RESEARCH PAPER

Exploring the Relationship Between Periodontitis and Atrial Fibrillation: A Comprehensive Narrative Review

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ABSTRACT
Background: Periodontitis (Periodontal Disease) is a widespread chronic inflammatory condition that progressively destroys the supporting structures of the teeth. Beyond causing local tissue breakdown, the persistent inflammatory response and microbial invasion characteristic of the disease can influence systemic physiology. Atrial fibrillation (AF), which is a common cardiac disease worldwide, is also strongly shaped by inflammatory and oxidative processes that alter atrial structure and electrical function. These parallels have prompted growing interest in whether chronic periodontal conditions connect to AF risk. This review synthesizes current evidence on the link between periodontitis and AF, also explores the biological structures that may link the two conditions, evaluates areas of agreement and disagreement across studies, and identifies research gaps requiring further investigation.
Methods: A narrative review methodology was used. PubMed, Scopus, and Web of Science were searched up to January 2025 using terms related to periodontal disease, atrial fibrillation, inflammatory pathways, endothelial dysfunction, and oral microbiota. Observational, interventional, mechanistic, and review studies addressing the periodontitis–AF relationship were included, while articles lacking clear diagnostic criteria or relevance were excluded.
Results: Large observational datasets generally show that individuals with moderate to severe periodontitis exhibit a higher likelihood of developing AF. Experimental studies support plausible mechanistic links, showing that periodontal pathogens and inflammatory mediators can disrupt endothelial integrity, intensify oxidative stress, and promote atrial fibrosis—all processes central to AF development. Treatment of Periodontal disease has been shown to reduce systemic inflammatory markers and improve vascular parameters; however, direct evidence showing reduced AF incidence or recurrence following periodontal therapy remains sparse.
Conclusion: Current evidence points toward a meaningful connection between chronic periodontal inflammation and AF vulnerability, although the available data do not yet confirm a causal relationship. More rigorous longitudinal and interventional research is required to determine whether improving periodontal health can play a protective influence on the prevention or management of AF...
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INTRODUCTION

Periodontitis is a chronic condition that gradually destroys the periodontal ligament, alveolar bone, and other tooth-supporting structures caused by an imbalance in the oral microbial community. It continues to be a major cause of tooth loss and affects almost half of the adult population, making it the most common chronic disease in the world[1]. Periodontitis is increasingly seen as a systemic inflammatory burden in addition to its local effects. Periodontal Diseases necrosis factor-alpha (TNF-α), and C-reactive protein (CRP). This suggests that oral infection is a contributing cause of extensive physiological disorders[2].
There is mounting evidence to suggest that this persistent inflammatory load may impact disorders outside of the oral cavity. For example, inflammation of the periodontal ligament has been constantly associated with cardiovascular diseases. An estimated 33 million people are affected by atrial fibrillation (AF), the most common persistent arrhythmia in the world, which is becoming more common as populations age[3]. Electrical abnormalities, atrial myocardial structural remodelling, and changes in autonomic control interact intricately in the pathogenesis of AF. Elevated inflammatory markers are commonly observed both before the onset of AF and during recurrence following rhythm-control treatments, indicating that systemic inflammation is a recognised contributor to both processes.
Researchers have shown that periodontitis may act as a modifiable factor influencing the formation of AF, as it is a chronic source of microbial products and inflammatory mediators. This notion is supported by a number of biologically viable mechanisms. During ordinary oral

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activities, periodontal infections like Porphyromonas gingivalis can enter the systemic circulation and cause dysfunction of the endothelial system, oxidative damage, and fibrotic responses—changes that are similar to those linked to atrial remodeling[4]. Furthermore, persistent oral inflammation may upset autonomic homeostasis, making the environment more vulnerable to arrhythmic episodes. While epidemiological studies have shown that those with severe periodontitis have a greater prevalence of AF, experimental models have demonstrated that periodontal infection can speed up structural changes inside heart tissue[5].

The relationship is still unclear in spite of these discoveries. According to certain research, correlations become weaker when common risk variables, including age, smoking, and cardiometabolic disorders, are taken into consideration. Interpretation is made more difficult by differences in study design and inconsistent periodontal assessment techniques. However, the confluence of clinical, mechanistic, and epidemiological findings highlights the need for a closer look at this relationship[6].

By analyzing epidemiological trends, biological processes, and results from treatment interventions, this study summarizes the state of knowledge about the possible connection between periodontitis and AF. It also describes the limitations of the research and explores the potential clinical benefits of incorporating periodontal care into frameworks for cardiovascular prevention.

METHODOLOGY

In order to collect and compile the most recent data on the possible connection between periodontitis and atrial fibrillation (AF), this narrative review used an organized and transparent methodology. The methodological framework was created to ensure thorough coverage and academic rigor while capturing results from mechanistic studies, clinical investigations, and epidemiological research.

Major scientific databases, such as PubMed, Scopus, Web of Science, Google Scholar, and the Cochrane Library, were searched extensively for relevant literature. To maximize the retrieval of pertinent studies, both free-text phrases and Medical Subject Headings (MeSH) were employed. Combinations of "periodontitis," "atrial fibrillation," "periodontal inflammation," "systemic inflammation," "endothelial dysfunction," and "oral microbiota" were among the search terms used. To improve the search technique and guarantee specificity, Boolean operators (AND/OR) were used. In order to reflect current clinical understanding, special attention was given to recent findings published between 2013 and 2023, even though older fundamental publications were not omitted.

Randomized or non-randomized interventional trials, observational designs (cross-sectional, cohort, and case-control), systematic reviews and meta-analyses, and experimental research utilizing human or animal models were among the studies that qualified for inclusion. Articles that evaluated the effect of periodontal therapy on cardiovascular or arrhythmia-related outcomes, examined inflammatory or microbial biomarkers pertinent to both conditions, or evaluated the relationship between periodontal disease and AF incidence or prevalence were kept. Publications that lacked clear diagnostic criteria, were published in languages other than English, or were opinion papers, case reports, or non-peer-reviewed abstracts were all excluded.

Design features, sample size and demographics, periodontal diagnostic techniques (for instance, standardized measures of probing depth and clinical attachment level, bleeding indices, or radiographic measures), AF diagnosis techniques (such as electrocardiography, Holter monitoring, or medical records), and mechanistic insights about inflammatory or microbial processes were among the important data taken from each study. Quantitative pooling by meta-analysis was inappropriate due to variations in study designs, demographics, and outcome measurements. Rather, a narrative synthesis was employed to emphasise increasing knowledge gaps, methodological strengths and limits, and common discoveries.

This methodical approach facilitated the evaluation of the data fairly and thoroughly, revealing the potential influence of periodontal inflammation on the risk of AF and highlighting aspects that warrant more investigation.

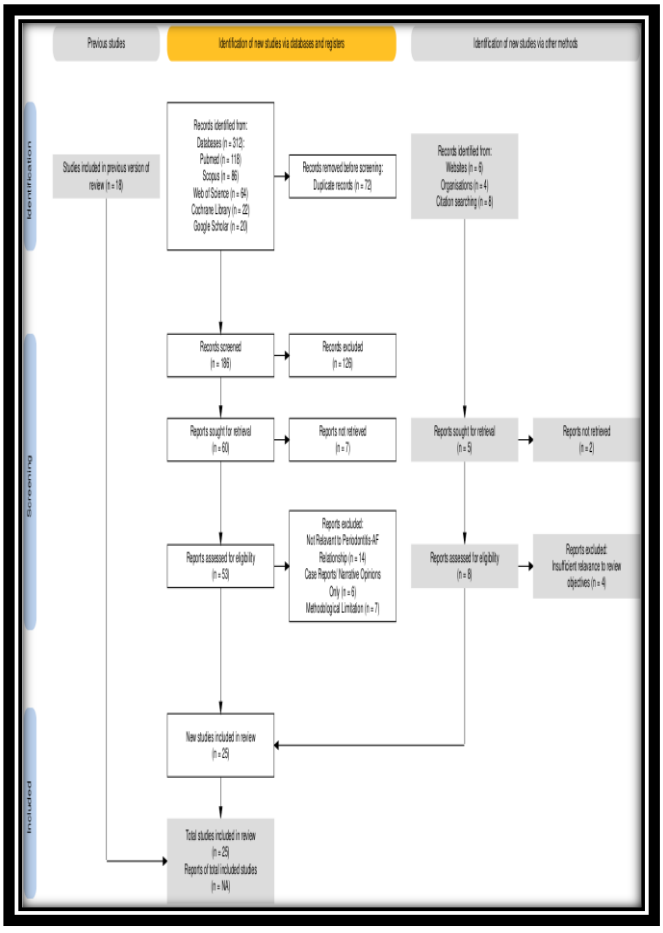

Fig 1: PRISMA 2020 flow diagram depicting the study selection process[7]

RESULTS

The Eligibility for participation was determined based on the following criteria, satisfied by 25 studies published within the period from 2004 to 2024. When taken as a whole, these studies—which ranged from extensive population-based analyses to mechanistic and interventional studies—revealed a number of recurring themes about an emerging connection between periodontal disease and atrial fibrillation (AF).

Evidence from Population-Based Studies

Numerous extensive epidemiological datasets point to a significant correlation between the likelihood of atrial fibrillation onset and periodontal health. Even after adjustment for established cardiovascular risk factors and demographics, a historic Korean cohort of more than 3.7 million persons indicated that periodontal disease was an independent predictor of new-onset AF[8]. Regular professional dental cleanings seemed to lower the risk of developing AF, suggesting a possible preventive function for preserving periodontal health.

Similar patterns were seen in Japanese cohort data, where a higher frequency of AF with time was linked to markers of severe periodontal deterioration, including deep periodontal pockets and considerable clinical attachment loss[9]. These results were confirmed by analyses of Western populations. For instance, the U.S. NHANES dataset indicated that subjects exhibiting moderate to severe periodontal disease had a higher frequency of AF; this association persisted despite adjustment for metabolic abnormalities, smoking, and obesity. Numerous studies conducted in Europe have reported similar findings[10].

But not all the evidence is in agreement. After controlling for coexisting cardiovascular illnesses, the link significantly decreased, according to a thorough nationwide study conducted in Denmark. This observation suggests overlapping risk profiles rather than periodontal disease may independently explain part of the observed relationship[11]. These inconsistent results emphasize the necessity for meticulously regulated studies that more thoroughly take confounding factors into consideration.

Inflammatory Link Between Periodontitis and AF

Accumulating evidence indicates that individuals with chronic periodontitis have a significantly higher inflammatory burden. Reports of elevated systemic concentrations of TNF-α, CRP, IL-6, and IL-1β—biomarkers strongly associated with the development, maintenance, and recurrence of AF—were common. These mediators are known to promote ion-channel dysregulation, extracellular matrix deposition, and fibroblast activation in atrial tissue—processes essential to atrial remodelling[12].

Therefore, a systemic biochemical milieu that is favourable to arrhythmogenesis may be created by persistent periodontal inflammation. One of the main mechanisms maintaining AF, atrial fibrosis, also seems to be accelerated by the persistent release of pro-inflammatory cytokines.

Microbial Translocation and Pathogen-Driven Mechanisms

Microbial translocation has been identified in several investigations as an additional factor involved in the pathogenesis of atrial fibrillation. Periodontal bacteria can enter the bloodstream through routine actions like chewing or brushing. Vascular tissues and atherosclerotic lesions have been shown to harbour Porphyromonas gingivalis, a keystone pathogen in periodontal deterioration. Its virulence agents, including gingipains, can prolong inflammatory reactions and elude immune defences[13].

Microbes like lipopolysaccharides (LPS) trigger strong cytokine production and oxidative stress reactions by activating Toll-like receptors. These mechanisms may exert indirect effects on the atrial substrate by impairing endothelial function and increasing vascular permeability. Periodontal pathogen-infected animal models exhibit extensive systemic inflammation, vascular dysfunction, and inflammatory infiltration of cardiac tissues, shedding light on the underlying mechanisms through which mouth infection may affect alterations related to arrhythmia[14].

Oxidative Stress and Vascular Effects

Another common connection between the two illnesses was found to be oxidative stress. Reactive oxygen species (ROS) are produced excessively in periodontitis due to ongoing microbial assault. Increased ROS levels contribute significantly to the pathogenesis of AF by impairing calcium handling, interfering with normal action potential propagation, and encouraging ectopic atrial activity.

The two conditions are also connected by endothelial dysfunction, a known precursor to cardiovascular disease. People with periodontal disease frequently exhibit diminished bioavailability of nitric oxide, elevated vascular stiffness, and a pro-thrombotic condition. Clinical research shows that periodontal therapy can lessen arterial stiffness and partially restore endothelial function, indicating a reversible aspect linked to better dental health[15].

Immune Dysregulation and Autonomic Mechanisms

Persistent immunological activation, including modified T-cell responses, changes in macrophage phenotype, and increased neutrophil activity, is a hallmark of chronic periodontitis. Autonomic regulatory pathways may be impacted by these immunological changes. Immune-driven autonomic abnormalities may increase arrhythmic vulnerability because autonomic imbalance, especially sympathetic overactivity or vagal oscillations, can trigger AF episodes[16].

Outcomes After Periodontal Therapy

Despite the paucity of interventional evidence, the trials that have since become available offer positive indications. It has been shown that non-surgical periodontal therapy enhances endothelial function and lowers systemic inflammatory indicators like CRP and IL-6. Additionally, some studies show improvements in lipid metabolism and decreases in arterial stiffness after treatment.

Early findings indicated that patients receiving periodontal therapy in addition to conventional care had lower recurrence rates while undergoing catheter ablation for AF. However, these results need to be evaluated carefully because of limited sample sizes and methodological limitations. Nevertheless, the trend shows that increasing periodontal health may have additive cardiovascular advantages[17].

DISCUSSION

The information gathered in this analysis suggests that atrial fibrillation (AF) and chronic periodontal disease demonstrate a meaningful and physiologically reasonable association. The convergence of epidemiological patterns, mechanistic insights, and new interventional data offers a solid foundation for examining periodontal inflammation as a potential contributor to AF pathogenesis, even though causality cannot yet be conclusively demonstrated.

Persons affected by moderate-to-severe periodontitis have a higher likelihood of developing AF, according to population-based studies. Increased AF incidence is correlated with signs of periodontal damage, including deep pockets and clinical attachment loss, according to several large cohorts, especially from the United States[18]. Even when smoking, obesity, and cardiometabolic variables are taken into consideration, this relationship frequently remains. Not all studies agree, though; results from a sizable Danish registry indicate that the association can become weaker when comorbidities are completely taken into account[19]. These disparities demonstrate the impact of population factors, variations in periodontal evaluation, and residual confounding that is still challenging to remove in observational designs.

It is becoming more evident how the two illnesses are connected biologically. Periodontitis represents a chronic inflammatory load that can raise circulating levels of cytokines, including CRP, TNF-α, and IL-6—biomarkers that are frequently linked to the onset and recurrence of AF[20]. By encouraging fibroblast activation, changing ion-channel expression, and upsetting calcium homeostasis, these inflammatory mediators have an impact on atrial electrophysiology. These alterations make it easier for arrhythmias to start and persist. The idea that periodontal inflammation may contribute to atrial remodelling is strengthened by this mechanistic overlap.

Another convincing cause is the spread of periodontal infections and their byproducts. Among other microorganisms, Porphyromonas gingivalis has been discovered in cardiovascular tissues and can enter the bloodstream during regular dental activities. Its virulence factors cause inflammatory cascades, endothelial dysfunction, and oxidative stress, all of which are established causes of atrial structural alterations. This pathogen-driven theory is supported by evidence obtained from animal experimental models that show cardiac infiltration and increased inflammatory signalling after periodontal infection[21].

The relationship is further strengthened by oxidative stress. Elevated reactive oxygen species (ROS) disrupt intracellular signalling and atrial electrical stability in people with periodontal disease. Additionally, endothelial dysfunction caused by ROS creates a vascular milieu that is favourable to arrhythmogenesis. Significantly, several studies demonstrate that periodontal therapy has been shown to attenuate oxidative stress and enhance endothelial function, indicating that at least some of the systemic effects of periodontitis are reversible[22].

Notwithstanding these encouraging discoveries, there are still important restrictions. Concluding causality is limited since most of the evidence that is currently accessible comes from observational studies[23]. Variability is also introduced by several methods that are employed to diagnose periodontitis, from self-reported measurements to thorough periodontal charting[24]. Similar to this, different research uses different methodologies for diagnosing AF, and many studies only use data derived from registries, which could underestimate cases that are asymptomatic or paroxysmal. These methodological differences highlight the necessity for standardized diagnostic criteria in subsequent studies, thereby contributing to conflicting results[25].

Despite their small number, interventional trials indicate that periodontal therapy could be beneficial. Numerous studies have demonstrated improvements in vascular function and decreases in circulating inflammatory biomarkers, which are consistent with biological mechanisms linked to AF. Treating periodontal disease may additionally contribute to the prevention of AF recurrence following catheter ablation, according to a small study. Although intriguing, before periodontal therapy is suggested as part of AF care procedures, these results need to be confirmed in large, well-controlled trials[26].

Given the significant worldwide burden of both AF and periodontitis, comprehending their interaction has important clinical consequences. Periodontal care may play an important role in cardiovascular risk reduction measures if further longitudinal and interventional research confirms a causal relationship. Combining dental and cardiology services could help identify at-risk persons earlier and promote a more comprehensive approach to cardiovascular health.

CONCLUSION

The combined results of mechanistic, epidemiological, and early interventional studies point to a plausible connection between a higher risk of atrial fibrillation and chronic periodontitis. Atrial structural and electrical remodelling may be influenced by overlapping pathophysiological mechanisms, including endothelial dysfunction, oxidative stress, microbial translocation, and persistent oral inflammation. Despite the strength of these correlations, there is still not enough data to prove direct causation. Conclusions are still limited by differences pertaining to the study design, diagnostic techniques, and confounding factor control.

Future investigations should prioritise well-controlled clinical trials to determine whether periodontal therapy can significantly affect AF incidence or recurrence, along with

large-scale, longitudinal studies incorporating standardised definitions for both periodontal and AF assessment. Incorporating periodontal care into cardiovascular prevention frameworks may provide a new, approachable, and adjustable method of lowering the risk of AF if a causal association is established. Therefore, improving cooperation between the cardiac and dentistry fields may be crucial to improving patient care and scientific knowledge.

RELEVANCE OF THE STUDY

Atrial fibrillation and periodontitis are both highly prevalent chronic conditions that impose a significant burden on healthcare systems worldwide. AF is a leading cause of stroke and heart failure, while periodontitis affects a large proportion of the adult population and is often underdiagnosed and undertreated. The public health relevance of this study lies in its exploration of a potential link between these two widespread conditions through shared inflammatory and pathophysiological pathways.

This review underscores the possibility that chronic periodontal disease may contribute to atrial fibrillation risk by increasing systemic inflammation, oxidative stress, and endothelial dysfunction. Given the modifiable nature of periodontal disease, improving oral health at the population level may offer a novel and cost-effective approach to reducing cardiovascular risk. Preventive dental care, early periodontal intervention, and improved public awareness of oral–systemic health links could therefore have broader cardiovascular benefits.

By identifying gaps in current evidence and emphasizing the need for longitudinal and interventional research, this study informs future public health strategies aimed at integrated care. Strengthening collaboration between dental and medical services may support more comprehensive prevention frameworks and help reduce the growing burden of atrial fibrillation and its associated complications.

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