Peri-implant Mucositis

Peri-implant mucositis has been defined as a reversible inflammatory reaction in the soft tissues around a functioning implant with no bone loss. This paper reviews the prevalence, etiology, risk indicators, prevention, and treatment of mucositis. Relying on the literature concerning mucositis, the bacterial etiology is discussed as well as the varying ranges of prevalence, reported to be from 20% to 80% of subjects (13% to 62% of implants) after a minimum of 5 years of implant function. A discussion of the definition of mucositis questions the assumption of it being completely reversible following treatment and challenges the concept of mucositis “transfer” (conversion) to peri-implantitis.


Stuart J. Froum, DDS
Eduardo González de la Torre, DDS
Paul S Rosen, DMD, MS

Peri-implant mucositis has been defined as a reversible inflammatory reaction in the soft tissues surrounding a functioning implant. This has been differentiated from peri-implantitis, which has been described as an inflammatory reaction of the peri-implant mucosa with loss of supporting bone in the tissues surrounding a functioning implant.1 Mucositis, although discussed and studied extensively, has been compared to gingivitis and is considered by many to be easily diagnosed, prevented, managed, and successfully treated (complete resolution of the inflammation).2 It has also been assumed that untreated or unsuccessfully treated mucositis can progress to peri-implantitis and result in the loss of an affected implant(s).3

The aim of the present paper was to review the prevalence, etiology, risk indicators, prevention, and treatment of mucositis and evaluate whether mucositis is completely reversible following treatment, as determined by complete elimination of bleeding on probing (BOP). Moreover, the paper will discuss if mucositis that is untreated or unsuccessfully treated will invariably lead to peri-implantitis.

1Ashman Department of Periodontology and Implant Dentistry, New York University College of Dentistry, New York, New York; Private Practice, New York, New York, USA.
2Ashman Department of Periodontology and Implant Dentistry, New York University College of Dentistry, New York, New York, USA.
3Department of Periodontology, Baltimore College of Dental Surgery, University of Maryland Dental School, Baltimore, Maryland, USA; James Cook University Dental School, Cairns, Australia; Private Practices, Yardley, Pennsylvania and New York City, New York, USA.

Correspondence to: Dr Stuart J Froum, 17 West 54th Street, Suite 1 c/d, New York, NY 10019, USA.

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Materials and Methods

Search Strategy

An electronic Medline (PubMed) search was performed for articles published from 1993 to 2018. The search was limited to the English language. Additional full-text articles of narrative and systematic reviews between 1994 and 2018 were obtained. An additional hand-search was performed to identify relevant studies by screening the systematic reviews as well as the reference lists of all obtained full-text articles. Search terms were “mucositis” or “peri-implant mucositis” combined with each of the following terms: “prevalence,” “etiology,” “risk indicators,” “prevention,” “treatment,” “progression,” or “conversion” to peri-implantitis.

Inclusion criteria: Human or relevant animal studies, reviews (including systematic reviews and meta-analyses), and clinical studies with at least a 5-year follow-up, including clinical trials, controlled clinical studies, comparative studies, chart studies, and congress/consensus reports.

Exclusion criteria: In vitro and preclinical studies, reports based on questionnaires, studies not meeting the inclusion criteria, studies not available in English, and studies published before 1994.

Selection of Studies

Two authors (S.J.F. and E.G.T.) independently screened titles derived from the online search based on the inclusion criteria. Subsequently, the abstracts of the selected titles were obtained and screened to see if they met the inclusion criteria. If no abstract was available, the abstract of the printed article was used. If the title and abstract did not provide sufficient information regarding the inclusion criteria, the full text was obtained. Disagreements were resolved by discussion among all three authors. The final selection of full-text articles was based on inclusion/exclusion criteria. The authors then screened and selected the articles that best addressed the topics of prevalence, etiology, risk indicators, prevention, treatment, and progression or conversion to peri-implantitis. The search identified 720 articles for “implant mucositis” and 582 articles for “peri-implant mucositis,” with 146 of the total articles satisfying the inclusion/exclusion criteria. Of these, 66 were determined to best address the above subtopics.

Etiology

Three separate human studies on experimentally induced peri-implant mucositis have demonstrated a cause-and-effect relationship with bacteria. In the first study, following active periodontal therapy, implants were placed in 20 partially edentulous patients who were asked to refrain from all oral hygiene practices for 3 weeks. At the end of this period, the following clinical parameters were obtained: Plaque Index, Gingival Index, Sulcus Bleeding Index, probing pocket depths, and recession (mm). At any given evaluation or from one measurement period to another, there were no significant differences between mean values of all parameters at implant sites compared to tooth sites.

Prevalence

Various studies have reported a prevalence of mucositis ranging from 20% to 80% of subjects (13% to 62% of implants) after a minimum of 5 years in function. Although the search included partially and fully edentulous studies, only one study reported on the prevalence of mucositis. One reason for this wide range is that the definition of mucositis differs in many of the studies. This was discussed at length in a recent publication of the 2017 World Workshop’s findings, citing 22 articles with various definitions of mucositis. While all refer to inflammation in the soft tissue, determined by BOP, some definitions include “and/or suppuration,” specific probing depths (PDs), and no bone loss or bone loss ≤ 2 mm. The common element in all studies that looked at the prevalence of mucositis was the use of BOP to determine soft tissue inflammation for inclusion. However, few studies looked at reversibility following treatment or whether mucositis without treatment or successful treatment, adequate oral hygiene, and maintenance leads to peri-implantitis.
<table>
<thead>
<tr>
<th>Author</th>
<th>Definition</th>
<th>Prevalence</th>
<th>Time</th>
<th>Maintenance interval (mo)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Roos-Jansåker et al⁴</td>
<td>PD ≥ 4 mm BOP</td>
<td>48.0%</td>
<td>76.6%</td>
<td>9–14 y</td>
</tr>
<tr>
<td>Ferreira et al⁵</td>
<td>BOP</td>
<td>62.6%</td>
<td>64.6%</td>
<td>6 mo to 5 y</td>
</tr>
<tr>
<td>Zitzmann and Berglund⁶</td>
<td>Reversible inflammatory reaction in the soft tissues surrounding a functioning implant</td>
<td>50%</td>
<td>80%</td>
<td>≥ 5 y</td>
</tr>
<tr>
<td>Koldsland et al⁷</td>
<td>Presence of inflammation + BOP without detectable BL</td>
<td>82/300 27.3%</td>
<td>41/104 39.4%</td>
<td>8.4 y</td>
</tr>
<tr>
<td>Meijer et al⁸</td>
<td>&lt; 2 mm radiographic BL + BOP and/or S</td>
<td>41.2%</td>
<td>47.0%</td>
<td>5 y</td>
</tr>
<tr>
<td>Aguirre-Zorzano et al⁹</td>
<td>BOP + clinical signs of inflammation with no BL around implant</td>
<td>101/786 12.8%</td>
<td>59/239 24.0%</td>
<td>63 to 41 mo</td>
</tr>
<tr>
<td>Daubert et al¹⁰</td>
<td>BOP and/or gingival inflammation with no evidence of radiographic BL beyond normal remodeling</td>
<td>33%</td>
<td>48%</td>
<td>8.9 to 14.8 y</td>
</tr>
<tr>
<td>Derks and Tomasi¹¹</td>
<td>Presence of a plaque-related inflammatory soft tissue infiltrate without concurrent BL</td>
<td>N/A</td>
<td>19% to 65%</td>
<td>N/A</td>
</tr>
<tr>
<td>Jepsen et al¹²</td>
<td>BOP gentle (&lt; 0.25 N)</td>
<td>N/A</td>
<td>43%</td>
<td>N/A</td>
</tr>
<tr>
<td>Konstantinidis et al¹³</td>
<td>At least 1 implant surface with BOP</td>
<td>57%</td>
<td>64.5%</td>
<td>&gt; 5</td>
</tr>
<tr>
<td>Tonetti et al¹⁴</td>
<td>BOP</td>
<td>N/A</td>
<td>43%</td>
<td>N/A</td>
</tr>
<tr>
<td>Papathanasiou et al¹⁵</td>
<td>Reversible inflammatory process causing redness and swelling localized to the soft tissue around implants without signs of BL following initial bone remodeling during healing</td>
<td>N/A</td>
<td>Survey to 280 periodontists reported 71% prevalence own practice</td>
<td>N/A</td>
</tr>
<tr>
<td>Rokn et al¹⁶</td>
<td>Radiographic BL ≤ 2 mm and concomitant presence of BOP+ and/or S</td>
<td>191/478 40%</td>
<td>65/132 48.5%</td>
<td>1–11 y</td>
</tr>
<tr>
<td>Salvi et al¹⁷</td>
<td>Development of mucosal inflammation around implants without BL</td>
<td>N/A</td>
<td>43% (range: 19%–65%)</td>
<td>N/A</td>
</tr>
<tr>
<td>Schwarz et al¹⁸</td>
<td>BOP on at least one aspect of the implant and no changes in the radiographic BL compared</td>
<td>35.6%</td>
<td>41.6%</td>
<td>26.44 ± 16.57 mo</td>
</tr>
<tr>
<td>Ramanauskaite et al¹⁹</td>
<td>BOP</td>
<td>43%</td>
<td>20.83%</td>
<td>N/A</td>
</tr>
<tr>
<td>Renvert et al²⁰</td>
<td>BOP + and/or S without BL</td>
<td>82.6%</td>
<td>58.1%</td>
<td>9–14 y</td>
</tr>
</tbody>
</table>

PD = probing depth; BL = bone loss; BOP = bleeding on probing; S = suppuration; N/A = not applicable; NM = not mentioned.
It was found that the undisturbed plaque accumulation induced an inflammatory response, increasing parameters of T and B cells in the infiltrate of both gingiva and peri-implant mucosa that was not statistically different. The last of the three studies compared clinical and biologic inflammatory changes around implants and natural teeth after 21 days of undisturbed plaque accumulation in 20 partially edentulous subjects aged 70 years or older. As in the other two experimental studies, plaque accumulation induced an inflammatory reaction around teeth and implants. The authors noted that although there was less plaque on the implants, the mucosa demonstrated a more-intense clinical response than that seen in the gingiva around the teeth. The development of mucositis from the accumulation of plaque over a 3-week period was similar to the development of gingivitis as originally demonstrated in the classic study by Löe et al. Moreover, at 42 days after professional plaque removal and 21 days of resumed oral hygiene, BOP scores and different biomarkers returned to scores “comparable to baseline.” Although the study in patients aged 70 and older showed more severe inflammation around implants compared to teeth, the mucosa demonstrated a more-intense clinical response than that seen in the gingiva around the teeth.

Risk Indicators

As stated previously, the etiology of peri-implant mucositis is primarily attributed to the accumulation of plaque and the development of biofilm on the surrounding structures of a dental implant. Hence, plaque related to inadequate oral hygiene is the main risk factor associated with the development of peri-implant disease. However, different authors have suggested other risk indicators. A systematic literature review was carried out to identify risk indicators for the development of peri-implant mucositis. Only 15 articles out of 3,135 were included, indicating that “only a few studies provided risk indicators for the development of peri-implant mucositis.” The factors were divided into three groups: local (abutment surface characteristics, abutment geometry, dimensions of keratinized tissue, and residual cement), systemic (smoking, radiation therapy, and diabetes), and patient-related (time in function of the implant, genetics, gender, and maintenance visits). It was concluded that plaque accumulation, as discussed previously, is the main risk indicator as it has the strongest supporting data. Other suggested risk indicators include smoking, radiation therapy for peri-implant mucositis, and diabetes with poor metabolic control, albeit the latter needs more confirmatory data. Roos-Jansäker et al mentioned the relevance of dimensions of keratinized tissue. However, Zigdon et al could not confirm a direct relation between keratinized-tissue width and peri-implant mucositis.
e50

evidence for including residual cement as a risk indicator, especially in patients with a history of periodontal disease. Abutment surface characteristic and abutment geometry demonstrated low evidence since plaque accumulation did not vary in relation to the abutment shape or texture. Patient-related factors presented very low evidence as risk indicators, even though a patient’s understanding that plaque accumulation is the main etiologic factor may improve oral hygiene motivation. Dalago et al also found a strong relation between patients with previous periodontal disease and the development of peri-implant diseases. When looking at all of these items, one must conclude that plaque development secondary to inadequate oral hygiene, smoking, and patient compliance should be considered the main risk indicators for peri-implant mucositis as they have the strongest evidence to date.

**Prevention**

The first step in a mucositis prevention program is to accurately diagnose the peri-implant condition. Prevention should start with healthy peri-implant tissues. A clinical determination as to whether inflammation exists in the peri-implant tissues involves light probing around the dental implant using a round-end probe with a force that is ≤ 0.25 N and observing whether or not BOP is present. Others have used additional parameters to diagnose mucositis (Table 1). However, if there is a PD ≥ 4 mm as well as BOP, an x-ray is indicated to determine if bone loss exceeds physiologic modeling/remodeling or is increasing compared either to previously exposed radiographs or to one taken at the time of restoration completion. If there is BOP, increased PD, and bone loss beyond physiologic remodeling, a diagnosis of peri-implantitis should be made. This is important, as this combination of factors has been shown to be nonresponsive to nonsurgical therapy. If the inflammation resides in the soft tissues, treatment measures for mucositis are indicated (see Treatment). However, to successfully prevent mucositis, treatment must target the cause of the disease and the possible risk factors. Since bacterial plaque has been demonstrated as the primary etiology of mucositis, preventive programs must focus on effective at-home oral hygiene procedures and regular professional maintenance and monitoring. The former should include demonstrating to the patient how biofilm removal should be accomplished around the implant restoration using brushes, floss, interproximal aids, and water irrigating devices, where necessary. These at-home procedures should be customized for each patient. A review on the efficacy of self-performed care on implant restorations reported a slight superiority of powered compared to manual toothbrushes. Restorations must allow the patient (and clinician) access for removal of plaque, food, and debris. Unfortunately, with the desire on the part of the patient and restorative dentist to avoid black triangles or any incompletely closed interproximal spaces, long contact points and pink and white porcelain or composites are often used (especially in the esthetic zone) to close these spaces. These measures, in addition to overbuilt flanges on splinted implants or hybrid cases, also make effective at-home oral care difficult if not impossible. Any dentist restoring an implant must make provisions in the restoration (suprastructure) to allow access for at-home care. A well-planned implant-supported restoration is therefore essential to the primary prevention of peri-implant mucositis.

Professional mechanical plaque removal using appropriate hand and ultrasonic instruments, alone or combined with adjunctive methods (air-abrasive device, antiseptic therapy phosphoric acid gel, chlorhexidine gluconate, ozone therapy, hydrogen peroxide, or locally delivered tetracycline HCl or systemic azithromycin), showed initial improved results that diminished over time and had only a minor benefit by the end of multiple studies. A recent cross-sectional study on 206 implants in 115 patients who were healthy and partially edentulous concluded that peri-implant maintenance therapy ≥ two times per year “seems to be crucial to prevent peri-implantitis in healthy patients.”

Finally, a consensus report based on four systematic reviews on the current epidemiology of peri-implant disease and potential risks of mucositis concluded “that lack of regular supportive therapy in patients with peri-implant mucositis was associated with increased risk for onset of peri-implantitis” and “patient-administered mechanical plaque...
control (with manual or powered toothbrushes) has been shown to be an effective preventive measure.\textsuperscript{44} In addition, professional intervention consisting of oral hygiene instruction and mechanical debridement in all cases revealed a reduction in clinical signs of inflammation.\textsuperscript{44} These studies and reviews underline the importance of mucositis prevention and attending regular maintenance for maintaining implant health. Mechanical therapy with appropriate instruments (with or without adjunctive therapy), reinforcement of at-home care procedures, and strict adherence to daily plaque control on the part of the patient appear to be the most effective preventive measures. Diligence on the part of the treating clinician to make sure all implant-supported restorations are made “cleansable” for at-home oral care is a primary contributor to maintaining peri-implant health. Other than plaque control, elimination of proven risk factors, such as smoking cessation and treatment of any existing periodontal problems, are currently considered to be the most effective methods of mucositis prevention.

**Treatment**

In evaluating treatment strategies for peri-implant diseases, it has been noted that case definitions and prevalence vary considerably when referring to peri-implantitis.\textsuperscript{7} This is the result of differences in the various studies regarding PD and radiographic bone level thresholds that define not only whether the disease exists (ie, patient inclusion) but also its severity.\textsuperscript{3,7,45,46} (Table 2). Mucositis, described as a reversible inflammatory process in the soft tissue around an implant, is usually easier to diagnose by identifying bleeding on light probing around one or more aspects of an implant without loss of supporting bone.\textsuperscript{45} However, as shown in Table 1, the definition of mucositis varied in many prevalence and treatment studies, some of which included PDs, bleeding indices, and BOP in the diagnosis. If the presence and resolution of BOP is used as the sole criteria to determine the efficacy of nonsurgical mucositis treatments, a systematic review and meta-analysis performed by Schwarz et al provides one with the means to evaluate the effectiveness of alternate or adjunctive measures compared to conventional treatment alone.\textsuperscript{47} In Schwarz et al’s review,\textsuperscript{47} the authors evaluated the outcomes of two randomized clinical trials (RCTs) and one nonrandomized controlled clinical trial in humans who underwent nonsurgical treatment of peri-implant mucositis with alternative or adjunctive measures. The three studies by Ji et al,\textsuperscript{48} DeSiena et al,\textsuperscript{49} and Riben-Grundström et al\textsuperscript{50} compared the control treatment (mechanical debridement alone with ultrasonic scalers with carbon-fiber or plastic-coated tips, Teflon curettes, and/or polishing) with the same mechanical debridement with the addition of an air abrasive with glycine.\textsuperscript{49–51} Study periods varied from 3 months, 6 months, to 3 months with a 6-month follow-up, respectively, in the three studies. The first RCT study showed 29.3% of test sites and 42% of control sites without bleeding at 3 months posttreatment.\textsuperscript{47} The results of the second study had 13 test patients compared to 9 control patients with no bleeding at 6 months posttreatment.\textsuperscript{48} The third study demonstrated a reduction in the number of diseased sites with bleeding from 38% to 8% (subject level) in the test treatment and from 52% to 17% (subject level) in the control treatment at 12 months.\textsuperscript{49}

In none of the three studies was BOP eliminated at all sites in either the test or control groups. A second group of three RCTs evaluated mechanical therapy (control) vs test groups consisting of mechanical therapy plus phosphoric acid gel every month for 5 months; mechanical therapy and chlorhexidine irrigation, gel, and mouthwash for 3 months; or mechanical therapy and chlorhexidine rinses and gel at 8 months posttreatment.\textsuperscript{51–53} Again, BOP was significantly reduced in both test and control groups in all three studies despite it never being completely resolved at either the subject or implant level. The final two RCTs compared the control group treated by mechanical therapy (MT) to MT plus adjunctive antibiotic therapy.\textsuperscript{54,55} The first RCT had 45 patients and compared MT with or without the use of locally delivered tetracycline over 6 months.\textsuperscript{54} In the second study, 500 mg Azithromycin was administered on the first day and 250 mg for the following three days in the test group.\textsuperscript{55} In the first study, mean BOP was reduced in the test group and increased in the control group at 6 months posttreatment.\textsuperscript{54}
second study, BOP was reduced in the test (82.6% to 27.3%) and control (80.0% to 47.5%) groups at the subject level at 6 months.55 Again, neither mechanical therapy alone nor in conjunction with adjunctive anti-biotics completely resolved BOP in any group.

These results appear to stand in juxtaposition to a review on the management of peri-implant mucositis and peri-implantitis, which concluded that “peri-implant mucositis seems to be successfully treated by professional maintenance debridement, independent of the adjunctive use of an antimicrobial”56 (Fig 1). Another RCT mentioned in this review evaluated anti-infective treatment of peri-implant mucositis in 29 patients (15 test and 14 control) and reported a significant reduction in BOP.57 However, following this treatment only 38% of implants diagnosed with mucositis had complete resolution of BOP at 3 months. Moreover, in looking at other clinical studies as well as the current anti-infective treatment, the authors concluded that “none of the treatment protocols tested have reported

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**Table 2 Definition and Prevalence of Peri-implantitis**

<table>
<thead>
<tr>
<th>Author</th>
<th>Definition</th>
<th>Prevalence</th>
<th>Time</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lindhe et al53</td>
<td>BOP + BL after 1 year of function</td>
<td>28%–58% subjects</td>
<td>N/A</td>
</tr>
<tr>
<td>Roos-Jansåker et al46</td>
<td>BL ≥ 3 threads, minimum BL of 1.8 mm following the first year in function, BOP and/or S</td>
<td>16% subjects</td>
<td>9–14 y</td>
</tr>
<tr>
<td>Ferreira et al55</td>
<td>PD ≥ 5 mm BOP and/or S Vertical BL</td>
<td>8.9% subjects</td>
<td>42.5 mo</td>
</tr>
<tr>
<td>Zitzmann and Berglundh6</td>
<td>BOP + PD &gt; 6 mm or BL ≥ 2.5</td>
<td>28% and ≥ 56% subjects</td>
<td>≥ 5 y</td>
</tr>
<tr>
<td>Koldsland et al7</td>
<td>Inflammation and detectable BL Radiographic peri-implant BL ≥ 2.0 mm and BOP/S at PD ≥ 4 mm</td>
<td>47.1% subjects 36.6% implants</td>
<td>8.4 y</td>
</tr>
<tr>
<td></td>
<td>Radiographic peri-implant BL ≥ 2.0 mm and BOP/S at PD ≥ 6 mm</td>
<td>20.4% subjects 11.4% implants</td>
<td>10.2 y</td>
</tr>
<tr>
<td></td>
<td>Radiographic peri-implant BL ≥ 3.0 mm and BOP/S at PD ≥ 4 mm</td>
<td>15.1% subjects 7.7% implants</td>
<td>9.9 y</td>
</tr>
<tr>
<td></td>
<td>Radiographic peri-implant BL ≥ 3.0 mm and BOP/S at PD ≥ 6 mm</td>
<td>11.7% subjects 6.0% implants</td>
<td>9.5 y</td>
</tr>
<tr>
<td>Fransson et al45</td>
<td>≥ 3 threads of BL</td>
<td>27.8% subjects 12.4% implants</td>
<td>5–20 y</td>
</tr>
<tr>
<td>Renvert et al46</td>
<td>≥ 3 threads of BL between the 1st year and final radiographic examinations + BOP</td>
<td>14.9% subjects</td>
<td>10.8 y</td>
</tr>
</tbody>
</table>

BOP = bleeding on probing; BL = bone loss; PD = probing depth; S = suppuration.

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**Fig 1** Maxillary left second-premolar implant restoration. (a) Pretreatment, with peri-implant mucositis and bleeding on probing. (b) Complete resolution of inflammation 2 months after mechanical therapy with titanium curettes, air powder abrasion with glycine, and rubber polishing cup with chlorhexidine.
complete resolution of inflammation at all implant sites\textsuperscript{56} (Fig 2). A significant number of test and control treatments mentioned above can (and did, in many cases) significantly reduce or eliminate inflammation (BOP) following mechanical therapy, good oral hygiene, and professional maintenance. Recently, authors have suggested the use of lasers to treat peri-implant mucositis.\textsuperscript{58,59} Here, too, lasers have shown mixed results (Figs 3 and 4). No study or review, as stated above, showed complete resolution of the disease in all implants with mucositis treated with a variety of nonsurgical or laser protocols and improved oral hygiene. A consensus report based on four systematic reviews answered the question “Is resolution of peri-implant mucositis achievable?” by stating that it was “possible” with the caveat that “current data also indicate that resolution of inflammation was not achieved in all patients.”\textsuperscript{43}

Based on these and other studies of treatment, one can question whether the term “reversible” should be part of the definition of peri-implant mucositis.

**Mucositis Progression to Peri-implantitis**

Lastly, a number of cross-sectional studies on the prevalence of mucositis and peri-implantitis have referred to “the conversion from mucositis to peri-implantitis characterized by crestal bone level changes in conjunction with BOP.”\textsuperscript{21} Others have stated that “diagnosis and management of peri-implant mucositis...”

**Fig 2** Maxillary right central incisor. (a) Pretreatment, with peri-implant mucositis, bleeding on probing, and suppuration with 4-mm probing depths and no bone loss. (b) Radiograph showing no bone loss. (c) Clinical view immediately following mechanical treatment using curettes and polishing cups with chlorhexidine. (d) Clinical view 6 weeks after mechanical therapy. There is decreased inflammation and probing depth but residual bleeding on probing and suppuration.

**Fig 3** Maxillary left central-incisor implant. (a) Clinical photo showing peri-implant mucositis bleeding on probing, suppuration, and 4-mm probing depth. (b) Radiograph showing no bone loss. (c) Treatment with the laser-assisted peri-implant mucositis procedure using an Nd:YAG laser. (d) Clinical appearance 5 months posttreatment with a probing depth of 3 mm and no bleeding on probing or suppuration.
should be implemented to prevent the onset of peri-implantitis. Moreover, studies that reported prevalence found a higher prevalence of mucositis than peri-implantitis at all subject and implant levels. For example, in a study of 134 patients with 478 implants placed during a 10-year period with a mean loading time of 4.43 years, the subject and implant-level prevalence of mucositis were 48.5% and 40%, respectively, compared to peri-implantitis diagnosed in 20% of patients and 8.8% of implants. In the introduction of a review on anti-infective treatment of peri-implant mucositis, the study’s authors state, “It is assumed that peri-implant mucositis is the precursor for peri-implantitis as gingivitis is the precursor for periodontitis,” citing unpublished data. An additional reference suggested peri-implant mucositis may transfer to irreversible peri-implantitis, stating “it is important to establish health conditions through effective interceptive treatment of peri-implant mucositis.” However, the mechanism and proof of this “transfer” (conversion) is lacking. Lastly, in an article on the prevention of peri-implantitis by managing peri-implant mucositis, the authors state, “there is emerging evidence on the patient level from a retrospective study that the lack of annual supportive therapy in patients diagnosed with peri-implant mucositis was associated with increased risk for conversion of mucositis to peri-implantitis.”

As proof of this “emerging evidence” or conversion, all the above authors reference the same study by Costa et al. 2012. In this 5-year follow-up without preventive maintenance, patients with pre-existing peri-implant mucositis were associated with a higher incidence of peri-implantitis (44% vs 18%). In another case-controlled study, 48% of implants with no accessibility/capability for proper oral hygiene were affected by peri-implantitis compared to only 4% of the implants with accessibility/capability. These are the articles most cited when conversion (progression of mucositis to peri-implantitis) is stated. However, neither of these articles is a randomized control study with parallel, implant test and control groups that observe an implant with mucositis (going untreated) progressing to peri-implantitis. Albeit, a study with this protocol would be deemed unethical as it would be malfeasance to observe a diagnosed disease without attempting to treat it. However, if this was allowed to occur, it may result in loss of the implant and prosthesis that could have been prevented. Although the etiology of mucositis is allegedly known, there is a poor understanding of the host response accountable for peri-implant bone destruction and loss of osseointegration. Therefore, the assumption that mucositis will progress to peri-implantitis (treated or not) is one that lacks concrete proof.

The conclusions based on the research currently available and reviewed in this paper are:

The prevalence of mucositis is widespread (regardless of the type of implant surface) on implants in function for > 5 years (20% to 80%, subject level).

The etiology of mucositis is related to biofilm forming on the implant, causing inflammation in the peri-implant tissues.

Fig 4 Mandibular left first-molar implant. (a) Mucositis: bleeding on probing and inflamed mucosa. (b) Radiograph suggesting no bone loss. (c) Clinical view following the Nd:YAG laser procedure. (d) Improvement of mucositis, but bleeding on probing and inflammation are still present.
There is strong evidence for including additional risk factors beyond biofilm formation on the implant surface, as other factors may help initiate the diseases. The impact of factors such as smoking, a previous history of periodontal disease, and certain cements cannot be undervalued in this complex and potentially multifactorial process.

Effective treatment should include mechanical debridement with or without chemotherapeutic adjuncts, effective home care procedures on the part of the patient, and regular professional maintenance visits.

The assumption that mucositis is reversible should be reconsidered since most methods of treatment were effective in eliminating inflammation (BOP) of the affected peri-implant soft tissue but few, if any, have been shown to completely eliminate the disease in the vast majority of treated implants. In one well-controlled study using an anti-infective approach including mechanical therapy plus antibiotics, the authors following treatment reported only 38% of implants diagnosed with and treated for mucositis had complete resolution of disease. Thus, the treatment of mucositis may require different strategies than the ones currently being used, and one might have reservations describing mucositis as reversible.

Lastly, there are no human studies or data demonstrating that implants with mucositis will progress to peri-implantitis. The authors are in agreement that successful treatment of mucositis is a preventive method for avoiding peri-implantitis. However, no prospective studies have followed a group of implants with mucositis and demonstrated progression to peri-implantitis. Randomized controlled research studies, at least in an animal model, are needed to see if this is true or not. There is also a lack of research on the recurrence of mucositis once successfully treated.

Conclusions
The focus of much research has been on peri-implantitis, for obvious reasons, since this disease entity may result in implant loss due to progressive loss of bone. However, based on the prevalence of mucositis and the fact that it may be a precursor to peri-implantitis, more research is needed to study the dynamics of mucositis and develop new strategies for prevention and treatment.

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References


