REVIEW ARTICLE

Radiographic Patterns of Periodontal Bone Loss in Diabetic versus Non-Diabetic Patients: A Narrative Review

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ABSTRACT

Periodontal disease is a chronic inflammatory condition characterized by the progressive destruction of the supporting structures of the teeth, ultimately resulting in alveolar bone loss. Diabetes mellitus, a globally prevalent metabolic disorder, is well recognized as a major systemic risk factor that influences both the initiation and progression of periodontitis. Radiographic imaging plays a pivotal role in the diagnosis, monitoring, and evaluation of periodontal bone loss, providing objective evidence of disease severity and distribution. This narrative review aims to comprehensively evaluate and compare radiographic patterns of periodontal bone loss in diabetic and non-diabetic patients. A structured literature search was conducted across major databases(Medline/PubMed, Embase, Scopus) from 2000 to 2025, focusing on studies that utilized conventional and advanced imaging modalities such as bitewing radiographs, periapical radiographs, panoramic radiographs, and cone-beam computed tomography (CBCT). Evidence consistently demonstrates that diabetic patients, particularly those with poor glycemic control, exhibit greater severity and extent of alveolar bone loss than non-diabetic individuals. Differences in bone loss patterns, including generalized horizontal bone loss and increased prevalence of vertical defects, have been reported. The underlying mechanisms involve hyperglycemia-induced inflammatory dysregulation, advanced glycation end-product accumulation, impaired bone remodeling, and altered host immune responses. Despite substantial evidence linking diabetes with increased radiographic periodontal destruction, heterogeneity in study design, imaging protocols, and confounding variables limits definitive conclusions. This review highlights existing evidence, discusses biological and radiographic correlations, and identifies gaps in current research that warrant further investigation.

KEYWORDS: Periodontal bone loss; Diabetes mellitus; Radiographic patterns; Alveolar bone; Panoramic radiography; Bitewing radiography; Cone-beam computed tomography.

INTRODUCTION

Periodontal disease represents one of the most common chronic inflammatory diseases affecting humans and is a major cause of tooth loss worldwide. It is characterized by gingival inflammation, periodontal pocket formation, connective tissue attachment loss, and resorption of alveolar bone. While dental plaque biofilm is the primary etiological factor, the severity and progression of periodontal destruction are profoundly influenced by systemic conditions, behavioral factors, and host immune responses. Among systemic conditions, diabetes mellitus has been consistently identified as one of the strongest and most biologically plausible risk factors for periodontal disease progression [1,2].

Diabetes mellitus is a chronic metabolic disorder characterized by persistent hyperglycemia resulting from defects in insulin secretion, insulin action, or both. The global prevalence of diabetes continues to rise, with type 2 diabetes mellitus accounting for the majority of cases. A well-established bidirectional relationship exists between diabetes and periodontal disease, wherein diabetes increases the risk and severity of periodontitis, and periodontal inflammation negatively affects glycemic control [3]. This relationship has significant implications for oral health assessment, diagnosis, and long-term management.

Radiographic imaging constitutes an indispensable component of periodontal diagnosis. Clinical parameters such as probing depth and clinical attachment loss provide information about soft tissue changes but do not directly visualize alveolar bone. Radiographs allow clinicians and researchers to assess the extent, pattern, and progression of bone loss over time. Advances in imaging technology, particularly CBCT, have further enhanced the ability to detect subtle osseous defects and three-dimensional bone changes. Understanding how diabetes influences radiographic patterns of bone loss is essential for accurate diagnosis, risk assessment, and treatment planning.

This review synthesizes available evidence on radiographic periodontal bone loss in diabetic versus non-diabetic patients, focusing on differences in severity, distribution, and defect morphology, as well as the biological mechanisms underlying these differences.

AIM

The primary aim of this review is to evaluate and compare the radiographic patterns and severity of periodontal bone loss in diabetic and non-diabetic patients based on available clinical and imaging evidence.

MATERIALS AND METHODS

A narrative review methodology was adopted to comprehensively synthesize available literature on radiographic periodontal bone loss in diabetic and non-diabetic populations. Electronic databases (Medline/PubMed, Embase, Scopus) from 2000 to 2025 searched for relevant articles published in English

up to 2025. Keywords and MeSH terms used in various combinations included "periodontal bone loss," "diabetes mellitus," "radiographic evaluation," "alveolar bone," "bitewing radiograph," "panoramic radiograph," "CBCT," and "periodontitis."

Studies were included if they reported radiographic assessment of periodontal bone loss in diabetic patients with comparison to non-diabetic controls. Observational studies, cross-sectional studies, case-control studies, retrospective radiographic analyses, and imaging-based clinical investigations were considered eligible. Reviews were consulted for background understanding but were not the primary source of outcome data. Studies focusing exclusively on clinical periodontal parameters without radiographic evaluation, animal studies, or case reports were excluded.

A PRISMA-inspired narrative screening process was followed. Titles and abstracts were initially screened for relevance, followed by full-text assessment of potentially eligible articles. Data extraction focused on study design, population characteristics, type of diabetes, imaging modality used, method of bone loss measurement, and key radiographic findings.

Radiographic Assessment of Periodontal Bone Loss

Radiographic evaluation of periodontal bone loss is traditionally performed using two-dimensional imaging modalities such as bitewing and periapical radiographs. Bitewing radiographs are particularly useful for assessing interproximal bone levels in posterior teeth and detecting early bone loss. Periapical radiographs provide detailed visualization of the entire tooth and surrounding alveolar bone but are limited by angulation errors and superimposition. Panoramic radiographs offer a comprehensive overview of the dentition and supporting structures, making them useful for large-scale epidemiological and retrospective studies, although their lower resolution may underestimate early bone changes [3].

The introduction of CBCT has significantly improved periodontal imaging by providing three-dimensional visualization of alveolar bone morphology. CBCT allows accurate assessment of intrabony defects, furcation involvement, and buccal and lingual bone plates, which are often obscured in conventional radiographs. However, higher radiation dose, cost, and limited availability restrict its routine use in periodontal assessment [4].

Radiographic Patterns of Bone Loss in Diabetic Patients

Multiple radiographic studies consistently demonstrate that diabetic patients exhibit greater alveolar bone loss compared to non-diabetic individuals. Retrospective panoramic radiographic analyses have shown significantly increased distance between the cementoenamel junction and alveolar crest in diabetic patients across both maxillary and mandibular arches [5]. The bone loss observed in diabetic patients tends to be more generalized rather than localized, reflecting the systemic nature of the disease.

Bitewing radiographic studies have revealed that both type 1 and type 2 diabetic patients have a higher prevalence of moderate to severe interproximal bone loss. In a well-controlled study of non-smoking adults with type 1 diabetes, radiographic bone loss was significantly greater than in matched non-diabetic controls, even after adjusting for age and plaque levels [6]. Importantly, poorly controlled diabetes has been associated with accelerated bone loss and more pronounced radiographic destruction.

CBCT-based studies further support these findings, demonstrating increased prevalence of vertical defects and intrabony lesions in diabetic patients. Although some studies report that diabetes does not independently predict the presence of vertical defects after adjustment for confounders, the overall burden of bone loss remains higher in diabetic populations [7].

Radiographic Patterns in Non-Diabetic Patients

In non-diabetic individuals, periodontal bone loss typically follows a pattern influenced primarily by local factors such as plaque accumulation, calculus, and occlusal trauma. Radiographic bone loss in non-diabetic patients is often more localized, corresponding to sites of poor oral hygiene or anatomical susceptibility. Horizontal bone loss is the most common pattern observed, with vertical defects developing in advanced or aggressive disease.

Comparative studies consistently show that non-diabetic patients demonstrate lower mean bone loss measurements and fewer sites with advanced destruction when compared with diabetic counterparts of similar age and periodontal status [8]. These findings underscore the modifying effect of systemic metabolic control on periodontal bone metabolism.

Biological Mechanisms Linking Diabetes and Bone Loss

The increased radiographic bone loss observed in diabetic patients can be explained by several interconnected biological mechanisms. Chronic hyperglycemia leads to the formation and accumulation of advanced glycation end-products (AGEs), which interact with their receptors (RAGE) on inflammatory and bone cells. This interaction amplifies the inflammatory response, resulting in increased production of pro-inflammatory cytokines such as tumor necrosis factor-alpha and interleukin-6 [9].

Diabetes also disrupts normal bone remodeling by altering the balance between osteoblast-mediated bone formation and osteoclast-mediated bone resorption. Increased expression of receptor activator of nuclear factor kappa-B ligand (RANKL) and decreased osteoprotegerin levels promote osteoclastogenesis and bone resorption. Additionally, impaired angiogenesis and reduced collagen synthesis compromise bone healing and regeneration, contributing to persistent bone loss [8].

The immune dysfunction associated with diabetes further exacerbates periodontal destruction. Neutrophil dysfunction, impaired chemotaxis, and exaggerated inflammatory responses result in ineffective bacterial clearance and prolonged tissue damage, which is reflected in more severe radiographic bone loss [10].

DISCUSSION

The present review highlights consistent and compelling evidence that diabetes mellitus exerts a significant influence on the radiographic manifestation of periodontal bone loss. Across diverse populations, imaging modalities, and study designs, diabetic individuals demonstrate greater severity, extent, and generalization of alveolar bone destruction when compared to non-diabetic counterparts. These differences are not merely quantitative but also qualitative, reflecting distinct biological mechanisms that modify the host response to periodontal infection. Radiographic findings thus serve as an objective reflection of the complex interplay between systemic metabolic dysregulation and localized periodontal tissue breakdown.

One of the most prominent observations across radiographic studies is the increased magnitude of alveolar bone loss in diabetic patients. Panoramic and bitewing radiographs consistently reveal increased cementoenamel junction to alveolar crest distances in diabetic individuals, indicating more advanced periodontal destruction. This finding has been reported in both type 1 and type 2 diabetes, although the

effect appears more pronounced in patients with long-standing disease and poor glycemic control. Importantly, this radiographic bone loss often appears generalized rather than site-specific, suggesting that diabetes acts as a systemic modifier rather than a localized risk factor. In contrast, non-diabetic patients typically exhibit bone loss patterns that correlate more closely with local etiological factors such as plaque accumulation, calculus, and anatomical predispositions [11].

The pattern of bone loss observed radiographically in diabetic patients is predominantly horizontal, although vertical and angular defects are also reported with greater frequency in advanced cases. Horizontal bone loss may reflect a chronic, sustained inflammatory burden driven by persistent hyperglycemia and systemic inflammation. Vertical defects, while less common, are clinically significant as they are often associated with rapid disease progression and poorer prognosis. CBCT-based studies have enhanced the detection of such defects, revealing that diabetic patients may harbor complex intrabony lesions that are not readily apparent on conventional two-dimensional radiographs. However, variability in CBCT findings suggests that diabetes alone may not be sufficient to determine defect morphology, and that disease duration, glycemic control, and coexisting risk factors play crucial roles [12].

A critical aspect influencing radiographic outcomes is glycemic control. Numerous studies emphasize that well-controlled diabetic patients exhibit radiographic bone loss patterns more comparable to non-diabetic individuals, whereas poorly controlled diabetes is associated with accelerated and extensive bone destruction. This observation underscores the importance of metabolic control in modulating periodontal disease progression. Hyperglycemia promotes the accumulation of advanced glycation end-products (AGEs) within periodontal tissues, which interact with their receptors on immune cells, endothelial cells, and osteoblasts. These interactions amplify inflammatory signaling pathways, leading to increased production of pro-inflammatory cytokines and matrix metalloproteinases that directly contribute to connective tissue degradation and alveolar bone resorption. Radiographically, these molecular events manifest as progressive crestal bone loss and reduced bone density [13].

Altered bone metabolism represents another key mechanism underlying the increased radiographic bone loss observed in diabetic patients. Diabetes disrupts the physiological balance between bone formation and resorption by enhancing osteoclast activity while simultaneously impairing osteoblast differentiation and function. Increased expression of receptor activator of nuclear factor kappa-B ligand (RANKL) and reduced osteoprotegerin levels favor osteoclastogenesis, resulting in heightened bone resorption. Moreover, impaired angiogenesis and reduced collagen synthesis compromise bone repair and regeneration. These alterations contribute not only to greater bone loss but also to diminished healing capacity following periodontal therapy, a phenomenon frequently noted in radiographic follow-up studies of diabetic patients [11,14].

The immune dysfunction associated with diabetes further exacerbates periodontal destruction. Neutrophil dysfunction, impaired chemotaxis, and defective phagocytosis compromise the host's ability to control periodontal pathogens effectively. At the same time, macrophages and monocytes exhibit a hyper-responsive inflammatory phenotype, producing excessive cytokines even in response to minimal bacterial challenge. This exaggerated inflammatory response leads to collateral tissue damage and sustained bone resorption. Radiographically, this translates into more severe and widespread bone loss, even in the presence of comparable plaque levels between diabetic and non-diabetic patients. These findings reinforce the concept that diabetes alters disease susceptibility rather than simply increasing bacterial burden [15].

From a diagnostic perspective, the choice of radiographic modality significantly influences the detection and interpretation of periodontal bone loss. Panoramic radiographs are widely used in epidemiological and retrospective studies due to their convenience and broad anatomical coverage. However, their inherent limitations, including image distortion and reduced resolution, may underestimate early bone changes. Bitewing radiographs offer superior sensitivity for detecting interproximal bone loss and are particularly valuable in identifying early disease progression. CBCT provides unparalleled three-dimensional visualization, allowing precise assessment of intrabony defects and buccolingual bone loss, which are often missed on conventional radiographs. Nevertheless, the routine use of CBCT in periodontal diagnosis remains limited due to concerns regarding radiation exposure, cost, and accessibility [16].

Interpretation of the existing literature is complicated by methodological heterogeneity. Differences in study design, population characteristics, imaging protocols, and measurement techniques limit direct comparisons across studies. Many investigations rely on retrospective data and lack standardized calibration procedures, increasing the potential for measurement bias. Additionally, important confounding factors such as smoking status, oral hygiene practices, duration of diabetes, and use of periodontal or antidiabetic medications are inconsistently controlled. These variables may independently influence periodontal bone loss and confound the observed association between diabetes and radiographic findings [17].

Despite these limitations, the consistency of findings across diverse settings supports the conclusion that diabetes is a significant modifier of periodontal bone loss. The radiographic evidence aligns with clinical and biological data, reinforcing the bidirectional relationship between periodontal disease and diabetes. From a clinical standpoint, these findings highlight the importance of comprehensive periodontal evaluation in diabetic patients, including routine radiographic monitoring. Early detection of bone loss allows timely intervention, which may not only preserve periodontal health but also contribute to improved glycemic control.

The discussion also underscores the need for an interdisciplinary approach to patient management. Collaboration between dental professionals, physicians, and endocrinologists is essential to address the shared inflammatory pathways linking diabetes and periodontal disease. Radiographic findings should be interpreted in conjunction with clinical parameters and metabolic indicators to provide a holistic assessment of disease status and risk.

In summary, discussion emphasizes that radiographic patterns of periodontal bone loss in diabetic patients reflect the cumulative effects of metabolic dysregulation, immune dysfunction, and impaired bone remodeling. While radiographic imaging remains an indispensable diagnostic tool, future research should focus on longitudinal, standardized, and technologically advanced approaches to further elucidate the impact of diabetes on periodontal bone dynamics.

CONCLUSION

Diabetes mellitus significantly influences the radiographic presentation of periodontal bone loss. Diabetic patients consistently demonstrate greater severity, extent, and generalization of alveolar bone loss compared with non-diab-

-etic individuals. These differences are attributable to metabolic, inflammatory, and immunological alterations associated with chronic hyperglycemia. Radiographic assessment remains a critical tool in identifying and monitoring periodontal destruction in diabetic patients. Early diagnosis, strict glycemic control, and comprehensive periodontal management are essential to mitigate bone loss and improve overall outcomes.

GAPS IN RESEARCH

Despite substantial evidence, several gaps remain in the current literature. There is a need for longitudinal radiographic studies that evaluate the progression of bone loss in relation to glycemic control over time. Standardized radiographic measurement protocols are lacking, limiting comparability across studies. The role of emerging imaging technologies such as artificial intelligence-assisted radiographic analysis remains underexplored. Additionally, limited data exist on radiographic bone changes in prediabetic individuals, representing an important area for future research.

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