Peri-implant diseases: The etiology, clinical features, diagnosis and treatment

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Abstract
Rehabilitation of missing teeth by root form endosseus dental implants has revolutionized the treatment of partially and fully edentulous patients. Due to improved implant materials, armamentarium, and surgical techniques implants have got a very high success and survival rate. At the same time, the implants are associated with failures by peri-implant diseases due to various reasons related to improper diagnosis and treatment plan, and poor maintenance.

Keywords: Dental implant, Peri-implant mucositis, Peri-implantitis, Prevention.

Introduction
Man has tried to replace a mutilated tooth by a foreign material since long. Implant therapy has undoubtedly been a very successful alternative to restore functions in areas where teeth are missing. Though excellent long-term results are often presented, biological complications may occur in the form of soft tissue inflammation with or without bone loss around the implant, and pose a threat to long-term implant survival (Fig.1).¹,²

Fig. 1: Periimplant mucositis. Arrow indicates the area of involvement

The 6th European Workshop in 2008 introduced a term peri-implant mucositis where gingival coronal tissues, ie, free-gingiva, and interdental gingiva, are first affected without involvement of alveolar bone.³ If not taken proper maintenance care at this stage of peri-implant mucositis, this disease entity leads to peri-implantitis with definite loss of alveolar bone and it is discernable through radiograph. Both of these two disease entities are the result of the imbalance of bacterial load and host defensive mechanism.⁵

Definition
The term peri-implantitis was introduced by Mombelli et al. (1992), who in a study on the microbiota at implants with and without bone loss concluded that "peri-implantitis can be regarded as a site specific infection which yields many features in common with chronic periodontitis".⁵

At the first European Workshop on Periodontology, Peri-implant mucositis was defined as reversible inflammatory changes of the peri-implant soft tissues without any bone loss. Peri-implantitis was defined as an inflammatory process affecting the tissues around an osseointegrated implant in function, resulting in loss of supporting bone.⁶ Periimplantitis can be classified into early, moderate and advanced (Table 1).⁷

Prevalence
Koldsland et al. (2010) showed that the prevalence of periimplantitis ranged from 11 to 47% on 109 patients with implant treatment.⁸ According to Fransson et al. (2010), after the first year of function, there were bone losses of ≥ 2 mm (32%) and ≥ 3 mm (10%) around infected implant. The severity of peri-implantitis associated with bone loss increases in proportion to the function time of the implant.⁹

Normal periodontium and peri-implant mucosa
The hard and soft tissues surrounding an osseointegrated implant show some similarities to the periodontium around natural dentition. The gingiva around dental implant is called peri-implant mucosa, and consists of well-keratinized oral epithelium, sulcular epithelium and junctional epithelium with underlying connective tissue.

Between the implant surface and epithelial cells are hemi-desmosomes and the basal lamina. Implants lack periodontal ligament. The collagen fibres are unattached and parallel to the implant surface rather than in functional contact from the bone to the cementum. The titanium screw attaches directly to the alveolar bone which is in direct and tight contact with the implant surface. The differences between dento-gingival and implant-gingival junction are shown in Table 2.
Table 1: Classification of peri-implantitis (Forum et al. 2012)\(^7\)

| Early | PD≥4mm (bleeding and/or suppuration on probing)  
Bone loss <25% of the implant length |
|-------|-----------------------------------------------|
| Moderate | PD≥6mm (bleeding and/or suppuration on probing)  
Bone loss 25% to 50% of the implant length |
| Advanced | PD≥8mm (bleeding and/or suppuration on probing)  
Bone loss > 50% of the implant length |

Table 2: The differences between dento-gingival junction and implant-gingival junction

<table>
<thead>
<tr>
<th></th>
<th>Tooth –gingival junction</th>
<th>Implant –mucosal junction</th>
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<tbody>
<tr>
<td>Gingival fibers</td>
<td>perpendicular to long axis of tooth</td>
<td>parallel to long axis</td>
</tr>
<tr>
<td>fibroblast-fiber ratio</td>
<td>more fibroblasts</td>
<td>Altered</td>
</tr>
<tr>
<td></td>
<td>less collagen</td>
<td></td>
</tr>
<tr>
<td>Vascularity</td>
<td>more</td>
<td>less</td>
</tr>
<tr>
<td>biologic width</td>
<td>strong</td>
<td>Weak</td>
</tr>
<tr>
<td>p d l</td>
<td>present</td>
<td>Absent</td>
</tr>
<tr>
<td>tactile sensation</td>
<td>Present</td>
<td>Absent</td>
</tr>
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Etiopathogenesis

Due to the reduced vascularisation and parallel orientation of the collagen fibres, peri-implant tissues are more susceptible for inflammatory disease than periodontal tissues. This phenomenon can be verified immunohistochemically through increased formation of inflammatory infiltrate, nitric oxide, lymphocytes, leukocytes.

An accumulation of microbes in plaque at the peri-implant or mucosal margin causes a local inflammation, which is a complex reaction of the body in response to infectious agents. Inflammatory cells such as macrophages, neutrophils, lymphocytes and plasma cells, provoke considerable tissue damage. The degradation of connective tissue are followed by bone destruction, which marks the borderline between peri-implant mucositis and peri-implantitis.

Two primary etiologic factors in peri-implant marginal bone loss:
1. Bacterial infection
2. Biomechanical overload

Bacterial infection

Healthy peri-implant sulcus is characterized by high proportions of cocci, a low ratio of anaerobic/aerobic species, a low number of gram-negative species and low detection frequencies for bacteria associated with periodontitis.

Quirynen et al. (2002) studied early microbial colonization of the “pristine” periimplant sulcus and reported that a complex microbiota was established within a week after abutment insertion\(^10\). Peri-implantitis is a poly-microbial anaerobic infection which includes following micro-organisms: Prevotella intermedia, Prevotella nigrescens, Streptococcus constellatus, Aggregatibacter actinomyctemcomitans, Porphyromonas gingivalis, Treponema denticola and Tannerella forsythia.

Biomechanical Overload

Bone loss at the coronal aspect of implants can result from biomechanical overloading and the resultant micro fractures at the coronal aspect of the implant-bone interface. The loss of osseointegration in this region results in apical down growth of epithelium and connective tissue. The speed and degree of loss of implant-bone contact depends upon the frequency and magnitude of the occlusal loading as well as superimposed bacterial invasion. The role of over loading is likely to increase in four clinical situations:
1. Poor quality bone.
2. The implant's position
3. The patient has a pattern of heavy occlusal function associated with parafunction.
4. The prosthetic superstructure does not fit the implants precisely.

Risk factors

1. Smoking with additional significantly higher risk of complications in the presence of a positive combined IL-1 genotype polymorphism.
2. History of periodontitis.
3. Poor oral hygiene
4. Systemic diseases (e.g. diabetes mellitus, cardiovascular disease, immunosuppression.)
5. Iatrogenic causes (e.g. "cementitis").
6. Soft tissue defects or poor-quality soft tissue at the area of implantation (e.g. lack of keratinized gingiva).
8. Alcohol consumption
9. Implant surfaces
Diabetes
1. A study by Ferreira et al. (2006) showed that patients with diabetes (fasting blood sugar ≥ 126 mg/dl or had been taking anti-diabetic medicine over the past 2 week) were more prone than those without diabetes to develop peri-implantitis.11
2. The presence of periodontitis and diabetes were statistically associated with greater risk of peri-implantitis.
3. The poor metabolic control in subjects with diabetes was associated with peri-implantitis.

Alcohol consumption
Only one study by Galindo-Moreno et al. (2015) concluded that peri-implant marginal bone loss was statistically linked to alcohol consumption >10 gm. per day and that alcohol induced more serious peri-implantitis than cigarette. Alcohol consumption is associated with deficiencies of the complement system, alteration of the neutrophil function and modulating T-associated with deficiencies of the complement system, affect bone destruction and reduces bone formation.12

Implant surfaces
1. Most recent titanium implants with a rough surface showed more favourable osseointegration than those with smooth surfaces.
2. However, a rough surface also favoured the formation and retention of dental plaque.
3. Roughness increased both the adhesive surface of bacteria and the difficulty in cleaning the implant.
4. In a study by Astrand et al. (2004) ITI system dental implants (Straumann and Waldenburg, Switzerland) with a plasma-sprayed surface had a statistically higher incidence of peri-implantitis than Branemark system implants (Nobel Biocare, Gothenburg, Sweden) with a smooth surface.13
5. In a review by Renvert et al. (2011) there was no evidence that implant surface characteristics can have a significant effect on the initiation of peri-implantitis.14

Diagnosis
Peri-implantitis can be diagnosed by following features;
1. Bleeding on probing (Fig. 2 )
2. Peri-implant crevicular fluid
3. Microbial testing
4. Radiographic evaluation (Fig. 3a,3b)
5. Suppuration (Fig. 4)
6. Mobility

Peri-implant probing
Standardized plastic probes should be recommended for peri-implant probing. Presence of bleeding on gentle probing (0.25 N) is a useful parameter for diagnosis of mucosal inflammation (Fig. 2). Experimental study showed that healthy peri-implant sites had absence of BOP while there was increased BOP in mucositis (67%) and peri-implantitis (91%).15 Penetration of the probe up to 1.6 mm. into the connective tissue occurred in the peri-implantitis lesion.16

Peri-implant crevicular fluid (PICF) analysis
Levels of biochemical mediators secreted into the PICF have been studied with the aim of identifying a diagnostic marker to monitor peri-implant health. Markers in PICF including cytokines such as IL-1β, IL-6, IL-8, IL-17 and TNF α, enzymes, and proteases have been investigated.17

Microbial testing
A microbiological test of sub-gingival microflora using a bacterial culture, checkerboard DNA-DNA hybridization, polymerase chain reaction (PCR), monoclonal antibody and enzyme assays could suggest antibiotic therapy.

Radiographic evaluation
Radiographic techniques including intra-oral periapical radiography (OPIA) and panoramic tomography (OPG) (Fig. 3a, 3b) using long cone parallelising techniques have been widely used to monitor marginal bone levels at implants and to diagnose interproximal bone loss. Here the distance from a fixed reference point (e.g. implant shoulder or implant–abutment junction) to the inter-proximal bone level is recorded at baseline and monitored longitudinally. Multi-slice computer tomography (CT) and cone beam volume imaging have been used in implant dentistry offering the advantage that osseous structures can be represented in three planes, true to scale and without overlay or distortion.
Fig. 3: Peri implant bone loss: a. IOPA radiograph; b: OPG

Experimental study in pig mandibles evaluated the accuracy and quality of the representation of prepared peri-implant defects by intraoral radiography, panoramic radiography, CT and cone beam radiography. Both CT and cone beam imaging techniques provided accurate three-dimensional representations of the peri-implant bone defects. The CT scans showed a slight artefact immediately adjacent to the implant while the cone beam scans showed the better imaging quality.18

Suppuration

Suppuration is one of the positive objective sign of periimplantitis (Fig. 4).19 In a report of 218 patients who were examined for biological complications at existing implants 9-14 years after implant placement, the presence of pus was identified as explanatory for periimplantitis resulting in apical level of bony defect at or apical to the third thread which is ≥3.1 mm apical to implant shoulder.20

Table 3: AKUT concept

<table>
<thead>
<tr>
<th>Stage</th>
<th>Result</th>
<th>Therapy</th>
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<tbody>
<tr>
<td>Normal</td>
<td>Pocket dept (PD) &lt;3 mm., no plaque or bleeding</td>
<td>No therapy</td>
</tr>
<tr>
<td>A</td>
<td>PD &lt;3 mm., plaque and/or bleeding on probing</td>
<td>Mechanically cleaning, polishing oral hygiene instructions.</td>
</tr>
<tr>
<td>B</td>
<td>PD 4-5 mm., radiologically no bone loss</td>
<td>Mechanically cleaning, polishing, oral hygienic instructions plus local antiinfective therapy (e.g CHX).</td>
</tr>
<tr>
<td>C</td>
<td>PD &gt;5 mm., radiologically bone loss &lt;2 mm.</td>
<td>Mechanically cleaning, polishing, microbiological test, local and systemic antiinfective therapy.</td>
</tr>
<tr>
<td>D</td>
<td>PD &gt; 5 mm., radiologically bone loss ≥2 mm.</td>
<td>Resective or regenerative surgery.</td>
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Nonsurgical therapy
1. Local debridement
2. Implant surface decontamination
3. Anti-infective therapy
4. Laser-assisted treatment of peri-implantitis
5. Photodynamic therapy

Surgical therapy
1. Resective surgery
2. Regenerative surgery

Non surgical therapy
Local debridement

The implant should be cleaned by instruments softer than titanium, such as polishing with a rubber cup and paste, floss, interdental brushes, or using plastic scaling instruments, teflon, carbon, plastic and titanium curettes (Figs. 5a,b).22

1. These have been shown not to roughen the implant surface unlike metal and ultrasonic scalers.
2. Karring et al. (2015) demonstrated that submucosal debridement alone, using either with an ultrasonic device or carbon fiber curettes, is not sufficient for the decontamination of the surfaces of implants with peri-implant pockets ≥ 5 mm and exposed implant threads.\textsuperscript{23}

3. So it has been suggested that mechanical or ultrasonic debridement alone may not be an adequate modality for the resolution of peri-implantitis.


**Fig. 5: Local debridement: a) Carbon fiber curettes; b) Plastic scaler**

**Implant surface decontamination**

1. There are several methods for implant surface decontamination, such as citric acid, photosensitizing treatment, stannous fluoride, tetracycline-HCl, chlorhexidine gluconate, hydrogen peroxide, sterile water, a plastic sonic scaler tip and an air powder abrasive unit.

2. An abrasive air polishing medium can modify the surface of implants.

3. After air powder treatment cell attachment and cell viability still showed sufficient levels, but cell response was decreased compared with sterile surfaces.

**Anti-infective therapy**

1. Antiseptic rinses in relation to different parameters.

2. Application of systemic and locally delivered antibiotics in relation to pocket depth or different parameters.

3. Schwarz et al. (2006) demonstrated that the treatment of peri-implant infection by mechanical debridement with plastic curettes combined with antiseptic (0.2% chlorhexidine) therapy may lead to statistically significant improvements in bleeding on probing, peri-implant probing pocket depth, and clinical attachment level at 6 months compared with baseline.\textsuperscript{24}

4. Renvert et al. (2008) showed that combined mechanical treatment with a repeated local application of minocycline microspheres (Arestin, OraPharma, Warminster, PA, USA) after 30 and 90 days and showed benefits in the therapy of peri-implantitis. This study also indicated that mechanical treatment combined with the local application of an antibiotic achieved a better result, but in addition of antiseptic therapy to mechanical debridement does not provide adjunctive benefits in shallow peri-implant lesions where the mean probing pocket depth was ≤ 4 mm.\textsuperscript{25}

5. Astasov-Frauenhoffer et al. (2014) found complete growth-inhibiting effects of amoxicillin and metronidazole on Streptococcus sanguinis, Porphyromonas gingivalis and Fusobacterium nucleatum apart from each other, but the combination was found to be more efficient than metronidazole alone.\textsuperscript{26}

**Laser-assisted treatment of peri-implantitis**

1. A study by Schwarz et al. (2006) on the efficiency of the ErYAG laser showed the improvement of bleeding on probing was greater in laser treatment than that of using a mechanical treatment (plastic curettes) combined with an antiseptic agent (0.2% chlorhexidine digluconate) in the non-surgical therapy of peri-implantitis.\textsuperscript{27}

2. Sajjad et al. (2014) proposed different laser systems with bactericidal effects, tissue ablation and detoxification for the therapy of periimplantitis. Erbium-doped yttrium, aluminum and garnet (ErYAG) laser has the ability to remove dental plaque and calculus on the smooth or porous surface of implants without causing alterations. The ErYAG laser also showed reduction of the peri-implant pocket and attachment gain after 6 months.\textsuperscript{28}

**Photodynamic therapy**

1. Bombecari et al. (2013) stated in a study after manual debridement by titanium curettes and glycerine 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-1\(\beta\) decreased significantly in both groups without significant differences between them.\textsuperscript{29}

2. Deppe et al. (2013) stated that photodynamic therapy generates reactive oxygen species by multiplicity with help of a high-energy single-frequency light (e.g. diode lasers) in combination with photosensitizers (e.g. toluidine blue). In a wave length range of 580 to 1400 nm and toluidine blue-concentrations between 10 and 50 \(\mu\)g/ml, photodynamic therapy generates bactericidal effects against aerobic and anaerobic bacteria (Aa, P. gingivalis, P. intermedia, Streptococcus mutans, Enterococcus faecalis).\textsuperscript{30}

**Surgical therapy**

1. The surgical therapy combines the concepts of the already mentioned non-surgical therapy with those of resective and/or regenerative procedures.

2. Classification of the morphology of peri-implant lesions was important for choosing a reliable type
of bone regeneration and for prognosis of surgical therapy of periimplantitis.

A predictable bone growth in human around failing implant was demonstrated for the first time by Fletcher et al. (2018).31

**Classification of peri-implant bone defects** (Schwarz et al. 2007)12

A. Class I consisted of infrabony destruction.

B. Class II characterized by horizontal bone loss.

Class I subdivided into:

1. Class Ia (buccal dehiscence)
2. Class Ib (buccal dehiscence + semicircular bone resorption to the middle of the implant body)
3. Class Ic (buccal dehiscence + circular bone resorption under maintenance of the lingual compacta)
4. Class Id (buccal dehiscence + circular bone resorption under loss of the lingual compacta)
5. Class Ie (circular bone resorption under maintenance of the buccal and oral compacta).

The bone defects most frequently found in peri-implantitis are class Ie (55.3%) > class Ib (15.8%) > class Ic (13.3%) > class Id (10.2%) > class la (5.4%).

The application of bone regeneration seems to be more favourable in class I bone destruction, but it is very limited in class II defects.

The best results for reducing pocket and clinical attachment gain are found in class Ie defects.

**Resective therapy**

1. Peri-implantitis with suprabony destruction, one-wall infrabony defects or buccal dehiscences in non-aesthetic regions, the resective surgery including osteotomy or osteoplasty associated with the polishing of the transmucosal implant part and apically positioned flap has been suggested.

2. The goals of resective surgery are the reduction of the peri-implant pocket and the morphological reconstruction of soft tissue to promote patients' oral-implant hygiene.

3. A clinical study by Romeo et al. (2005) demonstrated that resective therapy associated with implantoplasty improved the survival of infected implants.33

4. Implantoplasty is also a form of surface decontamination as it involves eliminating the implant threads to achieve a smooth polished surface to decontaminate and reduce the ability of plaque to adhere to the implant surface, but four concerns exist:
   i. Heat production
   ii. Deposit of implant material into the surgical field
   iii. Damage to the implant surface
   iv. Weakening of the implant structure.

   Heat production is easily managed by effective irrigation and appropriate bur selection.

In a dog study, titanium debris from implantoplasty produced a histological inflammatory cell infiltrate in adjacent tissue but the debris was very minor, undetectable via computed tomography by Schwartz et al. (2011). It has been suggested that these depositions are not associated with any adverse events clinically.34

**Regenerative therapy**

1. Resective surgical therapy may result in re-osseointegration in only minor superficial defects. From functional, aesthetic and long-time-survival point of views, full regeneration and re-osseointegration is aspired (Fig. 6).

2. A study of autogenous bone graft by Behneke et al. (2000) on 17 patients with 25 treated implants showed a reduction of the peri-implant pocket from 6.9 to 0.7 mm (P = 0.001), corresponding to 90% bone reconstruction and Median marginal bone loss was reduced from 6.2 to 2.3 mm after 2 and 3 years, respectively. The median vertical bone resorption of 4.5 mm was completely repaired.35

3. The application of a membrane barrier gave protection from blood clots and created a space around peri-implant defects to promote bone regeneration as well as to avoid competition from other tissues.

4. Collagen membranes were recommended for their convenient properties like hemostatic function, early stabilization, chemotactic activity attracting fibroblasts and semi-permeability.

5. In a recent prospective case series by Matarasso et al. (2014), a combined resective and regenerative approach including a bovine bone mineral and a collagen membrane infracrestally and implantoplasty supracrestally showed a significant peri-implant probing depth reduction and an increased radiographic defect fill after 12 months of follow-up.36

Fig. 6: Surgical Correction: a. Pre-operative radiograph; b. Placement of bone graft material; c. Post-operative follow-up radiograph

**Laser-assisted surgical treatment of peri-implantitis**
The contamination of the implant surface limited the success of this bone regeneration procedure. The efficiency of bacterial reduction by lasers has been confirmed by several studies. The effect of coagulation by laser permitting good stabilization of blood clots and good contact of substitutive materials with the implant surface promoted re-osseointegration. The clinical cases reported by Haas et al. in 2000 using combined methods (curettage + laser assisted decontamination of implant surfaces + autogenous bone graft + ePTFE membrane + systemic antibiotic therapy for 5 days) showed great results for peri-implantitis treatment with a 36.4% bone gain after 10 months.37-40

The new classification of peri-implant diseases and conditions

A new classification scheme for peri-implant diseases and conditions has been recently suggested in World Workshop held at Chicago on November 9 to 11, 2017 under the aegis of American Academy of Periodontology (AAP) and European Federation of Periodontology (EFP). This is published in Journal of Clinical Periodontology in its March issue of 2018 as a supplement by ten authors. It is stated that there are four terminologies to be used for designating different varieties of status for peri-implant diseases and conditions around an implant. These are: peri-implant health, peri-implant mucosities, peri-implantitis, and peri-implant hard and soft tissue deficiencies.

Conclusion

Prognosis of the affected implant will be contingent upon early detection and treatment of peri-implant mucositis and peri-implantitis. Differentiating peri-implantitis with and without pus formation is an important criterion that influences the outcome of nonsurgical and surgical therapy.

References


