Two-Year Outcome with Nobel Direct® Implants: A Retrospective Radiographic and Microbiologic Study in 10 Patients

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ABSTRACT

Introduction: The Nobel Direct® implant (Nobel Biocare AB, Göteborg, Sweden) was developed to minimize marginal bone resorption and to result in "soft tissue integration" for an optimized aesthetic outcome. However, conflicting results have been presented in the literature. The aim of this present study was to evaluate the clinical and microbiologic outcomes of Nobel Direct implants.

Materials and Methods: Ten partially edentulous subjects without evidence of active periodontitis (mean age 55 years) received 12 Nobel Direct implants. Implants were loaded with single crowns after a healing period of 3 to 6 months. Treatment outcomes were assessed at month 24. Routine clinical assessments, intraoral radiographs, and microbiologic samplings were made. Histologic analysis of one failing implant and chemical spectroscopy around three unused implants was performed. Paired Wilcoxon signed-rank test was used for the evaluation of bone loss; otherwise, descriptive analysis was performed.

Results: Implants were functionally loaded after 3 to 6 months. At 2 years, the mean bone loss of remaining implants was 2.0 mm (SD ± 1.1 mm; range: 0.0–3.4 mm). Three out of 12 implants with an early mean bone loss >3 mm were lost. The surviving implants showed increasing bone loss between 6 and 24 months ($p = .028$). Only 3 out of the 12 implants were considered successful and showed bone loss of <1.7 mm after 2 years. High rates of pathogens, including *Aggregatibacter actinomycetemcomitans*, *Fusobacterium* spp., *Porphyromonas gingivalis*, *Pseudomonas aeruginosa*, and *Tanerella forsythia*, were found. Chemical spectroscopy revealed, despite the normal signals from Ti, O, and C, also peaks of P, F, S, N, and Ca. A normal histologic image of osseointegration was observed in the apical part of the retrieved implant.

Conclusion: Radiographic evidence and 25% implant failures are indications of a low success rate. High counts and prevalence of significant pathogens were found at surviving implants. Although extensive bone loss had occurred in the coronal part, the apical portion of the implant showed some bone to implant integration.

KEY WORDS: bone remodeling, dental implants, implant failures, one-piece implant, one-stage surgery

INTRODUCTION

Oral implantology has shifted from a basic scientific background in the early 1970s to a well-established clinical procedure in today's daily dental practice. Dental implant treatment is now considered simple and unfussy, provided the practitioner does not disregard certain guidelines to support an evidence-based treatment. Some implant systems and treatment protocols

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have sufficient long-term scientific evidence to sustain their usage for the successful treatment of partial and complete edentulism.[1–10]

From the clinician's perspective, there is a consensus that long-term scientific evidence is needed before changing an aspect of the oral implant equipment.[11] However, research and clinical trials run behind clinical reality, and sometimes, implants are already outdated at the time of publication. As a consequence, what is published seems often "old school" for clinicians visiting industry-sponsored meetings. More and more, the dental implant industry is dictating today's practice, and science seems overrun by commerce. Nowadays, the evolution of implant design, together with the patient's demands, is pushing the boundaries of oral biology. Implant companies claim treatment concepts dominating the implant environment and advice surgical and prosthetical protocols without sustained evidence. What happens if an implant company forgets the scientific time frame to quench their commercial thirst?

The Nobel Direct® implant (Nobel Biocare AB, Göteborg, Sweden) was commercially launched in 2004. This one-piece implant was designed to minimize marginal bone resorption as there is no submucosal microgap.[12] Furthermore, the rough TiUnite™ (Nobel Biocare AB) surface, which is left toward the mucosa, would form a "soft tissue integration" for an optimized aesthetic outcome. This soft tissue integration was a new concept without any scientific and clinical support. The company advised and sustained by their brochure to install the implant with a flapless approach and immediately prepare the supramucosal part with carbide burs for immediate function (Nobel Biocare, 2003). The first papers in the peer-reviewed literature are published starting from 2005 and report on inconclusive success rates from 46.1 to 97.9%.[13–18]

The aim of this present study was to evaluate the clinical and radiographic outcome and failure rate of Nobel Direct implants and relate those findings to the current knowledge of successful dental implants.

MATERIALS AND METHODS

The present study was conducted in accordance with the principles of the Declaration of Helsinki (1975). Informed consent was obtained from all subjects prior to the clinical examination. The Ethical Committee of the Ghent University Hospital approved the study protocol.

Subjects

A total of 10 subjects were consecutively treated by a periodontal surgeon (E.T.) with 18 years of experience. They were treated after referral for implant treatment in the partially edentulous mandible or maxilla in spring 2005. At the time of surgery, a presurgical assessment involved a clinical and radiographic examination. The subjects were required to be healthy and to have adequate bone for the placement of at least one implant in the edentulous space. The opposing teeth were natural teeth or complete or partial removable dentures. Heavy smokers (>10 cigarettes/day) were not excluded. The subjects underwent periodontal treatment whenever considered necessary on the remaining teeth. Exclusion criteria were general contraindications for oral implant surgery and inadequate bone volume or infection at the implant recipient site. Previous tooth extractions at the implant recipient site were performed at least 3 months prior to implant insertion.

Clinical Procedure

Surgical treatment was performed under local anesthesia. The flap design was kept as minimally invasive as possible. When indicated, a flapless procedure was performed with a soft tissue punch ($n = 4$). For the flap cases, a crestal incision was made to raise a full thickness mucoperiosteal flap ($n = 8$). At least one Nobel Direct implant was installed in every edentulous space according to the manufacturer's guidelines. Implant installation was prosthetically driven by a surgical guide. Implants were intentionally installed with the implant threads completely covered by bone. To achieve perfect initial stability the insertion torque value was set at 30 Ncm for the diameter 3.5 mm, and 40 Ncm for the diameter 4.3- and 5-mm implants. Periapical radiographs were taken immediately after surgery (baseline). The subjects received a postsurgical analgesic (ibuprofen 600 mg or paracetamol 500 mg) and were supplied with an ice pack to reduce postsurgical swelling.

Postoperative Care

The subjects were given the advice to rinse with chlorhexidine 0.12% (PerioAid, Dentaid, Barcelona, Spain) and to perform standard oral hygiene measures. No special dietary advice was given. The subjects were advised to take ibuprofen 600 mg or paracetamol 500 mg painkillers at their own discretion. Amoxicillin antibiotics were administered 2 g daily for 4 days. After

10 days, the sutures were removed. Oral hygiene was reinstructed with a soft manual toothbrush.

PROSTHETICS

Implants were left unloaded during the early healing phase. The supramucosal part of the implant was not prepared until the subjects were recalled 3 to 6 months after surgery for radiographic evaluation and evaluation of clinical mobility, pain, or infection. The subjects were then referred to the general dentist for implant prosthetics and regular professional maintenance.

Preparation of the abutment portion of the implant was done with purpose-made drills (Nobel Biocare AB) on a high-speed handpiece. A circumferential margin was prepared in order to create a prosthetic pillar. Excessive water cooling was used during drilling to protect the implant from overheating. The type of the implant restoration was left at the discretion of the general dentist. Subjects with a history of periodontal disease were regularly followed up by the periodontist.

Research Examination

The subjects were recalled after 2 years. Digital periapical radiographs were taken with a commercially available film holder. The lower corner of the coronal cylinder of the Nobel Direct implant was used as a reference point[14] from where on marginal bone level was calculated at the mesial and distal sites. Each radiograph was calibrated using the known implant length to correct for magnifications. True bone resorption was calculated comparing the marginal bone to implant level on the postoperative radiograph with the follow-up radiographs. The mean of the mesial and distal measurements was taken as the individual implant value and used to calculate mean bone loss on patient level, and those were used for the statistical analysis of bone loss over time by means of the paired Wilcoxon signed-rank test. The criteria used to discriminate surviving from successful implants allowed a maximal bone loss of 1.5 mm during the first year and a further loss of <0.2 mm yearly[19] and were done on implant level.

Microbiologic Sampling and Processing

Bacterial samples were taken after 2 years of function. The bacterial samples were collected at the implant site with five sterile endodontic paper points. The paper points remained in situ for 10 seconds. The five samples from each implant were placed in a dry Eppendorf vial. The samples were shipped to the oral microbiology laboratory at the University of Berne, Switzerland, and immediately processed by the checkerboard DNA–DNA hybridization method as described elsewhere.[20–22] A total of 74 bacterial strains were analyzed. The checkerboard DNA–DNA panel had been developed by using known species provided by the Forsyth Institute (Boston, MA, USA) or had been purchased from the ATCC collection of species (LGC Promochem Sarl, Molsheim Cedex, France). In order to receive a fully detailed account of the identified bacteria, the digitized information was analyzed by a software program (ImageQuant, Amersham Pharmacia, Piscataway, NJ, USA), allowing the comparison of signals against standard lanes of known bacterial amounts (10^5 cells) in the appropriate checkerboard slot. Signals were converted to absolute counts by comparison with these standards and studied as the proportion of sites defined as having $\geq 1.0 \times 10^4$ and $\geq 1.0 \times 10^5$ bacterial cells. Cross-reactivity is routinely tested in the microbiology laboratory between known pure bacterial standards, with the results consistent with those reported elsewhere by others.[21]

Chemical Analyses

Surface chemical analyses were performed on three unused implants delivered in unbroken containers with different Lot numbers. Two regions per implant were investigated using the PHI 5500 instrument (Al Ka monochromatic radiation, Physical Electronics Industries, Inc., Edina, MN, USA).

Retrieved Sample Preparation

Because of ongoing bone resorption and continuous infection, one implant needed to be removed after 21 months to preserve remaining peri-implant tissues. The subject consented to have the trephined implant histologically examined. The specimen was immersed in fixative and processed according to the so-called Exakt technique (Exakt Apparatebau, Norderstedt, Germany) initially described by Donath and Breuner.[23] This preparation results in undecalcified cut and ground sections of 10 µm. The sections were stained by Toluidine blue mixed with pyronin G. The light microscopic investigation involved qualitative and quantitative analyses of tissue surrounding the implant. The latter was done with a computerized image analysis tool[24] and involved bone to implant contact and bone area inside the threads.

TABLE 1 Overview of the Subjects, Age, Implant Position, Implant Type, Surgical Flap Design, and True Bone Loss after 6 Months and 2 Years Compared with the Baseline

Patient	Age (years)	Implant Position	Implant Type	Flapless	Bone Loss (mm) 0 to 6 Months			Bone Loss (mm) 0 to 2 Years		
					Mesial	Distal	Mean	Mesial	Distal	Mean
B.L. ♀	51	46	RP 4.3 × 13	No	0.67	1.9	1.30	1.67	1.85	1.76
B.L. ♀	51	36	RP 4.3 × 10	No	2.72	3.45	3.09	Failure	Failure	Failure
D.J. ♀	53	46	RP 4.3 × 10	No	1.25	1.5	1.38	1.62	1.89	1.76
D.J. ♀	53	36	RP 4.3 × 10	No	4.83	4.17	4.50	Failure	Failure	Failure
L.J. ♂	56	47	WP 5 × 10	No	—	—	—	1	1.4	1.20
P.H. ♂	50	33	NP 3.5 × 13	No	2.64	0.05	1.35	3.51	2.4	2.96
P.J. ♀	64	15	RP 4.3 × 10	Yes	—	—	—	1.85	2.71	2.28
P.L. ♂	46	14	NP 3.5 × 16	Yes	1.1	2.35	1.73	2.06	2.82	2.44
S.T. ♂	57	26	NP 3.5 × 10	Yes	0.4	0.65	0.53	0.45	0.84	0.65
S.G. ♀	54	14	NP 3.5 × 10	No	4.9	3.86	4.38	Failure	Failure	Failure
V.W.J. ♂	62	34	RP 4.3 × 10	No	0.87	1.28	1.08	1.6	1.34	1.47
D.S.G. ♂	54	24	NP 3.5 × 13	Yes	3.18	2.01	2.60	2.7	2.44	2.57

Statistical Methods

Descriptive statistics were used for the clinical and microbiologic data and to report the histologic findings. The radiographic bone level data were used as the primary outcome measure of implant success. The paired Wilcoxon signed-rank test was used for the assessment of radiographic bone loss, declaring a significant difference at $p < .05$.

RESULTS

Implants were on average 23.4 months (SD 2.7 months; range 19–27) in function. A total of 12 implants were installed: one of 16-mm length, three of 13-mm length, and eight of 10-mm length. Implant diameter was 5 mm in one implant, 4.3 mm in six implants, and 3.5 mm in five implants. Of the 10 treated subjects, four were women and six were men, with a mean age of 54.7 years (SD 5.4 years; range 46–64 years). Only one subject was a smoker. The implant in this subject belonged to the successful implants and remained in the study.

All implants were installed with a maximum of 40-Ncm insertion torque. A summary of subject selection, implant type, surgical procedure, and true bone loss is summarized in Table 1.

After 6 months and 2 years of loading, respectively, 12 and 9 implants were checked and all were found to be clinically stable and without clinical signs of inflammation. Between the two intervals, however, three implants had to be removed because of ongoing bone resorption and infection. The clinical survival rate was 100% at 6-month follow-up and 75% at 2-year follow-up. Based on marginal bone levels, the individual implant success was after 6 months and 2 years, 42 and 25%, respectively. A four-field distribution of one-piece implants according to the success criteria is shown in Tables 2 and 3. As an example of the variability in bone loss, radiographs of

TABLE 2 Four-Field Table Representing the Proportion of One-Piece Implants ($n = 12$) at 6 Months with Outcome: Success, Unaccounted for, Survival, and Failure according to Albrektsson and Zarb[25]

Success	42.6%
Survival	42.6%
Unaccounted	16.7%
Failure	0%

TABLE 3 Four-Field Table Representing the Proportion of One-Piece Implants ($n = 12$) at 2 Years with Outcome: Success, Unaccounted for, Survival, and Failure according to Albrektsson and Zarb[25]

Success	25%
Survival	50%
Unaccounted	0%
Failure	25%

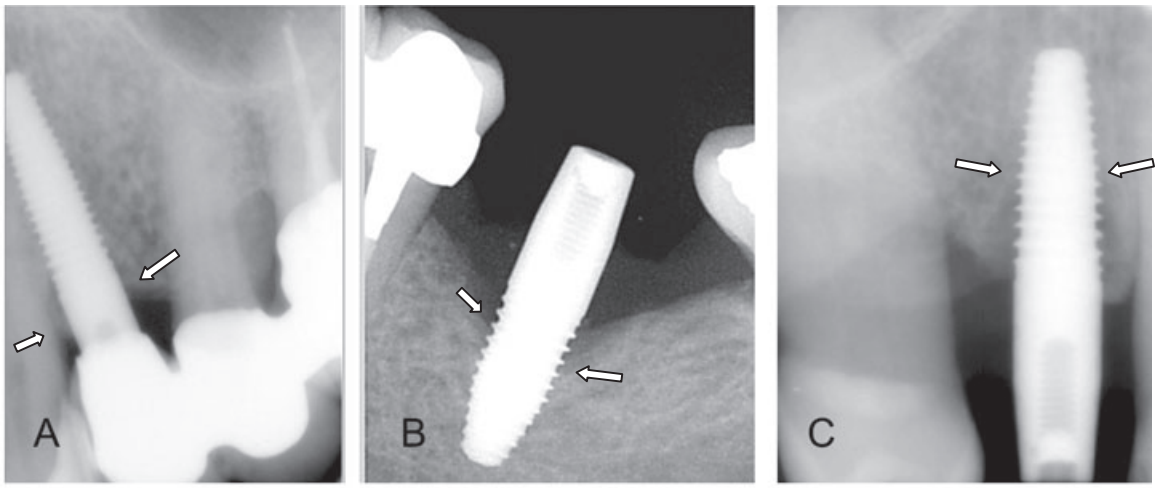

Figure 1 Composition of radiographs of three implants with 2 years of loading. Note the variability in bone loss and in some cases of extensive character (B and C). The white arrows are indicative for the radiographic bone level.

three implants with 2 years of loading are shown in Figure 1. Mean marginal bone levels at patient level are shown in Table 4 and illustrated at implant level in Figure 2. Readings were available from seven subjects for all evaluation intervals (Table 5). Statistically significant bone loss was detected after 6 months and 2 years.

Bacterial Samples

The distribution of bacteria present $\geq 1 \times 10^5$ cells are presented for implant samples (Table 6). A total of 45/76 species assessed were present in one or more samples from implant sites. The highest prevalence rates were found for *Fusobacterium* spp., *Leptorichia buccalis*, *Prevotella melaninogenica*, and *Veillonella parvula*, and all were present in all implant samples. In addition, *Aggregatibacter actinomycetemcomitans (Y4)*, *Staphylococcus aureus*, *Pseudomonas aeruginosa*, *Prevotella intermedia*, *Tannerella forsythia*, *Treponema denticola*, and *Treponema socransky* were also commonly present.

Chemical Analyses

Irrespective of the analyzed region, similar findings were obtained. Moreover, all three implants revealed similar contaminations with some minor differences between different implants. Besides the signals from Ti, O, and, C, peaks of P, F, S, N and Ca could be observed. The results from the chemical analyses on two different implants are shown in Figure 3.

TABLE 4 Mean Bone Level, SD, Range, and Number of Subjects in the Respective Intervals

Interval	Bone Level (mm)	SD (mm)	Range (mm)	*n*
Baseline	−0.74	0.57	−1.67 to 0.00	10
6 months	1.43	1.65	−1.14 to 4.24	8
2 years	1.11	1.02	−1.02 to 2.40	9

Retrieved Sample

The survey picture of the cut and ground section demonstrated the bone tissue to be located in the lower part of the implant. About 50% of the entire implants were lacking tissue in the upper region (Figure 4A). The distance from the implant neck to the first bony contact level was about 4 mm. The mean bone to implant contact based on all available threads was 19%, and the bone area inside the threads was 46%.

Along some of the bone free upper region of the implant surface, blue-stained areas/rims could be observed. No other signs of soft tissue or inflammatory cells could be observed in this area, that is, the implant surface was possibly covered by biofilm.

The uppermost bone tissue surface (cranial part) seemed to have been under resorption (clear reversal lines); however, new-formed woven bone-type tissues were located on the old preresorbed surface.

Focusing on the tissue outside the implant, that is, in the thread regions, in higher magnification, Haversian canals with clearly visible cement lines were located close to the implant surface (see Figure 4B), and several regions with compact mature-like bone tissue in "direct contact" to the implant surface could be seen. With the aid of polarizing filters, mostly lamellar bone (with

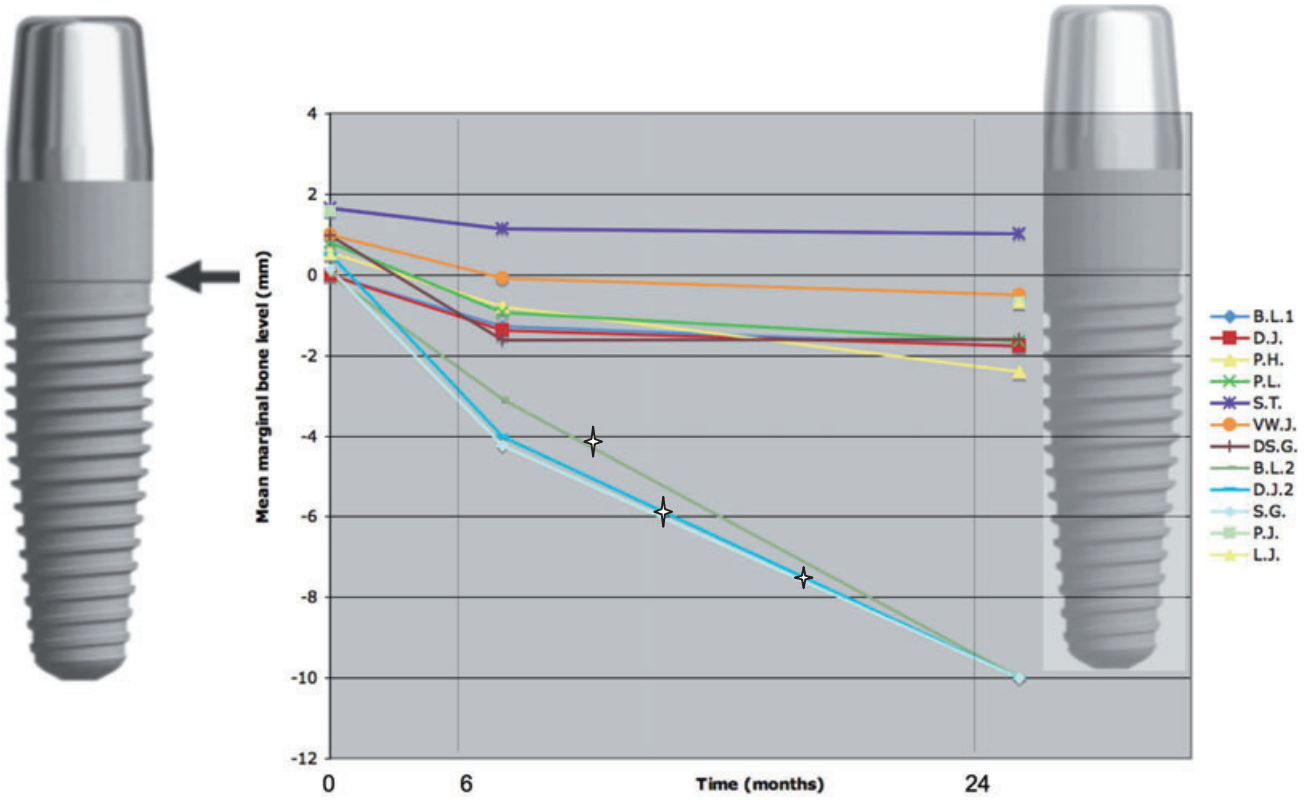

Figure 2 Marginal bone level according to the reference point for all implants. The lower corner of the coronal cylinder of the Nobel Direct implant was used as a reference point[14] from where on marginal bone level was calculated at the mesial and distal site. Lines marked with "*" represent the imaginary bone loss that occurred for 3 out of 12 implant failures.

various directions of the lamella) could be observed inside the threads (see Figure 4C). Moreover, some immature woven bone was also noted. Remodeling cavities were observed both inside the bone and close to the implant, revealing both active bone-forming and bone-resorption sites. Regions with entrapped "bone-dust particles" were seen being encapsulated in the bone. Osteocytes with empty and/or pycnotic nuclei, that is, devitalized osteocytes, were observed in close relation to the implant in some places. The innermost interfacial tissue had a darker stained rim in close apposition to the implant coat. If this "ceramic-like" tissue rim is because of the implant surface treatment, we cannot judge. In an even higher magnification, the presence of an irregular implant-surface coat (appearing brownish) could be observed, and at some parts, nonmineralized, osteoid-like tissue (lacking vital cells) was observed in close relation to the coat (see Figure 4D). Soft tissue regions were also observed in close relation to the implant surface. In these regions, macrophages and polymorphonuclear

TABLE 5 Marginal Bone Level Measured in Millimeter from the Implant Reference Point for Seven Subjects with All Evaluation Intervals Available up to 2 Years

Interval	Bone Level (mm)	SD (mm)	Range (mm)	Bone Loss (mm)	*p* Values
Baseline	−0.71	0.59	−1.67 to 0.00		
6 months	0.71	0.96	−1.14 to 1.63	1.42	0.018*
2 years	1.23	1.14	−1.02 to 1.76	0.52	0.028*

Changes with respect to previous interval are measured with Wilcoxon signed-rank test, and $p < .05$ is statistically significant and indicated with an asterisk.

TABLE 6 The Distribution of Bacteria Present ≥1 × 10^5 Cells for Implant Samples

Microorganism	Implant ($n = 8$) (%)
Aggregatibacter actinomycetemcomitans (Y4)	75.0
Aggregatibacter actinomycetemcomitans (b)	12.5
Actinomyces israelii	12.5
Actinomyces naeslundii	12.5
Actinomyces odontolyticus	12.5
Bacteroides ureolyticus	37.5
Campylobacer rectus	50.0
Campylobacter gracilis	37.5
Campylobacter showae	12.5
Capnocytophaga gingivalis	25.0
Capnocytophaga ochracea	37.5
Capnocytophaga sputigena	50.0
Eikenella corrodens	12.5
Eubacterium saburreum	12.5
Fusobacterium nucleatum ss.nucleatum	100.0
Fusobacterium periodonticum	100.0
Haemophilus influenzae	12.5
Lactobacillus acidophilus	37.5
Lactobacillus cispatus	62.5
Lactobacillus iners	12.5
Leptotrichia buccalis	100.0
Mobiluncus curtisii	12.5
Neisseria mucosa	62.5
Peptostreptococcus micros	37.5
Prevotella bivia	37.5
Prevotella disiens	12.5
Prevotella intermedia	62.5
Prevotella nigrescens	37.5
Porphyromonas gingivalis	37.5
Proteus mirabilis	12.5
Prevotella melaninogenica	100.0
Pseudomonas aeruginosa	25.0
Staphylococcus aureus	50.0
Streptococcus anginosus	12.5
Streptococcus constellatus	12.5
Streptococcus gordonii	50.0
Streptococcus intermedius	12.5
Streptococcus mitis	12.5
Streptococcus mutans	12.5
Streptococcus oralis	25.0
Streptococcus sanguinis	12.5
Tannerella forsythia	62.5
Treponema denticola	50.0
Treponema socransky	50.0
Veillonella parvula	100

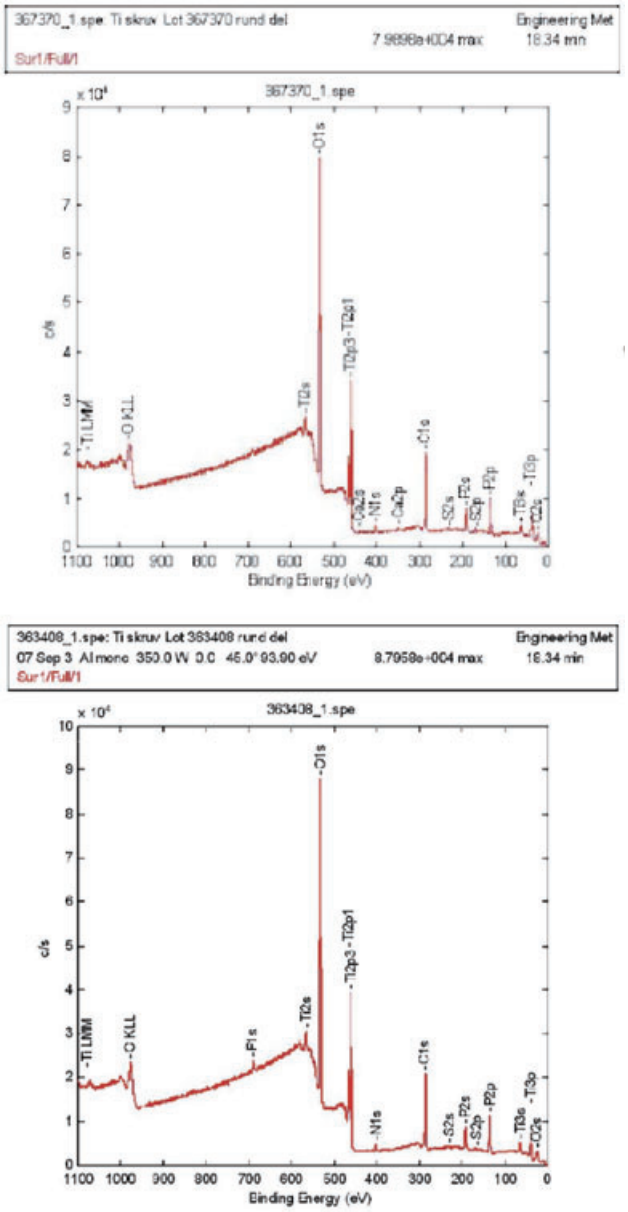

Figure 3 Results of the chemical analysis of two Nobel Direct implants with a different Lot number. Note the differences in peaks for Ca_2s/Ca_2p and F_1s between the two implants.

granulocytes (PMNGs) were noted, as well as some cells with possibly oxide particles internalized.

DISCUSSION

Implant success criteria have been described to scientifically evaluate a treatment outcome.[25] It is highly important that new implant designs are carefully evaluated preferably in multicenter prospective randomized clinical trials in comparison to a well-established successful implant system. The clinical reality is, however, often

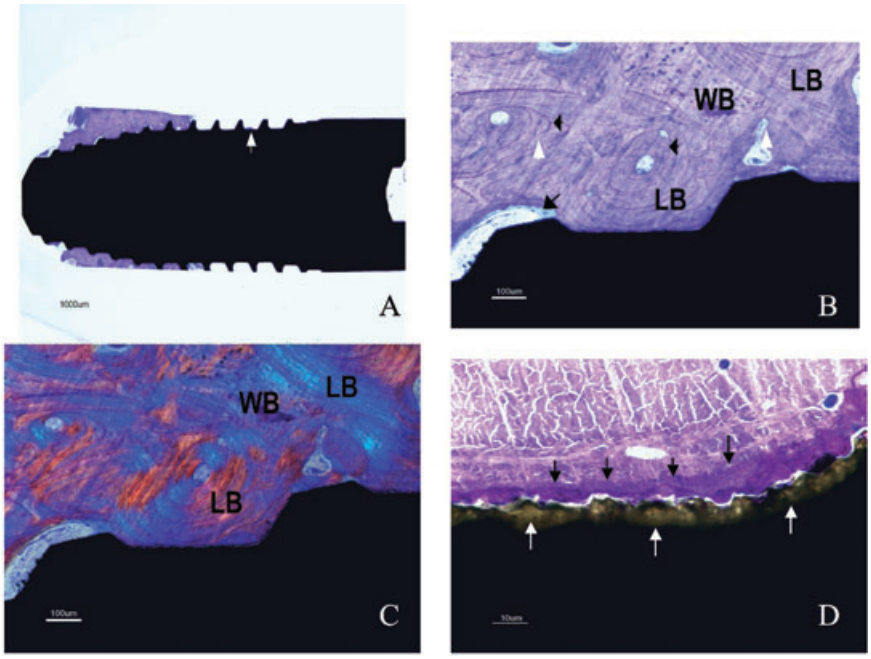

Figure 4 *A*, Survey picture of the 10-µm-thin undecalcified cut and ground section stained in Toluidine blue mixed with pyronin G. Arrow pointing at possibly biofilm remnants. Bar = 1,000 µm. *B*, Higher magnification of *A* revealing mostly mature, lamellar bone (LB) and some areas with immature, woven bone tissue (WB). Cement lines are clearly visible (small short arrows) in the LB. In some regions close to the implant surface, the presence of nonmineralized, osteoid-like tissue (lacking vital cells) could be observed. These regions could possibly be "disturbed bone mineralization areas" (longer arrow). Bar = 100 µm. *C*, Similar figure as in *B*, however, prepared with the aid of polarizing filter as well as a lambda filter. The directions of especially the LB are clearly visible. Bar = 100 µm. *D*, A dark-stained, a-cellular rim revealing a "ceramic-like tissue" (black arrows) could be observed outside the irregular brownish coat (white arrows). The white space between the tissue and the coat may be because of fixative artifacts, that is, shrinkage of the tissue. Bar = 10 µm. Note pycnotic-like osteocytes to the left. Bar = 10 µm.

more demanding than objective scientific criteria. From the patient's point of view, aesthetics, comfort, and treatment satisfaction need to be considered as the patient's treatment outcome can only be successful or unsuccessful. In order to be clinically used, an implant system should be thoroughly investigated during a reasonably long period of time. If a significant change in implant configuration is proposed, the advantages of the new design have to be scientifically proven by means of randomized controlled clinical trials.[11] The literature concerning the Nobel Direct implant system lacks clear evidence of good clinical results. Hahn[26] reported a cumulative survival rate (CSR) of 97.9% after 3 years. The proportion of subjects, however, who reached the 2- and 3-year follow-up was only 16/30 and 4/30, respectively. CSRs are a statistical way of boosting scientific figures as they do not take into account the dropouts or the subjects who have not passed a certain time period. According to the study by Hahn,[26] CSR after 3 years was 97.9%, but only 75% if the success was assessed according to Albrektsson and Zarb.[25] Furthermore, lack of baseline radiographic information makes it impossible to assess the study outcome based on clearly defined radiographic criteria.

Finne and colleagues[15] evaluated 152 Nobel Direct and Nobel Perfect® one-piece implants. They reported radiographic bone level measurements at baseline, 6 months, 1 year, and 2 years, reporting mean values of 0.33 mm, −0.77 mm, −0.98 mm, and 0.17 mm, respectively. Although they started with 152 implants, they only had radiographs of 141, 138, 123, and 26 implants at respectively implant placement, 6 months, 1 year, and 2 years. After 1 year of function, 21 implants (18%) showed bone levels of >2.0 mm under the reference point. A retrospective study of Siepenkothen[18] reported a mean bone loss during the first year of 0.91 mm (±1.27 mm). The studies of Finne and colleagues[15] and Siepenkothen[18] have similar results compared with studies evaluating radiographic bone levels around two-piece implants.[10,27] However, the authors of both studies failed to relate the radiographic bone measurements to their implant success criteria. Finne and colleagues[15]

modified the success criteria as proposed by Albrektsson and colleagues,[28] ignoring the radiographic bone level, and Siepenkothen[18] did not mention success rates.

Östman and colleagues[17] evaluated Nobel Direct implants according to success criteria somewhat less strict than those proposed by Albrektsson and colleagues.[28] They accepted more bone loss dividing the studied implants in grade 1 (<2-mm bone resorption at 1-year follow-up) and grade 2 success (<3-mm bone resorption at 1-year follow-up). When applying those less strict criteria, implant success grade 1 was 46.1% for Nobel Direct implants compared with 85.5% for a two-piece implant control group. The corresponding success rates when applying the grade 2 criteria were 72.2 and 91.6% for one-piece implants compared with two-piece implants.

The Nobel Direct one-piece implant system was believed to preserve peri-implant soft tissue and marginal bone levels.[15] When using a two-piece implant system, abutments need to be changed during the treatment period before the delivery of the final prosthesis. This leads to disruption of the soft tissue seal and additional bone resorption.[29] The implant-abutment connection of clearance fit implant systems seems to be unstable under loading conditions.[30] In this in vitro study, the author showed the micromotion of the implant-abutment interface when load was put on the abutment. This could theoretically, in a clinical situation, lead to the formation of a wider zone of inflammatory cell tissue around the microgap, resulting in more peri-implant bone loss.[31] A one-piece implant system combines the intraosseous threaded implant body, the transmucosal abutment, and the pillar for crown cementation in a single piece. The abutment portion of the implant can be prepared, which makes it possible to create an individualized preparation borderline that follows the anatomy of the soft tissue margin, without violating the soft tissue seal. The Nobel Direct one-piece implant system was, in this philosophy, believed to have better soft and hard tissue responses compared with a two-piece implant system.[12] However, this could not be shown in clinical reports as discussed above. Moreover, the success rates are inconclusive and when critically analyzed, are worryingly low, indicating that the Nobel Direct implants perform worse than conventional implants.

It was therefore decided by the authors to retrospectively evaluate the treatment outcome of a series of Nobel Direct implants placed in a private practice setting. The results of this study clearly show that the failure rate and the proportion of unsuccessful Nobel Direct implants are higher compared with validated implant systems. According to the success criteria of Albrektsson and Zarb,[25] the individual implant success was after 6 months and 2 years, respectively, 42 and 25%. This means that after 2 years, only one of four subjects had acceptable bone levels when radiographically evaluated. Sennerby and colleagues[32] indicated that flapless implant placement and immediate loading might be a possible reason for a higher failure rate of Nobel Direct implants. It can be assumed that a nonguided flapless surgical technique affects the correct positioning of implants within the alveolar bone, resulting in perforations and exposure of the treaded part of an implant. This was showed in an in vitro study[33] evaluating the possible complications encountered with nonguided flapless implant surgery. The positioning of implants placed with a flapless approach deviated significantly from the ideal position, resulting in perforations in 59.7% of the investigated implant sites.

There are few studies that have assayed on microbiologic distributions in implant health and disease by checkerboard DNA hybridization over longer time periods. A recent study by Renvert and colleagues[34] demonstrated that at healthy implant sites, *S. aureus* was found at 8% of the sites, *Porphyromonas gingivalis* at 8%, and *A. actinomycetemcomitans (Y4)* at 3%. In the present study, these bacteria were found at 50, 75, and 38%, respectively. The implants investigated in the study by Renvert and colleagues[34] were all Brånemark implants with a machined surface and placed before the year 2000. The present study suggested that the rough surface not covered by bone could act as a microbial niche for select bacterial colonization of significant pathogens.

The histologic analysis of the retrieved implant in this study showed some bone to implant contact on the lower part of the implant. The upper part of the implant surface was covered by biofilm and lacked bone. This means that the infection and bone loss around the upper half of the implant did not affect the osseointegration below. Some pycnotic cells and an atypical ceramic-like tissue in close relation to the surface of the implant could be seen. No conclusions can be drawn, however, based on this individual sample.

CONCLUSIONS

Radiographic evidence showed a very low success rate for the Nobel Direct implants in this study. High counts and prevalence of significant pathogens were found at surviving implants. Although extensive bone loss had occurred in the coronal part, the apical portion of the implant showed some bone to implant integration.

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