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Periodontal Disease as a Predictor of Undiagnosed Diabetes or Prediabetes in Dental Patients: A Comprehensive Review

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ABSTRACT

Periodontal disease and diabetes mellitus represent two of the most prevalent chronic diseases affecting the global population, with emerging evidence demonstrating a bidirectional relationship between these conditions. Recent studies have identified periodontal disease as a potential clinical marker for undiagnosed diabetes and prediabetes in dental patients. The presence of severe periodontal parameters, particularly clinical attachment loss greater than 3 mm, has demonstrated significant predictive value for identifying subjects with dysglycemia who have not yet been formally diagnosed with diabetes. This comprehensive review synthesizes current scientific evidence regarding the utility of periodontal examination findings in screening for undiagnosed diabetes and prediabetes in dental populations, examines the underlying biological mechanisms linking these conditions, and evaluates the potential for integrating diabetes screening protocols within dental clinical practice. Systematic review of peer-reviewed literature indicates that patients presenting with advanced periodontal disease exhibit a two to three-fold increased risk for undiagnosed diabetes or prediabetes. Logistic regression models incorporating periodontal parameters alongside traditional risk factors demonstrate predictive accuracy ranging from 69.4% to 73%, with clinical attachment loss emerging as the most significant periodontal predictor. Furthermore, treatment of periodontitis improves glycemic control in patients with established diabetes, reducing glycated hemoglobin (HbA1c) levels by 0.3–0.6%. This review underscores the importance of interprofessional collaboration between dental and medical practitioners, advocates for the integration of chairside diabetes screening tools in dental settings, and discusses the clinical implications of identifying undiagnosed dysglycemia through periodontal assessment.

Keywords: *periodontal disease; periodontitis; undiagnosed diabetes; prediabetes; clinical attachment loss; diabetes screening*

INTRODUCTION

Diabetes mellitus represents one of the most significant public health challenges of the twenty-first century, with the International Diabetes Federation reporting more than 537 million adults living with diabetes worldwide. Alarmingly, approximately 44% of individuals with diabetes remain undiagnosed, creating a substantial disease burden in terms of preventable complications and increased mortality risk (Saeedi et al., 2019). Concurrently, periodontitis affects nearly 50% of the global population to varying degrees of

severity, making it one of the leading causes of tooth loss in adults and a significant contributor to oral morbidity (Tonetti et al., 2017).

Substantial epidemiological and clinical evidence has established a bidirectional association between periodontal disease and diabetes mellitus (Demmer et al., 2021). Individuals with poorly controlled diabetes demonstrate a two to three-fold increased risk of developing moderate to severe periodontitis compared with non-diabetic individuals (Sanz et al., 2018). Conversely, emerging evidence indicates that periodontal inflammation may compromise glycemic control and increase the risk of incident diabetes in previously non-diabetic individuals (Demmer et al., 2012). This bidirectional relationship has prompted considerable research interest into whether periodontal findings might serve as clinical indicators of systemic dysglycemia.

The dental profession encounters large populations of individuals who may not have regular contact with medical practitioners. Studies demonstrate that 30–50% of dental patients with severe periodontitis exhibit abnormal glucose regulation when screened chairside (Lalla & Papapanou, 2011). Furthermore, approximately 1 in 18 dental patients screened with chairside HbA1c testing are detected with previously undiagnosed diabetes (Lalla et al., 2003). These findings suggest that dental practices represent an underutilized but potentially powerful frontline screening point for identifying individuals at risk of undiagnosed dysglycemia.

The global prevalence of undiagnosed diabetes is substantial, particularly in resource-limited settings and populations with limited access to healthcare services. In developed nations, the proportion of undiagnosed diabetes ranges from 15% to 40%, while in developing countries this proportion may exceed 50% (Saeedi et al., 2019). Prediabetes, defined as fasting plasma glucose between 100–125 mg/dL or HbA1c between 5.7%–6.4%, affects approximately 374 million adults globally, yet awareness rates remain below 20% (American Diabetes Association [ADA], 2024)).

A foundational study investigating the utility of periodontal parameters in identifying undiagnosed dysglycemia enrolled 61 dental patients aged 35 years and older, all of whom possessed at least one risk factor for diabetes but had no prior diagnosis of diabetes mellitus (9). Of the cohort, 64.5% reported a family history of diabetes, and 59% were diagnosed with advanced periodontal disease. Using multiple logistic regression analysis with backward elimination methodology, the investigators developed a predictive model incorporating smoking status, hypertension, family history of diabetes mellitus, and percentage of clinical attachment loss exceeding 3 mm as significant predictors of dysglycemic status. The final model demonstrated statistical significance ($P < 0.001$) and correctly classified 69.4% of cases. Notably, participants with a family history of diabetes demonstrated a 4.98-fold increased likelihood of exhibiting prediabetic or diabetic status. Critically, each unit increase in the percentage of clinical attachment loss >3 mm increased the likelihood of the participant presenting with prediabetes or diabetes by 1.104 times (Heji et al., 2020).

LITERATURE REVIEW

Clinical attachment loss, defined as the distance from the cemento-enamel junction to the base of the periodontal pocket, has emerged as the most sensitive periodontal parameter for predicting undiagnosed dysglycemia (9). This measurement captures the cumulative effect of periodontal destruction over time and integrates both bone loss and soft tissue destruction. Probing pocket depth, while useful for assessing current periodontal inflammation, may not reflect the historical burden of periodontal disease as comprehensively as clinical attachment loss.

Advanced bone loss, visualized on radiographic examination, has also demonstrated an association with elevated fasting blood glucose levels in dental patients (Holm-Pedersen et al., 2008). Dental patients presenting with generalized advanced bone loss and clinical attachment loss ≥3 mm in 30% or more of teeth

exhibited significantly higher mean fasting blood glucose concentrations compared with patients with mild bone loss, even after accounting for other metabolic risk factors (Heji et al., 2020).

Mechanisms Linking Periodontal Disease and Dysglycemia

The hallmark mechanism linking periodontal disease to dysglycemia involves chronic systemic inflammation. Periodontitis is characterized by a dysbiotic shift in the subgingival microbiota, triggering a robust host immune response that extends beyond the oral cavity. The ulcerated epithelium of the periodontal pocket presents a chronic source of systemic microbial challenge and bacterial products (Preshaw et al., 2012). The high vascularity of inflamed periodontal tissue, coupled with the magnitude of inflamed tissue surface area in advanced periodontitis, contributes to substantial systemic levels of inflammatory mediators.

Inflammatory Cytokine Production and Glycemic Control

Multiple cytokines have been identified as pathogenic mediators linking periodontal inflammation to impaired glycemic control. Tumor necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), and interleukin-1 beta (IL-1β) represent the primary pro-inflammatory cytokines elevated in periodontitis and associated with dysglycemia. These mediators are produced at elevated levels by macrophages, monocytes, and activated lymphocytes at periodontal sites (Preshaw et al., 2012; Chapple & Genco, 2013).

A comparative study of cytokine production in periodontal tissues examined four study groups: systemically healthy subjects without periodontal disease, systemically healthy patients with chronic periodontitis, well-controlled type 2 diabetic patients with chronic periodontitis, and poorly controlled type 2 diabetic patients with chronic periodontitis (13). Results demonstrated that patients with poorly controlled diabetes and concurrent periodontitis exhibited significantly elevated levels of IL-4, IL-10, IL-17, and interferon-gamma (IFN-γ) compared with other groups ($P < 0.05$). The elevations in IL-17 and IFN-γ were particularly pronounced, supporting the hypothesis that glycemic control influences the immune inflammatory response at periodontal sites. IL-17, produced by T-helper 17 (Th17) lymphocytes, has been implicated in osteoclast activation and bone resorption in periodontitis, and its elevated expression in poorly controlled diabetic patients may explain the accelerated bone loss observed in this population (Duarte et al., 2015).

Advanced Glycation End Products and Receptor Binding

Hyperglycemia promotes the formation of advanced glycation end-products (AGEs) through non-enzymatic glycation of proteins and lipids. These AGEs accumulate in periodontal tissues and circulating blood, where they bind to their cell surface receptors (RAGEs) on macrophages, endothelial cells, and fibroblasts (Brownlee, 2001). The AGE-RAGE interaction triggers a cascade of pro-inflammatory signaling, including activation of nuclear factor-kappa B (NF-κB), which amplifies the production of TNF-α, IL-6, and other inflammatory mediators (Brownlee, 2001). This AGE-RAGE pathway has been implicated in increased osteoclastic activity, leading to accelerated alveolar bone loss in diabetic patients with periodontitis.

Furthermore, inflamed periodontal tissues and certain oral bacteria have been identified as potential endogenous sources of AGEs, creating a bidirectional amplification loop where periodontal inflammation promotes AGE formation, and AGEs, in turn, perpetuate inflammation and exacerbate periodontal destruction (Genco & Borgnakke, 2022).

Oxidative Stress and Tissue Destruction

Both periodontitis and type 2 diabetes mellitus are characterized by excessive production of reactive oxygen species (ROS) and oxidative stress. Hyperglycemia promotes ROS generation through multiple pathways, including mitochondrial electron transport chain dysfunction and activation of NADPH oxidase enzymes.

In periodontal tissues, periodontal pathogens and activated immune cells generate ROS in abundance (D'Aiuto et al., 2013).

Oxidative stress impairs the function of periodontal tissue-constructing cells, including gingival fibroblasts and endothelial cells. Hyperglycemic conditions reduce the viability of periodontal ligament fibroblasts and enhance their expression of pro-inflammatory cytokines, particularly in response to lipopolysaccharide stimulation from gram-negative periodontal pathogens (Zhao et al., 2023). ROS also activates matrix metalloproteinases (MMPs), collagenolytic enzymes responsible for degradation of the extracellular matrix in periodontal tissues (*Pathogenic mechanisms that may link periodontal disease and type 2 diabetes mellitus*, 2024).

Dysbiotic Microbiota and Metabolic Endotoxemia

The periodontal microbiota undergoes substantial dysbiotic changes in the context of dysglycemia. Hyperglycemia and impaired immune function select for pathogenic bacteria, including Porphyromonas gingivalis, Tannerella forsythia, and Treponema denticola, which are gram-negative organisms producing substantial quantities of lipopolysaccharide (LPS) (Kumar et al., 2003). This LPS translocation across the ulcerated epithelium of the periodontal pocket contributes to systemic lipopolysaccharide levels and metabolic endotoxemia.

Bacterial species cultured from periodontal sites have been demonstrated to stimulate insulin secretion via toll-like receptor 4 (TLR4) and phosphoinositide 3-kinase/protein kinase B (PI3K/AKT) signaling pathways, potentially promoting compensatory pancreatic beta-cell responses and contributing to insulin resistance over time (*Bacterial supernatants elevate glucose-dependent insulin secretion*, 2018).

Insulin Resistance and Impaired Glucose Metabolism

Chronic periodontal inflammation impairs insulin signaling through multiple mechanisms. TNF-α and IL-6, the elevated levels of which characterize both periodontitis and diabetes, are known suppressors of insulin receptor signaling in skeletal muscle and adipose tissue. These cytokines interfere with insulin receptor substrate phosphorylation, impairing the insulin signaling cascade and promoting insulin resistance (Hotamisligil, 2006).

Additionally, emerging evidence indicates that impaired insulin action on periodontal tissue-constructing cells, particularly gingival fibroblasts and endothelial cells, contributes to the pathogenesis of diabetes-related periodontitis (*The bidirectional association between diabetes and periodontitis*, 2023). Insulin resistance therefore, represents a bidirectional link, whereby periodontal inflammation promotes systemic insulin resistance, which in turn impairs periodontal tissue homeostasis.

Clinical Utility of Periodontal Parameters in Diabetes Screening

The diagnostic utility of periodontal parameters in identifying undiagnosed dysglycemia has been systematically evaluated in multiple clinical cohorts. In a Danish population study, periodontal disease status demonstrated a sensitivity of 0.91 for identifying diabetes and prediabetes, although specificity was lower at 0.19 (Holm-Pedersen et al., 2008). The lower specificity reflects the multifactorial nature of periodontitis, which cannot be attributed exclusively to dysglycemia.

Strauss and colleagues, analyzing data from the National Health and Nutrition Examination Survey (NHANES-3), developed a model incorporating periodontal pocket depth and clinical attachment loss measurements to predict probabilities of undiagnosed diabetes (Strauss et al., 2015). Their model predicted probabilities of undiagnosed diabetes ranging from 27% to 53%, demonstrating the substantial predictive capacity of periodontal measurements in a nationally representative sample.

Acharya and colleagues presented a comprehensive prediction model that incorporated demographic data, medical history, and detailed dental examination findings (Acharya et al., 2017). Their model identified 70% of dental patients with undiagnosed diabetes, though the investigators acknowledged that access to comprehensive medical records might not be feasible in many dental practice settings.

Clinical Attachment Loss as the Optimal Screening Parameter

Among periodontal variables assessed in multivariable prediction models, clinical attachment loss has emerged as the most powerful and consistent predictor of undiagnosed dysglycemia. The fundamental study on this topic, conducted at Umm Al-Qura University, included multiple periodontally-relevant variables in their logistic regression analysis: age, gender, nationality, family history of diabetes, gestational diabetes, smoking status, hyperlipidemia, hypertension, body mass index, number of missing teeth, percentage of pocket depth exceeding 5 mm, percentage of clinical attachment loss exceeding 3 mm, and presence of advanced bone loss (Heji et al., 2020).

The final parsimonious model retained only four variables as statistically significant independent predictors: smoking status, hypertension, family history of diabetes mellitus, and percentage of clinical attachment loss exceeding 3 mm. This model correctly classified 69.4% of subjects into correct dysglycemic categories and explained 47.7% of the variance in diabetes status (Heji et al., 2020).

Family history of diabetes emerged as a critical risk factor in the predictive models. Subjects reporting a family member with diabetes were 4.98 times more likely to present with undiagnosed prediabetes or diabetes (Heji et al., 2020). This finding underscores the importance of comprehensive medical history assessment during initial dental consultation, as familial clustering of diabetes reflects shared genetic susceptibility and lifestyle factors.

Chairside HbA1c Testing as an Adjunctive Screening Tool

The evidence base strongly supports the integration of chairside HbA1c testing within dental practice settings as a complementary screening modality. The landmark study by Lalla and colleagues demonstrated that dental-based HbA1c screening identified 30% of previously undiagnosed diabetic or prediabetic individuals among high-risk dental patients (Lalla et al., 2003). These findings have been replicated in multiple dental populations, with consistency suggesting that approximately 30–50% of dental patients with severe periodontitis demonstrate abnormal glucose regulation when screened with chairside tools.

A more recent study employing chairside HbA1c testing alongside body mass index and waist circumference measurements in patients with known clinical attachment loss consistent with periodontitis found that patients with elevated HbA1c (≥5.7%) were significantly more likely to exhibit clinical attachment loss exceeding 5 mm than those with lower HbA1c levels (<5.7%) (Mataftsi et al., 2021). This reciprocal association strengthens the rationale for bidirectional screening.

Periodontal Disease as a Bidirectional Predictor of Metabolic Status

While cross-sectional studies have consistently demonstrated associations between periodontal disease and prevalent dysglycemia, prospective cohort studies have established temporal precedence, supporting a causal role for periodontitis in the development of incident diabetes. Analysis of National Health and Nutrition Examination Survey (NHANES I) data incorporating 9,296 nondiabetic participants followed over two decades demonstrated that baseline periodontal disease independently predicted incident type 2 diabetes over extended follow-up (*Periodontal disease and incident type 2 diabetes*, 2008).

A national cohort study from the Continuous National Health and Nutrition Examination Survey (2009-2010) enrolled 1,165 participants and investigated the relationship between periodontal disease, impaired fasting glucose, and impaired glucose tolerance (Islam et al., 2014). Periodontal disease burden at or

exceeding the 75th percentile was associated with a 105% increase in the odds of impaired glucose tolerance, with an odds ratio of 2.05 (95% confidence interval 1.24–3.39, P = 0.005). This prospective association persisted after adjustment for confounding variables, including age, sex, body mass index, smoking status, and hypertension, supporting an independent contribution of periodontal disease to glucose dysregulation (Islam et al., 2014).

RESULTS AND DISCUSSION

A comprehensive systematic review and meta-analysis examining the bidirectional prospective association between periodontal disease and diabetes mellitus by Demmer and colleagues synthesized evidence from observational cohort studies while accounting for risk of bias in the original investigations (Demmer et al., 2021). The analysis included studies investigating both the effect of baseline periodontal disease on incident diabetes and the effect of baseline diabetes on incident periodontitis. Results demonstrated a significant positive bidirectional association between periodontal disease and diabetes mellitus, providing level-1 evidence supporting the need for mutual screening—i.e., screening for diabetes in patients with periodontitis and vice versa (Demmer et al., 2021).

The therapeutic efficacy of periodontal treatment in improving glycemic control represents compelling evidence supporting a causal relationship between these conditions. Multiple randomized controlled trials and systematic reviews have documented that non-surgical periodontal therapy (scaling and root planing) reduces HbA1c levels by approximately 0.3–0.6% in patients with established type 2 diabetes and concurrent periodontitis (D'Aiuto et al., 2011; Teeuw et al., 2014).

A study evaluating the effect of non-surgical periodontal therapy incorporating topical antibiotics on glycemic control in patients with type 2 diabetes and periodontitis assessed inflammatory mediators, including TNF-α, IL-6, and C-reactive protein, before and after periodontal treatment (*Improvement of glycemic control after periodontal treatment*, 2012). The investigators found significant reductions in systemic inflammatory markers alongside improvements in glycemic control, supporting the hypothesis that resolution of periodontal inflammation mechanistically improves metabolic homeostasis.

Another investigation of 40 type 2 diabetic patients with stage III periodontitis who underwent non-surgical periodontal therapy demonstrated significant improvements in periodontal parameters and systemic inflammatory markers, specifically reductions in TNF-α and high-sensitivity C-reactive protein levels and increases in anti-inflammatory IL-10 levels (*Effect of nonsurgical periodontal therapy on metabolic control*, 2022). Blood glucose levels and HbA1c levels both improved following periodontal treatment, with changes reaching statistical significance. These findings suggest that periodontal treatment reduces the systemic inflammatory burden, enabling improved insulin action and glycemic control (*Effect of nonsurgical periodontal therapy on metabolic control*, 2022).

Feasibility and Acceptability of Dental-Based Screening

Evidence indicates that both patients and dental professionals view the integration of diabetes screening into routine dental practice as feasible and acceptable (32). Dental patients, particularly those with limited medical care access, express willingness to complete diabetes screening assessments in the dental setting. From the provider perspective, dental professionals acknowledge their role in oral-systemic health and recognize the value of incorporating simple screening protocols that do not substantially prolong clinical visits.

Recommended Screening Algorithm in Dental Practice

Current evidence and consensus guidelines support the following integrated approach to diabetes screening in dental patients (Lalla et al., 2003; Goh et al., 2021):

1. **Comprehensive periodontal assessment**: Complete periodontal probing should be conducted on all patients, with particular attention to clinical attachment loss and probing pocket depths. Radiographic assessment to evaluate alveolar bone status should be obtained as clinically indicated.

2. **Risk factor assessment**: Detailed medical and family histories should specifically query family history of diabetes, presence of hypertension, hyperlipidemia, and overweight/obesity status (BMI ≥25).

3. **Risk stratification**: Patients presenting with advanced periodontal disease (clinical attachment loss ≥5 mm), particularly those with additional risk factors including family history of diabetes, hypertension, or obesity, should be considered candidates for chairside diabetes screening.

4. **Chairside HbA1c testing**: For high-risk patients, chairside HbA1c measurement using point-of-care devices provides rapid determination of glycemic control status. HbA1c ≥5.7% warrants medical referral for confirmatory testing and diabetes risk assessment.

5. **Referral pathway**: Dental practices should establish clear referral protocols with primary care physicians or endocrinologists for patients identified with elevated HbA1c or other diabetes risk indicators.

Barriers to Implementation and Solutions

Despite compelling scientific evidence, several barriers to widespread implementation of diabetes screening in dental settings persist. Limited reimbursement for chairside testing, time constraints, and concerns about scope of practice remain significant challenges. However, these barriers can be partially addressed through:

- **Collaborative pathways**: Formalizing written agreements between dental and medical providers clarifying roles and responsibilities
- **Electronic health record integration**: Enabling electronic communication of screening results between dental and medical providers
- **Professional education**: Providing continuing education to dental professionals regarding the bidirectional periodontal-metabolic relationship
- **Evidence dissemination**: Engaging professional organizations to endorse dental-based screening through position statements and guidelines

Several periodontal risk assessment models exist, including the hexagonal Periodontal Risk Assessment, modified PRA, UniFe/PerioRisk, SmartRisk, DentoRisk, and the Periodontal Risk Calculator. These tools systematically integrate multiple risk factors including periodontal parameters, smoking, age, and systemic conditions. Integration of diabetes screening into these existing risk assessment frameworks represents an efficient means of enhancing their clinical utility (Carra et al., 2018).

Salivary Biomarkers as Non-Invasive Screening Tools

Recent research has explored the potential utility of salivary biomarkers as non-invasive alternatives to blood-based testing for diabetes screening. Salivary glucose levels have demonstrated comparability to blood glucose in diagnosing and monitoring type 2 diabetes, with the distinct advantage of non-invasiveness (*Salivary glucose as a predictor of blood glucose*, 2019). Similarly, investigations of salivary glycated hemoglobin have shown promise, though standardization of analytical processes remains necessary for widespread clinical application.

Inflammatory biomarkers in saliva, including C-reactive protein, IL-6, TNF-α, and 1,5-anhydroglucitol, show promise for early diagnosis and risk prediction of diabetes, demonstrating consistent positive results in dysglycemic patients (*Exploring salivary biomarkers and health parameters in type 2 diabetes mellitus*, 2024). These markers reflect both local oral inflammation and systemic inflammatory status, potentially providing integrated assessment of periodontal-metabolic health. However, further research is needed to standardize analytical processes and establish clinical cutoff values. AGEs in saliva and gingival crevicular fluid represent emerging biomarkers of glycemic control and periodontal-systemic interactions. The accumulation of AGEs in these oral fluids correlates with systemic AGE levels and may provide non-invasive assessment of hyperglycemic burden without venipuncture (*Are inflamed periodontal tissues endogenous sources of advanced glycation end-products*, 2022).

Risk Prediction Models and Artificial Intelligence

Emerging applications of machine learning and artificial intelligence show substantial promise in enhancing the predictive accuracy of diabetes screening algorithms. Studies utilizing machine learning models to analyze combined periodontal parameters, demographic data, and laboratory findings have achieved accuracy levels approaching or exceeding 90% in identifying undiagnosed dysglycemia (*Artificial intelligence in periodontal disease prediction*, 2025).

Future diabetes screening algorithms will likely integrate multiple data modalities, including clinical periodontal parameters, radiographic findings, salivary biomarkers, and oral microbiome data, analyzed through machine learning algorithms. A study applying machine learning to oral microbiome data successfully categorized diabetic patients into "low-risk cardiovascular disease" and "high-risk cardiovascular disease" groups, suggesting that microbial dysbiosis data can enhance risk stratification (*Integration of oral microbiome data with machine learning*, 2024).

The challenges associated with machine learning implementation in clinical dentistry include lack of standardization of diagnostic criteria, limited dataset size in individual practices, and the need for external validation of models developed in specific populations. Multi-center collaborative research efforts are needed to develop robust, universally applicable prediction models.

Clinical and Public Health Implications

The substantial burden of undiagnosed diabetes—estimated to affect nearly half of all diabetic individuals globally—translates directly into delayed diagnosis and late-stage complications. Early identification of dysglycemia through dental screening offers the potential to advance diagnosis by months to years, enabling timely medical intervention and primary prevention of diabetes complications.

In patients with already-diagnosed diabetes, severe periodontitis is associated with increased risk of vascular complications, including coronary heart disease and cerebrovascular events, retinopathy, renal disease, and overall mortality (*Severe periodontitis and macrovascular complications in diabetes*, 2020). By improving periodontal health through treatment and facilitating glycemic control improvement, dental-medical collaboration may reduce the incidence and severity of these catastrophic complications.

Health economic analyses support the cost-effectiveness of dental-based diabetes screening, particularly in high-risk populations. The relatively low cost of periodontal assessment and chairside HbA1c testing, compared with the substantial costs associated with diabetes complications, creates a compelling economic case for integrating screening into dental practice. Dental screening represents a particularly valuable strategy for identifying undiagnosed diabetes in populations with limited access to primary care, including rural populations, certain ethnic minorities, and individuals with low socioeconomic status. Dental practitioners often see these populations more regularly than primary care physicians, positioning dental care as a potential equalizer in diabetes detection.

Future Directions and Research Gaps

While cross-sectional and retrospective studies provide consistent evidence that periodontal parameters predict dysglycemia status, randomized controlled trials evaluating the impact of systematic dental-based screening on clinical outcomes—including diagnosis rates, time to diagnosis, glycemic control, and complications—remain limited. Such pragmatic trials are essential to establish the effectiveness of screening implementation at the population level.

Although the inflammatory and glycosylation pathways linking periodontal disease to dysglycemia have been characterized at the molecular level, additional research is needed to elucidate the relative contributions of various mechanisms and to identify intervention targets beyond conventional periodontal therapy. Investigation of the role of insulin resistance specifically at the level of periodontal tissue-constructing cells represents a promising direction. The development of improved prediction algorithms incorporating newly identified biomarkers, genetic factors, and oral microbiome composition would enhance screening precision and facilitate risk stratification. Integration of multi-omics approaches—combining genomics, transcriptomics, proteomics, and metabolomics data—may reveal novel predictive relationships.

Systematic investigation of barriers and facilitators to implementation of dental-based screening in diverse practice settings, including solo practices, group practices, and community health centers, would facilitate widespread adoption. Research on training methods, practice-level interventions, and referral pathway optimization is needed. Additional research examining the magnitude and durability of glycemic improvements following periodontal treatment, the optimal timing and frequency of periodontal re-evaluation in diabetic patients, and the potential additive benefits of combination medical-dental intervention strategies would strengthen clinical guidance.

CONCLUSION

Substantial and growing scientific evidence demonstrates that periodontal disease can serve as a clinically useful predictor of undiagnosed diabetes and prediabetes in dental patient populations. The presence of advanced clinical attachment loss and alveolar bone loss identifies dental patients at substantially elevated risk for dysglycemia. Logistic regression models incorporating periodontal parameters alongside traditional risk factors achieve predictive accuracy of 69–73%, approaching or equaling that of conventional screening approaches such as fasting glucose testing. The mechanisms linking periodontal disease to dysglycemia involve multiple pathways, with chronic inflammation emerging as the central pathogenic pathway. Elevated pro-inflammatory cytokines, including TNF-α, IL-6, and IL-1β, generated at periodontal sites and entering systemic circulation, impair insulin signaling and promote insulin resistance. Advanced glycation end-products accumulating in periodontal tissues and blood trigger additional pro-inflammatory signaling cascades. Oxidative stress, dysbiosis, and dysregulated immune function create a milieu favoring both periodontal destruction and metabolic dysfunction.

The therapeutic efficacy of periodontal treatment in improving glycemic control in diabetic patients provides compelling evidence supporting a causal relationship between these conditions. Dental patients presenting with severe periodontitis, particularly those with additional risk factors such as family history of diabetes, hypertension, or overweight/obesity, should be considered candidates for chairside diabetes screening using tools such as HbA1c measurement. Widespread implementation of systematic dental-based diabetes screening has the potential to identify undiagnosed dysglycemia in millions of individuals globally, enabling early medical intervention and prevention of serious complications. Integration of diabetes screening into dental practice requires collaborative relationships with medical practitioners, clear referral pathways, and ongoing professional education. Future research should employ rigorous pragmatic trial designs to evaluate the effectiveness of dental-based screening at the population level, explore emerging

biomarkers and machine learning approaches to enhance predictive accuracy, and investigate implementation strategies that overcome practice-level and systemic barriers to screening adoption.

The convergence of epidemiological evidence, mechanistic understanding, and demonstration of therapeutic efficacy compels a paradigm shift toward integration of diabetes risk assessment into routine periodontal evaluation. Such integration represents a significant opportunity for dental professionals to contribute meaningfully to the prevention and early detection of a major public health threat—undiagnosed diabetes mellitus—while simultaneously advancing the care of their patients' oral and systemic health.

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