Soft Tissue Exposure of Endosseous Implants Between Stage I and Stage II Surgery as a Potential Indicator of Early Crestal Bone Loss

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Implants are well accepted as a means of dental rehabilitation. While integration success rates are high, crestal bone loss can occur, and it may not become apparent until stage II surgery and implant uncovering. The purpose of this study was to quantify the relationship between exposure of implants through the oral mucosa between stage I and stage II implant surgery and early changes in crestal bone height. Bone levels were measured during placement of 275 implants in the maxillae of 50 subjects. Repeated bone height measurements were obtained at implant uncovering. Fourteen implants in 7 patients were exposed to the oral cavity through the mucosa at stage II surgery. Patients with 1 or more exposed sites demonstrated a likelihood of bone loss 3.9 times greater than patients with nonexposed sites (Fisher exact test, $P = .0003$). These results suggest that exposure of an implant between stage I and stage II implant surgery might serve as a potential indicator of the occurrence of early bone loss. (INT J ORAL MAXILLOFAC IMPLANTS 1999;14:436–441)

Key words: bone loss, endosseous dental implants, implant exposure

Endosseous cylindric implants are well accepted as safe and effective for general use in dental rehabilitation. Through the biodynamic process of osseointegration, a structural and functional connection has been shown to develop at the interface between the implant and the surrounding osseous tissue bed following successful surgical placement.[1] Implant surgery commonly proceeds in 2 stages. During stage I, the soft tissue is reflected and the implant is placed into the arch following careful preparation of the osseous tissue bed. The soft tissue is then sutured over the implants, obtaining primary closure of the wound. After an adequate healing period, stage II proceeds with the exposure of the implant by reflection of the soft tissue and the subsequent placement of an interconnecting transmucosal abutment. Prosthetic rehabilitation may then commence.

The literature uniformly describes high early implant integration success rates.[2,3] However, loss of the supporting bone during the period between stage I and stage II surgery can occur and becomes clinically apparent only at the time the implant is uncovered. Factors implicated in this bone loss include surgical complications, a less-than-ideal initial fit between the implant and the surrounding bone, insufficient osseous tissue volume to adequately surround the implant, premature loading with resulting micromovement of the implant prior to integration, harmful patient habits including tobacco product abuse, and healing impairment resulting from poor overall patient health.[4,5] Identification of early bone loss

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prior to uncovering of implants is difficult, since patients are frequently asymptomatic. If significant bone loss is encountered at the time of implant uncovering, further surgical treatment may be required, resulting in delays in rehabilitation and patient dissatisfaction.

The availability of a noninvasive clinical indicator of implant bone loss following stage I surgery would be a valuable adjunct to overall patient care. Early identification of such integration complications might permit surgical or other intervention to minimize osseous tissue damage and limit overall delay in implant treatment. In a review of 5-year results following the placement of 772 hydroxyapatite-coated implants, the exposure of an implant through the mucosa as noted clinically after stage I but prior to stage II surgery appeared qualitatively to be associated with an increased incidence of crestal bone loss.[6] The following study was undertaken to further evaluate the relationship between implant exposure and the level of bone loss noted at stage II implant uncovering.

Materials and Methods

Data were collected from 50 subjects currently participating in an ongoing 5-year prospective trial designed to assess the long-term efficacy of press-fit hydroxyapatite-coated endosseous implants (Omniloc Implants, Sulzer Calcitek, Carlsbad, CA) in dental rehabilitation. Subjects were enrolled from a population of patients under care at the University of Chicago, Zoller Memorial Dental Clinics, using a protocol approved by the university's institutional review board. Informed written consent was obtained from each subject. A total of 275 implants was placed in this subject population (mean 5.5 implants per subject; range, 2 to 8 implants per subject). All implants were placed into maxillae following a commonly accepted 2-stage surgical protocol. After placement but prior to closure of the soft tissue flap, the crestal bone height relative to the top of each implant was recorded to the nearest millimeter using a hand-held periodontal probe. A score of 0 was assigned if the implant was placed level with the prepared osseous tissue bed.

Bone density was assessed qualitatively at this time, based on surgical findings at implant placement, and graded on a scale from 1 to 3, with a score of 1 representing the densest bone and 3 representing the least dense bone encountered. Three unplanned surgical events were prospectively selected to ascertain possible correlations with postimplantation complications. These were sinus perforation, crestal bone dehiscence, and buccopalatal bony plate fenestration. Sinus perforations were surgically managed by obtaining primary closure of the wound over the implant. Bony dehiscences or fenestrations were managed through the use of a combination of autologous and allograft material. In addition, barrier membranes were employed in 5 subjects (1 membrane per subject). The decision to use these materials was made for each subject at the time of surgery so as to maximize clinical outcomes based on clinical judgment. The occurrence of these events was graded as either present or absent.

Subjects were examined 1, 2, and 4 weeks postoperatively to assess the adequacy of healing. Where indicated, previously fabricated temporary removable prostheses were lined with tissue conditioner and placed no sooner than 1 week after surgical implantation so as not to disrupt healing. Instructions for prosthesis use included the maintenance of a soft diet and the removal of the prosthesis while sleeping. Prosthetic follow-up was then maintained on a 4- to 6-week basis throughout the treatment period, at which time soft tissue health at the surgical sites was monitored and revisions of the prostheses were performed as indicated.

At stage II implant uncovering, all subjects were clinically examined, and any sites of oral mucosal dehiscence with implant exposure were identified and recorded (Fig 1). The mucosa was then reflected, exposing the implants, and the crestal bone height relative to the top of each implant was again measured and recorded to the nearest millimeter using a hand-held periodontal probe (Fig 2). This measurement was made at the point of greatest change seen in the crestal bone. In this manner, bone loss was defined as a decrease of 2 mm or more in crestal height between stage I and stage II measurements. If the bone was found to be level with the implant or at a variance of no greater than 1 mm between stage I and stage II measurements, a score of 0 was assigned.

Data summaries were generated for all implants combined, as well as by patient. Statistical analyses were generated by patient to uphold the assumption of independence. Patients presenting with at least 1 implant exposed to the oral cavity were defined as "exposed." Bone loss and surgical complications were defined in a similar manner. The highest bone density value obtained for all implants was retained for each patient. Univariate analyses using chi-square and Fisher exact tests were conducted to independently determine the association between bone loss and each variable.

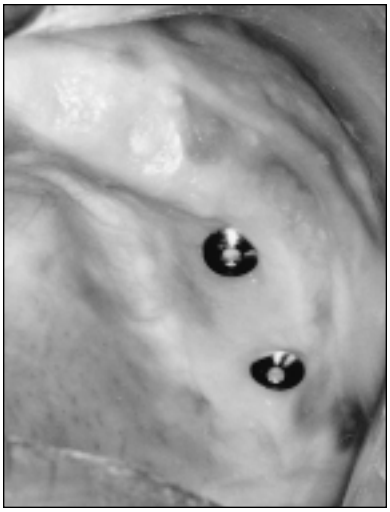

Fig 1 Oral exposure of implants, as noted immediately prior to stage II implant uncovering.

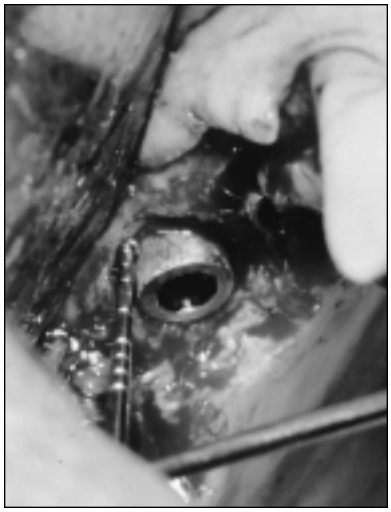

Fig 2 Crestal bone height measurement relative to implant, using hand-held periodontal probe, during stage II implant uncovering.

Results

The mean healing period between stage I implantation and stage II implant uncovering was 7.2 months (SD 1.5 months; range, 4.7 to 13.1 months). All 275 implants were found to be integrated at stage II surgery, based on the lack of clinical mobility as determined by tightening of the soft tissue healing abutment with finger pressure.

A total of 14 implants in 7 patients was identified as having become exposed to the oral cavity through the mucosa at the time of the stage II procedure. The overall mean crestal bone loss for all implants was found to be 0.55 mm (SD ± 1.23), with a mean bone loss of 2.71 mm (SD ± 1.78) for exposed implants and 0.43 mm (SD ± 1.08) for nonexposed implants. No evidence of clinical pathology was noted as exposed sites, and all subjects were asymptomatic. No implant exposures occurred at sites where barrier membranes were employed. No etiology for implant exposure was identified. Changes in bone heights as measured at stage I and stage II surgeries for all exposed sites are listed in Table 1.

Twenty-three percent of implants having a bone density of 3 (least dense) experienced bone loss, whereas 17% of implants with a bone density of 1 or 2 experienced bone loss. Six percent of implants experiencing surgical complications were noted to have bone loss; 22% of implants without identified complications experienced bone loss.

Since some patients experienced multiple implant exposures, the data were then examined on a per-patient basis (Tables 2a to 2f). A statistically significant association was found between patients experiencing implant exposure and those experiencing bone loss (Fisher exact test, P = .0003). Exposed patients were noted to have a risk of developing bone loss 3.9 times greater than unexposed patients (95% confidence interval between 2.3 and 6.5). A statistically significant inverse relationship was found between surgical complications and bone loss (chi-square = 5.824, P = .016). The odds of encountering bone loss were seen to decrease by a factor of 0.2 for patients with reported unanticipated surgical events, as compared to patients who experienced no unanticipated events. No statistically significant relationship was found between bone density and bone loss.

Discussion

The results of this study demonstrated a statistically significant relationship between implant exposure through the oral mucosa between stage I and stage II surgeries and an increased risk for crestal bone loss in the maxilla as discovered at implant uncovering for the subject population under review. The relative risk for bone loss increased by a factor of 3.9 in patients presenting with exposed implants at stage II uncovering, as

Table 1 Comparison of Stage I and Stage II Crestal Bone Levels for All Dehisced Sites

		Implant position above/below crestal bone		
Subject	Site	Stage I (placement)	Stage II (uncovering)	Total change (mm)
M.A.	Maxillary right first premolar	0 mm (level)	2 mm above	–2
	Maxillary left central incisor	0 mm (level)	5 mm above	–5
	Maxillary left second premolar	0 mm (level)	4 mm above	–4
L.H.	Maxillary right first molar	1 mm above	2 mm above	–1
	Maxillary left first premolar	1 mm below	1 mm above	–2
J. J.	Maxillary left canine	1 mm below	1 mm above	–2
D.M	Maxillary right lateral incisor	1 mm below	2 mm above	–3
	Maxillary left canine	1 mm below	2 mm above	–3
	Maxillary left second premolar	1 mm below	1 mm above	–2
H.B.	Maxillary right lateral incisor	1 mm below	4 mm above	–5
	Maxillary left canine	1 mm below	1 mm below	0
L.R.	Maxillary right first premolar	1 mm below	2 mm above	–3
	Maxillary left canine	1 mm below	4 mm above	–5

compared to those individuals who did not experience implant exposure throughout the healing period. This finding has implications for patient management between stage I and stage II surgeries. Following implant placement, asymptomatic patients are typically discharged after 1 postoperative examination and do not return until implant uncovering. If excessive bone loss is first identified at this late stage, then significant additional surgery, with resubmergence of the implant, may be required. Extreme bone loss may result in removal of the implant. Using exposure of the implant as a noninvasive clinical indicator of potential bone loss might permit more conservative early intervention surgery to prevent or limit crestal bone loss and related implant complications.

While the determination of the types of surgery that would be most appropriate in this situation needs further elucidation, effective treatment options for the general management of bony defects associated with implants have been described elsewhere.[7] Further study is needed to determine how to apply these surgical procedures when implant exposures occur so as to limit bone loss around the implants and avoid complications resulting from hard and soft tissue manipulation. Such procedures could include enlargement of the exposed site, both to establish a healing environment, similar to that used in single-stage implant placement, and to permit easy access for hygiene. Alternatively, a soft tissue flap could be raised to reclose the dehisced mucosal site, with the inclusion of graft and/or barrier membrane materials if early crestal bone clefting is detected. Finally, more frequent follow-up between stage I and stage II surgeries would be also required to identify and address implant exposure at an early stage.

Bone loss was found not to be associated with bone density for both exposed and nonexposed implant subsets. These findings were not predicted or consistent with other reports.[5] It is commonly accepted that the risk for integration complications and/or failures is higher in the maxilla and posterior mandible than in the symphyseal area of the mandible. Lack of stabilizing cortical bone and overall bony volume, as well as

Table 2a Implant Bone Loss Versus Implant Exposure

	Bone loss		
	Yes	No	Total
Exposed	11	3	14
Not exposed	39	222	261
Total	50	225	275

Table 2b Patient Bone Loss Versus Implant Exposure

	Bone loss		
	Yes	No	Total
Exposed	7	0	7
Not exposed	11	32	43
Total	18	32	50

Table 2c Implant Bone Loss Versus Bone Density

	Bone density			
	1	2	3	Total
Bone loss	15	24	11	50
No bone loss	71	118	36	225
Total	86	142	47	275

Table 2d Patient Bone Loss Versus Bone Density

	Bone density			
	1	2	3	Total
Bone loss	1	6	11	18
No bone loss	4	18	10	32
Total	5	24	21	50

Table 2e Implant Bone Loss Versus Surgical Complications

	Complications		
	Yes	No	Total
Bone loss	4	46	50
No bone loss	63	162	225
Total	67	208	275

Table 2f Patient Bone Loss Versus Surgical Complications

	Complications		
	Yes	No	Total
Bone loss	8	10	18
No bone loss	25	7	32
Total	33	17	50

decreased trabecular density, have been implicated.[8] One might then predict that the risk of crestal bone loss would increase at implant sites with low bony density, when compounded with the risk elicited by implant exposure to the oral cavity. In the current study, only the maxilla was involved, and few exposed implants were seen at stage II implant uncovering. Findings based on a narrow range of conditions make comparisons to reports that included more broad-based data difficult. Further studies appear to be needed to fully assess the potential correlation between bone density and the risk for crestal bone loss following implant exposure.

An inverse relationship was found to exist between unanticipated surgical events and crestal bone loss. The identification of unanticipated crestal bone defects and penetration of the bony floor of the maxillary sinus during implant surgery was prospectively selected in an attempt to identify potential correlates for implant failure within the broader parameters of a 5-year trial designed to evaluate the long-term efficacy of hydroxyapatite-coated implants. In this study, unanticipated crestal bone defects, when encountered, were routinely treated with the placement of a combination of autologous and allograft material, with or without the use of a barrier membrane. This interceptive treatment was performed to enhance bony fill while excluding epithelial and connective tissue at the defect site.[4] No exposure of implants through the oral mucosa occurred at sites when barrier membranes were employed. The inverse effect noted might be an indicator of the effectiveness of the treatment. Penetration of the bony floor of the maxillary sinus during surgery, a common occurrence in implant treatment, may have no impact on crestal bone loss if the implant is well immobilized within the prepared osseous bed.[9] These results suggest that the unanticipated discovery of crestal bone defects and penetration of the sinus floor during implant placement may not represent significant surgical complications, but rather a variant treatment finding that can be effectively managed during surgery without complications.

Conclusion

A direct positive relationship was found between exposure of the implant through the oral mucosa and the occurrence of crestal bone loss in the maxilla at stage II implant uncovering in the patient population studied. This finding has implications for the management of patients between stage I and stage II surgeries, with respect to the frequency of follow-up and the indication for interceptive treatment to limit bone loss and prevent delay of rehabilitation. No relationship was found between the bone density at the site for implantation and the level of bone loss at implant uncovering, a finding that requires further evaluation. An inverse relationship was noted between the incidence of unanticipated surgical events and the level of crestal bone loss. The nature of the event and the surgical response at the time of implantation may account for this finding.

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