

# Biological Implant Complications and Their Management

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## Abstract

**Background:** Background: With the increasing popularity of dental implants the presence of implant complications is rising, and the question of how to best manage these complications still lingers in most clinicians' minds. This paper aims to provide clinicians with an overview of the most commonly encountered biologic implant complications as well as to provide guidelines as to how to treat them.

**Methods:** Available English literature was reviewed, including peer-reviewed journal publications and online resources. Several treatment modalities have been proposed to manage these complications, including non-surgical mechanical debridement, antiseptics, local and/or systemic antibiotics, lasers, resection with or without implantoplasty and regenerative approaches.

**Results:** In this guideline, it is suggested that the treatment modalities should be chosen based on the severity of peri-implant diseases, amount of bone loss and the morphology of peri-implant bony defects. For peri-implant mucositis or peri-implant defects with less than 2 mm destruction, non-surgical treatments are recommended. For peri-implant defects with more than 2 mm destruction, surgical treatments (e.g., resection with or without implantoplasty, guided bone regeneration) are suggested that include removal of the implant if the bone loss is beyond repair.

**Conclusion:** The prevention of biological implant complications relies on careful planning, a thorough examination to assess etiological factors and a regular maintenance recall schedule. With early diagnosis, biological implant complications should be managed based on the severity of peri-implant disease, the amount of bone loss and the morphology of the peri-implant bony defects.

*Key words: Dental implants, implant complications, implant failure, biological*

## Introduction

Osseointegration is considered the foundation of implant stability and is defined as the direct contact between living bone tissue and the implant surface (Glossary of Periodontal Terms 2001). With the increasing popularity of dental implants, the presence of implant complications has been on the rise. Consequently, how to best manage these complications remains a question in all clinician's minds.

Prior to the discussion of implant complications, it is essential that we review the definitions of "implant success" and "implant failure" (Table 1). In 1986, Albrektsson and colleagues proposed the well-known criteria for implant success based on his treatment results using the classic Branemark systems (Albrektsson *et al.*, 1986). A common cited implant success criterion proposed by Albrektsson states, "no more than 1 mm of marginal bone loss during the first year" has been included in the updated implant success criteria that were proposed 10 years later (Roos *et al.*,

1997). It is also important to note that these success criteria are only referring to pure titanium (smooth) surface implants and not rough surface-coated implants. In contrast, "implant failure" has been categorized as: ailing, failing and failed implants. A "failed" implant is characterized as not only having radiographic bone loss but also mobility and is essentially considered an untreatable situation (Torosian and Rosenberg, 1993). The "ailing" implant presents with radiographic bone loss without clinical signs of inflammation, whereas a non-mobile implant with both radiographic bone loss and consistent deterioration is defined as a "failing" implant (Sakka *et al.*, 2012). Fortunately, both "ailing" and "failing" implants are considered to be treatable.

Generally, implant complications can be classified into three groups: biological, biomechanical and esthetic complications. Biological implant complications result from the biological process that affects peri-implant tissue and ultimately disturbs implant function. In other words, these complications include implant loss and inflammation of the peri-implant tissue (Berglundh *et al.*, 2002). Because of loss of osseointegration, loss of implant can be further divided into "early" or "late" implant failure based on the timing of implant removal or lack of osseointegration. Histologically, an implant with loss of

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osseointegration has a predominant fibrous tissue capsule, preventing direct contact between implant and bone and resulting in impaired implant function. Other biological implant complications, which occur more commonly, are peri-implant diseases. Peri-implant diseases are comprised of peri-implantitis and peri-implant mucositis, which are characterized by the presence or absence of bone loss, respectively. In the consensus reports of the Sixth European Workshop on Periodontology (6<sup>th</sup> EWOP), “peri-implant mucositis” was defined as inflammatory lesions limited to the mucosa, whereas the lesions in “peri-implantitis” sites extend to supporting bone (Lindhe and Meyle, 2008). Recently, the 7<sup>th</sup> EWOP has confirmed that the key diagnostic feature of peri-implant mucositis is the presence of bleeding on probing when using a force <0.25 Newtons. Moreover, the essential parameter for the diagnosis of peri-implantitis is evidence of progressive bone loss at the site of the implant (Lang and Berglundh, 2011). However, clinicians should remember to distinguish inflammation-induced bone loss from biological bone remodeling when diagnosing peri-implantitis. *Table 2* summarizes the definitions and the clinical characteristics of both peri-implant mucositis and peri-implantitis.

The incidence of implant loss varies from the type of prosthesis, location, and timing of implant loss. From a meta-analysis including studies with more than five years follow-up, the reported rate of implant loss prior to function was 2.16-2.53%. In the late stage, the incidence of implant loss was 2-3% and >5% with implant-supported fixed prosthesis and overdentures, respectively (Berglundh *et al.*, 2002). In addition, higher survival rates were reported in implants placed in partially edentulous patients compared with those in fully edentulous ridges (Esposito *et al.*, 1998; Goodacre *et al.*, 2003). A higher incidence of implant loss was observed in the maxilla in cases of patients who were treated with a full-arch prosthesis (Goodacre *et al.*, 2003). The incidence of peri-implant diseases varies from that reported in some previous literature because of a lack of consistent criteria/definition. The prevalence of peri-implant mucositis ranges from 38.9% to 90.9% (Fransson *et al.*, 2008; Rinke *et al.*, 2011). Similarly, bone loss has been reported in 10% - 28% of implants during various experimental periods (Fransson *et al.*, 2008; Karoussis *et al.*, 2004). An example may explain how the definition of disease affects the prevalence.

In 1999, Roos-Jansaker and co-workers reported that the incidence of peri-implantitis was 16% by their definition, i.e., more than 1.8 mm bone loss (e.g., 3 threads in the Branemark system) following the first year of function, although their results showed >56% of implants demonstrating bone loss  $\geq 1$  threads with or without bleeding on probing (BOP; Roos-Jansaker *et al.*, 2006).

Even though there are minimal absolute

contraindications to implant placement, several factors may contribute to implant loss and the reaction of peri-implant tissues. The primary etiology of biological implant complications is bacterial infection. The microbial profile of peri-implant disease is complex. In spite of the diversity, the most predominant species are Gram-negative anaerobic bacteria (Mombelli and Decaillet, 2011). Unlike the microbiota of successful osseointegrated implants (Lee *et al.*, 1999), periodontal pathogens (from both the orange and red complex) have been predominantly associated with peri-implant diseases (Al-Radha *et al.*, 2012; Charalampakis *et al.*, 2012). In a recent study by Al-Radha and coworkers, 22 patients with signs of peri-implant disease were evaluated and there was reportedly a positive correlation between the percentage of red complex bacteria and the severity of disease (i.e., pocket depth and gingival index; Al-Radha *et al.*, 2012). In addition to microbiota, environmental factors including plaque and individual susceptibility (Dereka *et al.*, 2012; Mombelli and Decaillet, 2011), smoking (Bain and Moy, 1993; De Boever *et al.*, 2009; DeLuca *et al.*, 2006; Vervaeke *et al.*, 2012), systemic diseases/past head and neck radiation (Anderson *et al.*, 2013; Marchand *et al.*, 2012; Moy *et al.*, 2005; Oates *et al.*, 2009; Yerit *et al.*, 2006), and periodontal stability (De Boever *et al.*, 2009) all can potentially influence the healing capacity of the host and ultimately affect the incidence of implant loss. Another factor to consider is bone density, as implants placed in type IV bone are more prone to failure than those placed in type I bone (Goodacre *et al.*, 2003). Next, implant-related factors include considerations such as implant length and diameter (Alsaadi *et al.*, 2008; Baqain *et al.*, 2012; Chung *et al.*, 2007; Monje *et al.*, 2012), and even modification of implant design has been introduced to control the effects of the microgap and minimize the reestablishment of biological width (Oh *et al.*, 2002; Tatarakis *et al.*, 2012). Moreover, peri-implant bone loss may result from surgical trauma (Eriksson and Albrektsson, 1984; Oh *et al.*, 2002) and implant malpositioning (Evans and Chen, 2008; Hermann *et al.*, 2000). Another important restorative/iatrogenic factor is residual cement. Among 42 implants with signs of peri-implant disease, Wilson found that 80.95% of the cases were associated with residual cement. The resolution of the clinical and endoscopic signs was observed in most of the cases (76%) 30 days after the cement was removed (Wilson, 2009).

To control inflammation and regain osseointegration, several decision trees have been proposed. In 1997, Lang and coworkers published a decision tree, named “Cumulative Interceptive Supportive Therapy (CIST).” In this chart, the treatment decision is based on the pocket depth, plaque index, morphology of defects and the presence of BOP (Lang *et al.*, 1997). Later, a flow chart was suggested by Mombelli. In this flow chart, the treatment is given according to the findings from clinical and radiographic

examination and microbial tests (Mombelli, 2002). In 2011, Okayasu and Wang recommended a decision tree for the management of peri-implant diseases. For the first time, the amount of bone loss was proposed to be a critical factor in determining treatment strategies (Okayasu and Wang, 2011). More recently, Aljateeli and colleagues recommended another decision tree to manage “peri-implant bone loss” (Aljateeli *et al.*, 2012). In this decision tree, both etiology and defect morphology were taken into consideration. Thus, it should be noticed that this is a guideline for the treatment of not only biological but also biomechanical implant complications. This purpose of this manuscript is to provide a guideline for the management of biological implant complications. In addition, common biological implant complications are discussed as well as a review of the currently available treatment strategies.

### **Decision tree: the management of biological implant complications**

Focusing on the management of biological implant complications (i.e., implant loss, peri-implant mucositis and peri-implantitis), a decision process is proposed in *Figure 1*. In addition to accurate diagnosis of the etiologic factors, the treatment modalities should be chosen based on the severity of peri-implant diseases, amount of bone loss and the morphology of peri-implant bony defects.

In order to control the inflammation and stop disease progression, numerous nonsurgical and surgical treatments have been proposed. To gain additional benefits, adjunctive therapy may be given such as antiseptics, or local and/or systemic antibiotics, as well as application of laser therapy. It should be kept in mind that it is difficult to compare the results of many of these studies because of the heterogeneity of experimental designs and the diverse definitions of peri-implant diseases that were used throughout the literature. Thus, clinicians should remember and take note of the clinical significance and potential applications of these treatments when interpreting these data.

### **Non-surgical approaches**

To disrupt the biofilm around implants, mechanical debridement has been applied using hand instruments, sonic instruments, ultrasonic instruments and air-abrasive devices. For the treatment of peri-implantitis, a double-blinded randomized trial was conducted by Renvert and co-workers (2009). Thirty-one patients were enrolled in the study and infected implants were treated using either titanium curettes or ultrasonics. Although there was improvement in plaque and bleeding scores, both treatment modalities failed to reduce pocket depth or bacterial counts during the 6-month experimental period (Persson *et al.*, 2010; Renvert *et al.*, 2009). The minimal effectiveness of mechanical debridement was also confirmed by Sahn

and colleagues (2011). Although the authors reported improved BOP scores with the air-abrasive device, the reductions in probing depth (PD) were less than 0.6 mm (Sahn *et al.*, 2011).

In contrast, positive outcomes have been demonstrated with treatment of peri-mucositis utilizing mechanical therapy. In both animal and human studies, research suggests that mechanical debridement alone is effective in controlling peri-implant mucositis in terms of PD reduction, clinical attachment loss (CAL) gain, plaque reduction and control of inflammation. However, the results of these studies did not lend support to the additional benefit of adjunctive antiseptic therapy in conjunction with mechanical treatment (Porras *et al.*, 2002; Trejo *et al.*, 2006).

As an adjunct therapy to mechanical debridement, local and systemic antibiotics have also been evaluated. Compared with chlorhexidine gel, significantly better outcomes have been observed with the use of minocycline microspheres for the treatment of peri-implant diseases. Additionally, the authors claimed that repeated antimicrobial therapy sustained the PD reduction and the level of microbial pathogens up to 12 months following treatment. Nevertheless, the mean PD reduction in the deepest pockets was 0.6 mm at 12 months in both the single and repeated antibiotic delivery groups (Renvert *et al.*, 2006; Renvert *et al.*, 2008). Significant but minimal benefits on probing attachment loss (PAL) gains (0.6 mm) were also observed with the use of doxycycline hyclate gel (Buchter *et al.*, 2004). On the other hand, limited studies have been conducted that investigate the effects of systemic antibiotics. In terms of reduction of bleeding index and PD, the use of ornidazole appeared to be effective, as was reported in a case series ( $n = 9$ ) with nine implants that had a 12-month follow-up (Mombelli and Lang, 1992). A recent randomized clinical trial with a larger sample size failed to show any benefit of systemic azithromycin administration in the treatment of peri-implant mucositis (Hallstrom *et al.*, 2012). Based on the scarcity of data that are currently available, more studies are needed to provide conclusive evidence regarding the effects of adjunctive systemic antibiotics for the treatment of peri-implant diseases.

In recent years, the application of laser therapy has been introduced to treat peri-implant diseases. Without surgical approaches, some studies were conducted to compare the effects of laser devices with mechanical debridement. A series of studies published by Schwarz and coworkers evaluated the non-surgical treatment outcomes of Er:YAG laser treatment within 12 months. In spite of significant improvement in BOP reduction during the experimental periods, the laser application only exhibited significant CAL gain at 3 and 6 months post-operatively compared with baseline. However, there were no significant differences in PD or CAL changes between laser-treated and control (mechanical debridement using plastic curettes in combination with

**Table 1.** Definitions of implant success

Albrektsson <i>et al.</i> , 1986	<ul style="list-style-type: none"> <li>• That an individual, unattached implant is immobile when tested clinically</li> <li>• That a radiograph does not demonstrate any evidence of peri-implant radiolucency</li> <li>• That vertical bone loss be less than 0.2 mm annually following the implant's first year of service</li> <li>• That individual implant performance be characterized by an absence of persistent and/or irreversible signs and symptoms such as pain, infection, neuropathies, paresthesia, or violation of the mandibular canal</li> <li>• That, in the context of the above, a successful rate of 85% at the end of a 5-year observation period and 80% at the end of a 10-yr period be a minimum criterion for success.</li> </ul>	
Smith <i>et al.</i> , 1989	<ul style="list-style-type: none"> <li>• The individual unattached implant is immobile when tested clinically</li> <li>• No evidence of peri-implant radiolucency is present as assessed on an undistorted radiograph</li> <li>• The mean vertical bone loss is less than 0.2 mm annually after the first year of service</li> <li>• No persistent pain, discomfort, or infection is attributable to the implant</li> <li>• The implant design does not preclude placement of a crown or prosthesis with an appearance that is satisfactory to the patient and dentist</li> <li>• By these criteria, a success rate of 85% at the end of a 5-year observation period and 80% at the end of a 10-year period are minimum levels for success.</li> </ul>	
Roos <i>et al.</i> , 1997	Grade 1	<ul style="list-style-type: none"> <li>• Absence of mobility is checked by individual stability testing of the unattached implant, using a light tightening force of an abutment screwdriver without simultaneous counteracting of the force via an abutment clamp. Any mobility or sensation/pain from the anchorage unit is regarded as a sign of lost osseointegration.</li> <li>• Radiographic evaluation of each implant reveals not more than 1.0 mm of marginal bone loss during the first year of loading, followed by not more than 0.2 mm resorption per year, as well as absence of peri-implant pathosis, such as a peri-implant radiolucency.</li> <li>• Severe soft tissue infections, persistent pain, paresthesia, discomfort, etc., are absent.</li> </ul>
	Grade 2	<ul style="list-style-type: none"> <li>• Radiographic evaluation of each implant reveals not more than 1.0 mm of marginal bone resorption during the first year of loading, followed by not more than 0.2 mm of resorption per year, as well as absence of peri-implant pathosis, such as peri-implant radiolucency.</li> <li>• Severe soft tissue infections, persistent pain, paresthesia, discomfort, etc., are absent.</li> </ul>
	Grade 3	<ul style="list-style-type: none"> <li>• Radiographic evaluation of each implant reveals not more than 0.2 mm of marginal bone resorption during the first year, but previously more than 1.0 mm of bone loss has taken place. Peri-implant pathosis, such as peri-implant radiolucency, is absent.</li> <li>• Severe soft tissue infections, persistent pain, paresthesia, discomfort, etc., are absent.</li> </ul>
ICOI implant health scale (Misch <i>et al.</i> , 2008)	Success (optimal health)	<ul style="list-style-type: none"> <li>• No pain or tenderness upon function</li> <li>• No mobility</li> <li>• &lt;2 mm radiographic bone loss from initial surgery</li> <li>• No exudate history</li> </ul>
	Satisfactory survival	<ul style="list-style-type: none"> <li>• No pain on function</li> <li>• No mobility</li> <li>• 2-4 mm radiographic bone loss</li> <li>• No exudate history</li> </ul>
	Compromised survival	<ul style="list-style-type: none"> <li>• May have sensitivity on function</li> <li>• No mobility</li> <li>• Radiographic bone loss &gt;4 mm (less than 1/2 of implant body)</li> <li>• Probing depth &gt;7 mm</li> <li>• May have exudates history</li> </ul>
	Failure (clinical or absolute failure): any of following	<ul style="list-style-type: none"> <li>• Pain on function</li> <li>• Mobility</li> <li>• Radiographic bone loss &gt;1/2 length of implant</li> <li>• Uncontrolled exudate</li> <li>• No longer in mouth</li> </ul>

**Table 2.** The definitions and the clinical characteristics of both peri-implant mucositis and peri-implantitis

	Peri -implant mucositis	Peri -implantitis
Definition	An inflammatory lesion that resides in the mucosa	An infectious disease that also affects the supporting bone
Characteristics	<ul style="list-style-type: none"> <li>•Bleeding on probing (&lt;25 Newtons force;key feature)</li> <li>•Redness</li> <li>•Swelling</li> </ul>	<ul style="list-style-type: none"> <li>•Changes in the level of the crestal bone</li> <li>•Bleeding on probing</li> <li>•Possible concomitant deepening of peri-implant pockets</li> <li>•The presence of pus</li> </ul>

0.2% chlorhexidine (CHX) irrigation) groups at any timepoint (Schwarz *et al.*, 2006a; Schwarz *et al.*, 2005). The result was further confirmed by later studies comparing the use of the Er:YAG laser with an air-abrasive device (Persson *et al.*, 2011; Renvert *et al.*, 2011). In regards to microbiological changes, a single episode of laser application may reduce the counts of *Fusobacterium nucleatum naviforme* and *Fusobacterium nucleatum nucleatum* within one month after therapy. Nevertheless, the antimicrobial effects failed to be maintained at the 6-month follow-up time point (Persson *et al.*, 2011).

### Surgical approaches

In the treatment of peri-implantitis, surgical approaches appear to be a predictable method over a short-term period (Renvert *et al.*, 2012). In general, surgical therapy consists of access flap surgery, degranulation and decontamination of the implant surface. To gain access and facilitate home care, resective surgery is performed either alone or in conjunction with implant surface modification (implantoplasty). In contrast, regenerative procedures should be considered to regenerate bone (Renvert *et al.*, 2012).

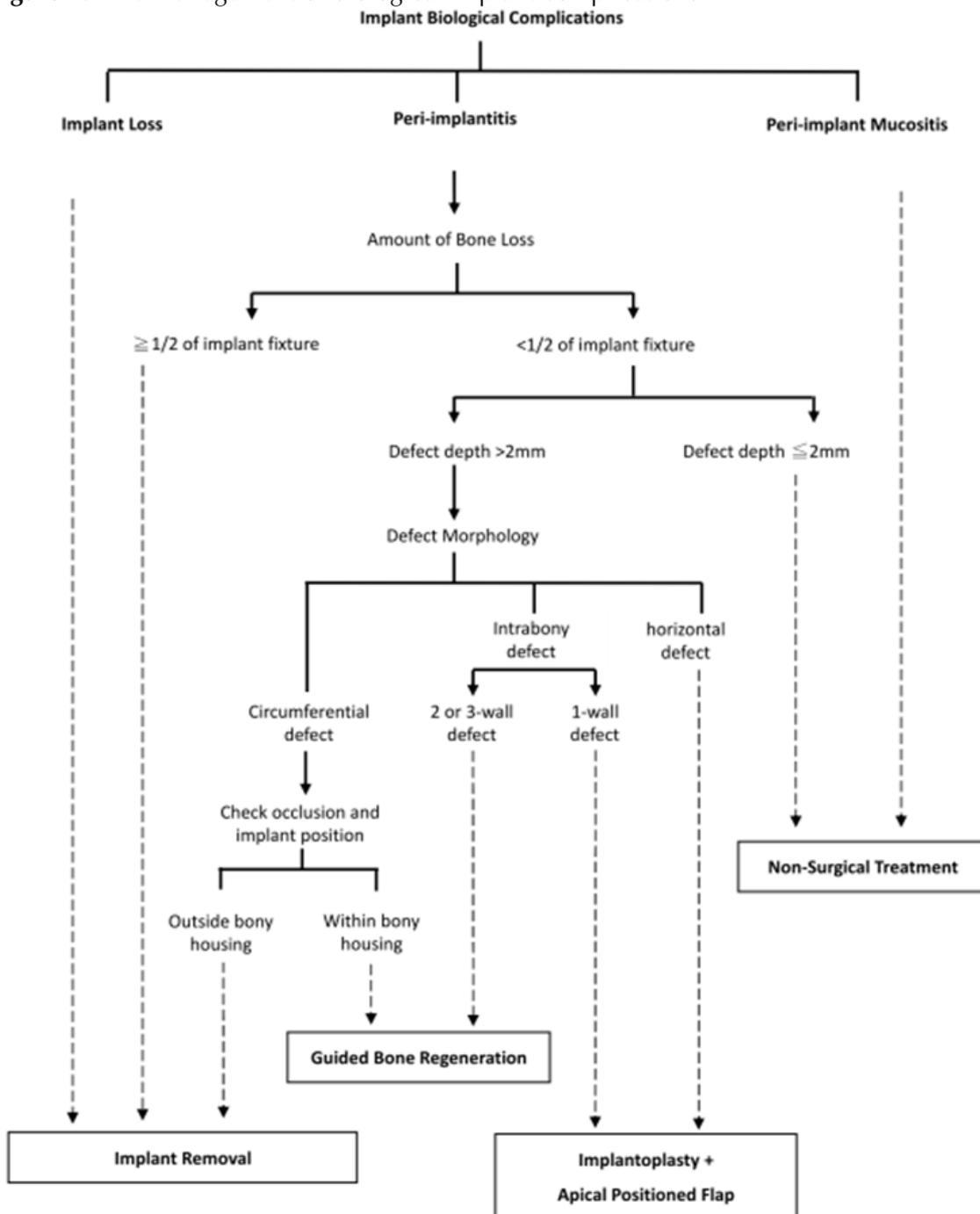
Resective surgery consists of an apically repositioned flap (APF) along with bone re-contouring, which ultimately leads to pocket reduction. With a 2-year follow-up, Serino and Turri (2011) reported positive outcomes of resective treatment on 86 implants with peri-implantitis. In addition, more implants (74%) with a minimal amount of initial bone loss (2-4 mm) returned to healthy status (no signs of peri-implant diseases) compared to those implants with >5 mm initial bone loss (40%; Serino and Turri, 2011). Without implantoplasty, a recent double-blind randomized controlled trial evaluated the effects of resective surgery with surface debridement on a total of 79 implants from 30 patients. Significant clinical improvements in terms of PD and BOP reduction were observed over a 12-month follow-up time period (de Waal *et al.*, 2013). On the contrary, some authors proposed that implantoplasty may augment the benefits

of resective therapy for peri-implantitis. In the comparative studies published by Romeo and co-researchers (2005, 2007), implantoplasty groups exhibited higher implant survival rates and less alveolar bone loss over the 3-year experimental periods. Better clinical results (lower PD, PAL and modified bleeding index) were also observed at the sites with surface modification (Romeo *et al.*, 2005; Romeo *et al.*, 2007). As for concerns of thermal changes during implantoplasty, an *in vivo* study indicated that minimal temperatures (approximately 1.5°C) were generated during implantoplasty with a properly selected bur and cooling system (Sharon *et al.*, 2011).

In addition to resective procedures, regenerative therapy is another treatment modality to re-establish osseointegration around a dental implant. Before any regenerative procedures can work, a surface detoxification must be done first. Decontamination of the implant surface can be performed by way of several methods. Similar to non-surgical mechanical debridement, the main goal of mechanical decontamination is to rupture the implant biofilm.

In addition, chemical modalities have been introduced to suppress bacterial load in peri-implantitis sites. They include hydrogen peroxide (Roos-Jansaker *et al.*, 2011; Roos-Jansaker *et al.*, 2007b), saline (Behneke *et al.*, 2000; Schwarz *et al.*, 2008), 35% phosphoric acid gel, CHX (Hammerle *et al.*, 1995; Khoury and Buchmann, 2001; Wiltfang *et al.*, 2012), citric acid (Khoury and Buchmann, 2001), and EDTA (Roccuzzo *et al.*, 2011). Another choice for implant surface decontamination is laser application, such as the CO<sub>2</sub> laser (Deppe *et al.*, 2007; Romanos and Nentwig, 2008), the diode laser (Bach *et al.*, 2000) and the Er:YAG laser (Schwarz *et al.*, 2011b). Despite decontamination modalities that have been widely applied in combination with surgical treatments, some authors questioned the effects of these procedures. In a recent meta-analysis, Renvert and coworkers refuted the benefits from laser decontamination (Renvert *et al.*, 2012). Compared with those who received conventional mechanical debridement (plastic curettes), the laser-treated group

**Figure 1.** The management of biological implant complications



did not exhibit better inflammation control (i.e., higher BOP reduction and CAL gain) in the treatment of advanced peri-implantitis (Schwarz *et al.*, 2011a). With resective surgery, the CHX/cetylpyridinium chloride (CPC) group achieved greater reduction of bacterial load, but failed to show any clinical superiority when compared to the control group (without CHX/CPC; de Waal *et al.*, 2013). However, it is difficult to compare the effects of different treatment modalities because more than one decontamination method has been used in most studies. Furthermore, systemic antibiotics were given in most of the studies. To reach optimum re-

osseointegration, decontamination of implant surfaces via chemical or mechanical techniques are still the most highly recommended (Subramani and Wismeijer, 2012). To date, there is no consensus on the indications and/or criteria for when to perform peri-implant regeneration. From the criteria of case selection in previous studies, the defect types that have been suggested include crater-like or saucer-shaped defects (Behneke *et al.*, 2000; Rocuzzo *et al.*, 2011; Wiltfang *et al.*, 2012), intrabony defects with  $\geq 3$  mm depth (Schwarz *et al.*, 2006b; Schwarz *et al.*, 2008) and  $>3$  threads of progressive loss (Roos-Jansaker *et al.*, 2011; Schwarz *et al.*, 2011b). As is

seen with the natural dentition, generally, the treatment of deeper defects is more predictable for regenerative purposes (Renvert *et al.*, 2012). Numerous grafting or barrier materials have been used either alone or together for purposes of peri-implant regeneration, such as autogenous bone grafts, bone blocks (Behneke *et al.*, 2000; Wiltfang *et al.*, 2012), xenografts (Schwarz *et al.*, 2009; Wiltfang *et al.*, 2012), alloplasts (Schwarz *et al.*, 2009), expanded polytetrafluoroethylene (e-PTFE) membranes (Jovanovic *et al.*, 1992; Khoury and Buchmann, 2001) and collagen membranes (Khoury and Buchmann, 2001; Schwarz *et al.*, 2009). Overall, promising outcomes have been reported with regeneration using bone grafts alone (Behneke *et al.*, 2000; Wiltfang *et al.*, 2012) or in combination with the barrier membranes (Roos-Jansaker *et al.*, 2011; Schwarz *et al.*, 2008). In contrast, some authors have claimed that the application of membranes may not resolve peri-implantitis (Jovanovic *et al.*, 1992; Roos-Jansaker *et al.*, 2007a; Roos-Jansaker *et al.*, 2007b). These results may derive from non-submerged techniques and the following membrane early exposure. However, Roos-Jansaker *et al.*, demonstrated in a case series that the submerged technique resulted in better treatment outcomes (Roos-Jansaker *et al.*, 2007a). Furthermore, Khoury and Buchmann reported a high incidence (58.6%) of post-operative complications in the membrane-treated sites (Khoury and Buchmann, 2001). In addition, a recent case series reported successful treatment with concomitant bone gain using a combination of enamel matrix derivatives (EMD), platelet-derived growth factors (PDGF), xenografts/allografts and collagen membranes/connective tissue grafts (Froum *et al.*, 2012).

To sum up, treatment modalities of implant biological complications should be determined with regard to three factors: the severity of disease, amount of bone loss, and the morphology of peri-implant bony defects. For implants with a large amount of bone loss or loss of osseointegration, implant removal is highly recommended because of the unfavorable treatment prognosis. Guided bone regeneration is indicated in peri-implant defects when there is bone loss affecting less than half of the implant fixture. Although there is no consensus among previous studies, peri-implant defects, including circumferential defects within bony housing and 2/3-wall intrabony defects, appear to have more regenerative potential. On the contrary, resective therapy (i.e., an apical positioned flap) should be considered in defects with moderate bone loss that do not have a favorable regenerative potential. Additionally, to reduce plaque accumulation and facilitate patient home care, treatment with implantoplasty is suggested at the time of resective surgery. Mild peri-implant disease cases can be maintained by non-surgical treatment modalities.

## Conclusion

With the increasing popularity of implant therapy, biological implant complications are important issues that cannot be ignored. In addition to comprehensive examination and a thorough treatment plan, proper surgical technique and regular maintenance play roles in the prevention of implant complications. Clinicians should be fully aware of the signs and symptoms of these complications and treat them as early as possible. Although more and more studies have been conducted in the treatment of peri-implant diseases, the effects of these treatment modalities should be evaluated in the future literature.

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