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Research Article

Soft Tissue Considerartion In Implantology: A Review

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ABSTRACT

The condition of the peri-implant tissues influences the long-term success of dental implants. The absence of keratinized gingiva (KG) may be a risk factor for developing recession or peri-implantitis. However, the need for keratinized gingiva around dental implants remains ambiguous. The preservation and reconstruction of soft tissue around dental implants is an integral component of dental Implantology. There is no long-term evidence that enhanced soft tissues can be sustained over time and alter peri-implant bone levels. The purpose of this review is to critically evaluate the importance of KG around implants, as well as the approaches for preserving and augmenting KG.

KEYWORDS: Keratinized Mucosa, Biologic Width, Peri-Implant soft tissue, Implant, Soft tissue Augmentation

INTRODUCTION

Implants have made tremendous advancements in dentistry ever since the osseointegration theory was first proposed. Osseointegration, being the main stay in implant dentistry, has been the ultimate goal for the dentists to achieve.[1]

In current research, initial bone modelling around implants in the first year after implantation is a challenging issue. The quality and quantity of soft tissue surrounding the implant have been demonstrated to influence hard tissue maintenance and preservation, which is a necessity for long-term aesthetic and functional implant-supported restorations. [2]

The increased prevalence of peri-implant bone loss and the more rapid progression of periodontal disease around implants compared to teeth has resulted in a paradigm shift with a focus on the importance of the peri-implant soft tissue in its ability to protect the underling bone and limit early marginal bone loss. Albrektsson [3] recently coined this a new era of mucointegration. When examining a site for dental implant placement, the current paradigm shift dictates that we must evaluate not just the availability of bone to house the implant but also the quality of the soft tissue at the site including the width of Keratinized Mucosa (KM) and the thickness and height of the mucosa.

The main purpose of this review is to provide overview about the functional significance of biological width around implant, the need for KG around implants and the techniques to preserve and augment KG around implant.

IMPLANT-SOFT TISSUE INTERFACE

The tissue that surrounds implants is known as peri-implant tissue and is comprised of soft (mucosa) and hard (bone) tissues. The peri-implant soft tissue has similar features to the soft tissue that surrounds teeth. It consists of a Junctional Epithelium (JE) and Connective Tissue (CT). The JE is attached to the implant and/or abutment surface through a hemi-desmosomal attachment. CT is present apical to the JE and coronal to the crest of alveolar bone.[4]

CT fibres are found to be positioned close to the implant surface but not attached to it, and predominantly arranged in a circular manner. CT fibres also arise from the crest of alveolar bone and from the periosteum and are oriented parallel to the implant/ abutment surface and extend towards the oral epithelium. Thus, the JE

and CT form a protective seal between the oral environment and the peri-implant bone which plays a vital role in the success of the implant treatment outcome. The JE and the CT are collectively known as the biologic width, which is comparable to that found around teeth.[4]

BIOLOGICAL WIDTH

The concept of biologic width in natural tooth was proposed in 1961. The biological width around natural tooth is 2 mm which is composed of 0.97 mm wide junctional epithelium and 1.07mm wide fibrous connective tissue, which is a distance between gingival sulcus and alveolar crest. The vital significance of biological width around the natural tooth is to protest it from external stimulation. **Berglund et al**[5] proposed that biological width existed around implant in 1991. Similar to that around the natural tooth, the biological width around dental implant differs in many aspects, such as concept, formation, remodeling, dimension, structure and function. The biological width in oral implantation seems to be 3-4 mm wide distance from the top of the peri-implant mucosa to the first bone-to-implant contact.

The remodeling of biologic width in soft tissue thickness affects the initial bone reconstitution. The margin level of soft tissue mainly depends on the thickness of peri-implant soft tissue, namely biological width rather than the bone width. A thin biotype is more susceptible to biologic width with less peri-implant mucosal dimensions than a thick biotype resulting in marginal tissue recession and initial crestal bone loss to form effective biologic width. The presence of KM has a positive effect on peri-implant tissue health to maintain soft tissue marginal position and accelerate the formation of soft tissue seal.

RELATION OF SOFT TISSUE BIOTYPE OR PERIODONTAL BIOTYPE WITH DENTAL IMPLANTS

According to **Ochsenbien and Ross**[6] in 1969**,** gingival biotypes are of two types. They are scalloped and thin or flat and thick gingiva. They proposed that the contour of the gingiva closely followed the contour of the underlying bone. Later **Seibert and Lindhe**[7] categorized the gingiva into “thick-flat” and “thin-scalloped” biotypes. Thick gingival tissue is associated with a broad zone of the keratinized tissue and flat gingival contour suggestive of thick bony architecture and also is more resistant to inflammation and trauma. Thin gingival tissue is associated with a thin band of the keratinized tissue, scalloped gingival contour suggestive of thin bony architecture and is more sensitive to inflammation and trauma. Inflammation of the periodontium results in increased pocket formation and gingival recession in thick and thin tissues respectively.

According to **Claffey and Shanley**[8] in 1986, they defined gingival thickness of >2 mm was considered as thick tissue biotype and a gingival thickness of <1.5 mm was referred as thin tissue biotype. They reported 1.8 mm of marginal mucosal recession in thin-biotype sites, compared to 0.6 mm in thick-biotype sites, when implants were placed slightly buccally.

The thicker biotype showed a lesser reduction in thickness from placement to restoration and higher rebound from restoration to the 1-year follow-up, as compared to the thin biotype cases. This is due to the higher amount of the CT and vascularity in thicker as compared to the thin biotype tissues, and thus the ability of these tissues to re-organize is better than thinner tissues. The effect of tissue thickness change on the marginal bone levels as the stability of the marginal bone levels has been used as a benchmark for implant success.

SIGNIFICANCE OF KERATINIZED MUCOSA AROUND IMPLANTS

Reduction in the crestal bone height reduces the distance between the mucogingival junction (MGJ) and the bone crest and hence, reduces the dimensions of the KM. KM consists of dense, collagen-rich connective tissue lined by a keratinizing epithelium and the lamina propria is firmly attached to the periosteum of the bone. The lining mucosa on the other hand, is comparatively collagen-poor, covered by a nonkeratinized epithelium, and is attached to muscles/periosteum of the underlying bone by collagen and elastic fibers. If implants are surrounded by a sufficient width of attached/keratinized mucosa, the long-term prognosis of these implants is improved. Clinical parameters of inflammation were higher for those implants with a narrow zone (2 mm) is associated with less mucosal recession and periodontal attachment loss as compared with a narrow. [9]

Soft tissue remodeling occurs after implant placement. An apical displacement of the facial mucosa by 0.6 mm occurs within the first 6 months, with relatively little change thereafter. Thus, in situations, which require an appropriate dimension of KM soft tissue augmentation should be contemplated.[9]

INFLUENCE OF MUCOSAL THICKNESS ON SOFT TISSUE INTEGRATION

A minimum of 3 mm of peri-implant mucosa is required for a stable epithelial connective tissue attachment to form. If a minimal dimension of gingival tissue is not available, bone loss may occur to ensure the proper development of the biologic width. The transition of the alveolar mucosa to the peri-implant mucosa is a difficult and a complex process.[10]

Linkevicious et al[11] in a human study found that positioning an implant 2 mm supracrestally did not prevent crestal bone loss if thin gingival tissues were present at the time of implant placement. Implants with thin tissue underwent additional bone loss interproximally versus the group with thick tissue pattern, which had significantly less bone loss. The conclusive findings of this study were that if initial tissue thickness was less (2.5 mm), marginal bone recession can be avoided. If the implant abutment junction is 2 mm or above the bone level, a negligible amount of bone loss (around 0.2 mm) would occur.

Augmentation of thin mucosa should be considered before implant placement. The maturity of the soft tissue sealing around the implants promotes increase in papillary height within 2 years without any manipulation of the soft tissue. Loss of implant papilla is one of the most troubling dilemmas in implant dentistry. The "black triangle" around the implant-supported restoration causes not only phonetic difficulties and food impaction but also unpleasant aesthetics. Factors that may affect the appearance of the peri-implant papilla are crestal bone height, interproximal distance, tooth form/shape, gingival thickness, and keratinized gingival width.[12]

SOFT TISSUE AUGMENTATION AROUND IMPLANT

Soft tissue augmentation procedures are frequently performed in partially and fully edentulous patients prior to, simultaneously with, and post-implant placement. These interventions are proposed to optimise aesthetic, functional and biological outcomes of implant therapy on the short- and long run.[13]
The biology of the peri-implant soft tissues warrants a careful manipulation of the soft tissues (flap design, reflection and suturing). Various modifications of the incisions/sutures during the implant placement and at the time of abutment placement conserve the soft tissue, enable stability of the flaps, and create papilla by coronal traction and thus, favor the soft tissue dimension.[14]

Modified incisions

- Modified Palacci technique (Grossberg 2001).
- T-shaped incision (Jemt T 1997).
- U-shaped incision (Nemcovsky 2000).
- Split-finger technique (Misch 2004).
- Beveled pericrestal incision (Sclar AG (2003).
- Curvilinear incisions (Miller 1988).
- Ramp sutures (Tinti and Benfenati 2002).
- Use of tissue punch.

Flap designs

- Rolled labial pedicle flap (Abrams 1980)
- Modified palatal roll technique (Scharf and Tarnow 1992)
- Buccal "envelope" technique for sliding a connective tissue graft on the labial aspect of the implant (Langer and Calagna 1982).
- The palatal or lingual based U-shaped peninsula flap (Miller PD Jr 1988).
- Papilla preserving modified roll technique (Dhir S 2014).

SURGICAL APPROACHES FOR SOFT TISSUE MANAGEMENT

Surgical approaches to augment the width of deficient mucosa are usually performed prior to implant placement or when undesired exposure of submerged implants occurs.[15]
1. Apically positioned flaps (APFs) — using a midcrestal/ lingual positioned incision.
2. APF/Vestibuloplasty (APF/V) in combination with autogenous tissue [subepithelial connective tissue graft (SCTG)/free gingival graft (FGG)].
3. FGG.
4. Epithelialized palatal graft technique.
5. SCTG technique or a soft tissue substitute [Acellular dermal matrix graft (ADMG)/collagen matrix (CM)].
6. Vascularized interpositional periosteal connective tissue flap vascularized interpositional periosteal connective tissue (VIP-CT flap).

CONCLUSIONS

An implant needs to achieve more than osseointegration for a successful outcome. Esposito et al. (2007) report that there is no sufficient evidence to recommend increase in KT or to indicate specific techniques for peri-implant soft tissue management. Although scientific evidence in most part is lacking, soft tissue augmentation at implant sites may need to be considered in some clinical situations. Innovative flap designs can be used to achieve better surgical and aesthetic results. Soft tissue augmentation either at implant placement, or at the second stage or after the implant placement as rescue operation can be predictably achieved with APF, APF/V, FGG, SCTG, and their modifications.

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