

The Association between the Frequency and Severity of Cover Screw Exposure in Dental Implants and Some Effective Factors

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Article Type	ABSTRACT
Research Paper	Background and Objective: Early exposure of the cover screw between two implant placement stages is a common problem that can impair the initial healing and success of the implant treatment. The aim of this study was to investigate the relationship between the frequency and severity of dental implant cover screw exposure and some effective factors. Methods: In this cross-sectional study, 159 implant units were examined in 35 patients who referred to Yazd Dental School for the second stage of implant placement in 2022-2023 after a 3-6-month initial healing period. Before the second stage, the area was examined for exposure severity based on Tal classification, and then the relationship between this event and the variables of gender, presence of systemic disease, keratinized gingiva width, thickness of the covering tissue on the buccal side, location and type of edentulism, time interval from extraction to replacement, and bone regeneration were examined. Findings: Of the 159 implants, 18 had spontaneous exposure. The most common exposure in terms of severity was class 4. There was a significant association between exposure and male gender ($p=0.009$), systemic disease ($p=0.041$), buccal overlying tissue thickness (3.00 ± 1.17 vs. 0) ($p<0.001$), and keratinized gingiva width (2.16 ± 1.03 vs. 2.82 ± 1.30) ($p=0.042$). No significant association was found between exposure and other selected factors. Conclusion: The results of this study showed that the severity of cover screw exposure is related to factors such as systemic diseases, keratinized gingiva width, and buccal tissue thickness. Keywords: <i>Dental Implant, Cover Screw, Attached Gingiva.</i>
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Introduction

When a tooth is lost, it is often replaced with an implant to facilitate chewing, speech, and aesthetic purposes (1, 2). Dental implant procedure is performed in a single-stage or two-stage procedure (3). Although the placement of dental implants using the single-stage and two-stage procedures both show similar results in terms of the size and thickness of the soft and hard tissues around the implant, the disadvantages of the single-stage procedure include the exposure of the implant to the oral environment and contamination of the surgical site. In the two-stage procedure, the implant is placed in the bone and covered with a flap to separate it from the oral environment and undergo the initial healing process under sterile, stress-free, and trauma-free conditions. After a few months, the implant is reopened and prosthetic treatment continues (4).

The success of dental implants depends on fundamental factors such as the ability of the implant structure to integrate with the surrounding tissues, which is influenced by several factors including implant materials, occlusion conditions, quality and quantity of the surrounding bone, and absence of early exposure (5). Factors such as personal characteristics, surgical technique, and implant design affect the initial healing of the bone and surrounding tissue around the implant (6). The longevity of dental implants generally depends on the potential ability of the surrounding bone to withstand the applied forces, but sometimes early exposure of the implant stimulates plaque accumulation, followed by marginal bone resorption, tissue inflammation, and impaired osseointegration (6). Perforation of the gingival tissue following implant placement and subsequent premature exposure of the cover screws between two-stage implant placements are common (7). Various factors may lead to the development of primary perforations, the most common cause of which is mechanical trauma and traction on the flap (8).

In a study by Mendoza et al., the presence of sufficient keratinized tissue at the implant site was found to be effective in protecting the implant from premature exposure (9). Hertel et al. reported the association between higher probability of cover screw exposure and male gender and posterior implant placement and believed that despite the importance of detecting these early exposures to the oral environment, this is often done late and the patient does not notice it.

Therefore, attempts to improve oral hygiene or therapeutic interventions do not occur at the right time and lead to bone loss around the implant. Therefore, timely diagnosis and identification of predisposing factors should be provided, and follow-up sessions should be arranged between the two stages of implant placement (10). Considering the importance of the issue of spontaneous cover screw exposure to the oral environment and its adverse effect on implant success, this study was designed and implemented with the aim of investigating the frequency and severity of spontaneous cover screw exposure after dental implant placement and identifying possible factors associated with its occurrence.

Methods

After obtaining informed consent and receiving the ethics code IR.SSU.DENTISTRY.REC.1401.094 from Yazd University of Medical Sciences, this cross-sectional study was conducted on all patients who had at least one dental implant in Yazd School of Dentistry in 2022-2023. This study was conducted on 35 patients with 159 dental implants. Patients with a history of having at least one UFII DIO implant unit (manufactured by DIO Implant System, South Korea), which was performed in two stages and at least 3 to 6 months after the first stage surgery were included in the study. Patients with a history of any oral mucosal lesion, alcohol, cigarette or drug use, and signs of implant failure such as looseness, active abscess or fistula were excluded from the study.

First, after referring to the Periodontics Department of Yazd Dental School and reviewing the patients' files, all their personal was recorded, which included age, gender, systemic diseases (diabetes, cardiovascular, hypothyroidism and hyperthyroidism, vesicular ulcer diseases, etc.), implant dimensions including regular (greater than 3.3 mm), narrow (between 3 and 3.3 mm), implant length (short: between 5-7 mm, long: between 10-14 mm), implant location in the jaw (anterior: implant in the location of incisor and canine teeth, posterior: implant in the location of premolar and molar teeth), type of edentulism (single-tooth/partial/complete), simultaneous use of guided reconstruction with bone substitutes, keratinized gingiva width and buccal tissue thickness, and placement time relative to tooth extraction time (immediate or delayed). Before starting the second stage of implant surgery, the target area was examined for the presence and severity of spontaneous exposure based on Tal classification (8) (Table 1).

Table 1. Classification of cover screw exposure based on Tal classification

Exposure class	Description
Class 0	The cover screw is completely covered by the soft tissue of the mucosa. Registration in this class means there is no exposure.
Class 1	There is a gap in the mucosa over the implant that can be detected using a periodontal probe, but the surface of the cover screw is not visible without mechanical intervention.
Class 2	The overlying mucosa is perforated and the cover screw is visible, but the boundaries of the perforation do not reach the outer perimeter of the cover screw at any point.
Class 3	The overlying mucosa is perforated and the screw cover is visible, and the boundaries of the perforation have reached the outer periphery of the screw cover in some places.
Class 4	The cover screw is fully visible and exposed to the oral environment. This class registration means the highest level of exposure.

During the second stage of surgery, before removal, the width of the attached gingiva and the thickness of the covering tissue on the buccal side of the cover screw were measured using a Williams periodontal probe. In this study, all patients used the same type of implant. In addition, the type of suture (simple), suture thread (4-0 Vicryl, Sutures Iran), and the type of postoperative medications prescribed (antibiotic amoxicillin 500 mg, Ibuprofen 400 mg, and chlorhexidine mouthwash 0.2%) were the same in all patients, and no temporary prosthesis was used to control occlusal trauma.

Implant and patient data were collected and samples were evaluated for the presence and severity of spontaneous cover screw exposure based on Tal classification. To examine the effect of different variables (assessment of risk factors associated with spontaneous cover screw exposure), SPSS version 21 and Chi-square test at the bivariate level and logistic regression test at the multivariate level were used, and $p < 0.05$ was considered significant.

Results

35 patients with a mean age of 52.74 ± 12.06 years (range 27-74 years) received 159 implants. Because all implants were regular in size and long in length, the two variables of implant size and length were excluded from the statistical analysis. The prevalence rate of spontaneous exposure was 25.7% in patients and 11.3% per implant.

Based on the demographic characteristics of the patients, the mean age of patients with at least one exposure was 55.55 ± 10.90 years and the mean age of patients without exposure was 51.77 ± 12.49 years, which was not statistically significant.

The frequency distribution of implant-related data such as the presence and severity of exposure based on its class, implant location in the jaws, type of implant based on extraction time, guided bone regeneration, average keratinized tissue width, and buccal mucosal thickness is shown in Table 2.

The highest frequency of exposure was class 4 and the lowest was class 1. The highest exposure was seen in men (44.4%), but in women only one case (9.5%) of exposure occurred, and this difference was statistically significant ($p < 0.05$) (Table 3).

The mean width of keratinized tissue in exposed implants (2.16 ± 1.03) was significantly less than that of unexposed implants with a keratinized tissue width of (2.82 ± 1.30) ($p = 0.042$). The mean thickness of buccal overlying mucosa in unexposed implants was 3.00 ± 1.17 , which was significantly greater than that of exposed implants, such that the mucosal thickness in all exposed implants was zero and there was virtually no overlying mucosa ($p < 0.001$), which was considered equivalent to exposure class 4 (Table 4).

By controlling for implant location in the binary logistic regression model, a significant negative association was observed between exposure and keratinized tissue width; meaning that for every unit increase in keratinized tissue width, the odds of exposure decreased by 0.47 ($p < 0.017$). By controlling for the width variable, the association between exposure and implant location was no longer statistically significant. However, the results of the odds ratio of exposure based on implant locations in the jaws are as follows: The odds of exposure in anterior maxillary implants were 3.76 times higher than in posterior mandibular implants ($p = 0.68$). Also, the odds of exposure in posterior maxillary implants were 1.95 times higher than in posterior mandibular implants ($p = 0.368$). On the other hand, anterior mandibular implants had 0.48 times less exposure than posterior mandibular implants.

Table 2. Frequency distribution of implant-related data

Variable	Number(%)
Exposure class	
Class 0	141(88.7)
Class 1	0(0)
Class 2	1(0.6)
Class 3	3(1.9)
Class 4	14(8.8)
Implant location	
Anterior maxilla	32(20.1)
Anterior mandible	39(24.5)
Posterior maxilla	49(30.8)
Posterior mandible	39(24.5)
Implant type based on extraction time	
Immediately	17(10.7)
Delayed	142(89.3)
Bone regeneration	
Yes	32(20.1)
No	127(79.9)

Table 3. Comparison of exposure frequency according to patient-related variables

Variable	At least one exposure		p-value*
	No Number(%)	Yes Number(%)	
Gender			
Male	10(55.6)	8(44.4)	0.009
Female	16(94.1)	1(5.9)	
Systemic disease			
Yes	17(65.4)	9(34.6)	0.041
No	9(100)	0(0)	
Edentulism type			
Single tooth	4(100)	0(0)	0.198
Partial	14(82.4)	3(17.6)	
Complete	8(57.1)	6(42.9)	

*Chi-square test

Table 4. Comparison of the frequency of exposure types based on implant-related variables

Variable	Exposure		p-value*
	No Number(%)	Yes Number(%)	
Implant location			
Anterior maxilla	25(78.1)	7(21.9)	0.197
Anterior mandible	37(94.9)	2(5.1)	
Posterior maxilla	44(89.8)	5(10.2)	
Posterior mandible	35(89.7)	4(10.3)	
Implant type based on extraction time			
Immediately	15(88.2)	2(11.8)	0.999
Delayed	126(88.7)	16(11.3)	
Bone regeneration			
Yes	30(98.8)	2 (6.3)	0.374
No	111(87.4)	16(12.6)	

*Chi-square test

Discussion

In the present study, there was a significant association between the occurrence and severity of exposure with male gender, history of systemic disease, thin buccal covering tissue thickness, and narrow keratinized gingiva width. Tal et al. reported impaired osteointegration and marginal bone loss following early cover screw exposure (11). However, there are limited studies on the underlying causes of this occurrence and it remains controversial (7). Toljanic et al. reported that they failed to identify causes related to exposure (12). Early and spontaneous exposure creates a site for plaque accumulation and bacterial growth, making it difficult for patients to perform oral hygiene measures in these cases. In addition, persistent plaque formation during the healing period may lead to tissue destruction around the implant (13). Therefore, prompt identification of early exposure may be crucial.

The present study reported a prevalence rate of spontaneous exposure of 25.7% in patients and 11.3% in implants, while in the study of Negahdari et al., it was 22.5% and 9.3% for patients and implants, respectively (7). However, Mendoza et al. reported a prevalence of 63%, which was significantly different from other studies (9). They believed that this higher prevalence could be due to the quality and type of suture, flap tension, and the presence of release flaps during the healing process.

In this study, the highest number of exposures, if any, was observed in class 4 according to the Tal classification, and there were no implants with class 1 exposure severity. However, in the study by Negahdari et al., spontaneous exposure of class 1 type with 7 implants was the most prevalent and class 3 with only one implant was the least prevalent (7). In the study by Mendoza et al., class 4 had the least prevalence and class 2 had the highest prevalence (9), which was inconsistent with our study. This difference in exposure may be due to different inclusion criteria in the aforementioned study. Mendoza et al. excluded patients from the study if they had systemic disease or did not have optimal oral hygiene, while in our study, having systemic problems was considered as a variable.

In the present study, the confounding effect of sutures used, type of postoperative medication, and use of temporary prosthesis were matched as much as possible. Since the implant surface is in direct contact with the bone, it can greatly affect the biological response and affect the mechanical strength of the interaction between the implant and the tissue, and play an important role in determining the fate of the implant. Therefore, in this study, an attempt was made to eliminate the effect of this variable as a confounder by considering only one type of implant (DIO). However, for example, in the study by Hertal et al., different types of implants were used (10).

In this study, there was a significant relationship between exposure and male gender, which may be due to women being more careful about maintaining oral hygiene. In the study of Hertal et al., the exposure rate was higher in men (10), which is consistent with our study. However, in the study of Negahdari et al., no significant difference was found between the two genders (7), which may be due to the same level of oral hygiene in male and female participants. In the present study, there was no statistically significant difference between the mean age of patients who had at least one exposure (55.55 ± 10.90) and patients who had not (51.77 ± 12.49), which is consistent with the study of Negahdari et al. (7).

A systematic review found a significant increase in peri-implant bone loss in diabetic patients compared with non-diabetic subjects (14). In this study, there was a significant association between systemic disease and the occurrence of exposure, but in the study by Negahdari et al., no significant association was found between diabetes and early cover screw exposure (7), which may be due to the fact that only four conditions were considered as systemic diseases in the study: diabetes, hypothyroidism, hypertension, or seizures.

Mendoza et al. examined the effect of fresh socket implant placement on exposure and found no significant relationship (9), which is consistent with the present study, which found that exposure rates were approximately the same in immediate and delayed placement. However, Negahdari et al. found that immediate implant placement had a significant effect on implant exposure (7), which could be related to the greater number of immediate implants that underwent bone regeneration. Mendoza et al. did not find a relationship between this variable and implant exposure (9), which is consistent with our study. A double-blind study by Cehreli et al. showed that immediate implants (mean time between extraction and placement was 40 days) were more likely to be exposed than delayed implants (15). In contrast, another study of 124 patients with 493 implants showed that the probability of exposure was significantly higher in implants placed more than three months apart (16), which was not consistent with the results reported by Cehreli et al. (15).

The effect of bone regeneration on spontaneous exposure was investigated in the present study, and the results indicated that although more exposures occurred in implants that did not undergo regeneration, there was no statistically significant relationship between regeneration and exposure, which was consistent with the studies of Negahdari et al. (7), Mendoza et al. (9), and Hertel et al. (10).

The highest exposure occurred in the anterior maxilla (21.9%) and the lowest exposure (5.1%) occurred in the anterior mandible, but there was no statistically significant relationship between implant location and exposure, which could be due to differences in the thickness of the overlying mucosa. There was a higher number of exposures in complete edentulism (42.9%) than in other types of edentulism, but no statistically significant relationship was observed, and implants placed as a replacement for only one tooth showed no exposure. Also, in the study of Negahdari et al. (7), no exposure was observed in single tooth replacement, which was consistent with the present study. This could be due to the greater amount of ridge resorption in complete edentulism or the greater protective effect of adjacent teeth against trauma in single tooth replacement compared to complete or partial edentulism.

In this study, the odds of exposure in anterior maxillary implants were 3.76 times higher than in posterior mandibular implants. Also, the odds of early exposure in posterior maxillary implants were 1.95 times higher than in posterior mandibular implants. On the other hand, the odds of exposure in anterior mandibular implants were 0.48 times lower than in posterior mandibular implants. In the study of Negahdari et al., no significant difference was found between the maxillary premolar region and other regions in the rate of exposure, and finally, logistic regression showed a higher chance of exposure in the maxilla than in the mandible (7). Moreover, the study of Hertel et al. indicated a higher rate of exposure in the posterior regions of the jaws (10), which could be due to the fact that most of the implants in that study were placed posteriorly.

The presence of sufficient keratinized gingiva around the implant is important during the loading and prosthesis placement phase. Some studies have reported excessive plaque accumulation and further soft tissue resorption in the absence of keratinized gingiva (17). Baqain et al. also considered the absence of keratinized gingiva to be a strong predictor of early implant failure before the loading phase (18). The results of the present study showed that the mean width of keratinized tissue in exposed implants was significantly less than that in non-exposed implants. The mean mucosal thickness in exposed implants was also significantly less than that in non-exposed implants, with mucosal thickness in all exposed implants being zero, which was somewhat predictable because thick periodontal tissue is more resistant to exposure than thin periodontal tissue. Also, by controlling for implant location in the logistic regression model, a significant negative association was observed between exposure and keratinized tissue width; This means that for every one unit increase in the width of keratinized tissue, the chance of exposure decreases by 0.47. The results of the study showed that the severity of cover screw exposure is related to factors such as systemic diseases, keratinized gingiva width, and tissue thickness in the buccal region.

Despite the limitations of the present study, a significant relationship was found between spontaneous exposure of the implant cover screw with male gender, history of systemic disease, keratinized gingiva width, and thickness of the covering tissue. However, further prospective studies are required to confirm the results of the present study. Also, by confirming the hypothesis in the present study that the thin thickness of the covering mucosa and the lack of keratinized gingiva in the implant placement area are factors for early exposure, it is recommended that these two variables be examined before implant placement.

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References

- 1.Na HJ. Implant Surgery and Assistant Guide. *Int J Clin Prev Dent*. 2022;18(3):96-109.
- 2.Kim JC, Lee M, Yeo ISL. Three interfaces of the dental implant system and their clinical effects on hard and soft tissues. *Mater Horiz*. 2022;9(5):1387-1411.
- 3.Joly JC, de Lima AF, da Silva RC. Clinical and radiographic evaluation of soft and hard tissue changes around implants: a pilot study. *J Periodontol*. 2003;74(8):1097-103.
- 4.Aivazi M. Detection of Accelerated Osteointegration in Surface Modified Dental Implants with Sensor Nodes Located in Wireless Network of Body Areas. *Int Multi Proc Commun Syst (IMPCS)*. 2022;3(3):1-10. [In Persian]
- 5.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(4):514-21.
- 6.Tal H, Reiser V, Naishlos S, Avishai G, Kolerman R, Chaushu L. Screw-Type Collar vs. Non-Screw-Type Collar Implants-Comparison of Initial Stability, Soft Tissue Adaptation, and Early Marginal Bone Loss-A Preclinical Study in the Dog. *Biology (Basel)*. 2022;11(8):1213.
- 7.Negahdari R, Ghoreishizadeh A, Ghavimi MA, Soltanpour A, Bohlouli S. Occurrence and severity of spontaneous exposure of cover screw after dental implant placement. *J Adv Periodontol Implant Dent*. 2021;13(2):61-8.
- 8.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(1):76-9.
- 9.Mendoza G, Reyes JD, Guerrero ME, De La Rosa G M, Chambrone L. Influence of keratinized tissue on spontaneous exposure of submerged implants: classification and clinical observations. *J Osseointegration*. 2014;6(3):47-50.
- 10.Hertel M, Roh YC, Neumann K, Strietzel FP. Premature exposure of dental implant cover screws. A retrospective evaluation of risk factors and influence on marginal peri-implant bone level changes. *Clin Oral Investig*. 2017;21(6):2109-22.
- 11.Tal H. Spontaneous early exposure of submerged implants: I. Classification and clinical observations. *J Periodontol*. 1999;70(2):213-9.
- 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(3):436-41.
- 13.Kim TH, Lee DW, Kim CK, Park KH, Moon IS. Influence of early cover screw exposure on crestal bone loss around implants: intraindividual comparison of bone level at exposed and non-exposed implants. *J Periodontol*. 2009;80(6):933-9.
- 14.Chrcanovic BR, Albrektsson T, Wennerberg A. Diabetes and oral implant failure: a systematic review. *J Dent Res*. 2014;93(9):859-67.
- 15.Cehreli MC, Kökat AM, Uysal S, Akca K. Spontaneous early exposure and marginal bone loss around conventionally and early-placed submerged implants: a double-blind study. *Clin Oral Implants Res*. 2010;21(12):1327-33.
- 16.Cassetta M, Pranno N, Calasso S, Di Mambro A, Giansanti M. Early peri-implant bone loss: a prospective cohort study. *Int J Oral Maxillofac Surg*. 2015;44(9):1138-45.
- 17.Barboza EP, Caúla AL. Diagnoses, clinical classification, and proposed treatment of spontaneous early exposure of submerged implants. *Implant Dent*. 2002;11(4):331-7.
- 18.Baqain ZH, Moqbel WY, Sawair FA. Early dental implant failure: risk factors. *Br J Oral Maxillofac Surg*. 2012;50(3):239-43.