International Journal of Community Medicine and Public Health
Mhaske S et al. Int J Community Med Public Health. 2026 Feb;13(2):1086-1090
http://www.ijcmph.com
pISSN 2394-6032 | eISSN 2394-6040

DOI: https://dx.doi.org/10.18203/2394-6040.ijcmph20260332

Review Article

Bacterial endocarditis and dental infections in the post-COVID-19 era: a review of current concepts

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Received: 01 December 2025
Revised: 22 January 2026
Accepted: 23 January 2026

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ABSTRACT

The COVID-19 pandemic has profoundly impacted global healthcare, leading to shifts in the epidemiology, diagnosis, and management of various infectious diseases. Infective endocarditis (IE), a severe and life-threatening infection of the endocardial surface of the heart, has seen a resurgence in recent years, with oral streptococci remaining a predominant causative agent. This review explores the evolving relationship between dental infections, oral health, and the risk of IE in the post-COVID-19 landscape. We examine the pathophysiological mechanisms linking dental bacteremia to endocardial colonization, emphasizing the role of endothelial damage induced by SARS-CoV-2 as a potential novel risk factor. The pandemic led to significant disruptions in routine dental care, resulting in an accumulation of untreated dental disease and a cohort of patients with advanced oral infections. Concurrently, altered immune responses and the pro-thrombotic, pro-inflammatory state associated with COVID-19 may create a more susceptible environment for bacterial adhesion and vegetation formation. This article synthesizes current evidence on these interactions, discusses updates in antibiotic prophylaxis guidelines, and highlights the critical importance of restoring access to dental care and reinforcing collaborative efforts between cardiologists, infectious disease specialists, and dentists in the post-pandemic period.

Keywords: Infective endocarditis, Dental infection, Oral health, SARS-CoV-2, Post-COVID syndrome, Antibiotic prophylaxis, Bacteremia, Healthcare disruption

INTRODUCTION

Infective endocarditis (IE) is a formidable disease characterized by microbial infection of the endocardium, most commonly the heart valves. Despite advances in diagnostic imaging and antimicrobial therapy, IE continues to be associated with high morbidity and mortality rates, often exceeding 20-30% in modern series.[1] A well-established portal of entry for the pathogens responsible for IE is the oral cavity. Transient bacteremia resulting from daily activities like chewing, tooth brushing, and, more significantly, from dental procedures involving manipulation of gingival tissue or the periapical region of teeth, can seed bacteria onto susceptible cardiac structures.[2]

The global landscape of healthcare was irrevocably altered by the COVID-19 pandemic caused by the SARS-CoV-2 virus. Beyond the acute respiratory illness, the pandemic's secondary effects have been far-reaching. These include the disruption of essential healthcare services, such as routine dental care, leading to a "deferred care backlog" of untreated dental pathologies.[3] Furthermore, emerging evidence suggests that SARS-CoV-2 infection itself can cause endothelial damage (endotheliitis) and a persistent pro-inflammatory state, which may theoretically increase the risk of bacterial superinfection, including IE.[4]

This review aims to synthesize current concepts regarding the interplay between dental infections and bacterial endocarditis in the context of the post-COVID-19 era. It will explore the traditional pathophysiology of IE, analyze the impact of pandemic-related dental care disruption, and investigate the potential mechanisms by which SARS-CoV-2 infection could modulate IE risk. Finally, we will discuss updated clinical guidelines and the imperative for a multidisciplinary approach to prevention and management.

PATHOPHYSIOLOGY: THE ORAL-CARDIAC AXIS OF INFECTION

The development of IE is a complex process that requires the confluence of several factors, elegantly described by Rodbard's classic triad: endothelial damage, bacteremia, and a susceptible host 5 (Figure 1).

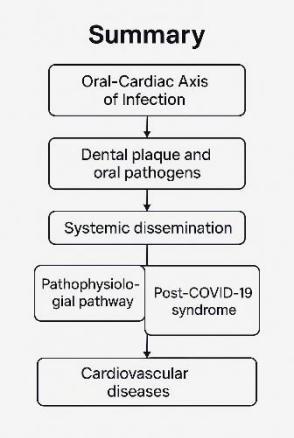

Figure 1: Basic flowchart suggesting role of dental procedures in post COVID causing risk for infective endocarditis.

The role of dental infections and bacteremia

The oral cavity hosts a complex and diverse microbiome that exists in equilibrium with the host under healthy conditions. In the presence of periodontal disease (gingivitis, periodontitis) or periapical infections such as pulpitis and apical abscess, this balance is disrupted, leading to a substantial increase in pathogenic anaerobes and facultative bacteria. Tissue breakdown within the gingival sulcus, periodontal pockets, or periapical region compromises epithelial integrity and creates a direct portal for bacteria to enter the bloodstream.

Different dental procedures release distinct microbial profiles into circulation. Tooth extraction commonly induces bacteremia dominated by *Porphyromonas gingivalis*, *Streptococcus pyogenes*, *Aggregatibacter actinomycetemcomitans*, and *Fusobacterium nucleatum*, organisms strongly associated with deep periodontal and endodontic lesions. Dental cleaning and scaling dislodge supragingival and subgingival plaque flora, including Streptococcus mitis, Neisseria species, Capnocytophaga species, and other Porphyromonas spp. Periodontal therapy introduces highly virulent subgingival anaerobes into the bloodstream, particularly A. actinomycetemcomitans, Tannerella forsythia, Treponema denticola, and Selenomonas spp., well-known contributors to advanced periodontitis pathogenesis.[10-12] Even routine tooth preparation may produce low-grade bacteremia with organisms such as Streptococcus viridans, Prevotella intermedia, Staphylococcus aureus, and Actinomyces spp.

Importantly, significant bacteremia is not limited to invasive procedures; daily activities such as mastication, flossing, and tooth brushing can produce transient bacteremia in patients with inflamed periodontal tissues. Evidence suggests that the magnitude and frequency of bacteremia from daily oral activities cumulatively exceed that associated with single dental procedures.[6] Among all oral microorganisms, the viridans group streptococci, particularly Streptococcus sanguinis, S. oralis, and S. mitis, remain the most frequently implicated pathogens in

infective endocarditis due to their strong affinity for adhering to damaged endocardial surfaces 20. figure 2.

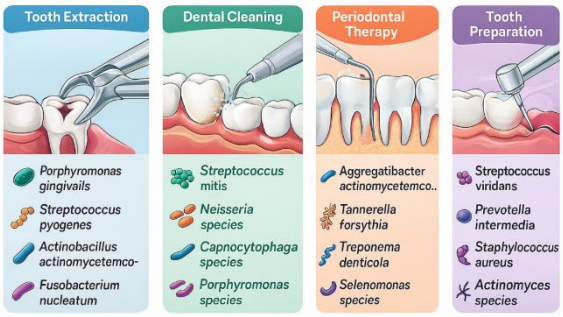

Figure 2: Microbiology in bacteremia involved in different dental procedures.

Schematic diagram for endocardialmicrobial colonization [6,8,11-13]

For IE to establish, the endothelial surface must first be predisposed to bacterial adhesion. Hemodynamic turbulence from congenital or acquired valvular heart disease can cause endothelial microtrauma. At these sites

of injury, a sterile platelet-fibrin thrombus forms. When bacteremia occurs, circulating bacteria can adhere to these thrombi via surface adhesins. Subsequent bacterial proliferation and further deposition of fibrin and platelets lead to the formation of the characteristic lesion of IE: the vegetation.[7] This protected nidus allows bacteria to proliferate to high densities, shielded from host immune defenses, and serves as a source for continuous embolization.

THE POST-COVID-19 LANDSCAPE: COMPOUNDING RISK FACTORS

The COVID-19 pandemic has introduced or exacerbated a confluence of factors that may significantly influence the incidence, etiology, and severity of IE of dental origin. The interplay between a degraded state of population-wide oral health and the unique pathophysiological sequelae of SARS-CoV-2 infection creates a high-risk environment for the development of odontogenic bacteremia and its systemic complications.

Disruption of dental care and the backlog of oral disease

The mandated suspension of non-essential dental services during the pandemic was a critical public health measure to control viral transmission. However, this resulted in a global deferral of routine and elective dental care, creating a substantial backlog of untreated oral disease. The consequences extend far beyond delayed appointments, fundamentally altering the oral microbiome and the host's inflammatory status.

Progression of undiagnosed disease

The absence of routine professional interventions allowed for the unchecked progression of common oral diseases. The progression from early caries to pulpal necrosis involves a shift in the microbial ecology from predominantly streptococcal species to a more complex, anaerobic consortium within the root canal system, including Parvimonas micra and Dialister pneumosintes.[8] The resulting apical periodontitis establishes a protected, hypoxic environment where these bacteria proliferate. The inflamed periapical tissues, rich in neovascularization, provide a direct conduit for these bacteria to enter the circulatory system, even in the absence of a clinical procedure.[9] This state of chronic, low-grade bacteremia can persist for months or years, continuously challenging the host's immune system and providing repeated opportunities for cardiac seeding.

Similarly, the lack of subgingival debridement allowed periodontal biofilms to mature, increasing the proportion of Gram-negative, anaerobic species such as Porphyromonas gingivalis, Treponema denticola, and Fusobacterium nucleatum.[10] The ulcerated epithelial lining of periodontal pockets, a vast wound surface, readily introduces these periodontopathogens into the bloodstream during mastication or tooth brushing.[11] The bacteremia from periodontitis is often polymicrobial, and synergistic interactions between species can enhance their collective pathogenicity and adhesion potential.[12]

Delayed management and amplified procedural bacteremia

The "emergency-only" model of care created a scenario where patients presented with more advanced disease, requiring more invasive procedures. These interventions are associated with a higher incidence and greater magnitude of bacteremia.[13] Furthermore, the microbial ecology in these advanced infections is more diverse and virulent, meaning the ensuing bacteremic shower is composed of more resilient and adhesive bacterial strains.

The systemic inflammatory burden and endothelial priming

Chronic oral infections are a significant source of systemic inflammation, associated with elevated serum levels of C-reactive protein (CRP), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-α).[14] This persistent inflammatory state can upregulate the expression of adhesion molecules such as E-selectin, ICAM-1, and VCAM-1 on the endocardial lining, effectively "priming" the cardiac valves for bacterial adhesion.[15] Concurrently, constant antigenic challenge can lead to immune dysregulation, potentially impairing the clearance of transient bacteremias.[16]

SARS-COV-2 and endothelial dysfunction: a novel predisposition

A key pathophysiological feature of severe COVID-19 is widespread endothelial injury. SARS-CoV-2 virus binds to angiotensin-converting enzyme 2 (ACE2) receptors on endothelial cells, leading to endotheliitis, microthrombosis, and a profound pro-inflammatory "cytokine storm".[4] This virus-induced endothelial damage could theoretically create the non-bacterial thrombotic endocardial lesions that serve as the nidus for bacterial adhesion in individuals who otherwise lack traditional high-risk cardiac conditions.[17] While a direct causal link requires further validation, several case reports have documented IE in patients with recent COVID-19 and no prior known heart valve disease, suggesting this is a plausible and concerning phenomenon.[19]

Immune modulation in post-COVID syndrome

Post-COVID-19 syndrome, or "Long COVID," is often characterized by dysregulation of the immune system, including T-cell exhaustion and persistent inflammation.[19] This state of immune dysregulation could potentially impair the host's ability to clear transient bacteremias, allowing a higher inoculum of bacteria to reach and adhere to a susceptible endocardial site.

CURRENT CONCEPTS IN PREVENTION AND MANAGEMENT

In light of these evolving risks, adherence to and reinforcement of established preventive strategies is paramount.

Antibiotic prophylaxis: updated guidelines and rationale

The role of antibiotic prophylaxis (AP) for the prevention of IE remains a focused topic. Current guidelines from major professional bodies, such as the American Heart Association (AHA) and the European Society of Cardiology (ESC), have significantly narrowed the indications for AP. The consensus is that AP should be reserved only for patients with the highest risk of adverse outcomes from IE undergoing highest risk dental procedures.[20,21]

Highest-risk cardiac conditions include prosthetic cardiac valves, previous history of IE, unrepaired cyanotic congenital heart disease, and cardiac transplant recipients with valvulopathy.

Highest-risk dental procedures are those that involve manipulation of the gingival tissue or the periapical region of teeth, or perforation of the oral mucosa (e.g., extractions, periodontal surgery) (Table 1).

Table 1: Dental conditions and their pathophysiological contribution to increased risk of infective endocarditis.

Dental condition	Pathophysiological consequence of disruption	Mechanism of increased ie risk	Source of study findings
Dental caries and pulpal necrosis	Maturation of a protected, anaerobic biofilm within the root canal system. Shift to a more virulent microbial consortium.	Establishes a chronic, low-grade bacteremia from the inflamed periapical tissues. Invasive treatment causes a high-magnitude bacteremia.	Siqueira et al; Cope et al[8,9]
Periapical abscess	Delayed drainage leads to local tissue destruction and potential fascial space involvement.	Creates a high-density bacterial focus under pressure, leading to spontaneous or procedure-induced septic embolization.	Cope et al; Parahitiyawa et al[9,12]
Chronic periodontitis	Increased pocket depth, clinical attachment loss, and biofilm pathogenicity ("dysbiosis"). Ulceration of the pocket epithelium.	Expands the surface area of the port of entry, facilitating frequent bacteremia from daily activities. Introduces highly adhesive, proteolytic pathogens.	Offenbacher; Lockhart et al; Hajishengallis[6,10,11]
Acute gingivitis/periodontitis	Unchecked inflammation leads to rapid tissue breakdown and increased vascular permeability.	Amplifies the bacteremic response to even minor trauma (e.g., chewing). The acute inflammatory milieu can transiently suppress local immune surveillance.	Offenbacher; Lockhart et al[6,11]

It is critical to emphasize that the focus has shifted from AP to the maintenance of excellent oral health as the primary preventive strategy. The risk of bacteremia from daily activities over time far exceeds that from a single dental procedure. Therefore, preventing dental diseases that cause chronic bacteremia is more impactful than prophylactic antibiotics for procedures.[20]

The imperative of oral hygiene and interprofessional collaboration

The post-COVID-19 era demands a renewed focus on restoring and maintaining oral health.

Public health initiatives: Efforts must be made to clear the backlog of dental care, with prioritization for high-risk cardiac patients.

Patient education: Patients, especially those with cardiac conditions, must be educated on the vital link between oral and systemic health. Consistent, effective oral hygiene practices and regular professional dental cleanings are the cornerstone of IE prevention.

Interprofessional collaboration: Cardiologists and primary care physicians should routinely inquire about their patients' oral health and dental care access. Dentists must be vigilant in taking comprehensive medical histories, including COVID-19 history and cardiac status. This collaborative model is essential for risk stratification and timely intervention.

CONCLUSION

The confluence of the COVID-19 pandemic has created a "perfect storm" of factors that may elevate the risk of bacterial endocarditis stemming from dental origins. The massive disruption in routine dental care has led to a higher prevalence of severe oral infections, while the biological sequelae of SARS-CoV-2 infection itself—

endothelial damage and immune dysregulation—may have created a more susceptible host population. This synergy between an enhanced microbial insult and a primed cardiovascular system necessitates a heightened clinical awareness.

Moving forward, a multi-pronged approach is essential: robust epidemiological surveillance to track IE trends, mechanistic research into the long-term effects of SARS-CoV-2, and unwavering clinical vigilance. Ultimately, strengthening the collaboration between medical and dental professions and reinforcing the principle of optimal oral health as a cornerstone of systemic well-being are the most critical strategies to mitigate the enduring and evolving threat of infective endocarditis in the post-pandemic world.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

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Cite this article as: Mhaske S, Ahmed SA, Babu A, Bakr MA, Magwa K. Bacterial endocarditis and dental infections in the post-COVID-19 era: a review of current concepts. Int J Community Med Public Health 2026;13:1086-90.