Width of Keratinized Gingiva and the Health Status of the Supporting Tissues Around Dental Implants

Anil Bouri, Jr, DDS, MSD[1]/Nabil Bissada, DDS, MSD[2]/Mohammad S. Al-Zahrani, MSD, PhD[3]/
Fady Faddoul, DDS, MSD[4]/Imad Nouneh, DDS, MSD[5]

Purpose: *This cross-sectional study was performed to determine whether an association exists between the width of keratinized mucosa and the health of implant-supporting tissues.* ***Materials and Methods:*** *Data on 200 dental implants were collected. Periodontal parameters measured included Plaque Index, Gingival Index, width of keratinized mucosa, thickness of keratinized mucosa, radiographic bone level, and bleeding on probing. Statistical analysis was accomplished with the* t *test, Wilcoxon rank sum test, and logistic and linear regression models. Significance was established when* P *was less than .05.* ***Results:*** *The mean Gingival Index score, Plaque Index score, and radiographic bone loss were significantly higher for those implants with a narrow zone (< 2 mm) of keratinized mucosa. Implants with a narrow zone of keratinized mucosa also were more likely to bleed upon probing, even after adjusting for Plaque Index, smoking, thickness of the gingiva, and time since implant placement (adjusted odds ratio, 2.37; 95% confidence interval, 1.04 to 5.83). Significant independent association also was found between the width of keratinized mucosa and radiographic bone loss in favor of wider zone of keratinized mucosa.* ***Conclusion:*** *Increased width of keratinized mucosa around implants is associated with lower mean alveolar bone loss and improved indices of soft tissue health.*
(Cross-sectional Study) Int J Oral Maxillofac Implants 2008;23:323–326

Key words: *dental implants, keratinized gingiva, peri-implant tissues*

The presence of an adequate zone of keratinized mucosa was thought to be necessary for the maintenance of gingival health and prevention of periodontal disease progression. Lang and Loe suggested a width of at least 2 mm of keratinized mucosa, of which 1 mm was to be attached.[1] Subsequently, several authors have challenged this concept and have shown that gingival health can be maintained with almost no attached gingiva.[2–6] In teeth with subgingival restorations, however, it has been reported that a narrow zone of keratinized mucosa is associated with a higher chance of gingival inflammation.[7]

Peri-implant and periodontal tissues may differ in their resistance to bacterial infection.[8–10] Thus, the necessity of a zone of keratinized tissue adjacent to dental implants has been suggested.[11] This is especially important because the implant-supported restoration is located beneath the oral mucosa. Furthermore, the implant-mucosa interface differs from the interface between the mucosa and natural teeth, and these differences are important to the understanding of the susceptibility of implants to infection.[12–16] Supracrestal collagen fibers are oriented in a parallel rather than a perpendicular configuration. This creates a much weaker mechanical attachment compared to natural teeth.[12–16] In addition, Lindhe and Berglundh[17] suggested that the ability of the peri-implant mucosa to regenerate itself is limited by its compromised number of cells and poor vascularity.

Few studies have examined the relationship between the width of keratinized mucosa and the health of peri-implant tissues.[11,18–20] The results of these studies are contradictory. Further studies are therefore required to clarify the role of the width of the keratinized mucosa around dental implants and their overall soft and hard tissue health. The purpose of the present cross-sectional study was to deter-

[1]Private Practice, Chicago, Illinois.
[2]Professor and Chair, Department of Periodontics, School of Dental Medicine, Case Western Reserve University, Cleveland, Ohio.
[3]Assistant Professor, Division of Periodontics, Faculty of Dentistry, King Abdulaziz University, Jeddah, Saudi Arabia.
[4]Associate Professor, Department of Comprehensive Care, School of Dental Medicine, Case Western Reserve University, Cleveland, Ohio.
[5]Assistant Clinical Professor, Department of Periodontics, School of Dental Medicine, Case Western Reserve University, Cleveland, Ohio.

Correspondence to: Dr Nabil F. Bissada, Professor and Chair, Department of Periodontics, Case School of Dental Medicine, 10900 Euclid Avenue, Cleveland, OH 44106-4905. Fax: +216 368 3204. E-mail: nabil.bissada@case.edu

mine whether the width of the keratinized gingiva around dental implants has an effect on the health of the surrounding soft and hard tissues.

MATERIALS AND METHODS

Patients for this study were randomly selected from those who presented to the clinics of the Advanced Education for General Dentistry or Graduate Periodontics of the Case School of Dental Medicine for their biannual maintenance appointments between August 2005 and February 2006. Subjects were eligible for inclusion if they were 18 years of age or older and had an implant-supported restoration placed at the Case School of Dental Medicine that had been in place for a minimum of 12 months. Patients were included only if they provided the written consent to participate, if the dental implant and its restoration were still in place, and if their radiographic records and periodontal charting were complete at the time of implant placement. The following data were recorded for each implant by a single calibrated examiner: number and anatomic location of the implant, Plaque Index, Gingival Index, width of keratinized tissue, thickness of gingival tissue, probing pocket depth, mobility of implant, radiographic bone level, time since implant placement, and smoking history. The Plaque Index and Gingival Index for each implant were measured according to the modified indices defined by Mombelli et al.[21]

The width of the keratinized mucosa was measured at the mid-facial aspect of each implant to the nearest half-millimeter with a University of North Carolina (UNC) periodontal probe. Each measurement was made from the gingival margin to the mucogingival junction. The mucogingival junction was identified by the rolling technique, wherein the mucosa was rolled until the nonmovable portion of the attached keratinized tissue was seen. The thickness of the gingiva around the dental implants was measured approximately 2 mm apical to the gingival margin on the facial aspect of the implant. After topical application of anesthetic, the thickness was measured by gently inserting a 27-gauge needle with a rubber stopper in the tip until the underlying hard structure had been contacted, as described by Austria and Bissada.[22] The distance between the tip of the needle and the rubber stopper was measured to the nearest 0.5 millimeter using the UNC-graduated periodontal probe. Pocket probing depth was measured to the nearest millimeter using a 15-mm UNC-graduated plastic periodontal probe at the midfacial and interproximal surfaces of each implant. For statistical analysis, the periodontal probing depth measurements were averaged for a mean value.

Radiographic bone level was measured by comparing the periapical radiographs obtained using the paralleling technique at the time of implant placement and with those obtained at the maintenance visit. Bone level was measured from a fixed reference point on the implant to the crestal bone level. Time of implant placement was recorded in number of years. Smoking was recorded in number of packs per year. Occlusion was recorded to identify the implants' opposing surface: natural teeth, acrylic/porcelain removable prosthetic devices, or no opposing teeth.

Statistical Analysis

Data were analyzed using the *t* test and Wilcoxon rank sum test for normally and non-normally distributed data, respectively. Statistical significance was established when *P* was less than or equal to .05. Width of keratinized mucosa was dichotomized using a 2 mm as a cutoff point. Group A consisted of implants where the width of the keratinized mucosa was $\geq$ 2 mm, and group B comprised implants where the width of the keratinized mucosa was $<$ 2 mm.

Multivariable logistic and linear regression analyses, respectively, were used to examine whether the width of keratinized tissue is independently associated with bleeding on probing and mean alveolar bone loss. Smoking, time since implant placement, thickness of the gingiva, and Plaque Index were selected as explanatory variables.

RESULTS

This study included a total of 76 patients with 200 restored dental implants. Of the 200 implants, 110 implants were found to have $\geq$ 2 mm of keratinized tissue (group A), whereas 90 implants had a keratinized mucosa of $<$ 2 mm (group B). As shown in Table 1, the means for group B were significantly higher than group A for the following parameters: GI, PI, and RBL. Mean thickness of keratinized gingiva, however, was significantly higher for group A than group B. There was no statistical difference between the 2 groups with respect to periodontal probing depth or smoking. Implants in group B had been restored for a significantly longer period of time compared to those of group A (4.91 ± 2.76 years vs 4.10 ± 2.48 years; $P < .05$).

With regard to bleeding on probing, implants with a narrow zone of keratinized tissue had a significantly higher chance of bleeding than implants with a wider zone of keratinized mucosa (89% vs 71%; $P < .01$). In the univariate analysis, implants with a narrow zone of keratinized gingiva were 3 times more likely to have bleeding on probing than those with a wider zone of keratinized tissue. This difference remained

Table 1 Periodontal Parameters of Implants Placed in Varying Widths of Keratinized Gingiva

	Width of keratinized gingiva ≥ 2 mm		Width of keratinized gingiva < 2 mm		
	Mean	SD	Mean	SD	*P*
Gingival Index	0.91	0.72	1.50	0.77	< .001
Plaque Index	1.25	0.53	1.78	0.78	< .001
Thickness of keratinized gingiva	1.42	0.39	0.37	0.56	< .001
Periodontal probing depth	3.72	0.75	3.87	0.66	.132
Radiographic bone loss	1.24	0.69	1.72	1.18	< .001

Table 2 The Results of the Multivariable Linear Regression for the Association Between Width of Keratinized Mucosa and Alveolar Bone Loss*

	Unstandardized coefficients		
	B	Standard error	*P*
Constant	0.33	0.22	.138
Keratinized mucosa ≥ 2 mm	−0.57	0.18	.002
Time since implant placement	0.07	0.02	.002
Plaque Index	0.51	0.09	< .001
Thickness of the gingiva	0.39	0.13	.003
Smoking	−0.06	0.04	.101

*R^2 = 0.27 and adjusted R^2 = 0.25.

highly significant even after adjusting for time since implant placement, smoking, thickness of the gingiva, and plaque score (adjusted odds ratio, 2.37; 95% confidence interval, 1.04 to 5.83).

Implants with narrow zones of keratinized mucosa had significantly higher mean alveolar bone loss than implants with wider zones of keratinized mucosa. This association remained statistically significant even after adjusting for time since implant placement, smoking, thickness of the gingiva, and plaque score (Table 2).

DISCUSSION

The purpose of this cross-sectional study was to analyze the relationship between the width of keratinized mucosa and the health of implant-supporting tissues. A total of 200 implants from edentulous and partially edentulous patients were included in the study. Results 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). Implants with narrow zones of keratinized gingiva were found to be more prone to bleeding on probing even after taking into account time since implant placement, smoking, thickness of the gingiva, and Plaque Index. This observation supports the view that narrow zones of keratinized gingiva are less resistant to insult along the implant-mucosa interface. When inflammation is present, its apical proliferation may occur more rapidly compared to those sites with wider zones of keratinized gingiva that have an epithelial seal. Wider zones of keratinized gingiva may offer more resistance to the forces of mastication and frictional contact that occur during oral hygiene procedures. Thus, a lack of keratinized gingiva may create an environment that is less amenable to oral cleansing and more susceptible to irritation and discomfort during such routine procedures.[23] A recent study has shown an association between width of keratinized mucosa and gingival inflammation and plaque accumulation that is in line with the present findings.[20] The findings of the present study also are supported by Warrer et al,[11] who demonstrated that implants placed in areas lacking keratinized gingiva had a higher susceptibility to tissue breakdown due to plaque accumulation. Despite similar plaque levels, implants in nonkeratinized areas showed earlier loss of attachment.[20] In contrast, other investigators[18,19] reported no association between implant survival and width of keratinized tissue. For example, Wennstrom et al[19] found similar results regarding the contribution of the width of keratinized tissue to implant health.

With regard to the alveolar bone, the mean bone loss was higher for implants with narrow zones of keratinized mucosa. This relationship remained significant even after taking into account time since implant placement, smoking, thickness of the gingiva, and Plaque Index. Thus, width of keratinized mucosa appears to have an impact on the alveolar bone loss around implants. In contrast, other investigators reported no association between the width of keratinized mucosa and alveolar bone loss around dental implants.[20] In the present study, the independent association between the width of keratinized gingiva and the mean alveolar bone loss was examined using multivariable linear regression analysis. Chung et al,[20] however, calculated the annual bone loss per each subject by dividing alveolar bone loss by the time of follow-up. The latter method, however, assumes an equal rate of bone loss each year. Furthermore, no adjustments for other variables such as smoking or Plaque Index were employed by Chung et al.[20] It should be noted, however, that the present and previous studies were cross-sectional in nature. Thus, the width of keratinized tissue before implant placement and restoration is not available. Also, determination of the alveolar bone loss was made through comparison of nonstandardized radiographs at 2 different points in time. Yearly measurements of bone loss over time on standardized radiographs possibly would have allowed a calculation of rate of bone loss per year.

CONCLUSION

The findings of the present study suggest a relation between width of keratinized tissue and the health of the peri-implant tissues. Bleeding on probing as well as mean alveolar bone loss was higher for implants surrounded by less than 2 mm of keratinized mucosa than for those with a wider zone of keratinized mucosa.

REFERENCES

1. Lang NP, Loe H. The relationship between the width of keratinized gingiva and gingival health. J Periodontol 1972;43: 623–627.
2. Miyasato M, Crigger M, Egelberg J. Gingival condition in areas of minimal and appreciable width of keratinized gingiva. J Clin Periodontol 1977;4:200–209.
3. Hangorsky U, Bissada NF. Clinical assessment of free gingival graft effectiveness on the maintenance of periodontal health. J Periodontol 1980;51:274–278.
4. Dorfman HS, Kennedy JE, Bird WC. Longitudinal evaluation of free autogenous gingival grafts. A four year report. J Periodontol 1982;53:349–352.
5. Wennstrom J, Lindhe J. Role of attached gingiva for maintenance of periodontal health. Healing following excisional and grafting procedures in dogs. J Clin Periodontol 1983;10:206–221.
6. Kennedy JE, Bird WC, Palcanis KG, Dorfman HS. A longitudinal evaluation of varying widths of attached gingiva. J Clin Periodontol 1985;12:667–675.
7. Stetler KJ, Bissada NF. Significance of the width of keratinized gingiva on the periodontal status of teeth with submarginal restorations. J Periodontol 1987;58:696–700.
8. Ericsson I, Berglundh T, Marinello C, Liljenberg B, Lindhe J. Long-standing plaque and gingivitis at implants and teeth in the dog. Clin Oral Implants Res 1992;3:99–103.
9. 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 Implants Res 1992;3:9–16.
10. Berglundh T, Lindhe J, Marinello C, Ericsson I, Liljenberg B. Soft tissue reaction to de novo plaque formation on implants and teeth. An experimental study in the dog. Clin Oral Implants Res 1992;3:1–8.
11. Warrer K, Buser D, Lang NP, Karring T. Plaque-induced peri-implantitis in the presence or absence of keratinized mucosa. An experimental study in monkeys. Clin Oral Implants Res 1995;6:131–138.
12. Jansen JA, de Wijn JR, Wolters-Lutgerhorst JM, van Mullem PJ. Ultrastructural study of epithelial cell attachment to implant materials. J Dent Res 1985;64:891–896.
13. Gould TR, Westbury L, Brunette DM. Ultrastructural study of the attachment of human gingiva to titanium in vivo. J Prosthet Dent 1984;52:418–420.
14. Abrahamsson I, Berglundh T, Wennstrom J, Lindhe J. The peri-implant hard and soft tissues at different implant systems. A comparative study in the dog. Clin Oral Implants Res 1996;7:212–219.
15. Listgarten MA, Lang NP, Schroeder HE, Schroeder A. Periodontal tissues and their counterparts around endosseous implants [corrected and republished with original paging, article originally printed in Clin Oral Implants Res 1991;2(1):1–19]. Clin Oral Implants Res 1991;2(3):1–19.
16. Chavrier C, Couble ML, Hartmann DJ. Qualitative study of collagenous and noncollagenous glycoproteins of the human healthy keratinized mucosa surrounding implants. Clin Oral Implants Res 1994;5:117–124.
17. Lindhe J, Berglundh T. The interface between the mucosa and the implant. Periodontol 2000 1998;17:47–54.
18. Adell R, Lekholm U, Rockler B, Brånemark P-I. A 15-year study of osseointegrated implants in the treatment of the edentulous jaw. Int J Oral Surg 1981;10:387–416.
19. Wennstrom JL, Bengazi F, Lekholm U. The influence of the masticatory mucosa on the peri-implant soft tissue condition. Clin Oral Implants Res 1994;5:1–8.
20. 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;77:1410–1420.
21. Mombelli A, van Oosten MA, Schurch E Jr, Land NP. The microbiota associated with successful or failing osseointegrated titanium implants. Oral Microbiol Immunol 1987;2:145–151.
22. Austria M, Bissada N. Gingival thickness and the periodontal status in health and disease [abstract 1818]. J Dent Res 1992;71.
23. Albrektsson T, Zarb G, Worthington P, Eriksson AR. The long-term efficacy of currently used dental implants: A review and proposed criteria of success. Int J Oral Maxillofac Implants 1986;1:11–25.