

CHRONIC PERIODONTITIS: A SILENT KILLER

Arun brar¹, Arunima Guru², Monika kaur³

^{1,2}Reader, ³Intern, Desh Bhagat Dental College and Hospital, Mandi Gobindgarh, Punjab, India

ABSTRACT

Chronic periodontitis is much more than “bleeding gums” or a cause of tooth loss. It operates quietly, often undetected, yet contributes to a systemic inflammatory burden that may tip the scale toward major noncommunicable diseases. Its associations with cardiovascular disease, diabetes, adverse pregnancy outcomes, respiratory disorders, autoimmune disease, and increased mortality position it as a “silent killer”.

KEYWORDS: Chronic periodontitis, inflammatory disease, Systemic Diseases

INTRODUCTION

“Silent killer” is a term often applied to diseases that inflict harm gradually, often without clear symptoms until substantial damage has occurred (e.g. hypertension, diabetes). Chronic periodontitis, a long-standing inflammatory disease of the supporting structures of the teeth, fits this description in many ways: it progresses insidiously, often unnoticed by patients, yet may contribute not only to tooth loss but also to systemic damage across multiple organs. In recent decades, accumulating evidence has suggested that chronic periodontitis is not merely an oral disease but a contributor to systemic inflammation, and may influence risks for cardiovascular disease, diabetes, adverse pregnancy outcomes, and more. In this article, we explore the epidemiology, pathogenesis, systemic implications, clinical significance, and management of chronic periodontitis — arguing that its “silent” progression and systemic footprint make it akin to a hidden killer.¹

EPIDEMIOLOGY AND CLINICAL FEATURES

Chronic periodontitis (now more commonly called “periodontitis,” per modern classification) is a microbially driven, host-mediated disease that results in progressive destruction of periodontal attachment (i.e. periodontal ligament, alveolar bone) and pocket formation.

Key clinical signs include:

Increase in probing depth (≥ 4 mm)

Clinical attachment loss

Radiographic evidence of alveolar bone loss

Bleeding on probing, gingival inflammation, recession

Diagnosis involves clinical examination and radiographic assessment, as well as classification by severity and extent (mild, moderate, severe; localized vs generalized).

PREVALENCE & Public Health Burden

Globally, periodontitis is highly prevalent. An estimated 30–35% of adults have some form of periodontitis.

Severe periodontitis affects around 11–12% of the global population.

Its high prevalence, combined with its progressive and largely asymptomatic nature in early phases, underscores its significance as a public health issue.

Because tooth loss may occur gradually over many years, many patients and clinicians regard it as part of “normal ageing,” thereby underestimating the disease's impact. Even before frank tooth loss, however, the chronic inflammatory burden may quietly influence systemic health.²

PATHOGENESIS: FROM LOCAL INFLAMMATION TO SYSTEMIC RIPPLES

Understanding how chronic periodontitis might impact the body beyond the mouth involves dissecting both local and systemic pathways.

Microbial Initiation & Dysbiosis

The initiation of periodontitis is driven by a complex biofilm of subgingival bacteria. Key pathogens include *Porphyromonas gingivalis*, *Aggregatibacter actinomycetemcomitans*, and others.

Over time, dysbiosis — an imbalance in microbial ecology — further amplifies destructive inflammation.

Host Response, Inflammation & Tissue Destruction

The host's immune and inflammatory response is central to tissue damage in periodontitis. Immune cells—neutrophils, macrophages, T-cells—release cytokines (IL-1 β , IL-6, TNF- α , etc.), prostaglandins, matrix metalloproteinases (MMPs), and reactive oxygen species.

These mediators drive connective tissue degradation and alveolar bone resorption via osteoclast activation.

Bacteremia & Translocation of Bacterial Products

Inflamed and ulcerated gingival epithelium allows bacteria or their antigens (e.g. LPS) to enter the bloodstream, especially during routine activities like toothbrushing, chewing, or dental procedures.

The Periodontitis & Diabetes Hub

These oral bacteria or antigens may seed distant tissues (e.g. atheromatous plaques) or provoke immune activation in remote sites.

Systemic Inflammation & Mediator Spillover

Cytokines and acute-phase proteins produced locally (e.g. IL-6, IL-1 β , TNF- α , CRP) can diffuse into systemic circulation, raising systemic inflammatory tone.

Chronic low-grade systemic inflammation is a known driver or contributor to atherosclerosis, insulin resistance, endothelial dysfunction, and other pathologies.

Thus, chronic periodontitis can be conceptualized as an “inflammatory amplifier” that adds to the systemic burden of inflammation — sometimes pushing individuals over thresholds for clinically manifest disease.³

Links to Systemic Diseases: Evidence & Mechanisms

Over the past two decades, research has increasingly pointed to associations between periodontitis and a wide range of systemic conditions. While in many cases causality remains uncertain, the body of epidemiological, mechanistic, and interventional evidence is growing. Below, we examine key systemic conditions and what is known about their links to chronic periodontitis.

Cardiovascular Disease (CVD)

Numerous observational studies and meta-analyses have found that periodontitis is associated with an increased risk of coronary artery disease, stroke, and atherosclerosis.

In a large cohort, a 1-unit increase in periodontitis score was associated with a 40% higher risk of all-cause mortality, and the interaction of periodontitis with systemic inflammation added excess risk to both all-cause and cardiovascular mortality.

Mechanistic pathways: periodontal bacteria may be present in atherosclerotic plaques, and chronic inflammation promotes endothelial dysfunction, lipid oxidation, and thrombogenesis.

Some interventional trials (e.g. periodontal treatment) have shown modest improvements in endothelial function, though results are heterogeneous.

Diabetes Mellitus

The relationship between periodontitis and diabetes is bidirectional: diabetes is a risk factor for periodontitis, and periodontitis may worsen glycemic control. In patients with diabetes, periodontal therapy has been shown in some trials to lower HbA1c modestly (by ~0.3–0.4%) over 3–4 months. Mechanisms include systemic inflammation (IL-6, TNF- α) promoting insulin resistance, and infiltration of bacterial products into circulation further triggering metabolic dysregulation.⁴

Adverse Pregnancy Outcomes

Periodontitis has been linked in observational studies to increased risk of preterm birth, low birth weight, and preeclampsia. A meta-analysis of 30 studies showed a significant association between periodontitis and preeclampsia, especially in lower-middle-income settings. Proposed mechanisms: dissemination of inflammatory mediators, bacterial translocation to placental tissues, and systemic endothelial activation.

Respiratory Diseases & Pneumonia Periodontitis may increase risk of aspiration pneumonia, especially among older adults, via inhalation of oral pathogens.

The Periodontitis & Diabetes Hub

Associations have also been noted with chronic obstructive pulmonary disease (COPD).

Rheumatoid Arthritis & Other Autoimmune Diseases

A higher prevalence of periodontitis is observed in patients with rheumatoid arthritis. Shared inflammatory and immune pathways (e.g. citrullination, cytokines) may underlie this association. Systemic inflammation arising from periodontal disease might aggravate autoimmune processes or act as a co-trigger. Chronic Kidney Disease (CKD), Cognitive Decline, Metabolic Syndrome & More Emerging studies suggest associations with chronic kidney disease, cognitive decline/dementia, metabolic syndrome, obesity, and even certain cancers.

The concept of multi morbidity (multiple chronic diseases interacting) highlights how periodontitis may interplay with a host of non communicable diseases via shared inflammatory pathways.^{5,6}

CONCLUSION

Chronic periodontitis is much more than “bleeding gums” or a cause of tooth loss. It operates quietly, often undetected, yet

contributes to a systemic inflammatory burden that may tip the scale toward major noncommunicable diseases. Its associations with cardiovascular disease, diabetes, adverse pregnancy outcomes, respiratory disorders, autoimmune disease, and increased mortality position it as a “silent killer” — subtle in progression but potentially substantial in impact. Given its high prevalence, modifiable nature, and connection to systemic disease, periodontitis deserves recognition not just in dentistry but across medical and public health domains. Early detection, timely treatment, and integration with chronic disease care may help unmask and neutralize this hidden pathogen.

REFERENCES

1. Vaithilingam RD, Baharuddin NA. Periodontitis and Systemic Diseases. In: Immunology for Dentistry. 2023.
2. “Inflammatory Association between Periodontal Disease and Systemic Health” (2025).
3. Systemic effects of periodontitis: epidemiology of periodontal disease and cardiovascular disease.
4. Effects of periodontal disease on systemic health.
5. Linden GJ, working group of EFP/AAP. Periodontitis and systemic diseases: record of discussions. J Clin Periodontol.
6. State of the science: chronic periodontitis and systemic health.
7. Managing the Systemic Impact of Periodontitis. Medicina, 2022.