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Is Insufficiently Keratinized Mucosa a Risk Factor for Periimplantitis? A Literature Review

Yeterince Keratinize Olmayan Mukoza Periimplantit İçin Bir Risk Faktörü mü? Literatür Taraması

Özge TEZEL[1]
[1]Erzurum Oral and Teeth Health Center, Erzurum, Türkiye

Sevilay YEĞİNOĞLU[2]
[2]Karabük Oral and Teeth Health Center, Karabük, Türkiye.

Adnan TEZEL[3]
[3]Ankara University, Faculty of Dentistry, Department of Periodontology, Ankara, Türkiye.

ABSTRACT

In this study, a literature review was conducted including human studies assessing keratinized presence and extent in PubMed and Scopus whether insufficiently keratinized mucosa s risk factor for periimplantitis or not. The end of this review, we concluded that . The KM is important for masticatory stresses in the tooth margin or crown, and results in esthetics that are more favorable, overall comfort, and simplified brushing. Therefore, the presence of KM in the surrounding dental implant (DI) space is important for the prevention of inflammation, plaque buildup, gingival recession, and occurring periimplantitis.

Keywords : Keratinized mucosa, Periimplantitis, Dental implants

ÖZ

Bu çalışmada, PubMed ve Scopus'ta keratinize varlığı ve yaygınlığını değerlendiren, yetersiz keratinize mukozanın periimplantitis için risk faktörü olup olmadığını değerlendiren insan çalışmalarını içeren bir literatür taraması yapılmıştır. Bu incelemenin sonunda şu sonuca vardık. KM, diş kenarındaki veya krondaki çiğneme stresleri için önemlidir ve daha olumlu estetik, genel konfor ve daha basit fırçalamayla sonuçlanır. Bu nedenle, çevredeki diş implantı (DI) boşluğunda KM'nin varlığı, iltihaplanmanın, plak oluşumunun, diş eti çekilmesinin ve ortaya çıkan periimplantitin önlenmesi için önemlidir..

Anahtar Kelimeler: Keratinize mukoza, Periimplantitis, Diş implantları

Periodontal Tissue

The periodontium involves specialized tissues that support and surround the teeth. These tissues maintain the teeth within the mandibular and maxillary bones. Periodontium is derived from Greek word ‘peri-’ and ‘odont-‘, which translate to "around the tooth", respectively.[1] The dental speciality that focuses on care and maintenance of dental tissue is periodontics. The periodontium provides the teeth with the gum support to facilitate regular function. The four components included within the periodontium are: gingiva, periodontal ligament (PDL), cementum, and the alveolar bone proper.

The periodontium supports the teeth while they are being used. It relies on the stimulation received in order to preserve its own structure. As a result, there is a continuous state of balance, which exists between the periodontal structures and their external forces. The gingiva or gum tissue is located under the tooth and guards the core of the tooth or bone.[2] It surrounds to the tooth and forms a tight junction that provides a germ and infection barrier. The outer and inner connective tissue make up an epithial layer that is keratinized. The inner layer are gingival fibroblasts and these cells are critical for wound healing and tissue repair. The gingiva is the first line of defense and visually demonstrates the inflammatory response by swollen, red and bleeding tissues. Even though these characteristics do not mean periodontal complications, it is recommended to have these symptoms checked to rule out any possible issues.[3-5]

In general, gums may differ in color an range from pink to red pigmented, and visually the gingivia are stippled. Gingiva vary based location and functionality and include two types: the attached and free gingiva. The attached gingiva is keratinized, adheres to tooth/bone, and varies in height from 3 to 12 millimeters. Free gingiva is adjacent to the attached gums, forms a collar at the epithelial base of the tooth (sulcus), and is unattached at a depth of 1-3 mm.[3]

The necessity of keratinized tissue around the teeth to maintain periodontal health has been debated for years.[5] Some studies suggests that keratinized tissue is needed for the preservation of periodontal

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Sorumlu Yazar/Corresponding author: Özge Tezel
E-mail: ozge.tezel@hotmail.com

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tissues.[4] It has been believed that approximately two millimeters of keratinized gum and ~ 1 millimeter of adherent tissue are required to ensure the stability of the periodontium.[5] Conversely, multiple reports have questioned this phenomenon and suggest that the maintenance of gingival health can happen without adherent keratinized mucosa.[6,7]

1) Peri-implant Tissue

Dental implants (DIs) are artificial roots produced to replace teeth. DIs offer durable infrastructure permanent or removable dentures made in accordance with your natural teeth.

Advantages of DIs:

- Better aesthetic appearance. DIs are indistinguishable from real teeth. DIs are permanent because they are attached to the bone.
- Speech: Improperly secured dentures may lead to slipping of the teeth resulting in difficulty in communication. Permanent DIs are an option to circumvent the issue of teeth slipping.
- Comfort. Since the implants form a whole with you, you do not feel the discomfort of removable prostheses.
- Simplify eating. Removable dentures may cause chewing difficulties and permanent DIs improve eating and chewing food with ease.
- Improved self-esteem. DIs enhance your confidence and improve your smile.
- Ease of Use. DIs reduce complications and the inconvenience of having to remove and clean dentures, as well as the necessity for cements.

Peri-implant tissue surrounds DIs and are classified into soft and hard tissue groups. The soft group, or peri-implant mucosa/tissue, is generated through the wound healing process. The healing process occurs following implantation. The soft tissue interface is important due to its capacity to form a biological seal around the implant, which protects against infection or foreign material.

The peri-implant mucosa is made up of keratinized oral, sulcular, and junctional epithelium that includes a connective tissue base layer. Hemidesmosomes and basal lamina are located within the implant and epithelium. This is also known as the "biological width" (1 mm + 0.97 mm). It consists of the supra-alveolar connective tissue and connecting epithelium. Clinically, there should be no progression within two millimeters of the tooth. A comparable association of bone to the soft tissues occurs about the DIs and/or teeth. Relapses in this association may contribute to early crest loss of bone.[8] The comparisons between the tooth/soft tissue interface and the peri-implant is shown in Table 1.

Table 1. Comparison between tissue around teeth and the peri-implant

Features	Peri-Implant Tissues	Tissues around Teeth
Gingival fibers	No implant insertion: fibers are parallel or circumferential to the long axis of the implant	Complex array of fibers inserting into the cementum about the crestal bone and onto the periosteum
Junctional epithelium	Hemidesmosome attachment to titanium	Hemidesmosome attachment to enamel
Connective tissue attachment	Structure rich in collagen with no fibroblasts and vascularity Variable and dependent on the implant depth positioning	organized collagen bundles perpendicular to the root cementum average 1-9 mm
Gingival sulcus depth	Variable and dependent on the implant depth positioning and abutment length and restoration margin	Shallow on around 2–3 mm
Blood supply	Less blood vessels and supply come from the underlying periosteum	Numerous vascular anastomoses between the vessels from the PDL space, and gingival connective tissue
Biological width	JE = 1.88 mm and CT = 1.05 mm	Junctional epithelium – 0.97 mm; CTA – 1.07 mm

DIs do not have root cementum, PDL, or bone like natural teeth.[9] Dental alveolar and gingival fibers link the gums to the tooth. These fibers are noticeable in the peri-implant. In healthy areas, the gingival boundary follows the contours of the cement-enamel junction, while the mucosal margin around an implant tracks the crest bone for multiple DIs or adheres to the proximal teeth tissue of single DIs. While the implant is rigidly fixed to the surrounding host bone, it is mobile within the tooth socket at the physiological limit. There has become an increased demand for DIs and good clinical practice has become a mainstay. Peri-implant health is fundamental for DI survival.

2) Keratinization and Its Clinical Significance

The epithelial layer of the attached gingiva is hard, resistant to trauma, and firmly attached to the underlying connective tissue. This durability is due to the formation of keratin in the upper layer of the epithelium (also called keratinized mucosa (KM)). Such differentiation is called keratinization.[10]

KM is the chewing mucosa found around dental implants. KM encompasses the peri-implant mucosal edge and the mobile mucogingival mucosa. KM is covered by an orthokeratinized squamous epithelium and consists of lamina propria (fibroblasts and fibrous connective tissue containing type I/III collagen).[11-13] Crest bone loss after tooth extraction has been suggested to cause a reduction in KM. The KM thickness in the facial region more prominent in DIs compared to the teeth (2.0 mm vs 1.1 mm, respectively).[12] A minimal amount of KM for maintenance of peri-odontal and -implant health around teeth and implants is controversial.[14-17] A few studies failed to link KM deficiency with inflammation of the mucosa,[18-22] while others have shown plaque deposition and marginal inflammation are more common at implant sites that have less than two millimeters KM.[23-27]

Lang and Löe [4] reported a relationship between keratin-attached gingival width and periodontal health. It is known that a minimum of 2 mm keratinized tissue and 1 mm of attached gingiva are considered healthy in more than 80% of the surfaces. Clinical inflammation has been demonstrated in areas with less than one millimeter of keratinized and attached gingiva. Lang and Löe (1972) [4] and Berglundh et al.[28] showed that the mobile gingival margin could give rise to microorganism entry into cavity. These results showed that 2 mm of keratinized tissue and 1 mm of adherence was adequate for gingival health.

Esfahanizadeh et al.[29] demonstrated KM width was inversely associated with Marginal Bleeding Index (MBI), Marginal Plaque Index (MPI), Marginal Gingival Index (MGI) ($P < 0.05$). No relationship was determined for width KM and age, sex, Probing Deep, oral hygiene rinses, tooth brushings, or dental status ($P > 0.05$).

Miyasato and colleagues demonstrated [30] that gingival margin health may be maintained at a KM less than one millimeter. No differences were found in terms of clinical inflammation for patients with reduced or enhanced KM.[30-33]

Lindhe and colleagues [34] assessed the peri-implant and tooth tissue reactions following plaque buildup. They showed that there was a similarity between DIs and natural teeth.

There are authors who think that KM is important in maintaining the long-term health of the soft/hard tissues around the implant, as well as claims to the contrary.[35-41] Two millimeters of keratinized gingiva and 1 mm of attached gingiva is sufficient to preserve healthy gums in natural teeth.[36,37]

Various studies have reported that patients with “inadequate” amounts of KM (< 2 mm) may have pain and other complications during daily oral hygiene procedures at DI sites. It has also been shown that more gingival recession may be linked to increased plaque, bleeding during probing, and bone loss.[22,23,32,37] Therefore, mucogingival surgeries with free gingival grafts have been suggested to increase the narrow band of keratinized tissue.

Conversely, some reports have shown a reduction in KM surrounding the DI does not adversely disturb the health or stability of tissues around the implant in those with good oral hygiene.[37,42,43] Evaluations of the need for KM around implants show that health maintenance and tissue stability is lacking[15,17] while others state that KM is positive in preventing inflammation.[12,37,44,45]

When the previous literature is evaluated, the frequency of DIs surrounded with KM at less than 2 mm varies between 23.8% and 74% [37,46] Many factors influence the attached gingiva width that surround natural teeth, including tooth location, inflammation or other reasons, including tooth position, high frenulum and muscle attachments.[37,47]

KM consists of the area from the gingival margin to the mucogingival line. It has been suggested that the width of the KM is greater than or equal to 2 mm and the attached gingiva ≥1 mm is sufficient for gingival health.[6,17] It has been reported that the adjacent mucosa to DIs consists of a 2 mm high (long) marginal connecting epithelial layer and a more apical connective tissue region about 1.5 mm high (long).[17]

Mammalian studies have shown similarities between soft tissue responses to plaque in the surrounding teeth and DIs.[25] Up to now, the impact of KM on peri-implant health has been consistently discussed.[21,25,46] In a clinical study, the effect of the presence of KM on peri-implant soft tissue health could not be proven. The effect of KM on plaque deposition has not been clarified.

Kungsadalpipob[7] is used with implants without keratinized mucosa, showed three times more plaque buildup in comparison to DIs containing KM. This result is in line other reports that have shown higher plaque scores at implant sites that do not contain keratinized mucosa.[6,7,23,32]

Reduced KM may contribute to an environment of poor hygiene and augmented predisposition to mechanical irritation/discomfort during daily oral hygiene procedures.[7] The lack of KM has been linked to mucosal recession. DIs lacking KM were enhanced threefold to mucosal recession (≥ 1 mm) in comparison to DI sites with KM.

Zigdon and Machtei[22] demonstrated an enhanced decline and reduced pocket formation in areas with less KM. The absence of KM may facilitate inflammatory components to apically migrate. Nevertheless, mucosal recession and its development at DI sites following restoration are strongly debated. Improper DI placement, KM deficiency, thin tissue or buccal bone, and loss of alveolar bone height need consideration for their relationship to mucosal recession in DIs.[7]

3) The Role of Quantity and Characteristics

Strub et al.[48] have been considered while the debate on the amount of keratinized implants were placed in an area without KM. A higher rate of plaque accumulation and peri-implantitis is expected compared to implants placed in an area with keratinized mucosa.[49]

Wenström et al.[21] hypothesized that a reduction in MK zones hinder correct oral hygiene and is insufficient to protect against distress while brushing or chewing, as well as bacterial plaque load.[20]

Provided adequate plaque control is achieved, it is compatible with peri-implant soft tissue health even in the absence of marginal keratinized tissue.[17,18,20]

Warrer et al.[38] on the other hand reported that a higher rate of attachment loss and gingival recession was observed for dental implants with insufficient keratinized mucosa as a result of ligature-induced plaque accumulation for 9 months around 30 implants placed. In this study, they suggested that “ligature-induced plaque accumulation” may also be associated with patients with “insufficient oral hygiene”.

Bouri et al.[31] in their study of 200 dental implants in 76 patients found that the amount of bleeding, plaque and gingival index, and alveolar bone loss during probing was higher for implants with insufficient keratinized mucosa (less than 2 mm) when compared to Dis with sufficient KM. It was reported that it was high and suggested that KM is necessary for the preservation of tissue stability.

Zigdon and Machtei[22] reported that KM surrounding the DIs can be critical in the early diagnosis of mucosal recession. These investigators suggested that patients with less than two millimeters of KM had higher plaque deposition associated with peri-implantitis over a 3-year period.

The peri-implant, which takes care of the important points between the soft tissue connection of DIs and teeth, if the peri-implant is keratinized content or is the most beneficial, and the same 2 mm threshold for soft tissue health and natural teeth is also valid for dental implants.[20]

Kim et al.[19] followed 276 dental implants for 13 months. No difference was observed in terms of gingival index, plaque index, and pocket depth for parts with insufficient amount of keratinized thickness, while marginal bone loss and gingival rates were higher than those with insufficient keratinized coating. Observation of birth reveals the necessity of a keratinized coating for the successful maintenance of dental implants.

Schrott et al.[20] evaluated a total of 307 implants in their studies in which the keratinized layer canal was examined separately in both lingual and buccal tissues for peri-implant tissues. In the results of the study, there was no component to evaluate the amount of keratinized coating in the buccal region, plaque accumulation and bleeding on probing. Conversely, cells with a higher rate of gingival emission were observed. In the lingual class, higher values were obtained than the plaque index and gingival index values in areas where the keratinized coating was insufficient. These values were discussed with the difficulties of oral hygiene practices in the lingual region.

Dental implants have a wide range of indications. It is very important to follow the desired successful results with implant treatment. Due to the popularity of implant-supported prostheses and the consumption of photographs, there are many studies in the literature. However, while there are various examinations evaluating the satisfaction levels of patients after implant treatment [13], the number of studies revealed by the amount of keratinized gingiva is quite vast in the literature.

Whether adequate amount of KM in dental implants is necessary for peri-implant health is debatable. In the literature, sufficient KM for peri-implant health has been documented to be ≥ 2 mm. When KM is < 2 mm, it may increase peri-implant health and may cause peri-mucositis/peri-implantitis.[12]

Studies have shown that peri-mucositis development is more common with a band of keratinized tissue less than 2 mm.[20,32,45] Periodontal destructions can occur when the structures are inadequate in the size of the keratinized tissue band and when the plaque is made appropriately.[7,10]

CONCLUSIONS

This study is not a meta-analysis study but a systematic review. The search process resulted in identifying 65 potential articles. The articles scanned were completed using different groups of different materialss and methods made between the years of 1972-2023. Since all articles have deficiencies and advantages over each other, future standardized studies are needed to say that keratinized gingiva is a definite risk factor for periimplantitis. With this the consensus report of the 2017 World Workshop on the Classification of Periodontal and Peri-implant Diseases and Conditions recommended that the role of KM on long-term peri-implant tissue health is ambiguous. Despite this, KM may be necessary for overall oral comfort and to facilitate the removal of plaque. KM as a barrier fight against inflammation and gingival recession. The KM is important for masticatory stresses in the tooth margin or crown, and results in esthetics that are more favorable, overall comfort, and

simplified brushing. Therefore, the presence of KM in the surrounding DI space is important for the prevention of inflammation, plaque buildup, and gingival recession. Clinicians should be aware of this factor when placing dental implants at a particular site.

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REFERENCES

1. Al-Ghutaimel H, Riba H, Al-Kahtani S, Al-Duhaimi S. Common periodontal diseases of children and adolescents. *Int J Dent*. 2014;2014
2. Nebel D. Functional importance of estrogen receptors in the periodontium. 2012;
3. Hirschfeld J, Higham J, Blair F, Richards A, Chapple IL. Systemic disease or periodontal disease? Distinguishing causes of gingival inflammation: A guide for dental practitioners. Part 2: Cancer related, infective, and other causes of gingival pathology. *Br Dent J*. 2019;227(12):1029-1034.
4. Kissa J, El Kholti W, Laalou Y, El Farouki M. Augmentation of keratinized gingiva around dental implants. *J Stomatol Oral Maxillofac Surg*. 2017;118(3):156-160.
5. Lang NP, Löe H. The relationship between the width of keratinized gingiva and gingival health. *J Periodontol*. 1972;43(10):623-627.
6. Kennedy JE, Bird WC, Palcanis KG, Dorfman HS. A longitudinal evaluation of varying widths of attached gingiva. *J Clinical Periodontol*. 1985;12(8):667-675.
7. Kungsadalpipob K, Supanimitkul K, Manopattanasoontorn S, Sophon N, Tangsathian T, Arunyanak SP. The lack of keratinized mucosa is associated with poor peri-implant tissue health: a cross-sectional study. *International Journal Of Implant Dentistry*. 2020;6:1-9.
8. Oh TJ, Yoon J, Misch CE, Wang HL. The causes of early implant bone loss: myth or science? *Journal Of Periodontology*. 2002;73(3):322-333.
9. Schroeder HE. *The periodontium*. vol 5. Springer Science & Business Media; 2012.
10. Karagülle L. Yapışık keratinize dişeti genişliği ve dişeti sağlığı ile ilişkisi. İstanbul Üniversitesi Diş Hekimliği Fakültesi, Uzmanlık Tezi, İstanbul: 2019.
11. Chang M, Wennström JL. Soft tissue topography and dimensions lateral to single implant-supported restorations. A cross-sectional study. *Clin Oral Imp Res*. 2013;24(5):556-562.
12. Chang M, Wennström JL, Ödman P, Andersson B. Implant supported single-tooth replacements compared to contralateral natural teeth. Crown and soft tissue dimensions. *Clin Oral Imp Res*. 1999;10(3):185-194.
13. Parpaiola A, Cecchinato D, Toia M, Bressan E, Speroni S, Lindhe J. Dimensions of the healthy gingiva and peri-implant mucosa. *Clin Oral Imp Res*. 2015;26(6):657-662.
14. Brito C, Tenenbaum HC, Wong BK, Schmitt C, Nogueira-Filho G. Is keratinized mucosa indispensable to maintain peri-implant health? A systematic review of the literature. *Journal of Biomedical Materials Research Part B: Applied Biomaterials*. 2014;102(3):643-650.
15. Gobbato L, Avila-Ortiz G, Sohrabi K, Wang C-W, Karimbux N. The effect of keratinized mucosa width on peri-implant health: a systematic review. *International Journal of Oral & Maxillofacial Implants*. 2013;28(6)
16. Lin GH, Chan HL, Wang HL. The significance of keratinized mucosa on implant health: a systematic review. *J Periodontol*. 2013;84(12):1755-1767.
17. Wennström JL, Derks J. Is there a need for keratinized mucosa around implants to maintain health and tissue stability? *Clinical oral implants research*. 2012;23:136-146.
18. Heckmann SM, Karl M, Wichmann MG, Winter W, Graef F, Taylor TD. Cement fixation and screw retention: parameters of passive fit: An in vitro study of three-unit implant-supported fixed partial dentures. *Clin Oral Imp Res*. 2004;15(4):466-473.
19. Kim B-S, Kim Y-K, Yun P-Y, et al. Evaluation of peri-implant tissue response according to the presence of keratinized mucosa. *Oral Surg Oral Med Oral Pathol, Oral Radiol Endodont*. 2009;107(3):e24-e28.
20. Schrott AR, Jimenez M, Hwang JW, Fiorellini J, Weber HP. Five-year evaluation of the influence of keratinized mucosa on peri-implant soft-tissue health and stability around implants supporting full-arch mandibular fixed prostheses. *Clinical Oral Implants Res*. 2009;20(10):1170-1177.
21. Wennström J, Bengazi F, Lekholm U. The influence of the masticatory mucosa on the peri-implant soft tissue condition. *Clin Oral Implants Res*. 1994;5(1):1-8.
22. Zigdon H, Machtei EE. The dimensions of keratinized mucosa around implants affect clinical and immunological parameters. *Clin Oral Implants Res*. 2008;19(4):387-392.
23. Boynueğri D, Nemli SK, Kasko YA. Significance of keratinized mucosa around dental implants: a prospective comparative study. *Clin Oral Implants Res*. 2013;24(8):928-933.
24. Kikuchi T, Wada M, Mameno T, Hasegawa D, Serino G, Ikebe K. Longitudinal study on the effect of keratinized mucosal augmentation surrounding dental implants in preventing peri-implant bone loss. *Peer J*. 2022;10:e13598.
25. Mahardawi B, Jiaranuchart S, Damrongsirirat N, et al. The lack of keratinized mucosa as a risk factor for peri-implantitis: A systematic review and meta-analysis. *Scientific Reports*. 2023;13(1):3778.
26. Mameno T, Wada M, Otsuki M, et al. Risk indicators for marginal bone resorption around implants in function for at least 4 years: A retrospective longitudinal study. *J Periodontol*. 2020;91(1):37-45.
27. Sohn J-Y, Park J-C, Cho K-S, Kim C-S. Simultaneous placement of an interpositional free gingival graft with nonsubmerged implant placement. *J Periodont & Implant Sci*. 2014;44(2):94.
28. Berglundh T, Lindhe J, Ericsson I, Marinello C, Liljenberg B, Thornsen P. The soft tissue barrier at implants and teeth. *Clin Oral Implants Res*. 1991;2(2):81-90.
29. Esfahanizadeh N, Daneshparvar N, Motallebi S, Akhondi N, Askarpour F, Davaie S. Do we need keratinized mucosa for a healthy peri-implant soft tissue. *Gen Dent*. 2016;64(4):51-5.
30. Miyasato M, Crigger M, Egelberg J. Gingival condition in areas of minimal and appreciable width of keratinized gingival. *J Clin Periodontol*. 1977;4(3):200-209.

31. Bouri Jr A, Bissada N, Al-Zahrani MS, Faddoul F, Nouneh I. Width of keratinized gingiva and the health status of the supporting tissues around dental implants. *Int J Oral & Maxillofac Imp*. 2008;23(2)
32. Chung DM, Oh TJ, Shotwell JL, Misch CE, Wang HL. Significance of keratinized mucosa in maintenance of dental implants with different surfaces. *J Periodontol*. 2006;77(8):1410-1420.
33. Greenstein G, Cavallaro J. The clinical significance of keratinized gingiva around dental implants. *Compend Contin Educ Dent*. 2011;32(8):24-31.
34. Lindhe J, Berglundh T, Ericsson I, Liljenberg B, Marinello C. Experimental breakdown of peri-implant and periodontal tissues. A study in the beagle dog. *Clin Oral Imp Res*. 1992;3(1):9-16.
35. Freedman AL, Green K, Salkin LM, Stein MD, Mellado JR. An 18-year longitudinal study of untreated mucogingival defects. *J Periodont*. 1999;70(10):1174-1176.
36. Stetler KJ, Bissada NF. Significance of the width of keratinized gingiva on the periodontal status of teeth with submarginal restorations. *J Periodonthol*. 1987;58(10):696-700.
37. Wang Q, Tang Z, Han J, Meng H. The width of keratinized mucosa around dental implants and its influencing factors. *Clin Implant Dent Related Res*. 2020;22(3):359-365.
38. Warrer K, Buser D, Lang N, Karring T. Plaque-induced peri-implantitis in the presence or absence of keratinized mucosa. An experimental study in monkeys. *Clin Oral Imp Res*. 1995;6(3):131-138.
39. Wennström J, Lindhe J. Plaque-induced gingival inflammation in the absence of attached gingiva in dogs. *J Clin Periodontol*. 1983;10(3):266-276.
40. Wennström J, Lindhe J, Nyman S. Role of keratinized gingiva for gingival health: clinical and histologic study of normal and regenerated gingival tissue in dogs. *J Clin Periodontol*. 1981;8(4):311-328.
41. Wennström JL. Lack of association between width of attached gingiva and development of soft tissue recession: A 5-year longitudinal study. *J Clin Periodontol*. 1987;14(3):181-184.
42. Krekeler G, Kappert H, Schilli W. Scanning electron microscopic study of the reaction of human bone to a titanium implant. *Int J Oral Surg*. 1985;14(5):447-450.
43. Merickske-Stern R. Clinical evaluation of overdenture restorations supported by osseointegrated titanium implants: a retrospective study. *Int J Oral & Maxillofac Imp*. 1990;5(4)
44. Mehta P, Lim LP. The width of the attached gingiva—Much ado about nothing? *J Dent*. 2010;38(7):517-525.
45. Pranskunas M, Poskevicius L, Juodzbalys G, Kubilius R, Jimbo R. Influence of peri-implant soft tissue condition and plaque accumulation on peri-implantitis: a systematic review. *J oral & Maxillofac Res*. 2016;7(3).
46. Mericske-Stern R, Steinlin Schaffner T, Marti P, Geering A. Peri-implant mucosal aspects of ITI implants supporting overdentures. A five-year longitudinal study. *Clin Oral Imp Res*. 1994;5(1):9-18.
47. Bowers GM. A study of the width of attached gingiva. *J Periodontol*. 1963;34(3):201-209.
48. Strub J, Gaverthüel T, Grunder U. The Role of Attached Gingiva in the Health of Peri-Implant Tissue in Dogs. Part I. Clinical Findings. *International J Periodont & Rest Dent*. 1991;11(4)
49. Costa FO, Takenaka-Martinez S, Cota LOM, Ferreira SD, Silva GLM, Costa JE. Peri-implant disease in subjects with and without preventive maintenance: a 5-year follow-up. *J Clin Periodontol*. 2012;39(2):173-181.