The Importance of Keratinized Gingiva Surrounding Dental Implants

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Purpose: The purpose of this study was to determine if keratinized gingiva has an effect on the success of implants.

Materials and Methods: Sixty-nine implants were used in this study. The amount of keratinized gingiva was measured and divided into two groups; less than 2mm and greater than 2mm. The amount of keratinized gingiva was compared to clinical parameters such as bleeding upon probing, redness, and pocket depths to determine whether implant success was related to the amount of keratinized gingiva.

Results: Chi square and regression analysis were used to analyze the data. All implants survived independent of the amount of keratinized gingiva. Patients with less than 2mm of keratinized gingiva displayed increased bleeding upon probing and redness, which was statistically significant (p=0.023), indicating increased inflammation due to lack of keratinized gingiva.

Conclusion: Amount of keratinized gingiva did not affect the success rate of implants. However, implants with less than 2mm of

keratinized gingiva exhibited increased bleeding upon probing, redness, and inflammation, which may contribute to later failure.

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1.0 INTRODUCTION

The periodontium is composed of four structures, the cementum, alveolar bone, periodontal ligament, and the gingiva. It is considered the supporting structure of the teeth. These structures are derived from the dental follicle during tooth development. Each of the four components has a distinct location, composition, architecture, and function. The periodontium supports the teeth during function and allows the teeth to withstand considerable forces and insult.

Periodontal disease is an inflammatory disease of the periodontium resulting in the progressive destruction of the structures comprising the periodontium (Chung, 2006).

The protection and maintenance of periodontal health is thought to be related to the presence of an adequate zone of keratinized gingiva. Keratinized gingiva surrounds the necks of the teeth and is measured from the mucogingival junction to the free gingival margin. Histologic comparison of keratinized attached gingiva and nonkeratinized alveolar mucosa shows that attached gingiva is keratinized with thin, prominent epithelial ridges, and is firmly attached to the underlying bone and tooth allowing it be more protective and making it better able to withstand mechanical irritation (Bouri, 2008). Lang and Loe in 1972 stated that there must be at least 2 mm of keratinized gingiva, of which 1 mm must be attached (Lang

and Loe, 1972). Their study showed 80% of sites with keratinized gingiva 2 mm or greater remained healthy, while sites with less than 2 mm of keratinized gingiva demonstrated signs of clinical inflammation (Lang and Loe, 1972). These findings led to their conclusion that at least 2 mm of keratinized gingiva is required for stability of the periodontium (Lang and Loe, 1972). This conclusion also rationalized the introduction of numerous surgical procedures to increase the width of keratinized gingiva in deficient areas.

2.0 REVIEW OF THE LITERATURE

2.1 SURGICAL TECHNIQUES

Two commonly used surgical techniques by which to increase the width of keratinized gingiva are the free gingival graft and the subepithelial connective tissue graft (Oh, 2008). Bjorn in 1963 and Atkins in 1968 first described the free gingival graft procedure. This graft was initially used to increase the amount of keratinized gingiva, but later studies have demonstrated the ability of this graft to also attain root coverage (Oh, 2008). A free gingival graft involves grafting a donor piece of gingiva to a recipient site. The free gingival graft is a very predictable procedure to increase the amount of keratinized gingiva. Edel, in 1974, first described the subepithelial connective tissue graft. A subepithelial connective tissue graft refers to submerging gingival connective tissue under a partial thickness flap (Oh, 2008). The gingival connective tissue will induce the formation of keratinized gingiva. This procedure is not as predictable as a free gingival graft, but is also intended to increase the amount of keratinized gingiva and gain root coverage.

2.2 NEED FOR MINIMAL AMOUNT OF KERATINIZED GINGIVA

Later studies challenged this concept of the need for a minimal amount of keratinized gingiva, and have shown that by controlling inflammation with adequate oral hygiene, periodontal stability can be maintained with almost no keratinized gingiva. According to Wennstrom, a minimal amount of keratinized gingiva does not necessarily lead to gingival recession and inflammation (Wennstom, 2012). He stated that the narrow zone of keratinized gingiva located apically to an area of recession is the result of recession, not the cause (Wennstom, 2012). Some later studies state that even in areas of minimal keratinized gingiva, proper plaque control techniques can prevent gingival recession and soft tissue inflammation.

An exception to this was in teeth with subgingival restorations. There was a significant association between subgingival restorations and gingival inflammation in areas of minimal keratinized gingiva (Bouri, 2008). Stetler concluded that subgingival restorations placed on teeth surrounded by less than 2 mm of keratinized gingiva demonstrated an increased gingival index (Stetler, 1986). According to Steler in 1986, gingival grafting is recommended in areas where subgingival margins will be placed if the width of keratinized gingiva is less than 5 mm (Stetler, 1986). The rationale behind this is that the keratinized gingiva will provide a protective barrier against

inflammation and attachment loss (Stetler, 1986). A similar study by Lindhe and Ericsson demonstrated an increase in plaque and bacterial infiltrate in areas where subgingival restorations were placed with minimal keratinized gingiva (Greenstein, 2011).

2.3 DENTAL IMPLANT ANATOMY

In 1978, Dr. Branemark presented the titanium root – form implant (Abraham, 2014). This discovery was made accidently while studying blood flow in rabbit femurs (Abraham, 2014). He placed titanium chambers in their bone and noticed that over time the titanium became rigidly fixated to the bone and was not able to be removed (Abraham, 2014). This was later termed by Branemark as osseointegration, and was defined as a "direct structural and functional connection between ordered, living bone, and the surface of a load carrying implant" (Abraham, 2014). Several different types of implants were later introduced and the use of dental implants for replacement of missing teeth began to dramatically increase (Abraham, 2014). As the use of dental implants replacing natural dentition becomes increasingly the standard of care, the amount of keratinized gingiva surrounding dental implants to optimize gingival health also comes into question. Due to the structural and anatomical differences between implants and natural teeth, the same concepts cannot be applied to implants (Lin, 2013). Implants are more

susceptible to the development of inflammation and subsequent bone loss in the presence of plaque accumulation and bacterial infiltration due to several factors (Lin, 2013). The implant to mucosa interface is different from the interface between natural teeth and mucosa (Lin, 2013). While the junctional epithelium ends at a similar distance to the bone crest in both teeth and dental implants, the gingival fiber orientation is different (Lin, 2013). The gingival fibers of natural teeth run in a perpendicular configuration, whereas the gingival fibers of implants run in a parallel configuration to the implant and do not attach to the implant surface creating a much weaker mechanical attachment compared to natural teeth (Lin, 2013). This weaker attachment increases the susceptibility to bacterial infiltration leading to gingival inflammation and bone loss around the implant. If the surface of the implant is contaminated by bacteria, an inflammatory response is triggered in the connective tissue (Paiva, 2012). Unlike the periodontal ligament around natural teeth, the bone surrounding the implant cannot organize a defense mechanism against infection (Paiva, 2012). Therefore the apical extension of the inflammatory infiltrate around implants seems to result from the orientation of the supra-alveolar peri-implant fibers (Paiva, 2012).

2.4 IMPLANT-MUCOSA INTERFACE

As stated by Bouri in 2008 narrow zones of keratinized gingiva are less resistant to insult along the implant-mucosa interface. In the presence of an inflammatory response, implants placed in areas with narrow zones of keratinized gingiva have an increased susceptibility to tissue breakdown and showed earlier loss of attachment (Bouri, 2008). Greenstein, in a literature review, similarly stated that a narrow zone of keratinized gingiva, less than 2 mm, was associated with increased inflammation, plaque accumulation, and recession of the gingiva, ultimately resulting in tissue destruction (Greenstein, 2011). Wider zones of keratinized gingiva may offer more resistance to the forces of mastication and frictional contact that occurs during oral hygiene procedures and may create an environment that is less susceptible to tissue breakdown in the presence of inflammation (Bouri, 2008).

2.5 IMPLANT SUPPORTED RESTORATIONS

Also, the implant-supported restoration is often located subgingivally. As stated by Valderhaug and Birkeland the subgingival placement of the restoration was associated with a significantly increased rate of inflammation and attachment loss, especially in areas with minimal keratinized gingiva (Chung, 2006). An adequate biologic width is fundamental to the success of implants. The biologic width around implants ranges from 3-4 mm (Esper, 2012). It is composed of junctional epithelium and connective tissue. Prosthetic restorations extending subgingivally require a width of at least 5 mm of keratinized gingiva (Esper, 2012). These types of restorations often facilitate the accumulation of plaque bacteria and gingival inflammation by impinging on biologic width (Esper, 2012). According to Abrahamsson in 1996, a certain width of keratinized gingiva is required to promote an adequate epithelial and connective tissue attachment; otherwise bone resorption can occur in an attempt to establish an adequate biologic width around dental implants (Wennstrom, 2012).

2.6 HYPOTHESIS AND PURPOSE

There is a great deal of controversy in the literature about the importance of keratinized gingiva around dental implants and the amount, if any, which is required for implant health. Some studies concluded that peri-implant health could be maintained even in the absence of keratinized gingiva providing adequate oral hygiene is employed (Chung, 2006). Other studies suggest that areas of minimal keratinized gingiva have decreased tissue resistance allowing plaque accumulation, which increases the risk of gingival inflammation, marginal bone loss, and increased gingival recession (Chung, 2006). We hypothesize that implants surrounded by less than 2mm of keratinized gingiva are more susceptible to failure due to decreased resistance of the tissues to bacterial infiltration, leading to increased tissue breakdown, increased probing depths surrounding the implants, increased bleeding upon probing, and increased bone loss. The purpose of this study is to determine whether a minimum width of 2mm of keratinized gingiva around dental implants is necessary for the health and stability of the surrounding soft and hard tissues of the periodontium. Knowing this will help clinicians to determine whether or not gingival augmentation to increase the amount of keratinized gingiva is required prior to implant therapy.

3.0 MATERIALS AND METHODS

A cross sectional study was done to determine implant health/success when looking specifically at the amount of keratinized gingiva surrounding the implant. Implant health, as we determined, was the absence of bleeding upon probing, redness, inflammation, suppuration, mobility, pocket depths less than or equal to 3mm, and no radiographic evidence of progressive crestal bone loss.

Patients participating in this study were randomly selected from those who presented to the Graduate Periodontics Clinic or the Multidisciplinary Implant Center at the University of Pittsburgh School of Dental Medicine for routine maintenance appointments. Subjects included in this study were 21 years of age or older and have had an implant supported restoration placed a minimum of six months prior. Two examiners, one resident and one faculty member in the Graduate Periodontics Department, recorded data for this study. Both examiners were calibrated and inter and intra-examiner reliability was evaluated.

The following data was recorded for each implant: The number and site of the implant, width of keratinized gingiva, pocket depth, presence or absence of bleeding upon probing, presence or absence of suppuration, mobility of the implant, gingival color, radiographic bone level, time since implant placement, type of implant, smoking history in packs per year, age, and gender. The width of keratinized

gingiva was measured at the midfacial aspect of each implant using a Michigan Probe. Measurements were taken from the mucogingival junction to the free gingival margin and were measured to the nearest millimeter. Pocket depths were measured to the nearest millimeter using a Michigan Probe at six surfaces of each implant: mesial-buccal, midbuccal, distobuccal, mesial-lingual, midlingual, distolingual. Radiographic bone level was measured from a fixed reference point to the alveolar crest on periapical radiographs. The periapical radiographs taken at the time of implant placement were compared to periapical radiographs taken at the current maintenance appointment to assess crestal bone loss.

Width of keratinized gingiva was divided into two groups using 2 mm as a cutoff point: Group 1: implants where the width of the surrounding keratinized gingiva was 2 mm or greater, Group 2: implants where the width of the surrounding keratinized gingiva was less than 2 mm.

4.0 RESULTS

This study included 69 patients. Thirty-six patients (52.2%) of the patients were 26-50 years old and 33 patients (47.8%) ranged in age from 51-75 years. Forty-nine patients (71.0%) were male, while 20 (29.0%) were female. Thirty-four (49.3%) of the patients reported current use of tobacco products, while 35 (50.7%) of the patients denied use of tobacco. Eighteen patients (26.1%) had keratinized gingiva less than 2mm surrounding their implants, compared to 51 patients (73.9%) who had more than 2mm of keratinized gingiva surrounding their implants. Bleeding upon probing was seen in 17 patients (24.6%) and was absent in 52 patients (75.4%). Pocket depths of more than 3mm was noted in 22 patients (31.9%), compared to pockets depths less than 3mm noted in 47 patients (68.1%) (Table 1).

The data was analyzed using a Chi Square test to determine if the width of keratinized gingiva significantly affected probing depths and bleeding upon probing. The data was considered statistically significant if the p value is less than or equal to 0.05. Also, a multivariate regression analysis was done to determine whether the width of keratinized gingiva was independently associated with bleeding upon probing. Smoking and gender were adjusted. Age was

not distributed well enough to be used in the model. Other variables were not significant and were not included in the final model.

Chi Square analysis was done to evaluate the association between the amount of keratinized gingiva and pocket depths. Statistical analysis of the data for pocket depths shows that there is no significant association between pocketing and amount of keratinized gingiva; indicating that a lack of keratinized gingiva does not result in greater pocket depths (p = 0.878) (Table 2).

Statistical analysis of the data for bleeding upon probing shows that there is a statistically significant association between the amount of keratinized gingiva and bleeding upon probing (p = 0.023). Eighty-two percent of the implants with less than 2mm of keratinized gingiva experienced bleeding upon probing as compared to 56% of the implants with keratinized gingiva greater than 2mm (Table 3).

A logistic regression model was performed, and it was adjusted for smoking and gender. Since age was not distributed well enough, it was not used in the model. Variables such as, suppuration, mobility of the implant, timing of implant placement, and width of the implant were not significant and were not included in the final model.

Table 4 showed implants with less than 2mm of keratinized gingiva are 6.5 times more likely to experience bleeding upon probing

than those implants with greater than 2mm of keratinized gingiva (OR=6.5). The final model was adjusted for smoking and gender.

TABLE 1. KERATINIZED GINGIVA RESEARCH CASE REPORT

Characteristics	Number of Patients
Age	
<25 years	0
26-50 years	36
51-75 years	33
>75 years	0
Gender	
Male	49
Female	20
G 1	
Smoke	2.4
Yes	34
No	35
KG levels	
< 2mm	18
> 2mm	51
, 2	0.1
Bleeding	
Yes	17
No	52
Redness	
Yes	3
No	66
D. L. (D. C)	
Pocket Depth	2.2
> 3 m m	22
< 3 m m	47
Timing of Implant	
< 2 years	5
2-3 years	15
> 3 years	49
/ 5 years	47

TABLE 2. POCKETING VS KERATINIZED GINGIVA

Pocketing > 3mm	KG < 2mm Yes No		Total
Yes	6.00	16.00	22.00
	27.27	72.73	100.00
	33.33	31.37	31.88
No	12.00	35.00	47.00
	25.53	74.47	100.00
	66.67	68.63	68.12
Total	18.00	51.00	69.00
	26.09	73.91	100.00
	100.00	100.00	100.00

Chi square = 0.0236 **P = 0.878**

TABLE 3. BLEEDING VS KERATINIZED GINGIVA

Bleeding	KG < 2mm Yes No		Total
Yes	8.00 9.00		17.00
	47.06	52.94	100.00
	44.44	17.65	24.64
No	10.00 42.00		52.00
	19.23	80.77	100.00
	55.56	82.35	75.36
Total	18.00	51.00	69.00
	26.09 73.91		100.00
	100.00	100.00	100.00

Chi Square = 5.1455 **P** = 0.023

TABLE 4. REGRESSION FINAL MODEL

Bleeding	Odds	Standard	Z	P > z	95% CI
	Ratio	Error			
KG <	6.483607	4.868435	2.49	0.013	1.488 28.25
2 m m					
Gender	2.780876	2.123517	1.34	0.180	0.623 15.42
Smoking	12.19472	9.778232	3.12	0.002	2.533 58.71
_ cons	0.2460288	0.1948526	-1.77	0.077	0.521 1.162

5.0 DISCUSSION

The need for keratinized gingiva around dental implants has been a controversial topic. Several studies have suggested that a minimal width of keratinized gingiva around implants is necessary for health and stability of the implant, while other studies have failed to demonstrate the need for minimal width.

The results of this study suggest that implants surrounded by less than 2mm of keratinized gingiva have an increased amount of bleeding upon probing. Bleeding upon probing is a clinical indication of active inflammation. Prolonged inflammation around dental implants can result in subsequent attachment loss and bone loss, ultimately leading to failure of the implant.

As stated by Lang and Loe, the minimum width for healthy keratinized tissue surrounding the teeth is 2mm (Lang and Loe, 1972). This concept has been carried over to peri-implant keratinized tissue. However, several studies have challenged this concept as it pertains to teeth and also implants, and have stated that a minimum width of keratinized gingiva is not required provided adequate oral hygiene is maintained. Cox and Zarb in 1987 conducted a study in which they found that 80% of the implants evaluated had no keratinized gingiva but had healthy peri-implant tissue (Cox and Zarb, 1987). Similarly, Esper in 2012 showed no statistically significant difference between

bleeding upon probing and plaque control and the width of keratinized gingiva (Esper, 2012).

While the absence of keratinized gingiva around dental implants does not necessarily cause peri-implant disease, maintaining meticulous oral hygiene in areas of minimal keratinized gingiva is difficult because mobile mucosa is more susceptible to inflammatory changes (Ten Bruggencate, 1991). Proper oral hygiene may be better facilitated in areas of adequate keratinized gingiva (Salvi and Lang, 2004).

Healthy keratinized gingiva around dental implants results in more predictable success and maintenance of the implant, and also results in an improved esthetic outcome. Keratinized gingiva provides stabilization to the periodontium, protects the teeth and implants from masticatory and external trauma, and provides a barrier to inflammatory infiltrate (Paiva, 2012). While the sample size in this study is limited, we feel that implants should have a minimum amount of 2mm of keratinized gingiva to maintain health. We believe that reconstruction of the keratinized gingiva in deficient areas using techniques such as the free gingival graft or the subepithelial connective tissue graft should be employed prior to implant placement.

6.0 CONCLUSION

Despite limited data in this study, we concluded that regardless of the amount of keratinized gingiva present, implant placement was successful. However, implants with less than 2mm of keratinized gingiva exhibited increased bleeding upon probing, which is a clinical sign of inflammation. Persistent inflammation around an implant may possibly contribute to later failure. These findings may warrant gingival augmentation prior to implant placement in areas where minimal keratinized gingiva exists to prevent future failure. Further studies may be needed to confirm the findings from this study due to the small sample size.

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