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Features of the Relationship Between Periodontal Diseases and Somatic Pathology: A Literature Review

▷ **Background.** Periodontal diseases represent one of the leading causes of oral tissue destruction worldwide and continue to pose a significant challenge to both dental practice and public health. Increasing evidence indicates that chronic inflammation of the periodontal tissues is closely associated with a broad spectrum of systemic disorders, particularly those affecting the cardiovascular, endocrine, gastrointestinal, respiratory, and immune systems. Disturbances in general health may alter host immune responses and tissue metabolism, thereby promoting the initiation and progression of periodontal inflammation, whereas, conversely, chronic oral infection may contribute to the systemic inflammatory burden.

Objective. This review aims to critically evaluate contemporary scientific evidence regarding the relationship between periodontal diseases and systemic disorders. Particular attention is given to the biological mechanisms responsible for this bidirectional interaction, including the influence of periodontal inflammation on systemic health and the impact of chronic systemic diseases on the structure and function of periodontal tissues. In addition, the review seeks to summarize current knowledge of the pathogenic pathways involved and to determine their clinical significance for improving preventive and therapeutic strategies.

Materials and Methods. A comprehensive literature review was performed using a structured search of the PubMed, Scopus, and Web of Science databases between January and February 2026. The review methodology was developed in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) recommendations. Publications issued between 2010 and 2024 were screened using combinations of the following Medical Subject Headings (MeSH) terms: *Periodontitis, Periodontal Diseases, Systemic Diseases, Diabetes Mellitus, Cardiovascular Diseases, Atherosclerosis, Rheumatoid Arthritis, Respiratory Tract Diseases, Gastrointestinal Diseases, and Chronic Inflammation*. Boolean operators AND and OR were applied to maximize the retrieval of relevant studies.

Results. The available evidence demonstrates that chronic generalized periodontitis develops through complex interactions among microbial, immunological, metabolic, and environmental factors. Systemic diseases are frequently accompanied by impaired immune regulation, microvascular dysfunction, and metabolic abnormalities that accelerate periodontal tissue destruction. At the same time, persistent periodontal infection contributes to chronic low-grade systemic inflammation, which may adversely influence the onset, progression, and prognosis of several systemic disorders. The strongest evidence for a bidirectional association has been reported for diabetes mellitus, whereas substantial epidemiological data also support links with cardiovascular diseases, rheumatoid arthritis, respiratory disorders, and gastrointestinal diseases.

Conclusion. Current scientific evidence indicates that periodontal diseases and systemic disorders influence one another through multiple interconnected biological pathways. Recognition of these interactions supports the implementation of an integrated, multidisciplinary approach that combines periodontal management with the assessment of the patient's systemic health. Such an approach may improve treatment outcomes, reduce the burden of chronic inflammation, and decrease the risk of both oral and systemic complications.

Keywords: *Periodontitis, Systemic diseases, Chronic inflammation, Comorbidity, Periodontal-systemic interaction.*

Study Selection and Eligibility Criteria

Studies were considered eligible if they addressed the relationship between periodontal diseases and systemic disorders, were directly relevant to the objectives of the review, were published in peer-reviewed scientific journals, provided accessible full texts, and demonstrated acceptable methodological quality. The review included original clinical investigations, systematic reviews, and meta-analyses published in English or Russian. Conference proceedings, editorials, letters to the editor, case reports, duplicate publications, and articles that did not provide sufficient information relevant to the review topic were excluded from further analysis.

Identification and selection of the literature were performed according to the PRISMA framework using a multistep screening process. The initial database search yielded 186 records. After removing duplicate citations, 154 publications remained for preliminary assessment. Screening of titles and abstracts resulted in the exclusion of 97 studies that failed to meet the predefined eligibility criteria. The full texts of the remaining 57 articles were subsequently evaluated, and 24 publications were excluded due to limited methodological rigor or inadequate relevance to the objectives of the review. Ultimately, 33 studies satisfied all eligibility requirements and were included in the qualitative synthesis.

Each eligible publication was critically appraised with regard to study design, population characteristics, diagnostic approaches used for periodontal and systemic diseases, methodological quality, and the principal outcomes reported by the authors. Following data extraction, the available evidence was organized into thematic categories according to the systemic conditions investigated, including cardiovascular diseases, diabetes mellitus, rheumatoid arthritis, gastrointestinal disorders, and respiratory diseases.

The adopted review methodology allowed for a comprehensive synthesis of contemporary evidence concerning the role of periodontal diseases in the onset and progression of systemic disorders. Furthermore, it facilitated the identification of the principal biological pathways that could explain the reciprocal relationship between periodontal pathology and systemic health.

Results and Discussion

Periodontal Diseases and Cardiovascular Disorders

The possible association between periodontal diseases and systemic disorders has been discussed in the scientific literature for more than one hundred years. One of the earliest concepts was proposed by McCall and Box in 1923, who introduced the term periodontitis complex to describe periodontal conditions thought to be influenced by systemic health disturbances. At that time, diabetes mellitus, gastrointestinal disorders, autoimmune diseases, obesity, cardiovascular diseases, and several other chronic conditions were considered potential contributors to periodontal tissue destruction [1].

Despite the considerable number of studies examining the relationship between periodontitis and cardiovascular diseases, the available evidence should be interpreted with caution. Most published investigations are observational and, therefore, cannot establish a direct cause-and-effect relationship. In addition, common risk factors—including aging, tobacco use, obesity, low socioeconomic status, and the presence of chronic comorbidities—may partially explain the reported associations. Although adjustment for these confounding variables reduces the magnitude of the observed relationship in several meta-analyses, the association generally remains statistically significant. These findings suggest that chronic periodontal inflammation may contribute to systemic inflammatory activity and accelerate atherosclerotic processes; nevertheless, definitive proof of causality has not yet been established [2–5].

Recent epidemiological and clinical studies have further strengthened the evidence linking periodontal inflammation with cardiovascular diseases. Greater severity of periodontitis and dysbiosis of the oral microbiome have been associated with an increased incidence of ischemic heart disease, stroke, and other cardiovascular disorders. Individuals diagnosed with chronic generalized periodontitis have been reported to experience an approximately 19% higher risk of cardiovascular disease, whereas among adults older than 65 years, this excess risk increases to nearly 44% [6]. Longitudinal cohort studies from the United States and South Korea have also demonstrated that severe periodontal destruction is associated with nearly a twofold increase in the occurrence of ischemic stroke, including both cardioembolic and thrombotic subtypes, as well as a higher incidence of myocardial infarction [7–9]. Likewise, tooth loss resulting from advanced periodontal destruction has been associated with modest but significant increases in the risk of ischemic heart disease and overall cardiovascular morbidity [10].

Current evidence suggests that periodontitis may promote atherosclerotic cardiovascular disease through two complementary biological pathways. The direct pathway involves the dissemination of periodontal microorganisms into the bloodstream, allowing bacterial colonization of vascular tissues and the

induction of local inflammatory responses. The indirect pathway is mediated through persistent systemic inflammation, characterized by increased circulating cytokine levels and other inflammatory mediators that contribute to endothelial dysfunction and the progression of atherosclerotic lesions [11, 12].

Collectively, these findings indicate that periodontal disease should not be regarded solely as a localized oral infection, but rather as a chronic inflammatory condition capable of influencing systemic health, particularly cardiovascular function. Consequently, the management of patients presenting with both periodontal and cardiovascular diseases should involve close collaboration between dental and medical professionals to optimize long-term outcomes [13].

Beyond improving oral health, comprehensive periodontal treatment may also contribute to lowering cardiovascular risk. Effective therapy includes not only the elimination of the local microbial biofilm, but also the control of inflammatory activity, restoration of periodontal tissue homeostasis, improvement of endothelial function, enhancement of microvascular circulation, and a reduction in circulating inflammatory biomarkers, thereby supporting overall systemic health [14, 15].

Diabetes Mellitus

Compared with other systemic disorders, the relationship between diabetes mellitus and periodontitis has been investigated more extensively and is supported by robust clinical and epidemiological evidence [16–20].

Among endocrine diseases, diabetes mellitus exerts one of the greatest influences on periodontal tissues. Advanced periodontal destruction occurs considerably more frequently in individuals with both type 1 and type 2 diabetes and is currently regarded as one of the major chronic complications associated with impaired glycemic control [21].

The interaction between diabetes and periodontitis is widely recognized as a bidirectional one. Chronic hyperglycemia induces diabetic microangiopathy, resulting in structural and functional alterations of the periodontal microcirculation. Reduced tissue perfusion limits oxygen delivery, stimulates osteoclastic activity, accelerates alveolar bone resorption, and delays regenerative processes. Simultaneously, elevated glucose concentrations within the gingival crevicular fluid provide favorable conditions for bacterial growth and facilitate the rapid maturation of the dental biofilm, thereby increasing susceptibility to periodontal destruction [22].

Conversely, chronic periodontal infection may negatively influence metabolic control in diabetic patients. Periodontal pathogens and their virulence factors can enter the systemic circulation, maintaining a persistent inflammatory response that contributes to insulin resistance and the progression of diabetes. Clinical studies have demonstrated that periodontitis is associated with a two- to threefold higher risk of major diabetic complications, including macroalbuminuria, end-stage renal disease, and cardiorenal mortality. Importantly, successful management of periodontal inflammation has been shown to improve metabolic outcomes, whereas effective glycemic control may reduce the severity and progression of periodontal disease. These observations support routine diabetes screening in patients diagnosed with periodontitis, as well as comprehensive periodontal assessment in individuals with newly diagnosed diabetes mellitus [23].

Evidence further indicates that prolonged hyperglycemia substantially increases susceptibility to periodontal disease. Individuals with poorly controlled diabetes demonstrate approximately a 2.8-fold greater risk of developing periodontitis and a 4.2-fold increase in the rate of alveolar bone loss compared with metabolically healthy individuals. Conversely, patients with periodontal disease have been reported to exhibit a 19–33% greater likelihood of developing type 2 diabetes. Furthermore, mortality among patients affected by both severe periodontitis and diabetes is approximately 3.2 times higher than that observed in diabetic patients without significant periodontal involvement. Elevated concentrations of inflammatory mediators in the gingival crevicular fluid and saliva further support the presence of enhanced local and systemic inflammatory activity in these patients [24].

A substantial body of clinical evidence indicates that periodontal therapy can improve glycemic control in diabetic individuals. This beneficial effect is believed to result from the suppression of chronic inflammation, the reduction of systemic cytokine levels, and the improvement of insulin sensitivity following successful periodontal treatment [25].

Gastrointestinal Diseases

The relationship between periodontal diseases and gastrointestinal disorders has been increasingly investigated during the past decade. Most available evidence originates from epidemiological studies and analyses of the oral and gastrointestinal microbiomes. Although periodontal pathogens have frequently been identified within the digestive tract, their direct involvement in the initiation of gastric and intestinal diseases remains incompletely understood. One of the most extensively studied topics concerns the association between periodontitis and *Helicobacter Pylori* infection; however, the published findings remain controversial. Whereas several authors consider

the oral cavity to serve as an extra-gastric reservoir for *H. Pylori*, others have reported that periodontal treatment alone does not significantly decrease the risk of reinfection following successful eradication therapy. Therefore, current evidence indicates a potential biological association rather than a confirmed causal relationship [22–24].

Long-standing gastrointestinal disorders are often accompanied by nutritional deficiencies involving vitamins, minerals, proteins, and essential micronutrients. Such metabolic disturbances may adversely affect the oral environment by altering the structure and function of the oral mucosa and reducing the regenerative capacity of periodontal tissues. Furthermore, inflammatory diseases affecting the digestive tract may interfere with calcium and iron absorption, thereby compromising alveolar bone metabolism and increasing susceptibility to periodontal tissue destruction [25].

Accumulating evidence suggests that the composition of the oral microbiome may influence gastrointestinal health. Oral bacteria are continuously transported into the digestive tract through saliva, allowing prolonged interaction with the gastrointestinal mucosa. Several periodontal pathogens, particularly *Porphyromonas gingivalis*, have been implicated in gastrointestinal dysbiosis and have been associated with a variety of digestive disorders, including malignant diseases. Individuals diagnosed with gastric or colorectal cancer have been reported to exhibit greater oral microbial diversity and an increased abundance of specific periodontal pathogens compared with healthy controls [26].

Dental plaque has also been proposed as a potential ecological niche for *Helicobacter Pylori*. The highly organized structure of the oral biofilm protects microorganisms from systemic antimicrobial therapy, allowing bacterial persistence despite the successful eradication of gastric infection. As a consequence, residual microorganisms within the oral cavity may subsequently recolonize the stomach and contribute to recurrent *H. Pylori* infection.

Evidence from a large population-based investigation involving 4,955 adults aged 20–90 years further supports this association. Participants underwent a comprehensive periodontal examination together with serological testing for *H. Pylori*. Individuals affected by periodontal disease demonstrated a significantly higher prevalence of *H. Pylori* infection than subjects with healthy periodontal tissues. In addition, periodontitis was independently associated with increased all-cause mortality and a higher incidence of malignant gastrointestinal neoplasms, particularly gastric and colorectal cancer [27].

Taken together, these findings suggest that the preservation of periodontal health may represent an important component of strategies aimed at preventing or limiting gastrointestinal diseases. Incorporating periodontal care into the multidisciplinary management of patients with digestive disorders may improve therapeutic outcomes and reduce the likelihood of disease progression.

Rheumatoid Arthritis

An increasing number of epidemiological and experimental studies indicate a close relationship between periodontal disease and rheumatoid arthritis (RA). Considerable attention has been focused on the potential contribution of periodontal microorganisms to the initiation and progression of autoimmune joint inflammation. Among these microorganisms, *Porphyromonas gingivalis* has emerged as the principal candidate because of its unique ability to interfere with host immune regulation and promote autoimmune responses.

Population-based studies consistently demonstrate that individuals with periodontitis have a greater probability of developing rheumatoid arthritis than those without periodontal disease. This relationship appears to be mediated through common biological mechanisms, including persistent systemic inflammation, dysregulation of innate and adaptive immunity, and alterations in the oral microbiome. Of particular importance is the ability of *P. gingivalis* to express peptidylarginine deiminase, an enzyme capable of inducing protein citrullination. This process contributes to the generation of anti-citrullinated protein antibodies, one of the characteristic immunological features of rheumatoid arthritis. Although available findings strongly support a biological association, current evidence remains insufficient to establish a definitive cause-and-effect relationship, thus highlighting the need for additional prospective and randomized clinical studies [18, 25].

Published epidemiological data indicate that the presence of periodontal disease is associated with an approximately 27% increase in the risk of developing rheumatoid arthritis. Because both diseases share multiple inflammatory, immunological, and molecular pathways, the therapeutic management of one condition may positively influence the clinical course of the other. The reduction of the periodontal bacterial burden together with the suppression of systemic inflammatory activity has been proposed as a strategy capable of slowing disease progression and improving outcomes in patients affected by both chronic periodontitis and rheumatoid arthritis[28].

Respiratory Diseases

Increasing scientific evidence suggests that the condition of the periodontal tissues is closely associated

with respiratory health. Numerous oral microorganisms commonly implicated in periodontitis, including *Aggregatibacter actinomycetemcomitans* (formerly *Actinobacillus actinomycetemcomitans*), *Actinomyces israelii*, *Capnocytophaga spp.*, *Eikenella corrodens*, *Prevotella intermedia*, *Porphyromonas gingivalis*, and *Streptococcus constellatus*, have also been isolated from patients with pneumonia, pulmonary abscesses, and other lower respiratory tract infections. Because the oral cavity is anatomically connected to the respiratory system, pathogenic microorganisms may readily gain access to the lower airways, particularly in individuals with impaired swallowing, diminished cough reflexes, or other conditions that facilitate aspiration [29].

Evidence synthesized from systematic reviews indicates that poor oral hygiene is associated with an increased risk of the aspiration of oral microorganisms, which may subsequently contribute to the development of pneumonia or acute exacerbations of chronic obstructive pulmonary disease (COPD) [30,31]. This complication is especially common among elderly individuals, patients with neurological disorders such as stroke, and those who are unable to maintain adequate oral hygiene during hospitalization [32].

In addition to direct aspiration, periodontal pathogens may influence respiratory disease through indirect biological mechanisms. Certain oral bacteria produce proteolytic enzymes capable of altering the integrity of the oropharyngeal epithelium. The degradation of surface proteins, including fibronectin, exposes epithelial receptors that facilitate the attachment and colonization of respiratory pathogens. These microbial and structural changes create favorable conditions for the infection of the lower respiratory tract and may contribute to both the onset and the progression of respiratory diseases [33].

Collectively, these observations emphasize the importance of maintaining periodontal health as part of strategies aimed at preventing respiratory infections. Effective plaque control, professional periodontal care, and the early treatment of periodontal inflammation may reduce the microbial burden within the oral cavity and consequently decrease the risk of aspiration-related pulmonary complications, particularly in medically compromised individuals.

Conclusions

The findings summarized in this review demonstrate that periodontal diseases should be regarded as chronic inflammatory conditions with consequences extending far beyond the oral cavity. Accumulating evidence supports a close relationship between periodontal pathology and a wide range of systemic disorders, including cardiovascular, endocrine, gastrointestinal, respiratory, and autoimmune diseases. Although the strength of evidence differs among individual conditions, the biological interactions between periodontal inflammation and systemic health are becoming increasingly well established.

The underlying mechanisms appear to involve the dissemination of periodontal microorganisms and their virulence factors into the systemic circulation through the ulcerated periodontal epithelium. This process promotes transient or persistent bacteremia, the activation of systemic inflammatory pathways, endothelial dysfunction, and immune dysregulation, all of which may contribute to pathological changes in distant organs and tissues. These observations reinforce the concept that the successful management of periodontitis requires not only the elimination of local infection, but also the consideration of its potential systemic consequences.

From a therapeutic standpoint, periodontal treatment should be incorporated into a comprehensive multidisciplinary approach to patient care. In addition to conventional mechanical periodontal therapy, clinical management may benefit from interventions aimed at improving microvascular function, supporting tissue metabolism, enhancing endothelial integrity, and reducing systemic inflammatory activity. Such an integrated strategy has the potential to lower the risk of systemic complications, delay the progression of chronic comorbid conditions, and improve long-term clinical outcomes.

Overall, the preservation of periodontal health should be recognized as an important component of general healthcare. Close collaboration between dental practitioners and physicians may facilitate the earlier identification of systemic disorders, optimize preventive measures, and improve individualized treatment planning. A better understanding of the bidirectional relationship between periodontal diseases and systemic pathology will contribute to the development of more effective preventive and therapeutic strategies that promote both oral and overall health.

Conflict of interest

The authors declare no conflict of interest.

Consent to publication

The authors have given their consent to the publication of the manuscript.

Use of Artificial Intelligence

The authors state that no artificial intelligence was used in the writing of the article.

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Особливості взаємозв'язку між захворюваннями пародонта та соматичною патологією: огляд літератури

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Актуальність. Захворювання пародонта є однією з основних причин руйнування тканин порожнини рота в усьому світі та продовжують становити значний виклик як для стоматологічної практики, так і для системи охорони здоров'я в цілому. Все більше даних свідчать про те, що хронічне запалення тканин пародонта тісно пов'язане із широким спектром системних розладів, зокрема тими, що уражають серцево-судинну, ендокринну, шлунково-кишкову, дихальну та імунну системи. Порушення загального стану здоров'я можуть змінювати імунні

реакції організму та метаболізм тканин, сприяючи виникненню та прогресуванню запалення пародонта, тоді як хронічна інфекція порожнини рота, навпаки, здатна підсилювати системне запальне навантаження.

Мета. Цей огляд спрямований на критичну оцінку сучасних наукових даних щодо взаємозв'язку між захворюваннями пародонта та системними розладами. Особливу увагу приділено біологічним механізмам, відповідальним за цю двосторонню взаємодію, зокрема впливу запалення пародонта на системне здоров'я та впливу хронічних соматичних захворювань на структуру й функцію тканин пародонта. Крім того, огляд має на меті узагальнити сучасні уявлення про залучені патогенетичні шляхи та визначити їхнє клінічне значення для вдосконалення профілактичних і терапевтичних стратегій.

Матеріали та методи. Комплексний огляд літератури було проведено на основі структурованого пошуку в базах даних PubMed, Scopus та Web of Science. Методологію огляду було розроблено відповідно до рекомендацій «Переважні параметри для звітів про системні огляди та метааналізи» (PRISMA). Публікації відбиралися із використанням комбінацій таких предметних медичних рубрик (MeSH): *Periodontitis*, *Periodontal Diseases*, *Systemic Diseases*, *Diabetes Mellitus*, *Cardiovascular Diseases*, *Atherosclerosis*, *Rheumatoid Arthritis*, *Respiratory Tract Diseases*, *Gastrointestinal Diseases* та *Chronic Inflammation*. Для максимального охоплення релевантних досліджень застосовувалися булеві оператори AND та OR.

Результати. Наявні дані свідчать про те, що хронічний генералізований пародонтит розвивається внаслідок складної взаємодії між мікробними, імунологічними, метаболічними чинниками та факторами навколишнього середовища. Системні захворювання часто супроводжуються порушенням імунної регуляції, мікросудинною дисфункцією та метаболічними відхиленнями, які прискорюють деструкцію тканин пародонта. Водночас персистуюча пародонтальна інфекція сприяє розвитку хронічного системного запалення низької інтенсивності, що може негативно впливати на початок, прогресування та прогноз перебігу низки системних розладів. Найпереконливіші докази двостороннього зв'язку зафіксовано для цукрового діабету, тоді як значні епідеміологічні дані також підтверджують наявність зв'язку із серцево-судинними захворюваннями, ревматоїдним артритом, захворюваннями дихальних шляхів та шлунково-кишкового тракту.

Висновки. Сучасні наукові дані свідчать про те, що захворювання пародонта та системні розлади взаємно впливають одне на одне через численні взаємопов'язані біологічні шляхи. Урахування цих взаємодій підтверджує необхідність впровадження інтегрованого мультидисциплінарного підходу, який поєднує лікування пародонта з оцінкою загального стану здоров'я пацієнта. Такий підхід дає змогу покращити результати лікування, зменшити навантаження хронічного запалення та знизити ризик розвитку як стоматологічних, так і системних ускладнень.

Ключові слова: *пародонтит; системні захворювання; хронічне запалення; коморбідність; пародонтально-системна взаємодія.*

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