Coated vs Uncoated Implants: Bone Defect Configurations After Progressive Peri-implantitis in Dogs

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In this study, hydroxyapatite coated vs uncoated implants were used to evaluate the type and dimensions of bone defects after progressive peri-implantitis in dogs. Thirty-two dental implants with 4 different surfaces—machined (M), sandblasted acid-etched (SA), 1-μm thin sputter hydroxyapatite (HA)-coated (S), and plasma-sprayed HA-coated (P)—were inserted into the mandibles of 4 beagle dogs after extracting all mandibular premolars. Experimental peri-implantitis was induced after 3 months using ligature to allow for plaque accumulation. After 4 months, ligatures were removed and plaque accumulation continued for 5 months (progression period). The open flap surgery demonstrated 3 patterns of peri-implantitis bone defect: (1) Class I defect: represented as circumferential intra-alveolar bone loss; (2) Class II defect: circumferential intra-alveolar defect with supra-alveolar bone loss exposing the implant surface; and (3) Class III defect: represented as circumferential intra-alveolar defect with supra-alveolar bone loss and buccal dehiscence. Class I was the most frequent (62.5%) defect pattern around implant types M, SA, and S; while implant type-P showed a recurring majority of Class II (62.5%). Comparison among the 4 implant groups revealed a significant defect width (DW) in implant type-P relative to other types ($P < 0.01$). However, no statistically significant differences were noted for defect depth (DD) ($P > 0.05$). We concluded that the shape and size of peri-implantitis bone defects were influenced by the type and thickness of the HA coat together with the quantity of the available peri-implant bone. Plasma-sprayed HA-coated implants showed larger peri-implant defects than did thin sputter HA-coated implants.

Key Words: dental implants, peri-implantitis, bone defects, hydroxyapatite coat

INTRODUCTION

The use of dental implants to restore missing teeth has become an appealing treatment approach over the past 20 years. Improved osseointegration significantly enhanced the long-term prognosis of dental implants.[1] Nevertheless, implant complications and failures do develop. Peri-implantitis is considered a significant complication that leads to late implant failure.[2] Inflammation of the peri-implant mucosa is called peri-implant mucositis, which is a reversible condition. However, peri-implantitis is an inflammatory process in the soft tissue with the loss of the peri-implant supporting bone. Further progression of peri-implantitis can lead to more pronounced vertical and horizontal bone destruction, and eventually to the loss of the implant.[3–5] Therefore, mobility is not a main early manifestation for implants affected by peri implantitis.[6]

Currently, considerable evidence is present that supports the hypothesis that microbial colonization has a fundamental role in the etiology of peri-implantitis.[7] After the installation of dental implants, a bacterial colonization similar to that associated with periodontitis is recognized. Bacterial biofilm is subsequently produced that acts as a scaffold for

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resident flora. Consequently, bacterial products elicit a host response consisting of the expression of inflammatory mediators and signaling molecules that stimulate inflammatory cells. Comparable to periodontitis, bacterial colonization around dental implants results in hard and soft tissue destruction.[8] Peri-implant diseases have been related to mainly Gram-negative anaerobic bacteria, while failing implants have been associated with bacteria similar to that of advanced periodontitis around natural teeth.[9]

An experimental ligature model to induce peri-implant infection was established and used in both dogs and monkeys to study the pathogenesis of peri-implantitis. In this model, peri-implantitis lesions were induced by placing cotton ligatures submarginally around the implant and terminating the plaque control regimen to enhance plaque accumulation.[10] This results in the establishment of inflammatory lesions in the peri-implant tissue with consequent rapid breakdown of the supporting bone.[11] Thereafter, this model was used to compare the influence of different implant surfaces and systems on peri-implantitis progression.[12,13]

Different preclinical models have been used to assess the osseointegration process of dental implants.[12,14,15] However, experimental studies in dogs are of clinical relevance in implant dentistry, due to the evidence that dogs can serve as appropriate representative models for periodontal disease as well as bone-implant interaction.[7,10,16,17] A previous study[7] compared induced peri-implant bone defect configurations in dogs and humans, and concluded that peri-implant bone defects in dogs bear a resemblance to naturally occurring lesions in humans regarding their shape and size. Although searching further for relevant issues regarding the etiology and diagnosis of peri-implantitis is essential, discovering an optimum treatment approach is an appealing research modality as well. Peri-implantitis treatment aims to attain re-osseointegration, which could be achieved by eliminating the cause of peri-implantitis, followed by establishment of conditions favorable to re-osseointegration.[18] Various treatment procedures such as conservative, resective, and regenerative have been described.[6] The outcomes of these different methods depend mainly on the dimensions of remaining bony walls.[19,20]

The highly osteoconductive properties of calcium phosphate compounds (called hydroxyapatite or HA) is well documented both experimentally and clinically. When used as coating material, they enhanced the bone-implant fixation. Plasma spraying is the conventional technique for depositing an HA coat on a core metal substrate. The resultant coat thickness is more than 50 μm with a very rough surface.[2] Although the biocompatible property of plasma-spraying HA coating has been observed in several studies, concerns have been raised regarding the potential risk of its delamination and degradation. As an alternative, other coating deposition methods were introduced to produce more adherent thin HA film. Radiofrequency magnetron sputtering (RFMS) is a process of depositing thin calcium phosphate films with a uniform thickness while preserving the roughness of the underlying substrate.[21,22]

Thus, the aim of this study was to assess the shape and size of the peri-implant bone defects after the progression of ligature-induced peri-implantitis around two types of HA-coated implants (thin sputter HA coat and plasma-sprayed HA coat) and uncoated implants (machined and sandblasted acid-etched implants) in canine mandibles.

Material and Methods

Animals

Animal selection, management, and study protocol was approved by the Animal Care and Use Committee of Tokyo Medical and Dental University. The study was carried out on four healthy female beagle dogs (age 22–24 months, mean weight: 14 kg). During the experiment, the dogs were fed once a day with a soft consistency laboratory diet and water. Prior to experiment initiation, an adaptation period of 4 weeks was allowed. All surgical operations were performed under general anesthesia with 0.05 mL/kg medetomidine hydrochloride (Dormitor, Orion Corporation, Espoo, Finland) as intramuscular premedication, and 2 ml/10kg ketamine (Ketararu, Daiichi Sankyo Co., Tokyo, Japan). Infiltration anesthesia at the surgical site was performed with 2% xylocaine/epinephrine (1:80 000) (Dentsply, Sankin, Tokyo, Japan). Prophylactic antibiotic of clindamycin (11 mg/kg body weight) was given postoperatively for 3 days.

TABLE 1

Characteristics of different implant types used in the study*

Implant	Surface	Dimensions	Type	Company
M	Machined	3.3 × 10 mm	custom on-piece	μ-one HA implant Tokyo, Japan
SA	Sandblasted acid-etched	3.3 × 10 mm	custom on-piece	
S	Radiofrequency magnetron sputter HA coated (1 μm)	3.3 × 10 mm	custom on-piece	
P	Pressurized post plasma-spraying HA coat (50 μm)	3.25 × 10 mm	MP-1 Spline Reliance implant	Zimmer Dental Inc., Carlsbad, Calif. ICA.

*HA indicates hydroxyapatite.

Surgical procedures and experimental peri-implantitis

The study was performed in 3 surgical phases: In the first phase, extraction of all mandibular premolars was performed bilaterally. After reflection of full-thickness mucoperiosteal flaps and tooth separation, premolars were removed. The flaps were repositioned by means of interrupted sutures and surgical sites were allowed to heal for 3 months. In the second surgical phase, bilateral mandibular full-thickness mucoperiosteal flaps were elevated. Four surgical implant sites were prepared bilaterally using a low-trauma surgical technique under copious irrigation with sterile saline. Four dental implants, with 4 different surface treatments—(1) implant type M: machined; (2) SA: sandblasted acid etched; (3) S: 1 μm sputter HA-coated; and (4) P: plasma spraying HA-coated (Table 1)—were inserted in anterior-posterior randomized order ($n = 8$ implants per dog).

All implants had coating levels in line with the alveolar crest and showed good primary stability. Healing abutments for implant type P (3 mm length, 3.5 mm diameter) were installed after implant insertion to allow for nonsubmerged healing for all implants. Adequate width of attached gingiva was preserved to avoid any mucogingival stress. Flaps were repositioned and approximated and then closed with interrupted sutures without tension. After 2 weeks, sutures were removed and for the following 3 months, a plaque control program using 2% chlorhexidine rinse (chlorhexidine gluconate 20%, ICI Pharmaceutical Group, Wilmington, Del), 40 ml of a 2% solution 3 times/week and scaling once a month was maintained.

Ligature-induced peri-implantitis was initiated after the healing period. Thus, oral hygiene procedure was terminated and cotton ligatures were placed around the implants neck in a submarginal position. Ligatures were examined twice a week and changed every 3 weeks. After 4 months, when approximately 20% of the initial bone support was lost (assessed on standardized radiographs), ligatures were removed. A 5-month plaque accumulation period with progression of experimental peri-implantitis was initiated. After 5 months of peri-implantitis progression, open flap surgery was performed. The third surgical phase will be reported elsewhere; in brief, bilateral full-thickness mucoperiosteal flaps were elevated to expose the subsequent peri-implant bone defects. Following granulation tissue removal, configuration of the peri-implant bone defects for the 4 different implant types ($n = 8$ implants) were evaluated.

Peri-implant bone defect configuration assessment

During open flap surgery, two pre-calibrated examiner assessed the peri-implant bone defect around the 4 implant types using a periodontal probe (PCP 12, Hu-Friedy Co, Chicago, Ill). The mean of the double assessments were recorded for the following measurements: Figure 1: Defect depth (DD), measured as the vertical distance from implant coating level to the bottom of the defect, and defect width (DW), measured as the linear distance from implant surface to the adjacent alveolar bone surface.

Statistical analysis

SPSS statistical software (SPSS18, IBM, Armonk, NY) was used for statistical analysis. The mean values of all parameters were calculated for each implant in each animal ($n = 8$). Normal distribution was assessed using the Shapiro-Wilk test. Differences between group means were analyzed using analysis of variance and the Scheffe posthoc test. The level of significance was set at $P < 0.05$.

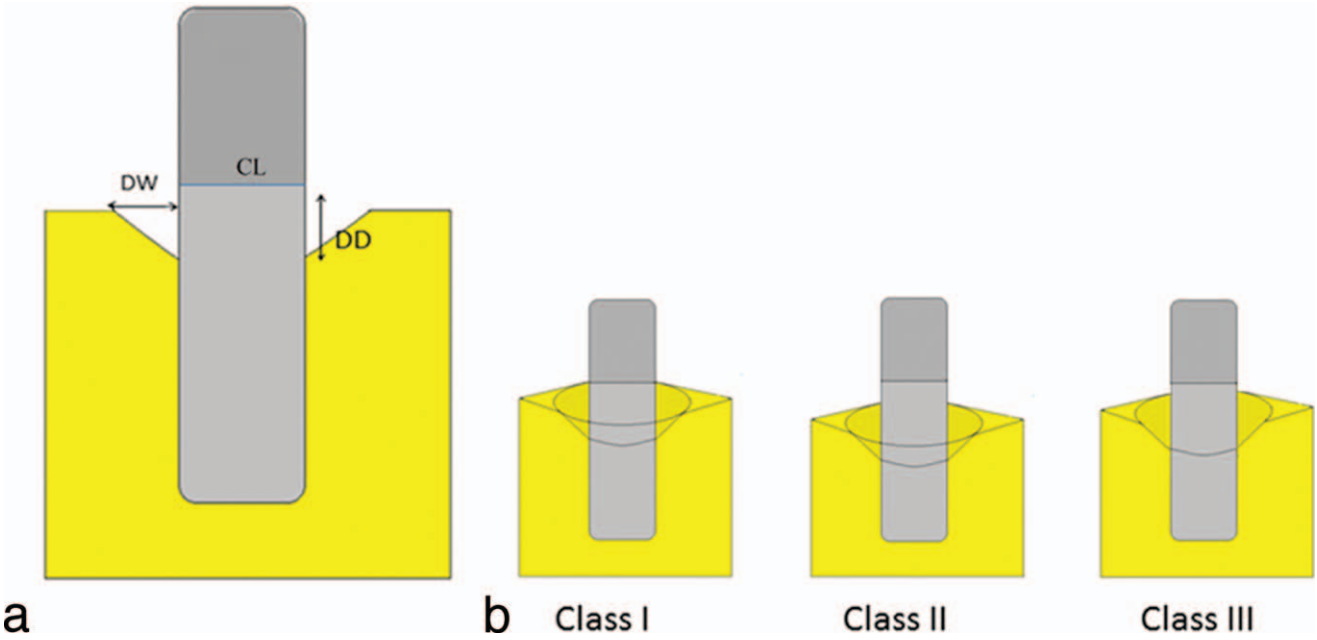

Figure 1. Illustration of defect measurements recorded for Class I, II, and III defects. (a) DD: defect depth, DW: defect width, CL: coating level. (b) Different defect classes.

Results

The three surgical phases were uneventful, and all animals recovered normally.

Peri-implantitis bone defect assessment

The open flap surgery demonstrated 3 patterns of peri-implantitis bone defect: (1) Class I defect: represented as circumferential intra-alveolar bone loss; (2) Class II defect: circumferential intra-alveolar defect combined supra-alveolar bone loss exposing the implant surface; and (3) Class III defect: represented as circumferential intra-alveolar defect with supra-alveolar bone loss and vertical bone loss of the adjacent buccal wall leading to dehiscence type defect (Figures 2 and 3).

Regarding the mean DD, implant type P showed increased values, while implant types S and SA showed the lowest values. The DD values for the buccal and lingual surfaces were slightly higher than their corresponding proximal values in implant type SA and S. Implant type P showed the highest defect width DW (1.7 mm) among the implants, a significant difference was detected between implant type P and implant type M, SA, and S ($P = 0.01$) (Figure 4).

Regarding the defects distribution, a Class I defect was more observed (62.5%) among implant types M, SA, and S than in implant type P (25%), while Class II defect was most frequently observed in implant type P (62.5%) than in implant type M (37.5%), SA (25%), or S (37.5%). Class III was observed in 12.5% of implant types SA and P.

Discussion

Assessment of defect configuration after peri-implantitis progression around different implant surfaces–namely, M, SA, S, and P—in canine mandible was evaluated in this study. The results of the present study have demonstrated 3 patterns of bone defects; in particular, circumferential bone defect (Class I), combined circumferential bone defect with supra-alveolar bone loss (Class II), and circumferential intra-alveolar defect combined with buccal dehiscence (Class III). The circumferential bone defect was the most frequently observed (62.5%) type of defect around machined, sandblasted acid-etched, and thin sputter HA coated implants. Circumferential bone defect with supra-alveolar bone loss was most observed around plasma-sprayed HA-coated implants (62.5%).

Ligature induced peri-implantitis animal models have been commonly used to study the pathogenesis and influence of different implant surfaces as well as different treatment modalities of peri-implantitis. Peri-implantitis has been defined as

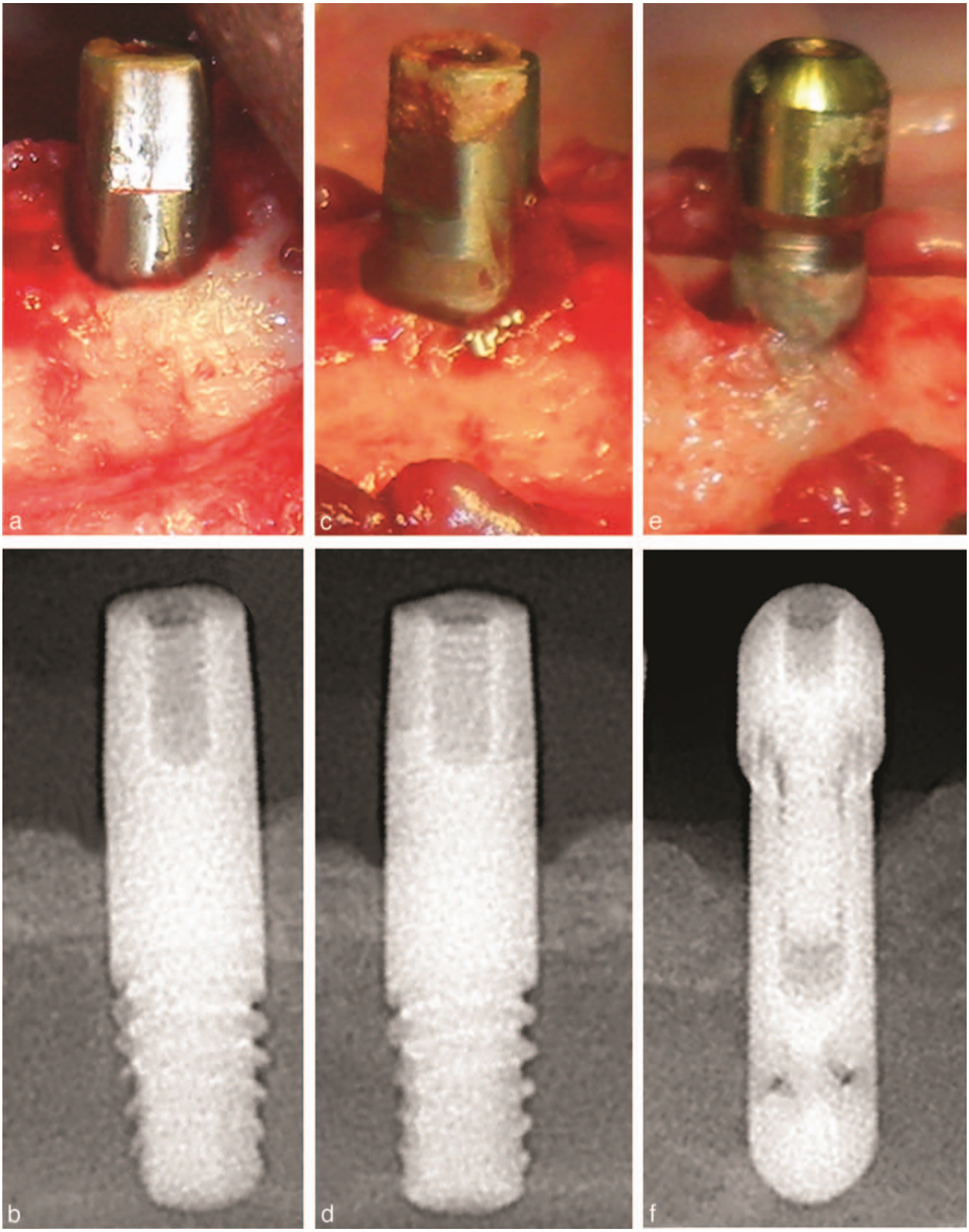

Figure 2. Clinical and radiographic view of induced peri-implantitis defects around different implant types. (a and b) Class I defect around machined implant (type M). (c and d) Class I defect around thin sputtered hydroxyapatite (HA)-coated implant (type S). (e and f) Class II defect around plasma-sprayed HA-coated implant (type P).

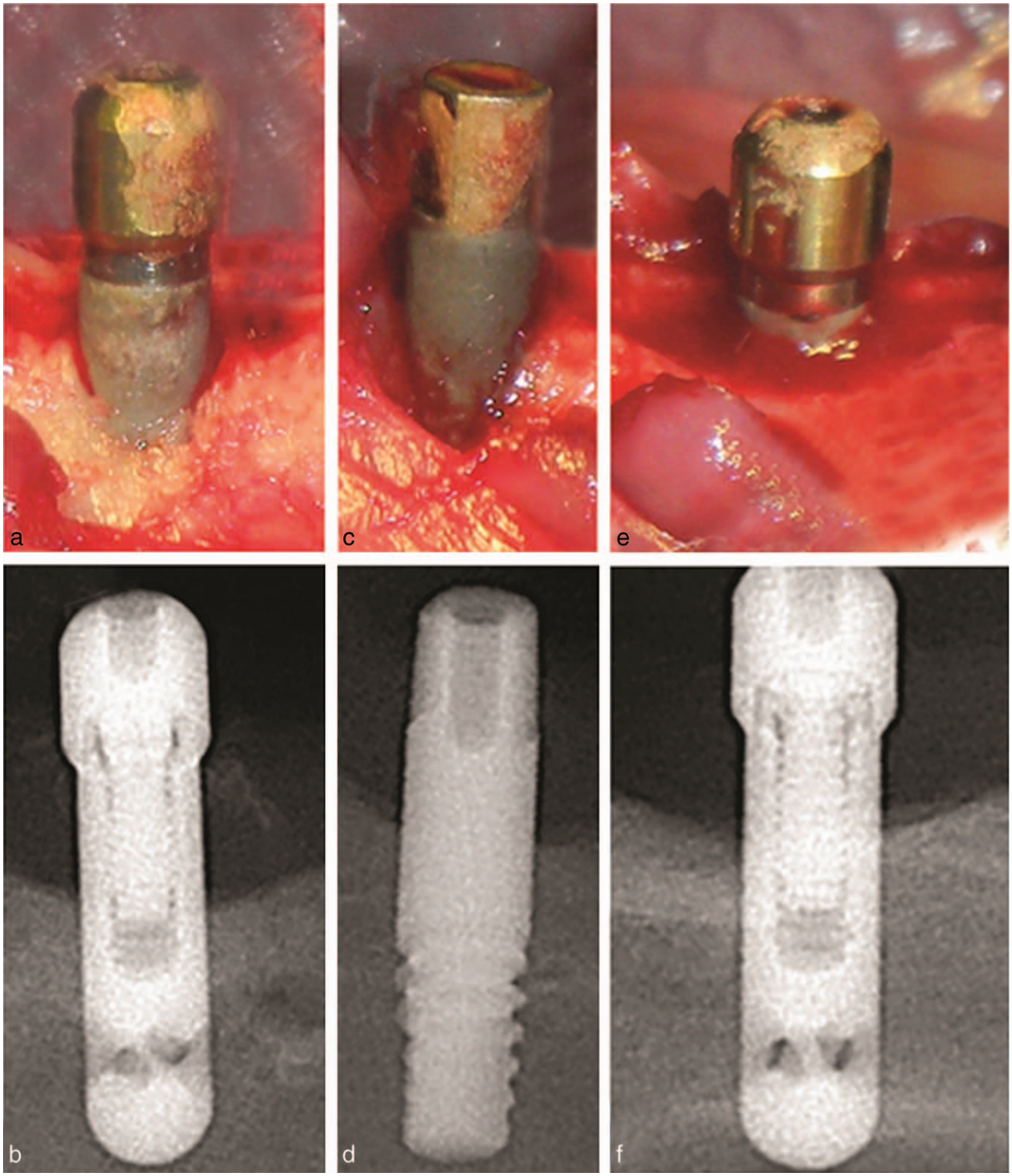

Figure 3. Clinical and radiographic view of induced peri-implantitis defects around different implant types. (a and b) Class III defect around plasma-sprayed hydroxyapatite (HA)-coated implant (type P). (c and d) Class III defect around sandblasted acid-etched implant (type SA). (e and f) Class II defect around plasma-sprayed HA-coated implant showing wide horizontal defect width.

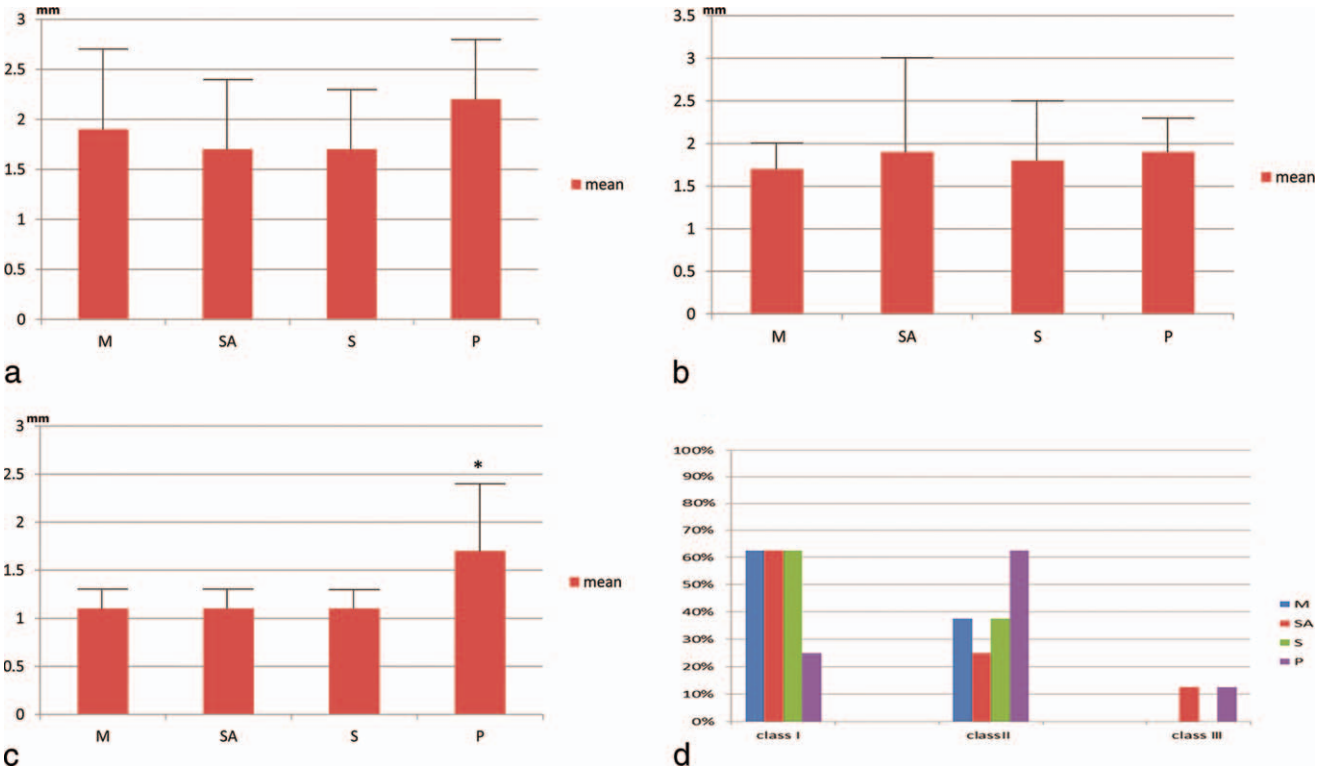

Figure 4. Induced peri-implantitis bone defect assessment. (a) Defect depth for proximal implant surfaces (mean ± SD in mm). (b) Defect depth for buccal and lingual implant surfaces (mean ± SD in mm). (c) Defect width (mean ± SD in mm). *Indicates significant difference ($P < 0.05$) between implant type P and other implant types. (d) Frequency distribution of defect classes around implant types.

plaque-induced progressive marginal bone loss that is observed on radiographs and associated with clinical signs of peri-implant soft tissue infection.[21] The consequential relationship between bacterial plaque accumulation and the development of soft tissue inflammation surrounding dental implants has been reported by Pontoriero et al.[23] Thus, untreated reversible peri-implant mucositis will lead to progressive destruction of the supporting bone (peri-implantitis) and eventually to implant failure.[5]

Dental plaque bacteria associated with periodontitis in beagle dogs has been shown to be similar to that observed in human periodontal disease. Additionally, canine bone has been observed to resemble human bone regarding its composition and density.[12,16,17] Schwarz et al[7] observed that bone defects resulting from ligature-induced peri-implantitis in beagle dogs closely represent the configuration of peri-implantitis bone defects in human.

Previous reports[7,11,24] showed that peri-implantitis was induced by placing a ligature around implants in a submarginal position, which lead to increased plaque accumulation and the rapid breakdown of peri-implant hard and soft tissue. This is in agreement with our results, in which placing ligature for 4 months in a submarginal position resulted in plaque accumulation with consequent peri-implant bone loss. The additional bone loss that took place after ligature removal during the subsequent 5 months (progression period) reflects the performance of different implant surfaces regarding peri-implantitis progression. This is in agreement with previous studies[2,11] that considered the progression period to be a more valid representation of the naturally occurring peri-implant bone loss, and can reflect the behavior of different implant designs and characteristics in peri-implantitis conditions.

Previous experiments using a canine model reported ligature removal after radiographic examination revealing 20–30% of initial bone loss around threaded implants.[4,11,25,26] However, regarding the shape of the resultant defects, limited data were available describing defect configuration following peri-implantitis progression. In contrast to these

studies, in the present study, cylindrical implants were used to avoid the influence of different thread designs on peri-implantitis progression.

Hayek et al[25] reported that after 120 days of ligature-induced peri-implantitis followed by an additional 120 days of plaque accumulation without ligature, the observed bone defects consisted of horizontal bone loss accompanied by localized vertical defect. However, these defects were prescribed based on clinical and radiographic evaluation. A limitation of this type of assessment is that the radiographic image is limited to the proximal implant surfaces without providing precise information about the buccal and lingual surfaces. Other studies[27–29] merely reported that circumferential bone defect associated with supra-alveolar bone loss was observed after open flap surgery to treat ligature induced peri-implantitis.

These observations are consistent with our results in which combined circumferential bone defect and supra-alveolar bone loss was observed in our study (Class II). However, in contrast to previously reported observations, our results reported another defect type—Class III—which exhibited circumferential bone loss and buccal dehiscence. This type of defect was also reported by Schwarz et al[7]; however, a limitation of the former study was the use of only one type of threaded implant (sandblasted acid-etched). Consequently, they did not evaluate the effect of different implant surfaces on defect shape, and only recorded the defect dimensions after the active-break down period (in presence of the ligature).

Bone loss at implants with the same design but with different surface treatments placed at the same time did not show the same progression rate. This proposes that local factors could modify the progression of bone loss at implants during peri-implantitis. This is in agreement with Serino et al.[30] In their study, they observed a different progression rate of bone loss in patients with peri-implantitis.

Regarding the influence of different implant surface treatments, in our study, we observed a difference in the horizontal and vertical components of the defect. Plasma-sprayed HA-coated implants showed wider and relatively deeper defects than did the other implant types. This could be explained by the high surface roughness (Ra = 5 μm) of plasma-sprayed HA coat in comparison to sandblasted acid-etched (Ra = 2 μm) and thin sputter HA coat (Ra = 1 μm) that could facilitate more plaque accumulation. In this context, it must be emphasized that 1 μm thin sputter HA-coated implants exhibited a Class I defect more frequently (62.5%), similar to machined and sandblasted acid-etched implants. While plasma-sprayed HA-coated implants exhibited a Class II defect more frequently (62.5%). However, remnants of bone tissue observed on the buccal defect of one plasma-sprayed implant may be due to the biocompatible properties of the HA coat that allows bone to bind to the coat.

A limitation of this study was the use of cylindrical implants to compare the effect of different surface treatment on peri-implantitis bone defects configuration. However, most of the commercially available implants are the threaded type, which might reveal a different behavior due to the influence of the thread shape on plaque accumulation and stress distribution. Another study using the same HA coat with a different threaded implant design is recommended to evaluate the combined influence of the thread shape and the hydroxyapatite coatings.

Within the limitation of the current study, it was concluded that the observed peri-implant bone defect's shape and size were influenced by the quantity of the available peri-implant bone (specifically the marginal bone thickness) together with the implant surface properties that enable more plaque accumulation. The current research has shown some promise for thin 1 μm sputter HA coat that may eliminate some of the problems associated with the plasma-spraying process. Thin sputter HA-coated implants showed comparable defect shape and depth similar to machined and sandblasted acid-etched implants.

Abbreviations

DD: defect depth
DW: defect width
HA: hydroxyapatite

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