<s>Restorative Dentistry

<t>Peri-Implant Disease Part 1: Diagnosis and Assessment Parameters

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<a/c>**Abstract**: Where suitable, dental implants are praised as a method of providing fixed solutions with the greatest longevity, and providing greatly improved retention for removable prostheses, resulting in increased levels of patient satisfaction and quality of life.

However, with increasing evidence of long-term follow-up, there is a growing recognition of the susceptibility of dental implants to peri-implant diseases; peri-mucositis and peri-implantitis. This paper discusses the features of peri-implant disease and important aspects of assessment criteria.

<a/c>**CPD/Clinical Relevance:** This paper highlights the importance of supportive maintenance care for patients with dental implants, as well as the features and assessment of peri-implant disease.

<b/f>Patient demand for dental implants as tooth replacements has increased rapidly over the decades and there are now a wide range of centres providing implant treatments. Dental implants are subject to failure, however, and dental practitioners should be prepared to monitor dental implants for disease and failure such that appropriate management can be implemented. This paper will present an overview of peri-implant diseases including peri-implant mucositis and peri-implantitis and assessment criteria based on current guidelines and evidence.

<ch1/1>Mucosa at teeth and implants in health

The peri-implant mucosa provides an important biological barrier which protects the rigid fixation of the implant to the bone from factors released from plaque

and the oral environment. This soft tissue attachment to the coronal portion of an implant is necessary for the maintenance of osseointegration and long term survival of dental implants.

Clinically, peri-implant health is characterized by the absence of erythema, bleeding on probing, swelling, and suppuration.[1] In health there are no visual differences between peri-implant and periodontal tissues, however, histologically a number of distinctions can be made (Figure 1)DELETE. The peri-implant mucosa is lined by keratinized oral epithelium on the outer aspect, which is continuous with the sulcular epithelium, and a junctional epithelium which attaches to the implant surface by hemi-desmosomal attachment. The junctional epithelium is approximately 2 mm long. The underlying connective tissue is collagen rich, with fewer vascular structures and fibroblasts than that in its periodontal counterpart. The peri-implant tissues lack a periodontal ligament, and derive blood supply from the supra periosteal vessels only. Collagen fibres tend to run in a direction parallel to the implant surface and are said to offer less resistance to irritation and inflammatory effects than that around natural teeth.[2,3]

The microflora that establishes in the healthy peri-implant sulcus is comparable to that found on adjacent teeth in health. Once an abutment is connected to the implant, salivary proteins and other substances soon form a pellicle similar to that on natural teeth. The microbial communities that colonize the site are characterized by gram-positive cocci and small numbers of gram-negative species.[4] In health, there exists a homeostasis between the peri-implant tissues and the microbial biofilm, comparable to that of the gingival sulcus in health (Table 1).

Peri-implant Mucosa	Periodontium
Direct bone to implant contact (osseointegration)	Presence of cementum and periodontal ligament
More collagen fibres, fewer fibroblasts, less vascular	More fibroblasts and more vascular
Connective tissue fibres run parallel to implant surface without insertion, creating a 'cuff' around the implant	Multiple orientations of connective tissue fibres, insertion of fibres into root cementum

Table 1. Comparison of peri-implant mucosa with physiological periodontium

<ch1/1>Peri-implant diseases

Peri-implant diseases include peri-implant mucositis and peri-implantitis. These diseases have been likened to gingivitis and periodontitis, respectively, in that peri-mucositis represents a reversible, contained inflammation around the dental implant whereas peri-implantitis is associated with the irreversible loss of attachment and surrounding alveolar bone. The prevalence of peri-implantitis is reported to affect 10% of implants and 20% of patients over a minimum of 5 years,[5] but might range from 6.6%–36.6% of implants and 11.2%– 47.1% of patients.[6] The prevalence of peri-implant mucositis is higher than that of peri-implantitis; occurring in about 50% of implants and just under 80% of patients.[7] Each will be discussed in more detail below.

<ch1/1>Peri-implant mucositis

Biofilm accumulation in the peri-implant sulcus can disrupt the host–microbe homeostasis, resulting in a shift towards microbial communities which are inclined to favour and contribute towards inflammatory conditions, resulting clinically in a lesion known as peri-implant mucositis.[6,8]

Features of peri-implant mucositis include erythema and bleeding on probing, with or without increased pocket depths, but without any loss of supporting

bone. Clinically, in terms of presentation, diagnosis and treatment, it is comparable to chronic gingivitis and is reversible.[9] On a biological level, the immune-inflammatory response has been suggested to be more intense in peri-implant mucositis than gingivitis, with greater increases in matrix metalloproteinase 8 and interleukin-1β in the crevicular fluid from implants, as compared with that from teeth,[10] although the size of the inflammatory infiltrate does not appear to be significantly different.[11]

There is some suggestion that a higher prevalence of peri-implant mucositis is associated with patients with mucosal diseases such as oral lichen planus and gingival desquamation, however, it is difficult to say whether this is due to the conditions or higher plaque scores that might be associated with them[12–15] as evidence remains limited.

Because such lesions are considered a precursor for peri-implantitis, the clinical implication is that peri-implant mucositis should be prevented and managed through optimal biofilm removal and addressing risk factors where possible.[12,16,17]

<ch1/1>Peri-implantitis

In susceptible patients, where peri-implant mucositis lesions are persistent, the inflammatory lesion can become extensive. A large inflammatory infiltrate consisting of plasma cells, macrophages and neutrophils[1,18] starts to spread in an apical direction, beyond the junctional epithelium and connective tissue to involve the peri-implant crestal bone.[19] The host response mediates bone resorption and loss of clinical attachment. Once clinical attachment loss is detectable, these lesions are then termed peri-implantitis.

According to the 2017 World Workshop,[1] the diagnosis of peri-implantitis requires:

- Presence of bleeding and/or suppuration on gentle probing;

- Increased probing depth compared to previous examinations (probing depths of ≥6 mm in the absence of previous data);
- Presence of bone loss beyond crestal bone level changes resulting from initial bone remodelling (bone loss ≥3 mm in the absence of previous data).

In a similar fashion to periodontal disease, increasing depths of peri-implant pockets are associated with increasingly dysbiotic biofilms[20] in which the occurrence and frequency of periodontal pathogens, such as *Porphyromonas gingivalis* and *Aggregatibacter actinomycetemcomitans*, are similar to those in periodontal disease. However, peri-implantitis lesions also harbour bacteria distinct from the microbiota typically associated with periodontal disease, such as *Campylobacter*, *Eubacterium*, *Prevotella*, *Campylobacter*, *Treponema* and *Staphylococcus*.[10,21] Resistance to at least one antimicrobial substance has been reported in numerous peri-implant biofilms.[22,23] In particular, *Staphylococcus aureus* appears to play a predominant role for the development of a peri-implantitis, showing a high affinity to titanium and being closely linked to deep pockets with suppuration and bleeding on probing.[24,25]

In comparison to periodontal lesions, peri-implantitis shows greater numbers of inflammatory mediators and increased osteoclasts.[1] It seems that the pathology associated with peri-implantitis tends to progress more rapidly and in a non-linear pattern.[19,26] In part, the explanation might be found with the anatomical differences between peri-implant and periodontal tissues.[27] In periodontitis, a connective tissue capsule is present between the base of the inflammatory lesion and the bone, but is absent in peri-implantitis.[19,27] It may be that this tissue barrier serves to limit the apical extent of the lesion to some degree. Furthermore, the progression of the lesion may also be affected by differences in vascularity. The absence of a periodontal ligament in implant lesions means that vascular supply is comparatively diminished in the zone between the alveolar crest and the base of the junctional epithelium. It is likely that the

flow of infiltrating immune cells and growth factors to the lesion would be slow compared to periodontitis cases.[2,6] The roughness of the implant surface might also be a contributing factor towards the progression of the lesion, rougher surfaces being more plaque retentive and establishing a biofilm which is more difficult to disturb.[28]

‹ch1/1›Foreign body reaction

It has been suggested that a chronic foreign body reaction is inevitable around dental implants.[29] Whereas the periodontal complex is the result of evolution, the soft-tissue barrier around implants is akin to an induced scar tissue response. Albrektsson *et al* consider osseointegration itself to be the result of a foreign body reaction, which results in osseous encapsulation to shield off the implant from the tissues. This results in direct bone-implant contact and the establishment of a dense zone of bone surrounding the implant which has less vasculature relatively compared to surrounding bone. They suggested that, in health, an equilibrium exists between the hard and soft tissues and the implant foreign body.[29] The term osseoseparation[30] has been used to describe marginal bone loss caused predominantly by immune osteoclastic activity following disturbance of an equilibrium between the host and implant 'foreign body', rather than being initiated by an infective or inflammatory process.[31] This foreign body response might act in combination with inflammation or superimposing infection to define the final distance that bone is resorbed from around the implant, so that what is resorbed may be predominantly, if not exclusively, the foreign body bone, whereas resorption of the properly vascularized host bone is potentially lacking.

‹ch1/1›Risk factors

A range of factors have been suggested as risk indicators for periodontal disease, with varying levels of supporting evidence. There is stronger evidence that there is an increased risk of developing peri-implantitis in patients who

have a history of chronic periodontitis, poor plaque control skills, and no regular maintenance care after implant therapy.[18]

There is some limited evidence linking peri-implantitis to other factors, such as post-restorative presence of submucosal cement, lack of peri-implant keratinized mucosa, and positioning of implants that make it difficult to perform oral hygiene and maintenance.[18]

It is suggested that peri-implant keratinized mucosa, occlusal overload, titanium particles, bone compression necrosis, overheating, micromotion and biocorrosion may be associated with the development of peri-implant disease, but strong evidence for these is lacking.[1]

Risk factors include:

- Plaque accumulation; and
- Host susceptibility.

<ch2/1>Plaque accumulation

- Poor oral hygiene;
- Non-enrolment on supportive maintenance programmes;
- Non-keratinized peri-implant tissue;
- Extrusion of excess cement;
- Implants placed in close proximity making cleaning difficult;
- Poorly designed prosthesis – not allowing access for plaque control;
- Rough implant surface – increased risk of plaque accumulation if exposed;
- Xerostomia.

<ch2/1>Host susceptibility

- Previous or current history of periodontally involved teeth;
- Genetic susceptibility;
- Uncontrolled Diabetes;
- Previous or current history of smoking;
- Radiotherapy;

- Medications affecting bone metabolism;
- Presence of autoimmune oral disease;
- Thin gingival biotypes;
- Poor bone quality;
- Parafunction and bruxism.

<ch2/1>Assessment THIS IS A HEADING SAME AS RISK FACTORS <ch1/1>?

Early signs of peri-implant disease can be difficult to detect.[32] The importance of regular supporting maintenance appointments in preventing and managing peri-implant disease has been well reported in the literature.[33–35] Sustained and repeated contact with patients enables clinicians to modify and reinforce oral hygiene, provide prophylactic care and recognize signs of developing disease so that treatment can be initiated at an early stage.[36] Assessment should focus on detecting the presence or absence of plaque accumulation, bleeding, suppuration, and clinical or radiographic bone loss.

<ch2/1>Plaque accumulation

There is a significant dose-dependent association between plaque scores and peri-implant disease[37,38] and should always be evaluated.[39] Plaque control around implants is difficult to achieve.[38] The prosthetic structure must be designed to allow access for oral hygiene at the implant site. Failure to do this may predispose to plaque accumulation and peri-implant disease.[40] This problem can be minimized by constructing a suprastructure that makes it easy for the patient to perform oral hygiene. In some instances, it may be necessary to modify or replace existing prosthetic supra-structures to allow access for cleaning.[41] Provisional prostheses are useful in trialling designs and oral hygiene in addition to the prerequisite for impeccable oral hygiene prior to implant planning. Whenever possible, margins of implant-supported prostheses should be placed at or above the peri-implant mucosal margin to facilitate access for biofilm control. The intracoronal compartments of screw-retained fixed

restorations, internal implant cavities, and the microgap at the abutment-implant interface are heavily contaminated.[42] Rough surfaces collect more biofilm than smooth surfaces and are more difficult to clean once exposed.[28] Extruded cement in the subcrevicular peri-implant space can be challenging to clean up and remove. It will act as a nidus for biofilm accumulation and result in an inflammatory lesions.[43] Therefore, only a small amount of radio-opaque cement has been suggested to be used, placed around the rim of the prosthesis, which might then be seated on an abutment replica to remove any large excess prior to cementation. Limiting the gingival extent of the crown to no more than 1.5 mm beyond the gingival margin may provide some protection;[3,6] restorations should be cemented on individualized abutments allowing proper cement removal(12)

The amount of keratinized tissue may negatively influence treatment outcomes and the patient's ability to maintain adequate plaque control due to discomfort when performing oral hygiene.[44] Sites with less than 2 mm thickness of keratinized mucosa might be more disposed to plaque accumulation therfore.[45] However, the presence of keratinized mucosa to maintain peri-implant health does not seem to be necessarily essential in patients who are able to maintain adequate plaque control and are enrolled in long-term regular supportive therapy.[46]

Mechanical biofilm control by patient and professional must be considered during implant planning and prosthesis design. Oral hygiene measures must be individually adapted to patients, evaluated and reinforced through attendance on a regular supportive review programme.

‹ch2/1›Peri-implant probing depth

In the periodontium, probing is met with resistance from the connective tissues at the base of the sulcus in health, which prevents the probe from reaching the apical portion of the epithelial tissue. In disease, the resistance is reduced and

the probe extends further to the base of the cell infiltrate. Less resistance exists to probing the peri-implant sulcus in health due to the orientation of connective tissue fibres and lack of insertion into the implant or abutment surface.[47] In disease, greater penetration of the peri-implant tissues can be expected and the probe may reach the alveolar crest.[48] Use of consistent, light forces (0.25 N) have been advised but is challenging to produce clinically. Probing error is greater around implants with peri-implant disease than in health and when compared to probing conditions around teeth but, despite this, recent working groups have advocated peri-implant probing.[49]

Peri-implant probing should be performed to determine the location of the base of a pocket relative to a known and documented fixed landmark. Conventional probing does not appear to damage the peri-implant tissues or implant surface.[50] Although usually between 2–4 mm, the depth of placement, mucosal biotype, as well as presence and contour of the prosthetic reconstruction, may increase probing depth readings around dental implants.[51] The finding of a probing depth of 5 mm or more should therefore be further assessed, however, this cannot be seen as a sign of pathology when taken alone, but rather must be used in combination with other assessment parameters. Evidence of increasing probing depth compared to previous findings is a more convincing measure of loss of attachment and bone loss than a single reading on one occasion.

‹ch2/1›Bleeding on probing

Assessment of mucosal inflammation is primarily made by observing bleeding following light probing. Bleeding on probing is thought to be diagnostic of peri-implant mucositis. The presence of bleeding on probing may not indicate the presence of acute inflammation in the peri-implant mucosa,[52] but may be due to the scar tissue–implant interface or forceful probing. As with periodontitis, it is the absence of bleeding on probing which has a high negative predictive value for peri-implant disease, and is a good prognostic indicator.[53]

‹ch2/1›Swelling or suppuration

Swelling and/or suppuration implies the presence of inflammation and infection at the implant site. Suppuration in particular is most likely to occur where there is peri-implant crestal bone loss[18] and has been detected in 30% of the patients or 17% of implants.[52]

‹ch2/1›Mobility

Implant mobility is a concerning finding which suggests complete loss of direct bone-implant contact. Mobility should be checked for all free-standing implants, but routine removal of fixed prosthesis to facilitate this is not advisable, especially in the absence of other findings. Mobility is a terminal clinical sign requiring removal of the implant.[51]

‹ch2/1›Radiographs

The normal bone remodelling processes will result in marginal bone loss following initial loading, in the region of 0.5–2 mm.[22,54] It is recommended that a baseline radiograph is taken after connection of the definitive transmucosal structure to establish the level of the supporting bone.[55] This provides a reference for comparison to radiographs at future assessments should there be clinical suspicion of peri-implant bone loss.[6] Panoramic radiographs are not suitable for this comparison as the image produced is less likely to be consistent. Intra-oral long-cone periapical radiographs are the appropriate choice, which enables measurement of interproximal loss of bone from fixed reference points, such as implant shoulder or implant threads. Vertical bone loss of less than 0.2 mm annually following the implant's first year of service has been proposed as one of the major criteria for success, but this amount is difficult to determine by visual inspection of radiographs in the clinical setting. If no previous data are available, over 3 mm bone loss is considered evidence of peri-implantitis. Radiographic evaluation of crestal bone levels over time is a reliable tool for identifying peri-implantitis.[55] However, radiographs cannot be taken at every

recall visit due to concerns regarding radiation exposure. A sensible protocol would be that radiographs are taken after the first year, and biannually thereafter, extending up to 5 year intervals, or in the presence of clinical signs or symptoms of disease.

‹ch1/1›Summary

The diagnosis of peri-implant diseases require assessment of multiple diagnostic parameters and should be based on clinical signs of inflammatory disease. The rate of progression of peri-implant disease is usually more aggressive and occurs at a more rapid rate than periodontitis. As for patients susceptible to periodontal disease, establishing effective home care with regular supportive professional maintenance appointments is essential for long-term survival.

‹ch1/1›References

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