Resective peri-implantitis therapy with implantoplasty in Crohn's disease

Lucas A. Greilich, Dr Mischa Krebs, PD Dr Maximilian Moergel, Germany

In 1977, Per-Ingvar Brånemark defined osseointegration as a functional ankylosis of the bone on the surfaces of titanium implants.[1,2] Since then, dental implants have evolved and now offer most patients a predictable option for long-term rehabilitation of their masticatory function. However, despite high healing rates of 90 to 95 per cent, certain risk factors predispose to peri-implant inflammation with bone resorption (peri-implantitis).[3,4] This article reviews peri-implantitis and describes resective therapy with implantoplasty in a patient with Crohn's disease.

The risks that can lead to implant failure can be categorised as either generalised systemic factors or localised factors. Table 1 provides an overview of these factors.

Some of these factors can be influenced by the patient (e.g. oral hygiene, smoking); others can be avoided by the clinician through advance treatment planning (e.g. cement residue, implant position). Still others, however, cannot be influenced (e.g. osteoporosis, diabetes mellitus). However, no valid therapy has been established that would result in complete healing of the progressive bone loss.

Prevalence

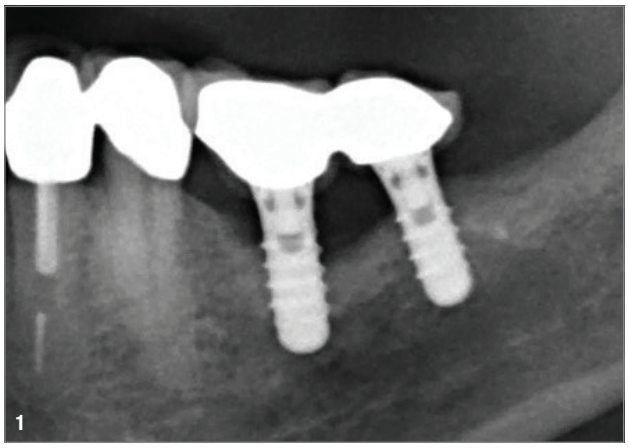
Peri-implantitis affects a significant number of patients.[5] Derks et al. reported the prevalence of peri-implant mucositis to be 19 to 65 per cent and peri-implantitis to be 1 to 47 per cent. The wide variation in the literature is due to the high degree of variability in the underlying definition of peri-implantitis, particularly with regard to the type and extent of bone resorption.[6,7]

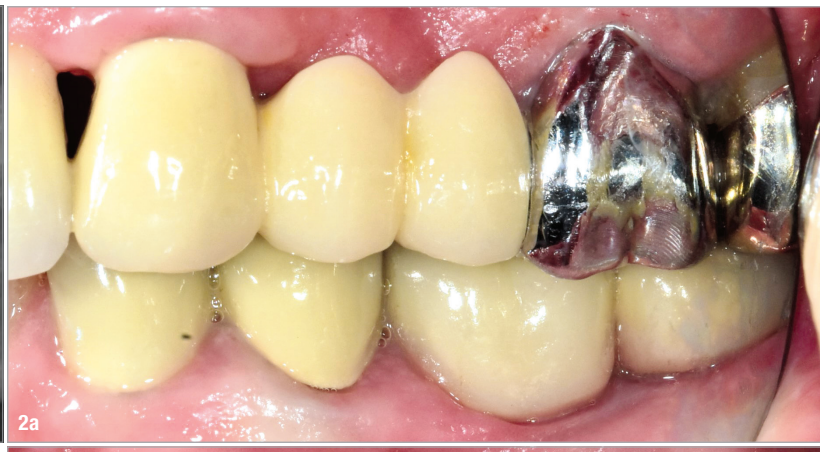
Aetiology

Peri-implantitis is primarily caused by anaerobic oral pathogens (e.g. *T. forsythia*, *P. nigrescens*, *A. actinomycetemcomitans*, *P. gingivalis*, *T. denticola*).[8–12] Titanium-

Generalised systemic risk factors	Localised risk factors
Diabetes mellitus	Incorrect implant position
Rheumatoid arthritis	Locally limited oral hygiene ability
Osteoporosis	Keratinised mucosa < 2 mm
Periodontitis	Cement residue
Radiation exposure	Mechanical overload
Antiresorptive drugs	Frequently replaced abutments
Crohn's disease	Abutment emergence profile too steep (< 30°)
IL-1 polymorphism	
Previous implant loss	
Poor oral hygiene	
Irregular recall schedule	
Nicotine abuse	

Table 1: Factors that can lead to peri-implantitis.





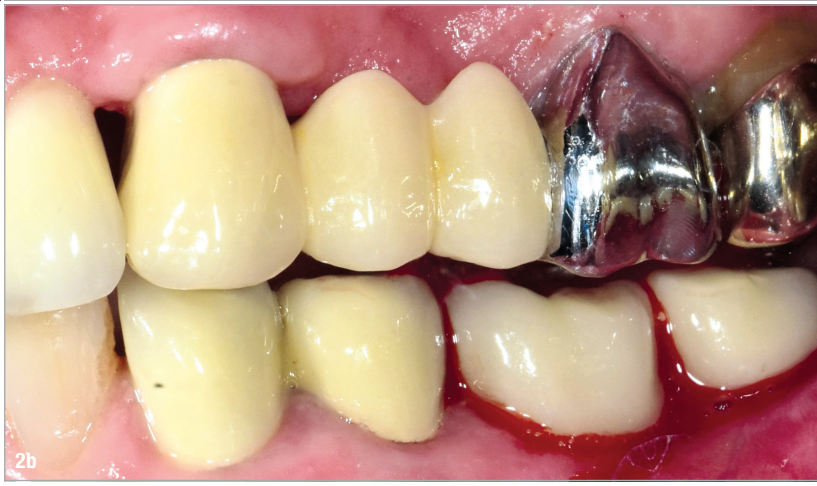


Fig. 1: Panoramic radiograph. Bowl-shaped peri-implant bone resorption at implant 36, less pronounced horizontal bone resorption at implant 37, splinted crowns on implants 36 and 37. **Fig. 2a:** Initial situation (photographed indirectly with a mirror). Narrow keratinised mucosa at sites 36 and 37. **Fig. 2b:** Ten seconds after probing with a WHO probe. Bleeding on probing as a sign of an inflammatory event in combination with radiographic bone resorption > 2.0 mm; diagnosis of peri-implantitis.

affine *S. aureus* also appears to play an important role in the development of peri-implantitis.[13] Histological analysis also shows that leukocytes, B cells, and T cells are significantly increased.[14,15]

Definitions

In 2017, at the World Workshop in Chicago, USA Schwarz et al. defined peri-implantitis as a pathological inflammatory condition in the peri-implant soft tissue that induces progressive bone resorption.[16] Bleeding on probing has been established as a mandatory finding for the diagnosis of mucositis, while radiographic evidence of bone loss, in combination with clinical signs of inflammation, is indicative of peri-implantitis.[16–20]

Untreated mucositis can progress to peri-implantitis.[20–22] The distinction is important because mucositis may be reversible with consistent plaque removal, whereas peri-implantitis cannot be brought to long-term healing. Progressive bone resorption subsequently poses a risk of implant loss.[16]

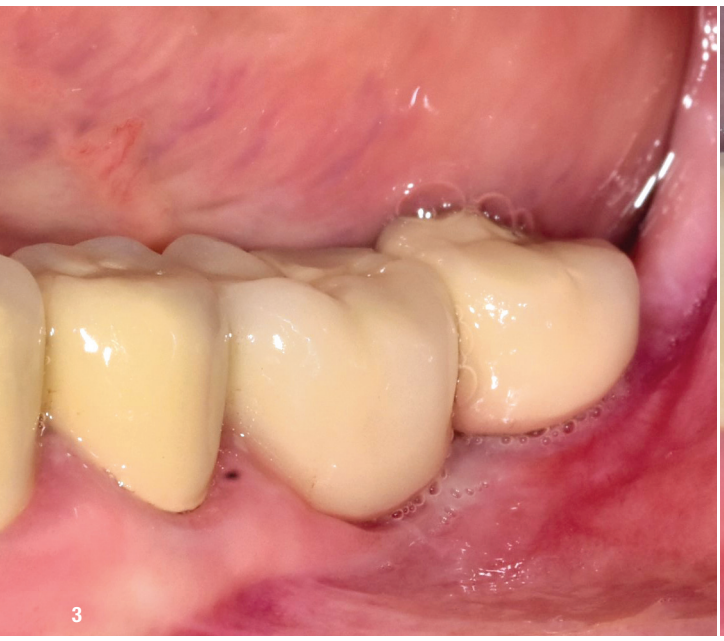
In daily clinical practice, the diagnostic problem is to decide when the extent of bone resorption can still be considered bone remodelling or when peri-implantitis must be assumed. In the absence of baseline radiographs after implant placement, Sanz and Chapell recommend diagnosing peri-implantitis at 2.0 mm vertical bone loss. If baseline radiographs are available after implant placement, a more sensitive value may be used. Krebs et al. compared different definitions of peri-implantitis. They recommend a threshold of 1.5 mm of radiographic bone loss in the presence of postoperative radiographs.[7]

Treatment

Treatment of peri-implantitis can be divided into conservative and surgical approaches; the latter of which may be regenerative or resective in nature. Derived from periodontology, the core issue is adequate plaque control.[12] Plaque reduction is performed with plastic or carbon curettes to avoid damaging the delicate titanium surfaces with metal curettes.[23,24] Other plaque reduction options include ultrasound, air-abrasive devices, diode lasers, or antiseptics (e.g. citric acid or chlorhexidine).[25–27] Treatment may be combined with topical or systemic antibiotics.[28]

The surgical therapeutic approach to peri-implantitis is derived from that of open periodontal surgery.[29] Regenerative therapy approaches form a narrow range of indications, namely those in which (mainly) three-wall defects—which must be sufficiently steep and deep—can be filled with bone substitute.[30] The therapeutic success of these regenerative measures is largely determined by whether complete decontamination of the implant surface has been achieved.

Often, however, generalised bone resorption occurs with successively exposed implant threads. Here, implantoplasty is an option. In this procedure, the contaminated



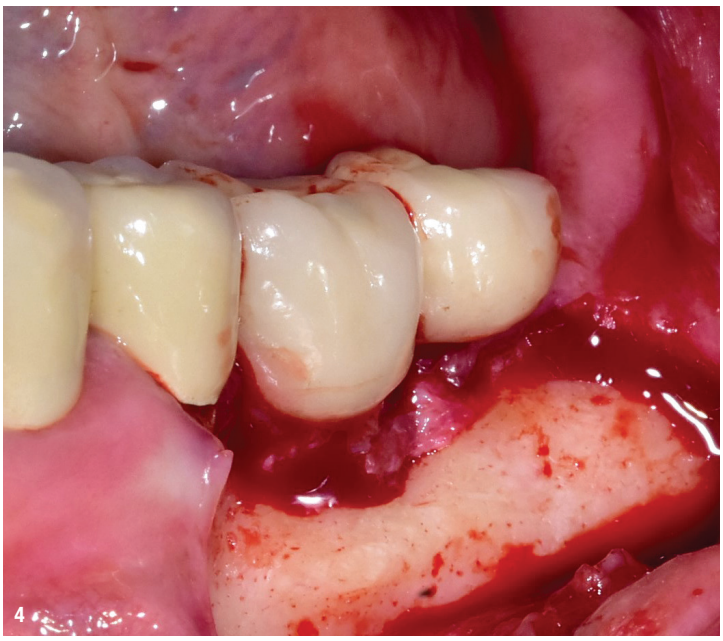


Fig. 3: Preoperative situation. Hyperaemic peri-implant mucosa with inflammatory changes at sites 36 and 37. **Fig. 4:** Mobilisation of a mucoperiosteal flap after a trapezoidal incision. Granulation tissue infiltrating the pronounced bone defect at site 36. Adequate individualised plaque control is no longer possible for this patient. The exposed, submerged implant threads provide optimal conditions for pathogens.

implant surface is smoothed by ablation of the exposed implant threads (red diamonds, yellow diamonds, Arkansas stones), making it more difficult for plaque to accumulate.[31]

It is important not to treat the implant surface with silicone polishers ("brownie", "greenie"), as silicone residues in the peri-implant soft tissue are not biocompatible and can lead to foreign body reactions and reinflammation.[32] Free titanium particles do not interfere with cellular activity (based on research to date), but may cause metallic discoloration of soft tissue, which constitutes an aesthetic compromise.[33–35]

Case report

Medical history

A 61-year-old female patient was referred to our day clinic for maxillofacial surgery. She presented with complaints related to implants placed in 2009. The patient's medical history included Crohn's disease, which had been diagnosed in adolescence and was currently well controlled. She had been in remission for eight years and was not taking any medication at the time.

Clinical findings

After clinical examination and evaluation and a panoramic radiograph, peri-implantitis was diagnosed on the splinted implants 36 and 37 based on clinical bleeding on probing and radiographic bone loss > 2.0 mm (Figs. 1 & 2). The width of the keratinised mucosa was less than 2.0 mm on implant 36 and completely lost on implant 37. The surrounding free mucosa showed reactive hyperaemic changes with associated oedematous swelling (Fig. 3).

> "In daily clinical practice, the diagnostic problem is to decide when the extent of bone resorption can still be considered bone remodelling or when peri-implantitis must be assumed."

Treatment

After detailed consultation and explanation, the patient was scheduled for resective peri-implantitis therapy by implantoplasty. Under local anaesthesia (Articaine 1:200,000), a strictly marginal incision was made (to preserve the remaining keratinised mucosa) and a trapezoidal flap was elevated with distal relief incisions at implant 37 and mesial relief incisions at tooth 35 (Fig. 4). After mechanical decontamination with a curette, an implantoplasty was performed on the exposed implant surfaces (Fig. 5). The implant threads were removed until a smooth implant surface was achieved with less risk of plaque accumulation and recontamination. During an implantoplasty, it is important to strictly level only the im-

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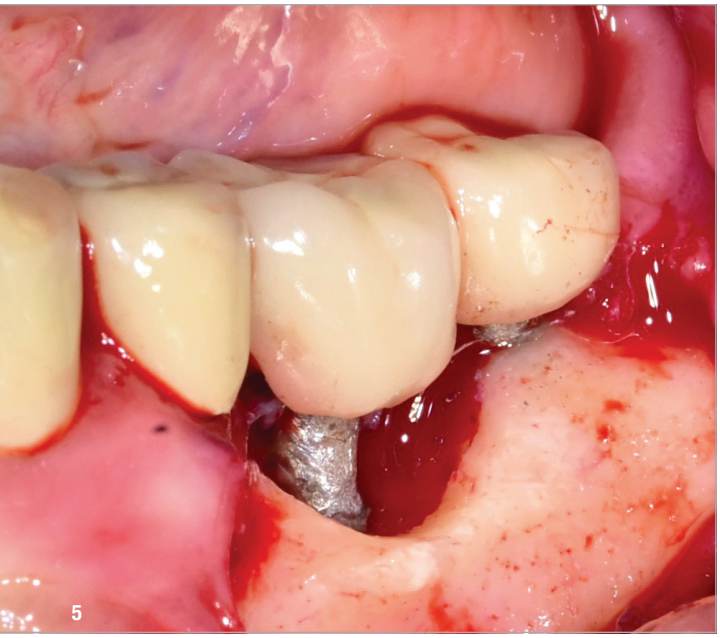

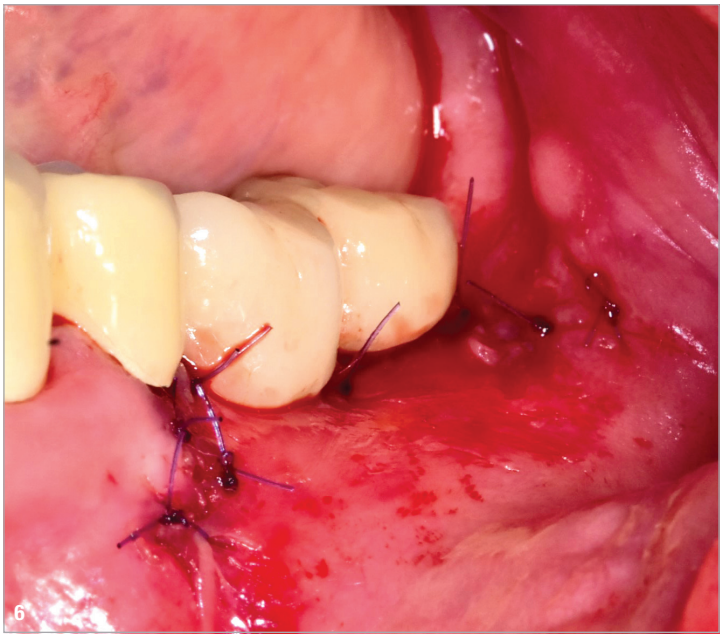

Fig. 5: After decontamination and implantoplasty of implants 36 and 37. The levelling of the implant threads is intended to prevent early recontamination of the implant surface by pathogens. Leaving the superstructure in place makes an implantoplasty more difficult. **Fig. 6:** Wound closure. Monofilament sutures (Monofast; mectron) are used for tension-free and saliva-tight adaptation of the wound margins.

plant threads, so as not to reduce the implant diameter (risk of fracture). Finally, the surface was polished with an Arkansas stone. The wound was closed with a 5/0 monofilament suture material (Monofast; mectron) after thorough irrigation with chlorhexidine (Chlorhexamed forte alcohol-free 0.2%; GSK) and saline solution (Fig. 6).

The patient received postoperative instructions and analgesic therapy (paracetamol 1 g). Sutures were removed after seven days. The patient was referred back to the referring general dentist with the request to re-evaluate the case and to perform regular dental prophylaxis in six months at the earliest. Further appointments were scheduled at our day clinic for expansion of the keratinised mucosa with a free mucosal graft after healing. Figure 7 illustrates the situation 20 days postoperatively.

Discussion

This case report demonstrates how risk factors (reduced height of keratinised mucosa, splinted superstructure, limited hygiene ability, Crohn's disease) can influence the development of peri-implantitis.

Crohn's disease and ulcerative colitis have become more prevalent in developed industrialised countries in recent decades, making this condition increasingly relevant for dentists. In Germany, 322 new cases of Crohn's disease are diagnosed per 100,000 inhabitants per year. Patients in their third and fourth decades of life are the most likely to develop the disease, although apparently young and healthy people can also be affected.[36]

Crohn's disease is an inflammatory bowel disease characterised by transmural ulcers of the bowel wall. Unlike Crohn's disease, ulcerative colitis can affect the entire digestive tract (from the mouth to the anus). In the dental office, therefore, close examination of the oral mucosa should be performed in these patients in order to detect any lichenoid/leukoplakic changes, lip and gingival swelling, pseudo-polyps or aphthoid/ulcerative lesions ("cobblestones") at an early stage.[37]

The disease progresses in phases, being completely asymptomatic in remission, while patients suffer from abdominal cramps, diarrhoea, weight loss, vomiting and fever during an active phase. The disease is treated with various pharmacological drugs, prescribed according to a graduated scheme.

Therefore, when examining the patient's medical history, the dentist should pay close attention to immunosuppressants (prednisolone, mesalazine, azathioprine, methotrexate) and biologics (infliximab, adalimumab, vedolizumab, ustekinumab). Given the patient's chronic inflammatory bowel disease, non-steroidal anti-inflammatory drugs (ibuprofen, aspirin, diclofenac) should be avoided, as they may irritate the gastric mucosa and trigger an episode.

In a systematic review, Voina-Tonea et al. identified a statistically significant association between Crohn's disease and early implant loss.[36] Malnutrition has been implicated as a cause of impaired osseointegration; autoimmune inflammatory events may have a direct effect on bone formation. In addition, possible side effects of long-term

cortisone therapy on implant survival are conceivable. Other known side effects that may directly or indirectly affect implant survival include hypertension, diabetes mellitus, gastritis type C, osteoporosis, glaucoma, and an increased risk of infection.

Three retrospective studies and one prospective study were evaluated In the above-mentioned review, although the studies by van Steenberghe et al. and Alsaadi et al. were limited by the very small number of participants of n = 2 and n = 3, respectively.[38,39] The extent to which Crohn's disease played a specific role in the development of peri-implantitis in the present case remains hypothetical, but must be considered in the search for a therapy.

Due to the horizontal (site 37) and bowl-shaped (site 36) bone defect configuration, a regenerative therapy approach was not considered promising (Figs. 1 & 5). Shallow bone defects create extremely poor conditions for a regenerative therapeutic approach and are difficult to augment stably over the long term.[40,41] To slow down peri-implantitis, especially outside the aesthetic zone, a resective therapeutic approach was chosen, which facilitates complete decontamination by "levelling" the implant threads and makes early recontamination of the implant surface more difficult.

> "In a systematic review, Voina-Tonea et al. identified a statistically significant association between Crohn's disease and early implant loss."

Removal of the superstructure should always be discussed with the patient and the general dentist prior to any proper implantoplasty. Leaving the superstructure in place will make levelling the exposed implant threads much more difficult and may compromise the result. In complex cases, it may even be advisable to close the implants with cover screws and allow them to re-heal subgingivally after bone grafting. In the present case, the patient chose not to have the superstructure removed for economic reasons.

The clinical and radiographic success of peri-implantitis therapy can only be evaluated retrospectively after several years, and the patient and clinician should be aware that long-term implant retention depends on many factors. Because peri-implantitis is a multifactorial process, there are factors that are beyond the control of either the patient or the clinician (Table 1), and despite the best efforts of both, implants may ultimately have to be explanted.

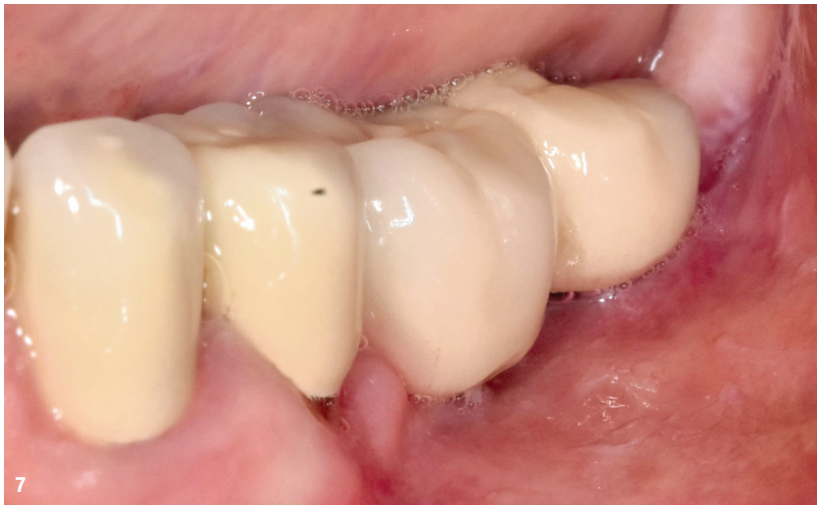

Fig. 7: Progress of wound healing after 20 days.

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contact

Lucas A. Greilich
Burgstraße 2–4, 65183 Wiesbaden, Germany
lucas.greilich@helios-gesundheit.de

Dr Mischa Krebs
Schillerplatz 3, 55232 Alzey, Germany

PD Dr Dr Maximilian Moergel
Burgstraße 2–4, 65183 Wiesbaden, Germany
maximilian.moergel@mkg-burgstrasse.de

Literature | Lucas A. Greilich | Dr Mischa Krebs | Dr Maximilian Moergel