Peri-implantitis diagnosis and treatment by New Zealand periodontists and oral maxillofacial surgeons.

Assil A Russell, Andrew Tawse-Smith, Jonathan M Broadbent, Jonathan W Leichter

ABSTRACT

Objectives: This study aimed to investigate the understanding, diagnosis and management of peri-implantitis by New Zealand periodontists and oral maxillofacial surgeons (OMFS).

Design: Telephone interviews (in combination with a postal and electronic survey) were conducted of all 25 periodontists and 32 OMFS listed as specialists on the New Zealand Dental Register. A seven item multi-choice and short answer questionnaire was used to investigate: their definition of peri-implantitis; the number of annual referrals received in their practice for this condition; their diagnostic, preventive and treatment strategies for peri-implantitis; and their perception of the role of general dental practitioners in its management.

Results: The participation rate was 84.6%. Most respondents defined peri-implantitis as a disease of multifactorial aetiology that leads to destruction of the bone supporting an implant. The average number of cases seen annually differed between periodontists (11 cases/year) and OMFS (4 cases/year). The criteria used by the respondents to diagnose peri-implantitis included increased probing depths and radiographic evidence of bone loss. Each type of specialist used mechanical debridement for treatment, but a higher proportion of OMFS performed surgical procedures as treatment. The prevention strategies used smoking cessation advice and ensuring good plaque control. All respondents agreed that peri-implantitis is an important disease that can lead to implant failure, and all acknowledged the role of general dental practitioners in diagnosis, referral for treatment and long-term implant maintenance.

Conclusion: The definition, diagnostic criteria and management strategies used by New Zealand specialists are generally consistent with those found in the literature. No evidence-based, gold standard treatment protocol for peri-implantitis has been identified in the literature, and New Zealand specialists use a range of treatment modalities.

INTRODUCTION

Dental implants are an integral part of modern dentistry. In New Zealand, it is estimated that 1.02% (95% CI 0.57, 1.81%) of adults (aged 18 and over) have at least one dental implant. Put another way, 1 in every 100 adults has an implant, and we are 95% sure that the true figure is somewhere between 0.6% (1 in 167) and 1.8% (1 in 56). Implants are twice as common (1 in 50) among those in the least deprived quintile and half as common among those in the most deprived quintile.1

Peri-implant disease is an umbrella term that refers to all inflammatory lesions that develop in tissues surrounding dental implants (Zitzmann and Berglundh, 2008). It includes the two main conditions of peri-implantitis and peri-implant mucositis. In 2008, the 6th European Workshop on Periodontology defined peri-implantitis as an inflammatory disease characterised by the destruction of the supporting bone around an osseointegrated, functioning dental implant (Lindhe and Møyle, 2008; Zitzmann and Berglundh, 2008). This inflammation can compromise the longevity of an implant and, if left untreated, may lead to implant loss (Esposito et al., 2003).

Mombelli and Lang proposed a guideline in 1998 for the detection and treatment of peri-implantitis. It is commonly known as Cumulative Interceptive Supportive Therapy (CIST) and is based on clinical findings of plaque, bleeding on probing, suppuration, pocket depth and bone loss (Mombelli and Lang, 1998). CIST is limited, in that it does not completely address the multifactorial aetiology of peri-implantitis. Table 1 presents a summary of the suggestions outlined in the Mombelli and Lang CIST protocol. These form a stepwise set of responses to increasingly severe manifestations of the condition.

The number of patients with dental implants is likely to rise in future, and peri-implant disease will become an increasingly common presenting condition. General dental practitioners and specialists alike will require understanding of this disease and its clinical management, but achieving that is complicated by a lack of consensus in the literature on the best diagnostic criteria and treatment modality for peri-implantitis.

This study was undertaken to investigate the way in which NZ-registered periodontists and oral maxillofacial surgeons (OMFS) define, diagnose, and manage peri-implantitis.

METHODS

A survey was conducted of all 25 periodontists and 32 OMFS listed as specialists in the New Zealand Dental Council register. Specialists who did not hold a current practising certificate were excluded, along with whose contact details were not disclosed, or who were not based in New Zealand. Telephone interviews were used in combination with postal and electronic questionnaires. A seven question multi-choice and short answer questionnaire was

1 Personal communication – 2009 national oral health survey data analysed on 17 January 2013 by Professor WM Thomson
designed following a review of the literature. Questions focused on the specialists’ definition of peri-implantitis, the average number of cases seen annually, the way in which they diagnose and treat peri-implantitis, any preventive strategies they have in place, and their perception of the role of the general dental practitioner (GDP) in its management. Specialists were also asked how significant a problem they perceived peri-implantitis to be, and whether they thought it would become more significant with time.

A cover letter, consent form and postage-paid envelope was provided. Responses were collected over a period of four months. Questionnaires were first sent by post, and then by email to those who had not responded. Non-responders were telephoned and invited to complete the survey by telephone interview. All three survey modalities contained the same set of questions, and 68% of respondents completed the electronic questionnaire, 27% used the postal option, and 5% were interviewed by telephone.

The responses were collated and data entered into an electronic database. All analyses were cross-tabulations, with the chi-square test used to examine the statistical significance of observed differences. Statistical analyses were conducted in Intercooled Stata 10.0.

**RESULTS**

The overall response rate for the survey was 84.6% (22 periodontists and 26 OMFS responded). When respondents were asked to provide a definition of peri-implantitis (using an open-ended question), they consistently described peri-implantitis as an inflammatory disease that affects the supporting tissues around dental implants. Only five of the specialists described it as occurring around implants in function. Periodontists reported seeing an average of 11 cases of peri-implantitis in the previous year (range 1 to 32), whereas OMFS reported an average of 4 cases (range 0 to 10). Periodontists

### Table 1. Cumulative Interceptive Supportive Therapy (adapted from Mombelli and Lang, 1998)

<table>
<thead>
<tr>
<th>Clinical Findings</th>
<th>Recommended Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plaque and bleeding on probing</td>
<td>Regimen A&lt;br&gt;Mechanical cleaning with rubber cup and instruments softer than titanium. Oral hygiene instruction</td>
</tr>
<tr>
<td>Suppuration with/without bone loss 4-5mm pocket depths</td>
<td>Regimen B&lt;br&gt;Regimen A + local antiseptic (0.2% chlorhexidine irrigation of peri-implant pockets twice daily.)</td>
</tr>
<tr>
<td>Pocket depth &gt;5mm Radiographic evidence of early bone loss</td>
<td>Regimen C&lt;br&gt;Regimen B + systemic antimicrobials specific against anaerobes</td>
</tr>
<tr>
<td>Advanced bone loss</td>
<td>Regimen D&lt;br&gt;Regimen C + surgical intervention for guided tissue regeneration and/or to correct tissue morphology</td>
</tr>
<tr>
<td>Loss of osseointegration</td>
<td>Regimen E&lt;br&gt;Removal of implant</td>
</tr>
</tbody>
</table>

### Table 2. Peri-implantitis diagnosis and preventive care by periodontists and oral & maxillofacial surgeons

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Periodontists N (%)</th>
<th>Oral &amp; Maxillofacial Surgeons N (%)</th>
<th>Overall N (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinical probing</td>
<td>21 (95.5)</td>
<td>22 (84.6)</td>
<td>43 (89.6)</td>
</tr>
<tr>
<td>Radiographic bone loss</td>
<td>21 (95.5)</td>
<td>22 (84.6)</td>
<td>43 (89.6)</td>
</tr>
<tr>
<td>Previous history of periodontitis</td>
<td>6 (27.3)</td>
<td>4 (15.4)</td>
<td>10 (20.8)</td>
</tr>
<tr>
<td>Implant mobility</td>
<td>4 (18.2)</td>
<td>11 (42.3)</td>
<td>15 (31.2)</td>
</tr>
<tr>
<td>Implant exposure and gum recession</td>
<td>6 (27.3)</td>
<td>13 (50.0)</td>
<td>19 (39.6)</td>
</tr>
<tr>
<td>Other</td>
<td>5 (22.7)</td>
<td>0 (0.0) a</td>
<td>5 (10.4)</td>
</tr>
<tr>
<td><strong>Preventive treatment/strategies</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Regular scaling</td>
<td>22 (100.0)</td>
<td>7 (26.9) c</td>
<td>29 (60.4)</td>
</tr>
<tr>
<td>OHI and plaque control</td>
<td>21 (95.5)</td>
<td>19 (73.1) b</td>
<td>40 (83.3)</td>
</tr>
<tr>
<td>Smoking cessation</td>
<td>22 (100.0)</td>
<td>18 (69.2) a</td>
<td>40 (83.3)</td>
</tr>
<tr>
<td>Treatment of periodontitis</td>
<td>22 (100.0)</td>
<td>13 (50.0) c</td>
<td>35 (72.9)</td>
</tr>
<tr>
<td>Patient selection (prior to implant treatment)</td>
<td>10 (45.5)</td>
<td>4 (15.4) a</td>
<td>14 (29.2)</td>
</tr>
<tr>
<td><strong>Overall</strong></td>
<td>22</td>
<td>26</td>
<td>48</td>
</tr>
</tbody>
</table>

a P<0.05  b P<0.01  c P<0.001
reported that 84% of the cases seen were referrals, whereas OMFS reported this proportion to be 34%. The majority of the referrals to either specialty had been made by general dentists, followed by other dental specialists and dental hygienists.

The diagnostic criteria applied by respondents are summarised in Table 2. Most (84% of periodontists and 69% of OMFS) reported using clinical probing and radiographs. Some 16% of periodontists and 34% of OMFS reported assessing implant mobility in diagnosis.

Prevention strategies applied by the specialists are summarised in Table 2. In general, a higher proportion of periodontists than OMFS reported using preventive measures.

Table 3 summarises how frequently periodontists and OMFS apply various treatment modalities for peri-implantitis treatment. Both specialties commonly used mechanical debridement with plastic scalers. Proportionally more OMFS than periodontists reported never using local antisepsics, and chlorhexidine was the only agent specified. A higher proportion of OMFS than periodontists carried out surgical procedures to treat peri-implantitis. No respondents reported using the CIST protocol. The use of lasers was the least common treatment modality reported.

The majority of periodontists (68%) believed peri-implantitis to be a “very” significant disease, whereas the majority of OMFS (73%) believed it to be “somewhat” significant. No respondents considered it to be insignificant. Most (91% and 74% respectively) reported believing that it will become more significant with time.

Some 71% of periodontists and 58% of OMFS agreed that GDPs should be able to diagnose the condition and have preventive strategies (such as OHI and monitoring plaque control) in place for all patients with dental implants in order to maintain peri-implant health. Both specialist groups recommended that GDPs refer cases to them for treatment of the disease.

**DISCUSSION**

This study set out to investigate the way in which NZ-registered periodontists and oral maxillofacial surgeons (OMFS) define, diagnose, and manage peri-implantitis. It found that, although they were able to define peri-implantitis, most believed that peri-implantitis is a disease which does not affect implants in function (contrary to the current literature).

Studies of peri-implantitis have indicated a broad range of prevalence estimates for the condition (Fransson et al., 2005, Roos-Jansaker et al., 2006). Fransson and colleagues investigated 3413 Branemark implants in 662 patients and found that 27% of patients in their study had developed peri-implantitis based on radiographic evidence of progressive bone loss (Fransson et al., 2005). Other studies with smaller sample sizes produced more modest findings (Roos-Jansaker et al., 2006). Karoussis et al. (2004) studied 89 patients with 166 dental implants and observed that 15% of implants developed peri-implantitis over 10 years. Their findings were based on the criteria of probing depth greater than 5mm, bleeding on probing, and radiographic evidence of bone loss greater than 0.2mm annually. Methodological differences (in definitions and diagnostic criteria) among studies (and different inclusion and exclusion criteria for patients) limit their comparability. There are no New Zealand prevalence estimates for peri-implantitis, but the current study showed that the two surveyed specialties differ in their referral rates.

Ideally, diagnostic criteria for the condition should be sensitive and accurate enough to detect its initial signs and symptoms (Mombelli and Lang, 1998). At present, there are no universally standardised diagnostic criteria specific for peri-implantitis. Nonetheless, many authors agree that a combination of deep (>5mm) clinical probing, the presence of bleeding, and the presence of suppuration with evidence of radiographic bone loss are useful indicators (Heitz-Mayfield and Lang, 2010; Karring et al., 2005; Klinge et al., 2005; Kotsovilis et al., 2008; Mombelli and Lang, 1998; Schou, 2008).

In the current study, a number of respondents (24% of periodontists and 13% of OMFS) reported using history of previous periodontal disease as a diagnostic tool. Although periodontitis is a known risk factor for developing peri-implantitis and can be included in the assessment of the patient, the dental literature does not support it being used in the diagnosis of peri-implantitis. This study also revealed that implant mobility is used as part of

**Table 3.** Peri-implantitis treatment modalities used by periodontists and oral & maxillofacial surgeons

<table>
<thead>
<tr>
<th>Treatment Modality</th>
<th>Frequency of use N (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Never</td>
</tr>
<tr>
<td><strong>Mechanical Debridement</strong></td>
<td></td>
</tr>
<tr>
<td>Periodontists</td>
<td>2 (11.1)</td>
</tr>
<tr>
<td>Oral &amp; Maxillofacial surgeons</td>
<td>4 (19.0)</td>
</tr>
<tr>
<td>Overall</td>
<td>6 (15.4)</td>
</tr>
<tr>
<td><strong>Surgical Procedures</strong></td>
<td></td>
</tr>
<tr>
<td>Periodontists</td>
<td>1 (5.6)</td>
</tr>
<tr>
<td>Oral &amp; Maxillofacial surgeons</td>
<td>2 (10.5)</td>
</tr>
<tr>
<td>Overall</td>
<td>3 (8.1)</td>
</tr>
<tr>
<td><strong>Pharmacological Therapy</strong></td>
<td></td>
</tr>
<tr>
<td>Periodontists</td>
<td>1 (11.1)</td>
</tr>
<tr>
<td>Oral &amp; Maxillofacial surgeons</td>
<td>4 (16.7)</td>
</tr>
<tr>
<td>Overall</td>
<td>5 (15.2)</td>
</tr>
</tbody>
</table>

* P<0.05  
  * Question not answered by 9 periodontists
diagnosis, mostly by OMFS (34% of OMFS, 16% of periodontists). This finding is likely to be of clinical significance because implant mobility—although part of patient assessment—is not sensitive in detecting early stage peri-implantitis and is thus limited as a diagnostic factor (Chen and Darby, 2003). Even though bone may have been lost from the coronal portion of the implant (indicating the presence of peri-implantitis), the implant can remain relatively immobile and stable as long as there is some bone-to-implant contact (Mombelli, 2002; Mombelli and Lang, 1998). These findings highlight a need to emphasise the use of acceptable diagnostic tools by New Zealand specialists.

Similarly, there is no gold standard for the treatment of peri-implantitis (Klinge et al., 2005; Kotsovilis et al., 2008). Nevertheless, the literature identifies various treatment strategies, such as mechanical debridement, antiseptics, and antibiotic therapy, surgical procedures or laser intervention. The objective of any treatment should be to disrupt the biofilm contributing to the disease, stop further progression, and return peri-implant tissues to a healthy state.

Mechanical debridement may damage and roughen the surface of implants if carried out with instruments which are harder than titanium (Mombelli and Lang, 1998). The roughened surface will (in turn) make implants more susceptible to biofilm adherence, potentially exacerbating the disease process. Thus, plastic or carbon fibre hand/ultrasonic scalers or rubber polishing cups are preferred over metallic instruments (Mombelli and Lang, 1998). Encouragingly, the current study found that New Zealand specialists were found to consistently prefer plastic over metal scalers.

For deeper peri-implant pocketing, mechanical methods alone may be inadequate in the management of the plaque biofilm, and other strategies may be needed to resolve the inflammatory lesion (Kotsovilis et al., 2008). Pharmacological interventions include the use of antibiotics as an adjunct to mechanical debridement or surgery. Antiseptics such as chlorhexidine are also used. Both specialist types in the current study reported using it to treat peri-implantitis. However, it is not known whether this was used as the main treatment option or whether it was used in combination with mechanical and surgical interventions (the latter option is preferred).

The surgical treatment of peri-implantitis usually involves raising a mucoperiosteal flap to expose the contaminated implant surface; this is followed by mechanical or laser therapy (Heitz-Mayfield and Lang, 2010). Both periodontists and OMFS reported using this approach, and all did so in combination with mechanical and or pharmacological intervention. This is supported by the dental literature, which suggests the use of surgical procedures as an adjunct to mechanical and pharmacological therapy (Heitz-Mayfield and Lang, 2010).

Surgical procedures also include implantoplasty, and one OMFS reported using it. Implantoplasty essentially involves the removal of rough implant threads that have become exposed following bone resorption, to provide a smoother implant surface that is less plaque-retentive (Romeo et al., 2005; Suh et al., 2003). Studies on this technique have shown promising outcomes when it is used alongside mechanical and pharmacological therapy (Claffey et al., 2008; Kotsovilis et al., 2008; Romeo et al., 2005; Suh et al., 2003). The use of lasers (such as CO2, diode, Nd:YAG and Er:YAG lasers) has also been suggested for the treatment of moderate to advanced peri-implantitis, where there are lesions with 4mm or greater pockets, radiographic bone loss and suppuration or bleeding on probing (Kotsovilis et al., 2008, Romanos et al., 2003, Kreisler et al 2002). Lasers were found to be the least common treatment modality used by New Zealand specialists.

Despite the lack of agreement in the literature on the best treatment approach, the CIST protocol can be a useful starting point for clinicians (Mombelli and Lang, 1998). In this study, none of the respondents reported using the CIST or other treatment protocols, and it appears that there is no systematic use of treatment guidelines by New Zealand specialists.

The identification of disease risk factors is crucial for appropriate patient selection and the development of targeted prevention protocols. A review of the available literature reveals that the risk factors for peri-implantitis include poor oral hygiene, a history of periodontitis, smoking, diabetes mellitus and genetic predisposition. There is evidence that poor plaque control is associated with greater incidence of disease (Heitz-Mayfield and Lang, 2010; Tonetti, 1998). Patients with a previous history of periodontal disease have been found to be more susceptible to peri-implantitis (Kotsovilis et al., 2008; Quirynen and Teughels, 2003; Schou, 2008; Tonetti, 1998). A review by Klinge and colleagues (2005) suggested that patients with a history of periodontitis have a higher incidence of peri-implantitis and experience earlier implant failure than those with no previous periodontal disease. Tobacco smoking is another well-documented disease risk factor. Its effects are dose-dependent, and it has been associated with greater radiographic marginal bone loss and consequently greater rates of implant failure (Heitz-Mayfield and Lang, 2010; Klinge et al., 2005; Schou, 2008; Tonetti, 1998). The current study found that, across all categories and risk factors, periodontists placed a greater emphasis on prevention than did OMFS. This suggests a need to establish standardised prevention protocols for New Zealand specialists.

The increasing significance of peri-implantitis is a common view held by both periodontists and OMFS. None of the specialists considered it to be insignificant. Despite this, there is a lack of New Zealand prevalence studies and limited consensus in the literature on its diagnosis and treatment. This highlights the need for further research.

CONCLUSIONS

The definition, diagnostic criteria and management strategies applied by New Zealand specialists are mostly in line with the current literature. There appears to be no set treatment protocol outlined in the literature, and New Zealand specialists use varying treatment modalities. Mombelli and Lang’s Cumulative Interceptive Supportive Therapy (CIST) protocol can be a useful reference point, but it is not universally accepted. All specialists surveyed acknowledged that peri-implantitis is significant and may become more so with time. They perceive the role of the general dentist in managing peri-implantitis to be one of prevention, diagnosis and referral.
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REFERENCES

AUTHOR DETAILS
Assil Amir Russell BDS
River Road Dental Centre
608 River Road, Hamilton

Jonathan Leichter BA, DMD, CertPeriodontology
Andrew Tawse-Smith DDS, CertPeriodontology
Jonathan Broadbent BDS, PGDipComDent, PhD
Sir John Walsh Research Institute
School of Dentistry
The University of Otago
PO Box 647
Dunedin

Corresponding author: Dr. Assil Russell
assilrussell@yahoo.com

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