

Oral Infections and Cardiovascular Disease: An Evolving Perspective

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Abstract

Oral infections and cardiovascular disease (CVD) are interconnected conditions. The oral cavity harbors a diverse microbiome, and dysbiosis can lead to systemic inflammation, which is a key factor in the development of atherosclerosis. This review discusses the pathophysiological mechanisms linking oral infections to CVD, highlighting the role of periodontal disease and the oral-systemic connection. It also explores the evolving perspective on the infection hypothesis and the implications for clinical practice.

Introduction

Periodontal disease (PD) is an inflammatory disease primarily

Cardiovascular disease (CVD) remains a leading cause of mortality

hypothesis were abandoned for a long period in the nineteenth century. Osler, often cited as the first to postulate the causal role of the infection hypothesis in arteriosclerosis, wrote in 1908 about the existence of four great factors in the causation of arteriosclerosis - normal wear and tear of life, the acute infections, the intoxications, and those combinations of circumstances that keep blood tension high [4]. Osler described fatty streaks in atheroma as well as their high prevalence in children. It was evident that the authors distinguished between other types of aortitis, as seen in conditions such as syphilis.

Periodontal disease and inflammation

Periodontal disease is characterized by the destruction of the gums, periodontal ligament, and alveolar bone. It results from the long-term accumulation of dental plaque and the subsequent inflammatory response triggered by bacteria present in the biofilm [5]. Chronic inflammation releases various pro-inflammatory mediators, such as cytokines and acute-phase reactants, which can enter the bloodstream and exert systemic effects.

Inflammation and atherosclerosis

Atherosclerosis, the buildup of fatty plaques within the arteries, is a key underlying cause of cardiovascular disease. Chronic inflammation plays a pivotal role in the initiation and progression of atherosclerosis. Inflammatory mediators released from periodontal infections can contribute to endothelial dysfunction, leading to the disruption of the arterial wall and the infiltration of immune cells [6]. This sets the stage

The oral-systemic connection

Oral health is not limited to the mouth alone; it has been increasingly

The infection hypothesis

The infection hypothesis was introduced in 1823 by Rayer, who

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for the formation of atherosclerotic plaques.

Bacterial translocation and endothelial dysfunction

One proposed mechanism linking oral infection to cardiovascular disease is the translocation of oral bacteria into the bloodstream. Several studies have detected oral pathogens, such as Porphyromonas gingivalis and Streptococcus mutans, within atherosclerotic plaques.
