Periodontitis and peri-implantitis in implant dentistry

An interview with Dr Inga Boehncke, Germany

Dr Inga Boehncke is a member of the German Association of Oral Implantology.

Peri-implantitis continues to be one of the greatest challenges for dental professionals. According to studies, the prevalence of the condition will continue to rise in the coming years. implants—**international magazine of** oral implantology spoke with implant specialist Dr Inga Boehncke, who has been running her own practice in Bremen in Germany since 2009, about specific features of implant therapy in the case of previous periodontal disease, surgical and non-surgical treatment protocols for peri-implantitis and how the treatment of the condition will develop in the future.

Dr Boehncke, what is especially challenging about implant treatment for patients with previous periodontal disease?
First, a stable periodontal situation must be established by determining and discussing the individual risk profile of the patient. The type and extent of previous periodontal disease play an important role and several questions need to be addressed. Have there been recurrences? Is the periodontal situation stable? Does the patient smoke? Does the patient have diabetes? In addition, it is important to establish and monitor the haemoglobin A1c level, which should normally be determined every three months and should not exceed a value of 6.5–7.0%.

Interleukin polymorphisms also need to be considered, especially in combination with several of the aforementioned factors, as about 10% of patients are high responders, meaning that an inflammatory change can be associated with a stronger to excessive reaction. Ultimately, the compliance of patients plays a decisive role regarding the removal of plaque at home and the willingness to undergo oral hygiene at short intervals in order to determine individual biofilm management depending on their risk profile.

If periodontal conditions are stable—free of bleeding and with probing depths of no more than 5 mm after previous periodontal disease—we apply a 0.2% chlorhexidine rinse three times for 30 seconds before implantation. If a film is visible on the tongue, it is removed with a tongue scraper and a chlorhexidine spray is used if necessary. Additionally, periodontally compromised patients are advised to undergo systematic plaque removal in the practice two to three days before the procedure. We also administer vitamin C and vitamin K2 and determine the vitamin D3 level, supplementing depending on the value. During follow-up, we closely monitor, in particular, the adequate removal of plaque. Postoperatively, patients are advised to rinse twice daily with a 0.2% chlorhexidine rinse for one minute. Alternatively, a chlorhexidine gel can be applied to the wound area. During subsequent follow-ups, depending on the severity, risk profile and cooperation of the patients, an individual schedule of short intervals of monitoring involving biofilm removal and determination of the bleeding on probing is indicated.

In addition, we work with the active matrix metalloproteinase-8 (aMMP-8) biomarker from Bioscientia, which determines collagenase activity and thus represents a kind of destruction marker. Inflammatory tissue destruction can thus be detected at an early stage before it becomes clinically visible. A value of 0 for the patient is determined two to four weeks after prosthetic restoration of the implant. One year later, another aMMP-8 test is carried out.

Nowadays, dental implants have a high survival rate, but peri-implant infections are among the most common complications. In the 2019 Delphi study of the European Association for Osseointegration, experts agree that the prevalence of peri-implantitis will increase in the coming years. How can dentistry meet this challenge?
The prevalence of peri-implant mucositis, which is roughly comparable to gingivitis and is initially limited to the inflammatory change in the soft tissue, is currently approximately 43%. Peri-implantitis, which is associated with inflammatory bone resorption that has already occurred, meaning it is comparable to periodontitis, affects approximately 22% of patients. In my opinion, early detection of inflammatory signs and timely intervention are the pillars of postoperative care. Regular bleeding on probing assessment is a key diagnostic tool to detect inflammatory changes at an early stage. As already mentioned, destruction markers can also be used. These are helpful in explaining the therapy to the patient.

In addition, patients with implants should be enrolled in special oral health programmes that include regular systematic removal of the microbial biofilm and early inflammatory diagnostics.

Recent studies have found that mesially and distally splinted implants, as well as implants with an over-contoured restoration, pose an increased risk of peri-implantitis. The more difficult plaque removal for patients and the resulting accumulation of plaque plays a central role in this.

The design of the implant superstructure in terms of facilitation of cleaning and adequate attachment of the soft tissue should be given high priority from the outset to guarantee patients easy and pain-free plaque removal at home. An unattached and thin mucosa often leads to discomfort during cleaning as well as to faster pocket formation and thus plaque accumulation. We often use CAMLOG's NovoMatrix to thicken and secure the peri-implant tissue.

Peri-implantitis can be treated both surgically and non-surgically or with a combination of both methods. However, there is no standardised surgical protocol yet. How can dentists guarantee good care for their patients?
The decision of whether to treat peri-implantitis surgically or non-surgically depends mainly on the severity and the implant surface. It must be clarified whether a rough surface or even already exposed contaminated threads are present. The first step is to eliminate mechanical risk factors such as overhangs that have contributed to plaque formation. The superstructures should be removed. For both surgical and non-surgical cases, the main focus lies on decontamination. Removal of the microbial biofilm and thus reduction of bacterial colonisation is achieved with hand instruments, ultrasonic tips and powder-blasting devices that use glycine powder. In addition, we use multiple 3% hydrogen peroxide rinses and chlorhexidine rinses applied directly and alternately. Local antibiotics can also be administered as a supportive measure. We use Ligosan Slow Release from Kulzer with the aim of keeping the tissue free of bleeding and reducing pocket depths.

In the case of defect morphologies that limit access or already advanced bone resorption, we apply a surgical therapy that involves flap elevation, analogous to open periodontal therapy, to achieve better visibility over the contaminated parts and thus better accessibility. For this purpose, we use fine nickel–titanium brushes for decontamination, as well as glycine powder and repeated 3% hydrogen peroxide and chlorhexidine rinses.

Furthermore, regenerative work can be carried out, and the success of this is directly related to the morphology of the defect. If a small bowl-shaped intraosseous defect is present, it is easier to regenerate with augmentation than are already developed supra-crestal defects which show screw threads that are above bone level. If this occurs, the threads are removed and the protruding implant surface is smoothed as far as possible to eliminate roughness, with the aim of preventing plaque colonisation anew. There are also combined defects for which both procedures can be used. We use autogenous bone chips and a bone substitute material covered with a collagen membrane for regeneration. The main aim is to support the soft tissue, and in many cases thickening owing to scarring is observed.

How do you think the prevention and treatment of peri-implantitis will develop in the future?
Particular attention should be paid to postoperative care at short intervals in order to be able to intervene as early as possible. Immunomodulatory therapies are currently under discussion. Through the anti-inflammatory effect of natural cranberry extract on the tissue-destroying macrophages, topical application should directly intervene in the intensity of the inflammatory reaction.

Furthermore, I could imagine that new carrier materials for local antibiotics or natural extracts—as already mentioned—will be developed. Modified implant surfaces, possibly with anti-infection or plaque-inhibiting properties, are also conceivable. However, I think it is most important to raise patients' awareness and motivate them to have regular and thorough follow-up care and to remove plaque at home, irrespective of the therapeutic approach. After all, prevention is better than cure.

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