Influence of Early Cover Screw Exposure on Crestal Bone Loss Around Implants: Intraindividual Comparison of Bone Level at Exposed and Non-Exposed Implants

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Background: The objective of this study was to evaluate the influence of early exposure of the cover screw on crestal bone loss around implants.

Methods: The study population consisted of 19 patients who were treated with dental implants placed using a two-stage surgical protocol and had early exposed and non-exposed implants (20 early exposed and 20 non-exposed implants). The crestal bone loss at exposed and non-exposed implants in each patient was evaluated with a periapical radiograph taken at the first surgery and at suprastructure insertion using a computerized image-analysis system. The bone loss was compared using the Wilcoxon signed-rank test.

Results: The mean crestal bone loss at exposed implants was 0.40 ± 0.53 mm, and it was 0.18 ± 0.26 mm at non-exposed implants. The Wilcoxon signed-rank test revealed a statistically significant difference in crestal bone loss between exposed and non-exposed implants in the same patient ($P = 0.02$).

Conclusions: The early exposure of the cover screw that results in breakdown of the mucosal seal seems to accelerate early peri-implant crestal bone loss. Periodic follow-up after the first surgery may be critical for minimizing the influence of early exposure. *J Periodontol 2009;80:933-939.*

KEY WORDS

Alveolar bone loss; dental implants; infection/prevention and control; osseointegration; plaque; wound healing.

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The peri-implant mucosa has many features in common with gingival tissues; it establishes a cuff-like barrier, and the junctional epithelium adheres to the implant surface through a basal lamina and hemidesmosomes.[1,2] The dimension of the junctional epithelium is ~2 mm in the apico-coronal direction, and the zone of connective tissue attachment is ~1 mm high. Once the implant is exposed to the oral environment and is functioning, a mucosal attachment of a certain minimal dimension is required to protect osseointegration.[3]

Loss of supporting bone can occur between the first and second surgeries, and it only becomes clinically apparent when the fixture is uncovered. Numerous factors may contribute to the early bone loss; however, many researchers[4-6] believe that sealing the communication between the implant and the oral cavity early in the development of osseointegration is crucial for its success. On the contrary, other investigators[7-10] reported that peri-implant soft and hard tissues of intentionally non-submerged (one-stage protocol) implants have similar dimensions and composition as those of submerged (two-stage protocol) implants; these studies included a strict plaque-control program.

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Adell et al.[11] observed early exposure of the mucosa in 4.6% of their treated patients, consisting of 304 fixtures in 22 upper and 24 lower jaws. Toljanic et al.[12] and Tal[13] reported 5.1% and 13.7% exposure rates, respectively. In histopathologic examinations of perforated soft tissue specimens, hyperplastic epithelium showed a gradual tendency toward invagination closer to the exposed site. A space that formed between the epithelial margins induced a direct communication between the implant cover screw and the oral cavity.[14] The breach between the perforated mucosa and the cover screw is an ideal place for plaque to accumulate and for bacteria to colonize during osseointegration.[13] If plaque accumulates on the implant surface, the subepithelial connective tissue becomes infiltrated with inflammatory cells.[15,16] When the apical migration of the plaque front continues, clinical and radiographic signs of tissue destruction are seen around the implants.[16,17]

The aim of this study was to evaluate the influence of spontaneous early exposure of the cover screw on crestal bone loss around implants.

MATERIALS AND METHODS

This study was approved by the Institutional Review Board of Yonsei University. The patients were informed of the study procedures, and all provided written informed consent.

Patients

The study subjects were selected from 355 patients who received two-part implants using a two-stage surgical protocol at the Department of Periodontology, Gangnam Severance Hospital, between August 2000 and August 2007. Seven hundred eighty-one implants were placed; the patients had good general health at the time of the implant procedure.

Twenty-eight implants in 24 patients were spontaneously exposed to the oral cavity before uncovering surgery. Among the 24 patients, those who also had a non-exposed implant were selected for the intraindividual comparison of crestal bone loss between the exposed and non-exposed implants.

Fourteen males and five females with a mean age of 54.4 years (range, 34 to 82 years) were included in the present study. One of the patients had two exposed and two non-exposed implants. As a result, 20 exposed and 20 non-exposed implants in 19 patients were evaluated (Table 1).

Procedures

Treatment procedure. The threaded conical implants[§] were placed following a two-stage surgical protocol. The first surgery was performed ≥3 months after tooth extraction, when the intact healing state of the extraction socket was confirmed clinically and radiographically. The top of the fixture was inserted at or below the marginal bone level. However, small variations in insertion depth occurred depending on the anatomy of the crest. All implants were inserted without guided bone regeneration or any other augmentation procedure.

After surgery, patients were instructed to avoid brushing the surgical site and to use antiseptic rinse[‖] (twice a day) for 10 days. A cold, soft diet was recommended for 2 days, and smokers were asked to avoid smoking for 7 days postoperatively.

The sutures were removed after 10 days. The patients were checked 1, 3, and 7 weeks after suture removal and 1 week before the second surgery.

The second surgery was performed after a healing period of 3 months in the mandible and 6 months in the maxilla; the suprastructure was inserted 3 to 4 weeks after the second surgery.

An uncovering surgery was performed immediately when cover screw exposure through the oral mucosa was observed between the first and second surgeries. A crestal mini-incision and local undermining in the gingiva at the center of the implant site were performed without flap elevation when placing the healing abutment after cover screw removal. Patients were asked to perform strict plaque control around the healing abutments (Fig. 1). The suprastructure was inserted after a proper healing period: 3 months in the mandible and 6 months in the maxilla after implant insertion.

Radiographs and evaluation. Standardized periapical intraoral radiographs were taken at the first surgery and at suprastructure insertion by a paralleling technique using films[¶] and a computed dental radiography digital sensor.[#]

The films were digitized using a digital scanner[**] at an input resolution of 2,400 dots per inch with 256 gray scales. Digital images were converted to the tagged image file format (tiff) by a picture archiving and communicating system.[††] All files were transferred to a personal computer[‡‡] and examined using the same monitor,[§§] which was set to a resolution of 1,024 × 768 pixels.[18]

The radiographs of exposed and non-exposed implants were evaluated for the distance between the implant shoulder and the bone/implant contact point at the mesial and distal surfaces using a computerized image-analysis system,[‖‖] and the average value was

§ Astra Tech implants, Astra Tech, Mölndal, Sweden.
‖ Freshburst Listerine, Pfizer Consumer Healthcare, Walton-on-the-Hill, Surrey, U.K.
¶ Kodak Insight F-speed film, Eastman Kodak, Rochester, NY.
# Schick Technologies, Long Island City, NY.
** EPSON GT-12000, EPSON, Nagano, Japan.
†† PiViewSTAR, Infinitt, Seoul, Korea.
‡‡ Processor, Intel Celeron D, Intel, Santa Clara, CA; operating system, Windows XP Professional 2002, Microsoft, Redmond, WA.
§§ Flatron 775FT Plus, LG, Seoul, Korea.
‖‖ Adobe Photoshop 7.0, Adobe Systems, San Jose, CA.

Table 1.

Overview of Data

				Exposed Implants		Non-Exposed Implants	
Patient	Gender	Age* (years)	Smoker	Tooth #	Bone Loss (mm)	Tooth #	Bone Loss (mm)
1	Female	67	No	30	0.19	19	0.05
2	Male	52	No	30	0.51	3	0.23
3	Male	33	No	19	–0.15	31	0.00
4	Female	61	No	29	1.28	30	0.76
5	Male	51	Yes	14	0.15	19	0.00
6	Male	57	No	20	2.15	29	0.19
7	Male	42	No	3	0.05	14	0.05
8	Male	47	No	18	0.42	31	0.05
9	Male	82	No	23	0.16	26	0.85
10	Male	41	No	14	0.03	15	–0.03
11	Male	40	No	18	0.48	21	0.34
12	Male	64	No	19	0.00	18	0.00
13	Male	53	Yes	3	0.05	14	0.14
14	Female	56	No	3	0.42	12	0.14
15	Male	67	No	2	0.77	3	0.60
16	Female	40	No	8	0.00	30	0.08
17	Male	54	No	5	0.00	12	0.00
18	Male	50	No	19	0.52	30	0.09
				18	0.33	3	0.00
19	Female	60	No	4	0.65	3	0.00
Mean					0.40		0.18
SD					0.53		0.26
Median					0.26		0.07
P value (Wilcoxon signed-rank test)						0.02	

* Age at the time of the first surgery.

obtained. Crestal bone loss, calculated as the difference between the distance at the first surgery and suprastructure insertion, was determined (Figs. 2 and 3). The measurements were made to the nearest 0.01 mm.

The measurements were done by a single operator (TK). Prior to taking part in the present investigation, intraobserver variability was tested under the supervision of the director (ISM). The bone loss in 40 periapical films with exposed and non-exposed implants that were selected arbitrarily for the calibration of intraobserver variability was measured twice, with an interval of 1 week between measurements. The Wilcoxon test was used to assess the statistical significance of differences between the first and second measurements. Pearson correlation coefficients were calculated to examine the relationship between the two measurements. The Wilcoxon test revealed no significant difference between the first and second readings. Also, correlation of the two measurements

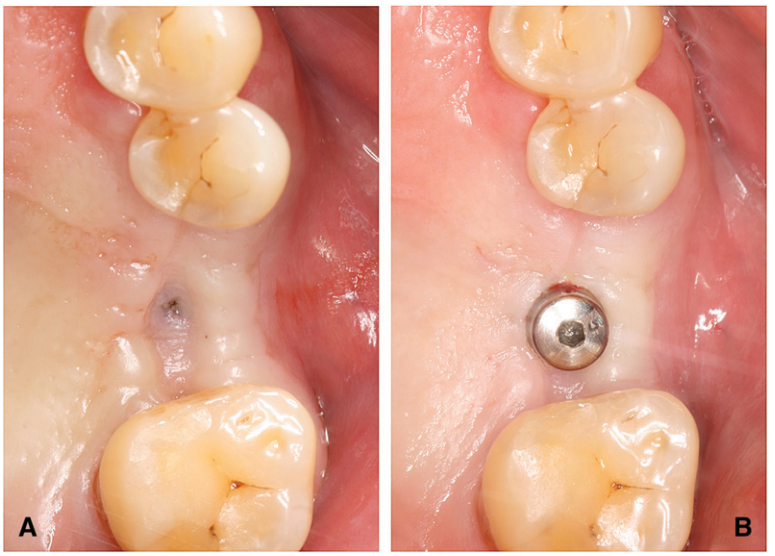

Figure 1.
Clinical features of an exposed implant. ***A)*** *Exposed cover screw.* ***B)*** *After treatment of the exposure, a healing abutment connection was created.*

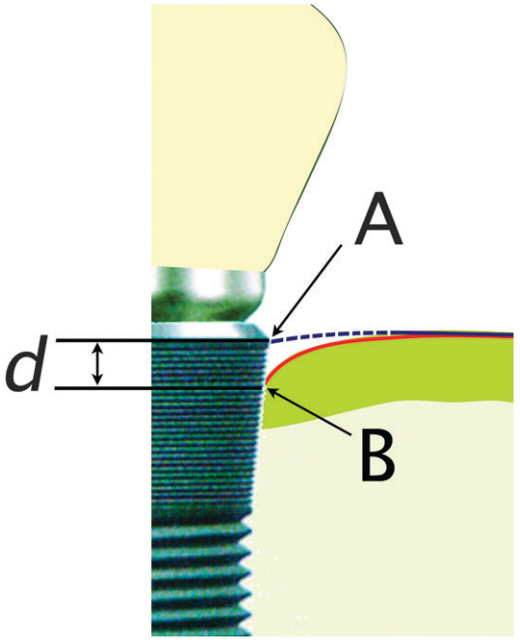

Figure 2.
Schematic presentation of measurements. A = bone/implant contact point at the first surgery. B = bone/implant contact point at suprastructure insertion. d = crestal bone loss, i.e., the difference between the marginal bone level at the first surgery and at suprastructure insertion.

was significant (Pearson correlation coefficient = 0.99; P <0.01). The intraobserver variability and correlation coefficient were comparable to previous studies.[19,20]

Statistics

The null hypothesis was that there would be no difference between the amounts of crestal bone loss in the exposed versus the non-exposed implants.

Mean values and ranges were calculated for the two groups (exposed and non-exposed implants in the same patient). The normality of the distribution was tested with the Kolmogorov-Smirnov test. Data analysis for marginal bone loss was performed by applying the Wilcoxon signed-rank test when the distribution was normal. A P value of 0.05 was considered statistically significant.¶¶

RESULTS

The mean crestal bone loss of the exposed implants was 0.40 ± 0.53 mm (range: –0.15 to 2.15 mm). The mean crestal bone loss in the non-exposed implants was 0.18 ± 0.26 mm (range: –0.03 to 0.85 mm).

The Kolmogorov-Smirnov test showed that the distribution was normal. There was a statistically significant difference in the crestal bone loss between the exposed and non-exposed implants (Wilcoxon signed-rank test; P = 0.02).

DISCUSSION

In the present study, there was a statistically significant difference in the crestal bone loss between exposed and non-exposed implants (P = 0.02), which might be due mainly to plaque accumulation.

Adell et al.[11] believed that isolation of a submerged implant from the oral environment by primary closure was an important factor for successful osseointegration. The investigators proposed that active surgical measures be taken, with excision of the bordering gingiva and full flap coverage of the perforated site when early exposure of the cover screw was observed.[11] Barboza et al.[21] stated that the space between the cover screw and the overlying mucosa formed by spontaneous early exposure is an ideal area for the accumulation of food debris and bacterial growth, and it is very difficult for patients to perform adequate oral hygiene procedures in these areas. Continuous plaque formation during the postoperative period after cover screw exposure may result in tissue destruction around the implants.[16,17] Tal et al.[22] described the

¶¶ SPSS for Windows, release 13.0, SPSS, Chicago, IL.

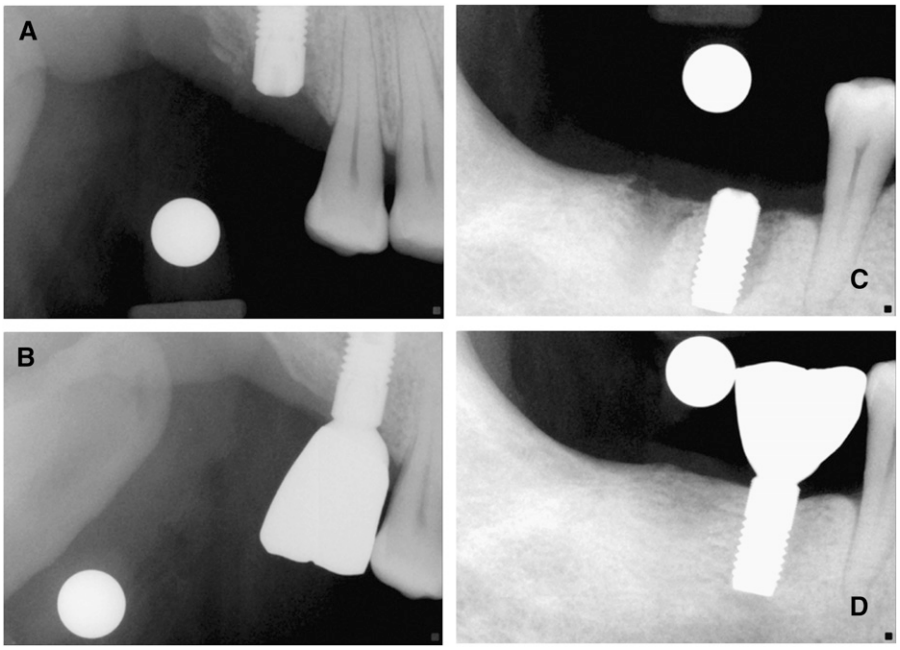

Figure 3.
*Periapical radiographs of a patient who had exposed and non-exposed implants. Non-exposed implant at the first surgery **(A)** and at suprastructure insertion **(B)**. Exposed implant at the first surgery **(C)** and at suprastructure insertion **(D)**.*

pathologic structure of the perforated lesions as a plaque-retentive site that could increase bone loss. Toljanic et al.[12] demonstrated a statistically significant relationship between implant exposure through the oral mucosa between the first and the second surgeries and an increased risk for crestal bone loss.

A two-part implant is commonly inserted using a two-stage surgical procedure. Some studies compared the radiographic bone loss between two-part implants followed by a two-stage surgical protocol and those followed by one-stage surgical protocol and including a plaque-control program. Abrahamsson et al.[9] concluded that radiographic bone loss was 0.4 mm for the submerged group and 0.3 mm for the non-submerged group. Ericsson et al.[23] and Collaert and De Bruyn[24] also reported that there were no statistically significant differences between these treatment modalities.

The difference in crestal bone loss between intentionally non-submerged implants under a meticulous plaque-control program and submerged implants was not statistically significant.[9,23,24] If the early exposure can be detected immediately, and patients are instructed to perform oral hygiene procedures around the exposed implants after the uncovering surgery, it was assumed in the present study that there would be no difference in the crestal bone loss between the exposed and non-exposed implants. However, there was a considerable period of time between exposure of the cover screw and the uncovering surgery, which would allow plaque accumulation, leading to statistically significant differences in crestal bone loss between the exposed and non-exposed implants. It may be critical to identify early exposure immediately.

Van Assche et al.[25] compared the early marginal bone level changes between the two-stage exposed and non-exposed groups. They found that the bone loss in the exposed group was significantly greater (1.96 mm). The mean bone loss was greater than that of exposed implants in this study (0.49 mm). The difference may be due to the treatment modality for the cover screw exposure; there was no intervention after the diagnosis of perforation in the study by Van Assche et al.,[25] unlike in this study.

It is recommended to excise the migrated epithelium of the perforated mucosa as soon as possible and to connect the healing abutments after cover screw removal for the treatment of spontaneous early exposure because it allows for better hygiene and minimizes the risk for infection.[12,14,26,27] During healing of the wound in the soft tissue, an attachment is formed between the mucosa and the healing abutment. After it is properly matured, this attachment effectively reestablishes the soft tissue barrier and separates the bony tissue from the oral cavity.[28,29] Our protocol was in accordance with this treatment modality.

A limitation of the present study is that it was unable to identify the exact time of exposure, thus making it impossible to factor the plaque-accumulation period into the statistical analysis. It was difficult to detect early exposure of the cover screw immediately because patients were commonly asymptomatic. The exposure (plaque accumulation) period, the time left untreated, should be determined to clarify the relationship between plaque accumulation and the occurrence of crestal bone loss.

Within the limits of a human study, there are ethical considerations involved in creating early exposure of the cover screw by factitious manipulation, and the influence of spontaneous early exposure of the cover screw on crestal bone can only be studied radiographically or biometrically.[14] A further investigation with animals treated with intentionally exposed implants may be required to evaluate the relationship between the exposure period without any intervention and early crestal bone loss.

CONCLUSIONS

The aim of this study was to evaluate the influence of spontaneous early exposure of the cover screw on crestal bone loss around the implant.

The mean crestal bone loss of exposed implants was 0.40 ± 0.53 mm (range, –0.15 to 2.15 mm). The mean crestal bone loss in non-exposed implants was 0.18 ± 0.26 mm (range, –0.03 to 0.85 mm). There was a statistically significant difference in the crestal bone loss between exposed and non-exposed implants (Wilcoxon signed-rank test; $P = 0.02$).

Early exposure of the cover screw that results in breakdown of the mucosal seal seems to accelerate peri-implant crestal bone loss. Periodic follow-up after the first surgery may be critical for minimizing the influence of early exposure.

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REFERENCES

1. 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.
2. McKinney RV Jr., Steflik DE, Koth DL. Evidence for a junctional epithelial attachment to ceramic dental implants, a transmission electron microscopy study. *J Periodontol* 1985;56:579-591.
3. Berglundh T, Lindhe J. Dimension of the periimplant mucosa. Biological width revisited. *J Clin Periodontol* 1996;23:971-973.
4. Bränemark PI, Adell R, Breine U, Hansson BO, Lindström J, Ohlsson A. Intra-osseous anchorage of dental prostheses. I. Experimental studies. *Scand J Plast Reconstr Surg* 1969;3:81-100.
5. Adell R, Lekholm U, Bränemark PI. Surgical procedures. In: Bränemark PI, Zarb GA, Albrektsson T, eds. *Tissue Integrated Prosthesis*. Chicago: Quintessence; 1985:223-225.
6. Albrektsson T. Bone tissue response. In: Bränemark PI, Zarb GA, Albrektsson T, eds. *Tissue Integrated Prostheses*. Chicago: Quintessence; 1985:129-143.
7. Buser D, Weber HP, Donath K, Fiorellini JP, Paquette DW, Williams RC. Soft tissue reactions to non-submerged unloaded titanium implants in beagle dogs. *J Periodontol* 1992;63:225-235.
8. Abrahamsson I, Berglundh T, Wennström 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.
9. Abrahamsson I, Berglundh T, Moon IS, Lindhe J. Peri-implant tissues at submerged and non-submerged titanium implants. *J Clin Periodontol* 1999;26:600-607.
10. Cochran DL, Hermann JS, Schenk RK, Higginbottom FL, Buser D. Biologic width around titanium implants. A histometric analysis of the implanto-gingival junction around unloaded and loaded nonsubmerged implants in the canine mandible. *J Periodontol* 1997;68:186-198.
11. Adell R, Lekholm U, Rockler B, Brånemark PI. A 15-year study of osseointegrated implants in the treatment of the edentulous jaw. *Int J Oral Surg* 1981;10:387-416.
12. Toljanic JA, Banakis ML, Willes LA, Graham L. Soft tissue exposure of endosseous implants between stage I and stage II surgery as a potential indicator of early crestal bone loss. *Int J Oral Maxillofac Implants* 1999;14:436-441.
13. Tal H. Spontaneous early exposure of submerged implants: I. Classification and clinical observations. *J Periodontol* 1999;70:213-219.
14. Tal H, Dayan D. Spontaneous early exposure of submerged implants: III. Histopathology of perforated mucosa covering submerged implants. *J Periodontol* 2000;71:1231-1235.
15. Ericsson I, Berglundh T, Marinello CP, Liljenberg B, Lindhe J. Long-standing plaque and gingivitis at implants and teeth in the dog. *Clin Oral Implants Res* 1992;3:99-103.
16. 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.
17. Marinello CP, Berglundh T, Ericsson I, Klinge B, Glantz PO, Lindhe J. Resolution of ligature-induced peri-implantitis lesions in the dog. *J Clin Periodontol* 1995;22:475-479.
18. Lee DW, Choi YS, Park KH, Kim CS, Moon IS. Effect of microthread on the maintenance of marginal bone level: A 3-year prospective study. *Clin Oral Implants Res* 2007;18:465-470.
19. Webber RL, Ruttimann UE, Heaven TJ. Calibration errors in digital subtraction radiography. *J Periodontal Res* 1990;25:268-275.
20. Wyatt CCL, Bryant SR, Avivi-Arbjer L, Chaytor DV, Zarb GA. A computer-assisted measurement technique to assess bone proximal to oral implants on intraoral radiographs. *Clin Oral Implants Res* 2001;12:225-229.
21. Barboza EP, Caúla AL, Carvalho WR. Crestal bone loss around submerged and exposed unloaded dental implants: A radiographic and microbiological descriptive study. *Implant Dent* 2002;11:162-169.
22. Tal H, Artzi Z, Moses O, Nemcovsky CE, Kozlovsky A. Spontaneous early exposure of submerged endosseous implants resulting in crestal bone loss: A clinical evaluation between stage I and stage II surgery. *Int J Oral Maxillofac Implants* 2001;16:514-521.
23. Ericsson I, Randow K, Glantz PO, Lindhe J, Nilner K. Clinical and radiographical features of submerged and nonsubmerged titanium implants. *Clin Oral Implants Res* 1994;5:185-189.
24. Collaert B, De Bruyn H. Comparison of Brånemark fixture integration and short-term survival using one-stage or two-stage surgery in completely and partially edentulous mandibles. *Clin Oral Implants Res* 1998;9:131-135.
25. Van Assche N, Collaert B, Coucke W, Quirynen M. Correlation between early perforation of cover screws

and marginal bone loss: A retrospective study. *J Clin Periodontol* 2008;35:76-79.
26. Rosenquist B, Grenthe B. Immediate placement of implants into extraction sockets: Implant survival. *Int J Oral Maxillofac Implants* 1996;11:205-209.
27. Barboza EP, Caúla AL. Diagnoses, clinical classification, and proposed treatment of spontaneous early exposure of submerged implants. *Implant Dent* 2002;11: 331-337.
28. Berglundh T, Lindhe J, Ericsson I, Marinello CP, Liljenberg B, Thomsen P. The soft tissue barrier at implants and teeth. *Clin Oral Implants Res* 1991;2:81-90.
29. Moon IS, Berglundh T, Abrahamsson I, Linder E, Lindhe J. The barrier between the keratinized mucosa and the dental implant. An experimental study in the dog. *J Clin Periodontol* 1999;26:658-663.

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