RCTs have drawn different conclusions regarding the therapeutic relevance of the procedures listed below.

7.1 Peri-implant mucositis
A recent 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 [16, 42, 47]. There is no evidence that one type of curette-material is superior to another. Photodynamic therapy was shown to be as effective as local antibiotic therapy [3]. Some system modifications available for photodynamic therapy have limited support in the literature for peri-implantitis therapy [14]. There is no evidence that laser therapy is effective in initial peri-implantitis [25]. Meta-analysis suggests that systemic antibiotic adjuvant therapy is not indicated [20, 23].

7.2 Surgical treatment
In advanced peri-implantitis, surgical procedures are more likely than closed procedures to improve probing depths and attachment levels [15]. The use of membranes when augmenting defects may improve results [7]. Various materials are used for defect augmentation in addition to autologous bone. No conclusive statement on the effectiveness of the materials can be made [24]. In surgical treatment approaches, the additional use of laser therapy [25, 31, 34, 49] (RCT and meta-analysis) or chlorhexidine applications has not been shown to improve long-term success [10].

8. Therapeutic success
The treatment outcome is considered less predictable in peri-implantitis than in periodontal disease. Currently, the goal is to reduce the signs and symptoms of inflammation and to avoid progression. It is important to identify and eliminate all possible causes; in susceptible patients, a close recall scheme is essential.

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References


