PERI-IMPLANTITIS – A PROBLEM.

Infections around implants are stubborn. What can help? What doesn’t? How can infections be prevented?
How prevalent is peri-implant disease?

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Peri-implantitis is such a recent phenomenon that there is still virtually no dependable data on the prevalence of the infection. Estimates put its incidence at around 1% per year.

The question of how frequently peri-implant disease crops up is not easy to answer. To begin with, there is a lack of specially designed epidemiological studies on the topic. As a result we can only infer the number from retrospective cohort studies. Next, studies define peri-implantitis differently, so results cannot always be compared between studies. Third, the frequency of peri-implantitis in a patient group is subject to diverse factors; therefore, the frequency differs by patient group.

Diverse definitions – varying prevalence

The definition of peri-implantitis, of course, plays a crucial role in calculating the prevalence and incidence. Peri-implantitis is such a recent medical condition that it was rarely treated as a biological complication in studies published prior to 2000. Soft tissue lesions were specified in a small number of cases, but not defined, or peri-implantitis was defined according to a few arbitrary radiological bone heights, which were made public after a conference in 1986. Therefore, data originating from earlier studies frequently cannot be used to ascertain the prevalence of peri-implant disease. In addition to bone loss, there is now also probing pocket depth (PPD) as a relevant clinical parameter, especially when the goal is to diagnose peri-implantitis at an early stage. An increasing probing pocket depth is very likely the first indication of the onset of peri-implantitis and suggests the need for a radiographic examination of the state of the bone.

Different studies have defined different probing pocket depth thresholds for diagnosing peri-implantitis. As a rule, a probing pocket depth of ≥5 mm has been taken as a basis for an early indication or Stage 1 peri-implantitis, and a probing pocket depth of ≥6 mm for more advanced peri-implantitis (Stage 2).

Different thresholds for the probing pocket depth inevitably change the recorded prevalence of the disease. For example, in a contemporary study involving a group of 70 patients with treated periodontitis and with implants averaging eight years, it was observed that 22.2% of the implants were affected by Stage 1 peri-implantitis (PPD ≥5 mm) in a high percentage of the patients (38.6%). If the peri-implantitis threshold had been set at a probing pocket depth of ≥6 mm (Stage 2), the peri-implantitis prevalence would have decreased to 8.8% in 17.1% of the patients.

In corollary, this means that peri-implantitis affecting one in twelve implants was diagnosed in one in six patients after an eight year “incubation period”.

Prevalence subject to patient group

Prof. Giovanni Salvi, Switzerland, has listed the risk factors for peri-implantitis in his article (p. 9–11). The presence of these risk factors – e.g., smoking, previous periodontitis, hard-to-clean reconstructions and cement residue from implant-supported crowns – also affects the prevalence of peri-implantitis in a patient group. As an example, residual cement from implant-supported crowns initiated peri-implantitis in 85% of patients prone to periodontitis, whereas prevalence was
only 1.08% in control patients with screw-retained crowns. On the other hand, after removal of residual cement, fiber-optic magnification revealed no further peri-implantitis in 74% of patients. Peri-implant disease correlates strongly with patient susceptibility to periodontal disease. Prevalence in susceptible patients can be influenced by residual periodontal pockets following active periodontal treatment or untreated periodontal pockets.

**Systematic review of prevalence**

For the 3rd EAO Consensus Conference in Pfäffikon, Switzerland – February 2012, a systematic review was undertaken to determine peri-implantitis prevalence and incidence. As the studies included in the analysis were heterogeneous, no meta-analysis could be performed, and no unequivocal, exact and relevant proportion of implants could be calculated following a specific peri-implant disease “incubation period.” The analysis therefore concentrated on describing all the relevant studies, and it was estimated that “five to ten years after implantation, approximately 10% of the implants and 20% of the patients were affected by peri-implantitis.” It needs to be taken into account, however, that this cumulative prevalence of about 1% per year of “incubation” is a very rough estimate subject to the above-mentioned “patient specific” risk factors.

**Estimation of the incidence**

To calculate the assumed incidence of peri-implantitis would necessitate accurately defining an additional peri-implantitis symptom – most likely the loss of bone of ≥2 mm within a specific time period. From the prevalence we can only speculate that the incidence of new cases of peri-implantitis is around 1% per year.

**References**

Infections around teeth and infections around implants have aspects in common. But in comparison with periodontitis, peri-implantitis exhibits various characteristics that make treatment more difficult.

Consensus reports from European Workshops on Periodontology have stated that peri-implant mucositis and peri-implantitis are infectious diseases. Peri-implant mucositis describes an inflammatory lesion that resides in the mucosa, whereas peri-implantitis also affects the supporting bone. In addition, peri-implantitis is characterized by changes in the height of the crestal bone in conjunction with bleeding on probing, with or without concomitant deepening of peri-implant pockets. Pus is a common finding in peri-implantitis sites.

Mucositis vs. Gingivitis

Results from clinical and experimental studies have revealed that peri-implant mucositis and gingivitis have many features in common. Gingivitis and peri-implant mucositis lesions form in gingival and peri-implant connective tissues in response to plaque formation on teeth or implants and are similar in terms of locations, size and composition. Gingivitis and peri-implant mucositis lesions, if left untreated, may progress, become destructive and develop into periodontitis and peri-implantitis lesions, respectively.

More neutrophil granulocytes and osteoclasts

Although there are obvious similarities regarding clinical characteristics and the etiology of peri-implantitis and periodontitis, the two lesions have critical histopathological differences between them. Data from experimental studies and the analysis of human biopsy material have demonstrated that peri-implantitis lesions are poorly encapsulated and extend to the bone. They are larger and extend closer to the bone crest than periodontitis lesions. In addition, peri-implantitis lesions contain larger proportions of neutrophil granulocytes and osteoclasts than periodontitis lesions.

References

Timely peri-implantitis diagnosis

Prof. Giovanni E. Salvi | Switzerland
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In recalls following implant placement, the peri-implant tissue should undergo careful clinical and radiological monitoring so that changes will be promptly noted.

Implant probing plays a key role in diagnosing peri-implant disease, as does a radiological check, in which bone changes should be compared with baseline radiographs from the time of reconstruction.

Probing peri-implant soft tissue

A peridontal probe made of plastic or metal should be used to explore four to six sites around the implant. No probing should be done while the soft tissues are healing following implantation (6–8 weeks). The probing pocket depth should be compared with the baseline following reconstruction. The probing pressure should not exceed 0.2–0.25 N. An increasing probing pocket depth is an alarm requiring further investigation. In the case of implants that are set deeply in the aesthetic zone, 5–6 mm probing depths are possible in the approximal region, even in non-inflamed conditions.

Signs of inflammation and bleeding in response to probing

Clinical changes in the peri-implant mucosa, such as reddening and swelling, should be examined regularly. The absence of bleeding in response to probing is an indication of peri-implant health. A two-year observation period has shown that peri-implantitis progresses
MUCOSITIS
Diagnosed and untreated mucositis is more likely to develop into peri-implantitis than treated mucositis⁷.  
Conclusion: treat mucositis promptly.

SURFACE ROUGHNESS
Implants with a smooth or micro-rough surface show a comparable incidence of peri-implantitis over a 13-year observation period⁸.

PERIO CASE HISTORY
The survival and success rates of implants in patients with previously recorded periodontitis are lower than in patients without periodontal issues⁹.
Conclusion: a check for periodontal infection prior to implantation is highly recommended. Leaving residual pockets > 5 mm with bleeding on probing jeopardizes implant success rate⁹,⁵.

Implant mobility
Implant mobility is an indication of a complete loss of osseointegration, and therefore cannot be used for early diagnosis of peri-implantitis. Implant mobility, when there are no signs of bleeding on probing, increased probing pocket depths, suppuration or crestal bone loss, can indicate improper loading⁴.

SUPPURATION
A purulent secretion with or without formation of fistulae is the consequence of advanced inflammation. Suppuration is therefore also not suited for early diagnosis of peri-implantitis.

REFERENCES

Radiographic images
The radiographic depiction of the implant should always be linked to the clinical diagnosis. Intraoral dental imaging, orthopantomography (OPT) and, for special indications, digital volume tomography have been shown to be successful in radiographic diagnosis. The distance should be measured from a fixed reference point, for example the implant shoulder, to the crestal bone. The bone level at the time of reconstruction serves as a radiological reference (baseline).

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Suppuration
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if bleeding on probing occurs in more than half of the follow-up sessions¹.
Risk factors for peri-implantitis

Prof. Giovanni E. Salvi | Switzerland

**SUPPORT**
The 10-year survival and success rates of implants in patients with treated periodontitis are worse with irregular hygiene.

**Conclusion:** A regular 3–6 month recall interval tailored to a patient’s risk profile is recommended.

**SMOKING**
Smoking causes soft tissue complications and elevated peri-implant bone or implant loss.

**Conclusion:** A smoking cessation program boosts implant survival rate.

**CLEANING OPTION**
Poor access reconstructions exhibit increased peri-implantitis compared with good access.

**Conclusion:** A well-integrated reconstruction should provide unimpeded cleaning access.

**ORAL HYGIENE**
Poor oral hygiene raises the risk for peri-implantitis.

**Conclusion:** Optimum oral hygiene is key to maintaining inflammation-free, peri-implant health.

**KERATINIZED GINGIVA**
Insufficiently wide (<2 mm) keratinized gingiva is linked with elevated plaque accumulation, inflammation and recession.

**Conclusion:** Care should be taken during implantation and reopening to ensure that the keratinized gingiva is sufficient (≥2 mm).

**CEMENT RESIDUE**
Iatrogenic cement residue is linked to mucositis and peri-implantitis.

**Conclusion:** A great deal of attention should be paid to cementing; otherwise, a screw-retained reconstruction is preferable.

**IMPLANT STRAIN**
Despite animal experiments failing to detect strain as a cause for osseointegration loss, without evidence of infection, osseointegration loss cannot be ruled out in humans.

**FOCUS**
Prof. Giovanni E. Salvi | Switzerland
FOCUS

Treating peri-implantitis systematically

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There is no single measure for resolving peri-implantitis but rather a sequence of steps: First, causative actors should be identified and resolved, then infection around the implant should be managed and, finally, regeneration of the defect can be considered.

Step 1 – Assessing the situation

The implant-supported prosthesis should be evaluated to determine if there are any causative factors such as screw-loosening, excess luting cement, poor abutment fit or poor prosthesis contour. The prosthesis should also fit well and provide access for easy cleaning. Corrections should be made where necessary (this may involve removal of the prosthesis). Risk factors, including poor oral hygiene, smoking, diabetes or the presence of deep periodontal pockets, should also be addressed.

Step 2 – Non-surgical debridement

Non-surgical debridement using appropriate instruments, such as titanium curettes, air-powder abrasive devices, ultrasonic devices, photodynamic therapy, or Er:YAG laser, should precede surgical intervention. Systemic antibiotics, local antimicrobials and/or the use of topical antiseptics (e.g., chlorhexidine) may be concomitantly prescribed. Individual oral hygiene instruction should be provided to ensure good plaque control.

Step 3 – Re-assessment

A re-evaluation should be made approximately 4-weeks after non-surgical debridement to determine if there has been a resolution of peri-implantitis. Some cases of peri-implantitis will resolve following non-surgical management, in which case patients can commence at home maintenance care.

Step 4 – Surgical intervention

If the peri-implantitis has not resolved at re-evaluation, a surgical approach is recommended. Surgical intervention is frequently required when the peri-implantitis lesion is severe with advanced bone loss and deep peri-implant pockets. The presence of retained excess luting cement located submucosally usually requires a surgical access approach for cement removal. Surgical management involves elevating a full mucoperiostal flap and removing the inflammatory granulation tissue to allow thorough decontamination of the implant surface. Various implant surface decontamination methods have been investigated including: rubbing with gauze soaked in saline, chemical agents such as citric acid or hydrogen peroxide, mechanical cleaning with a curette or a titanium brush, laser treatment and air-powder abrasive devices. However, currently there is no one decontamination method that has proved to be superior.

Access flap approach

In the access flap approach, no attempt is made to regenerate the bone. Following thorough implant surface decontamination, the flap is closed and allowed to heal. Soft-tissue recession is frequently observed as a part of the healing process, but the main goal of this treatment approach is to resolve inflammation.
Resective approach
In some situations where aesthetic outcomes do not have high priority, the bone peaks around the implant can be removed or reshaped to allow the flap margins to be positioned apically. After healing, this technique results in a reduction in peri-implant pockets but also significant soft-tissue recession. Implantoplasty, i.e., modification of the implant surface using a carbide or diamond bur, has also been described in conjunction with this treatment modality. The aim of implantoplasty is to modify the implant surface to facilitate oral hygiene following healing.

Regenerative approach
Another treatment approach aimed at regeneration and re-osseointegration of the peri-implant bone involves filling the intrabony component of the defect with a bone graft or bone substitute material followed by coverage with a barrier membrane (Fig. 1). Contained intrabony defects are more suited to a regenerative approach than non-contained defects, where there are no residual bony walls to support the graft material.

In an attempt to regenerate the peri-implant defect, numerous graft materials have been studied, including autogenous bone, allogeneic decalcified freeze-dried bone, phytogetic calcium carbonate, hydroxyapatite, tricalcium phosphate or xenogeneic bone mineral. In some protocols, non-resorbable membranes of expanded polytetrafluoroethylene (e-PTFE), resorbable synthetic or collagen membranes have been used to cover the graft material.

Varying amounts of defect fill have been reported. Animal studies have shown that re-osseointegration of a previously contaminated implant surface is possible following a regenerative approach. Several studies have shown that regenerative approaches can provide successful long-term treatment outcomes in the majority of patients.

1 Peri-implantitis at implant site with deep probing depths, a draining sinus on the buccal mucosa and bleeding and suppuration following probing.
2 Periapical radiograph showing marginal bone loss and the presence of excess luting cement.
3 After flap elevation, removal of the excess cement and decontamination of the implant surface, the intrabony defect is filled with Geistlich Bio-Oss® graft material.
4 The Geistlich Bio-Oss® is covered with a resorbable collagen membrane (Geistlich Bio-Gide®).
5 Immediately after flap closure and suturing.
6 Clinical photograph 12-months after healing.
7 Periapical radiograph 12-months after treatment.
8 Materials used for a regenerative treatment approach: Geistlich Bio-Oss® and Geistlich Bio-Gide®.
Step 5 – Post-surgical care

During the immediate post-operative healing phase, daily rinsing with chlorhexidine is recommended to provide adequate biofilm control. Although there are currently no randomized controlled trials evaluating the effect of systemic antimicrobials for peri-implantitis, peri-operative systemic antimicrobials are commonly prescribed to suppress the microbial load, particularly with specific periodontal/peri-implant pathogens. The possible side effects of systemic antimicrobials should be discussed with the patient prior to administration.

Step 6 – Maintenance care

The final treatment phase involves the provision of an individualized maintenance care program. Regular monitoring, oral hygiene reinforcement and professional supra-mucosal biofilm removal is required to avoid re-infection or the recurrence of peri-implantitis.

The frequency of maintenance depends on the risk assessment for each patient. Relevant factors include smoking habits, periodontal status, diabetes and oral hygiene.

Removal of implants

When peri-implantitis treatment is unsuccessful, or when there is a severely compromised aesthetic result, removal of the implant may be required. The implant should be removed in a conservative manner, avoiding damage to neighbouring structures and preserving as much bone as possible.

Many implant manufacturers have a specific tool that can be used to remove their particular implant by reversing it at high torque. Following removal of the implant, augmentation of the site using a bone graft or bone substitute material in conjunction with a barrier membrane may be considered for regenerating the site.

Conclusions

A recent systematic review concluded that in most studies peri-implantitis treatment resulted in an improvement in clinical conditions for the majority of patients. However, in some patients, despite treatment, there was a recurrence or progression of disease requiring re-treatment or removal of the implant. It is important to note that it is the anti-infective treatment protocol in its entirety that contributes to a successful treatment outcome. With respect to the choice of treatment modality, the clinician should choose the most appropriate treatment method based on the individualized needs of the patient.

References

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LEADING REGENERATION
The microbiology of peri-implantitis

Are there any microbes which are complicit when peri-implantitis takes an especially severe course? And are microbiological tests worthwhile? Searching for clues at a micro-scale with Prof. Andrea Mombelli, Switzerland.

Prof. Mombelli, are peri-implantitis bacteria the same as periodontitis bacteria?

Prof. Mombelli: High microbial counts for various anaerobic bacteria can regularly be detected in implants with peri-implantitis. These include Fusobacteria, Prevotella, Porphyromonas, Spirochetes and Peptostreptococci. This anaerobic mixed flora is indeed very similar to periodontitis in natural teeth. But at times you find flora on an implant where Staphylococci pre-dominate. This is untypical with natural teeth. Staphylococci, however, very often have a part to play in infections of orthopaedic implants outside the oral cavity and infections in catheters, etc.

Is the implant colonised from the outset or do the bacteria arrive later?

Prof. Mombelli: All dental implants are inevitably contaminated at placement. Even so, the great majority of implants heal without infection. Peri-implant infections may be the consequence of primarily non-microbial events, which encourage the emergence of a pathogenic microflora. We have explored this relationship in an article on the significance of biofilms in peri-implant disease. An example is the subgingival persistence of adhesiveness, which can trigger a purulent bacterial infection that cannot solely be remedied through anti-infectious measures. The underlying cause must be eliminated for healing to take place. So the search for a specific cause always forms part of the differential diagnosis of peri-implantitis, even if pus or a biofilm point to a bacterial infection.

Do all patients have the same peri-implantitis bacteria?

Prof. Mombelli: The infection is typically a mix of bacteria that the patient also has elsewhere in the mouth. Then microecological factors influence the growth of the various microbes. For instance, a local mucosal inflammation may be due to deficient cleaning in an inaccessible niche.

“Peri-implantitis has no specific pathogen behind it.”

Are there specific bacteria which are complicit in severe peri-implant infections?

Prof. Mombelli: No. Peri-implantitis does not develop due to an infection originating from an external specific highly pathogenic trigger. You can find all microbes in low numbers in the mouth, nose or throat area, even in clinically healthy individuals. Staphylococci are no different. So total eradication is an unrealistic treatment goal. Rather, the aim is to prevent an excessive build-up of potentially pathogenic microbes in the form of a biofilm.

Is there a good test for peri-implantitis bacteria? Should such a test be performed?

Prof. Mombelli: There is no clinical evidence showing any extra benefit from such tests over and above a precise clinical and radiological investigation. There is no cost benefit analysis for such tests either.
Though I am very sympathetic toward colleagues and patients who would like to know more, I must say that the preventive and therapeutic options currently on the table do not require a bacterial test.

**What systemic antibiotics are suitable for therapy?**

**Prof. Mombelli:** From extensive studies in periodontology and the knowledge mentioned in relation to peri-implant flora, today we generally use a combination of amoxicillin and metronidazole. Our own multi-centric study and the work by other research groups have shown good results\(^2\). For cases of intolerance, such as allergy to penicillin, just metronidazole by itself can be prescribed, but it is not effective against all incriminated microbes. The additional remark that peri-implantitis cannot be successfully treated by purely pharmaceutical means is very important. It always requires meticulous cleaning of the whole contaminated implant surface. In order to completely remove the biofilm, it usually has to be uncovered surgically.

**References**

Peri-implantitis therapy using regenerative surgery: Case studies

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CASE 1

CASE 2

Photos: Schwarz
Regenerative therapy should be combined with implant plastic surgery, if the configuration of a defect is advanced and complex.

In the first case, two bar-supporting implants have an advanced, combined (supra- and intraosseous) defect configuration with vestibular dehiscences and a supracrestal exposed screw thread (>1 mm). In such cases, after completely removing the granulation tissue, we start out by performing plastic surgery on the implant to smooth the implant body in the supra-crestal and buccal defect region. The portions of the implant surface facing the defect are structurally preserved and decontaminated (e.g., with a curette, Er:YAG laser and sterile saline solution).

The intraosseous defect components are then augmented with a slowly resorbing bone replacement material. This is covered with a collagen membrane before the soft tissue flap is adapted tightly around the implants. The second case involves circumferential intraosseous defects with a supracrestal component (<1 mm) on two adjacent implants. Such defects can be regenerated using bone grafting without plastic surgery on the implant.

**What are the special considerations?**

The plastic surgery smooths the macro- and microstructure of the implant body in the areas beyond the physiological barrier provided by current augmentation techniques. This encourages soft tissue integration and reduces bacterial deposition. This therapy combined with Guided Bone Regeneration (GBR) in the intraosseous defect region reduces the probing pocket depths, increases the clinical attachment level and ensures a long-term stable bone level. The mucosal recession bone level can be offset by a simultaneous soft tissue augmentation with a connective tissue graft or a porcine collagen matrix. This allows treatment in the aesthetic zone. However, the complete loss of osseointegration necessitates explantation.

**References**