Peri-Implant Diseases

Abstract: Peri-implant diseases are inflammatory conditions that affect the soft and hard supporting tissues around implant fixtures. Peri-implant mucositis usually responds to oral hygiene instructions, scaling and prophylaxis, but peri-implantitis, which involves bone resorption, has less predictable treatment outcomes following non-surgical management. Adjunctive treatment for decontaminating sites may include the use of antimicrobials and resistant cases may sometimes be managed with a surgical approach.

Clinical Relevance: As dental implant-retained prostheses become more popular the prevalence of peri-implant complications will also increase. Dental practitioners and care professionals should appreciate their potential roles in the management of these conditions.

Dent Update 2010; 37: 511–516

Peri-implant disease

Dental implants, like natural teeth, are susceptible to inflammatory diseases that are predominantly driven by the accumulation of dental plaque. These conditions are categorized into those that are limited to the peri-implant soft tissues (peri-implant mucositis) and those that also affect the alveolar bone support (peri-implantitis).

Peri-implant mucositis

Peri-implant mucositis is a reversible, inflammatory lesion affecting the marginal soft tissues that surround osseointegrated dental implants but does not involve the resorption of the supporting bone (Figure 1a). This condition corresponds to gingivitis around natural teeth and presents as redness and swelling of the soft tissues. Bleeding on probing is the clinical sign that confirms diagnosis (Figure 1b). As with chronic gingivitis, a necrotizing ulcerative condition may develop when risk factors such as smoking are present.

Peri-implantitis

Peri-implantitis is an inflammatory lesion that affects the supporting bone as well as the surrounding soft tissues of a functioning implant. This condition broadly corresponds to periodontitis around the natural teeth. In addition to bleeding on probing, affected sites may exude pus (Figure 1c) and are always accompanied by marginal bone loss (Figure 1d). To distinguish pathological bone resorption from physiological remodelling of the alveolar crest the loss of bone height should involve ≥ 3 threads of the implant fixture (1.8 mm) following the first year in function.

Prevalence

Although there are few long term data on the prevalence of peri-implant
diseases, those data that are available are relatively consistent. Cross-sectional studies of implants that have been in function for 9 years or more suggest that peri-implant mucositis affects approximately 50% of implants and 80% of patients; peri-implantitis, again for implants that have been in function for 9–11 years, affects 28–56% of patients and 12–43% of implant sites.\(^4\)\(^5\)\(^6\)\(^7\)\(^8\)\(^9\)\(^10\)\(^11\) Whilst these data clearly indicate a need for sustained supportive care, it is reassuring to observe that overall survival rate over the same period is around 96%. Of the implants that are lost, the majority fail early, either before placement of the suprastructure, or within the first year of loading.\(^9\)

### Identifying patients at risk

For patients with periodontal diseases it is crucial that associated risk factors are identified, modified or eliminated if the benefit of treatment is to be maximized. The same strategy applies to patients with peri-implant diseases and, although it might be anticipated that risk factors are addressed adequately before the placement of implants, this may not always be the case. Patients who are unable to achieve a good standard of plaque control are at increased risk of peri-implant disease with an odds ratio of 2.9 for peri-implant mucositis and 14.3 for peri-implantitis.\(^10\)\(^11\) and the association between plaque and disease is ‘dose-dependent’. The ability of patients to clean effectively around implant fixture, however, may not be assumed from their ability to achieve a high standard of plaque control around the natural dentition.\(^11\) Therefore careful instruction in plaque control measures around implants in the immediate post-placement period is essential.

There is also an increased prevalence of peri-implantitis in patients with a history of periodontitis when compared to non-periodontitis patients.\(^12\)\(^13\)\(^14\)\(^15\) although the extent to which this might impact on overall implant survival in the long-term is unclear.\(^14\)\(^15\) A history of periodontitis does not preclude the placement of implants, but the disease should be stabilized with high level maintenance to minimize the opportunity for periodontal pathogens from periodontally-active sites to colonize peri-implant tissues.\(^14\)\(^16\) It is important to appreciate, however, that the risk for peri-implant infection is still present even when implants are placed in edentulous patients; an implant will still provide a favourable ecological niche for colonization by commensal oral bacteria.\(^17\) Indeed, a number of bacterial species such as Peptostreptococcus stomatitis, Mycoplasma salivarium and Pseudoraminbicter alactolyticus, that are not linked with healthy implant sites or periodontal disease, have been identified at sites of peri-implantitis.\(^18\)

Smoking is a known risk factor for periodontal diseases and there is a substantial evidence base of long-term studies that implicate the habit as a risk factor for peri-mucositis, peri-implantitis and total implant failure.\(^12\)\(^13\)\(^14\)\(^15\) These associations have been substantiated unequivocally by systematic review and meta-analysis\(^20\)\(^21\) and, whilst there are few data to suggest otherwise, it must be assumed that cigarette smoking will impact negatively on attempts both to prevent and manage peri-implant disease in the long-term.

### Diagnosis of peri-implant disease

#### Clinical assessment

Visual assessment alone will reveal an inflamed gingival cuff, although scar reactions in the soft tissues, for example after bone grafting and tissue manipulation, may mask inflammatory reactions of the mucosa. Probing is essential and may be undertaken circumferentially or at four sites around an implant fixture. A plastic (TPS or WHO 621) probe has greater flexibility than a metal counterpart, may cause less contamination and damage to the implant surface, and is potentially more accurate as it can adapt more readily to the abutment-suprastructure junction (Figure 2). A light probing force of around 0.15–0.2N is recommended; the absence of gingival connective tissue fibres attached to the implant surface means that there is minimal resistance to probing. In the presence of peri-implantitis there is an extensive, unencapsulated neutrophil infiltrate and the periodontal probe is likely to penetrate the entire depth of the

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**Figure 1.** (a) Peri-implant mucositis: gingival inflammation and enlargement around two mandibular implants. (b) Clinical appearance of peri-implantitis with circumferential inflammation around the implant at the LR3 site. (c) Clinical appearance of peri-implantitis with suppuration at the mesial buccal site after gentle probing with TPS plastic probe. Note how the flexibility of the probe allows it to follow closely the contour of the crown and the long axis of the implant fixture. (d) Radiographic appearance of fixture seen in (b). There is extensive bone loss on the mesial and distal surfaces of the fixture. In situ, the bone involvement affected the entire circumference of the fixture.
soft tissues and extend to the level of the alveolar bone.22

Bleeding following gentle probing is the characteristic sign for peri-implant mucositis and suppurative following probing is indicative of peri-implantitis. Probing depth may be influenced by the implant system and the superstructure used: a gingival cuff with a probing depth of 5 mm but with no bleeding on probing may be consistent with health. If initial probing depths are recorded about one month after implant placement, then future measurements, recorded for example on an annual basis, can be compared to the baseline record.

Any mobility within a year of placing an implant and/or its superstructure is more likely to be a sign of lack of osseointegration, fracture of the implant or mobility and fracture of the superstructure rather than an early sign of peri-implantitis. The only reliable method for assessing mobility (other than for a single tooth implant) is to dismantle the superstructure so that the implant fixture(s) can be assessed independently. Late mobility of a successfully integrated implant, however, may be an additional sign of advanced or progressing peri-implantitis.

Radiographic assessment

High quality, long cone periapical radiographs are the films of choice for assessing implant fixtures. The ideal exposure will reveal the external configuration of the implant and the thread profile is likely to be visible unless there is a vertical discrepancy of ≥ 15° from the perpendicular between the beam and the film surface.23 The normal bone density around osseointegrated fixtures was described originally as having perifixture trabeculations radiating from the implant surface24 and radiographic evidence of a bone trough extending apically to the first thread may be consistent with health being a manifestation of bone remodelling and re-establishment of the biologic width of peri-implant tissues. A radiographic observation of bone resorption mesial and distal to the implant of ≥ 3 threads will confirm a diagnosis of peri-implantitis.7 If a ‘baseline’ radiograph is taken at the time of placement of the superstructure, then further films can be exposed at 6 and 12 months at the time of greatest remodelling of bone and when complications are most prevalent, and then at 2-3 year intervals thereafter. More frequent exposures on an annual basis are indicated when there are persistent clinical signs of peri-implant disease and when there is an increased risk of complications, such as when implants are placed in a patient with a history of periodontal disease. It must, however, always be remembered that this type of radiography is purely two dimensional and therefore gives no indication as to any potential buccal bone loss that may have occurred.

Treatment of peri-implant disease

Comprehensive reviews25,26 of the treatment of peri-implant diseases have made important conclusions:

- That mechanical, non-surgical therapy may be effective in the treatment of peri-implant mucositis and that the adjunctive use of antimicrobial mouthrinses may enhance the clinical outcomes;
- That non-surgical therapy for peri-implantitis is not a predictable treatment even with adjunctive chlorhexidine mouthrinse. Adjunctive local or systemic antibiotics, however, may be effective in reducing probing depths and resolving bleeding on probing;
- Adjunctive Listerine mouthrinse can reduce dental plaque and marginal bleeding and may provide some benefit in the management of peri-implant mucositis.

The relatively superficial nature of peri-implant mucositis is the most likely reason that this condition responds favourably to intensive oral hygiene instruction, scaling, prophylaxis and mouthrinsing with chlorhexidine. As lesions develop, however, their local anatomy, the complex morphology of the exposed fixture and the profile of superstructure-implant fixture design will inevitably compromise the ability of the clinician to debride the site effectively and the ability of the patient to maintain the area free from dental biofilm (Figure 3).27 Mechanical, non-surgical treatment (using specifically designed titanium curettes and the Vector ultrasonic system with tips designed for treatment around dental implants) is effective in reducing dental plaque deposits and soft tissue bleeding, although the effects on probing depths and the pathogenic subgingival microflora are limited.28 Titanium curettes are less likely than conventional instruments to alter or damage the implant surface, but their restricted ability to ‘work’ horizontally and circumferentially between the threads on the part of a fixture exposed through peri-implantitis will limit effectiveness. Similarly,
plastic manual instruments which may be useful for scaling in the supragingival environment may be less effective when attempting to remove deposits from between the threads (Figure 4).

Because of the problem with access to the affected part of the fixture, the Implantic Debrider™ has been recently introduced (Figure 5a). This rotatory instrument cleans down to the titanium surface between the threads of the exposed fixture at 400 rpm with copious water cooling, but the suprastructure must be dismantled to allow access (Figure 5b).

Er-YAG Lasers have also been used for decontaminating peri-implantitis affected implant surfaces, but again, access has proven challenging and the outcome data from relatively small studies have yet to demonstrate significant benefit when compared to conventional mechanical therapy. Ultrasonic instruments with plastic tips (SoftTip™, Dentsply PA, USA) are also conceptually attractive for use on implant surfaces and, although their efficacy in removing deposits from affected surfaces has yet to be determined, there is evidence to suggest that they inflict far less damage on the titanium surface than do conventional metal ultrasonic instruments (Figure 6).

The objective of treatment of peri-implantitis is to reduce or eliminate the bacterial load and so allow healing to occur. When there is evidence of bone cratering and thread exposure, and when mechanical therapy alone may be effective, then adjunctive antimicrobial therapy may be an option. Minocycline microspheres (Arestin®, Orapharma, Johnson and Johnson) adhere to the walls of peri-implant pockets and are retained in high enough concentrations to be effective against biofilm. They appear to be a valuable adjunct to mechanical debridement in patients with peri-implantitis. When peri-implantitis sites appear to be unresponsive to non-surgical management, pockets continue to bleed or suppurate and there is bone loss of ≥2mm (involving 3 or more threads of the implant fixture), then referral to a specialist may be indicated for consideration of a surgical approach to achieve better access for decontamination, and perhaps consider regenerative procedures in the longer term.

**Peri-implant supportive care**

It is crucial that any patient with an implant-retained prosthesis is enrolled into an individually-tailored programme of supportive care, with the emphasis being on a preventive approach to potential peri-implant problems and early diagnosis of developing peri-implant disease. Risk assessment should be ongoing, for example to establish smoking status (has an ex-smoker relapsed?) and the overall maintenance of patients who have a history of periodontal disease. The design of suprastructures should facilitate oral hygiene practices and the use of soft bristle toothbrushes (manual or powered) should be reviewed. Single-tufted, interspace brushes, and interdental tape and brushes with plastic-coated wire hubs are essential for cleaning around fixtures and beneath the suprastructure. Scaling and prophylaxis should be undertaken with plastic manual or ultrasonic instruments with a view to minimizing the deposit of calculus, disrupting and removing biofilm and ensuring an environment that is consistent with peri-implant health.

**Conclusion**

Peri-implant diseases are likely to become more prevalent as the popularity of implant-retained prostheses continues to increase. Prevention is the optimal strategy but early identification of peri-implant complications is essential if inflammation in the supporting soft tissues is to be resolved. Peri-implantitis, however, is a more challenging condition and...
treatment success may be unpredictable. Nevertheless, a conventional, non-surgical approach should be attempted.

References


