A Double-Blind Randomized Trial Comparing Implants with Laser-Microtextured and Machined Collar Surfaces: Microbiologic and Clinical Results

Renzo Guarnieri, MD, DDS[1]/Giorgio Rappelli, MD, DDS[2]/Matteo Piemontese, MD, DDS[3]/
Maurizio Procaccini, MD, DDS[4]/Alessandro Quaranta, DDS, PhD[5]

Purpose: *The purpose of this study was to compare the clinical outcome of and determine the differences in periodonto-pathogenic microbiota around two types of implant collar surfaces: laser-microtextured (test) vs machined (control).* ***Materials and Methods:*** *Seventeen patients (11 periodontally healthy, and 6 periodontally compromised) were selected to receive the two different implants, placed randomly, in two edentulous sites. Six months following the surgical placement of the dental implants, subgingival plaque samples were collected using paper points from the peri-implant sulcus and from the sulcus of an adjacent tooth. The presence of five putative periodontal pathogens, namely,* Aggregatibacter actinomycetemcomitans, Porphyromonas gingivalis, Prevotella intermedia, Treponema denticola, and Tannerella forsythensis*, was assessed using real-time polymerized chain reaction (RT-PCR). Peri-implant parameters and intraoral radiographs were recorded up to 1 year after abutment connection.* ***Results:*** *In the main population, and in the periodontally compromised subgroup, the total number of periodontal pathogens around test implant sites was lower than control implant sites and adjacent tooth sites, with a statistically significant difference ($P < .05$). In periodontally healthy patients, the mean probing pocket depth for the test implant was 1.31 ± 0.51 mm, compared with 2.66 ± 0.83 mm for the control implant, while in periodontally compromised patients, it was 1.61 ± 0.58 mm for the test implant, compared with a mean value of 2.84 ± 1.0 mm for the control implant.* ***Conclusion:*** *Implants with a laser-microtextured collar surface are not more vulnerable to pathogenic microflora colonization than implants with a machined collar surface. In both of the subgroups of patients (periodontally healthy and periodontally compromised), implants with a laser-microtextured collar surface have a better clinical outcome at 1 year of loading, compared with implants with a machined collar surface.* Int J Oral Maxillofac Implants 2016;31:1117–1125. doi: 10.11607/jomi.4563

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Many implants have a rough or textured surface to facilitate osseointegration, while the collar segment features a machined-turned (also called smooth) surface to potentially inhibit or reduce the adhesion of oral bacteria and plaque. According to their roughness, oral implant surfaces were classified in 2004 by Albrektsson and Wennerberg[1] into three different types: minimally rough with Sa ± 0.5 mm, moderately rough with Sa between 1.0 and 2.0 mm, and rough with Sa 2.0 mm. Data from the literature agreed in reporting that supragingival surfaces with a high surface roughness (Ra value > 2.0 mm) increased biofilm formation, leading to peri-implant tissue inflammation.[2–4] The increased plaque deposition on rough surfaces, compared with smooth surfaces, seems to be due both to the greater initial bacterial adhesion and the greater difficulty of cleansing. Surface free energy, chemical composition, irregularities, and roughness of the surface have been previously reported as the main factors related to microbial adhesion to the implant.[3,5–8] However, among all the studied factors, the roughness seems to be what most influences the plaque accumulation on implants.[3,4,9,10] Compared with smooth surfaces, rougher surfaces accumulate

[1]Private Implant Practice, Rome, Italy; Freelance Researcher, SCS, Scientific Consulting Service, Rome, Italy.
[2]Associate Professor, Division of Prosthodontics, School of Dentistry, University "Politecnica" delle Marche, Ancona, Italy.
[3]Associate Professor, Division of Periodontology, School of Dentistry, University "Politecnica" delle Marche, Ancona, Italy.
[4]Director, Department of Clinical and Specialist Sciences, School of Dentistry, Polytechnic University of Marche, Ancona, Italy.
[5]Associate Professor, Discipline Lead and Program Convenor, Periodontics and Implantology, Oral Health Centre of Western Australia/University of Western Australia, Perth, Australia.

Correspondence to: Dr Renzo Guarnieri, Via Aldo Moro, 31, 31011 Preganziol, Treviso, Italy.
Email: renzoguarnieri@gmail.com

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and retain more plaque, characterized by an increased proportion of rods, motile organisms, and spirochetes.[3] Besides the roughness, literature data indicate that the microbial composition at the peri-implant site is also influenced by the periodontal status of the remaining dentition.[11–14]

Several similarities between the composition of microbiota at implant and teeth sites were reported and support the role of the remaining dentition as the major source for the microbiologic colonization of implants.[12,15,16] These similarities indicated that, at least in the partially edentulous patients, the teeth with periodontal pockets may act as a reservoir for (re) colonization of the subgingival region surrounding the implants. As the bacteria implicated in peri-implantitis are primarily those associated with periodontal disease,[17] several authors have suggested that the presence of periodontal pathogens may be correlated to a high risk of peri-implantitis.[18–20] However, a more recent comparative study between minimally and moderately rough implants[21] showed that the surface roughness is not correlated with a higher risk in the formation of peri-implant mucositis, while smoking seems to have a statistically significant correlation in the onset of peri-implant tissue inflammation regardless of the type of implant. On the other hand, in vitro studies have reported that moderately rough surfaces of collar implants may improve early healing of the soft tissue by influencing the attachment, orientation, and proliferation of epithelial and connective tissue cells.[22–27] Several clinical studies have verified the advantages of retention elements such as microthreads and microgrooves at the implant neck on the coronal portion of the implant, compared with a smooth neck, in terms of the established connective and bone-to-implant contact.[28–32] However, no data have been published on the in vivo subgingival plaque formation considering the influence of this kind of surface. Hence, this randomized clinical study aimed to compare the clinical results, and to determine the differences in periodonto-pathogenic microbiota around implants with a laser-microtextured collar surface vs implants with a machined collar surface. For this purpose, two different subgroups were evaluated: partially edentulous periodontally compromised patients and partially edentulous periodontally healthy patients.

MATERIALS AND METHODS

Patients

The study group included 17 partially edentulous patients (mean age at implant insertion: 57.1 years, two smokers, four women), who required, in 2013, implants for the rehabilitation of two edentulous sites.

Patients were included in this study according to the following criteria:

- No contraindications for oral surgery procedures, such as systemic diseases, pregnancy, regular use of prescription medications, or consumption of recreational drugs
- Recipient sites for implants healed for $>$ 3 months after tooth extraction, and with sufficient bone volume to place an implant with at least 3.8 mm in diameter and 9 mm in length
- Teeth adjacent to the implant area (mesially and distally) not affected by untreated periodontal and endodontic infections.

Exclusion criteria were:

- Poor oral hygiene
- Absence of opposing occlusion
- Parafunctional habits, severe maxilla-mandibular space discrepancies, and any drug/alcohol abuse

All patients gave their informed consent to the treatments, which were performed in accordance with the Helsinki Declaration.

Implants

Two implants with the same tapered macro design, but with different collar surfaces, were placed randomly: test—mount-free Laser-Lok Tapered Internal (BioHorizons), and control—mount-free RBT Tapered Internal (Biohorizons). Test implants had the most coronal 1.8 mm of the collar laser-microtextured (Fig 1), while the most coronal 0.3 mm of the collar of the control implants was smooth, machined metal and the next 1.5 mm of the collar was grit-blasted to create a moderately smooth surface (roughness between 0.72 and 1.34 μm; Fig 2).

Presurgical Assessment, Surgical Procedure, and Prosthetic Procedure

To perform a complete presurgical evaluation, a cone beam computed tomography (CBCT) scan examination, a waxup, and a surgical template were carried out for each patient.

All patients underwent the same surgical protocol, and in each patient, the two implants were placed in an alternating sequence determined by computer randomization. The implants were placed by two operators (A.Q., M.P.).

Patients scheduled for surgery were prescribed a systemic antibiotic (Amoxicillin/clavulanic acid [Augmentin, GlaxoSmithKline, Italy], 1 g, twice a day for 7 days) and a chlorhexidine digluconate solution 0.12% (Dentosan 0.12%, Johnson & Johnson) rinse (twice

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Fig 1 Test implant.



Fig 2 Control implant.

daily for 1 minute). After local anesthesia, by infiltration with articaine/epinephrine (Ecocain 20 mg/mL, Molteni Dental), surgical access was carried out with a full-thickness flap at the level of keratinized mucosa with a minimally extended release incision, to expose the crest and the vestibular limit of the bone. The utmost care in preserving the periodontal integrity of adjacent teeth was carried out. The receiving sites were prepared according to the recommendations of the manufacturer. A single-stage surgery with a nonsubmerged healing approach was performed in all sites. The flap was closed without tension using 4.0 or 5.0 monofilament sutures, which were left in place for 10 days. Patients were instructed to have a liquid or semiliquid diet for the first 3 days and then gradually return to a normal diet. An analgesic (Ibuprofen, 600 mg) immediately after the surgical intervention and after 8 hours was prescribed.

Abutment connection was carried out after 12 weeks with straight Simple Solution (BioHorizons) abutments, which were seated with a torque wrench device at 30 N. Prior to the impression, clinical parameters were collected (probing depth [PD], modified Gingival Index [mGI], and modified sulcular Bleeding Index [mSBI]). Impressions were made 15 days following abutment connection according to the Simple Solution BioHorizons prosthetic manual. Definitive restorations were delivered within 1 month. All partially edentulous areas were restored with fixed partial dentures by a single operator (G.R.).

Microbiologic Analysis

The microbiologic analysis was performed by means of real-time polymerized chain reaction (RT-PCR) technique using Carpegen Perio Diagnostics Test (Carpegen) according to the instructions of the manufacturer. Six months following the surgical placement of the dental implants, subgingival sampling with paper points of the peri-implant sulcus and adjacent tooth sulcus were collected. The sampling area was isolated with cotton gauzes, and accurate suction was performed as well as supragingival scaling with Teflon or plastic curettes for implant maintenance. Paper points were inserted in four different sites for each implant included in the study (buccal, mesiobuccal, distobuccal, and lingual). The paper points were removed after 20 seconds with careful attention not to touch the oral mucosa and coronal surfaces. The points were then dropped in plastic sterile falcon tubes. An adjacent tooth was also evaluated for microbiologic analysis. The RT-PCR analysis was consequently assessed to test the quantitative and qualitative presence of the following periodontal and peri-implant pathogens using species-specific probes: *Aggregatibacter actinomycetemcomitans*, *Porphyromonas gingivalis*, *Prevotella intermedia*, *Treponema denticola*, and *Tannerella forsythensis*. Results were expressed in Genome Equivalents (Geq)/mL $\times$ 10^3.

Clinical, Radiographic, and Statistical Evaluation

Peri-implant probing pocket depth (PPD), mGI, and modified Plaque Index (mPI) were assessed 15 days after implant uncovering, from 4 to 6 months following implant placement, at the time of implant position impression, after the microbiologic sampling visit, and at the 1-year follow-up recall. PPD was recorded with a millimeter-calibrated periodontal probe (PCP-UNC 15, Hu-Friedy) to the nearest millimeter. The measurements were registered for each implant on vestibular, oral, and proximal sites, and the mean was calculated. Five patients, each showing 2 teeth (single and multirooted) with probing depths $>$ 4 mm on at least one aspect of each tooth, were used to calibrate the investigator. Calibration was considered satisfactory when the two measurements were similar to the millimeter at $>$ 90% level.

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Radiographs were performed immediately and 6 months after surgery with a paralleling technique using a Rinn film holder with a rigid film-object x-ray source. For the radiographic procedures, a silicone index material was fixated to the maxillary dentition and a radiograph holder was constructed for each patient.

Periapical radiographs by means of a 70 kW/7.5 mA dental radiographic unit equipped with a long cone (TM 2002 Planmeca Proline CC, Planmeca Group) were exposed and developed in an automatic developer under standardized conditions.

The radiographs were then digitized using a dedicated scanner and converted into .TIFF files. Using image-processing software (ImageJ, National Institutes of Health), digitized images were stored at a resolution of 600 dpi, and the following radiographic measurements were evaluated using a digital trackball-driven cursor on a 10× magnified digitized image of the implant on the monitor:

- Radiographic implant length (IL): distance (in millimeters) between the implant shoulder and the implant apex as assessed at the midportion of the implant
- Residual bone height at the mesial (mBH) and distal (dBH) aspects of the implant: distance (in millimeters) between the implant shoulder and the first contact of the crestal bone on both the mesial and distal sides of the implant
- To account for radiographic distortion, radiographic measurements (ie, mBH, dBH) on each radiograph were adjusted for a coefficient derived from the ratio: true length of the implant/IL; mBH and dBH were reassessed at 6 months after adjustment for 6-month true length of the implant/IL.

For each implant, the MBL was calculated as the mean value of mBH and dBH.

To assess the reliability of the radiographic examination, 15 radiographs of patients restored with the same implant system and not included in the study (k-score for intra-examiner agreement: 0.952) were randomly selected and measured by a single trainer examiner twice with a 2-week interval.

Means and standard deviations were obtained for all samples in each group.

A public domain online software (Raosoft, http://www.raosoft.com/samplesize.html) was used to calculate the minimal number necessary for statistical evaluation. A randomization protocol was produced from two computer-generated lists for the assignment of the two different implants (test/control). The envelopes containing the randomization codes (1 or 2) were opened before every patient's surgery, to indicate the site of the implant placement (test or control).

The author who made the statistics was kept blind and has performed all the analysis without knowing the assignment group of the patients. Data were analyzed using SPSS software version 13.0 (SPSS).

Mean values and standard deviations (mean ± SD) were calculated for each considered parameter. The normal distribution of error terms and the validity of the model's assumptions were assessed using a Q-Q plot. Implant surfaces per patient subgroup and patient groups per implant surface were set up by means of multiple comparisions. One-sided *P* values were calculated and corrected according to Sidak's method for multiple hypothesis testing. Data were analyzed by means of the Mann-Whitney test and by repeated-measures analysis of variance (ANOVA). A P value $< .05$ was considered statistically significant. Results of the linear mixed effects were used to calculate the estimates of the differences between test and control implants and their 95% confidence intervals. Differences were considered as nonrelevant, when a clinically relevant difference was not included in the confidence interval.

RESULTS

Six patients received implants in the maxilla, and nine patients in the mandible. Six patients had a history of moderate to severe periodontitis that had been treated nonsurgically before implant insertion. At implant insertion, only a few 4- to 5-mm pockets remained, and all the patients were enrolled in a regular supportive periodontal therapy program. Nine patients were periodontally healthy. At the end of the observation period, the implant survival rate was 100% for test and control implants. In addition, there were no prosthetic complications regardless of the type of implant. The clinical measurement data for both test and control implants and for the adjacent tooth are listed in Table 1. In periodontally healthy patients, the mean PPD for the test implants was 1.31 ± 0.51 mm, compared with 2.66 ± 0.83 mm for the control implants ($P < .05$), while in periodontally compromised patients, the mean PPD was 1.61 ± 0.58 mm for the test implants, compared with a mean value of 2.84 ± 1.0 mm for the control implants ($P < .01$). No statistically significant differences of mGI and mPI were found in the subgroups of patients comparing test and control implants.

In the total amount of patients at the end of follow-up period, the mean radiographic marginal bone loss (MBL) was 0.188 ± 0.06 mm for test implants and 0.353 ± 0.17 mm for control implants. In the periodontally healthy subgroup, the mean radiographic MBL was 0.171 ± 0.04 mm for test implants and 0.333 ± 0.14 mm for control implants. In the periodontally compromised subgroup of patients, the radiographic MBL for the test

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Table 1 Mean PPD (mm), mGI, and mPI Scores of Study Groups

Study groups	Index (mean ± SD)								
	PPD			mGI			mPI		
	T	C	TT	T	C	TT	T	C	TT
Total (17)	1.31±0.51*	2.66±0.83	1.51±0.71	0.29±0.58	0.47±0.71	0.52±0.62	0.29±0.58	0.47±0.71	0.52±0.62
PC (6)	1.61±0.58**	2.84±1.0	2.25±0.6	0.0±0.0	0.5±0.54	0.33±0.51	0.0±0.0	0.16±0.40	0.66±1.03
PH (11)	1.72±0.4*	2.62±0.7	1.12±0.2	0.45±0.68	0.45±0.82	0.63±0.67	0.45±0.68	0.54±0.82	0.36±0.80

PPD = peri-implant probing pocket depth; mGI = modified Gingival Index; mPI = modified Plaque Index; T = laser-microtextured implants; C = smooth/moderately rough implants; TT = adjacent tooth; PC =-periodontally compromised patients; PH = periodontally healthy patients.
*Significant ($P < .05$).
**Highly significant ($P < .01$).

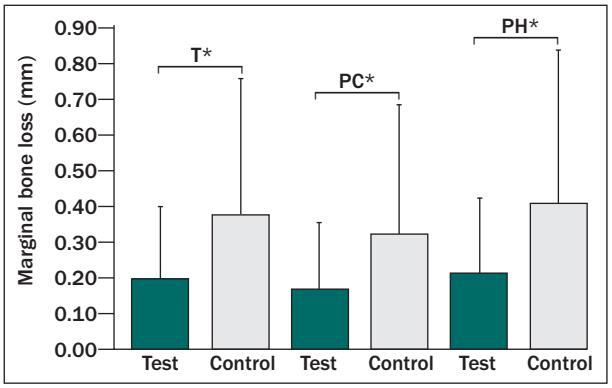

Fig 3 Mean radiographic marginal bone loss (mm) scores of study group. Test = laser-microtextured implants; control = smooth/moderately rough implants; T = total (17); PC = periodontally compromised patients (7); PH = periodontally healthy patients (9). *The differences between the test and control groups were statistically significant ($P < .05$).

Table 2 Subgingival Microbiota Along Test (Laser Microtextured), Control (Smooth/Moderately Rough) Implants, and Adjacent Teeth in the Study Groups, Analyzed Using RT-PCR

		Total bacterial load	*A.a.*	*P.g.*	*P.i.*	*T.d.*	*T.f.*
Total patients	T	0.8×10^3 (SD 2.3)	< 100 (SD 0)	0.8×10^3 (SD 1.9)	1.1×10^3 (SD 1.2)	1.0×10^3 (SD 2.0)	0.6×10^3 (SD 1.4)
	C	1.8×10^3 (SD 2.7)	< 100 (SD 0)	2.3×10^3 (SD 3.0)	1.2×10^3 (SD 2.4)	2.0×10^3 (SD 2.7)	2.2×10^3 (SD 2.9)
	TT	2.5×10^3 (SD 3.1)	< 100 (SD 0)	2.3×10^3 (SD 2.9)	2.3×10^3 (SD 3.2)	2.9×10^3 (SD 3.5)	2.2×10^3 (SD 3.1)
PH patients	T	0.6×10^3 (SD 1.5)	< 100 (SD 0)	0.6×10^3 (SD 1.1)	1.2×10^3 (SD 2.3)	0.6×10^3 (SD 1.5)	0.2×10^3 (SD 0.6)
	C	1.5×10^3 (SD 2.8)	< 100 (SD 0)	2.0×10^3 (SD 3.0)	1.0×10^3 (SD 2.7)	0.6×10^3 (SD 2.1)	2.2×10^3 (SD 3.2)
	TT	1.9×10^3 (SD 2.7)	< 100 (SD 0)	1.5×10^3 (SD 3.0)	2.1×10^3 (SD 3.0)	2.9×10^3 (SD 2.9)	1.0×10^3 (SD 2.0)
PC patients	T	1.3×10^3 (SD 2.3)	< 100 (SD 0)	1.3×10^3 (SD 3.1)		1.7×10^3 (SD 2.7)	1.4×10^3 (SD 2.2)
	C	2.8×10^3 (SD 2.5)	< 100 (SD 0)	2.9×10^3 (SD 3.3)	1.6×10^3 (SD 1.9)	4.6×10^3 (SD 2.8)	2.0×10^3 (SD 2.3)
	TT	3.5×10^3 (SD 3.5)	< 100 (SD 0)	3.9×10^3 (SD 1.9)	2.6×10^3 (SD 3.9)	3.0×10^3 (SD 4.7)	4.4×10^3 (SD 3.8)

A.a. = Aggregatibacter actinomycetemcomitans; P.g. = Porphyromonas gingivalis; P.i. = Prevotella intermedia; T.d. = Treponema denticola; T.f. = Tannerella forsythensis; PH = periodontally healthy; PC = periodontally compromised. *$P < .05$.

implants was 0.204 ± 0.09 mm, and 0.388 ± 0.21 mm for the control group. In the total amount of patients and in both subgroups, the difference between the test and control groups was noted to be statistically significant ($P < .05$; Fig 3).

Table 2 shows the amount of five periodontal pathogens present in the saliva samples identified by using RT-PCR along test (laser microtextured) implants, control (machined/moderately rough) implants, and adjacent teeth in all patients and in the subgroups

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analyzed. The total number of the five periodontal pathogens detectable by the PCR test was on average $0.8 \times 10^3 \pm 2.3$ for the test group, and $1.8 \times 10^3 \pm 2.7$ for the control group. The corresponding value in the adjacent tooth was $2.5 \times 10^3 \pm 3.1$. *Aggregatibacter actinomycetemcomitans* scored < 100 regardless of the type of implant (test and control). The same score was recorded for adjacent teeth as well. Around test implants, the mean score of *Porphyromonas gingivalis*, *Prevotella intermedia*, *Treponema denticola*, and *Tannerella forsythensis* was $0.8 \times 10^3 \pm 1.9$, $1.1 \times 10^3 \pm 1.2$, $1.0 \times 10^3 \pm 2.0$, and $0.6 \times 10^3 \pm 1.4$, respectively. Around control implants, the mean score of *Porphyromonas gingivalis, Prevotella intermedia, Treponema denticola,* and *Tannerella forsythensis* was $2.3 \times 10^3 \pm 3.0$, $1.2 \times 10^3 \pm 2.4$, $2.0 \times 10^3 \pm 2.7$, and $2.2 \times 10^3 \pm 3.1$, respectively. Around adjacent teeth, the mean score of *Porphyromonas gingivalis, Prevotella intermedia, Treponema denticola,* and *Tannerella forsythensis* was $2.3 \times 10^3 \pm 3.1$, $2.3 \times 10^3 \pm 3.2$, $2.9 \times 10^3 \pm 3.5$, and $2.2 \times 10^3 \pm 3.1$, respectively. Regarding the total bacterial load, the differences between the test and control, and between test and adjacent teeth were statistically significant ($P < .05$).

In periodontally compromised patients, the mean total number of periodontal pathogens around the test implants was $1.3 \times 10^3 \pm 2.3$, around the control implants it was $2.8 \times 10^3 \pm 2.5$, and around adjacent teeth it was $3.5 \times 10^3 \pm 3.5$. *Aggregatibacter actinomycetemcomitans* scored < 100 regardless of the type of implant (test and control). The same score was recorded for adjacent teeth as well. Around test implants, the mean score of *Porphyromonas gingivalis, Prevotella intermedia, Treponema denticola,* and *Tannerella forsythensis* was $1.3 \times 10^3 \pm 3.1$, $0.9 \times 10^3 \pm 1.5$, $1.7 \times 10^3 \pm 2.7$, and $1.4 \times 10^3 \pm 2.2$, respectively. Around control implants, the mean score of *Porphyromonas gingivalis, Prevotella intermedia, Treponema denticola,* and *Tannerella forsythensis* was $2.9 \times 10^3 \pm 3.3$, $1.6 \times 10^3 \pm 1.9$, $4.6 \times 10^3 \pm 1.8$, and $2.0 \times 10^3 \pm 2.3$, respectively. Around adjacent teeth, the mean score of *Porphyromonas gingivalis, Prevotella intermedia, Treponema denticola,* and *Tannerella forsythensis* was $3.9 \times 10^3 \pm 1.9$, $2.6 \times 10^3 \pm 3.9$, $3.0 \times 10^3 \pm 4.7$, and $4.4 \times 10^3 \pm 3.8$, respectively. The differences between test and control, and between test and adjacent teeth were statistically significant ($P < .05$).

In periodontally healthy patients, the total number of periodontal pathogens around the test implants was a mean of $0.6 \times 10^3 \pm 1.5$, around the control implants it was $1.5 \times 10^3 \pm 2.8$, and around adjacent teeth it was $1.9 \times 10^3 \pm 2.7$. *Aggregatibacter actinomycetemcomitans* scored < 100 regardless of the type of implant (test and control). The same score was recorded for adjacent teeth as well. Around test implants, the mean score of *Porphyromonas gingivalis, Prevotella intermedia, Treponema denticola,* and *Tannerella forsythensis* was $0.6 \times 10^3 \pm 1.1$, $1.2 \times 10^3 \pm 2.3$, $0.6 \times 10^3 \pm 1.5$, and $0.2 \times 10^3 \pm 0.6$, respectively. Around control implants, the mean score of *Porphyromonas gingivalis, Prevotella intermedia, Treponema denticola,* and *Tannerella forsythensis* was $2.0 \times 10^3 \pm 3.0$, $1.0 \times 10^3 \pm 2.7$, $0.6 \times 10^3 \pm 2.1$, and $2.2 \times 10^3 \pm 2.9$, respectively. Around adjacent teeth, the mean score of *Porphyromonas gingivalis, Prevotella intermedia, Treponema denticola,* and *Tannerella forsythensis* was $1.5 \times 10^3 \pm 3.0$, $2.1 \times 10^3 \pm 3.0$, $2.9 \times 10^3 \pm 2.9$, and $1.0 \times 10^3 \pm 2.0$, respectively. The differences between test and control, and between test and adjacent teeth were statistically significant.

DISCUSSION

The present study compared the microbiologic differences in partially edentulous patients, with and without a history of periodontal disease, between two commercially available implants: a BioHorizons mount-free Tapered Internal with a laser-microtextured collar surface (test) and a BioHorizons mount-free Tapered Internal with a machined collar surface (control). To the authors' knowledge, this is the first randomized study aimed at evaluating the microbiologic differences between these two types of implants characterized by the same body design and surface, but different collar surfaces. Data from the literature of in vivo subgingival plaque formation with the influence of surface roughness are scarce.[3,33,34] Quirynen el al[33] reported no significant differences in the subgingival density of periodontal pathogens between smooth and rough implant surfaces. Key pathogens like *P gingivalis*, *P intermedia*, and *F nucleatum* were found in comparable proportions on all surfaces. These data have been confirmed in a more recent randomized controlled trial with split-mouth design in which the same author, comparing minimally vs moderately rough implants, reported that an increased surface roughness does not influence the subgingival plaque formation.[34] Results of the present study agree with these data. They show that implants with a laser-microtextured collar surface are not more vulnerable to pathogenic microflora colonization than implants with a machined collar surface. Data present in the literature on the influence of implant surface roughness on plaque colonization are, however, contrasting. Some authors reported that implants with rough surfaces compared with machined surfaces accumulate and retain more plaque, characterized by an increased proportion of rods, motile organisms, and spirochetes.[4,6] However, more recent microbiologic studies highlighted that a higher surface roughness

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increases biofilm accumulation only in supragingival areas, whereas this influence was not detected in subgingival areas.[33–35]

In the present study, no statistical differences were found in the amount of periodontal pathogens in the subgroup of periodontally healthy patients, while statistically significant differences between test and control implants and between test implants and adjacent teeth were remarkable in periodontally compromised individuals. In this group of patients, the concentration of specific periodontal pathogens was found to be lower around test implants than control implants and adjacent teeth sites. These data might be seen as being in contrast with those reported by previous studies, in which it has been observed that the periodontally compromised dentition influences the periodontal microbiota composition at the implant site more than differences in surface roughness[12,13,20,36–41] Data from the literature indicate that in periodontally susceptible patients, the coexistence of teeth and implants seems to create a local environment in which subgingival periodontal pathogens around teeth can colonize newly inserted implant surfaces. However, more recent studies suggested that the simple geographic proximity seems to not be a sufficient determinant of colonization of topographically distinct niches, since the peri-implant and periodontal microbiomes represent microbiologically distinct ecosystems.[42–44]

The majority of studies on osseointegrated implant prognosis in partially edentulous patients showed that periodontally healthy and periodontally compromised individuals have the same short-term implant survival rates, and that the rough or smooth implant surfaces do not affect the implant prognosis.[45–47]

The results of the present study confirmed these data, showing 1 year after abutment connection an implant survival rate of 100% regardless of the type of implant used. Also, regarding clinical parameters, results from the present study are aligned with those of previous aforementioned studies, which reported, on a short-term basis, no statistically significant differences in PPD and marginal bone loss between patients treated for periodontitis and periodontally healthy subjects. In the present study, in periodontitis patients, a lower concentration of periodontal pathogens was found around test implants than control implants and adjacent teeth. In the authors' opinion, this can be explained by the different pocket probing depth found around the three different sites. The mean probing depth around test implants was 1.61 ± 0.58 mm, while around control implants it was 2.81 ± 1.3 mm, and around residual adjacent teeth it was 2.25 ± 0.69 mm. Previous clinical studies highlighted a consistent difference in probing depth and marginal bone loss between the implant pairs with and without the laser-microtextured implant collar treatment.[48–52] The laser-microtextured surface has been the object of in vitro research that has provided the hypothesis that this kind of microgeometry on the collar surface could be used to control soft tissue responses to dental implants, establishing a predetermined site to attract a physical connective tissue attachment.[26,27] This hypothesis has been afterward confirmed by histologic research in an animal model,[53] and in humans,[54] which have documented the presence of a physical connective tissue attachment onto laser-produced microgrooves on collar implant and abutment surfaces. Histologic data highlighted the presence of a physical connective tissue attachment, whose position is determined by the laser microgroove layout, with connective tissue fibers perpendicularly oriented to the implant surface, which act as a seal to apical migration of gingival epithelial cells. It is believed that the natural potential of the gingiva to prevent subgingival biofilm formation, and to respond to early biofilm accumulation, is associated with protective mechanisms, which depends on several components, such as the crevicular fluid, the junctional epithelium, a well-keratinized oral epithelium terminated at the crest of the gingival margin, and a network of collagen fibers.[12,55,56] As is the case around natural teeth, in which collagen bundles inserted into the root cementum deter the downgrowth migration of the overlying epithelium,[57] epithelial downgrowth around implants could be impeded by firm physical attachment between the soft connective tissue and the implant. In the case of a dental implant, a smooth collar does not permit connective tissue attachment; hence, the stability of the peri-implant soft tissue is lacking.[58] The laser-microtextured surface, although not analogous to the cemental surface of the natural teeth, seems to act in establishing a predetermined site to attract a physical connective tissue attachment, to restrict apical migration of gingival epithelium, and to provide higher soft tissue support compared with that provided only by the marginal bone. The present study did not demonstrate histologic evidence of a physical connective tissue attachment to the laser-microtextured implant collar surface; therefore, it is difficult to assess how these findings relate to the clinical results of the present study. However, what is clear on the basis of the present study is that the laser-microgrooved surface on the implant neck is not more vulnerable to pathogenic microflora colonization than implants with a machined collar surface. Also, in the presence of adjacent teeth affected by periodontal disease, a laser-microtextured surface, compared with a machined surface, seems to promote the formation of higher soft tissue support in the transmucosal area.

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Important limitations of the present study are the small sample size and the short follow-up period. Accordingly, final confirmation of findings needs further studies with longer periods of observation with an increased number of implants.

CONCLUSIONS

Within the limits of the present study (short follow-up period and small sample size), results showed that implants with a laser-microtextured collar surface are not more vulnerable to pathogenic microflora colonization than implants with a smooth/moderately rough collar surface. In addition, laser-microtextured implants have a better clinical outcome (at 1 year of loading) compared with smooth/moderately rough implants.

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