Long-Term Clinical, Microbiological, and Radiographic Outcomes of Brånemark™ Implants Installed in Augmented Maxillary Bone for Fixed Full-Arch Rehabilitation

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ABSTRACT

Purpose: The purpose of this study was to document the long-term outcome of Brånemark implants installed in augmented maxillary bone and to identify parameters that are associated with peri-implant bone level.

Material and Methods: Patients of a periodontal practice who had been referred to a maxillofacial surgeon for iliac crest bone grafting in the atrophic maxilla were retrospectively recruited. Five months following grafting, they received 7–8 turned Brånemark implants. Following submerged healing of another 5 months, implants were uncovered and restorative procedures for fixed rehabilitation were initiated 2–3 months thereafter. The primary outcome variable was bone level defined as the distance from the implant-abutment interface to the first visible bone-to-implant contact. Secondary outcome variables included plaque index, bleeding index, probing depth, and levels of 40 species in subgingival plaque samples as identified by means of checkerboard DNA–DNA hybridization.

Results: Nine out of 16 patients (eight females, one male; mean age 59) with 71 implants agreed to come in for evaluation after on average 9 years (SD 4; range 3–13) of function. One implant was deemed mobile at the time of inspection. Clinical conditions were acceptable with 11% of the implants showing pockets ≥ 5 mm. Periodontopathogens were encountered frequently and in high numbers. Clinical parameters and bacterial levels were highly patient dependent. The mean bone level was 2.30 mm (SD 1.53; range 0.00–6.95), with 23% of the implants demonstrating advanced resorption (bone level > 3 mm). Regression analysis showed a significant association of the patient ($p < .001$) and plaque index ($p = .007$) with bone level.

Conclusions: The long-term outcome of Brånemark implants installed in iliac crest-augmented maxillary bone is acceptable; however, advanced peri-implant bone loss is rather common and indicative of graft resorption. This phenomenon is patient dependent and seems also associated with oral hygiene.

KEY WORDS: bone augmentation, bone level, dental implant, maxilla, microbiology

INTRODUCTION

Implant treatment is a common and straightforward procedure in most patients. However, bone resorption, secondary to periodontal disease, tooth loss, or ill-fitting prostheses, may lead to severe atrophy,[1] requiring reconstructive surgery prior to implant placement. Several procedures using inlay and onlay techniques have been described. The former include Le Fort I osteotomy with

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interpositional bone grafting and sinus augmentation.[2,3] Onlay techniques are used for horizontal ridge augmentation usually using autogenous bone blocks. If limited bone gain is needed, the chin or retromolar region may serve as suitable donor sites. Large volumes require extraoral donor sites such as the iliac crest or calvarium.

An important concern following all bone augmentation procedures is volume stability of the graft. Even though no augmentation technique has detailed documentation or long-term follow-up studies as described in a recent systematic review,[4] graft resorption seems inevitable.[5] Bone blocks from intraoral donor sites may lose from up to 50% to 60% of their volume after 1 year.[6,7] When applied as an onlay graft, bone blocks from the iliac crest have also shown considerable resorption of nearly half of their volume after 1 year.[8]

Another issue relates to the survival and bone remodeling of dental implants installed in augmented bone. Implant survival rates of about 75% have been reported for turned titanium implants placed in the iliac crest-augmented maxilla after 3–5 years of function.[9,10] Although a recent long-term study described higher survival rates,[11] these findings suggest that turned titanium implants could be more prone to failure when installed in augmented maxillary bone. Limited data exist on bone adaptation around turned titanium implants installed in the iliac crest-augmented maxilla. Adell and co-workers[9] described a mean bone level of 1.49 mm in reference to the implant-abutment interface after 1 year, and about 0.10 mm annually thereafter. This corresponds quite well with recent findings by Nyström and colleagues[11] pointing to a mean bone level of 2.40 mm after 10 years of function. Still, frequency distributions and microbiological data have never been described and no attempt has been made so far to identify parameters that are associated with bone level under these conditions.

Hence, the primary goal of this study was to evaluate the long-term overall outcome of turned titanium implants installed in iliac crest-augmented maxillary bone. A secondary objective was to identify parameters that are associated with peri-implant bone level.

MATERIALS AND METHODS

Patient Selection

Patients of a periodontal practice were retrospectively recruited for a cross-sectional evaluation based on the following inclusion criteria:

1 all had been referred to a maxillofacial center (AZ Sint-Jan, Bruges, Belgium) for advanced bone augmentation in the fully edentulous atrophic maxilla;
2 bone augmentation was performed by one and the same experienced maxillofacial surgeon (CDC);
3 bone augmentation included onlay grafting using iliac crest bone blocks in combination with sinus augmentation if necessary;
4 implant surgery was performed by two experienced periodontists (HDB, BC) using turned Brånemark implants (Nobel Biocare, Göteborg, Sweden); and
5 implants were restored with a full-arch bridge.

The study was conducted in accordance with the Helsinki declaration of 1975 as revised in 2000 and the protocol was approved by the ethical committee of the University Hospital in Ghent (UZ Gent).

Surgical Procedures

Reconstructive surgery was performed under general anesthesia with oral endotracheal intubation. A small incision was made at the spina iliaca anterior superior making access to the iliac crest. Monocortical bone blocks and cancellous bone were harvested (Figure 1). Thereafter, the wound was closed in layers. Intraorally, a crestal incision was made to raise a mucoperiosteal flap exposing the alveolar process and lateral sinus walls if necessary. The cortical iliac bone blocks were used as saddle or veneer onlay grafts at the buccal side of the atrophic ridge and fixed with small titanium screws. If necessary, sinus augmentation was performed with cancellous bone as filling material. In order to avoid tension on the flap, a horizontal periosteal incision was made.

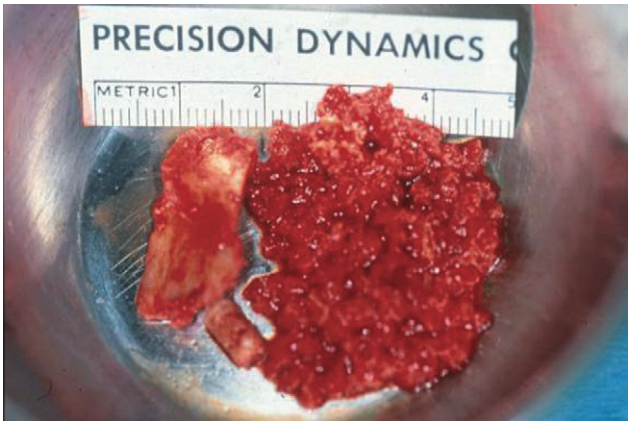

Figure 1 Harvesting of monocortical and cancellous bone from the iliac crest.

Closure of the flap was achieved by means of single sutures. All patients were discharged from the hospital after a few days and were not allowed to wear removable prostheses for at least 1 month.

After a healing period of 5 months, patients were sent back to the referring practice for implant surgery under local anesthesia. A full-arch mucoperiosteal flap was raised following crestal incision and all fixation screws were removed. Seven to eight turned Brånemark implants (Nobel Biocare, Göteborg, Sweden) were placed subcrestally to reduce the risk for perforation as a result of graft resorption. Following submerged healing of another 5 months, implants were uncovered and healing abutments were installed. The general dentist initiated the restorative procedure for fixed rehabilitation of 2–3 months thereafter.

Clinical Evaluation

All patients were examined by one and the same trained clinician who had not been involved in the treatment (SV). Plaque and bleeding indices were recorded at six sites per implant (mesial, central, distal, buccally as well as orally). Both included a score ranging from 0 to 3.[12] Following removal of the bridge, probing depth was measured to the nearest 0.5 mm using a manual periodontal probe (CP 15 UNC, Hu-Friedy®, Chicago, IL, USA) at the same six sites. Removal of the bridge also allowed for evaluation of implant mobility. Patient's records were scrutinized for implant failure that had occurred in the past.

Microbiological Evaluation

Subgingival microbial samples were obtained from the deepest pocket of each implant just prior to removal of the bridge. Supragingival plaque was removed with sterile cotton pellets, and a sterile paper point (Mynol Plus, Ada Products, Milwaukee, WI, USA) was then inserted into the pocket until resistance was felt. After leaving it in situ for 20 seconds, each paper point was placed in a separate sterile and dry Eppendorf tube. All samples were immediately stored at –20°C in Ghent and mailed to the processing center in Berne within 30 days. There, microbiological analysis was performed using the checkerboard DNA–DNA hybridization technique. This assay included a panel of 40 bacterial species. Details of the procedures have been described elsewhere.[13–16] Briefly, the samples were individually placed in Eppendorf tubes containing 0.15 mL TE (10 mM Tris-HCL, 1 mM ethylenediaminetetraacetic acid, pH 7.6). Within 30 minutes, 0.1 mL 5 M NaOH was added to each tube. Bacterial DNA was extracted, concentrated on nylon membranes (Roche Diagnostics GmbH, Mannheim, Germany), and fixed by cross-linking using ultraviolet light (Stratalinker 1800, Stratagene, La Jolla, CA, USA). The membranes with fixed DNA were placed in a Miniblotter 45 (Immunetics, Cambridge, MA, USA). A 30×45 "checkerboard" pattern was produced as described by Socransky and colleagues[13] and Katsoulis and colleagues.[14] Chemiluminescent signals were detected using the Storm Fluor-Imager (Storm 840, Amersham Biosciences, Piscataway NJ, USA). In order to receive a full detailed account of the identified bacteria, the digitized information was analyzed by a software program (ImageQuant, Amersham Pharmacia, Piscataway, NJ, USA), allowing comparison of signals against standard lanes of known bacterial amounts. Signals were converted to absolute counts by comparison with these standards and studied as the proportion of sites defined as having $\geq 1 \times 10^4$ bacterial cells. The outcome variables for all 40 species were detection frequency and bacterial level.

Radiographic Evaluation

A digital peri-apical radiograph was taken of each implant with the long-cone paralleling technique and an x-ray holder (XCP Bite Block, Dentsply Rinn, Elgin, IL, USA) with the implant bridge in place to enhance stability of the holder. The radiographic evaluation of the peri-implant bone level was done using Vixquick software (Amsterdam, the Netherlands) with an accuracy of 0.1 mm. The bone level was considered the primary outcome variable and was defined as the distance from the implant-abutment interface to the first visible bone-to-implant contact. All radiographs were analyzed twice by two clinicians (HDB, PB) in order to evaluate the intra- and interexaminer reliability. The implant success was rated based on the criteria by Albrektsson and Isidor.[17]

Statistical Analysis

Mean values were calculated for all parameters on an individual implant basis and descriptive statistics included frequency distributions (plaque index, bleeding index, probing depth, bone level) and detection frequencies (microbiota). Intraexaminer and interexaminer reliability on bone levels was assessed using

percent agreement within 0.2 mm deviation, Pearson's correlation coefficients, and the paired *t*-test.

The impact of the patient factor on clinical parameters and bacterial levels was evaluated using the Kruskal–Wallis test. In order to explore the association between clinical and microbiological conditions, the implant with the lowest and highest probing depth, respectively, were selected for each patient. Paired omparisons in terms of bacterial levels were performed using the Wilcoxon signed ranks test.

A general linear model was used to examine the association of the patient, clinical parameters, and total DNA count with bone level (dependent variable). Therefore, the patient was considered a random factor whereas plaque index, bleeding index, probing depth, and total DNA count were included as covariates. A residual analysis on linearity and homoskedasticity was performed to evaluate the model fit. The level of significance was set at 0.05.

RESULTS

Sixteen patients met the inclusion criteria. Four were lost to follow-up. The remaining 12 were contacted and nine (eight females, one male; mean age 59, SD 10; age range 52–73) agreed to come in for evaluation. These were all nonsmokers, in good general health, and receiving supportive care at least once a year. One patient had received seven implants, the others eight implants. The mean time in function was 9 years (SD 4; range 3–13). Fifty-four out of 71 implants were in function for at least 6 years.

Clinical Outcome

From the 71 implants, one presented with a buccal fistula and was found mobile when the bridge was removed. According to the patient's records, there had not been any other preceding failures.

Table 1 shows the clinical outcome in terms of plaque index, bleeding index, and probing depth on implant level and patient level. Overall, clinical conditions were acceptable albeit high variation was observed. Forty-four percent of the implants were plaque free; whereas 41% showed high plaque levels (plaque index ≥ 1). Thirty-eight percent of the implants were free of inflammation as determined by the bleeding index whereas 10% showed high bleeding tendency (bleeding index ≥ 1). Forty-nine percent of the implants demonstrated shallow pockets (probing depth ≤ 3 mm) whereas 11% showed deep pockets (probing depth ≥ 5 mm). Statistical analyses showed a significant impact of the patient on all clinical parameters ($p < .001$).

Microbiological Outcome

Table 2 shows detection frequencies and levels of the 40 species included in the panel. Twenty-five out of 40 species were detected around the vast majority of the implants (detection frequency ≥ 80%). In addition, 17 of these bacteria were found in high numbers (level ≥ 1×10^5). Large variation in bacterial levels was observed between implants. *Fusobacteria, Leptotrichia buccalis, Parvimonas micra, Veillonella parvula*, and especially *Tannerella forsythia* showed the highest levels well surpassing 2×10^5 bacterial counts.

When the implant with the lowest and highest probing depth was compared on a patient level, implants with shallow pockets showed significantly lower counts of the following species: *Actinomyces odontolyticus* ($p = .048$), *Campylobacter gracilis* ($p = .047$), *Fusobacterium nucleatum naviforme* ($p = .039$), and *Leptotrichia buccalis* ($p = .018$). The total DNA count was not significantly different between implants with shallow and deep pockets ($p = .097$).

TABLE 1 Clinical Outcome of Implants Installed in Augmented Maxillary Bone

	Implant Level ($n = 70$)				Patient Level ($n = 9$)			
Outcome Variable	Mean	SD	Minimum	Maximum	Mean	SD	Minimum	Maximum
Plaque index	0.65	0.68	0.00	2.00	0.63	0.60	0.00	1.48
Bleeding index	0.35	0.38	0.00	1.67	0.35	0.31	0.00	0.88
Probing depth (mm)	3.56	1.10	2.00	7.67	3.55	0.86	2.85	5.10

SD = standard deviation.

TABLE 2 Microbiological Outcome of Implants Installed in Augmented Maxillary Bone

Species	Collection	Detection Frequency (%)	Bacterial Level ($\times 10^5$)			
			Mean	SD	Minimum	Maximum
Aggregatibacter actinomycetemcomitans (a)	ATCC 29523	85	0.97	0.81	0.00	2.95
Aggregatibacter actinomycetemcomitans (Y4)	ATCC 43718	92	1.94	1.62	0.00	5.62
Actinomyces israelii	ATCC 12102	38	0.22	0.32	0.00	1.05
Actinomyces naeslundii (types I + II)	ATCC 43146	50	0.27	0.43	0.00	1.95
Actinomyces odontolyticus	ATCC 17929	60	0.49	0.65	0.00	3.16
Campylobacter gracilis	ATCC 33236	91	1.36	1.17	0.00	5.13
Campylobacter rectus	ATCC 33238	94	3.03	1.93	0.00	7.59
Campylobacter showae	ATCC 51146	98	1.34	1.42	0.06	6.03
Capnocytophaga gingivalis	ATCC 33612	85	1.07	0.93	0.00	4.47
Capnocytophaga ochracea	ATCC 33596	72	1.77	1.62	0.00	5.89
Capnocytophaga sputigena	ATCC 33612	58	0.89	0.96	0.00	3.09
Eikenella corrodens	ATCC 23834	52	0.34	0.45	0.00	1.82
Eubacterium saburreum	ATCC 33271	95	1.52	1.28	0.00	5.37
Fusobacterium nucleatum nucleatum	ATCC 25586	98	3.78	3.95	0.17	15.14
Fusobacterium nucleatum polymorphum	ATCC 10953	66	2.80	3.76	0.00	18.20
Fusobacterium nucleatum naviforme	ATCC 49256	91	3.43	6.11	0.00	30.90
Fusobacterium periodonticum	ATCC 33693	98	2.58	2.42	0.15	11.22
Lactobacillus acidophilus	ATCC 11975	81	0.71	0.77	0.00	2.82
Leptotrichia buccalis	ATCC 14201	97	2.98	2.17	0.00	6.92
Parvimonas micra	ATCC 19696	94	2.16	2.60	0.00	9.55
Neisseria mucosa	ATCC 33270	39	0.70	1.13	0.00	4.47
Prevotella intermedia	ATCC 25611	57	0.62	0.75	0.00	3.55
Prevotella melaninogenica	ATCC 25845	92	1.48	1.36	0.00	6.76
Prevotella nigrescens	ATCC 33563	54	0.51	0.76	0.00	4.17
Porphyromonas gingivalis	ATCC 33277	58	1.20	4.95	0.00	38.02
Propionibacterium acnes (types I + II)	ATCC11827/28	45	0.16	0.21	0.00	0.72
Selenomonas noxia	ATCC 43541	58	0.44	0.55	0.00	2.29
Staphylococcus aureus	ATCC 25923	88	1.14	1.26	0.00	4.90
Streptococcus anginosus	ATCC 33397	87	0.94	1.00	0.00	3.98
Streptococcus constellatus	ATCC 27823 M32b	84	0.83	0.77	0.00	3.09
Streptococcus gordonii	ATCC 10558	94	1.04	0.95	0.00	3.80
Streptococcus intermedius	ATCC 27335	95	0.75	0.75	0.00	2.95
Streptococcus mitis	ATCC 49456	91	0.88	0.82	0.00	2.16
Streptococcus oralis	ATCC 35037	92	0.56	0.61	0.00	2.57
Streptococcus sanguinis	ATCC 10556	82	0.67	0.70	0.00	2.88
Streptococcus mutans	ATCC 25175	95	1.47	0.90	0.00	3.71
Tannerella forsythia	ATCC 43037 (338)	75	6.32	16.86	0.00	77.62
Treponema denticola	ATCC 35405	85	1.93	2.56	0.00	16.22
Treponema socranskii	D40DR2*	85	1.53	1.64	0.00	10.00
Veillonella parvula	ATCC 10790	70	2.10	4.67	0.00	29.51

ATCC = American Type Culture Collection; SD = standard deviation.
*D: sample from Forsyth Institute, Boston, MA, USA.

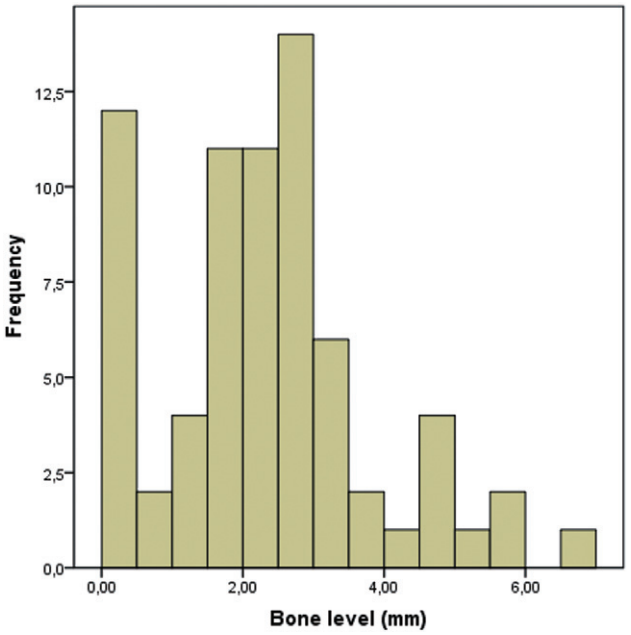

Figure 2 Frequency distribution of bone level.

Statistical analyses showed a significant impact of the patient on the levels of 37 out of 40 species ($p \leq .024$).

Radiographic Outcome

The intraexaminer repeatability on bone levels was high (84% agreement within 0.2-mm deviation; Pearson's correlation coefficient: 0.977-$p < .001$; paired t-test: $p = .761$), as was the interexaminer reproducibility (76% agreement within 0.2-mm deviation; Pearson's correlation coefficient: 0.948-$p < .001$; paired t-test: $p = .803$).

The mean bone level was 2.30 mm (SD 1.53; range 0.00–6.95) when analyzing data on implant level. A frequency distribution is given in Figure 2 illustrating high variation. Forty-one percent of the implants showed acceptable bone preservation (bone level < 2 mm) whereas 23% demonstrated bone levels extending to or beyond the third implant thread (bone level > 3 mm). Based on the success criteria by Albrektsson and Isidor,[17] 49 out of 71 (69%) implants could be considered successful.

When analyzing data with the patient as the experimental unit, the mean bone level was 2.29 mm (SD 1.06; range 0.10–3.52). Figure 3 indicates high variation within as well as between patients.

Parameters Associated with Bone Level

Table 3 shows the results of the regression analysis with bone level as the dependent variable. The covariates demonstrated no meaningful correlation with each other (Pearson correlation coefficient ≤ 0.401). Hence, the data set showed no multicollinearity, which is mandatory to have in all parameters included in the analysis. The regression model was highly significant ($p < .001$) and

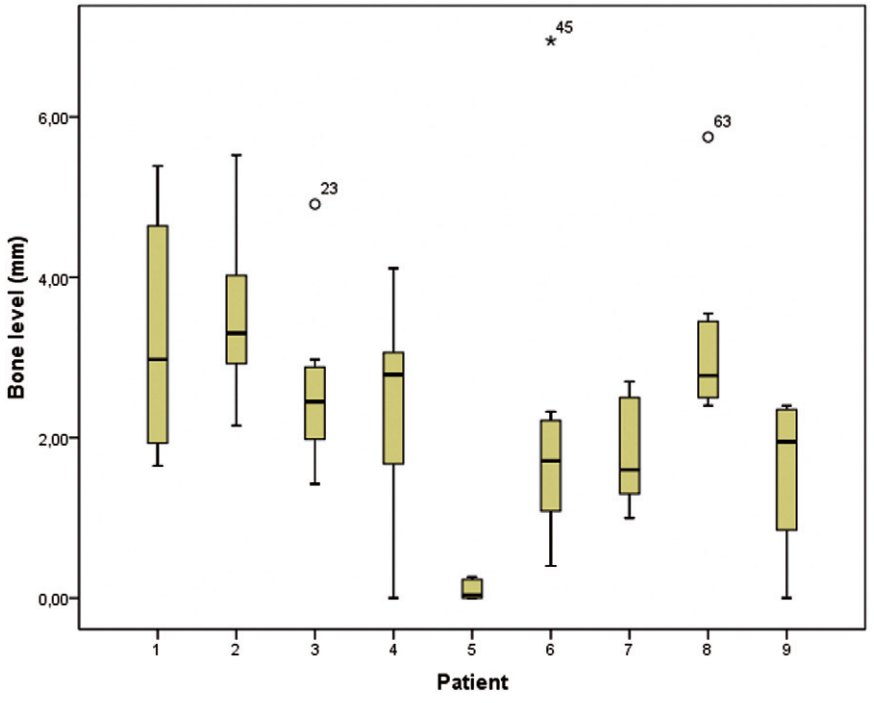

Figure 3 Boxplots illustrating the variability in bone level.

TABLE 3 Regression Analysis with Bone Level as Dependent Variable

Parameter	*p*-Value	Regression Coefficient B	Standard Error	95% Confidence Interval
Regression model	<.001	N/A	N/A	N/A
Intercept	.152	1.068	0.734	−0.407; 2.543
Patient	<.001	N/A	N/A	N/A
Plaque index	.007	1.035	0.368	0.296; 1.774
Bleeding index	.705	−0.233	0.610	−1.460; 0.994
Probing depth	.884	−0.027	0.185	−0.398; 0.344
Total DNA count	.100	0.005	0.003	−0.001; 0.011

N/A = not applicable.
$R^2 = 0.632$.

the patient was most decisive for bone level ($p < .001$) followed by the oral hygiene status (plaque index; $p = .007$). No other clinical nor microbiological parameters were significant predictors for bone level ($p \geq .100$). Sixty-three percent of the variability in the dependent variable could be explained by the regression model (R^2: 0.632). The model quality was satisfying given the linear relationship and homoskedasticity of the residuals (Figure 4). An illustration on the prediction of bone level by the regression model is given in Figure 5.

DISCUSSION

According to the international literature the survival of dental implants installed in native bone is about 93% in the long term.[18,19] Turned titanium implants installed in the augmented atrophic maxilla have shown lower survival rates ranging from 75 to 90%.[9–11] In contrast, high survival rates surpassing 96% have been described for surface-modified implants under comparable conditions.[20,21] These observations suggest that surface-modified implants may be less prone to failure than turned titanium implants when installed in the augmented atrophic maxilla. Interestingly, the fact that we only encountered one implant failure in our study may deviate from this viewpoint. However, one should take into account a possible oversimplification in this study having only evaluated nine out of 16 eligible patients. On the other hand, three additional patients were

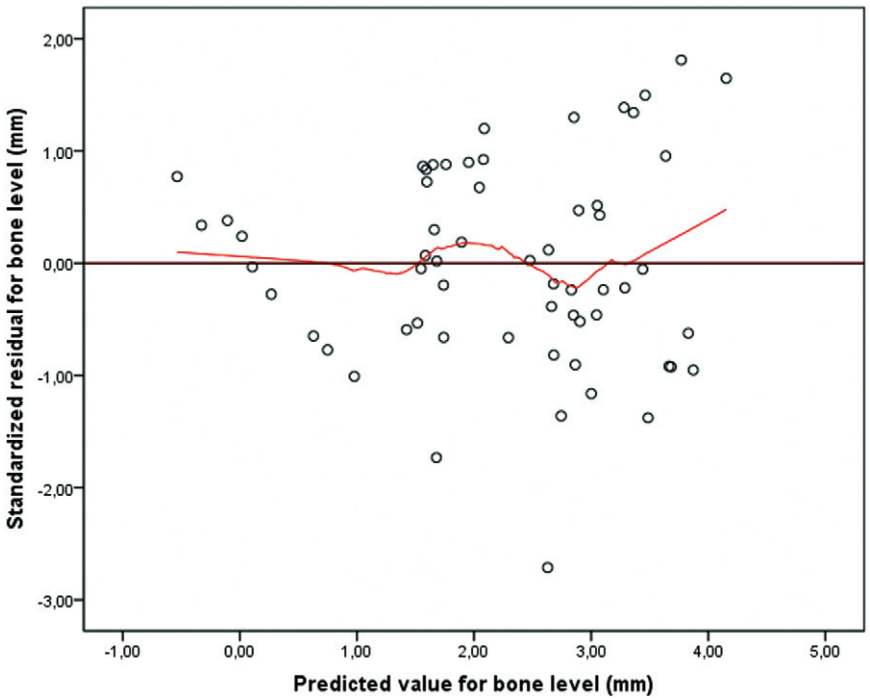

Figure 4 Scatterplot illustrating the model quality in terms of linearity and homoskedasticity.

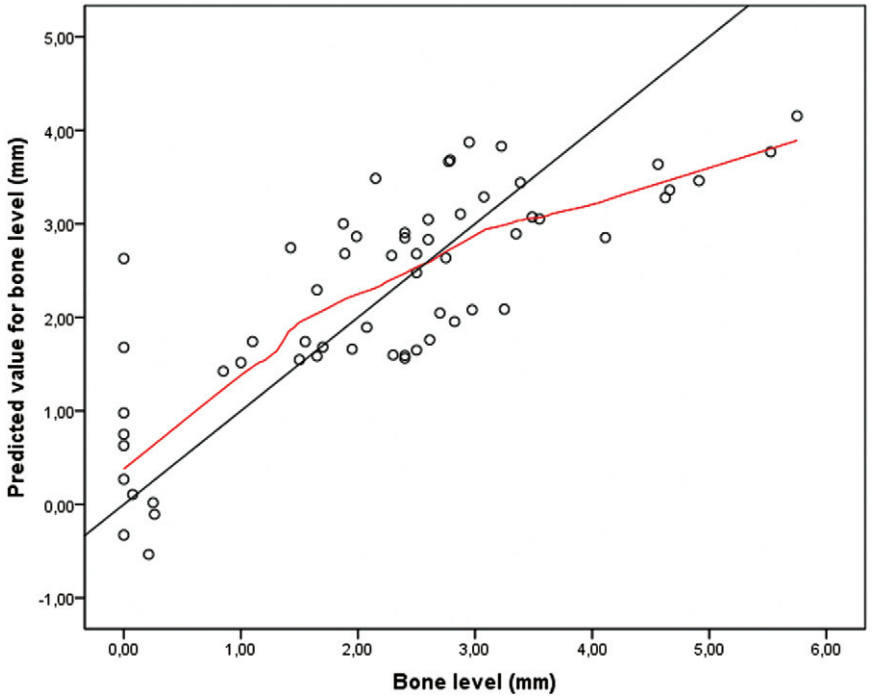

Figure 5 Scatterplot illustrating the prediction of bone level by the regression model.

contacted by phone and confirmed the presence of all implants, and the records of the remaining four patients did not reveal any failures up to their last visit.

For this study, it was decided to remove the implant bridge in all patients because of the following: first, it has been shown that limited access to the peri-implant sulcus is quite prevalent. This seems related to the prosthetic design and may compromise accurate registration in 15% of the sites.[22] Second, removal of the bridge enabled us to evaluate implant mobility which is considered one of the main criteria for success by Albrektsson and Isidor.[17]

By and large, the implants under investigation showed an acceptable clinical outcome. Still, periodontopathogens were frequently and in high numbers identified in the peri-implant sulcus, which is in accordance with previous findings based on checkerboard DNA–DNA hybridization.[15,23,24] An important observation of the present study was the high variation in clinical and microbiological conditions of the implants, which was principally patient-related. When controlling for the patient factor, a significant clinical-microbiological link was only found for four out of 40 species. Even though our study was clearly not designed for this purpose, the level of significance was of marginal magnitude for these species and probably related to multiple testing. Indeed, when 40 independent tests are performed each at the 0.05 significance level, the probability that one or more will achieve significance by chance is 87% ($1–0.95^{40}$). Therefore, we believe our data may not support a link between clinical and microbiological peri-implant conditions within the same patient. This is in agreement with Renvert and co-workers[23] showing trivial difference in the microbiota between healthy implants and implants suffering from peri-implantitis. However, the observation that the patient was a highly decisive factor for the peri-implant microbial profile in this study, suggests a pivotal impact of genetic background possibly overruling local factors. This view has never been addressed before, yet would reflect recent insights in periodontal disease basically showing that the microbial content of the periodontal pocket is determined by gene expression in the periodontal tissues.[25]

The primary outcome variable in this study was bone level defined as the distance from the implant-abutment interface to the first visible bone-to-implant contact. Mean bone level was 2.30 mm after an average of 9 years of function, which corresponds well with long-term findings by Nyström and colleagues[11] on the same treatment concept. Frequency analysis showed that 23% of our implants showed bone levels extending to or beyond the third implant thread. Even though this was

not a longitudinal investigation including data on baseline radiographs, we believe these cases are related to advanced bone loss because of the following. First, all implants had been inserted by two experienced implant surgeons according to a standard protocol of subcrestal implant placement. Second, a 3-mm bone level corresponding to the third implant thread clearly surpasses the measurement error of 0.8 mm (SD on the largest mean difference between duplicate readings multiplied by 2) for radiographic bone level analyses. Essentially, advanced bone loss, as we frequently encountered, could be the result of peri-implantitis and/or graft resorption. Peri-implantitis has been described as a complication of implant therapy with varying prevalence basically depending on the definition of the condition.[26] The present sample only included five out of 71 implants (7%) showing bone levels exceeding the physiological threshold as defined by Albrektsson and Isidor[17] and including clinical signs of inflammation (probing depth ≥ 5 mm and bleeding or pus). Interestingly, only two of these implants showed a circular crater indicative of peri-implantitis and therefore, it remains debatable whether even all five truly qualified as peri-implantitis cases. These findings suggest that graft resorption was the primary cause of advanced peri-implant bone loss, which would explain why the prevalence of implant cases with extreme bone levels was considerably higher than reported in the study by Jemt and Johansson[27] on turned Brånemark implants installed in native maxillary bone and in function for 10 years (23% vs 13% showing bone level ≥ 3 mm). Hence, iliac crest grafts seem prone to resorption in the long term. Regression analysis showed that this was predominantly patient related. Indeed, variability between patients was huge in terms of bone level ranging from 0.10 to 3.52 mm despite comparable reconstructive surgery. Bone level also varied substantially within patients, which was mainly attributed to disparities in plaque accumulation as shown by regression analysis.

In contrast to iliac bone grafts, calvarium bone grafts have shown limited resorption (<20%) in the short term when used for alveolar ridge reconstruction.[28] The latter seems also superior over iliac crest bone for sinus augmentation.[29,30] As a result and because of limited morbidity, bone grafting from the skull is more and more becoming part of current daily practice at the expense of iliac crest grafting. Given this evolution, it would be interesting to evaluate the long-term survival and bone adaption of implants installed in calvarium-augmented maxillary bone. This would preferably be assessed using surface-modified implants because these have become the standard in contemporary implant dentistry.

In conclusion, the long-term outcome of Brånemark implants installed in iliac crest-augmented maxillary bone is acceptable; however, advanced peri-implant bone loss is rather common and indicative of graft resorption. This phenomenon is patient dependent and seems to also be associated with oral hygiene.

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