FOREWORD

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ALBERTA SOCIETY OF DENTAL SPECIALISTS

PERI-IMPLANTITIS; STATE OF THE ART REVIEW AND OPINION PAPER

Part 1: *Diagnosis and Prevalence of Peri-Implantitis: Risks factors for Peri-implantitis*

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Definitions & Prevalence

Peri-implantitis is defined as the "inflammatory lesion of the soft tissues surrounding an endosseous implant AND Progressive crestal bone loss." This differentiates it from peri implant mucositis that is just inflammatory lesion of the soft tissues surrounding an implant with no bone loss. Peri implant mucositis is a critical diagnostic factor in peri-implantitis since bone loss at an implant can be caused by other factors such as failure to position the implant fully in bone, remodeling from abutment micro-gap area and failed bone grafting. The presence of bleeding on probing and progressive bone loss are thus the key features of peri-implantitis.

Prevalence is complicated by the variability in diagnostic criteria, for example, one review showed that the peri-implantitis prevalence reported in literature ranges from 1% to 47%. The consensus estimate is that 10 % implants and 20% patients have peri-implantitis (EAO Consensus 2012). The actual number though is still unknown, as there have been numerous thresholds used to determine what amount of bone loss and soft tissue condition represents actual disease.

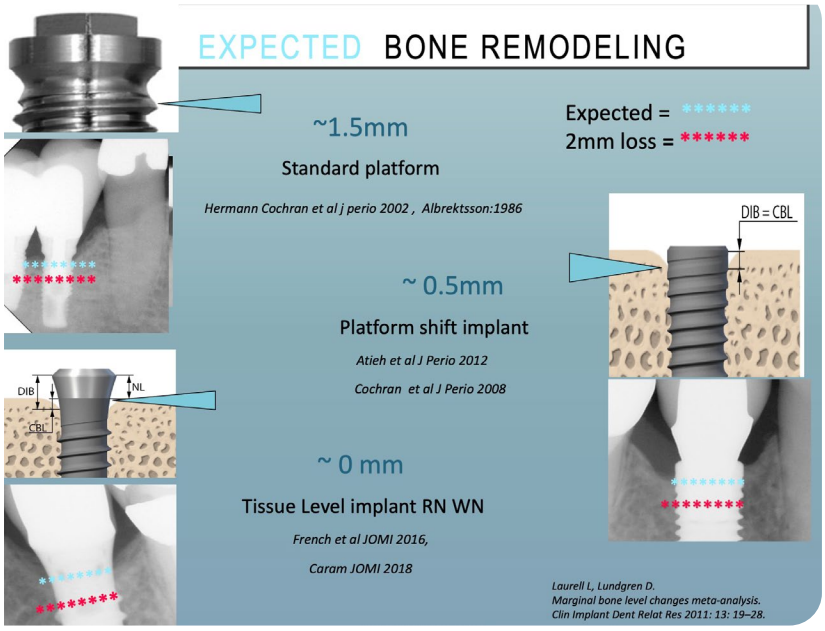
The definition proposed by Workgroup 4 of the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions highlights the need to monitor the radiographic bone loss in a longitudinal basis to validate the hypothesis that bone loss has occurred as consequence of pathology. Accordingly, a radiographic examination taken at a given timepoint should be compared to the baseline radiographic bone level (6-12 months after prosthesis). Otherwise, the following criteria can be used for the diagnosis of implants without baseline data:

- Presence of bleeding and/or suppuration on probing
- Probing depths of ≥6 mm
- Bone levels ≥3 mm from the implant neck - or - smooth rough interface.

How much bone loss is normal

Personally, I disagree with the above bone threshold of 3mm (although I do accept if you have no history on a case and inherit an implant case with some bone loss it is hard to know if it was a surgical placement issue or actual disease related bone loss). Nonetheless, if a 3mm loss is used, then the prevalence is underestimated and also action to treatment is delayed until about ¼ of the implant length has lost bone; this is simply too late. On my cases we use 1mm threshold from the known stage 2 level or the "expected" bone level as indicating bone loss to disease if inflammation is also present. The expected level of bone remodeling is as follows; tissue level = no bone loss past smooth rough interface, platform shift = 0.7mm from implant neck and standard platform = 1.5mm from abutment connection (French et al JOMI 2016 & Atieh et al J Perio 2012 & Cochran et al J Perio 2002).

Figure 1: Expected Bone Remodeling indicated by blue line and 2mm threshold by red line



BOP is meaningless, unless you grade it and include suppuration

To evaluate inflammation and soft tissue status at a dental implant is not so obvious as just scoring bleeding on probing. Most indices used in periodontal disease do not apply well to implants because implants differ in frequency, sensitivity and specificity for bleeding and suppuration as compared to teeth. Bleeding on probing (BOP) score as a "yes/ no" binary score is commonly used but is flawed because implants have a high false positive for BOP. This means if an implant has BOP + it does not predict bone loss. That is why I developed a graded index that includes both suppuration and degrees of BOP using a controlled force probe to overcome the limitations of the older indices for implants that led to the over-diagnosis mucositis in the current literature.

The rate of mucositis using the novel Implant Mucosal Index (IMI) compared to strict BOP criteria versus relaxed BOP criteria (which ignored light single point bleeding) revealed the prevalence of mucositis was 38.6% versus 14.2% strict versus relaxed, respectively. The prevalence of peri-implantitis was 4.7% and 3.6% when using strict versus relaxed criteria, respectively (French. J Periodontol. 2019). It is important to note that both IMI = 0 and IMI = 1 had no bone loss, which means even if an implant has a light BOP it is not at risk. There is doubling of bone loss with each subsequent IMI increase: IMI 2 = 0.33mm, IMI 3 = 0.71mm, IMI 4 = 1.52mm, thus, there is a predictive potential to find at risk cases (Mean IMI at 4 years, P value <0.01, French Cochrane Ofec JOMI 2016).

Table 1: Implant Mucosal Index, scored using controlled force probe of 17gram.

Score	Description
0	No
1	Minimal point
2	Moderate multiple-point bleeding
3	Profuse, multiple-point bleeding
4	Suppuration

The implant mucosal index (IMI) is now recognized as the most accurate diagnostic index for dental implants as proven in animal studies Monje et al J. Perio 2018) and is also the default index used in the world's largest database on dental implants, the Straumann "Registry" (www.straumann.com/en/discover/implant-registry-app.html).

Probing depth not diagnostic but is a relative value and indicates potential for risk

First, let us all agree that you need go probe dental implants. The arguments against probing implants are unfounded. Briefly, I will discount those myths before we discuss what is a normal probe depth

for implants. Does it damage the epithelial connection? No, it is reattached by one day and fully reformed by five days (Etter et al COIR 2012). Does it damage surface implant? There is no damage even with metal tip probe (Fakhravar et al Imp Dent 2012).

Does it transfer periopathogens from teeth to implants? No, the subgingival microbiome is resistant to change and returns to baseline by one week even if pathogens intentionally introduced (Christersson et al J Perio 1985).

Now on to what is a normal pocket depth (Pd) for a dental implant. In fact, unlike teeth where a 1-3mm is a normal healthy depth, there is no exact number for implants but they typically probe 2-4mm. The reason P implants as opposed to teeth are due to many factors such as, prosthetic contours limiting access, depth of implant placement relative to adjacent teeth, tissue inflammatory status, as well as probe type and probe pressures. A very good article on the topic of probe force is by Cha et al JOMI 2019, where they show probe force and dimensions standardized tip pressure of 120Ncm2 improves the diagnostic value to minimize false positive bleeding on probing on implants. This is achieved by a 0.4mm tip (Marquis) at 15 N or by a 0.5mm tip (UNC) at 23 N. A follow up study reported that most clinicians probe 2-3x higher than 120Ncm2 thereby, increasing the risk for high false positive BOP at implants. Therefore, given the weak hemidesmosome attachment to the implant/abutment surface, light probing is suggested (0.15gr-0.17gr), but this has been overlooked in most studies and accounts for the lack of diagnostic value and inter-examiner agreement in earlier studies as reported by Merli et al J clin perio 2014, using older BOP + /- scores ONLY 52% of examiners agree on peri-implantitis diagnosis.

Schøu et al. found that even mild marginal inflammation was associated with deeper probe penetration around implants in comparison to teeth where probe depth varied by 0.5mm on but varied by 1.5mm on implants with mucositis.

Even though, unlike teeth deeper probing depth on implants is not evidence of disease, this does indicate increased risk for disease where a Pd of ≥ 6mm is a recognized risk factor for the development of peri-implantitis.

Implants are NOT teeth, they are medical devices with potential for Peri-implantitis Tsunami

For a given amount of biofilm implants have more bone loss than teeth (AAP World Workshop, J Periodontol. 2018) because there is no periodontal ligament, there is only a weak epithelial hemidesmosome attachment.

Implants were once thought to be "resistant to periodontal disease" (Van steenbergh et al. J perio 1993) but this was a mistake of circumstance. Implants in the 1980s were machined surfaces (smooth) so did not readily harbour perio-pathogens, and implants were done in fully edentulous cases that had lost the perio-pathogen reservoir. The introduction of partial rough surface (Sa2) compared to machined implants had an "early stage benefit" in that it doubled bone to implant contact (BIC) from about 40% to about 75% of implant surface so implants integrated more reliably and faster. The rougher surfaces however have a "late stage disadvantage" of increased risk for peri-implantitis progression (ie; Straumann SLA and Nobel Ti-unite vs machined surface) have about 10x more bone loss once peri-implantitis is initiated (Berglundh et al, COIR 2007, Albouy et al, JCP 2012). Furthermore, by the late 1990s implants were routinely placed in dentate patients, many of whom lost teeth to periodontal disease so perio-pathogens present. Add to this the demand for esthetics and trend toward subgingival abutment connection (ie. bone level BL vs tissue level TL design) where the internal connection harbours pathogens at level of the bone that causes bone loss related to microgap and related exposure of rough surface which is then prone to peri-implantitis progression. Our own data on TL vs BL comparing over 7000 implants shows a double "intervention rate" required to control active peri implantitis disease, with BL intervention rate of 6% versus TL rate at 3%.

By 2012 more implants were placed by General Dentists than highly trained specialists and the ADA's own study (Da Silva et al, J Am Dent Assoc. 2014) showed drastically increased peri implant bone loss in cases treated by General Dentists versus Specialists, with 18% of implants losing 2mm of bone by 4 years, so > 3 x more bone loss as compared to our published number of 5% at 5 years (French, Cochran, Ofec JOMI 2016).

Given the shift to rough surfaces, treating dentate patients, subgingival abutment connection and shift to General Dentist placement; the stage is set for the "Peri-implantitis Tsunami" that we now see.

Risk factors for peri-implantitis

The bottom line in reducing peri-implantitis risk is placing and keeping the rough surface fully in bone and placing implants in healthy patients. Any bone loss that exposes rough surface increases the risk for progressive peri-implantitis.

The risk factors for peri-implantitis can be broken down into five categories: surgical site factors, implant factors, prosthetic factors, patient factors and provider factors.

Site factors

Implants placed in grafted bone, or with a bone graft at time of placement have about 2.5 times more bone loss on longer term follow up (French et al, J Periodontol 2019), (Shatta et al JOMI 2019). It is important then to also consider that an implant placed in a grafted socket is, by definition, an implant that is in grafted bone. Socket grafting, though a popular technique, is not the standard of care and overall has a poor evidence base being mostly animal studies and case reports and has no long-term follow up (the longest being only four years). Implants placed in narrow ridges where there is < 1.5mm bone volume around the implant are also known to lose bone during healing that exposes rough or threaded surfaces.

Implants placed immediately do not necessarily have more peri-implantitis risk but this is a more complex procedure and a novice surgeon is prone to errors that may lead to exposed rough surfaces over time.

The need for keratinzed tissue is debatable, with some studies showing no effect if OH is good (Wenstrom Derks. COIR 2012) where as some studies show more bone loss, especially if a bone level implant is used as the lack of soft tissue thickness leads to bone loss to develop a biologic width (Linkivicius series 2009 JOMI).

Implant factors

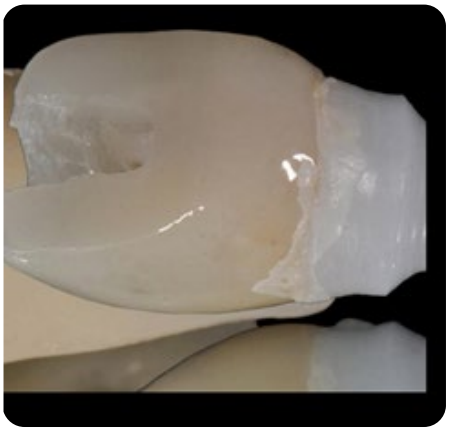
Tissue level implants with a smooth collar are the ONLY implant designs that do not lose bone from remodelling, the median bone loss is zero meaning the bone is at the smooth rough interface (French et al JOMI 2016) (Santiago et al 2018 JOMI). As previously noted, all other designs lose bone from the micro-gap of abutment connection; standard platforms like external hex and biocare replace lose 1.5mm of bone and platform shift designs that typically lose 0.7mm thus potentially exposing rough surface. There is some confusion driven by false promises from some companies that a conical internal connection does not leak. This is false, due to micro-mobility all internal connections harbour bacteria, usually within two weeks of loading (Tripodi et al J appl biomater. 2015) and the space harbours pathogens that are the same that cause peri-implantitis (Tallarico et al, J prost res. 2017). Using chlorhexidine in the connection does not prevent the problem (Koutouzis et al, CIDRR. 2015).

High insertion torque taper and aggressive thread designs can lose bone from the zone of necrosis that develops at the crestal bone area, this is especially found in dense bone cases, underprepared sites where insertion torque is over 50 Ncm (Stavropoulos, Cochran, et al . Adv Dent Res. 2016). Implant brand also matters, some implants show more bone loss over time (Derks et al JDR 2016) with an odds ratio of 3-5 x more bone loss as compared to Straumann Tissue level designs.

Prosthetic Factors

Retained Cement is certainly a risk factor for peri-implantitis and it is not the fault of the clinician to miss subgingival cement if the margins are sub-gingival. Linkevicious (COIR 2013) showed that only supragingival margins can be fully removed of residual cement, hence, if using cement retained solutions then always keep margins supragingival or only 1mm subgingival at facial margin only. The sad reality is this detail is lost in most custom abutment designs where the dentist does not direct the lab to really follow the gingival margin. The other sad reality is this typically does not become problematic until longer follow up of 6-12 years, so may be missed by the dental office that initially treated the patient. (Wilson et al J Periodontol. 2015)

Figure 2. Residual cement on a custom abutment even when treated by Perio-Prosth team (photo courtesy Dr S. Leziy)



Favourably, technology moved on and we have a variety of titanium bases that can be cemented in the lab and then delivered as screw retained solutions. This works very well if the screw access in line with occlusal fossa or cingulum but in 80% of anterior sites, proper implant alignment leads to a screw access at – or – facial to the incisal edge. Enter the "angle correcting" abutment systems to solve this problem and they work well BUT there are limitations, mostly due to manufacturers designs that typically use a 45 degree emergence of 1mm collar height for the angle correcting systems (ie Straumann angle correcting variobase). The science however shows that a 45 degree emergence on a bone level implant causes bone loss (Souza et al COIR) whereas a 15 degree , more narrow emergence preserves bone. This, in my opinion, is the biggest problem with platform shift designs but it is a problem that can be managed with communication with the lab and the use of more customized Ti-bases.

The other prosthetic factor that needs to be discussed is oral hygiene access.

This is especially problematic for fixed hybrid style prosthetics or platform shift implants in molars where an overhang is created. The lack of good OH access has been shown to have a 65% predictive value for peri-implantitis (Serino & Strom CIOR 2009).

Patient Factors

Various patient factors can affect peri-implantitis risk such as smoking, periodontal disease history, diabetes, bisphosphonates and auto immune disease.
Of these, a prior history of periodontal disease and smoking are most common and the most critical.

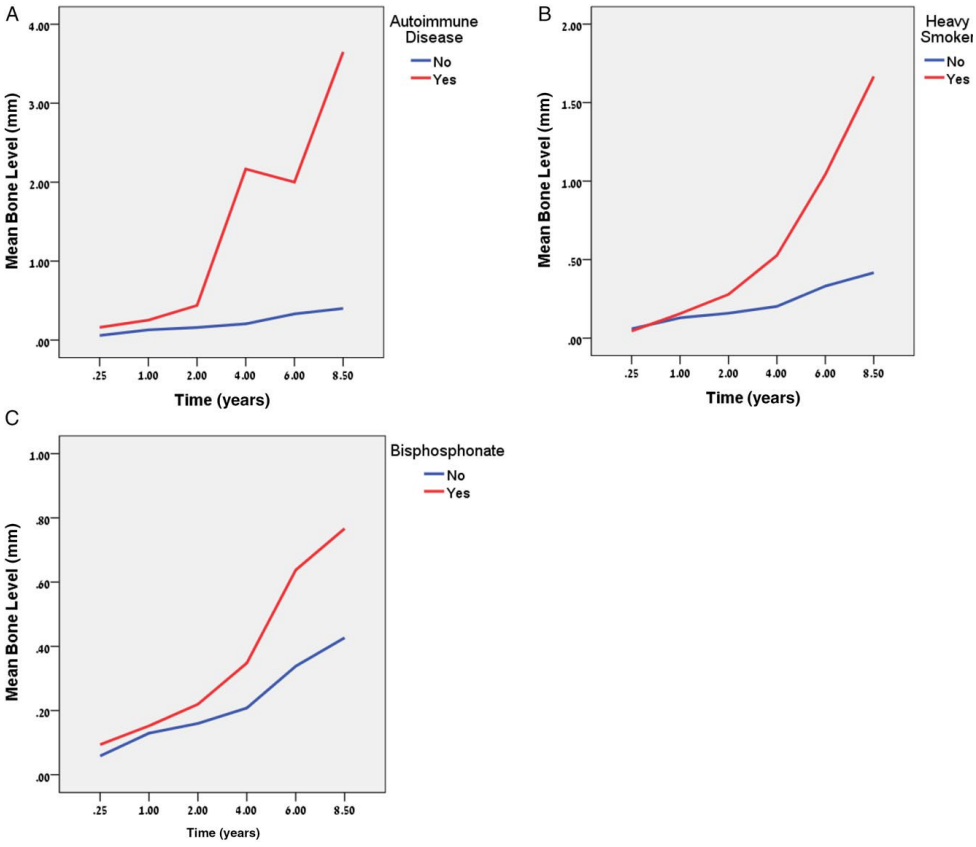
A history of periodontal disease is a known risk factor for developing peri-implantitis. One large retrospective cohort study of 588 patients with 2277 implants were followed by a nine year exam with clinical and radiographic analysis. The prevalence of moderate-severe peri-implantitis was 14% patients that had an average bone loss of 3.6mm and the authors report a 4x risk for peri-implantitis in patients with hx of periodontal disease. (Derks et al. Journal of Dental Research 2016). The risk posed by periodontal disease is also increased depending on the category of periodontal disease, for example, Mengel et al (J Periodontol. 2001) reported significantly more bone loss in "aggressive" disease profile patients (GAgP) that had 14x greater risk of peri-implantitis as opposed to Derks reported 4 x risk. It is critical that potential implant patients be evaluated for periodontal disease and if any noted then treat the periodontal disease prior to placing implants, furthermore in some cases one may choose a different implant system or surface as well as more conservative treatment protocols in a patient with periodontal disease history.

Smoking, especially if defined as heavy smoking (> 15 cigarettes per day), in our data on this with 4591 Straumann implants followed up to ten years revealed a significantly greater bone loss of 0.9mm more loss on average at four year (P<0.01). (French et al J Perio 2018). At the start (Stage 2) there was no difference in bone loss between heavy smokers and non-smokers but there was a significant interaction with time. Hence, in smokers one can expect at least 1mm bone loss by four to five years and more over longer period.

Other studies support this with Kan et al (J Prosthet Dent 2002) a five year bone loss > 1mm in 18% of non- smokers versus in 35% of smokers.

The best visual image to have in mind as to the impact of smoking on peri-implantitis is the red line in composite Figure 3, if you follow up long enough, bone loss is a certainty in smokers.

Figure 3.



Auto immune Patients

Patients on steroids and/or with known autoimmune conditions may be more prone to bone loss over time. There is not much in the literature on this and one of the first studies to report this was our research on 4591 straumman implants followed over ten years. Patients on steroids or with known autoimmune disease had at least 2mm more bone loss by six to eight years than healthy cohorts. Of note, is that it was not a factor of surgical healing between stage 1 and 2 as there was early bone loss effect in either group but there was an increase of loss increases over time P≤0.04. (French et al J Perio 2018).

Osteoporosis and Bisphosphonate Medication

As previously published in our implant survival report (French et al COIR 2015) and in in a systematic review of bisphosphonates and oral implants (Madrid C, Sanz M. COIR 2009) bisphosphonate therapy for osteoporosis, did not impact implant survival. However, in our analysis of bone loss over time it did pose as a significant risk for MBL over time (Figure 1C). This is a unique finding in the literature (French et al J Perio 2018). Land may reflect altered remodeling potential of bone, or it may also be the effect of a few outlier cases and the number of cases relative to larger cohort was small. More research is needed to verify if there is a longer-term impact on peri-implantitis.

Diabetes

Uncontrolled diabetes can hinder bone formation, delays wound healing and causes a reduction in BIC and bone volume and so complicates implant survival in addition to increasing risk for peri-implantitis. However, controlled diabetes does not affect implant survival or peri-implantitis significantly as shown by Chrvanovic (J dent res 2014) in a meta-analysis the risk ratio of 1.07 (95% ci) so effectively no difference on survival and had a minimal impact on peri-implant bone loss. What matters then is to ensure the patient is well controlled. This can be done by Hb1Ac test that measures glycosylated hemoglobin providing a snapshot of past six weeks blood sugar levels as opposed to single blood sugar test. The American Diabetes Association (ADA) defines good diabetic control at a cut-off of (Hb1Ac) at 7%.

Summary

Peri-implantitis is a complex problem for patients and clinicians to manage. It is estimated to be about 10% of implants placed but this may be an over or underestimation depending on the thresholds used to diagnosis and the numerous risk factors that can affect a single outcome. It is very important for clinicians to be aware or the risk factors and treat with additional caution regarding implant types and procedures used on patients who present with various risk factors. For example, an extraction immediate implant in a heavy smoker who also has a history of periodontal disease may not be the "best" plan.

Treatment of peri-implantitis is difficult and not always predictable, it is best to avoid the problem in the first place with proper diagnosis and planning.

Treatment will be discussed in part two of this review and opinion paper in the coming months.