Influence of Peri-Implant Soft Tissue Condition and Plaque Accumulation on Peri-Implantitis: a Systematic Review

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ABSTRACT

Objectives: To systematically examine influence of soft tissue condition and plaque accumulation around dental implants on peri-implantitis development.

Material and Methods: An electronic literature search was conducted of two databases - MEDLINE (Ovid) and EMBASE from 2011 to 2016. Sequential screenings at the title, abstract, and full-text levels were performed. Clinical human studies in the English language that had reported soft tissue condition or plaque accumulation influence on peri-implantitis development were included. The resulting articles were independently subjected to clear inclusion and exclusion criteria by two reviewers as follows.

Results: The search resulted in 8 articles meeting the inclusion criteria. These studies reported gingival index, plaque index, pocket depth, bleeding on probing/modified bleeding index for sites with “adequate” (≥ 2 mm) and “inadequate” (< 2 mm) width of keratinized mucosa. Results demonstrated that the amount of keratinized mucosa has little influence on soft-tissue inflammation in the presence of good oral hygiene. However, suboptimal oral hygiene due to difficulty in access for plaque control in the areas of minimal keratinized mucosa may lead to greater tissue damage.

Conclusions: In cases with insufficient keratinized gingiva in the vicinity of implants, the insufficiency does not necessarily mediate adverse effects on the hygiene management and soft tissue health condition. Nonetheless, the risk of the increase of gingival index, plaque index, pocket depth, bleeding on probing/modified bleeding index is present. Therefore, the presence of an appropriate amount of keratinized gingiva is required.

Keywords: dental implants; dental plaque; gingiva; peri-implantitis; risk factors.

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INTRODUCTION

Periodontitis can be considered as the consequence of broken balances in bacterial components of the dental plaque [1]. Its prevalence drives to its consideration as the most prevalent infectious disease in the community [2], with 75% of adults affected as reported in published studies [3-4]. Several studies have identified similarities in the pathogenesis of late periodontitis and peri-implantitis, showing intraoral translocation of periodontal pathogens from teeth showing chronic periodontitis to the peri-implant niche [5], producing at last the loss of affected teeth or implants. Previous history of periodontitis, poor oral hygiene and smoking are considered risk factors for peri-implantitis, and late dental implant failures are associated with peri-implantitis and/or biomechanical forces [6]. While peri-implantitis is defined on implant basis (an inflammatory process leading to deformation of the peri-implant pocket and bone loss around an implant in function) [7], periodontitis is defined on subject basis (individuals with more than one tooth [8] showing alterations not only in the classical measures of bone loss but also in additional parameters as bleeding on probing and probing pocket depth) [9].

The peri-implant keratinized mucosa is firmly bound to the underlying bone and constitutes a functional barrier between the oral environment and underlying dental implants. However, after teeth are extracted, the resorption of surrounding bone and keratinized gingiva occurs, which may result in deficiency of keratinized mucosa during subsequent implant placement. The need for keratinized mucosa around dental implants has been widely discussed. During the early development of endosseous dental implants, the establishment of a dense connective tissue around the implant collar for long-term implant stability was repeatedly addressed [10-12]. Nevertheless, a number of subsequent studies showed that implants had a high survival rate irrespective of the presence or absence of keratinized mucosa [13]. Nowadays, in addition to achieving high implant survival following implant therapy, maintenance of functionally loaded implants in an adequate status of health and aesthetics had become a prerequisite for long-term success of implant restoration. The need for keratinized tissue around the dental implant to maintain health and tissue stability is therefore becoming of increasing concern.

Because of the vast differences between natural teeth and dental implants, their maintenance is of critical importance for the longevity of successful osseointegrated implants. A study which purposely banned oral hygiene around dental implants for a short period of time demonstrated a cause-effect relationship between the accumulation of bacterial plaque and the development of peri-implant mucositis [14]. Recent studies have shown that bacterial colonization occurs within 30 minutes following implantation [15] and becomes stable after a 2-week period [16,17]. Thus, the primary objective of maintenance and recovery of any implant regiment is to remove the bacterial plaque and/or calculus. Of course, the dental provider has a role in guiding implant stability following osseointegration, however, proper maintenance of the peri-implant soft tissue health is largely in the control of the patient’s own oral hygiene regimen. Patients’ self-management includes mechanical methods and chemical ways to control biofilm formation and subsequent plaque/calculus accumulation.

Therefore, the aim of the present study is to determine influence of soft tissue condition and plaque accumulation around dental implants on peri-implantitis development.

MATERIAL AND METHODS

Protocol and registration

The methods of the analysis and inclusion criteria were specified in advance and documented in a protocol. The review was registered in PROSPERO, an international prospective register of systematic reviews. The protocol can be accessed at: http://www.crd.york.ac.uk/PROSPERO/display_record.asp?ID=CRD42016033672

Registration number: CRD42016033672.

The reporting of this systematic analysis adhered to the PRISMA Statement [18].

Types of publications

The review included studies on humans published in the English language. Letters, editorials, literature reviews, PhD theses, and abstracts were excluded.

Types of studies

The review included all human prospective and retrospective follow-up studies and clinical trials, cohort studies, case-control studies, and case series studies published between January 2011 and March 2016, on various soft-tissue conditions and plaque accumulation around dental implant influence on
peri-implantitis development. Case report studies were excluded.

**Information sources**

The search strategy incorporated examinations of electronic databases, supplemented by hand searches. A search was conducted on two databases - MEDLINE (Ovid) and EMBASE. Additionally, a hand search was carried out in dental implant related journals, including “Journal of Oral and Maxillofacial Implants”, “Clinical Oral Implants Research”, “European Journal of Oral Implantology”, “Journal of Oral and Maxillofacial Surgery”, “Journal of Clinical Periodontology”, “Journal of Periodontology”, “International Journal of Oral and Maxillofacial Surgery”, “The International Journal of Periodontics and Restorative Dentistry”. The references of each relevant study were screened to discover additional relevant publications and to improve the sensitivity of the search.

**Search**

The MEDLINE and EMBASE resource databases were explored through advanced searches. The keywords and search inquiries that were used during the primary stage were as follows: (("attached gingiva" OR "keratinized gingiva" OR “keratinized mucosa” OR “attached mucosa” OR “soft tissue condition” OR “soft tissue volume” OR “gingiva volume” OR “soft tissue height” OR “gingiva height” OR “soft tissue width” OR “gingiva width”)) AND ("peri-implantitis” OR “peri-implant pathology” OR “dental implant infections” OR “peri-implant bone loss” OR “peri-implant disease”) OR ("plaque” OR “plaque accumulation” OR “plaque index” OR “plaque control”)) AND (“peri-implantitis” OR “peri-implant pathology” OR “dental implant infections” OR “peri-implant bone loss” OR “peri-implant disease”). The choice of keywords was intended to be broad, in order to collect as much relevant data as possible without relying on electronic means alone to refine the search results.

**Selection of studies**

The resulting articles were independently subjected to clear inclusion and exclusion criteria by two reviewers as follows. Reviewers compared decisions and resolved differences through discussion, consulting a third party when consensus could not be reached. The third party was an experienced senior reviewer.

**Inclusion and exclusion criteria**

The applied inclusion criteria for studies were as follows:

- Investigated soft-tissue dimensions at implant sites and peri-implantitis occurrence;
- Followed-up plaque accumulation influence on peri-implantitis development;
- All human prospective or retrospective follow-up studies and clinical trials, cohort studies, case-control studies, and case series studies with at least 5 patients;
- A follow-up time period of at least 6 months after the placement of definitive prosthesis;
- Could not be excluded before careful reading.

The following articles were excluded:

- Studies that targeted soft-tissue condition around teeth;
- Studies where the effect of soft tissue condition and plaque accumulation on peri-implantitis could not be extracted from the data (e.g., a combination of other risk factors, including heavy smokers, systemic diseases, personal habits);
- Studies that included unclear data, with authors who could not be contacted for any reason.

**Sequential search strategy**

Following the initial literature search, all article titles were screened to eliminate irrelevant publications, review articles, case reports, and animal studies. Next, studies were excluded based on data obtained from screening the abstracts. The final stage of screening involved reading the full texts to confirm each study’s eligibility, based on the inclusion and exclusion criteria.

**Data extraction**

The data were independently extracted from studies in the form of variables, according to the aims and themes of the present review, as listed onwards.

**Data items**

Data were collected from the included articles and arranged in the following fields: year, follow-up period, patient number, implant number, plaque index, gingiva index, probing depth.

**Assessment of methodological quality**

The risk of bias assessment of the included trials was undertaken independently and in duplicate by at least two review authors as part of the data extraction process.
This was conducted using the recommended approach for assessing risk of bias in studies included in Cochrane reviews [19].

**Synthesis of results**

Relevant data of interest on the previously stated variables were collected and organised into table.

**Statistical analysis**

No meta-analyses could be performed due to the heterogeneity between the studies.

**RESULTS**

**Study selection**

Article review and data extraction were performed according to the PRISMA flow diagram. The initial search identified a total of 1071 articles. Following the screening of the article titles, 543 potentially relevant articles were identified. Independent screening of the abstracts resulted in the selection of 46 publications for possible inclusion. The inclusion and exclusion criteria were applied to the 46 full-text articles. Finally, 8 articles that met the predefined criteria were included in the systematic review (Figure 1).

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**NCBI PMC and PubMed database advanced search:**
- Search items: “attached gingiva” OR “keratinized gingiva” OR “keratinized mucosa” OR “attached mucosa” OR “soft tissue condition” OR “soft tissue volume” OR “gingiva volume” OR “soft tissue height” OR “gingiva height” OR “soft tissue width” OR “gingiva width” AND “peri-implantitis OR “peri-implant pathology” OR “dental implant infections” OR “peri-implant bone loss” OR “peri-implant disease” OR “plaque OR “plaque index” OR “plaque control” AND “peri-implantitis” OR “peri-implant pathology” OR “dental implant infections” OR “peri-implant bone loss” OR “peri-implant disease”;
- Investigated soft-tissue dimensions at implant sites and peri-implantitis occurrence;
- Followed up plaque accumulation influence on peri-implantitis development
- All human prospective or retrospective follow-up studies and clinical trials, cohort studies, case-control studies, and case series studies with at least 5 patients;
- A follow up-time period of at least 6 months;
- Could not be excluded before careful reading;
- Abstract available (n = 1071).

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**Figure 1.** Procedural flow of the literature search and selection process.
Five of them are prospective clinical studies (patients number: 287; implants number: 917), one cross-sectional study (patients: 109; implants: 202), one retrospective study (patients: 118; implants: 320) and one present cohort study (patients: 80; implants: 270). Total patients number: 594; total implants number: 1709.

Exclusion of studies

The reasons for excluding studies after full-text assessment were as follows: investigated soft-tissue dimensions at implant sites and peri-implantitis occurrence (n = 14), followed-up plaque accumulation influence on peri-implantitis development (n = 8), all human prospective or retrospective follow-up studies and clinical trials, cohort studies, case-control studies, and case series studies with at least 5 patients (n = 7), a follow-up time period of at least 6 months (n = 1), could not be excluded before careful reading (n = 7).

Study characteristics

The included studies were further divided into two groups: KM ≥ 2mm; KM < 2 mm. Also studies were compared regarding to the follow-up period, number of the patients, implants number and clinical parameters: plaque index (PI), gingival index (GI), probing depth (PD), bleeding on probing/modified bleeding index (BoP/mBI).

Synthesis of results

No meta-analysis could be performed due to the heterogeneity in the study designs and treatment modalities.

Influence of keratinized mucosa

Peri-implant soft-tissue inflammation, marginal tissue recession, PD, and attachment level are the clinical parameters commonly used for monitoring soft-tissue status of dental implants [20]. The clinical signs of BoP, mucosal recession, increasing PD, and loss of attachment level are always present with peri-implant disease [21]. Qualitative change of soft tissue, PI, GI, PD, bleeding index (BI), or BoP were used to determine the status of soft tissue inflammation (Table 1).

- **PI**: 6 of 8 studies showed significantly higher difference in the PI of the periimplant soft tissues [22-27].
- **GI**: four clinical studies reported [22-25] higher scores of GI in implants with narrow keratinized mucosa (< 2 mm).
- **PD**: 2 of 8 studies showed significantly smaller PD at implants with ≥ 2 mm width of keratinized mucosa [22,23].
- **BI/BoP**: using BI/BoP as an indicator of the presence of an inflammatory lesion in the peri-implant mucosa, 3 of 8 studies showed significantly higher prevalence of bleeding scores at implants with < 2 mm compared to ≥ 2 mm width of keratinized mucosa [24,26,27].

However, other studies showed that the width of keratinized mucosa around implants had no impact on PI, GI, PD and BI/BoP. The findings of those studies regarding the effect of the width of keratinized mucosa on soft-tissue inflammation are controversial, and impaired oral hygiene may play a role in the manifestation of mucosal inflammation around implants with minimal keratinized tissue. Several authors reported that significant elevation of GI and BI scores was accompanied by compromised plaque control at sites with narrow keratinized mucosa [22-25]. These results demonstrated that the amount of keratinized mucosa has little influence on soft-tissue inflammation in the presence of good oral hygiene. However, suboptimal oral hygiene due to difficulty in access for plaque control in the areas of minimal keratinized mucosa may lead to greater tissue damage. For the maintenance of soft-tissue health of dental implants, the capability to access oral hygiene at implant sites is more important than the width of keratinized mucosa.

Quality assessment

The quality assessment of the included studies revealed an unknown risk of bias (for one or more key domains) for the majority of the included studies [21-25,27,28], one study [26] was classified as low risk (of bias for all key domains) (Table 2).

**DISCUSSION**

Traditionally, the sufficient keratinized gingiva has been recognized to maintain healthy gingival tissues and to prevent gingival recession. Particularly, it has been believed that the success of implants is dependent on the ability of the mucosa endowing the appropriate biologic protective role between the oral environment and the implants [29]. According to several authors [30-32] reported that, in good oral hygiene conditions, the marginal gingiva around implants were clinically healthy, even when no keratinized mucosa was present.
### Table 1. Characteristics of the included studies with report on soft tissue condition around dental implants

| Study               | Year of publication | Follow-up period | Study design                  | Patients number | Implants number | PI KM ≥ 2 mm | PI KM < 2 mm | GI KM ≥ 2 mm | GI KM < 2 mm | PD KM ≥ 2 mm | PD KM < 2 mm | BoP/mBI KM ≥ 2 mm | BoP/mBI KM < 2 mm | P value  |
|---------------------|---------------------|------------------|--------------------------------|-----------------|-----------------|--------------|--------------|--------------|--------------|--------------|--------------|-----------------|-----------------|----------|
| Boynuegri et al. [21] | 2012 | 1 year | Prospective clinical study | 15 | 36 | 0.05 (0.19) | 0.28 (0.38) | < 0.05 | 0.07 (0.26) | 0.58 (0.6) | < 0.05 | 0.24 (0.3) | 0.39 (0.36) | > 0.05 |
| Chung et al. [22] | 2006 | > 3 years | Prospective clinical study | 69 | 339 | 1.26 (0.05) | 1.51 (0.09) | < 0.05 | 0.76 (0.04) | 0.94 (0.07) | < 0.05 | 2.9 (0.05) | 2.85 (0.06) | > 0.05 |
| Adibrad et al. [23] | 2009 | 2 years | Prospective clinical study | 27 | 66 | 1.2 (0.71) | 1.87 (0.59) | 0.02 | 1.01 (0.67) | 1.65 (0.78) | 0.01 | 2.98 (0.51) | 3.11 (0.56) | 0.115 |
| Bouri et al. [24] | 2008 | 1 year | Prospective clinical study | 76 | 200 | 1.25 (0.53) | 1.78 (0.78) | < 0.001 | 0.91 (0.72) | 1.5 (0.77) | < 0.001 | 3.72 (0.75) | 3.87 (0.66) | 0.132 |
| Romanos et al. [25] | 2015 | > 2.6 years | Retrospective study | 118 | 320 | 0.45 (0.56) | 0.69 (0.63) | 0.001 | 3.72 (0.75) | 3.87 (0.66) | 0.132 | 0.11 (0.31) | 0.31 (0.52) | < 0.0001 |
| Souza et al. [26] | 2015 | 1 year | Present cohort study | 80 | 270 | 0.6 (0.51) | 0.92 (0.52) | 0.008 | 2.36 (0.41) | 2.43 (0.65) | 0.582 | 51 (27.2)% | 63.8 (29.3)% | 0.033 |
| Kim et al. [31] | 2009 | 13 months | Prospective clinical study | 100 | 276 | 0.74 (0.83) | 0.74 (0.91) | 0.943 | 0.38 (0.66) | 0.44 (0.72) | 0.472 | 2.84 (1.8) | 2.62 (1.55) | 0.328 |
| Esper et al. [32] | 2012 | 1 year | Cross-sectional study | 109 | 202 | 0.6 (0.62) | 0.67 (0.71) | 0.487 | 1.25 (0.61) | 1.11 (0.58) | 0.127 | 3.02 (1.05) | 2.43 (1.02) | < 0.001 |

PI = plaque index; GI = gingival index; PD = pocket depth; BoP/mBI = bleeding on probing/modified bleeding index; SD = standard deviation.

### Table 2. Bias summary

| Study               | Year of publication | Random sequence generation | Allocation concealment | Blinding of outcome assessment | Incomplete outcome data | Selective reporting | Other sources of bias |
|---------------------|---------------------|---------------------------|------------------------|-------------------------------|------------------------|---------------------|----------------------|
| Boynuegri et al. [21] | 2012 | ? | ? | ? | + | + | + |
| Chung et al. [22] | 2006 | ? | ? | + | + | + | + |
| Adibrad et al. [23] | 2009 | ? | ? | + | + | + | + |
| Bouri et al. [24] | 2008 | + | ? | + | + | + | + |
| Romanos et al. [25] | 2015 | ? | ? | + | + | + | + |
| Souza et al. [26] | 2015 | + | + | + | + | + | + |
| Kim et al. [31] | 2009 | ? | ? | ? | + | + | + |
| Esper et al. [32] | 2012 | ? | ? | ? | + | + | + |

+= low risk; ?= unclear risk; - = high risk.
On the other hand, more investigators [20-27] with newer data reported an association between implant survival and width of keratinized gingival. In our findings, some researches [24,26,27] showed that implants with narrow zones of keratinized gingiva had more BoP. In the peri-implant sulcus, the collagen fibers are orientated parallel to the implant surface, in contrast to the collagen fibers adjacent to the natural teeth, which are perpendicularly orientated and anchored in the cementum. The absence of these horizontal collagen fibers will result in less resistance on probing. This will lead to a local tissue trauma and some bleeding, even in clinically healthy peri-implant tissue [19].

We did not find any correlation between KM widths and PD. Our finding is supported by previous studies [23-26,32] that showed that KM width was positively correlated to PD. It was reported that at sites with healthy mucosa or mucositis, the tip of the probe may identify the location of the apical level of the barrier epithelium [33]. At sites with peri-implantitis, however, the probe will penetrate apical to the epithelium and reach the base of the inflammatory lesion at the alveolar bone crest. Consequently, an increased probing depth will result. Schou et al. [34] reported that probing depth measurements at implant and teeth yielded different information, and small alterations in probing depth at implants may reflect changes in soft tissue inflammation rather than loss of supporting tissues.

This study showed that implants with narrow zones of keratinized tissue (< 2 mm) had significantly more plaque and signs of inflammation than those with wider zones of keratinized gingiva (≥ 2 mm). These findings are supported by previous studies [22,25-27] that demonstrated that the absence of adequate keratinized mucosa in endosseous dental implants, especially in posterior implants, was associated with higher plaque accumulation and gingival inflammation. In fact, good oral hygiene is very difficult to achieve around dental restorations without the protection of a band of keratinized gingival tissue. Therefore, in order to achieve long-term stable peri-implant health, it is important to achieve an adequate soft tissue seal around dental implant/restorations [35]. Several studies [36,37] have shown the use of free soft tissue grafts to augment keratinized gingiva in conjunction with implant placement, around present dental implant or following the restoration of an implant. The rationale for performing the procedures include making plaque control more effective, facilitating impression taking by the restorative dentist and dissipating muscular and frenal pull, and possibly preventing further recession [38,39].

In conclusion, the present study demonstrated that patients with < 2 mm of KM exhibited higher levels of peri-implant discomfort during brushing, plaque, and peri-implant inflammation. Further studies are necessary to evaluate whether patients reporting brushing discomfort at implant sites are more likely to develop peri-implantitis.

**CONCLUSIONS**

This systematic review has highlighted a number of studies examining the clinical relevance of keratinized mucosa around dental implants in preventing peri-implant disease. All studies concluded that the width of keratinized mucosa around dental implants was related with less mucosal inflammation, less plaque accumulation, increased stability of the peri-implant area, and prevention of mucosal recession leading to loss of implant. Within the limitations of the current review, the following conclusions may be drawn:

1. The absence of adequate keratinized mucosa around implants supporting overdentures was associated with higher plaque accumulation, gingival inflammation and bleeding on probing.
2. Only one study reported that in cases with insufficient keratinized gingiva in the vicinity of implants, the insufficiency does not necessarily mediate adverse effects on the hygiene management and soft tissue health condition.

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