Peri-implant Mucosal Tissues and Inflammation: Clinical Implications

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Purpose: *The purpose of this review was to explore the available literature and compile studies that discuss the relevance of the biofilm, onset and progression of disease, critical peri-implant pocket depth, frequency of supportive implant therapy, excess cement, and keratinized peri-implant tissues as related to peri-implant disease.* ***Materials and Methods:*** *PubMed, Cochrane Oral Health Group Specialized Trial Register, and hand searches of related journals were performed in relationship to the focused question. Reports describing techniques, preclinical studies, and case reports were excluded.* ***Results:*** *Due to the absence of controlled studies, a meta-analysis could not be performed. Summaries of relevant publications were completed for each topic area. Clinical recommendations were developed to provide guidance to the practitioner.* ***Conclusion:*** *The importance of proper diagnosis, planning, and clinical treatment cannot be overstated. Patient factors including systemic disease, periodontal status, and oral hygiene significantly impact peri-implant health. Clinician factors such as implant position, excess cement, and restorative design can contribute to development of peri-implant disease. Surveillance of implant status is essential and can be assisted by the assessment of risk factors, establishment of a proper recall program, and monitoring changes in bone and peri-implant pocket depths.* Int J Oral Maxillofac Implants 2019;34(suppl):s25–s33. doi: 10.11607/jomi.19suppl.g2

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Over the past almost five decades, dental implants have been a predictable therapy to replace missing teeth. The field has also evolved to include more aggressive loading protocols, osseous augmentation of deficient bone areas, and modification of the implant designs. These, along with other factors, have led to an expansion in the number of implants placed and their more widespread use by both general practitioners and dental specialists. The importance of proper diagnosis, planning, and clinical treatment cannot be overstated. However, survival/success have been found to be affected by a number of risk factors resulting in peri-implant diseases. Peri-implant diseases have been characterized as a condition of the tissues around osseointegrated implants with signs of inflammation (bleeding and/or suppuration on probing) with or without loss of supporting bone.

Early on, Meffert (1992) described the ailing and failing implant.[1] The ailing implant had bone loss with pocketing but was static at maintenance visits, whereas the failing implant also demonstrated bone loss with pocketing but presented additionally with bleeding on probing, purulence, and continued bone loss despite therapy. Misch (1998) also utilized clinical parameters to assess implant health.[2] A continuum of health to disease was described with disease status related to a progressive worsening of clinical parameters

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such as probing depth, bone loss, and pain. Froum and Rosen (2012) established thresholds for clinical parameters as a basis for classification of peri-implantitis.[3] Combinations of bleeding on probing and/or suppuration, probing depth, and radiographic bone loss were utilized to classify peri-implantitis into early, moderate, and advanced categories. Recently, a classification system based on etiology was proposed.[4] Its results indicated that the majority of bone loss was related to biofilm, followed by iatrogenic factors, exogenous irritants, absence of keratinized tissue, and extrinsic pathology.

Although the literature denotes many risk factors associated with peri-implant diseases, the purpose of this review was to explore the available literature and compile studies that discuss the relevance of the biofilm, onset and progression of disease, critical peri-implant pocket depth, frequency of supportive implant therapy, excess cement, and keratinized peri-implant tissues as related to peri-implant disease.

MATERIALS AND METHODS

Rationale

This systematic review evaluated the relationship of various risk factors as they pertain to dental implant disease.

Focused Question

In patients with dental implants, what is the relationship of biofilm, onset and progression of disease, critical peri-implant pocket depth, frequency of supportive implant therapy, excess cement, and keratinized peri-implant tissues with peri-implant disease?

Search Protocol

PubMed and Cochrane Oral Health Group Specialized Trial Register were searched. Hand searches were performed on *Clinical Oral Implants Research, The International Journal of Oral and Maxillofacial Implants, The International Journal of Periodontics and Restorative Dentistry, Journal of Clinical Periodontology, Journal of Periodontology,* and *Journal of Periodontal Research*.

Selection Criteria

Publications reporting mucositis of dental implants related to biofilm, keratinized peri-implant tissues, peri-implant pocket depth, frequency of recall, and residual cement were included in the analysis. Reports describing techniques were excluded.

Data Collection and Analysis

Due to the absence of controlled studies, a meta-analysis was not performed.

Biofilm and Inflammatory Response to Peri-implant Disease

The biofilm related to peri-implant diseases has not been extensively documented. The literature for peri-implant diseases follow those of gingivitis and periodontitis with regard to microflora and inflammatory response. As Loe et al (1965) induced an experimental gingivitis,[5] several studies have described the response around dental implants.[6–9] They indicate that the response to the cessation of oral hygiene around implants and its onset are similar to that around the natural teeth. The classic signs of inflammation—Gingival Index, bleeding on probing, and Plaque Index—were increased. Salvi et al (2012) also induced an experimental peri-implant mucositis and found similar results.[6] However, the Gingival Index was higher around dental implants when compared to teeth. The authors postulated that the inflammatory response for dental implants is more severe than for the natural dentition. In addition, they found that with the reintroduction of oral hygiene measures, the soft tissues recovered to baseline values.[6] Zitzmann et al (2001) evaluated the cellular response with an experimentally induced peri-implant mucositis with tissue biopsies.[7] The response of T- and B-cells was similar to that around teeth. At 21 days, there was an increase in the volume of these lymphocytes. Lastly, host biomarkers including IL1-β, TNF-α, and TGF-β2 have been evaluated in the induced peri-implant mucositis model.[10] Only IL-1β increased over the 3-week period in the gingival crevicular fluid. Following reinstitution of oral hygiene, IL1-β levels returned to baseline. In natural longstanding peri-implant mucositis lesions, sites that bled on probing and had some degree of redness and inflammation demonstrated on biopsy infiltrate in the connective tissue.[11]

The biofilm conversion from peri-implant mucositis to peri-implantitis has been only documented with cross-sectional studies describing the characteristics of peri-implantitis. The microbiome of healthy and/or diseased implants have been compared.[12,13] In general, species of the red complex such as *Porphyromonas gingivalis* and *Tannerella forsythia* have been found in diseased sites. Sanz-Martin et al (2017) studied healthy and diseased implants, illustrating that both states had a core microbiome, while health had taxa consistent with periodontal health and peri-implantitis had taxa consistent with periodontitis.[14] In addition, the findings regarding peri-implantitis biofilm have shown species including *Synergistetes* and *Tannerella*. In a systematic review by Lafaurie et al (2017), peri-implantitis was an infection with a diversity of microorganisms including periodontal pathogens, anaerobic Gram-negative rods, and rarely, entric rods and *Staphylococcus aureus*.[15] Overall, the majority of information

available indicates that a shift occurs from health to disease with regard to the biofilm. The biofilm associated with the diseased dental implant seems to be more pathogenic and somewhat like those in periodontitis.

Clinical Recommendations

- The clinician should be aware that a shift in the biofilm and inflammatory response occurs with the conversion of a healthy to diseased implant state. For example, with the deepening of a peri-implant pocket, the practitioner should consider additional measures to monitor and/or treat the failing implant.
- The biofilm associated with peri-implant disease may be similar to that in periodontitis. As a result, the susceptible periodontitis patient with dental implants should be carefully monitored.
- Given the inflammatory basis of peri-implant diseases, the surgical and restorative clinician should carefully consider the contour, function, and overall cleansability of the prosthesis throughout the planning process.

Time to Onset and Progression of Peri-implant Diseases

The development of peri-implant diseases seems dependent on many variables, such as oral hygiene, quality of tissue, and restoration type. However, unlike periodontitis, factors such as position of the implant within the edentulous ridge, implant surface texture, etc, impact the development of both mucositis and/or peri-implantitis. In addition, the pattern and rate of onset do not follow that of periodontal disease but may be influenced by the presence or history of periodontitis.

Studies involving experimentally induced peri-implantitis demonstrate clinical changes as in an induced gingivitis.[6] The cessation of oral hygiene produced increases in plaque, Gingival Index, and bleeding. When oral hygiene was reinstituted, clinical parameters recovered.[8] The analysis of biologic responses to the induction of peri-implant mucositis indicates an upregulation of peri-implant crevicular fluid factors and the cellular response involving an increase in the proportions of T and B cells.[7] Overall, these studies indicate that the clinical biologic response as well as timing of progression in the experimental induction of peri-implant mucositis collates with induced gingivitis.

In a retrospective review of dental radiographs, Fransson et al (2010) evaluated the onset and progression of bone loss.[16] A total of 182 patients with 419 implants were assessed for bone loss and had a mean follow-up period of 11.1 years. Bone loss after the first year averaged 1.68 mm. Interestingly, 32% of the implants had bone loss of ≥ 2 mm, and 10% of those had bone loss ≥ 3 mm. Analysis indicated that progression was nonlinear and the rate decreased with time. Derks et al (2016) also found that the pattern of bone loss was nonlinear.[17] A retrospective review of 105 implant dental radiographs indicated that the onset generally occurred within the first 3 years of function. However, compared to Fransson et al (2010),[16] the amount of bone loss was greater with an average of 3.5 mm. Bone loss of more than 3 mm occurred in 51% of the sample. The authors postulated that the bone loss could be related to a prior history of periodontitis. In a shorter-duration study, Schwarz et al (2017) found that of 512 implants, a total of 262 were diagnosed with peri-implant disease.[18] The majority of disease that occurred with implants was found between 12 and 48 months. After 48 months, the rate of disease was similar to that between 1 and 12 months. These results again indicate that peri-implant disease occurs early in the life of a functionally loaded implant. Recently, Sarmiento et al (2018) found that the timing of grafting influenced the rate of peri-implant disease.[19] In a staged grafting procedure where the implant is placed following a healing period, the rate of peri-implant mucositis was 8.1% and the rate of peri-implantitis was 4.4%. However, when grafting occurred at the time of implant placement, the rate of peri-implant mucositis was 7.5% and the rate of peri-implantitis was 9.7%. In addition, the onset of mucositis preceded the statistical detection of radiographic bone loss.

Clinical Recommendations

- Although limited, studies indicate that the onset and the majority of peri-implant disease progressions occur in the life of a functionally loaded implant generally prior to 36 months.
- Since the onset and progression of peri-implant diseases seem not to follow patterns of periodontitis, the clinician should have an enhanced awareness of risk and should structure an individualized recall program for the patient.

Critical Peri-implant Pocket Depth

The establishment of the biologic width with implant healing has been well documented. Although the dimensions seem to vary when compared to the natural dentition, the formation of an epithelial and connective tissue attachment occurs. The healing results in the formation of a peri-implant sulcus. Several factors, such as surface characteristics, implant height above the bone level, platform switching, microgap, abutment size, etc, influence the final measurements. With the onset of peri-implant disease, these structures begin to change much in the same manner as with periodontitis. Gingival inflammation can increase the peri-implant sulcus and with loss of supporting bone, apical migration of the connective tissue and

epithelium occurs. The disease around an implant with bone loss then forms a deepened peri-implant pocket.

Healthy peri-implant mucosa can be altered by factors that progress to conditions of peri-implant mucositis and peri-implantitis. Although clinical assessment can differentiate between a state of health and disease, a critical implant pocket depth can assist in defining this transitional state. Derks et al (2016) described healthy peri-implant tissue by the absence of bleeding on probing, mucositis as presence of bleeding on probing ± suppuration without radiographic bone loss, and peri-implantitis as bleeding on probing ± suppuration and radiographic bone loss > 0.5 mm (45% of cases presented).[17] The onset of disease occurred early, with the majority of peri-implantitis cases/implants occurring within 3 years of function. Schwarz et al (2018) measured peri-implant sulcus/pocket depth values of 1 to 3 mm and 4 to 6 mm for healthy and mucositis implants, respectively.[20] Peri-implantitis implants only recorded pocket values in the 4- to 6-mm range. Schwarz et al (2017) noted that mucositis and peri-implantitis could present within 12 to 48 months after initial implant placement.[18]

Recently, Monje et al (2018) evaluated 1,572 sites around 262 implants in 141 patients.[21] The clinical parameters included the evaluation of pocket depth. In this cross-sectional matched case-control study, healthy implants had a mean pocket depth of 2.63 mm, mucositis-classified implants had a mean pocket depth of 3.26 mm, and peri-implantitis–diagnosed implants had a mean pocket depth of 4.58 mm. The authors concluded that pocket depth might accurately discern between diagnoses among peri-implant conditions. Similarly, Ramanauskaite et al (2018) evaluated 269 dental implants with several different clinical parameters including peri-implant pocket depth.[22] A total of 77 dental implants diagnosed as healthy had a mean pocket depth of 2.95 mm. Peri-implant mucositis implants (n = 77) had a mean pocket depth of 3.10 mm, whereas peri-implantitis implants (n = 115) had a mean pocket depth of 4.91 mm. These differences in pocket depth between the three groups were significantly different.

Clinical Recommendations

- The documentation of dental implant health status is an important component of clinical care. During the treatment process, a baseline radiograph (at final prosthesis insertion) and clinical parameters (pocket depth, bleeding on probing, etc) should be recorded. The frequency of periodic documentation should be based on individual risk factors in order to assess changes in implant health over the life of the implant.
- The apparently healthy implant may still harbor an environment (biofilm, inflammation, etc) that could predispose the implant to convert to disease. Studies indicate that the clinician should be aware of the development of future markers for peri-implant disease and conditions including perfuse bleeding on probing, suppuration, bone loss, and critical implant pocket depth of ≥ 5 mm. This awareness includes but is not limited to continuing routine care, radiographic assessment, oral hygiene instruction, and shortened recall interval.

Frequency of Supportive Implant Therapy

According to contemporary studies, there is an estimated 43% prevalence of peri-implant mucositis and a 22% prevalence of peri-implantitis.[23–25] A majority of this peri-implant disease may share a pathogenesis with periodontitis.[26] Therefore, peri-implant maintenance seems to be critical to maintain the stability of the tissue around dental implants.[27,28]

Despite the understanding of the importance of peri-implant maintenance, there have been no well-controlled investigations that refine the peri-implant recall interval; this is due to various definitions of diagnosis, unclear terminology, varying follow-up periods, and ethical considerations. Thus, most of the evidence of supportive implant therapy interval has been obtained from retrospective studies or reviews.

The prevalence rates of peri-implant diseases were evaluated in 89 patients.[29] The patients were assigned to 3-month intervals during the first year after implant placement and later on 6-month intervals. Patients who did not participate in regular supportive implant therapy had an 11-fold higher chance of peri-implantitis than patients showing good compliance. On the contrary, patients who did not have regular supportive implant therapy were reported to have up to 48% of the prevalence of peri-implant mucositis during an observation period of 9 to 14 years.[30] These studies have confirmed the essential role of supportive implant therapy to maintain tissue health and that the lack of supportive implant therapy will lead to a higher prevalence of the peri-implant disease.

A retrospective study done by Frisch et al (2015) evaluated the compliance and the frequency of the supportive post-implant therapy program and indicated a positive correlation between lower compliance and increased probing depth and higher plaque rate.[31] The compliance rates were categorized into five different groups, from a 3-month interval to no compliance at all. The higher rates of patient compliance (86% to 94%) were observed during the first 3 years. A significant correlation was found between lower compliance and increased peri-implant probing depth. In addition,

higher plaque rates were found in individuals with lower compliance rates.

Dental implant patients may have a higher risk of peri-implant disease due to diabetes, poor oral hygiene, and smoking.[29,32–34] A history of periodontitis seems to be the most documented risk for future peri-implant disease.[28,35,36] Long-term studies done by Roccuzzo and coworkers evaluated the tissue around dental implants with supportive implant therapy for 10 years.[37–39] The study found that in periodontally healthy patients there were no statistical differences in clinical parameters if subjects adhered to their supportive implant therapy or not. In contrast, the patients who had moderate to severe periodontal disease had higher plaque scores, bleeding scores during the supportive implant therapy, and eventually implant loss. In a systematic review, Monje et al (2016) analyzed 13 studies to evaluate the impact of supportive maintenance on the implant.[40] This review successfully provides positive evidence of the patient with maintenance and concluded a minimum recall post-implant maintenance therapy of 5 to 6 months. However, the studies did indicate that the possibility of biologic complications might still occur and other risk factors should be appropriately considered.

In 2013, Aguirre-Zorzano et al stated that the prevalence of peri-implant inflammatory disease in periodontal patients who regularly undergo supportive implant therapy with a mean recall of 4 months is clinically significantly lower and the peri-implant disease could be even prevented.[41] A similar result was found in a retrospective study by Costa et al (2012), which suggested that the simple fact of enrolling subjects for supportive implant therapy may reduce the risk of peri-implantitis from 43.9% to 18% at the patient level with maintenance at least once a year.[42] These findings provide clinical evidence that the supportive implant therapy interval should be adjusted case by case, particularly in patients with a history of periodontitis.

Clinical Recommendations

- In general, a reasonable interval of supportive implant therapy is 5 to 6 months for a patient with low risk of peri-implant disease, but should this be evaluated case by case.
- When considering a patient with a history of risk, such as periodontal disease, it may be necessary to shorten the supportive implant therapy interval.
- During a supportive implant therapy appointment, reinforcement of oral hygiene, modification of factors such as smoking, and removal and cleaning of the prosthesis should be considered.

Role of Excess Cement in Peri-implant Diseases

Cemented restorations are commonly used to restore dental implants. It has been shown that excess cement left in the sulcus around implant-supported restorations can cause inflammation, ultimately leading to peri-implant disease. Cemented restorations offer several advantages, such as ease of prosthetic fabrication, reduced costs, increased framework passivity, and improved esthetics due to the absence of the buccal screw access hole. The main disadvantage of cement-retained restorations is the fact that cements are a flowable material that can spread unintentionally to the adjacent gingival tissues, making it difficult for the clinician to properly remove excess subgingival material. Therefore, caution on implant selection, placement, and prosthetic design should play a key role in the initial treatment plan. Although we aim for screw restoration as a primary election to restore a dental implant, it is not always feasible if angulation of the implant body is necessary.

A study published by Wilson (2009) demonstrated how the presence of peri-implantitis caused by dental cements was observed in 81% of cases.[43] Korsch and Walther (2015) compared various types of cements and associated peri-implant disease. The authors found that the frequency of excess cement depended on cement type.[44] For implants that used methacrylate cement, the frequency of excess cement was 62%, whereas the frequency of excess cement for zinc oxide-eugenol cement was 100%. Although several publications have documented different peri-implant complications, few have addressed the causative factor for the biologic breakdown. In a study published by Sarmiento et al (2016), the classification system based on etiologies included 5.5% of cases with peri-implantitis induced by an exogenous factor, eg, residual excess cement.[4] Another retrospective study by Linkevicius et al (2013) concluded that patients with previous periodontal disease may also be more predisposed to peri-implantitis due to excess cement.[45] Other studies have shown that the presence of suppuration around dental implants was greater on crowns that were cemented versus screw-retained.[46] Many authors have shown different techniques to reduce excess cement complications. Linkevicius et al (2013) in a clinical study showed that dental radiographs should not be considered as a reliable method for cement excess evaluation.[47] His results revealed that cement remnants were around 7.5% to 11.5% depending on the location of detection. Wadhwani et al (2010) proved that radiographic density of implant restorative cements is poor and depending

on the thickness of the cement, smaller pieces would remain unseen.[48] They suggested placing margins supragingival for proper cement removal.

Challenges for the clinician on when to place a cement-retained restoration should not be based on material selection to achieve higher esthetics, but rather the criteria should be those unique to cementation technique and proper treatment planning. Therefore, avoiding cement-retained restorations in difficult-to-access areas should be recommended for clinicians with less experience.

Clinical Recommendations

- All attempts to reduce excess submarginal cement should be made through the use of screw-retained restorations or abutments designed to minimize the depth of the cement line in relation to the peri-implant tissue architecture. The cement line should be located with a minimal depth for ease of removal and to minimize submarginal excess cement.
- A final radiograph should be carried out routinely to assess unwanted cement beyond the gingival margin.
- Postoperative assessment is recommended within the first weeks post–crown insertion. This should allow immediate clinical detection of excess cement and avoid peri-implant tissue inflammation.

Role of Keratinized Tissue Surrounding Dental Implants

Peri-implant health has been defined both clinically and histologically.[49] Peri-implant tissues surround an osseointegrated dental implant and can be divided into hard and soft tissue components. The hard tissue component forms a contact relationship to the implant surface, which contributes to implant stability.[50] The soft tissue component is formed during the healing process following implant/prosthetic placement.[51] The peri-implant tissues protect the bone that supports the implant. With the absence of healthy peri-implant tissues, the long-term implant success and survival becomes compromised and less predictable.[42] However, if there is an insufficient amount of keratinized tissue around implants, is there an increased risk of peri-implant mucositis? If so, what types of modalities are appropriate to address a lack of keratinized tissue of the peri-implant tissues?

Does the Absence of Keratinized Tissue Influence Peri-implant Mucositis? Keratinized tissue or mucosa, which extends from the margin of the peri-implant mucosa to the mucogingival junction, is composed of fibrous connective tissue with fibroblasts; type I, III, IV, V, and VI collagen; and an orthokeratinized squamous epithelium.[52,53] Keratinized gingiva has been defined as marginal and attached gingiva that excludes soft tissue of the interdental col region—interproximal gingival tissue between posterior teeth where epithelium is devoid of keratinization.[54] One theory explaining the reduction of keratinized tissue is the post–tooth extraction natural loss of crestal bone. The buccal thickness of keratinized tissue is greater at the base of implants than at teeth (2.0 mm vs 1.1 mm, respectively).[53] According to a new classification scheme for periodontal and peri-implant diseases and conditions, peri-implant mucositis is defined as an inflammatory lesion of the mucosa surrounding an endosseous implant without loss of supporting peri-implant bone.[34,55,56] This is clinically determined by the presence of redness, swelling, bleeding on probing, and suppuration. The dimensions of peri-implant keratinized mucosa may be a risk indicator for peri-implant mucositis. The need for a minimum amount of keratinized tissue in order to maintain peri-implant tissue health has been a controversial issue.[57–61] Some studies suggested that plaque accumulation that resulted in marginal inflammation was more frequent at implant sites with < 2 mm of keratinized tissue.[62–66] However, several studies suggested that the lack of a minimum amount of keratinized tissue was not associated with mucosal inflammation.[64,67–72]

A systematic review assessed seven cross-sectional and four longitudinal studies to determine if keratinized mucosa affected implant health, suggesting that a lack of adequate keratinized tissue around endosseous dental implants is associated with plaque accumulation, tissue inflammation, recession, and attachment loss.[58] This supported a meta-analysis that indicated a statistically significant difference between plaque scores and modified Gingival Index in favor of sites with a wider dimension for keratinized tissue.[59] The width of keratinized tissue at implant sites is another area of controversy.[73] The association of keratinized mucosa width at implant sites was studied in a small group of patients 5 to 10 years retrospectively. Statistical analysis failed to indicate an association between keratinized tissue or the mobility of marginal mucosa around implant sites with plaque accumulation, bleeding on probing, or probing depth.[69] Another longitudinal study of 339 implants measuring the amount of keratinized mucosa present over a period of at least 3 years showed a higher Gingival Index (0.9 vs 0.8) and modified Plaque Index (1.5 vs 1.3) in patients with keratinized tissue with < 2 mm and > 2 mm, respectively.[62] A 5-year study involving 307 implants in edentulous mandibles with fixed implant-retained reconstructions assessed sites with < 2 mm and > 2 mm of keratinized tissue. The investigators reported higher plaque scores (0.7 vs 0.4) and bleeding on probing (0.2 vs 0.1) at lingual sites as well as

recession (0.7 vs 0.1) at buccal sites.[72] Another study involving 15 patients with mandibular overdentures on four implants assessed the presence or absence of keratinized tissue on buccal aspects of implants, showing that 19 implants with at least 2 mm of keratinized mucosa had lower plaque (0.3 vs 0.6) and gingival indices (0.1 vs 0.6) than 17 implants without keratinized mucosa.[66] It has been suggested that the evidence is equivocal regarding the effect of keratinized tissue on the long-term health of the peri-implant tissue. The notable advantages are the ease of plaque removal and patient comfort.[74–78]

Does Grafting Help in the Management of Peri-implant Mucositis? Historically, scientific evidence reported that a lack of keratinized tissue was not critical to maintain peri-implant soft tissue health nor to result in more peri-implant diseases.[69,79] However, based on current evidence, it has been suggested that an increased amount of keratinized tissue may better preserve both soft and hard tissue stability, resulting in a favorable long-term outcome for dental implants (as well as better oral hygiene maintenance over time).[63,72,73] Also, an increased amount of keratinized tissue thickness may decrease the risk of recessions with immediate implants.[80] Therefore, periodontal surgical procedures that augment soft tissue volume are recommended for esthetic and dimensional advantages following tooth extraction and implant therapy for both immediate and delayed placement.[81–84]

Bleeding on probing was discussed in two studies with respect to grafting and nongrafting treatments after implant placement. According to a long-term study by Roccuzzo et al, there was an insignificant difference (23% and 27%) between groups with or without soft tissue grafting.[65] However, in another study there was notable improvement from mean baseline values of 85% to 30% with autogenous soft tissue grafting compared with 40% to 95% to 25% to 95% without soft tissue grafting.[83] In the same study, the mean Gingival Index shared a similar notable improvement comparing soft tissue grafting with no soft tissue grafting after follow-up periods of 6 to 12 months. Multiple studies indicated that there was a significant benefit to lower plaque values following surgical intervention when increasing keratinized tissue.[65] Conversely, one study compared Plaque Index of treated and untreated groups over time and found no significant difference at baseline and at 12 months.[85]

With regard to probing depth, there were no significant changes over time between the different treatment groups of apically positioned flap versus apically positioned flap plus free gingival graft in a meta-analysis.[86] The mean probing depth values at baseline for soft tissue grafting, 1.97 to 3.09 mm, reduced to 2.08 to 3.18 mm after 6 to 12 months, while no soft tissue grafting ranged from 1.76 to 3.25 mm at baseline and 1.60 to 3.62 mm after 6 to 12 months. Comparing these final probing depth values favored the apically positioned flap plus autogenous tissue. In a clinical study of 30 patients with keratinized tissue of < 1 mm at implant sites, half of the patients underwent surgery to widen the band of keratinized mucosa. After 10 years, there was a significant difference in the gain of keratinized mucosa of 3.1 mm versus 0 mm in patients who underwent surgery and patients who did not, respectively. However, the Plaque Index, bleeding on probing, and presence of peri-implantitis was not different between both groups.[31] Lastly, a meta-analysis assessing three different treatment modalities (autogenous, collagen matrix, and apically positioned flap) used for implant site maintenance with > 2 mm, < 2 mm, or no keratinized tissue resulted in statistically significant differences favoring apically positioned flap plus autogenous tissue[65] in a period of 6 months.

Although there has been an increase of literature to support the use of soft tissue augmentation for keratinized tissue around peri-implant tissues, unfortunately there is a lack of data regarding clinical long-term outcomes associated with peri-implant soft tissue augmentation procedures.

Clinical Recommendations

- The removal of plaque around peri-implant tissues should be performed as part of a routine periodontal maintenance program to prevent the progression of peri-implant diseases and conditions.
- Soft tissue augmentation surgery should be performed around dental implants to provide stable keratinized tissue.

CONCLUSIONS

The importance of proper diagnosis, planning, and clinical treatment cannot be overstated. Patient factors including systemic disease, periodontal status, and oral hygiene significantly impact peri-implant health. Clinician factors such as implant position, excess cement, and restorative design can contribute to development of peri-implant disease. Surveillance of implant status is essential and can be assisted by assessment of risk factors, establishment of a proper recall program, and monitoring changes in bone and peri-implant pocket depths.

DISCLAIMER

The authors have no direct financial interests with the products and instruments listed in the paper.

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