Cardiovascular Disease and Atherosclerosis

Atherosclerosis, the thickening and hardening of arteries produced by a build-up of plaque, is the underlying cause of cardiovascular disease (CVD). It is essentially an inflammatory disease, whereby an initial lesion, in response to injury to the endothelium of elastic and muscular arterial tissue, leads to a complex chronic inflammatory process. There is accumulating evidence of a role for infectious agents in atherogenesis; by causing endothelial injury, they may, in part, trigger the inflammatory response. The levels of inflammatory mediators in the systemic circulation, such as C-reactive protein (CRP) and fibrinogen, are indicators of a general inflammatory response and atherosclerosis. This link between inflammation and atherosclerosis suggests that chronic infections, such as oral infections from periodontal disease, may predispose to cardiovascular disease.

Significant similarities in the pathogenesis of atherosclerosis and periodontitis have suggested a common underlying biological mechanism for the two conditions. Based on this paradigm, several studies have investigated the relationship between periodontitis and cardiovascular disease.

Indirect Evidence: Epidemiological Studies

Most of the evidence supporting a relationship between periodontal disease and CVD comes from epidemiological studies. In the late 1980s, pioneer work showed that patients who had a history of myocardial infarction (MI) generally had worse oral health than control subjects. Subsequently, cross-sectional data from the Third National Health and Nutrition Survey (NHANES III) indicated that patients with severe clinical attachment loss were at greater risk for MI than subjects with a healthy periodontium (odds ratio: 3.8). Since then, systematic literature reviews have indicated that most studies report a modest association between periodontal disease and CVD, between a 1.3 and 2-fold increase in the risk of CVD in people with periodontitis.

Conversely, treatment of periodontitis was shown to decrease serum concentration of CRP, interleukin (IL)-6 and tumor necrosis factor (TNF)-α, indicating that infection of the periodontium can influence systemic conditions. What remains unclear from these studies, however, is whether periodontitis can predispose to atherosclerosis.

Direct Evidence: Experimental Studies

Direct evidence for the role of oral infection in predisposing to atherosclerosis comes from several lines. The presence of predominant oral pathogens such as Porphyromonas gingivalis, Tannerella forsythensis, and Prevotella intermedia was detected in atherosclerotic plaque, suggesting a possible invasion of atheromas by oral pathogens. In addition, P. gingivalis can invade endothelial cells and can also induce platelet aggregation, a key process in atheroma and thrombus formation. Whether these pathogens actively contribute to the development of atheroma, however, remains to be established.

Most of the experimental evidence supporting a relationship between CVD and periodontal disease comes from animal model studies. Using apolipoprotein E-deficient mice, research has shown that clinically induced bacteremia or oral infection with P. gingivalis increase atheroma size compared to non-infected controls.
injected with host antigens, enhancing the effects of some bacterial and human proteins (e.g., heat-shock protein HSPE60) raises the possibility that antibodies against bacterial versions of the protein may cross-react with the human protein, inducing an autoimmune response. In the case of HSPE60, disruption of arterial endothelial cells is thought to stimulate atherosclerosis. Proinflammatory response may also be enhanced by cross-reactive epitopes that stimulate T-cell response reactive with host antigens, enhancing the effects of bacterial pathogens on cardiovascular health.

of periodontitis. This is due to its 12-hour antibacterial action coupled with its ability to directly inhibit potent inflammatory mediators. Control of periodontal infection and inflammation will improve the oral health of patients, decrease the systemic chronic inflammation burden caused by oral inflammation, improve general health, and may ultimately contribute to the reduction of cardiovascular disease.

Establishing a direct link between periodontal disease and CVD is very difficult due to the multifactorial nature of both diseases with numerous confounding risk factors common to the two. Nevertheless, epidemiological observations coupled with the biological plausibility of this association between two inflammatory diseases warrant the need for further investigation and validation of the cardiovascular-periodontal link.

Management and Prevention

Oral inflammation and periodontal disease are generally chronic and can persist asymptomatically for many years in the absence of appropriate treatment. This results in chronic exposure to local and systemic inflammation, which may induce or enhance already existing inflammatory disease, including atherosclerosis. For this reason, appropriate oral preventive care is important not only to preserve oral health, but also systemic health. Management and prevention strategies must sensitize both dental care providers and patients to the importance of good oral health on systemic burden and chronic diseases.


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