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Published in final edited form as:

J Dent Res. 2009 April; 88(4): 367-371. doi:10.1177/0022034509334203.

Glycemic Control and Implant Stabilization in Type 2 Diabetes Mellitus

Thomas W. Oates, DMD, PhD¹, Scott Dowell, DDS, MS^{1,2}, Melanie Robinson, DDS, MS^{1,3}, and C. Alex McMahan, PhD⁴

¹ Department of Periodontics, University of Texas Health Science Center at San Antonio

⁴ Department of Pathology, University of Texas Health Science Center at San Antonio

Abstract

Diabetes mellitus is considered a relative contraindication to implant therapy. However, the effects of glycemic level on implant integration in patients with diabetes remains poorly understood. The hypothesis of the research was that poor glycemic control is directly related to short-term impairments implant stabilization. This prospective clinical study evaluated 10 non-diabetic patients (12 implants) and 20 type 2 diabetic patients (30 implants). Glycated hemoglobin (HbA1c) levels ranged from 4.7-12.6%. Implant stability was assessed using resonance frequency analysis over 4 months following placement. Minimum stability levels were observed 2-6 weeks following placement for all 42 implants. Patients with HbA1c \geq 8.1% had both a greater maximum decrease in stability from baseline and required a longer time for healing as indicated by return of stability level to baseline. This study demonstrates alterations in implant stability consistent with impaired implant integration for patients with type 2 diabetes mellitus in direct relation to hyperglycemic conditions.

Keywords

Implants; hyperglycemia; diabetes mellitus; resonance frequency analysis; implant stability

INTRODUCTION

Currently, diabetes mellitus is considered a relative contraindication to dental implant therapy, dependent on levels of glycemic control (World Workshop in Periodontics, 1996; Beikler, 2003; Blanchaert, 1998). Poor glycemic control and hyperglycemia have been directly associated with an increased risk of co-morbidities for type 2 diabetes mellitus, including compromised dermal wound healing and immune responses.

Consistent with these associations, hyperglycemia has been shown to adversely affect bone formation and implant integration in animal models, with levels of implant integration decreased approximately 30% relative to control animals (Nevins, 1998; Gerritsen, 2000; McCracken, 2000). Clinical studies, however, have reported highly varied implant failure rates, ranging from 0-14.7% of implants and 0-31.3% of patients, leaving the consequences of diabetes on implant success in question (Shernoff 1994; Garrett 1998; Fiorellini 2000; Olson 2000; Morris 2000; Abdulwassie 2002; Moy 2005). Furthermore, glycemic status of the patients defined as well-controlled was not clearly reported in these studies. Taken together,

Corresponding Author: Dr. Thomas W. Oates, Department of Periodontics, University of Texas Health Science Center at San Antonio, 7703 Floyd Curl Drive, San Antonio, Texas 78229-3900, (210) 567-3590, Fax: (210) 567-6858, oates@uthscsa.edu. currently private practice, Abilene, TX

³currently private practice, Austin, TX

Implant integration may be assessed using longitudinal measures of implant stability by resonance frequency analysis (Barewal 2003). Implant stability has been shown to correlate with bone density, insertion torque, changes in supporting matrix, and bone-implant contact (Barewal 2003, Meredith, 1996, Meredith, 1997a and 1997b; Friberg, 1999). Additionally, resonance frequency analysis has identified changes in implant stability consistent with resorptive and formative osseous healing following implant placement (Barewal 2003).

We previously reported the clinical findings from this study showing no evidence of diminished clinical success or early healing complications of implant therapy associated with glycemic control in type 2 diabetic patients (Dowell, 2007). In this paper, we challenge the hypothesis that poor glycemic control is directly related to short-term impairments implant stabilization.

MATERIALS AND METHODS

Study Design

Detailed descriptions of the study design and methods have been presented previously (Dowell, 2007). In brief, this prospective pilot study was designed to examine the effect of glycemic control on implant stabilization for type 2 diabetes patients over 4 months following implant placement. Subjects were recruited from dental patients seeking treatment within the University of Texas Health Science Center at San Antonio Dental School. The study enrolled patients missing one or more teeth and recognized as having the potential to benefit from dental implant therapy. Both healthy, non-diabetic patients and type 2 diabetes mellitus patients were enrolled. All diabetic subjects were under the care of a health care provider and study participation did not alter their medical management. This study was approved by the Institutional Review Board; all patients gave written, informed consent.

Implant sites were required to have at least 4 months of healing following tooth extraction prior to implant placement, and adequate bone dimensions for implant placement without the need for bone grafting. Patients with oral pathology, systemic disorders affecting surgical therapy protocols, history of bone grafting at the implant site, or currently smoking, were excluded. Type 2 diabetes mellitus status was self-reported and verified with physician report, glycemic level within 1 month prior to implant surgery, and/or treatment record. Diabetic patients with a history of treatment for microvascular or macrovascular complications were excluded. Type 2 diabetes patients could be on a modified diet, oral medication, insulin, or combination therapies. Of 50 implants placed in 35 patients, 1 non-diabetic patient (1 implant) and 1 implant (of 2) from a diabetic patient were excluded due to rotational movement during the 4 months following implant placement. Additionally, 6 implants and 2 patients with 42 implants were analyzed in this study.

Glycemic control

Glycemic control was assessed by glycated hemoglobin (HbA1c; Quest Diagnostics Laboratory); HbA1c level reflects average blood glucose levels over the preceding 2 to 3 month period (Derr 2003). Type 2 diabetic patients were stratified by HbA1c levels as well controlled (HbA1c 6.1–8.0%), moderately-controlled (HbA1c 8.1–10.0 %) or poorly-controlled (HbA1c \geq 10.1%). All non-diabetic patients had HbA1c \leq 6.0%. HbA1c measurements taken 8 weeks following implant placement were used as the assessment of glycemic control.

Surgical Implant Placement

Ten or 12 mm length, 4.1 diameter, rough-surfaced (SP, SLAR®) implants were placed consistent with manufacturer's (Institut Straumann AG, Basel, Switzerland) protocols, and covered with a transgingival healing cap. Non-diabetic patients were prescribed post-operative antibiotics for 3 days, and diabetic patients prescribed antibiotics for 7–10 days. Implants were not restored during the 4 month evaluation period, and prostheses were adjusted as needed to minimize inadvertent loading of the implant. The surgical visit was considered as the baseline (Week 0).

Clinical Measurements

Resonance frequency measurements as an assessment of implant stability (Implant Stability Quotient) were taken in triplicate using the Osstell® instrument (Integration Diagnostics Ltd., Oslo, Norway). The stability level was recorded at baseline, 2, 4, 6, 8, 12, and 16 weeks following implant placement. The mean of the three measurements was used in the statistical analysis. Subjective clinical assessments were made regarding bone type using a 4-tier scale based on mineral densities during osteotomy as being high density (type I), moderate density (type II), low density (type III), and very low density (type IV) (Lekholm and Zarb, 1985).

Statistical Analysis

Implant Stability Quotient data were analyzed for associations with HbA1c level and time following implant placement (Baseline, 2, 4, 6, 8, 12, 16 weeks) using analysis of variance for repeated measurements. HbA1c group was a between-subjects factor and follow-up time was a within-subjects factor. Multiple implants for a patient were considered nested within patient. Contrasts among means following the analysis of variance were analyzed using Scheffe's multiple comparison procedure. The minimum stability level among the values at 2, 4, and 6 weeks was identified. The follow-up time at which the minimum occurred was also determined. The follow-up time that the stability returned to a level \geq baseline value, defined as the "time to healing," was also determined; if the stability value never returned to baseline, a time to healing of 16 weeks was assigned. Differences in these responses (minimum stability, time of minimum, time to healing) among HbA1c classes measured at 8 weeks were analyzed by analysis of variance for repeated measurements. To better assess the stabilization of implants over time, the stability data were normalized for each time (2, 4, 6, 8, 12, 16 weeks) by calculating the relative change from baseline for each patient and each implant site. The fraction of subjects returning to baseline by 16 weeks were analyzed with generalized linear models (Liang and Zeger, 1986) using a binomial distribution with a logistic link function.

RESULTS

Patient Characteristics

Non-diabetic patients were significantly younger than type 2 diabetic patients (Table 1). Nondiabetic patients ranged in age from 29–61 years with HbA1c levels ranging from 4.7–5.8%. Diabetic patients ranged in age from 51–81 years with HbA1c levels 6.3–12.6% at the 8 weeks follow-up visit. When the subjects were classified into low (subjects with HbA1c \leq 8.0) and high (subjects with HbA1c \geq 8.1) HbA1c groups independent of diabetes diagnosis, there was no significant difference in age distributions between the two groups.

Implant Stability

Implant stability was significantly affected by the combination of HbA1c level and the time following implant placement [Interaction of HbA1c and follow-up time; p=0.0094; Figure 1]. The maximum decrease in implant stability relative to baseline was significantly greater for

the HbA1c 8.1–10.0% and HbA1c \geq 10.1% groups compared with the non-diabetic (HbA1c \leq 6.0%) and well-controlled (HbA1c 6.1–8.0%) diabetic groups (Table 2).

The time required for stability to return to baseline level (time to healing) for the moderatelyand poorly-controlled diabetic groups was approximately double that required for the nondiabetic and well-controlled diabetic groups (Table 2). In the poorly-controlled group (HbA1c \geq 10.1%) only 57.1% of the implants returned to or exceeded baseline stability levels after 16 weeks, compared with 80% or more for each of the other HbA1c groups. Although there was not a significant difference between HbA1c groups in the changes in stability from baseline to 16 weeks, the two HbA1c \geq 8.1% groups tended to show less improvement in stability from baseline.

Irrespective of HbA1c, a decrease in implant stability was evident at 2 weeks with progressively increasing stability beginning at 4 to 8 weeks following implant placement. After adjusting for HbA1c level, there were no significant associations of implant stability with gender, age, bone type, or implant site (maxilla or mandible).

Primary stability, that is, baseline stability obtained at the time of implant placement, was greater, but not significantly greater, for the HbA1c 8.1-10.0% and HbA1c $\geq 10.1\%$ groups compared with the non-diabetic (HbA1c $\leq 6.0\%$) and well-controlled (HbA1c 6.1-8.0%) diabetic groups (Table 2).

There were no significant differences among HbA1c groups in minimum stability levels measured between weeks 2–6 weeks following implant placement or in 16 week stability level (Table 2). The average time at which the minimum stability occurred in all HbA1c groups ranged from 2.4 to 3.6 weeks and did not differ significantly among HbA1c groups.

The results in Table 2 indicate similar responses for the two groups with HbA1c \leq 8.0% (low group) and for the two groups with HbA1c \geq 8.1% (high group), and that the changes in implant stability for the high HbA1c group were significantly different from that of the low HbA1c group. This dichotomous classification into low and high HbA1c groups (Figure 2) shows the decreases in stability at weeks 2 and 4 and longer time to healing for the high HbA1c group (Table 2) relative to the low HbA1c group. The differences in stability between low and high groups apparent from week 6 through week 12 were not statistically significant. In the low HbA1c group there were no significant decreases in stability from baseline, and there were significant increases in stability from baseline after 12 and 16 weeks following implant placement that were not evident for the high HbA1c group.

DISCUSSION

The importance of maintaining stringent glycemic control to minimize diabetic co-morbidities is becoming increasingly appreciated(UKPDS 2000). However, a majority of diabetic patients still suffer from an inability to maintain adequate glycemic control with HbA1c levels for individual patients frequently averaging between 8.5 and 9% (Kirk 2005). As diabetes mellitus remains a relative contraindication to dental implant therapy dependent upon the patient's level of glycemic control, many diabetic patients may be denied the benefits of implant therapy.

The detrimental effects on implant integration with HbA1c levels $\geq 8.1\%$ found in this study are consistent with several diabetes co-morbidity studies which suggested that the risk of microvascular complications does not rise dramatically until HbA1c levels are greater than 8%, and that the most prominent threshold for glycemic damage leading to renal and retinal complications is between 8 and 8.5% (DCCT, 1993). Our findings show that patients with HbA1c levels $\geq 8.1\%$ have compromises in implant stabilization that suggest alterations in the biologic integration of the implants in direct relation to glycemic control.

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The findings of the current study are consistent with previous studies that have demonstrated that hyperglycemic conditions lead to alterations in bone physiology (Funk 2000, Lu, 2003, Amir, 2002). Impaired osseous healing in association with hyperglycemia has been demonstrated in several cross-sectional and retrospective studies (White, 2003; Loder 1988). Animal and in vitro studies have extended these findings to include the effect of blood glucose control on fracture healing and bone turnover, with decreased consequences of the hyperglycemic state in animals receiving insulin treatment to reduce the hyperglycemia (Beam, 2002; Funk, 2000; Follak, 2004; Gebauer 2002). Consistent with these findings, an investigation using a murine model reported the reduced expression of two genetic markers of osteoblastic differentiation, Cbfa1 and Dlx5, found in response to hyperglycemia was reversed with insulin treatment controlling the hyperglycemia (Lu, 2003).

The effects of a hyperglycemic state have been shown to include inhibition of osteoblastic cell proliferation and collagen production during the early stages of callus development that results in reduced bone formation, as well as diminished mechanical properties of the newly formed bone (Beam, 2002; Gebauer, 2002; Gooch, 2000; Lu, 2003; Amir, 2002). The diminished bone formation may be exacerbated further by increased apoptosis of bone lining cells in a hyperglycemic state (He 2005). More recently, several animal studies have demonstrated a more persistent inflammatory response that may also lead to increased osteoclastic activity in a hyperglycemic state (Kayal, 2007; Liu 2006).

Together, the potential for alterations in bone metabolism in association with hyperglycemia are consistent with the longitudinal assessments of implant stabilization found in this study. It is noteworthy that the differences in implant stability change relative to low and high HbA1c levels are consistent with clinically relevant differences in bone density found between type 1 and type 4 bone (Barewal 2003; Lekholm and Zarb 1985). The implications for clinical treatment based on these stability changes remain to be determined.

In conclusion, the results of the current study justify the continued investigation of the effects of diabetes and glycemic control on bone metabolism, as well as the longer term effects of glycemic control on implant integration, success and complications for patients with type 2 diabetes. Findings from this study and future studies must be considered in light of the potential increased risk for long-term complications such as peri-implant inflammation and bone loss.

Acknowledgments

This study was supported by NIH/NIDCR grant R01 DE017882 (T.W.O.), the ITI Foundation, and the San Antonio Area Foundation. The authors would also like to thank the Institut Straumann AG, (Basel, Switzerland) for providing the implants used in this study.

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Figure 1.

Implant Stability Quotient by HbA1c level and time following implant placement. Error bars represent SE. '+' indicates significant ($p\leq0.05$) change from baseline for same HbA1c group. Dashed reference lines represent baseline stability level. HbA1c $\leq 6.0\%$ n=10 patients; HbA1c $\leq 6.1-8.0\%$, n=12 patients; HbA1c $\approx 1.10.0\%$, n=5 patients; HbA1c $\geq 10.1\%$, n=5 patients

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Figure 2.

Changes in Implant Stability Quotient (%) from baseline by HbA1c level and time following implant placement. \circ HbA1c \leq 8.0% (n=22 patients), \bullet HbA1c \geq 8.1% (n=10 patients). Error bars represent SE. '*' indicates HbA1c \ge 8.1% significantly (p \le 0.05) different from HbA1c \le 8.0% at same follow-up time. '+' indicates significant (p≤0.05) change from baseline for same HbA1c group.

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	u	u	Number female	Mean ± SE	Number mandible	Type 1	Type 2	Type 3	Type 4
≤6.0	10	12	L	$45.7 \pm 3.1 b$	L	4	3	5	0
6.1 to 8	12	18	9	66.0 ± 2.2	12	0	7	10	
8.1 to 10	5	5	2	54.4 ± 1.7	3	0	2	1	2
≥10.1	5	7	2	64.0 ± 5.1	5	2	2	2	Т
Total number	32	42	17^{C}		27^d	9	14^{e}	18^{e}	4
Significance ^{<i>a</i>}			p=0.7094	p=0.0001	p=0.9277		p=0.	0610	
^a Test of equality of Hb	Alc groups								

 b HbA lc \leq 6.0 significantly (p \leq 0.05, Scheffe's multiple comparison procedure) different from combined 6.1 to 8.0, 8.1 to 10.0, and \geq 10.1

 c 53.1% of subjects were female

 $d_{64.3\%}$ of implants were in the mandible

 $^e76.2\%$ of implants were in types II or II bone

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Table 2

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HbA1c at 8 weeks	Baseline	Minimum	Time to minimum (weeks)	Maximum change relative to baseline (%)	16 weeks	Change at 16 weeks relative to baseline (%)	Time to healing (weeks)	Fraction returning to baseline (%)
	Mean ± SE	Mean ± SE	Mean ± SE	Mean ± SE	Mean ± SE	Mean ± SE	Mean ± SE	
≤6.0	58.0 ± 1.5	55.3 ± 1.5	3.6 ± 0.4	-4.4 ± 1.8	62.2 ± 1.5	7.4 ± 2.3	5.1 ± 1.6	91.7
6.1 to 8	57.5 ± 1.3	55.7 ± 1.4	3.4 ± 0.3	-3.0 ± 1.5	61.6 ± 1.3	7.6 ± 2.0	6.4 ± 1.4	83.3
8.1 to 10	59.8 ± 2.1	53.5 ± 2.1	2.4 ± 0.6	$-10.4 \pm 2.5 b$	62.6 ± 2.2	4.8 ± 3.3	11.6 ± 2.4^{b}	80.0
≥10.1	$63.8\pm2.0^{\mathcal{C}}$	53.9 ± 2.1	3.2 ± 0.5	$-14.8\pm2.4b$	65.6 ± 2.0	3.9 ± 3.1	12.5 ± 2.2^b	57.1
Significance ^a	p=0.0673	p=0.7781	p=0.4216	p=0.0014	p=0.3356	p=0.7453	p=0.0248	p=0.6279

 b Combined HbAlc 8.1 to 10 and 10.1- significantly (p \leq 0.05, Scheffe's multiple comparison procedure) different from combined -6.0 and 6.1 to 8.0

^c The higher initial stability for the $\geq 10.1\%$ group appears to be largely due to an elevated initial stability for two implants (Implant Stability Quotients of 71 and 78) in one patient. Without this patient, the mean initial stability was 60.5 (SE=1.9).