Original paper

Impact of glycemic control on early implant failure: A retrospective cohort study

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Abstract

Background. The impact of diabetes mellitus (DM), as reflected by glycemic control, on dental implant outcomes remains debated. Specifically, there is a critical gap in our understanding of the impact of uncontrolled DM on early implant failure (EIF), despite the potential benefits of implant dentistry for these patients.

Objectives. The aim of the present study was to investigate the impact of glycemic control on EIF in patients with DM.

Material and methods. A retrospective analysis of electronic medical records (EMRs) was conducted over a 5-year period at a tertiary referral center. Data from 100 patients with 427 implants were collected, including demographic characteristics, systemic conditions, the glycated hemoglobin (HbA1c) values, procedural details, and implant-related variables. Statistical analyses were performed to assess the associations between glycemic control, the study variables and EIF, with statistical significance set at $p < 0.05$.

Results. Uncontrolled DM (HbA1c > 8%) was observed in 17 patients and was not a significant predictor of EIF. However, it was associated with a greater need for bone augmentation (90.8% vs. 45.0%), the placement of longer implants (11.8 mm vs. 11.4 mm), a higher proportion of implants placed in the anterior maxilla (22.4% vs. 11.1%), and younger patient age (62.8 years vs. 67.8 years). The EIF rates were higher for implants placed in augmented bone and lower for implants placed in the anterior and premolar regions of the maxilla.

Conclusions. This study indicates that, although uncontrolled DM may present certain challenges in implant dentistry, including a younger age at implant placement, an increased need for bone augmentation, and a preference for specific implant sites, it does not significantly affect the EIF rates.

Keywords: risk factor, implant, glycemic control, diabetes, early implant failure

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Highlights

- Uncontrolled diabetes mellitus (DM) did not significantly impact the early implant failure (EIF) rates.
- Uncontrolled DM was associated with a younger age at implant placement.
- Uncontrolled DM was associated with an increased need for bone augmentation.
- This study suggests uncontrolled DM is not an absolute contraindication to implant therapy.

Introduction

Diabetes mellitus (DM) is a common metabolic disorder that may lead to adverse oral health outcomes, including microvascular disease, delayed wound healing, functional alterations in the salivary glands, periodontitis, changes in the oral microbiota, and an impaired response to infection.[1–3]

Dental implants have become a popular treatment option for missing teeth, offering a stable and esthetic solution.[4–10] The influence of DM on implant dentistry has been debated for many years. While some studies have considered dental implant placement in patients with DM a relative contraindication,[11] others have reported that implant survival and osseointegration remain predictable and comparable to those in healthy individuals.[1,4,12,13] These studies have also highlighted the potential negative effects of hyperglycemia and uncontrolled hemoglobin A1c (HbA1c) levels on implant therapy outcomes.[4,13,14]

Glycemic control is a crucial factor in the management of patients with DM. Adequate control of blood glucose and HbA1c levels may help prevent microvascular and macrovascular complications, wound infections, and impaired bone formation.[14–16] Poor glycemic control has been suggested to compromise osseointegration. For example, Oates et al. conducted a prospective clinical study demonstrating that, in patients with type 2 diabetes mellitus (T2DM), poor glycemic control (HbA1c ≥ 8.1%) was associated with reduced initial implant stability and prolonged healing following implant placement.[17] However, after 16 weeks, implant stability was comparable to that in patients without DM, suggesting that successful osseointegration is achievable, although healing may take longer.[17] Other studies have reported that glycemic control does not significantly affect implant success.[1,18] Escow and Oates conducted a 2-year observational study involving patients with poorly controlled T2DM (HbA1c levels ranging from 6.3% to 13.1%) and found that elevated glycemic levels did not significantly affect the implant survival rates, challenging the prevailing notion that strict glycemic control is essential for early implant success.[18]

Although the influence of DM on dental implant outcomes has been investigated in previous studies, an important limitation remains in the current literature. Many studies have excluded patients with uncontrolled DM, despite the ongoing debate on the impact of glycemic control on early implant failure (EIF).[19–21] This exclusion creates a significant knowledge gap regarding the effect of uncontrolled DM on implant outcomes, particularly during the early stages of healing.

Traditionally, some oral surgeons have considered uncontrolled DM a relative contraindication to implant placement.[11] This is particularly relevant for patients who struggle to achieve adequate glycemic control, as they may be denied access to implant therapy based on limited evidence. Implant-supported prostheses offer potential benefits for patients with DM, particularly those who are partially or completely edentulous. By restoring masticatory function, dental implants may facilitate healthier dietary choices and potentially improve glycemic control. These benefits may, in turn, contribute to an improved quality of life (QoL), enhanced self-image, and better overall health outcomes in these patients.[22,23]

The evolving understanding of DM management, the limited evidence supporting the contraindication of implant therapy in patients with uncontrolled DM, and the growing emphasis on holistic, patient-centered care necessitate a re-evaluation of this approach.

Therefore, the aim of the present study was to assess the impact of glycemic control on EIF in patients with DM. The null hypothesis was that glycemic control is not a significant risk factor for EIF in patients with DM.

Material and methods

A retrospective cohort study, conducted in accordance with the STROBE (Strengthening the Reporting of Observational Studies in Epidemiology) guidelines for observational research, was performed at the Department of Oral and Maxillofacial Surgery, Rabin Medical Center, Petah Tiqwa, Israel, between January 2013 and December 2018. The study complied with the Declaration of Helsinki and its subsequent amendments, and received ethical approval (No. 0674-19-RMC). Written informed consent was obtained from all participants included in the study.

Patients were eligible for inclusion if they were older than 18 years, had undergone implant dentistry treatment, were treated consecutively during the study period, and had available HbA1c measurements obtained within the 3 months preceding implant placement. Patients with incomplete documentation were excluded from the study.

Surgical protocol

Preoperative radiological assessment was performed using cone-beam computed tomography (CBCT). All bone augmentation and implant placement procedures were performed by well-trained oral and maxillofacial surgeons with a minimum of 10 years of clinical experience. Implant placement began with a mid-crestal incision, followed by the elevation of a full-thickness flap. When the available bone volume was insufficient to accommodate an implant with a diameter of ≥3.3 mm and a length of ≥8 mm, or when augmentation was indicated for esthetic reasons, bone augmentation was performed using one of the following bone substitutes or a collagen membrane prior to implant placement:

– xenogeneic bone substitute (Bio-Oss; Geistlisch Pharma, Wolhusen, Switzerland);
– allogeneic bone substitute (OraGraft®; LifeNet Health, Virginia Beach, USA);
– collagen membrane (Ossix® Plus; Datum Dental Ltd., Lod, Israel).

When adequate primary implant stability could be achieved, implants were placed, with simultaneous bone augmentation when indicated, according to the implant manufacturer's protocol and using the dedicated surgical bur set. Upon discharge, patients were prescribed antibiotics (amoxicillin 500 mg 3 times daily for 5 days, or clindamycin 300 mg 3 times daily for 7 days in patients with a penicillin allergy), a chlorhexidine mouth rinse and an analgesic regimen.

Data collection

Clinical data were collected using the Chameleon Electronic Medical Records (EMR) system, while medical histories were retrieved from the Ofek EMR system. The Ofek EMR system provides comprehensive access to patients' medical histories across all Clalit Health Services hospitals and community clinics in Israel. The available information includes diagnoses, medications, complete blood counts (CBCs), histopathology results, and other clinically relevant data. By centralizing this information, the Ofek EMR system helps minimize the risk of disclosure and reporting biases. Data collection was performed independently by D.M. and E.J. Any discrepancies were discussed until a consensus was reached.

Demographic characteristics included patients' gender and age at the time of the procedure. In addition, systemic conditions, HbA1c levels, and the smoking status were recorded.

Patients were stratified into 3 groups according to HbA1c levels[17,18]: non-diabetic (HbA1c < 6.5%); controlled DM (HbA1c between 6.5% and 8%); and uncontrolled DM (HbA1c > 8%).

The following variables were taken into consideration:

– span length: short-span partial edentulism (≤2 missing teeth); long-span partial edentulism (>2 missing teeth; and total edentulism;
– implant:
  • brand;
  • length;
  • diameter;
  • location in the maxilla or the mandible, and in particular regions: anterior (central incisor to canine); premolar (first and second premolars); and posterior (first and second molars);
– number of implants per patient;
– need for bone augmentation (yes/no); and
– EIF defined as implant removal ≤1 year after permanent rehabilitation – primary outcome variable.

Statistical analysis

The data were analyzed using IBM SPSS Statistics, v. 25.0 (IBM Corp., Armonk, USA). Descriptive statistics were used to summarize continuous variables as means and standard deviations ($M \pm SD$) and categorical variables as frequencies. Comparisons among the 3 groups (patients without DM, patients with controlled DM, and patients with uncontrolled DM) were performed using the Kruskal–Wallis test for continuous variables and the χ^2 test for categorical variables.

To account for multiple implants within the same patient and the resulting within-patient correlation, a generalized estimating equations (GEE) model was used to estimate the risk of EIF. The GEE model accounted for subject-level clustering, binomial distribution and exchangeable correlation. Multicollinearity was assessed using the variance inflation factor (VIF). Variables with univariate p-values <0.1 and no evidence of multicollinearity were included in the multivariable models – multivariable GEE at the implant level and multivariable logistic regression at the patient level. Statistical significance was set at $p \leq 0.05$.

Results

Descriptive statistics

Cohort patient level

At the patient level (Table 1), the cohort ($N = 100$) comprised 40% males and 60% females. The mean age was 67.1 ±11.5 years, and 6% of the patients were smokers. Sixty-one percent of the patients received Zimmer implants (Zimmer Biomet TMT, Parsippany, USA; a microtextured (MTX) surface, treated by grit-blasting with hydroxyapatite, followed by washing with non-etching acid and distilled water), 26% received MIS implants (MIS Implants

Table 1. Demographic and baseline clinical characteristics of the cohort at the patient level ($N = 100$)

Variable	Subgroup	Characteristic
Gender	M	40 (40.0)
	F	60 (60.0)
Age [years]	–	67.1 ±11.5
Smoking	–	6 (6.0)
HbA1c level prior to implantation [%]	–	7.1 ±1.1
	non-diabetic	37 (37.0)
	controlled DM	46 (46.0)
Glycemic control	uncontrolled DM	17 (17.0)
	Ditron	13 (13.0)
	MIS	26 (26.0)
Implant brand	Zimmer	61 (61.0)
	short-span partial edentulism	49 (49.0)
	long-span partial edentulism	32 (32.0)
Span length	complete edentulism	19 (19.0)
Number of implants per patient	–	4.3 ±2.9
Bone augmentation	–	51 (51.0)
EIF	–	12 (12.0)

Data presented as mean ± standard deviation ($M \pm SD$), or as number (percentage) (n (%)).
M – male; F – female; HbA1c – hemoglobin A1c; DM – diabetes mellitus; EIF – early implant failure.

Technologies Ltd., Karmiel, Israel; a sandblasted and acid-etched surface), and the remainder received Ditron implants (MPI (Molecular Precision Implant); Ditron Dental, Ashkelon, Israel; a blasted and thermally acid-etched endosseous surface). The mean number of implants per patient was 4.3 ±2.9. Most of the cohort (51%) required bone augmentation.

The mean HbA1c level in the entire cohort was 7.10 ±1.08%. Seventeen percent of the patients were classified as having uncontrolled DM (HbA1c > 8%), 46% as having controlled DM (HbA1c 6.5–8%), and 37% as non-diabetic (HbA1c < 6.5%). Twelve patients had at least one EIF.

Cohort implant level

All data were likewise analyzed at the implant level ($N = 427$) (Table 2). Most implants (58.8%) were Zimmer implants, with 15–20% distributed across each sextant. Twenty-three percent of the implants were placed in patients with uncontrolled DM. The overall incidence of EIF was 2.8% at the implant level.

Table 2. Demographic and baseline clinical characteristics of the cohort at the implant level ($N = 427$)

Variable	Subgroup	Characteristic
Age [years]	–	66.6 ±10.8
Smoking	–	14 (3.3)
Glycemic control	non-diabetic	158 (37.0)
	controlled DM	171 (40.0)
	uncontrolled DM	98 (23.0)
Implant brand	Ditron	63 (14.8)
	MIS	113 (26.5)
	Zimmer	251 (58.8)
Implant length [mm]	–	11.5 ±1.5
Implant diameter [mm]	–	3.8 ±0.3
Implant location	anterior maxilla	58 (13.6)
	premolar maxilla	86 (20.1)
	posterior maxilla	66 (15.5)
	anterior mandible	76 (17.8)
	premolar mandible	70 (16.4)
	posterior mandible	71 (16.6)
Bone augmentation	–	228 (53.4)
EIF	–	12 (2.8)

Data presented as $M \pm SD$, or as n (%).

Statistical analysis

Univariate analyses

At the implant level (Table 3), uncontrolled DM was associated with a substantially higher need for bone augmentation (90.8%) as compared to the controlled DM group (45.0%) and non-diabetic individuals (39.2%). The younger mean age (62.8 ±12.2 years) and female predominance (63.3%) in the poorly controlled DM group were statistically significant. Differences were also observed in implant location, with a higher proportion of implants placed in the anterior maxilla among patients with uncontrolled DM. In addition, implants were significantly longer in this group. Notably, no significant differences were found between the non-diabetic, controlled DM and uncontrolled DM groups.

At the patient level (Table 4), the prevalence of bone augmentation was significantly associated with glycemic control, with a higher need among patients with uncontrolled DM (82.4%) than among those with controlled DM (45.7%) or non-diabetic individuals (43.2%). The number of implants per patient also differed between the groups ($p < 0.1$), reflecting the greater extent of tooth loss in patients with uncontrolled DM.

Table 3. Univariate tests at the implant level to assess the influence of glycemic control on the study variables

Variable	Subgroup	Non-diabetic (*n* = 158)	Controlled DM (*n* = 171)	Uncontrolled DM (*n* = 98)	Statistical analysis		
					Kruskal–Wallis test	χ^2 test	*p*-value
Gender	M	42 (26.6)	73 (42.7)	36 (36.7)	–	9.42	0.009***
	F	116 (73.4)	98 (57.3)	62 (63.3)			
Age [years]	–	67.6 ±10.8	67.8 ±9.6	62.8 ±12.2	13.23	–	0.001***
HbA1c level prior to implantation [%]	–	6.1 ±0.3	7.2 ±0.9	9.1 ±0.4	373.10	–	<0.001***
Implant length [mm]	–	11.3 ±1.7	11.4 ±1.5	11.8 ±1.4	10.49	–	0.007***
Implant diameter [mm]	–	3.9 ±0.3	3.8 ±0.4	3.8 ±0.2	1.61	–	0.142
Implant location	anterior maxilla	17 (10.8)	19 (11.1)	22 (22.4)	–	9.60	0.047**
	premolar maxilla	36 (22.8)	29 (17.0)	21 (21.4)	–	1.90	0.394
	posterior maxilla	29 (18.4)	25 (14.6)	12 (12.2)	–	1.90	0.391
	anterior mandible	21 (13.3)	36 (21.1)	19 (19.4)	–	3.60	0.165
	premolar mandible	28 (17.7)	25 (14.6)	17 (17.3)	–	0.66	0.719
	posterior mandible	27 (17.1)	37 (21.6)	7 (7.1)	–	9.48	0.009***
Bone augmentation	–	62 (39.2)	77 (45.0)	89 (90.8)	–	72.70	<0.001***
EIF	–	5 (3.2)	3 (1.8)	4 (4.1)	–	1.35	0.509

Data presented as *M* ±*SD*, or as *n* (%).
* $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$.

Table 4. Univariate tests at the patient level to assess the influence of glycemic control on the study variables

Variable	Subgroup	Non-diabetic (*n* = 37)	Controlled DM (*n* = 46)	Uncontrolled DM (*n* = 17)	Statistical analysis		
					Kruskal–Wallis test	χ^2 test	*p*-value
Gender	M	11 (29.7)	22 (47.8)	7 (41.2)	–	2.81	0.245
	F	26 (70.3)	24 (52.2)	10 (58.8)			
Age [years]	–	67.6 ±11.6	68.6 ±10.2	62.4 ±13.9	2.92	–	0.231
HbA1c level prior to implantation [%]	–	6.1 ±0.4	7.2 ±0.4	8.9. ±0.9	84.09	–	<0.001***
Span length	short-span partial edentulism	20 (54.1)	26 (56.5)	3 (17.6)	–	10.00	0.125
	long-span partial edentulism	10 (27.0)	13 (28.3)	9 (52.9)			
	complete edentulism	7 (18.9)	7 (15.2)	5 (29.4)			
Number of implants per patient	–	4.3 ±2.9	3.8 ±2.4	5.8 ±3.8	4.73	–	0.094*
Bone augmentation	–	16 (43.2)	21 (45.7)	14 (82.4)	–	8.10	0.017**
EIF	–	5 (13.5)	3 (6.5)	4 (23.5)	–	3.52	0.171

Data presented as *M* ±*SD*, or as *n* (%).
* $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$.

Analyses were performed at both the implant and patient levels to identify risk factors for EIF. These analyses revealed that implant placement in the augmented bone was a significant risk factor for EIF. Notably, no EIF events occurred among implants placed in the anterior or premolar regions of the maxilla, suggesting a lower risk of failure at these sites. Conversely, implants placed in the anterior mandible had significantly higher odds of EIF (*OR* = 3.46). Implants placed in the posterior maxilla showed a borderline statistically significant increase in the odds of failure (*OR* = 2.85; $p = 0.085$) (Tables 5 and 6).

Table 5. Univariate generalized estimating equations (GEE) analysis at the implant level to assess risk factors for early implant failure (EIF)

Variable	Subgroup	*OR*	95% *CI*	*p*-value
Gender	F	1	–	–
	M	2.62	0.82–8.41	0.105
Age	–	1.01	0.97–1.05	0.680
Smoking	–	2.82	0.35–22.76	0.330
HbA1c level prior to implantation	non-diabetic (healthy)	1	–	–
	controlled DM	0.54	0.13–2.30	0.410
	uncontrolled DM	1.30	0.33–5.06	0.700
ASA classification	ASA 2	1	–	–
	ASA 3	1.82	0.57–5.83	0.310
Hypertension	–	0.75	0.24–2.33	0.620
Hyperlipidemia	–	0.64	0.17–2.41	0.520
Osteoporosis	–	2.34	0.58–9.55	0.230
CVA	–	2.89	0.68–12.41	0.150
Implant brand	Ditron	1	–	–
	MIS	0.27	0.02–3.06	0.290
	Zimmer	1.13	0.25–5.20	0.870
Implant length	–	1.12	0.76–1.66	0.550
Implant diameter	–	1.88	0.48–7.40	0.360
Implant location	anterior maxilla	0[a]	0	0
	premolar maxilla	0[a]	0	0
	posterior maxilla	2.85	0.86–9.42	0.085*
	anterior mandible	3.46	1.10–10.91	0.033**
	premolar mandible	1.02	0.22–4.76	0.970
	posterior mandible	0.44	0.06–3.54	0.440
Bone augmentation	–	10.04	1.33–76.03	0.020**

OR – odds ratio; *CI* – confidence interval; ASA – American Society of Anesthesiology; CVA – cerebrovascular accident; [a] no failures occurred in these subgroups; * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$.

Table 6. Univariate statistical analysis at the patient level to assess risk factors for early implant failure (EIF)

Variable	Subgroup	Failure ($n = 12$)	Success ($n = 88$)	Statistical analysis		
				Kruskal–Wallis test	χ^2 test	*p*-value
Gender	M	7 (58.3)	33 (37.5)	–	1.14	0.280
	F	5 (41.7)	55 (62.5)			
Age	–	67.5 ±7.93	67.1 ±11.9	0	–	0.980
Smoking	–	1 (8.3)	5 (5.7)	–	0	1.000
ASA classification	ASA 2	5 (41.7)	51 (58.0)	–	0.57	0.450
	ASA 3	7 (58.3)	37 (42.0))			
Hypertension	–	7 (58.3)	54 (61.4)	–	0	1.000
Hyperlipidemia	–	3 (25.0)	32 (36.4)	–	0.20	0.650
Osteoporosis	–	2 (16.7)	11 (12.5)	–	0	1.000
CVA	–	2 (16.7)	7 (8.0)	–	0.20	0.650
Span length	short-span partial edentulism	6 (50.0)	43 (48.9)	–	3.97	0.140
	long-span partial edentulism	6 (50.0)	26 (30.0)			
	complete edentulism	0 (0.0)	19 (21.6)			
Number of implants per patient	–	3.5 ±2.1	4.4 ±3.0	0.72	–	0.400
Bone augmentation	–	11 (91.7)	40 (45.5)	–	7.27	0.007***

Data presented as *M* ±*SD*, or as *n* (%).
* $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$.

Multivariable analysis for EIF

Similar to the univariate analysis, the implant-level multivariable GEE model showed that implantation in augmented bone was a significant risk factor for EIF (*OR* = 17.48). Implants placed in the anterior and premolar regions of the maxilla had a significantly lower risk of failure, with no occurrences of EIF observed in these regions (Table 7).

The patient-level model showed that, after controlling for potential confounders, there was no statistically significant difference between the controlled and uncontrolled DM groups (Table 8).

Table 9 illustrates the characteristics of EIF. Notably, 11 of the 12 patients who experienced EIF in this study had augmented bone.

Table 7. Multivariable generalized estimating equations (GEE) model to predict early implant failure (EIF) at the implant level

Variable	Subgroup	*OR*	95% *CI*	*p*-value
Implant location	anterior maxilla	0	0	0[a]
	premolar maxilla	0	0	0[a]
	posterior maxilla	2.56	0.59–11.21	0.210
	anterior mandible	5.33	0.88–14.69	0.090*
Bone augmentation	–	17.48	2.27–34.20	0.002***

[a] no failures occurred in these subgroups; * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$. Adjusted for age, smoking, hypertension, and hyperlipidemia.

Table 8. Multivariable logistic regression model to predict early implant failure (EIF) at the patient level

Variable	*OR*	95% *CI*	*p*-value
Controlled DM	0.41	0.18–2.67	0.350
Uncontrolled DM	1.35	0.34–3.66	0.960
Bone augmentation	13.24	3.46–96.33	0.013**

* $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$. Adjusted for age, smoking, hypertension, and hyperlipidemia.

Discussion

Achieving successful osseointegration and long-term implant stability is a primary goal in implant dentistry. Various factors can influence implant outcomes, including systemic conditions such as diabetes.[24] Currently, the evidence regarding the impact of uncontrolled DM on EIF remains limited, primarily because individuals with poorly controlled DM are often excluded from clinical studies.[19–21] This study sought to address this gap by analyzing a substantial cohort of patients with varying levels of glycemic control, providing valuable insights into the impact of DM on dental implant outcomes, particularly among patients with uncontrolled DM. The results revealed several significant differences among non-diabetic patients and those with controlled and uncontrolled DM.

Table 9. Characteristics of failed implants at the patient level

No.	Age [years]	HbA1c level prior to implantation [%]	Span length	Implant brand	Implant length [mm]	Implant diameter [mm]	Implant location	Pristine/ augmented bone
1	64	6.8[2]	long-span partial edentulism	Zimmer	11.5	3.7	anterior mandible	pristine
2	68	8.2[3]	short-span partial edentulism	Ditron	11.5	4.1	anterior mandible	augmented
3	69	8.0[2]	short-span partial edentulism	Zimmer	11.5	3.7	posterior maxilla	augmented
4	61	8.6[3]	long-span partial edentulism	Zimmer	13.0	3.75	anterior mandible	augmented
5	52	7.9[2]	long-span partial edentulism	Zimmer	13.0	5.0	posterior maxilla	augmented
6	64	11.3[3]	long-span partial edentulism	Ditron	13.0	3.7	posterior maxilla	augmented
7	65	9.5[3]	long-span partial edentulism	Zimmer	11.5	3.7	premolar mandible	augmented
8	81	5.8[1]	short-span partial edentulism	Zimmer	11.5	3.75	anterior mandible	augmented
9	76	5.8[1]	long-span partial edentulism	Zimmer	8.0	4.2	posterior mandible	augmented
10	64	6.2[1]	short-span partial edentulism	MIS	13.0	3.75	anterior mandible	augmented
11	68	6.2[1]	short-span partial edentulism	Zimmer	10.0	3.75	premolar mandible	augmented
12	78	6.3[1]	short-span partial edentulism	Zimmer	13.0	3.75	posterior maxilla	augmented

[1] non-diabetic; [2] controlled DM; [3] uncontrolled DM.

Osseointegration is the biological process through which a direct interface forms between bone and an implant.[25] Dental implants rely on this process for their initial success; therefore, any disruption may increase the risk of EIF.[4] Prolonged hyperglycemia has been associated with several mechanisms that may impair osseointegration and contribute to EIF. These include the tissue damage resulting from insulin-independent glucose uptake by endothelial cells, endothelial dysfunction and vascular impairment. Additionally, hyperglycemia may impair polymorphonuclear leukocyte migration and phagocytosis while enhancing the inflammatory response to microbial stimuli.[4] These alterations may compromise angiogenesis and disrupt the inflammatory response, both of which are essential for successful osseointegration. Furthermore, in the context of hyperglycemia, the formation of advanced glycation end products (AGEs) is of particular concern. These compounds result from non-enzymatic reactions between sugars and proteins or lipids, a process accelerated by elevated glucose levels. The accumulation of AGEs has been associated with impaired osteoblast differentiation and function, alterations in bone matrix formation and quality, and compromised angiogenesis, all of which are critical for bone healing around implants.[26–33] By directly affecting bone cells and the structural integrity of bone tissue, AGEs may further compromise the achievement of successful osseointegration in individuals with hyperglycemia.[34]

The statistical analysis revealed that neither the DM status nor glycemic control was a statistically significant risk factor for EIF at either the patient or implant level. This finding is consistent with several previous studies. Ghiraldini et al. found no significant differences in implant stability 3 months after implantation between patients with well-controlled (HbA1c < 8%) and poorly controlled (HbA1c > 8.1%) DM.[35] Oates et al. similarly demonstrated that patients with poorly controlled DM did not exhibit significantly reduced implant stability at 16 weeks after implantation.[17] Shi et al. conducted a meta-analysis to assess the association between poorly controlled DM and EIF. Their findings did not demonstrate a significant difference in the implant failure rates between patients with controlled and uncontrolled DM.[36]

Patients with uncontrolled DM were notably younger, highlighting the potential oral health implications of poor glycemic control, which is associated with an increased risk of periodontitis and tooth loss. These patients may experience tooth loss at an earlier age, leading to the need for implant therapy at a younger age.[37] This finding is consistent with the existing literature, as several studies have reported an association between better glycemic control and an older age.[38,39] This observation underscores the importance of effective diabetes management from an early age to help delay or potentially prevent tooth loss and other oral health complications.

Consistent with previous studies, we found that the dental status of patients with uncontrolled DM prior to implant placement was significantly compromised.[14] A higher proportion of patients with poorly controlled DM required bone augmentation procedures. Type 2 DM may exacerbate the host inflammatory response to oral plaque, thereby aggravating bone resorption associated with periodontitis and peri-implantitis.[39–45] Patients with DM exhibit increased levels of pro-inflammatory cytokines in bone tissues, including interleukin (IL)-1β, IL-6, and tumor necrosis factor alpha (TNF-α).[46–49] Consequently, increased osteoclast activity may lead to greater bone resorption. Previous studies have also reported that good glycemic control improves osteoclast function, and helps prevent periodontal bone loss and tooth loss.[50,51] These findings are consistent with previous research, as well as with the results of the present study, which indicate a higher prevalence of EIF in patients with augmented bone.[22,52,53] This evidence underscores the importance of preserving natural bone and preventing bone loss associated with periodontal disease. Additionally, a significant difference was observed in the mean implant length between the 3 groups, with patients with well-controlled DM receiving shorter implants, on average, than those with poorly controlled DM. This difference may be attributable to the additional bone volume gained through the augmentation procedures, which may have allowed the placement of longer implants.

The results also revealed significant differences in implant location between patients with controlled and uncontrolled DM. Uncontrolled DM was associated with a higher likelihood of implants being placed in the anterior maxilla, whereas controlled DM was more frequently associated with implant placement in the mandibular molar region. These findings highlight the importance of considering anatomical factors when evaluating implant placement patterns in patients with DM. They may also reflect more pronounced bone resorption and periodontal damage in the anterior maxilla among patients with uncontrolled DM, although this interpretation requires further investigation. To the best of our knowledge, no previous studies have specifically reported differences in implant location according to glycemic control. However, these findings are supported by a previous study conducted in healthy individuals.[54]

Another finding regarding implant location was the lower incidence of EIF in implants placed in the anterior and premolar regions of the maxilla, compared with the higher incidence observed in the anterior mandible. This difference may be attributed to the superior vascular supply of the maxilla as compared to the mandible, which may promote osseointegration.[55] The relatively limited vascular supply in the anterior mandible may, in turn, contribute to the higher EIF rates observed in this region.[55] We hypothesize that this anatomical factor may be particularly relevant in patients with DM, who often experience microvascular damage.[1] Impaired blood flow could further compromise osseointegration in areas with inherently

limited vascularity, such as the anterior mandible, potentially increasing the risk of EIF. Further studies are needed to investigate this proposed mechanism.

The results of this study call for a reevaluation of the current approach to treating patients with uncontrolled DM who require dental implant therapy. Although achieving optimal glycemic control remains the ideal goal, this is not always attainable, particularly in individuals with complex health conditions or socioeconomic barriers.[56] Despite numerous studies investigating implant outcomes in patients with DM, definitive evidence regarding its independent negative impact on EIF remains inconclusive.[17–20,35,36,51,57] In fact, some studies suggest that implant therapy in patients with DM may indirectly contribute to improved glycemic control. The restoration of masticatory function through implant-supported prostheses may facilitate healthier dietary choices and improve overall health, potentially contributing to a reduction in the HbA1c levels.[22]

Furthermore, the improvement in QoL associated with implant therapy, both functionally and psychologically, is substantial and should not be underestimated.[58] The ability to eat comfortably, speak clearly and smile confidently can have a profound impact on a patient's overall well-being. Denying these benefits solely on the basis of uncontrolled DM may represent an overly restrictive approach. Our findings suggest that, although uncontrolled DM may present certain challenges in implant dentistry, including a potentially higher risk of peri-implantitis and consequently reduced implant survival, it should not be considered an absolute contraindication to implant therapy. Instead, treatment decisions should be individualized, taking into account the patient's overall health status, degree of glycemic control, and risk factors for implant complications.

Limitations

The limitations of this study stem primarily from its retrospective design and the involvement of multiple operators, which may have introduced variability in data recording and surgical techniques, thereby limiting the generalizability of the findings. Although this study represents one of the larger investigations of diabetic patients undergoing implant therapy, a larger sample size may help identify additional statistically significant predictors and subtle differences in implant failure risk, as suggested by the wide confidence intervals observed in our analyses. Additionally, data on medication use were unavailable. It is possible that antidiabetic medications may influence implant outcomes and potentially reduce the EIF rates in these patients.

Another potential limitation is the possibility of selection bias. Previous studies have raised concerns regarding the oral complications associated with chronic hyperglycemia, including periodontitis, hyposalivation, impaired inflammatory responses, and potentially EIF. These concerns may have prompted clinicians to adopt a more cautious approach when treating patients with uncontrolled DM. Consequently, such patients may have been advised to improve their glycemic control before undergoing the implant placement or bone augmentation procedures. This selection process could have reduced the observed risk of implant failure, even among patients with elevated HbA1c levels.

Lastly, although the HbA1c levels were measured within 3 months before implant placement, fluctuations in glycemic control over time were not accounted for. This may have resulted in the misclassification of the patients' glycemic status and potentially influenced the observed associations between glycemic control and EIF.

Conclusions

This study indicates that, although uncontrolled DM may present certain challenges in implant dentistry, including a younger age at implant placement, an increased need for bone augmentation, and a preference for specific implant sites, it does not significantly affect the EIF rates.

Ethics approval and consent to participate

The research adhered to the ethical standards outlined in the Declaration of Helsinki, and received approval from the institutional review board at Rabin Medical Center, Petah Tiqwa, Israel (No. 0674-19-RMC). All study participants provided written informed consent prior to their inclusion.

Data availability

The datasets supporting the findings of the current study are available from the corresponding author on reasonable request.

Consent for publication

Not applicable.

Use of AI and AI-assisted technologies

Not applicable.

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References

1. Chrcanovic BR, Albrektsson T, Wennerberg A. Diabetes and oral implant failure: A systematic review. *J Dent Res*. 2014;93(9):859–867. doi:10.1177/0022034514538820
2. Khader YS, Dauod AS, El-Qaderi SS, Alkafajei A, Batayha WQ. Periodontal status of diabetics compared with nondiabetics: A meta-analysis. *J Diabetes Complications*. 2006;20(1):59–68. doi:10.1016/j.jdiacomp.2005.05.006
3. Blumer S, Eliasi H, Peretz B, Kharouba J, Jonas E. Knowledge and decision-making among Israeli dentists treating young patients with type 1 diabetes mellitus: A cross-sectional survey. *J Clin Pediatr Dent*. 2022;46(3):225–232. doi:10.17796/1053-4625-46.3.9
4. Javed F, Romanos GE. Impact of diabetes mellitus and glycemic control on the osseointegration of dental implants: A systematic literature review. *J Periodontol*. 2009;80(11):1719–1730. doi:10.1902/jop.2009.090283
5. Nissan J, Zenziper E, Rosner O, Kolerman R, Chaushu L, Chaushu G. The effect of mucosal cuff shrinkage around dental implants during healing abutment replacement. *J Oral Rehabil*. 2015;42(10):774–778. doi:10.1111/joor.12315
6. Nissan J, Snir D, Rosner O, Kolerman R, Chaushu L, Chaushu G. Reliability of retrievable cemented implant-supported prostheses. *J Prosthet Dent*. 2016;115(5):587–591. doi:10.1016/j.prosdent.2015.10.013
7. Kolerman R, Qahaz N, Barnea E, et al. Allograft and collagen membrane augmentation procedures preserve the bone level around implants after immediate placement and restoration. *Int J Environ Res Public Health*. 2020;17(4):1133. Published 2020 Feb 11. doi:10.3390/ijerph17041133
8. Esteves Fernandes PR, Pelaez Otero AI, Hasse Fernandes JC, Nassani LM, Castilho RM, Fernandes GVdO. Clinical performance comparing titanium and titanium-zirconium or zirconia dental implants: A systematic review of randomized controlled trials. *Dent J (Basel)*. 2022;10(5):83. doi:10.3390/dj10050083
9. Remísio MJdS, Borges T, Castro F, Gehrke SA, Hasse Fernandes JC, Fernandes GVdO. Histologic osseointegration level comparing titanium and zirconia dental implants: Meta-analysis of preclinical studies. *Int J Oral Maxillofac Implants*. 2023;38(4):667–680. doi:10.11607/jomi.10142
10. Shrivastava D, Quadri SA, Alshadidi AA, et al. Clinical assessment of the relationship of dental implant materials (titanium and zirconia) and peri-implantitis: A systematic review. *J Maxillofac Oral Surg*. 2025;24(4):1010–1028. doi:10.1007/s12663-024-02409-9
11. Samara W, Moztarzadeh O, Hauer L, Babuska V. Dental implant placement in medically compromised patients: A literature review. *Cureus*. 2024;16(2):e54199. doi:10.7759/cureus.54199
12. Michaeli E, Weinberg I, Nahlieli O. Dental implants in the diabetic patient: Systemic and rehabilitative considerations. *Quintessence Int*. 2009;40(8):639–645. PMID:19639088.
13. Wagner J, Spille JH, Wiltfang J, Naujokat H. Systematic review on diabetes mellitus and dental implants: An update. *Int J Implant Dent*. 2022;8(1):1. doi:10.1186/s40729-021-00399-8
14. Tan SJ, Baharin B, Nabil S, Mohd N, Zhu Y. Does glycemic control have a dose–response relationship with implant outcomes? A comprehensive systematic review and meta-analysis. *J Evid Based Dent Pract*. 2021;21(2):101543. doi:10.1016/j.jebdp.2021.101543
15. Guarnieri R, Reda R, Zanza A, Miccoli G, Di Nardo D, Testarelli L. Can peri-implant marginal bone loss progression and a-MMP-8 be considered indicators of the subsequent onset of peri-implantitis? A 5-year study. *Diagnostics (Basel)*. 2022;12(11):2599. doi:10.3390/diagnostics12112599
16. Guarnieri R, Reda R, Di Nardo D, Miccoli G, Zanza A, Testarelli L. Clinical, radiographic, and biochemical evaluation of two-piece versus one-piece single implants with a laser-microgrooved collar surface after 5 years of functional loading. *Clin Implant Dent Relat Res*. 2022;24(5):676–682. doi:10.1111/cid.13118
17. Oates TW, Dowell S, Robinson M, McMahan CA. Glycemic control and implant stabilization in type 2 diabetes mellitus. *J Dent Res*. 2009;88(4):367–371. doi:10.1177/0022034509334203
18. Eskow CC, Oates TW. Dental implant survival and complication rate over 2 years for individuals with poorly controlled type 2 diabetes mellitus. *Clin Implant Dent Relat Res*. 2017;19(3):423–431. doi:10.1111/cid.12465
19. Chae SW, Kim YS, Kim WK, Lee YK, Kim SH. Effect of glycemic control on the incidence of dental implant complications in diabetic patients. *J Implant Appl Sci*. 2023;27(4):163–173. doi:10.32542/implantology.2023019
20. Tang D, Wang E, Xu Y, et al. Is hyperglycemia the only risk factor for implant in type 2 diabetics during the healing period? *Oral Dis*. 2021;27(6):1551–1563. doi:10.1111/odi.13685
21. De Angelis P, Manicone PF, Gasparini G, et al. The effect of controlled diabetes and hyperglycemia on implant placement with simultaneous horizontal guided bone regeneration: A clinical retrospective analysis. *Biomed Res Int*. 2021;2021:9931505. doi:10.1155/2021/9931505
22. Masri D, Masri-Iraqi H, Nissan J, et al. On the association between implant-supported prosthesis and glycemic control (HbA1c values). *Int J Environ Res Public Health*. 2022;19(11):6923. doi:10.3390/ijerph19116923
23. Masri D, Masri-Iraqi H, Naishlos S, Weinberg E, Reiser V, Chaushu L. Implant-supported prosthesis is a viable treatment alternative for American Society of Anesthesiology physical status 3 individuals – a retrospective cohort study. *J Clin Med*. 2022;11(7):2002. doi:10.3390/jcm11072002
24. Jonas E, Masri D, Avishai G, Masri-Iraqi H, Chaushu G, Chaushu L. The impact of antiplatelet and anticoagulant medications on early implant failure following sinus floor augmentation: A retrospective cohort analysis. *Clin Implant Dent Relat Res*. 2024;26(5):1046–1055. doi:10.1111/cid.13369
25. Albrektsson T, Brånemark PI, Hansson HA, Lindström J. Osseointegrated titanium implants. Requirements for ensuring a long-lasting, direct bone-to-implant anchorage in man. *Acta Orthop Scand*. 1981;52(2):155–170. doi:10.3109/17453678108991776
26. Park SY, Choi KH, Jun JE, Chung HY. Effects of advanced glycation end products on differentiation and function of osteoblasts and osteoclasts. *J Korean Med Sci*. 2021;36(37):e239. doi:10.3346/jkms.2021.36.e239
27. Jia T, Wang YN, Zhang D, Xu X. 1α,25-dihydroxyvitamin D3 promotes osseointegration of titanium implant via downregulating AGEs/RAGE pathway in T2DM. *Endocr Connect*. 2018;7(11):1186–1195. doi:10.1530/EC-18-0241
28. Phimphilai M, Pothacharoen P, Kongtawelert P. Age-influenced receptors of advanced glycation end product overexpression associated with osteogenic differentiation impairment in patients with type 2 diabetes. *Front Endocrinol (Lausanne)*. 2021;12:726182. doi:10.3389/fendo.2021.726182
29. Phimphilai M, Pothacharoen P, Chattipakorn N, Kongtawelert P. Receptors of advanced glycation end product (RAGE) suppression associated with a preserved osteogenic differentiation in patients with prediabetes. *Front Endocrinol (Lausanne)*. 2022;13:799872. doi:10.3389/fendo.2022.799872
30. Marin C, Luyten FP, Van der Schueren B, Kerckhofs G, Vandamme K. The impact of type 2 diabetes on bone fracture healing. *Front Endocrinol (Lausanne)*. 2018;9:6. doi:10.3389/fendo.2018.00006
31. Stirban A, Gawlowski T, Roden M. Vascular effects of advanced glycation endproducts: Clinical effects and molecular mechanisms. *Mol Metab*. 2013;3(2):94–108. doi:10.1016/j.molmet.2013.11.006
32. Asadipooya K, Uy EM. Advanced glycation end products (AGEs), receptor for AGEs, diabetes, and bone: Review of the literature. *J Endocr Soc*. 2019;3(10):1799–1818. doi:10.1210/js.2019-00160
33. Yamamoto M, Sugimoto T. Advanced glycation end products, diabetes, and bone strength. *Curr Osteoporos Rep*. 2016;14(6):320–326. doi:10.1007/s11914-016-0332-1
34. Cosola S, Butera A, Zergaw AH, et al. Glycemic control and implant stability in patients with type II diabetes: Narrative review. *Healthcare (Basel)*. 2025;13(5):449. doi:10.3390/healthcare13050449
35. Ghiraldini B, Conte A, Casarin RC, et al. Influence of glycemic control on peri-implant bone healing: 12-month outcomes of local release of bone-related factors and implant stabilization in type 2 diabetics. *Clin Implant Dent Relat Res*. 2016;18(4):801–809. doi:10.1111/cid.12339
36. Shi Q, Xu J, Huo N, Cai C, Liu H. Does a higher glycemic level lead to a higher rate of dental implant failure? A meta-analysis. *J Am Dent Assoc*. 2016;147(11):875–881. doi:10.1016/j.adaj.2016.06.011
37. Genco RJ, Graziani F, Hasturk H. Effects of periodontal disease on glycemic control, complications, and incidence of diabetes mellitus. *Periodontol 2000*. 2020;83(1):59–65. doi:10.1111/prd.12271

38. Berkowitz SA, Meigs JB, Wexler DJ. Age at type 2 diabetes onset and glycaemic control: Results from the National Health and Nutrition Examination Survey (NHANES) 2005–2010. *Diabetologia*. 2013;56(12):2593–2600. doi:10.1007/s00125-013-3036-4
39. Shamshirgaran SM, Mamaghanian A, Aliasgarzadeh A, Aiminisani N, Iranparvar-Alamdari M, Ataie J. Age differences in diabetes-related complications and glycemic control. *BMC Endocr Disord*. 2017;17(1):25. doi:10.1186/s12902-017-0175-5
40. Andriankaja OM, Barros SP, Moss K, et al. Levels of serum interleukin (IL)-6 and gingival crevicular fluid of IL-1beta and prostaglandin E(2) among non-smoking subjects with gingivitis and type 2 diabetes. *J Periodontol*. 2009;80(2):307–316. doi:10.1902/jop.2009.080385
41. Chaushu L, Tal H, Sculean A, Fernández-Tomé B, Chaushu G. Peri-implant disease affects systemic complete blood count values – an experimental in vivo study. *Clin Oral Investig*. 2020;24(12):4531–4539. doi:10.1007/s00784-020-03318-0
42. Chaushu L, Tal H, Sculean A, Fernández-Tomé B, Chaushu G. Effects of peri-implant infection on serum biochemical analysis. *J Periodontol*. 2021;92(3):436–445. doi:10.1002/JPER.20-0048
43. Chaushu L, Weinreb M, Beitlitum I, Moses O, Nemcovsky CE. Evaluation of a topical herbal patch for soft tissue wound healing: An animal study. *J Clin Periodontol*. 2015;42(3):288–293. doi:10.1111/jcpe.12372
44. Lo Bianco L, Montevecchi M, Ostanello M, Checchi V. Recognition and treatment of peri-implant mucositis: Do we have the right perception? A structured review. *Dent Med Probl*. 2021;58(4):545–554. doi:10.17219/dmp/136359
45. Fernandes GVdO, Martins BGdS, Fraile JF. Revisiting peri-implant diseases in order to rethink the future of compromised dental implants: Considerations, perspectives, treatment, and prognosis. *Dent Med Probl*. 2024;61(5):637–640. doi:10.17219/dmp/187215
46. Graves DT, Cochran D. The contribution of interleukin-1 and tumor necrosis factor to periodontal tissue destruction. *J Periodontol*. 2003;74(3):391–401. doi:10.1902/jop.2003.74.3.391
47. Graves D. Cytokines that promote periodontal tissue destruction. *J Periodontol*. 2008;79(Suppl 8):1585–1591. doi:10.1902/jop.2008.080183
48. Cochran DL. Inflammation and bone loss in periodontal disease. *J Periodontol*. 2008;79(Suppl 8):1569–1576. doi:10.1902/jop.2008.080233
49. Al-Askar M, Ajlan S, Alomar N, Al-Daghri NM. Clinical and radiographic peri-implant parameters and whole salivary interleukin-1β and interleukin-6 levels among type-2 diabetic and nondiabetic patients with and without peri-implantitis. *Med Princ Pract*. 2018;27(2):133–138. doi:10.1159/000488032
50. Araujo MG, Lindhe J. Peri-implant health. *J Clin Periodontol*. 2018;45(Suppl 20):S230–S236. doi:10.1111/jcpe.12952
51. Taylor GW, Burt BA, Becker MP, Genco RJ, Shlossman M. Glycemic control and alveolar bone loss progression in type 2 diabetes. *Ann Periodontol*. 1998;3(1):30–39. doi:10.1902/annals.1998.3.1.30
52. Chatzopoulos GS, Wolff LF. Dental implant failure and bone augmentation: A retrospective study. *J Clin Exp Dent*. 2023;15(3):e195–e204. doi:10.4317/jced.60171
53. Alshiddi IF. Survival rate and clinico-radiographic parameters around narrow-diameter dental implants for fixed dental prostheses in the posterior regions: A systematic review. *Dent Med Probl*. 2023;60(2):345–353. doi:10.17219/dmp/140757
54. Olate S, Negreiros Lyrio MC, de Moraes M, Mazzonetto R, Fernandes Moreira RW. Influence of diameter and length of implant on early dental implant failure. *J Oral Maxillofac Surg*. 2010;68(2):414–419. doi:10.1016/j.joms.2009.10.002
55. Tsaï GE, Volkov SI, Lavrent'ev PA, Lavrent'ev AA. Blood supply topographic features in maxilla and mandible [in Russian]. *Stomatologiia (Mosk)*. 2015;94(1):7–10. doi:10.17116/stomat20159417-10
56. Qaseem A, Wilt TJ, Kansagara D, et al. Hemoglobin A1c targets for glycemic control with pharmacologic therapy for nonpregnant adults with type 2 diabetes mellitus: A guidance statement update from the American College of Physicians. *Ann Intern Med*. 2018;168(8):569–576. doi:10.7326/M17-0939
57. Oates Jr. TW, Galloway P, Alexander P, et al. The effects of elevated hemoglobin A(1c) in patients with type 2 diabetes mellitus on dental implants: Survival and stability at one year. *J Am Dent Assoc*. 2014;145(12):1218–1226. doi:10.14219/jada.2014.93
58. Sargozaie N, Moeintaghavi A, Shojaie H. Comparing the quality of life of patients requesting dental implants before and after implant. *Open Dent J*. 2017;11:485–491. doi:10.2174/1874210601711010485