The Association Between Buccal Mucosa Thickness and Periimplant Bone Loss and Attachment Loss: A Cross-Sectional Study

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Purpose: *The aim of this study was to assess if there is an association between buccal mucosa thickness and periimplant attachment loss after 1 year of function.*

Materials and Methods: *A total of 28 patients (14 periimplantitis implants and 14 healthy implants) were included. The buccal mucosal thickness was assessed using K-files at 3 mm apical to the soft tissue margin of the implant. Probing depth, recession (REC), clinical attachment level (CAL), bleeding on probing, and radiographic bone loss on mesial and distal sites of the implant were recorded.*

Results: *The data showed that there was a statistically significant difference in midfacial REC between thin and thick buccal mucosa groups. However, the CAL was not statistically significant different between both groups. In addition, there was no statistically significant difference in mesial and distal bone loss between implants with thin and thick mucosa.*

Conclusion: *When the midfacial soft tissue thickness was thin, the midfacial REC was greater and the CAL also tended to be higher. There was no association between buccal mucosa thickness and periimplant bone loss on mesial and distal sites of the implant after 1 year of function. (Implant Dent 2018;27:1–7)*

Key Words: alveolar bone loss, dental implants, tissues, phenotype

Dental implants have been widely used as the treatment modality for partially and fully edentulous patient due to its high survival rates after 5 to 10 years.[1,2] However, despite its high survival rates, periimplant disease is not uncommon. A recent report showed a high frequency of periimplantitis, with 18.8% on a subject level and 9.6% on an implant level.[3] Periimplantitis has been defined as an inflammatory process around an implant that includes both soft tissue inflammation and progressive loss of supporting bone beyond biological bone remodeling, which may lead to implant failure if it is not properly treated.[4] It was hypothesized that lack of soft tissue seal at the base of the soft tissue-implant interface and absence of cementum with inserting collagen fibers may increase susceptibility for bone loss around implants.[5] In the gingiva, most of the collagen fiber bundles are originated from root cementum, whereas soft tissue formed around implants are originated from the mucosa of the edentulous alveolar ridge or masticatory mucosa. Thus, the collagen fibers in the gingiva are attached to the root cementum and oriented in perpendicular direction into lateral portions of the soft tissue, whereas in periimplant tissue, the fiber bundles are arranged in parallel direction to the surface of the titanium abutment and attached to the marginal bone.[6]

The formation of junctional epithelium around implants is resulted by proliferation and migration of epithelial cells on the exposed connective tissue and it continues until the epithelial continuity is restored. Immediately after abutment connection, the early bone remodeling begins,[7] and it may

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continue to remodel down to the first implant thread after the first year of function.[8,9] Therefore, it was suggested that connective tissue integration during the initial healing is critical to establish a biological barrier and to prevent further epithelial migration around implant.[6,10] Moreover, it has been suggested that a certain thickness of periimplant mucosa is needed to protect osseointegration after abutment connection and loading of the implant.[10] Kois[11] described thick tissue biotype as a more fibrotic tissue and is more resistant to recession (REC), whereas thin tissue biotype is more friable and has less vascularization or blood supply and thus it increases the risk of REC after implant surgery. Berglundh and Lindhe[10] evaluated the marginal bone level of implants placed in area where the connective tissue on the inside of the flap was excised to obtain a thin mucosa and compared it to the implants placed in the contralateral site where the flap was thick. The results showed that when the ridge mucosa was thin (≤2 mm), more bone resorption and angular bony defect were noted.[10] In agreement with the latter study, Abrahamsson et al[12] also observed that implants that were placed at sites where the mucosa of the ridge was thin showed angular bone defects occurring at periimplant marginal bone, whereas implants placed at sites with thick mucosa showed even pattern of alveolar crest. In contrast, a more recent study demonstrated that thinner keratinized tissue around implant did not influence the buccal bone plate remodeling even with flapless implant placement.[13] It was emphasized that the buccal bone thickness plays a more important role than tissue thickness on the periimplant bone loss.[13]

A human prospective clinical trial reported that tissue/mucosa thickness might affect crestal bone stability around implants.[14] The study showed that implants with initially thin tissue (≤2 mm) had bone loss up to 1.45 mm that occurred within the first year of function. Whereas thick tissues (>2 mm) only had 0.2 mm bone loss noted.[14] Puisys and Linkevicius[15] also reported that greater crestal bone loss in the thin tissue group could be seen approximately 2 months after healing, before implant loading, which suggested that biological width around implants formed from thin tissue was less stable than the one originated from thick or thickened mucosa. Nevertheless, the mucosal thickness was measured vertically to the bone crest and the bone loss was calculated from the implant abutment junction in these studies.[14–16] Therefore, it may be considered as a biological width formation phenomenon, and the significance of mucosal thickness on periimplant crestal bone loss remains questionable. Hence, the aim of this study was to assess if there is an association between buccal mucosa thickness and periimplant bone loss and attachment loss beyond initial biologic bone remodeling.

MATERIALS AND METHODS

Trial Design

This study was approved by the University of Michigan (U-M) Institutional Review Board (HUM00111621). Patients at the U-M School of Dentistry who received endosseous root-form dental implants and had the implants restored at least 1 year before the study were screened in this cross-sectional study. The prescreening was performed by reviewing the records of dental school patients. Patient's dental school records were reviewed for inclusion/exclusion criteria. A letter was sent to patients who qualified, inviting them to participate in the study and be scheduled for a screening visit. Upon the screening visit, the informed consent was obtained and medical history was reviewed. After potential subjects were screened, subjects were clinically evaluated and periapical radiographs were taken at the implant site. Disclosing solution was used to stain the subject's plaque, and the plaque score was calculated and recorded based on plaque control record described by O'Leary et al in 1972.[17] Subjects were categorized into: (1) healthy implants group and (2) implants with periimplantitis group. Subjects were diagnosed with periimplantitis if the probing depth (PD) of the implant sites was ≥4 mm with bleeding and/or suppuration on probing, and there was evidence of radiographic bone loss beyond the first thread of the implant.[18]

Subject Eligibility

Potential subjects were screened according to the inclusion criteria as follows: (1) male or female aged ≥18 years; (2) had implants in the anterior or posterior region loaded for ≥1 year; (3) had regular maintenance visits (every 3–6 months); (4) had ≥2 mm keratinized mucosa on the buccal and lingual of the implants; (5) had sharp and undistorted periapical x-rays at the time of implant placement; (6) implants with an internal or external connection, 1 or 2 components, and a rough or smooth collar; (7) implants placed crestally, supracrestally, or subcrestally; (8) had good oral hygiene; and (9) had stable periodontium. Subjects were excluded if they were a smoker or quit smoking less than 1 year before implant placement; were pregnant or plan to become pregnant over the next 6 months; had uncontrolled diabetes (HbA1C > 7) before implant placement; had history of intravenous bisphosphonates; had history of radiation therapy in the head and neck area within 4 years before implant placement; had poor oral hygiene (plaque score more than 40% based on O'Leary plaque score); had implants that were placed immediately after extraction or immediately loaded; or had implants with unclear and/or distorted radiographs.

Clinical Measurements

At the screening visit, probe transparency technique was used to assess gingival tissue thickness. When the tip of the probe was visible through the gingiva around implants, the tissue was considered thin. Clinical measurements (PD, REC, clinical attachment level [CAL], and bleeding on probing) were recorded using a University of North Carolina manual probe on the implant. REC was measured from free gingival margin to the junction implant/crown. CAL was measured from the junction implant/crown to the most apically probable portion in millimeters. Buccal mucosal thickness at 3 mm apical to the soft tissue margin measured at 3 locations (mesial, midfacial, and distal) of

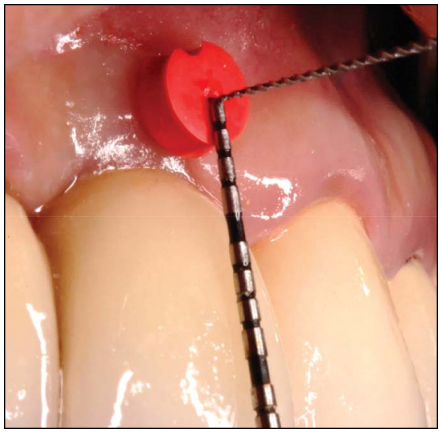

Fig. 1. Buccal mucosal thickness measurement at 3 mm apical to the soft tissue margin of the implant using K-files with endo stopper.

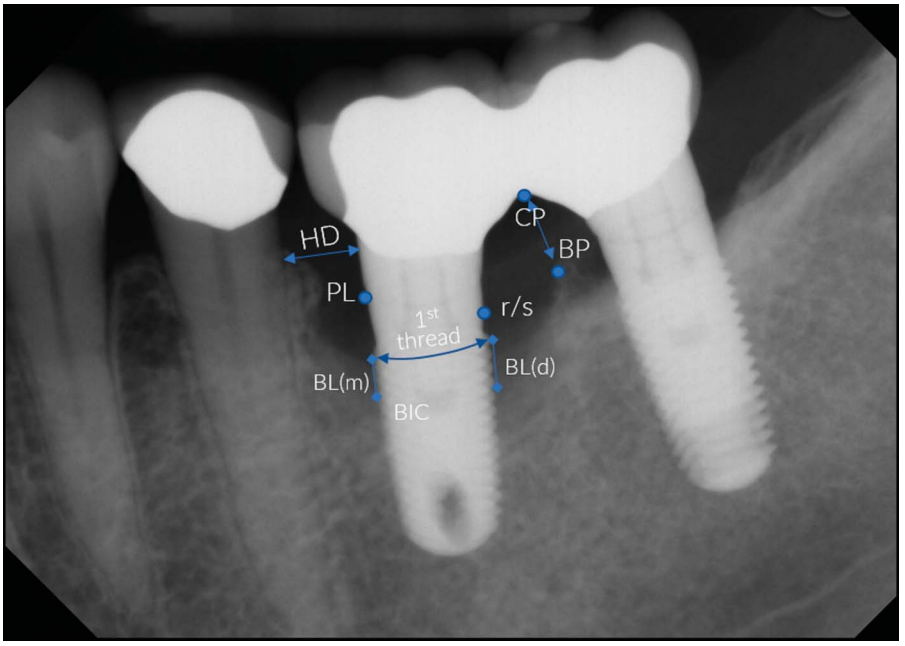

Fig. 2. Schematic drawing showing the selected reference points. PL, r/s, and first radiographic BIC were identified. HD represents horizontal distance between the adjacent tooth and PL, and BL-1st TD (m) and BL-1st TD (d) represent vertical distance between BL and the first implant thread at mesial ($BL_{(m)}$) and distal ($BL_{(d)}$) implant surfaces at the most recent follow-up appointment respectively. PL indicates implant platform; r/s, rough/smooth implant border; BIC, bone-implant contact.

the implant using K-files with endo stopper (30 K-file; Dentsply, Tulsa, OK) (Fig. 1). Before conducting buccal mucosal thickness measurements, a local infiltration was given on the buccal of implant sites using 2% lidocaine with 1:100,000 epinephrine at the depth of the vestibule to avoid increased tissue thickness at the measurement sites. When the tissue was ≤2 mm, it was considered as thin tissue. In contrast, if the tissue thickness was >2 mm, it was categorized as thick tissue. Inter examiner calibration was performed by 2 examiners (J. M. and M. A.) for soft tissue thickness and PD measurements. The *t* test analysis showed no statistically significant difference in soft tissue thickness and PD measurements between both examiners ($P = 0.5$). For midfacial soft tissue thickness and PD measurements, both examiners reached 100% agreement. Height of keratinized mucosa was measured from the soft tissue margin to the mucogingival line at the facial and lingual aspect of the implant. In addition, clinical photographs were also taken for further comparison.

Radiographic Assessment

The periapical or bitewing radiographs taken immediately after implant placement and at the time of implant restoration were digitalized at a resolution of 600 dpi and saved in JPEG format. Intraoral radiographs were taken at the most recent follow-up visit using a paralleling technique with

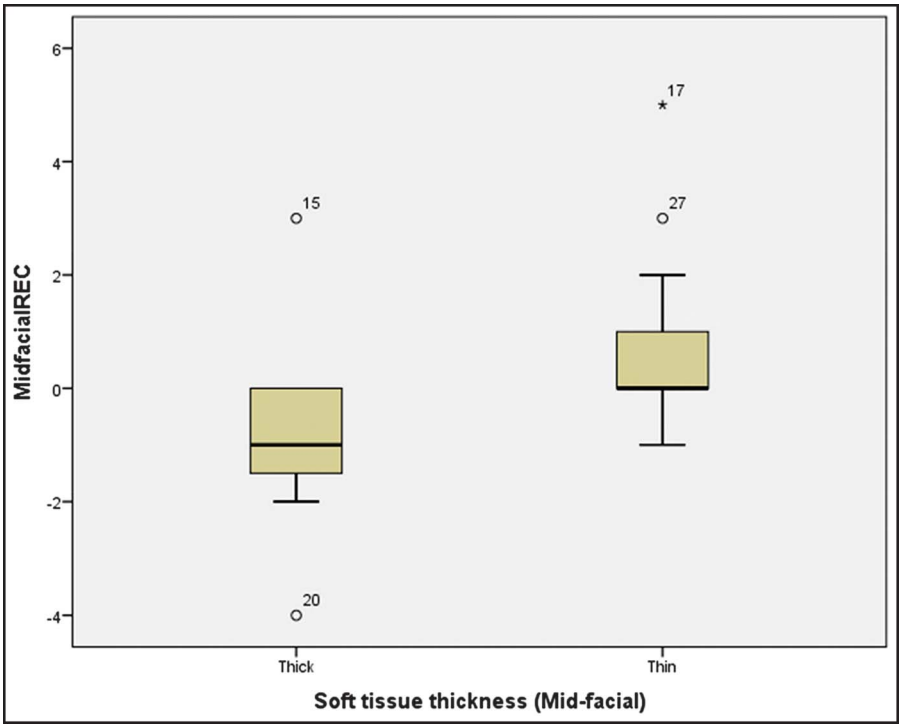

Fig. 3. The mean midfacial REC on implants with thick and thin buccal mucosa was –0.82 ± 1.72 mm and 0.71 ± 1.53 mm, respectively. There was statistically significant difference in midfacial REC between thin and thick buccal mucosa groups ($P = 0.021$).

a Rinn-type film holder. Changes in marginal bone level were evaluated using an open source software package (ImageJ; U.S. National Institutes of Health, Bethesda, MD).[19] The images were magnified and viewed under the full screen mode for better visualization. The known implant length was used to calibrate the measurements. A built-in digital caliper in the software was used for all measurements. Pixel values of a given linear measurement were converted to an international system for units in millimeter.

Vertical distance between first radiographic bone-implant contact and the first implant thread at mesial ($BL_{(m)}$) and distal ($BL_{(d)}$) implant surfaces were measured to calculate the amount of bone loss on the mesial and distal sites of the implant (Fig. 2). A single calibrated examiner (J. M.) performed all radiographic measurements. Intra-examiner calibration was performed by measuring the parameters of radiographs at 2 separate occasions that were 5 days apart. The intra-examiner reliability obtained had a kappa value of 95% as the differences in linear measurements were within 0.5 mm.

Statistical Analysis

In the present study, test significance level (α) used was 5% and the power analysis was 80%. Sample size for each group was calculated using a computer program with 2-sided equivalence for difference of proportions in 2 group designs (nQuery Advisor, version 7.0; Statistical Solutions Inc., Los Angeles, CA). According to the previous study,[14] mean bone loss of implant placed in thin tissue biotype (μ_1) was 1.450 mm and mean bone loss of implant placed in thick tissue biotype (μ_2) was 0.170 mm. The difference in mean values in between 2 groups ($\mu_1 - \mu_2$) was 1.280 mm and common SD was (σ) was 1.160 mm. Therefore, the minimal sample (n) needed in each group in this present study was 14 patients. Data were analyzed using a statistic software program (IBM SPSS Statistics for Windows, Version 24.0; IBM Corp., Armonk, NY). Fisher exact test was used to detect if there were any differences in buccal mucosal thickness between healthy implant and periimplantitis groups. Independent *t* test analysis was conducted to assess mean differences between the groups.

RESULTS

A total of 28 patients with rough surface implants placed between March 2001 and August 2013 fulfilled the inclusion criteria were included in this study. The patient population consisted of 14 men and 14 women, with a mean age of 67.5 ± 10.4 years. Statistical analysis using Fisher exact test and *t* test showed that there was no gender difference ($P = 0.057$) or age difference (mean age: 68 vs 66.9 years, $P = 0.793$) between healthy implant and periimplantitis groups. All implants were placed in healed sites without immediate loading and restored with fixed dental prostheses. There were 14 implants placed in mandible and 14 implants placed in maxilla. No statistical significant difference was found regarding implant location between healthy implant and periimplantitis groups ($P = 0.257$). The mean follow-up period was 7.65 ± 4.3 years, which was timing of measurement after the implants had been restored. At the measurement visit, clinical examination and radiographic assessment showed that 14 implants had periimplantitis and 14 implants were healthy. There was no statistical significant difference in mean follow-up time between healthy implant and periimplantitis groups (mean: 81.07 vs 102.5 months, $P = 0.283$).

In healthy implant group, 4 implants were placed supracrestrally, 6 implants were placed crestally, and 4 implants were placed subcrestally. In periimplantitis group, there were 4 implants placed supracrestally, 7 implants placed crestally, and 3 implants placed subcrestally. Pearson Chi-squared test showed no statistically significant difference in implant placement position in relation to the crest of the bone between both groups ($P = 0.896$). Majority of the implants were restored with cement restorations (25 implants), and 3 implants were restored with screw-retained restorations. There was no statistically significant difference in the type of implant restorations between groups ($P = 1$).

The mean soft tissue thickness at the mesial, midfacial, and distal sites in

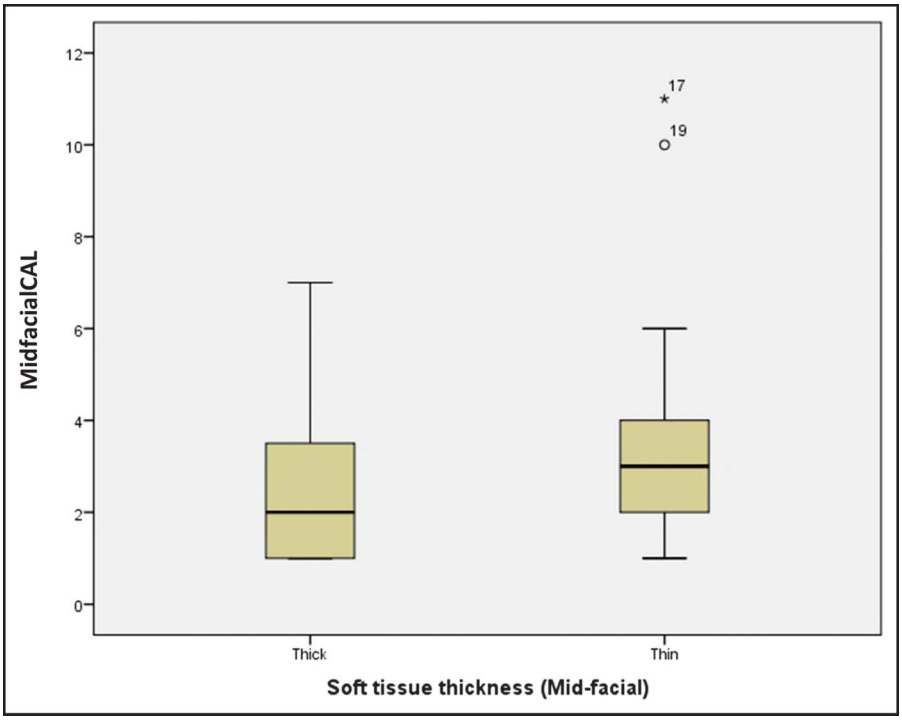

Fig. 4. The mean CAL in thin tissue and thick tissue group was 3.88 ± 2.85 mm and 2.73 ± 2.10 mm, respectively. However, the CAL difference between both groups did not reach statistically significant difference ($P = 0.259$).

Table 1. Mean Bone Loss of Implants With Thick Soft Tissue (>2 mm) and Thin Soft Tissue (≤2 mm) in Periimplantitis Group

	Thickness	N	Mean	SD	SEM
$BL_{(m)}$	Thick (>2 mm)	5	2.5720	1.67883	0.75080
	Thin (≤2 mm)	9	1.4733	0.71741	0.23914
$BL_{(d)}$	Thick (>2 mm)	6	1.3717	0.76971	0.31423
	Thin (≤2 mm)	8	2.5413	1.36904	0.48403

BL(m) indicates bone loss at mesial site; BL(d), bone loss at distal site.

healthy implant group were 2.86 ± 1.06 mm, 2.43 ± 0.68 mm, and 3.29 ± 1.24 mm, respectively. The mean soft tissue thickness at the mesial, midfacial, and distal sites in periimplantitis group were 2.54 ± 1.29 mm, 1.82 ± 0.50 mm, and 2.39 ± 1.04 mm, respectively. Independent *t* test showed there was statistically significant difference in soft tissue thickness on midfacial ($P = 0.013$) and distal ($P = 0.049$) sites between healthy implant and periimplantitis groups. In healthy implant group, there were 8 implants presented with thick soft tissue (>2 mm) and 6 implants presented with thin soft tissue (≤2 mm), whereas in periimplantitis group, there were 3 implants presented with thick soft tissue (>2 mm) and 11 implants presented with thin soft tissue (≤2 mm). Fisher exact test revealed there was no statistically significant difference in distribution of soft tissue thickness between healthy implant subjects and subjects with periimplantitis ($P = 0.120$).

The mean midfacial PD in healthy implant group and periimplantitis group were 2.43 ± 0.76 mm and 4.57 ± 2.53 mm, respectively. There was statistically significant difference in midfacial PD between healthy implant and periimplantitis groups ($P = 0.008$). The mean midfacial REC in healthy implant and periimplantitis groups were −0.57 ± 0.65 mm and 0.79 ± 2.22 mm, respectively. There was statistically significant difference in midfacial REC between healthy implant and periimplantitis groups ($P = 0.044$). The mean midfacial CAL in healthy implant and periimplantitis groups was 1.79 ± 0.97 mm and 5.07 ± 2.70 mm, respectively. There was significantly higher midfacial CAL in periimplantitis group than in healthy implant group ($P = 0.001$).

Mean midfacial PD on implants with thick and thin buccal mucosa was 3.64 ± 2.34 mm and 3.41 ± 2.06 mm, respectively. There was no statistically significant difference in midfacial PD between implants with thick and thin buccal mucosa ($P = 0.791$). The mean midfacial REC on implants with thick and thin buccal mucosa was −0.82 ± 1.72 mm and 0.71 ± 1.53 mm, respectively. There was statistically significant difference in midfacial REC between thin and thick buccal mucosa groups ($P = 0.021$) (Fig. 3). The mean CAL in thin tissue group and thick tissue group was 3.88 ± 2.85 mm and 2.73 ± 2.10 mm, respectively. However, the CAL difference between both groups did not reach statistically significant difference ($P = 0.259$) (Fig. 4).

Mean bone loss around implants in periimplantitis group was 1.87 ± 1.21 mm on the mesial ($BL_{(m)}$) site and 2.04 ± 1.26 mm on the distal ($BL_{(d)}$) site. The mean bone loss around implants with thin soft tissue (≤2 mm) in periimplantitis group was 1.47 ± 0.72 mm on the mesial site and 2.54 ± 1.37 mm on the distal site. The mean bone loss around implants with thick soft tissue (>2 mm) was 2.57 ± 1.68 mm on the mesial site and 1.37 ± 0.77 mm on the distal site (Table 1). No statistically significant difference in bone loss was found between implants with thin and thick soft tissue on the mesial site ($P = 0.108$) and distal site ($P = 0.086$) in periimplantitis group.

DISCUSSION

The current study evaluated buccal mucosa thickness in healthy implant and periimplantitis groups. Data showed that the mean midfacial REC in periimplantitis group was greater than healthy implant group. Furthermore, the mean midfacial REC in thin tissue group was also significantly greater than in thick tissue group. When the midfacial soft tissue thickness was thin, the CAL also tended to be greater; however, the difference did not reach the statistically significant level. This may be due to inadequate sample size. However, similar findings were also reported by Annibali et al, who retrospectively evaluated 53 first molar implants. They found that implants with thin soft tissue biotype seemed to be more susceptible to a greater amount of buccal REC and incomplete filling of interdental papilla than implants with thick tissue biotype in the first molar area.[20] In addition, it was also observed that sites with thick gingival biotype had significantly less facial gingival level change than sites with a thin gingival biotype at 1 and 4 years after implant placement.[21] The mean midfacial PD was also significantly higher in periimplantitis group than in healthy implant group. This finding is in accordance with the study reported by Lang et al, who evaluated probe penetration in healthy and inflamed periimplant tissues in an animal study. The results of the latter study showed that the probe penetration increased with higher degree of inflammation, and in periimplantitis sites, the probe penetration was almost close to the bone crest, resulting in deeper PD than healthy implant sites and periimplant mucositis sites.[22]

Results from this study also showed that there was statistically significant difference in mean mid-buccal and distal mucosa thickness between the healthy implant and periimplantitis groups. However, when implants with thin and thick buccal mucosa were compared, there was no statistically significant difference in mesial and distal bone loss between implants with thin and thick buccal mucosa in periimplantitis group. Nonetheless, the mean midfacial PD and CAL was found statistically significant different between periimplantitis group and healthy implant group. Quirynen et al evaluated 108 patients who had implants supported overdentures and measured the CAL around the implants using electronic probe and examined the relationship between radiographic bone level and CAL around implants. The observations showed that the mean probing attachment level correlated with

marginal bone level seen on radiographs. Thus, it was suggested that the attachment level determination around implants could be used as an indicator for the bone level around implants.[23]

It was observed that after successful placement and abutment connection of implants with various systems, mucosal barrier was formed around implants, which consisted of zone of junctional epithelium and zone of connective tissue.[10,12,24] The distance from the bone crest to the outer surface of the oral epithelium was found on average 3 mm.[10,12,24] The linear dimensions of these zones are also known as the biologic width. Therefore, soft tissue thickness measurements in the present study were performed at 3 mm from free gingival margin of the implants. When dental implants are placed into function, the crestal bone remodels as a result of stress concentration at the coronal region of the implant.[8] As it remodels, it changes in morphology to adapt to the existing external environment or load.[25] Radiographically, postrestorative "remodeled" crestal bone level generally lies at the first thread on most 3.5-mm implants.[8] Hence, any bone loss beyond the biologic bone remodeling after loading is considered as a pathologic process. For this reason, $BL_{(m)}$ and $BL_{(d)}$ in the current study were measured from implant first thread.

The results in the present study agrees with the study reported by Maia et al,[13,26] who evaluated the influence of gingival thickness after immediate implant placement in animal studies. To evaluate the influence of buccal gingival thickness on bone healing, the latter study included 8 young beagle dogs that had thin buccal bone plate. The buccal mucosa on one side of the mandible was made thin using high speed bur followed by traumatic brushing before implant placement until there was a statistically significant difference in gingival thickness between thick and thin buccal gingiva groups.[26] The authors reported that there was no significant difference in implant buccal bone loss observed between thin and thick buccal gingiva group, and thus, it was concluded that gingival thickness does not influence implant buccal bone resorption when the buccal bone is initially thin.[13,26] Chappuis et al[27] analyzed 39 patients with thin facial soft tissue biotype (0.7–0.8 mm) who received tooth extraction and evaluated the facial soft tissue changes and the underlying facial bone dimensions after 8 weeks of socket healing. The analysis based on digitized impressions and cone beam computed tomography during an 8-week of healing showed that in patients with thin buccal bone wall, there was a spontaneous soft tissue thickening after tooth extraction. However, in patients with thick buccal bone wall, the facial soft tissue thickness remained thin and did not change over time. This observation suggested that significant buccal bone resorption and facial soft tissue changes occurred when the buccal bone wall was thin, whereas in thick buccal bone wall, the horizontal bone loss was minimal and soft tissue dimensions remained unchanged.[27] The authors explained that facial soft tissue ingrowth and buccal bone resorption are more rapid in thin facial bone walls because soft tissue cells occupy the majority of the available space in the extraction socket. However, thick facial bone walls favor the ingrowth of cells from the socket walls and surrounding bone marrow space and thus minimizes soft tissue ingrowth and facial bone resorption.[27] Furthermore, in naturally thin buccal bone wall, the use of grafting material did not prevent buccal bone resorption, and the amount of bone resorption was similar between thin and thick buccal mucosa biotypes.[28]

On implant sites, a minimum of 2 mm of facial bone thickness has been proposed as the "critical bone thickness" for the prevention of vertical height loss of the facial plate.[29] When the distance of the buccal shoulder position of the implant to facial bone plate was below this critical thickness, an increased amount of facial bone resorption can be expected, which may in turn increase the chance of implant mucosal REC and failure. However, when the facial bone thickness approached 1.8 to 2.0 mm from the implant buccal shoulder position, the likelihood of facial bone loss was decreased and bone gain occurs more frequently.[29] Chen et al[30] evaluated the soft tissue and radiographic outcomes of implants placed in extraction sockets using a nonsubmerged protocol. The results showed that there was statistically significant higher marginal tissue REC at sites when implants were placed 1.1 mm from the inner buccal socket wall when compared to implants placed 2.3 mm from the inner buccal socket wall. Resorption of the buccal bone was significantly greater in sites with thin buccal plate. The authors concluded that a minimum of 2 mm distance from the implant shoulder to the buccal wall was needed to prevent implant marginal tissue REC and vertical bone resorption.[30]

There were several limitations in this study: (1) the radiographs were nonstandardized; (2) no information was available regarding ridge width and buccal bone thickness at the time of implant placement; and (3) the study sample is relatively small to illustrate a possible statistical significance difference.

CONCLUSION

Within the limitations of the study, it can be concluded that there was statistically significant difference in midfacial REC between thin and thick tissue groups. In addition, when the midfacial soft tissue thickness was thin, the CAL also tended to be greater; however, the difference was not statistically significant. There was no association between buccal mucosa thickness and periimplant bone loss on mesial and distal site of the implant after 1 year of function.

DISCLOSURE

The authors claim to have no financial interest, either directly or indirectly, in the products or information listed in the article.

APPROVAL

This study was approved by the University of Michigan (U-M) Institutional Review Board (HUM00111621).

ROLE/CONTRIBUTION OF AUTHORS

J. Mailoa: Conducting the research, attaining and analyzing the data, and drafting the manuscript. M. Arnett: Recruiting subjects, calibrating measurements, and critically revised the manuscript. H.-L. Chan: Contributing in data interpretation, drafting and critically revised the manuscript. F. M. George: Contributing in data interpretation, drafting and critically revised the manuscript. D. Kaigler: Contributing in data interpretation, drafting and critically revised the manuscript. H.-L. Wang: Contributing to conception, study design, data interpretation, drafting and critically revised the manuscript.

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