



Association
of Dental
Implantology

ADI GUIDELINES

On the Management of Peri-implant Diseases

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Scope of the Problem:

Long-term biological complications arising from peri-implant infection and inflammation result in progressive crestal bone loss (CBL) which puts the implant **at risk** of being lost. Although the technical and mechanical complications have been reduced up to a certain extent due to advances in implant technology and design, there is still no clarity on how to manage biological complications around implants. History of periodontal disease, presence of plaque, smoking, compliance and availability of adequate professional supportive therapies are important issues affecting implant patients. Furthermore, and based on the fact that in some studies it was shown that failures of implants may take place in clusters, some authors discuss the possibility for higher susceptibility of some patients to peri-implantitis which could be of similar aetiology as periodontal disease.

ADI Guidelines on Monitoring and Maintenance of peri-implant health have been published in a separate paper (see **ADI Guidelines on Monitoring and Maintenance**).

These guidelines are produced as a summary of ADI's Expert Panel Consensus meeting, held on 19th November 2012 in London, on the management of peri-implant diseases. The guidelines are not prescriptive protocols but good practice recommendations based on expert opinion informed by best available current evidence on the subject. Nevertheless, clinicians are strongly advised to seek up-to-date information and opinion from different sources when managing patients.

Introduction:

A cause-effect relationship has been demonstrated between dental plaque and peri-implant diseases (Mombelli et al, 2007). Reversible inflammation affecting the peri-implant soft tissues without any bony involvement is known as **peri-implant mucositis**. When this leads to **crestal bone loss** it is referred to as **peri-implantitis**, which is **irreversible**. Both conditions have similarities, respectively, to gingivitis and periodontitis in pathogenesis (Okayasu and Wang, 2011) but the disease progression might be faster around implants due to physiological differences in connective tissue morphology (Lang and Berglund, 2011).

Table 1: Aetiology of Peri-implant Diseases (PID):

Host Susceptibility
History of periodontal/peri-implant disease (strong evidence)
Genetic susceptibility (insufficient evidence)
Systemic disease/medical co-morbidities (e.g. uncontrolled diabetes, immunosuppression, radiotherapy) (insufficient evidence)
Prosthetic Design
Impassive fit (insufficient evidence)
Poor access for plaque control (good evidence)
Occlusal trauma or adverse loading conditions (inconclusive evidence)
Implant Design
Surface and/or geometry (inconclusive evidence)
Excessive stress concentration at Bone Implant Interface (mostly preclinical evidence)
Increased risk of plaque adhesion or retention (e.g. exposed endosteal implant surface)
Type of abutment connection (inconclusive evidence)
Patient Factors
Poor plaque control (strong evidence)
Smoking (strong evidence)
Bone quality and quantity (mostly preclinical evidence)
Iatrogenic Factors (dentist/surgeon related)
Surgical trauma (mechanical damage)
Implant malpositioning
Extrusion of excess luting cement*

This table summarises the biological and mechanical causative and predisposing factors that have been implicated in PID (see references) (Adapted from Heitz-Mayfield et al 2008).

* Cementitis is caused by extrusion of luting cement acting as plaque retention between the abutment and implant shoulder leading to peri-implant inflammation, fistula formation, discharge or bleeding. If untreated, this could also progress into deep seated infection with crestal bone loss. The use of radiopaque luting cements and satisfactory removal of all excess material after cementation is essential to eliminate cementitis.

Diagnosis of Peri-implant Diseases (PID):

Table 2: Peri-implant Mucositis:

Disease outcome: reversible

Signs and symptoms (S&S)		
Bleeding	Swelling	Pain or tenderness and/or fistula
Increased PD	Discharge or suppuration	Diagnostic criteria: no radiological sign of CBL

Table 3: Peri-implantitis:

Disease outcome: irreversible

Signs and symptoms (S&S)		
Bleeding	Pain or tenderness	Discharge or suppuration
Increased PD	Fistula	Diagnostic criteria: radiological sign of CBL
Swelling		

Table 4: Diagnosis of Peri-implant Diseases (PID):

To clinically diagnose the presence of peri-implant diseases the following should be evaluated:
Probing (using a calibrated probe <math><0.25N</math>): Increase in PPD (from a fixed reference point) indicates loss of attachment. Enables a clinical diagnosis of peri-implantitis.
BOP: Indicates inflammation. Diagnostic of peri-implant mucositis
Periapical radiography: Crestal bone loss confirms the diagnosis of peri-implantitis. Continual CBL indicates Implant at Risk (IaR)
Other S&S: Exudate; swelling and pain

Note: Any bone loss after implant loading that is observed is considered pathological (Berglundh, 2011)

Management of Peri-implant Diseases:

Treatment of Peri-implant Mucositis:

Non-surgical debridement, effective disruption of biofilm and plaque and improvement in personal oral hygiene with or without use of antimicrobials or antiseptics has been shown to be effective for the treatment of peri-implant mucositis, which is reversible (Heitz-Mayfield et al 2001; Lindhe and Meyle, 2008; Lang and Berglundh, 2011). Although peri-implant mucositis is indistinguishable from gingivitis in its pathogenesis, there is evidence that peri-implant mucositis may progress earlier than gingivitis. Therefore meticulous monitoring and supportive therapy individualised for each patient's circumstances is essential. The first line of treatment aims should be: effective plaque and biofilm disruption and control of predisposing factors (diabetes, smoking, plaque retention, occlusal trauma, inadequate access for oral hygiene etc). Clinical parameters should be periodically evaluated to control disease progression.

Treatment of Peri-implantitis:

Since peri-implantitis has an infective aetiology, its treatment is aimed at plaque removal, surface decontamination and anti-infective measures (Mombelli and Lang, 1998). Studies have shown that implant scaling and meticulous plaque removal should be the first line of treatment for eliminating peri-implant mucositis and early peri-implantitis lesions (Faggion and Schmitter, 2010). If there is no improvement in attachment levels and PPDs, surgical interventions aimed at better debridement and decontamination with or without adjunctive use of antibiotics should be considered as a second line of treatment. It should also be noted that according to Renvert (EFP 2008) non-surgical therapy does not result in treatment of peri-implantitis or resolution of the defect.

Table 5: Non Surgical Treatment Approaches (Subramani and Wismeijer, 2012)

Mechanical Cleansing		
Rubber cup	Titanium Scalers	Ultrasonic devices
Antiseptic Therapy		
Chlorhexidine rinse	Locally delivered antiseptics	
Antibiotic therapy		
Systematic antibiotics (e.g metronidazole and or amoxicillin)		Locally delivered antibiotics

Notes: The table lists some of the common non-surgical treatments for PID that have been cited in the literature

Treatment of Peri-implantitis and the Implant at Risk (IaR):

Although there is lack of reliable evidence to support the efficiency of surgical therapy, surgical procedures with or without bone regenerative techniques could be considered in order to control the disease progression and continual CBL (Faggion and Schmitter, 2010). Lack of evidence should not mean that there is no evidence that currently used techniques are not effective. On the other hand, there are studies that show that surgery alone does not guarantee success. For example, Charalambakis has shown that even after a surgical intervention, disease elimination and resolution occurs only in 47% cases. Nevertheless, regenerative techniques for the treatment of peri-implant intrabony defects have been reported successfully by several research groups (Schwarz et al, 2011; Rocuzzo et al, 2011; Roos-Jansaker et al, 2011 and Serino and Turri, 2011), but these techniques require general validation thus currently it is not known which is more effective intervention for treating peri-implantitis (Esposito et al, 2012). In this context it is proposed by some authors that early removal of a failing implant may be an advantage as this may prevent further and unnecessary loss of alveolar bone, thus reducing the complexity of future remedial treatment and the need for associated bone regeneration. At the same time though, evidence has shown that a second implant placed in a site of a previously failed implant has a high odds ratio for failure as well.

Objectives of Surgical Treatment:

The overall aim of the surgical treatment is to gain open access to the exposed implant surface and the associated intrabony defect for effective plaque and biofilm removal and surface decontamination. No one technique or method of surface decontamination (chemicals, lasers or air polishing) has been shown to be superior, although animal studies have confirmed that open decontamination was more effective in resolving peri-implantitis and promoting re-osseointegration compared with closed debridement (Lindhe and Meyle, 2008). Currently it is not known if use of systemic antibiotics may have any beneficial effect on peri-implant disease progression as an adjunctive therapy (Esposito et al, 2012; Subramani and Wismeijer, 2012). The goal of treatment is to stop the progression of crestal bone loss by controlling infection and inflammation (Subramani and Wismeijer, 2012). This may be achieved by a) conservative/access flap surgery for decontamination and disruption of the biofilm, b) respective therapy (e.g. apically positioned flap), c) regenerative therapy with the ultimate goal of achieving re-osseointegration d) explantation.

Table 6: Surgical Interventions for Peri-implantitis:

- a) Conservative/Access flap surgery: for decontamination of implant surface and decrease BOP
- b) Resective surgery (bone and implant surfaces including pocket depth including pocket depth reduction to improve PC)
- c) Regenerative surgery and re-osseointegration
- d) Explantation with or without concomitant grafting
- e) Re-implantation

Table 7: Outcome of PID Treatment Could be Affected by:

- a) Systemic Host factors (e.g. smoking/diabetes etc)
- b) Local factors (eg. defect type)
- c) Implant surface characteristics

Treatment Methods and Materials:

Long-term Monitoring and Outcome Measures:

Studies have identified certain risk factors and co-morbidities that may affect the pathogenesis and progression of peri-implant diseases. These include radiotherapy, uncontrolled diabetes, susceptibility to periodontal disease, bruxism and smoking. Although for some of these conditions the evidence is insufficient or inconclusive, these risk factors should be carefully monitored and managed in patients with dental implants. Diabetic control should be checked using HB1Ac tests at least annually and patients should be counselled to discontinue smoking. Meticulous plaque control and control of occlusal harmony becomes more critical in patients who are susceptible to PID.

Documentation of Long-term Monitoring of Implants:

- Records of individualised supportive therapy plan and recall attendance
- Periodontal indices including OH, BOP, pocket depth, mobility, recession and suppuration
- Baseline and serial radiographs
- Restorative parameters and function
- Technical problems
- Mechanical problems with abutments/screws etc
- Crestal bone support and maintenance of osseointegration
- General patient satisfaction including aesthetics

Success and Survival Criteria:

In absence of an objective test, implant success should be documented using surrogate outcome measures or treatment end-points such as pain, BOP, PD and crestal bone loss (CBL). Survival statistics simply report on the presence or absence of an implant at the time of evaluation and do not take into consideration the health of the peri-implant tissues, thus a failing implant with gross bone loss could be reported as “surviving”. It is mandatory for the clinician in charge of an implant patient’s maintenance to monitor and document implant outcome periodically and meticulously using the above-mentioned parameters in order to manage emerging problems in a timely fashion.

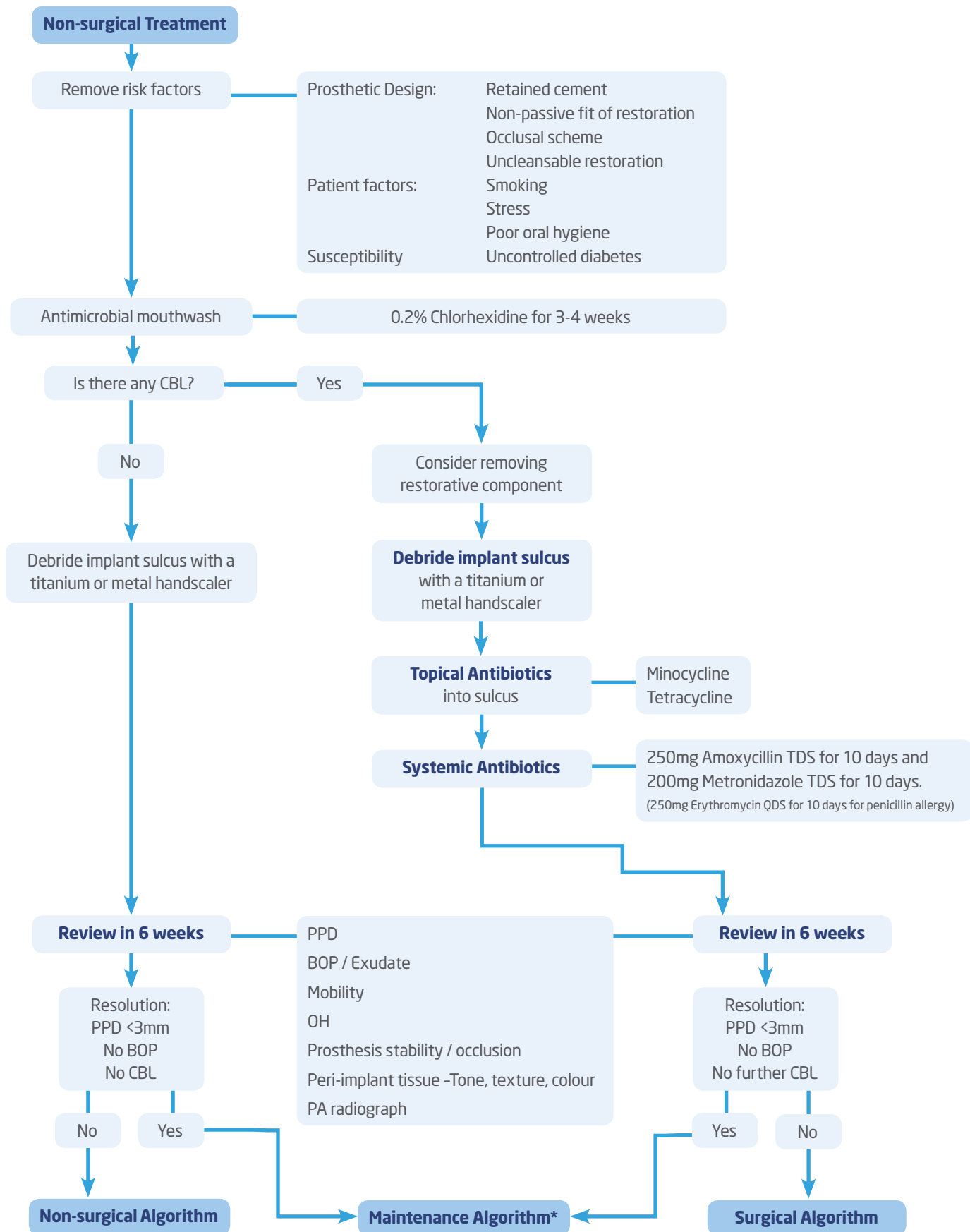
We suggest the following classification of implant success or failure:

Table 8: Definition of Implant Success and Failure:

Success	Failing
Stable marginal bone levels after an initial crestal bone loss of < 1.5mm No BOP PD < 4 mm No mobility Good plaque control (FMPI < 20%)	< 50% bone loss which has not stabilised Deep pocketing BOP Less than ideal PC Pain Exudate/discharge
At Risk	Failed
Initial bone loss of < 4mm but bone loss has been stabilised PD < 5mm No BOP immediate or delayed PI less than ideal	Deep pocketing Discharge BOP Mobility Lost or about to be lost

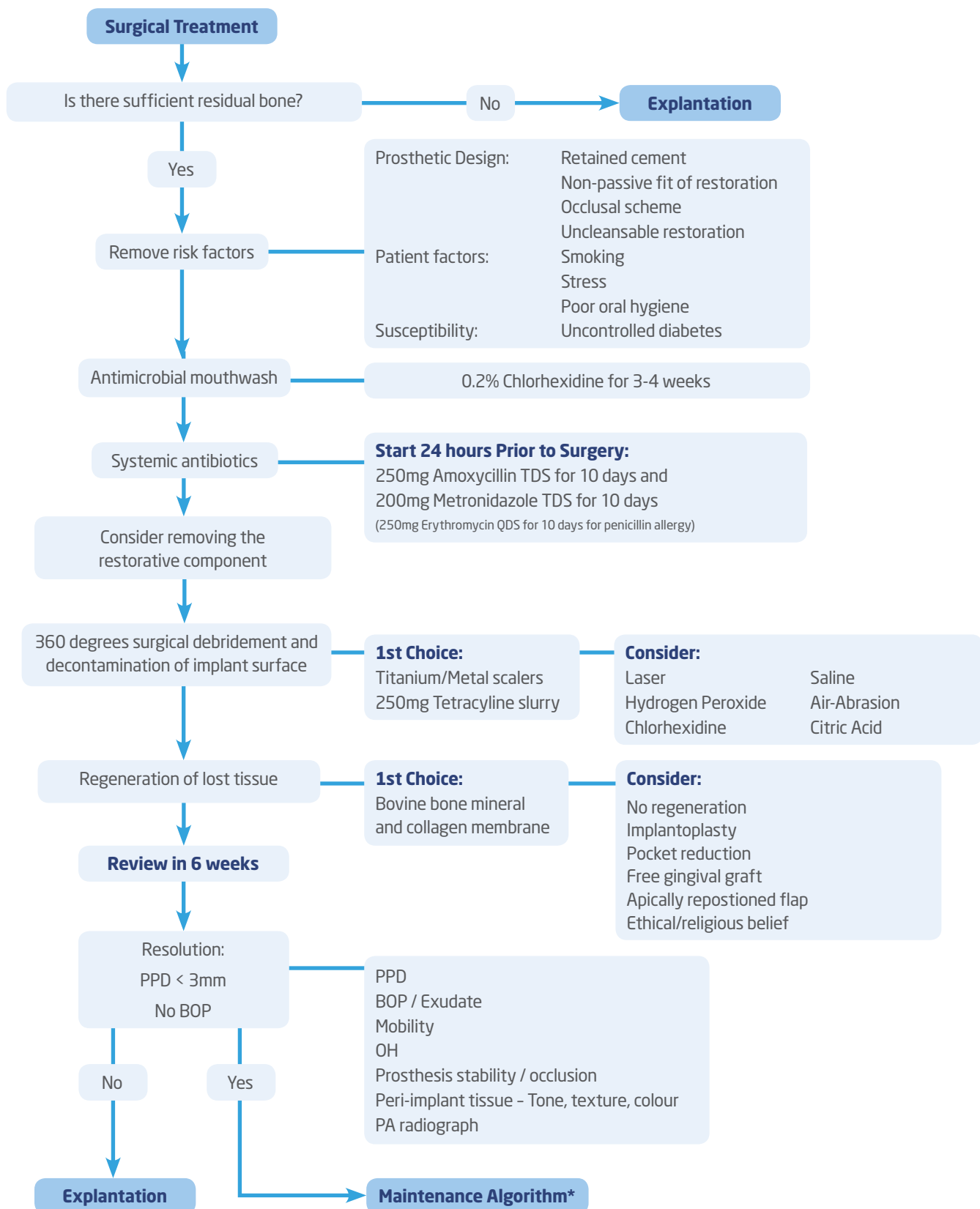
See next page:

Non-surgical Algorithm for Treatment of PID:



*Please refer to the ADI Guidelines on Peri-implant Monitoring and Maintenance for this algorithm.

Surgical Algorithm for Treatment of PID:



*Please refer to the ADI Guidelines on Peri-implant Monitoring and Maintenance for this algorithm.

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