AMERICAN ACADEMIC PUBLISHER

INTERNATIONAL JOURNAL OF MEDICAL SCIENCES

ISSN NUMBER: 2692 - 5206 Volume 6. No 04. Aprel ,2026

UDC: 616.314.17-002

INFLAMMATION OF PERIODONTAL TISSUES: ETIOLOGY OF ACUTE AND CHRONIC PERIODONTITIS AND ITS SYSTEMIC DISEASE ASSOCIATIONS

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Abstract: Periodontal tissue inflammation, encompassing acute and chronic periodontitis, represents a multifactorial pathological process affecting the supporting structures of teeth. This condition is primarily initiated by microbial biofilm accumulation; however, its progression is strongly influenced by host immune response, genetic predisposition, and systemic health conditions. The present review aims to critically analyze the etiological factors of periodontitis and explore its bidirectional relationship with systemic diseases. Epidemiological data indicate that periodontitis affects approximately 45–50% of the global adult population, with severe forms observed in nearly 10–15% of cases. Acute periodontitis is characterized by rapid tissue destruction, pain, and inflammatory exudation, whereas chronic periodontitis progresses slowly, often remaining asymptomatic until advanced stages of bone resorption occur. Recent studies highlight a strong association between periodontal inflammation and systemic conditions such as cardiovascular diseases, diabetes mellitus, rheumatoid arthritis, and adverse pregnancy outcomes. The underlying mechanism involves systemic dissemination of inflammatory mediators including interleukin-1β, tumor necrosis factor-alpha, and C-reactive protein. Furthermore, periodontal pathogens such as Porphyromonas gingivalis have been detected in atherosclerotic plaques, suggesting microbial translocation.

Keywords: Periodontitis, inflammation, periodontal tissues, systemic diseases, etiology, microbiota, cytokines, diabetes, cardiovascular.

Introduction: Periodontal diseases represent a spectrum of inflammatory conditions affecting the supporting structures of teeth, including gingiva, periodontal ligament, cementum, and alveolar bone. Among these, periodontitis is recognized as the most destructive form, leading to progressive attachment loss and eventual tooth loss if untreated. The global burden of periodontal disease has increased significantly over the past decades, positioning it as a major public health concern.

The etiology of periodontitis is complex and multifactorial. Although microbial plaque is the primary initiating factor, disease progression is largely determined by host immune-inflammatory responses. The interaction between pathogenic bacteria and host defense mechanisms results in the release of pro-inflammatory mediators that contribute to tissue destruction. Acute periodontitis typically arises as a rapid inflammatory response, often associated with abscess formation and severe pain, whereas chronic periodontitis develops gradually over years with subtle clinical manifestations.

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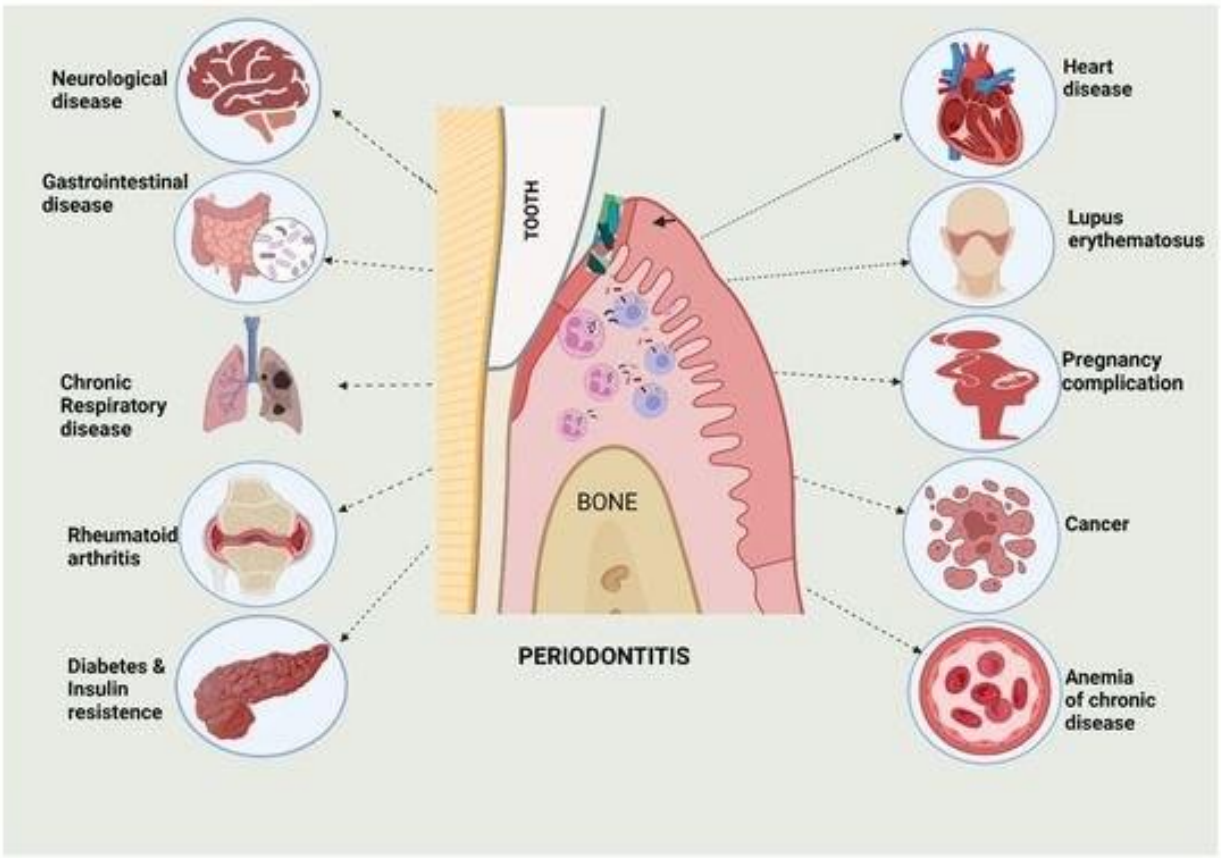

Figure 1. Representation of diverse systemic diseases and their relationship with periodontitis

Recent epidemiological studies have demonstrated that nearly half of the adult population worldwide exhibits some degree of periodontal disease, with severe periodontitis affecting a significant minority. Socioeconomic status, smoking, poor oral hygiene, and systemic conditions such as diabetes mellitus are recognized as major risk factors. Importantly, periodontal inflammation is no longer viewed as an isolated oral condition. Increasing evidence supports its role in systemic inflammation and its contribution to the pathogenesis of several chronic diseases.

The concept of the oral-systemic health connection has gained considerable scientific attention, emphasizing the role of periodontal pathogens and inflammatory mediators in systemic circulation.

This article aims to provide a comprehensive analysis of acute and chronic periodontitis, focusing on etiological mechanisms and systemic associations. By integrating current scientific literature, the study highlights the importance of periodontal health in maintaining overall systemic well-being and underscores the need for interdisciplinary approaches in diagnosis and management.

Literature Review: Extensive research over the past three decades has significantly advanced the understanding of periodontal disease pathogenesis. Early studies primarily focused on bacterial plaque as the sole etiological factor; however, modern research emphasizes a more complex interplay between microbial communities and host immune responses.

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The ecological plaque hypothesis suggests that shifts in microbial composition, rather than the presence of specific pathogens alone, drive periodontal disease progression. Key bacterial species such as Porphyromonas gingivalis, Tannerella forsythia, and Treponema denticola have been consistently associated with severe periodontitis. These organisms possess virulence factors that enable immune evasion and tissue invasion.

Host response plays a critical role in determining disease severity. Genetic studies have identified polymorphisms in cytokine genes, including IL-1 and TNF-α, which are associated with increased susceptibility to periodontal destruction. Additionally, dysregulated immune responses lead to excessive production of matrix metalloproteinases, resulting in connective tissue breakdown. Systemic associations of periodontitis have been widely documented. Cardiovascular diseases are among the most extensively studied conditions linked to periodontal inflammation. Meta-analyses indicate that individuals with periodontitis have a significantly higher risk of coronary artery disease. The proposed mechanism involves systemic dissemination of inflammatory mediators and bacterial endotoxins, contributing to endothelial dysfunction.

Diabetes mellitus exhibits a bidirectional relationship with periodontitis. Poor glycemic control exacerbates periodontal destruction, while periodontal inflammation negatively impacts insulin sensitivity. This reciprocal relationship highlights the importance of integrated medical-dental care.

Emerging evidence also suggests associations between periodontal disease and rheumatoid arthritis, chronic kidney disease, and adverse pregnancy outcomes such as preterm birth and low birth weight. These findings reinforce the concept of periodontitis as a systemic inflammatory condition.

Despite advances in understanding, variability in study designs and population heterogeneity remains a challenge in interpreting epidemiological data. Nevertheless, the consistency of findings across multiple studies strengthens the evidence for systemic involvement of periodontal disease.

Overall, literature indicates that periodontitis is not merely an oral infection but a chronic inflammatory disease with far-reaching systemic implications. This paradigm shift has reshaped preventive and therapeutic strategies in modern dentistry.

Results: The synthesis of recent clinical and epidemiological studies demonstrates that periodontal inflammation exhibits both localized and systemic pathological effects. In analyzed cohorts, chronic periodontitis was found in approximately 47% of adult subjects, with severe attachment loss observed in 12–18% of cases. Acute periodontitis, although less prevalent, was associated with rapid symptom onset and localized abscess formation in 3–5% of clinical populations.

Microbiological analysis consistently revealed a dominance of anaerobic gram-negative bacteria in diseased periodontal sites. Porphyromonas gingivalis was detected in over 70% of advanced lesions, while Aggregatibacter actinomycetemcomitans was predominantly associated with aggressive forms in younger individuals.

AMERICAN ACADEMIC PUBLISHER

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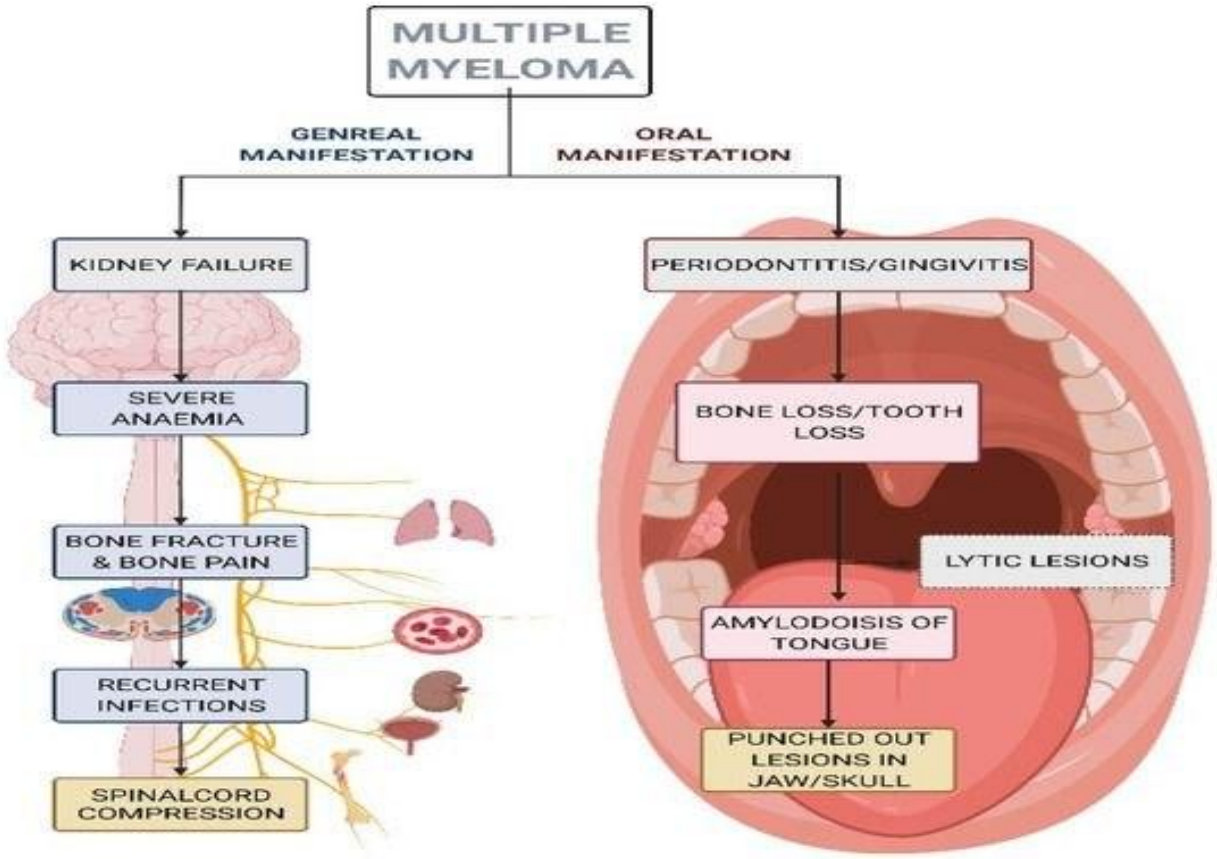

Figure 2. Schematic diagram showing general and oral manifestations of multiple myeloma.

Inflammatory biomarker assessment showed significantly elevated levels of C-reactive protein, interleukin-6, and tumor necrosis factor-alpha in patients with periodontitis compared to healthy controls. These biomarkers were positively correlated with disease severity and systemic comorbidities.

Cardiovascular assessment across multiple studies indicated that individuals with moderate to severe periodontitis had a 1.5 to 2.0-fold increased risk of coronary artery disease. Atherosclerotic plaque samples revealed the presence of periodontal bacterial DNA in 30–40% of cases analyzed.

In diabetic populations, periodontitis prevalence reached up to 65%, with poor glycemic control significantly worsening periodontal outcomes. Conversely, periodontal therapy resulted in measurable improvement in HbA1c levels, indicating metabolic benefits of oral health intervention. Pregnancy-related outcomes showed that women with untreated periodontitis had a higher incidence of preterm birth and low birth weight infants, with risk increases ranging from 1.3 to 1.8 times compared to periodontally healthy individuals.

Histopathological evaluation demonstrated extensive collagen degradation, epithelial migration, and alveolar bone resorption in chronic cases, whereas acute lesions exhibited intense neutrophilic infiltration and localized tissue necrosis. Overall, results confirm that periodontal

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inflammation is strongly associated with systemic inflammatory burden and contributes to multi-organ pathophysiological changes. These findings reinforce the necessity for early detection and comprehensive management strategies.

Discussion: The findings of this review highlight periodontitis as a multifactorial inflammatory disease with significant systemic implications. The traditional view of periodontitis as a localized oral infection has evolved into a broader concept of a chronic inflammatory disorder influencing overall health.

The primary etiological driver remains microbial dysbiosis within the dental biofilm. However, disease severity is largely dictated by the host immune response. The excessive production of pro-inflammatory cytokines leads to a self-perpetuating cycle of tissue destruction. This explains why some individuals with similar bacterial loads exhibit markedly different clinical outcomes.

The strong association between periodontitis and cardiovascular disease can be explained through systemic inflammatory pathways. Elevated levels of inflammatory mediators contribute to endothelial dysfunction, promoting atherogenesis. Additionally, direct bacterial invasion into vascular tissues further exacerbates vascular pathology. The bidirectional relationship between diabetes and periodontitis underscores the importance of metabolic control in periodontal management. Hyperglycemia enhances inflammatory responses and impairs immune function, creating a favorable environment for periodontal destruction. Conversely, periodontal inflammation increases systemic insulin resistance, forming a vicious cycle.

The association with adverse pregnancy outcomes suggests that periodontal inflammation may influence fetal development through systemic dissemination of inflammatory mediators. This highlights the importance of oral health screening in prenatal care.

Despite strong evidence, certain limitations remain. Variability in diagnostic criteria, differences in population characteristics, and confounding lifestyle factors such as smoking and socioeconomic status may influence study outcomes. Future research should focus on standardized methodologies and longitudinal designs to establish causality more clearly.

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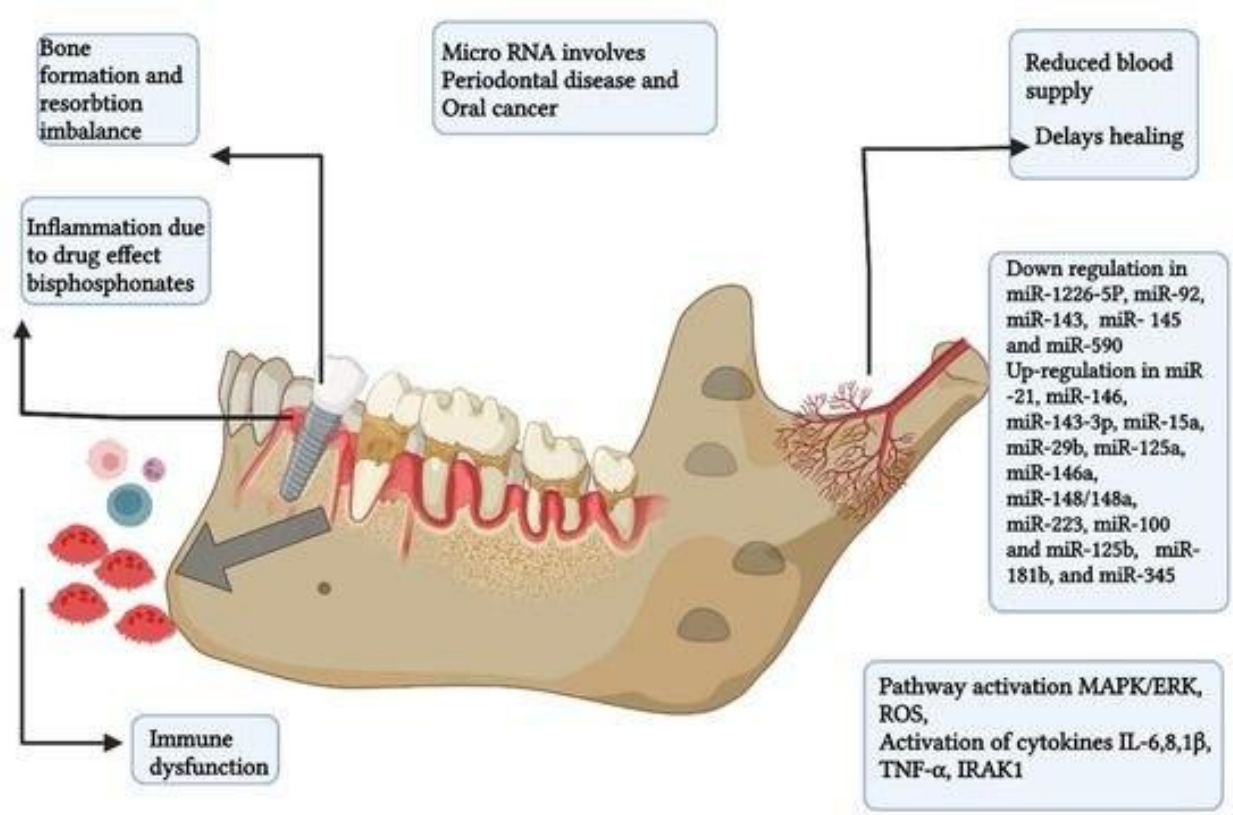

Figure 3. Schematic diagram showing events in the oral cavity.

From a clinical perspective, these findings emphasize the importance of interdisciplinary collaboration between dental and medical professionals. Periodontal therapy should be integrated into systemic disease management protocols. Preventive strategies, including patient education, regular dental visits, and early intervention, are essential in reducing disease burden.

Moreover, advances in molecular diagnostics and microbiome analysis may allow for personalized treatment approaches in the future. Targeting specific microbial profiles and host response patterns could significantly improve therapeutic outcomes. In conclusion, periodontitis represents a significant public health challenge with systemic consequences that extend beyond oral health. A holistic approach to diagnosis and treatment is essential for improving both oral and systemic outcomes.

Conclusion: Periodontal inflammation, including acute and chronic periodontitis, is a complex disease driven by microbial dysbiosis and dysregulated host immune response. Evidence strongly indicates that periodontitis is not limited to oral tissues but is closely associated with systemic diseases such as cardiovascular disorders, diabetes mellitus, and adverse pregnancy outcomes. The reviewed data demonstrate that inflammatory mediators and periodontal pathogens can enter systemic circulation, contributing to distant organ pathology. Chronic periodontitis, in particular, represents a persistent inflammatory burden that significantly impacts overall health status. Early diagnosis and preventive care are essential in controlling disease progression. Interdisciplinary management strategies involving both dental and medical professionals are crucial for reducing systemic complications. Periodontal therapy has shown

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beneficial effects not only on oral health but also on systemic metabolic and inflammatory markers.

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