

Impact of Glycemic Control on Dental Implant Survival and Peri-Implant Health in Patients with Diabetes Mellitus: A Narrative Review

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Abstract

Background: Diabetes mellitus (DM) is one of the most common systemic conditions encountered in implant dentistry. Hyperglycemia has been proposed to compromise osseointegration, delay wound healing, and heighten susceptibility to peri-implant inflammatory disease, but the clinical significance of glycemic control specifically — rather than the diagnosis of diabetes alone — remains incompletely defined.

Objective: To synthesise current evidence on the relationship between glycemic control, most commonly quantified by glycated haemoglobin (HbA1c), and dental implant survival, marginal bone loss, and peri-implant health in patients with diabetes mellitus.

Methods: A structured narrative review was conducted using PubMed/MEDLINE, Scopus, and Google Scholar. Eligible evidence included cohort studies, retrospective and prospective clinical studies, systematic reviews, and meta-analyses reporting implant survival, marginal bone loss, peri-implant probing depth, bleeding on probing, or peri-implantitis in relation to diabetes status or glycemic control, published between 2016 and 2026. Findings were synthesised thematically because of substantial heterogeneity in glycemic thresholds, follow-up duration, and outcome definitions.

Results: Implant survival in well-controlled diabetes (HbA1c approximately $\leq 7\%$) was consistently comparable to non-diabetic populations across long-term retrospective cohorts. Poorly controlled diabetes (HbA1c $> 8\%$) was associated with greater marginal bone loss, higher probing depth, increased bleeding on probing, elevated peri-implant inflammatory cytokines, and a higher pooled odds of implant failure, particularly in type 1 diabetes and the maxilla. Mechanistic evidence implicated advanced glycation end-product accumulation, oxidative stress, impaired angiogenesis, and dysregulated osteoblast–osteoclast coupling as pathways linking hyperglycemia to compromised bone healing. Most primary evidence remained observational and retrospective, limiting causal inference.

Conclusion: Diabetes mellitus is not an absolute contraindication to implant therapy. Glycemic control, rather than the diagnosis of diabetes per se, is the clinically decisive variable: implants placed in well-controlled diabetic patients achieve survival rates approaching those of non-diabetic patients, whereas poor glycemic control is associated with a measurably higher burden of peri-implant complications. Pre-surgical HbA1c assessment and glycemic optimisation should be integrated into implant treatment planning.

Keywords: Dental implants; diabetes mellitus; glycemic control; HbA1c; osseointegration; peri-implantitis; marginal bone loss

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1. Introduction

Dental implants have become a predictable and widely used method for the rehabilitation of partially and fully edentulous patients, with long-term survival rates typically exceeding 90–95% in the general population. As implant therapy has expanded, an increasing proportion of candidates present with systemic comorbidities, among which diabetes mellitus (DM) is one of the most prevalent. The global burden of diabetes continues to rise, driven by increasing obesity, sedentary lifestyles, and population ageing, and diabetic patients experience disproportionately higher rates of tooth loss and edentulism, making them a growing segment of the implant-seeking population (4,5,6).

Diabetes mellitus is a metabolic disorder impacting bone quality and bone healing through several interacting mechanisms. Chronic hyperglycemia promotes the accumulation of advanced glycation end products (AGEs) within collagen and bone matrix, increases oxidative stress and reactive oxygen species generation, disrupts angiogenesis, and impairs the coupled activity of osteoblasts and osteoclasts required for normal bone remodelling (16,17,18). These disturbances are most consequential during the early phases of osseointegration, when new bone formation and vascular ingrowth around the implant surface determine long-term mechanical stability. Beyond bone metabolism, diabetes is an established risk factor for periodontitis, and comparable inflammatory pathways are increasingly implicated in peri-implant mucositis and peri-implantitis, raising the concern that diabetic patients may also be more susceptible to biologic complications after successful initial healing (14,15).

For many years, diabetes was treated as a relative or even absolute contraindication to implant placement. However, accumulating clinical evidence suggests that the relationship between diabetes and implant outcomes is not uniform: it depends heavily on the quality of glycemic control, most frequently quantified using glycated haemoglobin (HbA1c), rather than on the diagnosis of diabetes in isolation. Several long-term retrospective cohorts have reported implant survival rates in well- to moderately-controlled diabetic patients that are statistically indistinguishable from non-diabetic controls, while other studies restricted to poorly controlled patients report significantly higher rates of marginal bone loss, delayed healing, and peri-implantitis (8,9,10,11,12,13).

This inconsistency in the literature has generated two parallel, and at times conflicting, bodies of evidence: pooled meta-analyses that treat diabetes as a single dichotomous exposure often report modest or non-significant associations with implant failure, whereas studies that stratify patients by HbA1c band or duration of disease more consistently identify a dose-related relationship between glycemic burden and adverse peri-implant outcomes (1,2,3,4). Clarifying this distinction has direct clinical relevance: it determines whether implant candidacy in diabetic patients should be assessed primarily by diagnosis or by a quantifiable, modifiable physiological threshold that can be optimised prior to surgery.

This narrative review therefore aimed to synthesise current evidence specifically addressing the influence of glycemic control — rather than diabetes status alone — on dental implant survival, marginal bone loss, and peri-implant soft- and hard-tissue health, and to translate this evidence into practical considerations for pre-surgical assessment, perioperative management, and long-term maintenance of implants in patients with diabetes mellitus.

2. Materials and Methods

A structured narrative review was undertaken to synthesise evidence concerning the relationship between glycemic control and dental implant outcomes in patients with diabetes mellitus. A narrative rather than systematic approach was selected because of substantial heterogeneity across the primary literature in diabetes classification, glycemic thresholds used to define “well-controlled,” “moderately controlled,” and “poorly controlled” disease, implant systems, follow-up duration, and outcome definitions, which precluded meaningful quantitative pooling within the scope of this review.

Relevant literature was identified through electronic searches of PubMed/MEDLINE, Scopus, and Google Scholar. Search concepts combined terms relating to implant dentistry (“dental implants,” “osseointegration,” “implant survival,” “peri-implantitis,” “marginal bone loss”) with terms relating to diabetes and glycemic status (“diabetes mellitus,” “hyperglycemia,” “glycemic control,” “HbA1c,” “glycated haemoglobin,” “prediabetes”). Reference lists of retrieved systematic reviews and primary studies were hand-searched to identify additional relevant publications.

Emphasis was placed on literature published between 2016 and 2026 to reflect contemporary implant protocols, diagnostic thresholds for peri-implant disease, and glycemic classification standards. Eligible study designs included retrospective and prospective cohort studies, case-control studies, cross-sectional clinical studies, systematic reviews, and meta-analyses that reported an identifiable implant-related outcome (survival, failure, marginal bone loss, probing depth, bleeding on probing, or peri-implantitis incidence) in relation to diabetes status or a quantified measure of glycemic control. Only English-language, peer-reviewed publications with sufficient methodological detail to support interpretation were included. Case reports, conference abstracts without adequate data, and studies that did not differentiate implant-related outcomes from unrelated oral conditions were excluded.

Data extracted from eligible studies included study design, population and sample size, method of glycemic classification (HbA1c thresholds where reported), implant-related outcomes assessed, follow-up duration, principal findings, and proposed mechanisms. Findings were organised thematically into domains addressing glycemic thresholds and implant survival, marginal bone loss, peri-implant soft-tissue inflammation, biological and molecular mechanisms, type 1 versus type 2 diabetes, and clinical and perioperative management implications. No quantitative meta-analysis was performed; instead, consistency of findings across study designs and populations was used to gauge the strength of the overall evidence base.

3. Results

3.1 Characteristics of the Evidence Base

The reviewed literature comprised long-term retrospective cohort studies, prospective clinical studies, systematic reviews, and meta-analyses, most originating from specialist implant, periodontology, or oral surgery centres. Glycemic status was most frequently classified using HbA1c, although thresholds defining “well,” “moderate,” and “poor” control varied: some studies used <7% versus >8% as well- versus poorly-controlled, others used three-tier classifications (e.g., <6%, 6–8%, >8%), and a minority relied solely on clinical diagnosis without reporting HbA1c values (4,6,8,9). This heterogeneity limited direct quantitative

comparison across studies but did not prevent identification of a broadly consistent thematic pattern: outcomes deteriorated as glycemic control worsened, and this gradient was more informative than a simple diabetic/non-diabetic dichotomy.

3.2 Glycemic Control and Implant Survival

Across long-term retrospective cohorts, implant survival in well- to moderately-controlled diabetic patients was consistently comparable to non-diabetic populations. Ormianer and colleagues followed 169 patients with 1,112 implants and reported survival and bone-loss outcomes in moderately controlled type 2 diabetes (HbA1c up to 8%) that were similar to reported non-diabetic rates, with the least bone loss observed following delayed implant insertion protocols (8). A related long-term study by the same group, focused on patients with moderately to poorly controlled HbA1c values up to 10%, still reported a 98.4% overall implant survival rate across 357 implants, although marginal bone loss increased progressively with higher HbA1c (9).

At the pooled level, the largest meta-analysis to date, incorporating 89 publications and more than 68,000 implants, found that implants placed in diabetic patients overall carried a 77.7% higher risk of failure compared with non-diabetic patients, with the differential risk significantly greater in type 1 than type 2 diabetes and more pronounced in the maxilla than the mandible (4). Other systematic reviews and overviews of systematic reviews similarly concluded that survival and failure estimates in diabetic cohorts were inconsistent across the literature, largely because studies rarely standardised glycemic control status, disease duration, or diabetes type when reporting outcomes (5,1,3).

3.3 Marginal Bone Loss and Osseointegration

Marginal bone loss (MBL) around implants showed one of the most consistent gradients with glycemic control. In the Lorean et al. cohort, mean MBL was significantly greater in the poorly controlled group (HbA1c 8.1–10.0%) than in the moderately controlled group (HbA1c 6.9–8.0%), and the maxilla demonstrated greater bone loss than the mandible in both groups (9). A meta-analysis by Jiang and colleagues similarly reported significantly higher MBL, probing depth, and bleeding on probing in diabetic compared with non-diabetic implant patients, with low to moderate heterogeneity for the bone-loss outcome specifically (6). A seven-year prospective study by Al Zahrani and Al Mutairi reported greater long-term crestal bone loss in type 2 diabetic patients regardless of whether implants were submerged or non-submerged at placement (10).

Mechanistic and translational evidence provides biological plausibility for these clinical observations. Chronic hyperglycemia drives accumulation of advanced glycation end products within the bone collagen matrix, disrupting normal intrafibrillar mineralisation and reducing bone mechanical strength even when bone mineral density appears preserved — a phenomenon described as the diabetic bone paradox (18). Reviews of bone healing under diabetic conditions further describe oxidative stress, impaired mesenchymal stem cell osteogenic differentiation, disrupted angiogenesis, and dysregulated macrophage polarisation as converging mechanisms that delay early peri-implant bone formation and may compromise the stability of the bone-to-implant interface during the critical early healing window (16,17).

3.4 Peri-Implant Soft-Tissue Inflammation and Peri-Implantitis

Beyond bone loss, diabetes and glycemic control were associated with heightened peri-implant soft-tissue inflammation. Alrabiah and colleagues found that higher AGE concentrations correlated with greater peri-implant inflammation across prediabetic, type 2 diabetic, and healthy comparison groups, suggesting a dose-related biological gradient rather than a binary diabetic effect (11). Al-Askar and colleagues reported elevated whole salivary interleukin-1 β and interleukin-6 levels in diabetic patients with peri-implantitis compared with diabetic patients without peri-implantitis and non-diabetic controls, supporting an amplified local inflammatory response in the presence of hyperglycemia (13). Broader epidemiological syntheses of peri-implantitis risk factors have repeatedly identified diabetes mellitus, alongside smoking and a history of periodontitis, as a consistent risk indicator, with pooled odds ratios in the range of approximately 2 to 4.5 depending on the population studied (14,15).

Conversely, when glycemic control was strictly maintained, soft-tissue and bone parameters around implants — including in complex scenarios involving adjacent implants — remained stable and comparable to non-diabetic patients over multi-year follow-up, reinforcing that the inflammatory burden associated with diabetes is substantially modifiable through metabolic control rather than fixed by diagnosis alone (12).

3.5 Type 1 versus Type 2 Diabetes and Disease Duration

Relatively few studies directly compared implant outcomes between type 1 and type 2 diabetes, but the available meta-regression evidence suggests type 1 diabetes carries a disproportionately higher failure risk, potentially reflecting earlier disease onset, longer cumulative hyperglycemic exposure, and a greater prevalence of microvascular complications by the time of implant placement (4). Disease duration and cumulative glycemic burden, rather than glycemic status at a single time point, may therefore be additional determinants of implant risk that are not fully captured by a single pre-operative HbA1c value.

3.6 Screening and Clinical Translation

An emerging area of evidence concerns opportunistic glycemic screening in implant candidates without a prior diagnosis of diabetes. In a retrospective cohort of implant candidates exhibiting oral signs suggestive of undiagnosed dysglycemia, pre-surgical HbA1c testing identified diabetes in approximately 42% and prediabetes in approximately 30% of those screened, indicating that a meaningful proportion of implant patients may have unrecognised glycemic abnormalities relevant to surgical planning (20). Regional clinical studies from varied practice settings have echoed the broader pattern: implant failure clusters disproportionately in the highest HbA1c strata, while patients with well-controlled diabetes achieve favourable, predictable outcomes (21,19,22).

Table 1 summarises representative primary studies illustrating the relationship between glycemic control and implant outcomes, and Table 2 presents a thematic synthesis of the evidence domains, interpretations, and principal limitations identified across the reviewed literature.

Table 1. Representative Studies Examining Glycemic Control and Dental Implant Outcomes

Study	Population / Design	Glycemic Assessment	Outcome(s) Assessed	Principal Finding
Ormianer et al., 2018 (8)	169 patients, 1,112 implants; retrospective cohort	HbA1c $\leq 7\%$ (well) vs $\leq 8\%$ (moderate)	Implant survival, marginal bone loss	Survival comparable to non-diabetic population; delayed insertion protocol showed least bone loss
Lorean et al., 2021 (9)	38 patients, 357 implants; long-term retrospective	HbA1c 6.9–10.0%	Survival, marginal bone loss, prosthesis type	98.4% survival; bone loss increased with higher HbA1c (moderate 1.86 mm vs poor 2.33 mm)
Al Zahrani & Al Mutairi, 2019 (10)	Type 2 diabetics vs controls; 7-year prospective	Clinical diabetes diagnosis	Crestal bone loss, submerged vs non-submerged	Diabetic patients showed greater crestal bone loss over long-term follow-up
Alrabiah et al., 2018 (11)	Prediabetes, T2DM, and healthy controls	HbA1c-based glycemic strata	Peri-implant inflammation, AGE levels	Higher AGE levels correlated with greater peri-implant inflammation across glycemic groups
Alasqah et al., 2018 (12)	Type 2 diabetics vs non-diabetics with adjacent implants; 6-year follow-up	Strict glycemic control criteria	Soft-tissue status, crestal bone levels	Adjacent implants remained stable when glycemic levels were strictly controlled
Al-Askar et al., 2018 (13)	Type 2 diabetics and non-diabetics with/without peri-implantitis	HbA1c measurement	Clinical/radiographic parameters, salivary IL-1 β and IL-6	Diabetic patients with peri-implantitis had higher inflammatory cytokine levels
Jiang et al., 2021 (6)	Meta-analysis of comparative studies	Variable HbA1c thresholds across studies	Implant complications (bone loss, probing depth, bleeding)	Diabetic patients showed significantly greater bone loss, probing depth, and bleeding on probing
Al Ansari, Shahwan & Chrcanovic, 2022 (4)	89 publications; 5,510 diabetic and 62,780 non-diabetic implants	Mixed glycemic definitions	Implant failure, marginal bone loss	77.7% higher failure risk in diabetics overall; risk greatest in type 1 diabetes and the maxilla
Cosola et al., 2025 (7)	Narrative synthesis of clinical cohorts	HbA1c-stratified glycemic control	Implant stability, prosthetic longevity	Hyperglycemia significantly associated with implant complications; well-controlled diabetes performs comparably to healthy controls
Sharma et al., 2023 (21)	30 diabetic patients; clinical follow-up study	HbA1c stratification (8.0–11.9%)	Implant failure vs HbA1c band	Failure concentrated in the highest HbA1c strata; well-controlled patients had favorable outcomes
Erfan, Qasemian & Anis, 2024 (19)	23 articles; systematic review, type 2 diabetics	Mean HbA1c 7.26% across pooled studies	Implant success/failure rate	Pooled success rate 93.67%; failure risk rose with HbA1c, implant number, and diabetes duration
Çoban & Altay, 2025 (20)	43 patients without prior DM diagnosis; retrospective cohort	Opportunistic pre-surgical HbA1c screening	Undiagnosed dysglycemia detection	42% met diagnostic criteria for DM and 30% for prediabetes among implant candidates screened

Note. AGE, advanced glycation end product; HbA1c, glycated haemoglobin; IL, interleukin; MBL, marginal bone loss; T2DM, type 2 diabetes mellitus.

Table 2. Thematic Synthesis of Glycemic Control and Peri-Implant Outcomes

Evidence Domain	Reported Findings	Interpretation	Key Limitation
Glycemic threshold (HbA1c)	Implant survival and MBL worsen progressively above HbA1c ~8%; well-controlled diabetics (<7%) approach non-diabetic outcomes	HbA1c functions as a graded, not binary, risk marker	Thresholds for “good/moderate/poor” control vary across studies
Osseointegration / bone healing	AGE accumulation, oxidative stress, and impaired osteoblast–osteoclast coupling delay early bone-to-implant contact	Hyperglycemia biologically compromises early healing more than late maintenance	Mostly animal/mechanistic data; limited human biomarker correlation
Peri-implant soft-tissue inflammation	Higher bleeding on probing, probing depth, and inflammatory cytokines (IL-1 β , IL-6) in diabetics, especially with poor control	Diabetes amplifies the inflammatory response to peri-implant biofilm	Cross-sectional designs limit causal interpretation
Peri-implantitis risk	Diabetes mellitus consistently identified as a risk indicator (OR approx. 2–4.5) for peri-implantitis and implant failure	Diabetes should be considered alongside smoking and periodontal history in risk stratification	Risk estimates heterogeneous across pooled reviews
Type 1 vs Type 2 diabetes	Implant failure more likely in type 1 than type 2 diabetes in pooled meta-regression	Disease type and duration may matter independently of HbA1c	Few studies directly compare type 1 and type 2 cohorts
Long-term survival with good control	Multiple long-term cohorts (5–7+ years) report survival rates comparable to non-diabetic patients when glycemic control is maintained	Diabetes is not an absolute contraindication to implant therapy	Most cohorts are retrospective and single-center
Undiagnosed dysglycemia	Opportunistic HbA1c screening before implant surgery identifies previously undiagnosed diabetes/prediabetes in a substantial minority of candidates	Pre-surgical metabolic screening may have clinical value beyond known diabetics	Evidence from limited retrospective cohorts; no prospective screening trials

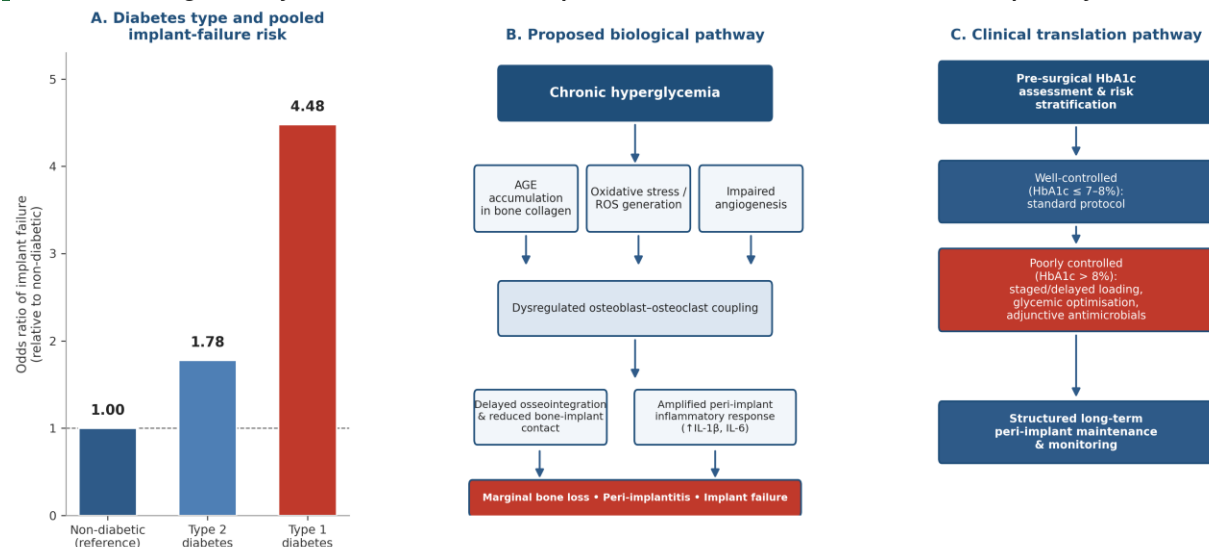


Figure 1. Glycemic control and dental implant outcomes: risk, mechanism, and clinical pathway. (A) Pooled odds ratio of implant failure by diabetes type relative to non-diabetic patients (reference = 1.0), plotted from the meta-analytic estimates of Al Ansari, Shahwan & Chrcanovic (ref. 4): overall diabetes OR 1.78 and type 1 diabetes OR 4.48. (B) Proposed biological pathway linking chronic hyperglycemia → advanced glycation end-product (AGE) accumulation, oxidative stress, and impaired angiogenesis → dysregulated osteoblast-osteoclast coupling → delayed osseointegration and an amplified peri-implant inflammatory response → marginal bone loss, peri-implantitis, and implant failure. (C) Clinical translation pathway: pre-surgical HbA1c assessment and risk stratification, standard protocol for well-controlled patients, staged/delayed loading with glycemic optimisation and adjunctive antimicrobials for poorly controlled patients, and structured long-term peri-implant maintenance.

4. Discussion

This narrative review synthesised evidence indicating that glycemic control, rather than the presence of diabetes mellitus per se, is the principal determinant of dental implant outcomes. Across multiple long-term cohorts, implants placed in patients with well- to moderately-controlled diabetes achieved survival rates statistically comparable to non-diabetic populations, whereas poorly controlled diabetes was consistently associated with greater marginal bone loss, higher probing depth, increased bleeding on probing, elevated peri-implant inflammatory cytokines, and a measurably higher pooled risk of implant failure (4,6,8,9,10). These findings are consistent with, and extend, earlier systematic reviews that identified poorly controlled diabetes, rather than diabetes as a diagnostic category, as the operative risk factor for impaired osseointegration and peri-implantitis (1,2,3).

The biological plausibility of this relationship is well supported. Chronic hyperglycemia promotes AGE accumulation within bone collagen, disrupting normal intrafibrillar mineralisation and weakening bone despite seemingly preserved mineral density (18). Oxidative stress, impaired osteogenic differentiation of mesenchymal stem cells, disrupted angiogenesis, and altered macrophage polarisation further compromise the early bone-healing response that is critical to primary implant stability (16,17). These mechanisms plausibly explain why the greatest divergence between diabetic and non-diabetic outcomes is often observed during early healing and in regions of lower bone density such as the posterior maxilla, and why the differential risk narrows once osseointegration is established and maintenance therapy is consistently delivered (4,8).

At the tissue level, diabetes appears to amplify the inflammatory response to peri-implant biofilm in a manner analogous to its well-established effect on periodontal tissues. Elevated local cytokine levels (IL-1 β , IL-6) in diabetic patients with peri-implantitis, together with epidemiological data identifying diabetes as a consistent risk indicator for peri-implant disease, suggest that hyperglycemia lowers the threshold at which bacterial biofilm triggers a destructive inflammatory response (11,13,14,15). This has direct implications for maintenance protocols: diabetic patients, particularly those with suboptimal glycemic control, may require more frequent professional maintenance and more rigorous oral hygiene reinforcement than non-diabetic implant patients.

The distinction between type 1 and type 2 diabetes, although under-studied, deserves greater clinical attention. Pooled meta-regression data suggesting disproportionately higher failure risk in type 1 diabetes may reflect differences in disease duration, microvascular complication burden, and glycemic variability rather than glycemic control at a single pre-operative time point (4). This raises the possibility that a single HbA1c measurement, while clinically useful, may not fully capture cumulative metabolic risk, and that disease duration and complication history should be incorporated into implant risk assessment alongside HbA1c.

An additional and clinically actionable finding concerns undiagnosed dysglycemia. Evidence that opportunistic HbA1c screening identifies substantial rates of previously unrecognised diabetes and prediabetes among implant candidates suggests that pre-surgical metabolic screening may have value beyond patients with an existing diagnosis, potentially altering surgical timing, perioperative protocols, or triggering referral for medical optimisation before elective implant placement (20).

These findings carry direct implications for clinical practice. First, diabetes mellitus alone should not be treated as an absolute contraindication to implant therapy; rather, pre-surgical HbA1c assessment should inform individualised risk stratification. Second, in patients with poorly controlled diabetes, clinicians may consider staged rather than immediate implant protocols, delayed loading, adjunctive antimicrobial or chlorhexidine measures, and closer perioperative glycemic monitoring, consistent with findings that delayed insertion protocols were associated with reduced bone loss in moderately controlled diabetic cohorts (8). Third, structured long-term peri-implant maintenance appears particularly important in diabetic patients given the amplified inflammatory response observed at the tissue level. Finally, collaboration with the patient's treating physician or endocrinologist to optimise glycemic control prior to elective implant surgery, where feasible, is a reasonable extension of the evidence reviewed, in line with established standards for glycemic classification and target-setting in diabetes care (23).

This review has several strengths. It integrates evidence across implant survival, marginal bone loss, peri-implant soft-tissue inflammation, and underlying molecular mechanisms rather than treating each domain in isolation, and it explicitly foregrounds glycemic control as the operative variable rather than diabetes diagnosis alone, which better reflects the modifiable, actionable nature of this risk factor in clinical practice.

Several limitations should be acknowledged. This was a narrative rather than systematic review, and formal risk-of-bias grading and quantitative pooling were not undertaken. The primary literature is dominated by retrospective, single-centre cohort studies with variable and inconsistently reported HbA1c thresholds, which limits precise quantitative comparison across studies and may inflate apparent heterogeneity in reported outcomes. Publication bias favouring studies reporting significant associations cannot be excluded.

Follow-up duration varied substantially across cohorts, and few studies adequately controlled for co-existing risk factors such as smoking, periodontal history, and implant surface characteristics when isolating the effect of glycemic control. Finally, restriction to English-language publications may have introduced language bias.

Future research should prioritise prospective, multicentre cohort studies using standardised HbA1c thresholds and consistent peri-implant disease case definitions, direct comparisons between type 1 and type 2 diabetes, and evaluation of disease duration and glycemic variability as independent predictors alongside single-point HbA1c. Randomised or controlled interventional studies evaluating pre-surgical glycemic optimisation protocols, staged versus immediate loading in poorly controlled patients, and structured maintenance intervals tailored to diabetic status would substantially strengthen the evidence base underpinning clinical decision-making.

5. Conclusion

Glycemic control, rather than the diagnosis of diabetes mellitus alone, is the principal clinically relevant determinant of dental implant survival and peri-implant health. Implants placed in patients with well- to moderately-controlled diabetes (HbA1c approximately $\leq 7\text{--}8\%$) achieve survival rates and bone-loss profiles approaching those of non-diabetic patients, whereas poorly controlled diabetes (HbA1c $>8\%$) is associated with a measurably higher burden of marginal bone loss, peri-implant inflammation, and implant failure, with type 1 diabetes and the maxilla appearing particularly vulnerable. These findings support incorporating pre-surgical HbA1c assessment, individualised risk stratification, and where necessary staged surgical protocols and reinforced long-term maintenance into implant treatment planning for diabetic patients, while affirming that diabetes mellitus, when well controlled, does not preclude predictable implant therapy.

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