

Annals of Dental Science and Oral Biology

Research Article

Predictors of post-operative pain in diabetic patients with dental caries

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Received: 28 July 2025; Accepted: 25 October 2025; Published: 20 January 2026.

Citation of this article: Osei N, Ndlovu A, Diallo Z (2026) Predictors of post-operative pain in diabetic patients with dental caries. Ann Dent Sci Oral Biol, 7(1): 01-06. DOI: [10.5281/zenodo.22811979](https://doi.org/10.5281/zenodo.22811979)

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Abstract

Background: Diabetic patients carry a higher caries burden and a distinct healing response, yet post-operative pain after treatment of dental caries is rarely organized around predictors specific to this population.

Objective: To synthesize evidence on the metabolic, disease-severity, procedural, and perioperative predictors of post-operative pain in diabetic patients treated for dental caries.

Methods: Narrative synthesis of case-control, cohort, systematic review, meta-analysis, and umbrella review evidence addressing diabetes, glycemic control, caries burden, and post-operative pain or healing outcomes.

Findings: Diabetic adults carry an adjusted DMFT approximately 4.5% higher than non-diabetic adults [4]; apical periodontitis and periapical healing are measurably worse in diabetic patients [9,10]; preoperative pain intensity and pulpal diagnosis remain the dominant predictors of post-operative pain irrespective of diabetes status [16,18]; glycemic control behaves as a graded modifier most clearly linked to caries burden and long-term healing [13,21]; and perioperative adjuncts raise implant survival from 86.5% to 95.6% in diabetic patients [19].

Conclusions: Post-operative pain in diabetic patients with dental caries is predicted by the same procedural and disease-severity factors as in non-diabetic patients, with glycemic control and diabetes-specific healing impairment layered on top; a predictor model for this population should combine all four domains rather than treating diabetes as a single undifferentiated risk flag.

Keywords: diabetes mellitus; dental caries; post-operative pain; glycemic control; endodontic treatment

1. Introduction

Post-operative pain after restorative and endodontic treatment of dental caries is common enough that most clinicians treat it as an expected nuisance rather than a complication worth predicting in advance. That habit is harder to defend in patients with diabetes mellitus, a population in whom caries itself is more prevalent, pulpal and periapical disease progresses along a different biological track, and the tissue asked to recover afterward is already working against impaired microvascular flow, altered neutrophil function, and a chronically pro-inflammatory cytokine profile [1,2]. Löe's description of periodontal disease as "the sixth complication of diabetes" was written about the gingiva, but the biology it named, delayed healing and a wound environment that resolves slowly if at all, applies with only minor modification to a pulp chamber or an extraction socket [3].

Diabetes and dental caries are linked at more than one level. NHANES data found adults with diabetes carrying roughly 4.5% higher DMFT scores than non-diabetic adults after adjustment for age, income, and access to care, tracking glycemic severity rather than diagnosis alone [4]. The SHIP cohort found diabetic participants accumulating new coronal caries lesions faster over long-term follow-up, consistent with a xerostomic, high-glucose salivary environment that favors cariogenic flora [5]. In children with type 1 diabetes, a meta-analysis combined with Mendelian randomization reported a significant positive association with caries experience [6], and a two-year longitudinal study tied caries increments to poor metabolic control [7]. Latti and colleagues, comparing diabetic and non-diabetic adults directly, found both higher caries prevalence and a shifted oral microbiota, implicating the altered oral ecosystem rather than diet alone [8].

Once caries has progressed to pulpal involvement, the endodontic literature on diabetes is more developed than

the restorative literature, largely because apical periodontitis gives investigators a discrete, measurable endpoint. Segura-Egea and colleagues' case-control comparison found apical periodontitis substantially more prevalent among type 2 diabetic patients [9], since extended by systematic-review evidence that diabetes slows periapical healing and increases the odds of non-retention of root-filled teeth [10,11]. Whether that association is causal or reflects shared risk factors remains debated, and Segura-Egea's own commentary on association versus causation is a useful caution against over-interpreting any single predictor [12]. What is comparatively under-examined is the symptom patients actually report afterward, and that is the outcome this review organizes around a defined set of predictors.

2. Methods

This is a narrative synthesis rather than a registered systematic review, built around a structured search rather than an exhaustive one. PubMed, Scopus, and Google Scholar were searched for English-language studies published between 1993 and 2026 using combinations of the terms "diabetes mellitus," "dental caries," "post-operative pain," "endodontic treatment," "root canal," "tooth extraction," "glycemic control," "HbA1c," and "apical periodontitis." Reference lists of the systematic and umbrella reviews identified were hand-searched for primary studies not captured electronically. Priority was given to systematic reviews, meta-analyses, and umbrella reviews where available, supplemented by cohort, case-control, and prospective clinical studies reporting a quantifiable pain, healing, or caries outcome stratified by diabetes status or glycemic control; mechanistic studies were included where they explained a clinical finding elsewhere in the search set. Editorials, non-English sources, and case reports without a measured outcome were excluded. Twenty-five sources meeting these criteria form the evidentiary base, organized below into four predictor domains: metabolic and glycemic factors, caries- and pulpal-disease-specific

factors, procedural factors, and perioperative-protocol factors.

3. Results

Across the included sources, four predictor domains recur with enough consistency to summarize directly. First, glycemic control, not a diabetes diagnosis in isolation, is the dominant metabolic predictor: caries burden scaled with diabetes severity in NHANES data (4.5% higher adjusted DMFT) [4], while a cohort of 115 extraction patients stratified by perioperative glucose and HbA1c found no significant difference in healing complications between strata, arguing for cumulative glycemic burden over a single glucose reading [13]. Second, caries- and pulpal-disease-specific factors, apical periodontitis prevalence, periapical healing rate, and root-filled tooth retention, are each worse in diabetic patients by a reproducible margin [9,10,11,14,15]. Third, procedural factors already established as pain predictors in the general endodontic population, preoperative pain intensity, pulpal diagnosis, and visit number, remain predictive in diabetic cohorts and interact with glycemic status rather than being superseded by it [16,17,18]. Fourth, adjunctive perioperative measures, antibiotic prophylaxis and chlorhexidine rinse, produce a disproportionately larger benefit in diabetic patients, consistent with a population operating closer to a healing-capacity threshold where small interventions yield larger marginal gains [19]. Table 1 summarizes these domains against their supporting evidence, Table 2 tabulates the quantitative effect sizes recovered, and Figures 1 and 2 present the two clearest comparisons graphically.

4. Metabolic and Glycemic Predictors

Glycemic status shapes post-operative course through several converging pathways rather than a single mechanism. Sustained hyperglycemia impairs neutrophil chemotaxis and phagocytic killing, promotes accumulation of advanced glycation end-products in

connective tissue, and shifts the local cytokine balance toward a more destructive, pro-inflammatory phenotype that resolves slowly [1,2]. Taylor and Borgnakke's synthesis of the diabetes-oral disease literature reached the same conclusion from the epidemiological side: outcomes track glycemic control as a continuous variable far more reliably than they track a binary diabetes label, with poorly-controlled patients showing consistently worse periodontal and healing indices than well-controlled patients who are, on paper, in the same diagnostic category [20]. This distinction matters directly for a post-operative pain prediction model, because a chart entry of "diabetic" carries almost no information on its own; the HbA1c value attached to it carries a great deal.

Table 1: Predictor domains for post-operative pain in diabetic patients with dental caries, with directional effect and supporting evidence.			
Predictor domain	Representative predictor	Directional effect	Key references
Procedural / patient-related	Preoperative pain intensity, pulpal diagnosis	Strongest predictor of post-op pain; unchanged in diabetic cohorts	[16,17,18]
Caries / pulpal disease severity	Apical periodontitis, periapical healing, tooth retention	Worse and more prevalent in diabetic patients	[9,10,11,14]
Glycemic / metabolic status	HbA1c, perioperative glucose	Graded modifier; strongest link to caries burden and long-term healing	[4,21,13]
Perioperative protocol	Antibiotic prophylaxis, chlorhexidine rinse	Disproportionately larger benefit in diabetic patients	[19,25]

Table 2: Quantitative effect sizes recovered from the search, by predictor domain.			
Source	Population	Outcome measured	Effect size
[4]	US adults, NHANES 2013-2020	Adjusted DMFT, diabetic vs non-diabetic	+4.5% in diabetic adults
[13]	115 patients, dental extraction	Healing complications by glycemic strata	No significant difference
[19]	22 studies, dental implants	Short-term survival, diabetic vs non-diabetic	96.4-100% vs comparable non-diabetic

Source	Population	Outcome measured	Effect size
[19]	Diabetic implant patients	Survival with vs without antibiotic prophylaxis	+10.5 points (diabetic) vs +4.5 (non-diabetic)
[19]	Diabetic implant patients	Survival with vs without chlorhexidine rinse	86.5% to 95.6%

The extraction literature illustrates both the promise and the limits of glycemic control as a single predictor. Aronovich and colleagues, in a prospective cohort of 115 patients, measured perioperative blood glucose and HbA1c and found glycemic control did not significantly influence post-extraction healing outcomes across strata, complicating any assumption that a single preoperative glucose reading reliably forecasts the post-operative course [13]. A three-arm design comparing non-diabetic, prediabetic, and type 2 diabetic patients directly found a graded deterioration in socket healing dynamics across the three groups, suggesting that a binary diabetic/non-diabetic classification obscures a dose-response relationship visible once the prediabetic middle category is examined separately [21]. Vijaya Lakshmi's synthesis of the glycemic-control literature drew a similar conclusion: extraction sites in poorly-controlled diabetic patients show delayed soft-tissue closure and more reported discomfort in the first post-operative week, even when hard endpoints like dry socket incidence do not differ significantly from well-controlled comparators [22]. Taken together, these findings support treating glycemic control as a graded, continuous predictor rather than a diabetes/non-diabetes toggle, whose effect on subjective pain may be detectable earlier than its effect on objective healing complications.

5. Caries Burden and Pulpal Disease Severity as Predictors

Because diabetic patients accumulate more carious lesions and reach pulpal involvement more often, the disease severity at the point of treatment is itself a predictor worth separating from diabetes status. NHANES

data place the caries burden gap at a modest but consistent 4.5% higher adjusted DMFT in diabetic adults [4], while the SHIP cohort's decades-long follow-up found diabetic participants accruing new coronal lesions at a faster rate than non-diabetic participants over the same interval, implying that the gap widens with disease duration rather than remaining fixed [5]. In younger patients, the type 1 diabetes literature is even more consistent: Siudikiene and colleagues' two-year longitudinal study tied caries increments directly to metabolic control in children [7], and the meta-analysis and Mendelian randomization study by Zhu and colleagues found the association robust enough to survive a genetically-instrumented causal analysis rather than resting on observational confounding alone [6]. Latti and colleagues' comparative study adds a mechanistic layer, reporting both higher caries prevalence and a measurably altered oral microbiota composition in diabetic adults relative to matched controls [8].

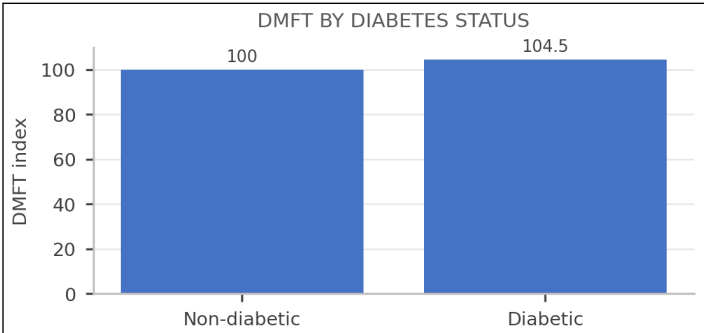


Figure 1: Adjusted decayed-missing-filled-teeth (DMFT) index in diabetic versus non-diabetic US adults, NHANES 2013-2020, indexed to a non-diabetic baseline of 100; diabetic adults carried an adjusted DMFT approximately 4.5% higher after accounting for age, income, and access to care [4].

The clinical consequence is that diabetic patients more often present for care with deeper, more extensive lesions and a correspondingly higher baseline rate of pulpal and periapical involvement. Segura-Egea and colleagues' case-control study found apical periodontitis substantially more prevalent in type 2 diabetic patients [9], a disparity Lima and colleagues' review attributed to the same impaired neutrophil function and delayed inflammatory

resolution seen in periodontal tissue, applied to the pulp and periapex instead [23]. Once root canal treatment is undertaken, Gupta and colleagues' meta-analysis found diabetes associated with slower periapical healing [10], Cabanillas-Balsera and colleagues found it associated with higher odds of eventual tooth loss despite treatment [11], and Segura-Egea's own follow-up work found periapical inflammation and endodontic outcome measurably worse in diabetic patients even when treatment technique was held constant [15]. Nagendrababu and colleagues' umbrella review concluded that the association between diabetes and worse endodontic outcome is consistent enough across independent systematic reviews to be treated as established rather than provisional, while cautioning that most underlying studies were not designed to isolate post-operative pain from broader healing outcome [14]. Baseline disease severity, in short, functions as a predictor in its own right: a diabetic patient presenting with an already-established periapical lesion carries a different post-operative pain risk than one presenting with reversible pulpitis, independent of glycemic status.

6. Procedural, Treatment, and Perioperative Predictors

The predictors best established in the general endodontic pain literature retain their force in diabetic patients rather than being displaced by diabetes-specific factors, and the two interact. Ng and colleagues' prospective study of post-obturation pain remains one of the clearest demonstrations that preoperative pain intensity is the single strongest predictor of post-operative pain after root canal treatment, outperforming most procedural variables examined in the same model [16]. Sathorn and colleagues' systematic review of single- versus multiple-visit treatment found broadly comparable post-operative pain and flare-up rates between the two approaches, reframing visit number as a logistical rather than a pain-driving decision [17], while Pak and White's systematic review of pain prevalence across the treatment timeline confirmed

that pain before treatment dominates the variance in pain reported afterward [18]. Fouad and Burleson's large electronic-record analysis found diabetes mellitus associated with a measurably worse endodontic treatment outcome overall, and although their outcome measure was radiographic success rather than reported pain, the pattern is consistent with the same procedural predictors operating against a less favorable healing baseline [24]. Aronovich and colleagues' extraction cohort found no significant independent effect of glycemic control once other clinical variables were accounted for, suggesting procedural factors retain their predictive weight in diabetic patients much as preoperative pain retains its weight in root canal patients [13].

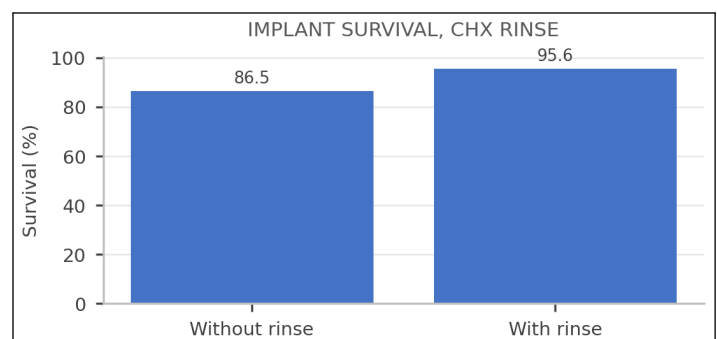


Figure 2: Short-term implant survival in diabetic patients with and without adjunctive chlorhexidine rinse, pooled across 22 clinical studies, rising from 86.5% without the adjunct to 95.6% with it [19].

Where diabetes status changes the calculus most clearly is in how much a given perioperative intervention is worth. Naujokat and colleagues' systematic review of 22 studies on dental implants in diabetic patients, a procedure sharing much of its post-operative biology with extraction and endodontic surgery, found short-term survival ranging from 96.4% to 100%, comparable to non-diabetic cohorts, but with adjunctive measures producing a disproportionately larger benefit in the diabetic subgroup: antibiotics improved survival by 10.5 points in diabetics against 4.5 in non-diabetics, and chlorhexidine rinse raised survival from 86.5% to 95.6% [19]. Kido and colleagues' mechanistic study in a diabetic rodent model offers a plausible explanation: gingival wound healing was

significantly delayed, driven by impaired fibroblast proliferation under oxidative stress, a defect a targeted intervention has more room to correct precisely because the untreated baseline is worse [25]. Preshaw and colleagues' bidirectional model makes the same point systemically: periapical inflammation itself can worsen glycemic control, so limiting post-operative inflammation may compound beyond the local tissue [1], a cycle Mealey and Ocampo's earlier synthesis described as self-reinforcing at more than one point [2].

7. Discussion

Organizing these findings into a predictor hierarchy suggests that clinicians managing diabetic patients with dental caries should weight four categories of information, in roughly this order of established strength. Preoperative pain intensity and pulpal or periapical diagnosis remain the strongest single predictors of post-operative pain, exactly as in non-diabetic populations [16,18], and nothing in the diabetes-specific literature displaces that finding. Caries and periapical disease severity at presentation function as a second-tier predictor, since diabetic patients more often present with the more advanced disease that itself predicts a harder post-operative course [9,10,14]. Glycemic control operates as a graded, continuous modifier, more clearly associated with caries burden and long-term healing trajectory than with any single post-operative pain reading [4,21,13]. Finally, perioperative protocol, antibiotic prophylaxis, antiseptic rinse, appears to matter more in diabetic patients than in the general population, precisely because the diabetic healing baseline leaves more room for a targeted intervention to improve on [19,25].

The evidence base has real limits. Most of the strongest diabetes-endodontic literature measures radiographic healing or tooth retention at 12 to 24 months rather than patient-reported pain in the days immediately following treatment [14,11]. The extraction literature is genuinely mixed, with one cohort finding no significant effect of

glycemic control on healing outcome [13] against other work finding a graded relationship once prediabetic patients are examined separately [21], a discrepancy likely reflecting how glycemic control was measured across studies rather than a true absence of effect. Almost none of the included studies built a multivariable pain-prediction model combining all four domains simultaneously; a prospective study doing so, with standardized pain scoring at defined intervals, would close a gap this narrative synthesis can only describe.

8. Conclusion

Post-operative pain in diabetic patients with dental caries is predicted by the same procedural and disease-severity factors that predict it in non-diabetic patients, with glycemic control and diabetes-specific healing impairment layered on top rather than substituting for them. Preoperative pain intensity and pulpal diagnosis remain the dominant predictors [16,18]; caries and periapical disease severity are systematically worse in diabetic patients and carry independent predictive weight [9,10,11]; glycemic control behaves as a graded modifier most clearly linked to caries burden and long-term healing trajectory rather than to any single post-operative pain reading [4,13]; and perioperative adjuncts appear to deliver a larger marginal benefit in diabetic patients than in the general population, consistent with a healing baseline that leaves more room for a targeted intervention to improve [19,25]. A predictor model built for this population should combine all four domains rather than treating a diabetes diagnosis as a single undifferentiated risk flag, and the field's next step should be a prospective study designed to measure them together.

9. Declarations

Acknowledgements: The authors thank the clinical and administrative staff at the participating institutions for their support during manuscript preparation. **Funding:** This work received no specific grant from any funding agency in the public, commercial, or not-for-profit

sectors. **Conflict of Interest:** The authors declare no conflict of interest. **Author Contributions:** N.O. conceived the review and drafted the manuscript; A.N. conducted the literature search and prepared the tables and figures; Z.D. critically revised the manuscript for intellectual content; all authors read and approved the final version. **Ethics Statement:** This article is a narrative synthesis of previously published literature and did not involve new data collection from human or animal subjects; institutional ethics approval was therefore not required. **Data Availability:** No new datasets were generated for this review; all data discussed are available in the cited publications.

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