by **Dr. Christopher Ho** and **Dr. Teck Tang**

Dr. Christopher Ho received his Bachelor of Dental Surgery with First Class Honours at the University of Sydney. He is a lecturer on aesthetic and implant dentistry locally and internationally and is involved with the evaluation and development of new dental products and materials. As a faculty member of the UCLA/Global Institute for Dental Education, his research interests are in immediate placement and loading of dental implants. Currently, he has a referral-based private practice in prosthodontic and implant dentistry in Sydney, Australia.

Dr. Teck Tang graduated with his Bachelor of Dental Science with honours at the University of Western Australia. He then completed his specialist training in Periodontics at the University of Adelaide and is a recipient of the APA scholarship where he received a student prize for his research publication. Currently, he works in restricted practices, specialising in Periodontics and Implant Dentistry and is a member of various organisations such as AOS (Australian Osseointegration Society) and ASP (Australian Society of Periodontology).

Fig.1: Peri-implant mucositis – bleeding on probing however no loss of bony attachment.

Failing implants, maintenance, recall

The introduction of dental implants has expanded the armamentarium of dental practitioners in replacing missing teeth, however implant rehabilitation is no longer restricted to restoring function, but it has become a multi-million industry driven by bone augmentation, soft tissue management and aesthetic restorations. Constantly, we, as dental practitioners, and our patients get bombarded with updates of dental implants by advertisements and other media. Very rarely we get to hear of the complications that may arise with implant rehabilitation.

In this article, we will deal with the issues of implant failures and complications and emphasise the importance of ongoing maintenance care.

Implant failures

Implant failures may be described as early or late. An early failure follows shortly after placement and osseointegration is never achieved. A late failure occurs in a successfully integrated implant some time after restoration.

Early failures

An early failure of an implant results from inability to establish an "intimate bone-to-implant contact" or osseointegration (Quirynen et al. 2002). This means that problems have occurred with the bone healing process after implant placement. Commonly, it is related to traumatic surgery and occurrence of micromotion at the interface during the critical post-implantation phase (Esposito et al. 2000).

Late failures

Late implant failures have been associated with both peri-implantitis and/or occlusal overloading (van Steenberghe et al. 1990, Quirynen et al. 2002). Since the

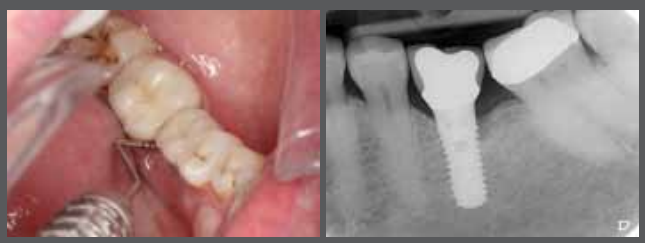

Fig. 2: Peri-implantitis – Inflammatory process characterised by early loss of peri-implant bone.

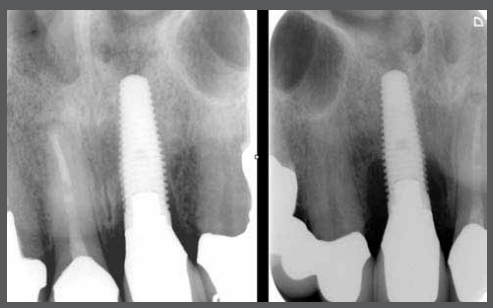

Fig. 3: Progression of peri-implantitis lesion with crater like loss of bone.

Fig. 4: Biologic and technical complications. Implants placed over 10 years ago, with technical complication in 16 implant with fractured abutment screw, and biologic complications with peri-implantitis and subsequent loss of bone.

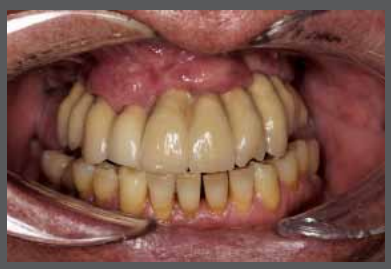

Fig. 5. Clinical picture of patient from Fig.4.

occlusal overload leads to failure shortly after implant fixture restoration this could be considered to be an "intermediate failure" and should be easily averted by careful and judicious treatment planning. Thus true failures are most likely attributed to peri-implant infections.

Implant survival and implant success

There has always been some confusion in the literature in terms of defining implant survival and implant success. Implant survival means the presence of implants irrespective of the conditions of the implants. Implant success means the presence of implants with no interventions required. As yet, consensus agreement on criteria of success has not been achieved (van Steenberghe et al, 1999).

Biological complications

Biological complications may include all the soft tissue complications that may arise from the implant reconstructions such as hyperplasia, excessive tissue swelling, peri-implant mucositis and peri-implantitis. It has been reported that 28 to 56 percent of subjects with dental implants suffer peri-implantitis while 10-15 percent of subjects exhibit severe peri-implantitis (Zitzmann & Berglundh 2008). These figures are interestingly very similar to the ones for periodontitis.

Peri-implant mucositis is defined as a reversible inflammatory process in the soft tissues surrounding a functioning implant, with no loss of bone. The inflammatory infiltrate adjacent to teeth and implants were found to be similar and suggests a similar host response in gingiva and peri-implant mucosa (Berglundh et al. 1992).

Peri-implantitis is an inflammatory process characterised by additional loss of peri-implant bone. It is important to recognise that peri-implantitis is not a synonym for "failing implant" or "ailing implant".

Susceptibility for periodontitis and peri-implantitis

Periodontitis is a multifactorial disease involving complex interactions between host and plaque, further modified by genetic and environmental factors. The most recognised factors related to susceptibility for periodontitis include plaque composition, smoking, genetics and various systemic conditions.

Since the bacteria cannot differentiate between implants and teeth, it would be reasonable to assume that all other factors for development of peri-implantitis remain the same for periodontitis. Therefore, the susceptibility for peri-implantitis should be dependent on the susceptibility for periodontitis.

Risk factors for peri-implantitis

Apart from a history of periodontitis, the other established risk factors for peri-implantitis include smoking and poor oral hygiene. It is imperative for clinicians to not only identify these risk factors but also to manage them to the best of their ability. This could include stabilising periodontal infections before implant therapy, providing

Risk factors for peri-implantitis
- History of periodontitis
- Smoking
- Poor oral hygiene
- Exposed threads
- Exposed surface coatings (roughened surfaces)
- Deep pockets (placed too deep, placed into deficiencies)
- No plaque removal access (ridge lap crown,connected prostheses)

Fig. 6: Risk Factors for Peri-implantitis.

Fig. 7: Poor design of fixed prosthesis with buccal flange. This does not allow cleaning of the tissue fitting surface of the bridge with subsequent soft tissue inflammation evident from food trapping.

Fig. 8: All-ceramic fixed implant supported bridge with convex tissue fitting surface allowing cleansability by the patient with superfloss, waterpick or interdental aids.

Fig. 9: Periodontitis v.s. Peri-implantitis. Note there is no periodontal ligament attachment with implants. (Courtesy of Astratech)

ongoing maintenance care, delivering smoking cessation advice and also designing prosthesis that would allows easy oral hygiene practices.

Does peri-implantitis progress faster than periodontitis?

Peri-implantitis is similar to periodontitis. They both involve alveolar bone loss. However, there are some differences. There is a zone of connective tissues being attached to the root surface in periodontitis. But in peri-implantitis, the connective tissue does not attach directly onto implants and there is no periodontal ligament, so the inflammatory lesion in peri-implantitis always extends closer to the bone surface (Gualini & Berglundh 2003). Therefore, it progresses faster and it is potentially a more aggressive disease and it is very hard to treat. Nevertheless, tissue degradation may be a slow process, as in chronic periodontitis, a function time exceeding 5 years for implants may be required to detect biological peri-implant complications.

Implants in patients with periodontitis

A number of studies have confirmed that patients with a history of periodontitis may yield lower success rate than patients without a history of periodontitis (Karoussis et al. 2004, Roos-Jansaker et al. 2006).

Aggressive periodontitis

So far, there are a few case reports for implant therapy in aggressive periodontitis patients. The evidence is not conclusive and some reported failure and some reported success (Fardal et al. 1999, Malmstrom et al. 1990, Yalcin et al. 2001, Wu & Chee 2007).

A study comparing implant success rates in aggressive periodontitis and chronic periodontitis patients showed that 3-year implant success rate is slightly lower in aggressive periodontitis patients, but still well above 95 percent with strict periodontal maintenance regime (Mengel & Flores-de-Jacoby 2005).

Refractory periodontitis

For refractory periodontitis, if the periodontal infections are not under control, implant therapy should be delayed.

Maintenance and recall

After successful periodontal and implant therapy the patient should be offered an individually tailored maintenance care program. It is important to assess mobility, probing depth, bleeding on probing and suppuration during a recall visit. Radiographic and microbiological parameters are to be added, depending on the primary clinical findings. In addition, the occlusion of the suprastructures should not be overlooked.

In the treatment of peri-implantitis, only limited scientific evidence is available to recommend any specific treatment modality. Most studies lack controls and randomization and are often handicapped by a small sample size. There are also limited reports on microbiological changes and histological changes following treatment.

Nevertheless, a systematic approach for monitoring tissues around implants in the prevention and treatment of peri-implant disease has been recommended by Lang and coworkers in Berne, Switzerland. This systematic protocol, referred to as Cumulative Interceptive Supportive Therapy (CIST), contains four cumulative treatment modalities (A–D)(Figure 10 Table 1). Each step of the procedures is used in a sequential manner with increasing antibacterial intervention, combined with surgical resective/regenerative treatment (A+B+C+D). The

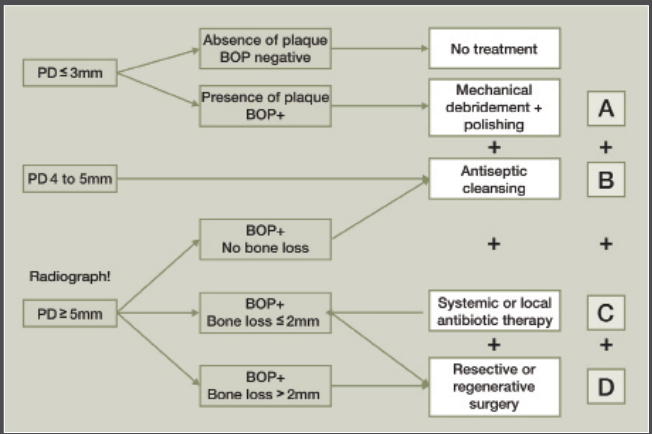

Fig. 10: *Cumulative Interceptive Supportive Therapy* (CIST). (Lang et al, 2004).

Table 1. Cumulative Interceptive Supportive Therapy (CIST) modalities (Lang et al, 2004).
A. Mechanical cleansing using rubber cups and polishing paster, acrylic scalers for chipping off calculus. Instruction for more effective oral hygiene practices.
B. Antiseptic therapy. Rinses with 0.1% to 0.2% chlorhexidine digluconate for 30 seconds using approximately 10ml, for 3 to 4 weeks, supplemented by irrigating locally with chlorhexidine (preferably 0.2% to 0.5%) using a Luer syringe or local
chlorhexidine gel application.
C. Antibiotic therapy:
1. Systemic ornidazole (2 x 500 mg/day) or metronidazole (3 x 250 mg/day) for 10 days or combination of metronidazole (500 mg/day) plus amoxicillin (375 mg/day) for 10 days.
2. Local: application of antibiotics using controlled release devices for 10 days (25% Tetracycline fibres).
D. Surgical approach:
1. Regenerative surgery using abundant saline rinses at the defect, barrier membranes, close flap adaptation and careful post-surgical monitoring for several months. Plaque control is to be assured by applying chlorhexidine gels.
2. Resective surgery. Apical repositioning of the flap following osteoplasty around the defect.

CIST protocol has been shown to be effective in improvement of clinical and microbiological parameters in clinical studies (Mombelli et al. 2001, Persson et al. 2006).

Conclusions

Dental implants are an excellent option for replacing missing teeth, but problems may arise with such treatment. Early implant failures occur at a global rate of about 2.5%. Late implant failures are mainly related to peri-implantitis. In addition, technical and biological complications are common in implant therapy.

Susceptibility for peri-implantitis is associated with susceptibility for periodontitis. Therefore, every partially edentulous patient should receive appropriate periodontal screening and treatment prior to implant therapy. It is reasonable to place implants in periodontitis patients but they are at much greater risks of developing problems.

Comprehensive treatment planning is paramount with regular recall and maintenance necessary to detect and intercept problems early. Instruction in oral hygiene and smoking cessation advice should be given. For periodontitis patients, regular supportive periodontal therapy and smooth and well contoured transmucosal abutments are required for the long term success of implant therapy.

The cumulative interceptive supportive therapy protocol can be adopted in the prevention and treatment of peri-implantitis. However, there is limited scientific evidence to recommend any specific treatment modalities for peri-implantitis, with more clinical controlled trials required for the management of this problem. **DA**

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