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ABSTRACT

Objectives: the purpose of this essay is to systematically review the diagnostic procedures, treatment methods and challenges associated with implant surface cleansing in peri-implant mucositis and to identify the best diagnostic and treatment methods based on reviewed literature, published in the last 10 years.

Aim and hypothesis of master’s thesis; - the aim of this essay is to determine how we can best diagnose and treat peri-implant mucositis, to highlight the main risk factors for the development of this disease and lastly, to explore the importance of early intervention after diagnosis of peri-implant mucositis.

Material and Methods: A literature investigation using the MEDLINE (PubMed) electronic database and manually reviewing dental journals to find articles concerning the challenges associated with cleansing implant surfaces in peri-implant mucositis. The search was restricted to English language articles published during the last 10 years, from December 2007 to March 2017.

Results: In the absence of standardized methods for the diagnosis and treatment of peri-implant mucositis, the task focuses on presenting an overview of methods for diagnosing and treating the condition. Peri-implant mucositis can be diagnosed based on clinical examination that identifies bleeding on probing (BoP). Other inflammatory signs such as suppuration and an increased periodontal pocket depth can also occur with peri-implant mucositis. Treatment of peri-implant mucositis produces the best results if the implant and surrounding tissues are cleansed mechanically, for example using titanium curettes or alternatively air-abrasion and airscalers. Antibacterial agents may also have a positive effect on the outcome. The patient must be involved in the treatment process. An individualized follow-up regimen for oral health, both before and after treatment, is crucial for a successful and lasting result.

Conclusion: Various diagnostic methods, as well as treatment strategies, have been developed throughout the years for dealing with this fairly frequent complication in the field of periodontology, following dental implant inflammation. Concurrently, the importance of early diagnosis and treatment was proven repeatedly. Essentially, it is recommended to establish the diagnosis within the first 36 hours. And use correct and effective instruments to remove the pathology slowly in addition to being careful during insertion of the implant, and cleansing it without changing the morphology and anatomy of the peri-implant tissues. Effectively, according to the results of the data analysis, a relatively high percentage of the practitioners successfully accomplished this target and achieved good treatment outcomes.

Key words: Peri-implant mucositis, periimplant mucositis development, periimplantitis vs peri.implant mucosotis, peri-implantmucositis treatment, peri-implant mucositis air powder abrasive, periimplant infection and peri-implant decontamination.
INTRODUCTION

Dental implants have revolutionized the treatment of edentulous patients for the last two decades \[^{[1]}\].

People today are living longer and longer, and those who grow up today have more teeth with fewer fillings and crowns at the age of retirement than those who grew up just a generation ago. Previously, "old" was all most synonymous with edentulous and it was not unusual to get dentures. That time is over, and there are fewer and fewer of those who are edentulous due to oral disease in developed countries. In modern dentistry, oral implants for the edentulous have become an important therapeutic modality for replacing missing teeth \[^{[2]}\].

There is a greater demand for permanent solutions and the implant treatment is becoming increasingly relevant. Today it is common to insert dental implants in patients, which was contraindicated and almost impossible just a few years ago.

Ingvar Brånemarks new discovery and invention was the source of the modern dental implant. He and some of his colleagues at the University of Gothenburg started making the precursor of todays implants. The first implant in the form we know it today, was inserted in 1965 in a totally edentulous patient by Professor Brånemark. The new treatment method did not show success, and had sufficient results in only half of the inserted implant cases. This naturally led to a huge disagreement within the academic society in Sweden about the use of the implant treatment in a clinical setting. There was so much scepticism at the time that Brånemark was refusing to treat patients at the University of Gothenburg. In the late 70s success rates of the procedure start increasingly improving. Nowadays, the success rate is high, and long-term studies have shown low rates of oral implant failures \[^{[2]}\].

The majority of implants inserted in the world, are inserted in the United States by private general dentists, and not by specialists, who previously were those who had scored the implants. Treatment with implants has now become a generally known treatment procedure, and there is not much else in dentistry being researched as much as this.
Aim of master thesis and hypothesis
The aim of this literature review is to create an overview of current knowledge about peri-implant mucositis based on published articles, and to mainly try and answer the problems listed further:

- To investigate the challenges of the diagnosis and treatment of peri-implant mucositis.
- To emphasize the risk factors, which may trigger the development of the disease.
- To explore the importance of immediate intervention after the diagnosis of peri-implant mucositis.
- To discuss follow-up protocols performed in relation to these treatments.

Clinical effect, demands for a correct diagnosis and follow-up, varies between different treatment methods and used materials.

The main anatomical difference of bonds in a natural tooth with surrounding structures is that the tooth's hard tissue is bound to the surrounding tissue by fibres of periodontal ligament which are enriched by nutrition, blood, immunologic and nerve supplies which provide the tooth's sensibility and tactility.

This direct connection between bone and tooth is ensured by alveolar collagen fibres in horizontal, vertical and oblique directions and periodontal ligament fluid, which is filtrated from blood plasma. These fibres function as pressure sensors and in addition allow the tooth some limited mobility during load. The periodontal ligaments are about 0.4-1.5nm thick and encircle the roots of teeth, in addition there are donto-alveolar, donto-periosteal, circular and transeptal types of collagen fibres which tightly connect mucosa and tooth hard structures together. Tooth implants do not have periodontal ligaments between the implant and bone, the connection is created through bone (Osseo-integration) which means over 60% of the implant surface is covered by bone (bone-to-implant contact).

The space between the implant and surrounding bone measures about 100-400nm. This space has interface fluid, proteins and proteoglycans. On the cervical bone there are collagen structures but none of them directly connect bone to implant, instead those fibres parallelly line the tooth implant and bind to the free mucosa.
The gingival fibre structures do not ensure an equal amount of strength for the implant as they do for natural teeth [2].

Peri-implant disease is one of the factors responsible for implant failure [3].

Treatment of missing teeth with tooth implant replacement has solved many problems for the edentulous, but this method is far from complication free.

Risk factors that are often highlighted in this aspect are a history of periodontal disease (Daubert et al. 2015; Renvert et al. 2014), genetic predisposition (Laine et al. 2006), smoking (Rinke et al. 2011), the individuals’ general health status (Renvert et al. 2014), diseases like diabetes mellitus (Daubert et al. 2015), and the individuals’ motivation to attend supportive care visits and willingness to perform adequate oral hygiene (Rinke et al. 2011; Roccuzzo et al. 2012). [4]

To name some of the complications which are mostly caused by practitioners: inappropriate implant material, lack of patient follow ups, mechanical complications such as fracture of implant, loosening the screws etc. But the most important factors are patients’ motivation to follow up, oral hygiene and plaque control.

Dental implant complications can be divided into early and late failures. The early ones occur due to surgical trauma during implant insertion, inadequate primary stability, lack of wound healing, lack of complete osseointegration, early load on the inserted implant, and infection at the site of the implant. The two latter ones being the most common causes of early failure [5]. It is important for practitioners to take these factors into consideration [5].

Observation for early inflammation in the implant site increases the chance of later implant survival [6]. Research shows that about 80% of implant patient’s develop some form of peri-implant mucositis [7]. And inflammation of tissue, surrounding the implant, is one of the risk factors to develop peri-implantitis [4]. Therefore, it should be a dentists’ goal to stop inflammation of tissue around the implant (peri-implant mucositis) to prevent progression to peri-implantitis, which is irreversible and the main cause of infection and implant failure.

In patients with natural teeth, reversible inflammation of gingival tissue around the
tooth, which is clinically recognised by swelling and redness, is diagnosed as gingivitis, but the same reversible mucosal inflammation for a patient with an endosseous implant is diagnosed as peri-implant mucositis.

Cases where the radiographs did not confirm the peri-implant bone loss were diagnosed as peri-implant mucositis [6].

The problem with the definition of peri-implant mucositis is that in literature it differs and changes accordingly, depending on various conditions. It can be defined as a primary infection at the implant site without loss of alveolar bone [2, 4, 8, 9, 31].

It could also be an infection of the soft tissue around the implant, which for various reasons is exposed threads, but where it is observed stable bone level over time. In this situation there is a low bone level, and threads on the titanium surface are exposed. No distinction is made between peri-implant mucositis with normal or low bone levels in the literature. These situations will result in differing adhesion of biofilm and unequal access for maintenance and daily cleaning.

Is there huge similarity between gingivitis and peri-implant mucositis? Due to anatomical differences between implants, natural teeth and their surrounding tissues, it’s not possible to compare them directly; the description of the inflammatory process of PIM around an implant is quite similar to gingivitis around natural teeth [10]. The most common type of gingivitis is a chronic form induced by plaque which very similar to the pathogenesis of PM.

PIM disease has been associated with gram negative anaerobic bacteria similar to those in patients with chronic periodontitis, its generally accepted that PM is the precursor of peri-implantitis as it is accepted that gingivitis is the precursor to periodontitis [10]. In addition a study by Leo et al showed a similar cause and effect relationship between bacterial accumulation and incidence of PM and gingivitis [11].

Signs such as swelling, redness and Bop which are clinical parameters for gingivitis around the tooth, is also accounted for PIM, with some differences in the procedure of diagnosis depending on the standards in different countries, such as plaque index (mPI), and gingival index (GI). High level periodontal pathogens, mainly gram negative including Porphyromonas gingivalis, Tannerella forsythia and Treponema
*denticula* have been identified\[^9\].

Absence of BOP has a positive predictive value for periodontal health of up to 98.5%, and can therefore be used to exclude inflammation at the implant site\[^9\]. However, BoP alone is not a good predictor of disease activity\[^5\].

In the lining around both teeth and implants keratinized tissue is desirable. This keratinisation may make tissues resistant to mechanical impact. Alveolar bone fibers and the cervical area of the tooth adhere to the keratinized part of the gingiva.

There have been discussions about the need for a minimum level of keratinized gingival tissue around teeth for maintenance. Similar studies have been done for implants. The results of the studies on implants and keratinized mucosa are discussed under the section of diagnostics of PM.\[^9,12\] Measurement of pocket depth is a crucial procedure for assessing the condition of both teeth and implants, and for subsequent selection of therapy. With pocket depth measurement around the teeth, a measure of about 2 mm normally indicates healthy periodontal conditions. Pocket depths around implants are reviewed under the section of diagnostics.

The occurrence of pus is a sure sign of both gingivitis and PM. Histological examination of the infected periodontal tissue, shows infiltration of neutrophils. High numbers of lymphocytes and plasma cells are also detected around implants with mucositis, which is natural in the presence of chronic inflammation. In a study on patients with periodontitis, biochemical markers were used for neutrophils, and a correlation was found between periodontal disease activity and high levels of the enzyme β-glucuronidase. This indicates that suppuration is associated with the level of disease activity\[^13\].
Material and Methods

Research was carried out using PubMed database English-language articles published in the last 10 years (2007-2017) relevant to the topic of peri-implant mucositis. The following key words were used in the main query: (peri-implant mucositis development), (peri implant mucositis ), (peri implant mucositis treatment), (periimplant mucosites ), (periimplant inflammation ), (peri-implant inflammation), (periimplant mucositis decontamination ).

A single search was done with keywords (treatment), (risk factors), and, "clinical, parameters ". Some articles, with direct relevance to the thesis, could not be detected through the main search and simple queries were handpicked from citations in review articles. The search resulted in a of total 30 articles published in various journals, and an abstract from one international convention.

These include systematic review articles, a sectional study, experimental animal studies, randomized controlled clinical attempts, controlled clinical trials, prospective and retrospective cohort studies.

RESULT

In the absence of standardized methods for diagnosis and treatment of peri-implant mucositis the task was focused on presenting an overview of methods for diagnosing and treating the condition. Peri-implant mucositis can be diagnosed based on a clinical examination that identifies bleeding on probing (BoP).

Other inflammatory signs such as suppuration and an increase in the periodontal pocket depth can also occur in peri-implant mucositis.
**Fig 1** Flow diagram of literature search and inclusion.


- 432 of records identified through Pub-med database

- 230 records after records err removed

- 130 records excluded because animal research

- 100 records, abstract reviewed

- 68 records excluded due not qualitative to search

- 30 studies included in qualitative and quantitative synthesis.
1.0 DIAGNOSTICS REVIEW AND DISCUSSION.

Peri-implant mucositis is a condition which general dentist should take control of to prevent the development of peri-implantitis.

<table>
<thead>
<tr>
<th>Types</th>
<th>Characteristics</th>
</tr>
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<tbody>
<tr>
<td>Early</td>
<td>Pd&gt;4mm (bleeding and/or suppuration on probing). Bone loss &lt;25% of the implant length</td>
</tr>
<tr>
<td>Moderate</td>
<td>PD&gt;6mm, (bleeding and/or suppuration on probing) bone loss 25-50% of the implant length</td>
</tr>
<tr>
<td>Advanced</td>
<td>Pd&gt;8mm, (bleeding and/or suppuration on probing) bone loss &gt;50% of the implant length</td>
</tr>
</tbody>
</table>

*Table 1. Classification of peri-implant disease* [13].

Furthermore, it follows an overview of which diagnostic methods, and clinical parameters are most important for diagnosis PM.

There are many challenges we encounter searching for the best way to diagnose PM, partially due to an unclear definition.

<table>
<thead>
<tr>
<th>Authors</th>
<th>Definitions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heitz-Mayfield et al. (2008)</td>
<td>Bop, no bone loss</td>
</tr>
<tr>
<td>Thone-Muhling et al.2010</td>
<td>Bop and/or Gingival index (GI)&gt;1 on at least one site</td>
</tr>
<tr>
<td>Ramberg et al. (2009)</td>
<td>Bop.</td>
</tr>
<tr>
<td>Porras et al. (2009)</td>
<td>Plaque,(p), probing depth(PP)&lt;5mm and evidence of infl.by modified bleeding index</td>
</tr>
<tr>
<td>Felo et al. (1997)</td>
<td>Bop, modified GI&gt;1.5, modified PI&gt;1.5 and PD&lt;3mm</td>
</tr>
<tr>
<td>Ciancio et al. (1995)</td>
<td>Bop, modified GI&gt;1.5 and modified PI&gt;1.7</td>
</tr>
</tbody>
</table>

*Table (2) shows some definitions of PM* [14].
Making a correct diagnosis is a challenge with so many different criteria for one condition such as PM, but despite all the differing definitions and varying criteria one, oral BoP, remains constant.

In others words, presence of plaque works as a positive predictor and should cause thought for consideration that an implant device will develop mucositis (risk indicator).

Clinically peri-implant evaluation is in need of early observation for signs of disease, needs proper definition of these diseases according to the indicated parameters.

<table>
<thead>
<tr>
<th>Score</th>
<th>Mobelli et al.(mPI)</th>
<th>Lindquist et al</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No detection of plaque</td>
<td>No visible plaque</td>
</tr>
<tr>
<td>1</td>
<td>Plaque only recognized by running a probe across the smooth margin of implant</td>
<td>Local plaque accumulation</td>
</tr>
<tr>
<td>2</td>
<td>Plaque can be seen by naked eye</td>
<td>General plaque accumulation greater than 25%</td>
</tr>
<tr>
<td>3</td>
<td>Abundance of soft matter</td>
<td>%</td>
</tr>
</tbody>
</table>

*Table 3* Indexes used to assess plaque accumulation around Oral implant.[15]*

Presence of plaque and periodontal (peri-implant) BOP at >30% of sites are associated with increased risk of PM and PI. [12]*

<table>
<thead>
<tr>
<th>Score</th>
<th>Mombelli et al.(mGI)</th>
<th>Apse et al</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No bleeding when a periodontal probe is passed along the mucosal margin adjacent to the implant</td>
<td>Normal mucosa</td>
</tr>
<tr>
<td>1</td>
<td>Isolated bleeding spots visible</td>
<td>Minimal inflammation with color change and minor edema</td>
</tr>
<tr>
<td>2</td>
<td>Blood forms a confluent red line on mucosal margin</td>
<td>Moderate inflammation with redness, edema, and glazing</td>
</tr>
<tr>
<td>3</td>
<td>Heavy or profuse bleeding</td>
<td>Severe inflammation with redness, edema, ulceration, and spontaneous bleeding without probing</td>
</tr>
</tbody>
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*Table 4* Indices Used to Assess Marginal Mucosal Conditions around Oral Implants[15].
In addition to clinical signs such as redness and edema around the implant mucosa, POB+ testing is the earliest sign of Peri-implant disease, especially in the case of PM, but then a question arises about POB. How gently should a dentist insert the probe? Most studies suggest that the optimal force for probe insertion should be no more than (0,25 N)\(^{12}\). Pocket depth measurements around implants performed by Lang et al in a histological animal study showed that healthy tissue around the implant was never positive Bop\(^{15}\). According to the study the pressure used was just 02N, however, in the case of inflammation in the pocket such as peri-implant mucositis or peri-implantitis BoP was positive 67% and 91% of the time respectively. At the same time other studies reject the POB+ during exploration as a diagnostic criterion for PM\(^{15}\). BoP is thus a positive predictor that there actually is inflammation in the pocket and absence of Bop indicates fresh tissue.

1.1 Clinical appearance of the mucosa

Mucosal inflammation can be graded. To determine the grade of peri-implant mucosal tissue inflammation it is advised to inspect the mucosa and look for tissue changes around the implant, investigate if there is any gingival obliteration of keratinized mucosal tissue\(^{15}\). On the other hand, Roos-jansåker (2006b) who examined 218 patients treated with titanium implants, could not find any association between the absence of keratinized peri-implant mucosa and peri-implant disease\(^{12}\). This has proven to be a poor predictive method.

When evaluating the peri-implant mucosa, there must be data on how the mucosa appeared, before the implant was inserted. If there is no access such data, it is not certain, whether the color changes and the alterations to mucosal surface and texture are a result of a reaction to the implant surface or if it arises from real PM. It may also be difficult to detect color changes in the mucosa and it cannot be decided with certainty, whether the mucosa is inflamed or not, based just on appearance alone. Peri-implant mucosa is not always erythematous and swollen, but may appear pale and bright pink even with severe infection / inflammation. Inspection of the mucosa therefore rarely gives useful parameter information.
1.2 Changes in pocket depth

A study published during the 6th European workshop on periodontology, came to a conclusion that measuring pocket depth is the necessary tool for precise diagnosis of peri-implant disease [13].

Pocket depth measurement for the diagnosis of PM is dependent on many factors: the force of probe insertion, the angle of the instrument, the diameter of the probe tip, the shape of the implant surface, the state and grade of inflammation of the tissue. Some implant designs make it difficult to insert the probe. Concave, shouldered and buoyed types are a couple of such examples.

The mini threads on the implant surfaces also play a great role in probing. A smooth implant surface makes it easier to explore around them. Plasma-coated, sandblasted and acid etched implants or those that have machined microstructures are good examples [22].

According to Pontoriero et al. [15] the deeper the peri-implant pocket, the higher the chance of inflammation is. This statement may be true in the case of periodontal pocket depth, but with peri-implant pocket depth, it will not always indicate inflammation when deep pockets are discovered.

1.3 Is pocket depth measurement safe?

There are several studies which discuss the probing force pressure during pocket measurement and some even register irreversible damage caused by excessive force, applied during the procedure [17]. In one study, complete attachment of the functional epithelium was re-established only after 5 days following probing, using a conventional periodontal probe [18].

This indicated that the force applied during pocket measurement should not exceed (0.25N). Therefore the force between (0.20-0.25N) should be considered optimal and in addition to being applied in controlled and correct fashion.

One study compares the pocket depth measurements in various implant types by
measuring pockets 2, 4, 8, and 12 weeks following implant insertion\textsuperscript{[16]}.

Is there a difference in optimal force applied during pocket depth measurement?

The indicated pocket depth measurement force is between 0.20-0.25N \textbackslash about 20g. This ensures optimal pocket depth measurement \textsuperscript{[12, 15, 17]}, however it is a big challenge. There are some companies, which introduced customized pocket measurement probes with a standard pressure of 0.25N \textsuperscript{[15]}. Two studies concluded that probing with optimal force, not higher than 0.25N, did not cause any damage to the mucosa during pocket measurement \textsuperscript{[12, 15]}. The same study concluded that probing with a force higher than 0.25N has a higher chance of causing different kinds of traumatic damage to the peri-implant epithelial tissue.

Now after all that data, questions arise: Is pocket depth measurement reliable enough for diagnostic purposes? What is the relation between measured pocket depth and inflammation?

The most popular procedures we repeat and register during control clinical check-ups of tissue around implants after implant insertion are PI, PPD, BoP.

The critical issues of implant/tooth loss due to periodontitis /peri-implantitis are concluded by a study which was done in 11 Spanish centers \textsuperscript{[18]}. According to the study the deeper the measured pocket, the greater the bone loss found on x-ray. The number of sites with observed plaque also correlated with bone loss observed on x-ray. The higher the number of implant sites with observed plaque, the greater the bone loss found on x-ray.

A total of 117 patients with 295 implants participated in the study, which was carried out 4-5 years after implant insertion. It studied the difference of bone levels, calculated from 2004 to 2009. During the study period they found 117 patients with no plaque in any site of implant or tooth surfaces, out of this number just 27.4\% had BoP, there was no suppuration and no pain under examination. But on the other hand there were 133 which had plaque found on probing, and 85 of the patients had visible plaque. Bi-directional relation with the percentage of finding Bop was 68.4\% and 90.6 \%, furthermore the pain ratio increased by 3.8\% and 5.9.

They concluded that the periodontal status of the patient is a critical factor in
predicting implant health/lesions, and that the parameters measured in daily practice (bone loss, plaque index, pocket depth and BoP), may provide quantitative values for assessing the longevity of implants \[18\].

In modified pocket depth measurement and modified mucosal tissue inflammation (table 2, 3), it is described that the level of bleeding under probing is significant, but there are other factors which play a role in the amount of bleeding beside the probe, control and pressure.

Renvert et al. describe grades of bleeding under probing and highlights that puncture bleeding can be caused by trauma \[14,8\]. On the other hand, patients being treated with anticoagulants, will always have a bleeding tendency higher than others, and patients who are heavy smokers and have high nicotine levels in the blood often have reactive hyperkeratosis throughout the oral mucosa. These and many other factors can distort the parameters of bleeding on probing, concurrently effecting the diagnosis of PM.

Mombelli et al. compare the measurement of pocket depth in natural teeth and implants, and conclude that probing around the implant is more painful than around natural teeth \[15\].

By using a special peri-probe with variable pressure control between (0.3-0.4g), measured pocket depth in periodontitis measured at 1.5mm but in the peri-implant pocket with mucositis it was only 0.5 mm away to bone surface.

1.4 Suppuration as an indicator for PM.
A high number of PMN cells have been detected around implants, which is associated with severe signs of mucosal inflammation \[15,17\]. The sing of pus around peri-implant tissue alone cannot differentiate PM from PI. To confirm the diagnosis we need an x-ray to prove that there is bone loss.

1.5 Presence of keratinized gingival cells and its relation to PM.
The role of keratinized mucosa in peri-implant disease was studied by Roos-Jansåker et al. \[20\]. In the study they concluded, that no association between the absence of keratinized peri-implant mucosa and peri-implant disease was found \[20\].

On the other hand, Lin et al. name the absence of keratinized tissue, which adjusts the
implant, as one of the risk factors for the progression of inflammation [4]. In an experimental study which was done on animals [15], dogs received plaque induced peri-implantitis and the significance of keratinized mucosa around implants was examined. No increased incidence of gingival retractions or bone loss has been demonstrated in dogs with "normal" amounts of keratinized mucosa compared to dogs without keratinized mucosa.

Good oral hygiene can prevent development of oral pathology. The presence and amount of keratinized mucosa, in fact absence of keratinized-mucosa around implants, could reduce the tissue resistance against pathogens and plaque induced PM and then progress to PI.

2.0 RISK INDICATORS FOR DEVELOPMENT OF PERI-IMPLANT MUCOSITIS.

Overall the most prominent etiological factor for developing peri-implant mucositis is the presence of biofilm related to the implant [19]. Oral hygiene plays an important role in developing PM and a number of clinical studies have reported a cause and effect correlation between experimental plaque accumulation and the development of PM [20].

The factor has only been demonstrated scientifically to be associated with the disease. It identified a number of risk indicators associated with the development of PM.

The definition of a risk indicator for peri-implant disease, according to Heitz-Mayfield et al. is any factor, which increases the risk to induce and/or facilitate peri-implant pathogens [4, 12, 20].

A systematic review in 2015, provided by the European federation of periodontology (EFP), categorized risk factors to: environmental, behavioral or biological [7, 27]. The presence of these mentioned factors directly increases the chance of peri-implant disease, but if removed (treated) the probability of disease development decreases.

In cross-sectional study named several risk factors such: history of periodontitis, diabetes, genetic traits, poor oral hygiene, smoking, alcohol consumption, absence of keratinized mucosa and implant surface properties [9, 12].
Smoking and alcohol consumption are identified as independent factors that induce PM, as well as exposure to radiation which can have same effect\textsuperscript{[7]}.

<table>
<thead>
<tr>
<th>Authors \ year</th>
<th>Study</th>
<th>Results around PM risk factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Michael K et al. (2016)</td>
<td>Cement- associated peri-implantatis</td>
<td>Excess cement was found around 60% of the implants with PIC. No excess cement was found around implants with TB. The removal of excess cement and recementation with TB had an anti-inflammatory effect on the peri-implant tissues after 1 year.</td>
</tr>
<tr>
<td>Stefan R. Et al. (2015)</td>
<td>Risk indicators for peri-implant disease</td>
<td>Plaque accumulation at implant sites will result in the development of an inflammatory process. A history of periodontal disease, smoking, excess cement, and lack of supportive therapy should be considered as risk indicators for the development of peri-implantitis.</td>
</tr>
<tr>
<td>Frank J.J Van Velzen et al (2015)\textsuperscript{[8]}</td>
<td>Dental floss as possible risk for development of peri-implant disease</td>
<td>Rough exposed implant surfaces may lead to tearing floss fibers, which in turn may lead to the development of plaque related peri-implant</td>
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<tr>
<td>Source</td>
<td>Study Details</td>
<td>Findings/Conclusion</td>
</tr>
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<td>----------------------------------------------------------------------</td>
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<tr>
<td>Mauro M. And Clinica Merli et al. (2016) [9]</td>
<td>Peri-implant bleeding on probing: across-sectional multilevel analysis of associated factors</td>
<td>A significantly higher risk was observed for interproximal surfaces as opposed to approximal surfaces. Peri-implant bleeding was associated with site-specific factors.</td>
</tr>
<tr>
<td>Nicola A. V. Et al. (2016)[21]</td>
<td>Peri-implant disease: what we know and what we need to know</td>
<td>-Microbial flora around teeth lead to plaque accumulation around implants.</td>
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<td></td>
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<td>- significantly elevated levels of PGE2, IL-1β, and PDGF detected in the cervical fluid of implants affected by peri-implantitis</td>
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<td>- smokers harbor more periodontal pathogens in the peri-implant sulci, thus implying a potential risk for the onset of peri-implant disease</td>
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Bernardo B. P. et al. (2014)\textsuperscript{[38]}

Does the number of implants have any relation with peri-implant disease?

The lack of KM seems to negatively influence mucositis.

The greater the accumulation of plaque, the higher the incidence of inflammation around the implant is, which leads to mucositis.

Lisa J. A. and Heitz-Myfield et al. (2008)

Peri-implant disease: diagnosis and risk factors

- Poor oral hygiene
- Ferreira et al. (2006) OR 14.3 (95% CI: 2.0–4.1),
- History of periodontitis
- Cigarette smoking

<table>
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<th>Table 5 overview over studies which they mentioned risk factors for PM.</th>
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2.1 Patient failure to comply.

As in most cases, treating peri-implant pathology is also linked to adequate patient information about the disease and may even self-initiate preventative measures.

Studies have proven that patients who do not comply with a structural maintenance program more frequently develop peri-implant disease compared to compliant patients (Roccuzzo et al., 2010, 2012, 2014, Rinke et al. 2011\textsuperscript{[4]}). A maintenance program should be based on the patients' individual local and systemic risk factors. A study by Roccuzzo et al. agreed that individual supportive therapy treatment regimens in patients with implants reduced a big number of biological complications\textsuperscript{[4,21]}.

2.2 Suboptimal design of supra construction and residual cement.

The design of the prosthetic supra construction is very important. Several studies mention that residual cement during crown cementation has direct effect on implant longevity. Recent microscopic analysis of soft tissue biopsies, taken from around implants with cemented restorations of patients suffering from peri-implantitis, revealed a mixture of subacute and chronic inflammation dominated by plasma cells,
indicating foreign bodies, primarily consisting of titanium and dental cement.\textsuperscript{[23]} It could be the rough surface from remaining cement providing desirable pathogen conditions. This area will be extra difficult to clean during plaque removal and especially during polishing. Moreover, excess cement was found to be related to peri-implant mucositis.

Another study also mentioned that, its surface topography might provide a positive environment for bacterial attachment\textsuperscript{[10]}.

The prosthetic design should not only pay attention to patient demands for esthetics, phonetics and chewing function, but also be designed so that both prosthetic abutments and any fixture can be kept clean. In addition, it should be possible to implement standardized diagnostic investigations around fixtures.

Just by removing the residual cement we do not ensure inflammation free tissue long term. The effect of remaining dental floss is almost the same as that of remaining residual cement\textsuperscript{[21]}.

2.3 Selection of implant.

As soon as the dental implant is exposed in the oral cavity, microbial colonization of the exposed implant surface occurs\textsuperscript{[4,17,24]}. After a couple of weeks established sub-mucosal micro-flora may already resemble what we see with gingivitis, which applies to all implants regardless of how the implant has been selected. Different implants may have different amounts of biofilm colonizing the implant surface.

The design of a tapered superstructure may further complicate adequate access to the affected surface\textsuperscript{[23]}. Thus, no access for adequate cleaning means no opportunity for removing pathology. Mostly, defects in peri-implant tissue occur due to chronic and respected irritations.

Poor alignment of the components that comprise an implant prosthesis system may foster the retention of bacterial plaque\textsuperscript{[26]}.
2.4 The position of implant in the dental arch.

Is there a connection between the implant position in the mouth and risk of peri-implant mucositis?

Aguirre-Zorzano et al. found a significant correlation if the implants inserted in maxillary especially anteriorly\textsuperscript{[8]}.

2.2.5 Smoking.

McDermott et al.\textsuperscript{[12]} studied 677 subjects (results based on 677 implants) 10.3\% of which were smokers and a multivariate analysis for inflammatory complications (including peri-implantitis) showed smoking was significant, Hazard ratio: 3.26 (95\% CI: 1.7–6.10), 69 of 677 implants had inflammatory complications. Although they indicated smokers had mucositis more frequently, bone level at $\geq$3 threads and peri-implantitis\textsuperscript{[13]}. A different meta-analysis by Sgolastra et al. showed, in the implant-based results, that smokers have a higher and significant risk for PM compared to non-smokers\textsuperscript{[13]}.

2.6 History of periodontal disease.

Study by Cosgarea et al. (2012) tested 24 patients, in that study they found a sufficient relationship between patients with a history of periodontitis and per-implant disease. Higher \textit{P. gingivalis} and \textit{A.a.} colonization at implants and teeth was detected in the chronic periodontitis group\textsuperscript{[25]}.

Javed et al. (2011) found that individuals susceptible to periodontitis have been reported to react differently to microbial challenges than patients who are less susceptible.

In a recently published cross-sectional analysis of 239 patients who had experienced periodontitis, 786 implants were studied. A correlation was not found between chronic periodontitis and PM\textsuperscript{[26]}.
Summary of first part of discussion (2.0)

This thesis concludes that the risk factors for developing PM are noncompliance, oral plaque, suboptimal design of the supra structure, residual cement, thread exposure and the location of the implant being anterior in the maxilla. There is a need for more research to determine whether smoking is a true risk indicator.

3.0 TREATMENT OF PERI-IMPLANT MUCOSITIS, AN OVERVIEW AND DISCUSSION.

In a recently published systemic review, in which 1497 patients and 6283 implants were followed for more than five years, PM was found in 63.4% of patients and approximately 30.7% of the implants, and PI. Was found in 18.8% of patients and 9.6% of implants, with smokers being at a higher risk of these conditions [2,27].

The main goal of treatment of PM is to reestablish healthy mucosa without damaging the tissue and without damage the implant surface [2,27,28].

The inflammation can be eliminated if the biofilm is removed around the implant. This is done professionally at the dentist and maintained by the patient involved in treatment.

Are there methods that perform better treatment result than others?

Several treatment protocols for peri-implant diseases have been proposed, but no gold standard has been established to date [2,12,17,29]. Those studies mostly research work but not a fact, the only point which is very clear is that PM is not treated surgically.

According many studies treatment methods should be after full understanding of the cause of peri-implant disease, so chosen effective treatment addressed after diagnosis are the most widely for dentists [2,12,17]. Because the disease can have multi factorial cause factors, therefore single therapy can solve the problems totally, because bacteria cause peri-implant diseases, treatment must include anti-infective measures.

Future studies must investigate the influence of defect characteristics or patient-related factors on treatment outcome.
3.1 Mechanical anti-infective therapies.

Mechanical anti-infective treatments including mechanically removing microbial biofilm with calculus deposits by professionals using different types of curettes then cleaning the location with air scalars, combined with airflow abrasives and finally polishing the prosthetic over contour and saline irrigations in the end.


Chosen clinical parameters were: probing around the implant in six sites by periodontal probe, (North Carolina–Hu-Friedy, Chicago, IL, USA) [19].

The peri-implant site with the deepest PD was selected for sampling. If two or more sites presented similar PD values, the most anterior site was chosen [19].

The PM treatment was 1- Mechanical removal of biofilm and/or calculus deposits using Teflon curettes (Hu-Friedy Co, Chicago, IL, USA; Straumann Institute, Waldenburg, Switzerland); 2- abrasive sodium carbonate air-powder system (Jet Sonic, Gnatus, Ribeirão Preto, SP, Brazil) and in addition, 3- prosthesis overcontour was removed with extra-fine diamond finishing drills, under abundant water irrigation, as indicated. The treatment period lasted 3 month.

<table>
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<th>Baseline</th>
<th>3 months</th>
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<tr>
<td></td>
<td>Number</td>
<td>%</td>
</tr>
<tr>
<td>PD ≥ 5 mm†</td>
<td>16</td>
<td>100</td>
</tr>
<tr>
<td>BOP (at least one site)</td>
<td>16</td>
<td>100</td>
</tr>
<tr>
<td>MB (at least one site)</td>
<td>16</td>
<td>100</td>
</tr>
<tr>
<td>PD ≥ 5 + BOP or MB (at least one site)</td>
<td>9</td>
<td>56.3</td>
</tr>
<tr>
<td>Peri-implantitis</td>
<td>20</td>
<td>100</td>
</tr>
<tr>
<td>PD ≥ 5 mm†</td>
<td>20</td>
<td>100</td>
</tr>
<tr>
<td>BOP (at least one site)</td>
<td>13</td>
<td>65</td>
</tr>
<tr>
<td>SUP (at least one site)</td>
<td>20</td>
<td>100</td>
</tr>
</tbody>
</table>

Table (6). PD=probing depth,BOP=bleeding in probing, MB=mucosal bleeding

At baseline and after 3 month treatment[19].

They concluded as the mechanical treatment, proposed in the present study to treat mucositis, effectively reduced clinical parameters of inflammation (Table 6), the levels of some pathogens (T. socranskii, P. gingivalis and F. nucleatunssnucleatum)
As well reduced the bacterial complex around the per-implant tissue, but due to limitations of study that the result didn’t gave total resolution of PM, and that because the treatment was closed debridement and the recent curettes tips are to thick to reach the deepest peri-implant pockets. But overall these initial improvements of the mechanical anti-infective therapies will be sustained over the long term.

In a study (cleaning potential of three implant debridement methods) which was published in 2016, they were comparing the Gracey curerette, the ultrasonic scaler and the powder abrasive device. Their conclusion supported the result that a complete surface cleaning could not be achieved regardless of the instrumentation method applied. The air powder abrasive device showed a superior cleaning potential for all defect angulations with better results at wide defects [23].

3.2 Er:YAG(yttrium aluminum garnet) laser system in treatment of PM.

Er:Yag laser has been used in medicine for long time, as well as in dentistry. Er:YAG laser is the most commonly used laser for nonsurgical treatment of peri-implantitis (24), Er:yag laser has shown significant effect on removing calculus and degranulation and debride infected implant surfaces [17].

I choose a study which been presented 2015, there Diode laser been used to evaluate effect of this non-surgical therapy for peri-implant disease.

27 subject r choose, age (36-67, 15 female, 12 male, 12 smokers and 15 non-smokers)

Plaque index (PI), pocket depth (PD), and bleeding on probing(BoP) were recorded at baseline. Patient in test group (TG) where total 606 sites taken in to account, and control group (CG) 144 sites.

(CG)-and (TG), received conventional non-surgical peri-implant treatment, patients in (TG) got in addition diode laser application (810 nm, 30 s, 1 W, 50 Hz, ton=100 ms, toff=100 ms, energy density=24.87 J/cm²). Repeated measurement recorded in (T0) baseline and (T1) after treatment.

Result concluded as (606) in (TG) and 144 sites from (CT) sites which were under
evaluation the Clinical variable such PD, and PoP were reduced more in TG then CG. As in table (6) describes PD difference in both groups as an effect of Diode laser non-surgical treatment.

![Pocket Depth](image)

*Table (7) Box and whisker plot of PD in TG and CG at T0 and T1*

(Er: YAG) laser showed no significance differences to the improvement of clinical parameters, such as bleeding on probing (BOP), pocket depth (PD) reduction of clinical attachment level (CAL) or bone fill, when compared with saline soaked cotton pellets at 12 and 24 months follow-up [2].

In a randomized controlled clinical trial, the therapeutic effect of an Er:YAG laser was compared with that of a newly developed air-abrasive device designed to be used sub-gingually. After 6 months, bleeding on probing and suppuration had decreased significantly in both groups [17].

Study conclusion, use of diode laser with both high or low power mode may represent a useful protocol for the maintenance of serious complications post-implant rehabilitation [30].

Light energy, including the ablation of tissue, killing bacteria, and inflammation control [30].

The semiconductor 809-nm, the CO2 and Er:YAG lasers are recommended, since it appears that they do not exert a negative impact on the implant surface [26].
3.3 Enamel matrix derivative (EMD), and sustained release micro spherical minocycline (MSM) in non-surgical treatment of PM.

Students in Tabriz University of medical science, divided 96 subjects in to 3 group one control group (CG) which been treated just with mechanical debridement, and two test group (TG1) was treated with (EMD), and (TG2) treated with (MSM). For the study they used (BOP), (DP) and peri-implant curricular fluid for microbial analysis.

Result was reduction of *Porphyromonas gingivalis* for all the groups, but more reduction in (TG1) and (TG2) compared to (CG), and the parameters as (BOP), (DP) MSM, EMD, and control groups were 60%, 50%, and 20%, respectively. The study was short-term control period [11].

All in all, there have been 7 studies carried out for 2007-2017 that kompered air-abrasive system effectiveness compared to different types peri-implant cleaning methods. Two of the studies concluded that glycine powder didn’t gave better result compared to sodium bicarbonate powder, both equally improved BOP, PD parameters.

Two other studies showed that sodium bicarbonate powder is more aggressive then glycine powder and can change the morphology of titanium implant surface. Therefor glycine power could be preferable method of implant surface cleaning.

One other article states that glycine powder is more effective method for peri-implant disease treatment then curettage or ultra sound scalars. On the other hand one other study shows that the air-abrasive method doesn’t achieve the better result then curettage, ultra sound or ER: YAG laser treatment method. Hydrogen Peroxide was shown to be the least effective when compared to the use of citric acid, plastic scalers, sonic tips and air powder abrasives [2].

Therefore more research needs to be conducted in the feature to determine which method is most effective.

<table>
<thead>
<tr>
<th>Authors /year</th>
<th>Study</th>
<th>Result</th>
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<tbody>
<tr>
<td>Frank Schwartx et al.</td>
<td>Compared Glycine +sodium bicarbonate</td>
<td>Both powder types were equally effective. Sodium</td>
</tr>
<tr>
<td>Reference</td>
<td>Methodology</td>
<td>Findings</td>
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<td>-----------</td>
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</tr>
<tr>
<td>(2018)(^{[16]})</td>
<td>Powder effectiveness, surface smoothness post treatment, residual biofilm</td>
<td>Bicarbonate powder changed surface morphology due to it larger, harder particles</td>
</tr>
<tr>
<td>Sahrmann et al. (2015)(^{[26]})</td>
<td>Used glycine powder</td>
<td>Glycine P. is more effective method for therapeutic implant debridement then ultrasound or curettes.</td>
</tr>
<tr>
<td>SM Lupi et al. (2016)(^{[8]})</td>
<td>Maintenance treatment with glycine powder on the periodontal health of peri-implant tissues</td>
<td>Treatment with glycine seems appropriate in the maintenance of peri-implant health and more effective than the traditional treatment with plastic curette and chlorhexidine.</td>
</tr>
<tr>
<td>Poliana M D et al. (2009)(^{[19]})</td>
<td>Clinical and microbial evaluation using MC. Removing biofilm and calculus using teflon curettes + and abrasive sodium carbonate air-powder system with overcontour was removed with extra-fine diamond finishing drills. Treatment period was 3 and 6 months.</td>
<td>All clinical parameters (BOP;PD; and calculus deposits) improved at 3 months post-therapy in mucositis. An insignificant reduction of microbial levels was also noted.</td>
</tr>
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</table>

*Table (8)* Role of anti-infective mechanical removing of biofilm in PM treatment
There are only two studies carried out on the effect of anti-bacterial agent when used alone to fight PM.

One study concluded that use of 0.12% chlorhexidine showed reduction of visible plaque index, bleeding on probing and pocked depth after 6 month of treatment but not big different then only mechanical decontamination [14], in other hand other study also proved the same result, in addition they mentioned that peri-implant mucositis seems to be successfully treated by professional mechanical debridement, independently of the adjunctive use of an antimicrobial [14].

Elena F et al. (2014). In case of home use oral hygiene some studies shows the good effect of use antiseptic in term of reduction of BoP and in same cases reduction of PI. The same study states local (tetracycline) Or systemic (azithromycin, 500 mg/day for 4 days) Antibiotics can be used in conjunction with mechanical debridement.

3.4 Follow up and recall protocols.

Søren J et al. (2015) stated that after insertion of implants patients should be followed up and put under recall protocol as a preventative and supportive prospective.

Professional supportive care should be established according to the individual needs of the patient (e.g. 3-, 6- or 12-month recall intervals) and their compliance has to be confirmed.

Particularly in patients with a history of treated aggressive periodontitis indicating an increased susceptibility to periodontal and peri-implant disease, shorter recall intervals may be considered.
CONCLUSION

Peri-implant mucositis is a condition that can progress to peri-implantitis if left untreated. That emphasizes the importance of early intervention. The condition can be diagnosed by BoP, suppuration or mBl. I Peri-implant mucositis should be treated with contributions from the patient and by measures made in the dental office. The patient must get a thorough oral hygiene instruction and, mastering the measures dentist instructs in and develops disease understanding. It is helpful with close supervision and frequent recall.

At the clinic, dentists remove the bacterial biofilm around the implant, using a curette and taking care not to damage the implant surface. It is also proposed to use "air-abrasive systems", Er: YAG laser or ultrasound to mechanically clean the implant surface. Experimental treatments such as topical antimicrobial application or irrigation with antibacterial agent chlorhexidine in the pocket have shown varying efficacy in clinical trials. Systemic antibiotics are not recommended to treat the PM condition. Risk factors have been identified that can be associated with an increased incidence of peri-implant mucositis.

These are: - noncompliant patients (plaque), suboptimal design of supra construction and presence of residual cement, exposed threads on the implant site. It is unclear if smoking is a true risk factor for the condition.

Proposals present on how to proceed in the diagnosis and treatment of peri-implant mucositis:

- Inspect the implants and surrounding mucosa. Look for signs of inflammation: redness and swelling of the mucosa.
- Palpate around the implant, hold a finger on the back and press down gently with another finger or the handle of periodontal probe. See if pus comes out of the pocket.
- Register if plaque or bleeding is present.

- Register pocket depths, measured from the mucosal rim down to the bottom of the pocket. This is performed with a pressure of 0.20N. Changes from previous measurements may indicate pocket pathology.
Periapical x-ray is a useful diagnostic tool. Radiographs of the implant should be taken immediately after insertion of implant, then repeat Radiographs immediately after functional load. This is to establish a "baseline". This is important in order to assess any future changes. Radiological examination of the implants is used to establish a diagnosis if inflammation is detected.

Treatment proposals:
- Patients must be educated in adequate oral hygiene.
- The distance? Can be cleaned with a rubber cup and suitable cleaning agent.
- Mechanical cleaning of the implant is necessary. Titanium curettes can be used to clean the implant surface. It is important that all possible calculi and biofilm are removed.

The surface of an implant is different than that of a tooth and depuration must be carried out in a different way for implants. A titanium curette must be used between any exposed threads, where the biofilm is undescended and consequently the movement pattern has to be different from that of conventional depuration.

- Curette may also be used in pocket away from the implant to the inflamed tissue to clean pocket bacteria and granulation tissue (curettage).

- Air Scalar or air-abrasives can also be used.

Lorenzo Drago et al. (2014)[10] concluded that a combination of erythrol and chlorhexidine had a greater antimicrobial effect and removed biofilm more successfully than glycine powder. This could be a great alternative for resolving peri-implant disease. One other alternative could be hydroxyapatite and titanium oxide powder, which caused minimal implant surface changes and removed 99% of biofilm on the implant surface.
REFERENCES


