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CARDIOVASCULAR DISEASE A HALL MARK RISK FOR PERIODONTAL DISEASE – A VIEW

DR PREETHI
MDS, READER, DEPARTMENT OF PERIODONTICS, SREE BALAJI DENTAL COLLEGE & HOSPITAL

DR. BHUVANESWARRI
MDS, PROFESSOR, DEPT OF PERIODONTICS, SREE BALAJI DENTAL COLLEGE & HOSPITAL

DR RAMYA.V
MDS, PROFESSOR, DEPT OF PERIODONTICS, SREE BALAJI DENTAL COLLEGE & HOSPITAL

GAAYATHRI K R
SAVEETHA MEDICAL COLLEGE, SAVEETHA INSTITUTE OF MEDICAL AND TECHNICAL SCIENCES

Abstract
Periodontal disease is a multi factorial disease that affects the gingiva and the tooth supporting structures Models of oral infections have become valuable resources for researching the idea that infection is a risk factor for cardiovascular disease (CVD)[1]. Research has indicated a link between periodontal disease and cardiovascular disease (CVD), identifying periodontal infections as a significant contributing factor . However, still evidence supporting this remains unclear and studies are emerging to prove this correlation. Periodontal disease and cardiovascular disease are related. Nevertheless, it remains uncertain whether this relationship is merely coincidental or if it is indeed causal.

Summary
In many instances, earlier studies replaced vague clinical and radiographic criteria for periodontal disease with measures of infectious exposure. However, not all research demonstrates a positive correlation between cardiovascular disease and periodontal disease. Recent studies have enhanced the precision of definitions related to infectious exposure through the evaluation of oral microbiota directly from subgingival dental plaque or by evaluating systemic antibodies to specific periodontal pathogens.Some results has shown the positive correlation between periodontal disease andCVD. There is still evidence linking vascular disease, atherosclerosis, and periodontal diseases. Large-scale clinical intervention study design may be influenced by ongoing observational and targeted pilot intervention studies.

Keywords: Cardiovascular Disease , Periodontitis ,oral microbiota, subgingival plaque, Heat shock protein

The impact of periodontitis on the occurrence of cardiovascular events

Research has shown that periodontitis elevates the likelihood of a first acute cardiovascular disease (ACVD) event, such as a heart attack or stroke, independent of other recognized cardiovascular risk factors. The extent of the increased risk is influenced by the type of ACVD event, along with factors such as age and gender. For example, periodontitis presents a greater supplementary risk for cerebrovascular disease compared to coronary heart disease.

Additionally, it presents a heightened risk for younger individuals and males, as research suggests that there is no increased risk for individuals aged 65 and older.[2] Nevertheless, the established prevalence clearly indicates that a low to moderate increase in risk has significant implications for public health. In particular, after evaluating the impact of various risk factors such as age, gender, diabetes, blood pressure, cholesterol levels, obesity, smoking habits, dietary practices, race/ethnicity, educational background, and socioeconomic status, most studies identified positive associations.[3,4]Furthermore, studies that adjusted for diabetic status revealed an increased incidence of periodontitis in individuals who had never smoked. However, it's feasible that unidentified hereditary variables could affect both of these inflammatory disorders and account for the correlation discovered in clinical research.

Is there a connection between periodontitis and acute cardiovascular disease?

A highly credible explanation for the role of periodontitis in the development of atherosclerotic cardiovascular disease (ACVD) is that endotoxins and various byproducts produced by periodontal bacteria infiltrate the bloodstream, triggering an acute phase response and eliciting an inflammatory reaction .

Atheroma, or fatty lesions in the arteries, is then encouraged to form, mature, and become unstable by mediators created as part of this host response, raising the possibility of an ACVD adverse event . Research indicates that the state of a patient's periodontal health affects the likelihood of periodontal bacteria entering the bloodstream (bacteraemia) during activities such as tooth brushing, flossing, or scaling. A highly credible explanation for the role of periodontitis in the development of atherosclerotic cardiovascular disease (ACVD) is that endotoxins and various byproducts produced by periodontal bacteria infiltrate the bloodstream, triggering an acute phase response and eliciting an inflammatory reaction.

Periodontitis may also elevate the risk of atherosclerotic cardiovascular disease (ACVD) through the generation of antibodies in response to plaque bacteria. This immune response can promote the formation of atheroma by interacting with blood lipids and endothelial cells within the arterial lining. Periodontal disease has the potential to induce inflammation in the periodontal tissues and also generates inflammatory mediators and products that may adversely affect cardiovascular health via the bloodstream[5].

Systemic mediators of inflammation: The second mechanism pertains to an intensified host reaction to proinflammatory mediators such as PGE2, TNF-α, and IL-1β, which may arise from the influence of lipopolysaccharide (LPS) or changes induced by microbial activity [6]. These mediators are associated with distinct differences in T cell receptor variations and the secretory functions of monocytes. Individuals exhibiting a hyperinflammatory monocyte phenotype typically release 3 to 10 times the amount of peripheral blood monocytes compared to those with a standard monocyte phenotype . Genes that govern the response of T cells and monocytes, as well as the host-microbe environment, can directly initiate and modulate the inflammatory response. Individuals suffering from periodontal disease exhibit a hyperinflammatory phenotype in their monocytes[7].

The connection between periodontitis' inflammatory and bacterial byproducts and cardiovascular disease may be the third mechanism. Periodontal bacteria can emit lipopolysaccharides (LPS) that can penetrate serum and induce bacteremia, or they can invade endothelium directly and cause atherosclerosis[8]. LPS has the ability to cause inflammatory cells to accumulate on major blood arteries, as well as to promote blood thrombocyte function, vascular lipid degeneration, vascular muscle degeneration, and intravascular coagulation. Biological mediators include PGs, ILs, and TNF-α that are activated in smooth muscle cause these alterations[9]. Furthermore, it has been demonstrated that the presence of LPS enhances the sensitivity of endothelial cells to P. gingivalis.

Ghorbani et al. reported that lipopolysaccharides (LPS) originating from P. gingivalis contribute to endothelial dysfunction and enhance the contractility of coronary arteries[9]. Recent studies have shown that the extent of bacteremia following scaling is significantly greater in patients with periodontitis compared to those with gingivitis or healthy controls[10] . Common periodontal infections from arterial plaques have been found through studies of carotid endarterectomy samples[11]. Oral microorganisms possess the capability to directly influence subclinical factors associated with cardiovascular events, including hypercoagulability and the progression of atherosclerosis, by gaining systemic access . An investigation on mice revealed that P. gingivalis administered intravenously speeds up the development of atherosclerosis.

Lalla and her team used oral inoculation with P. gingivalis to produce periodontal infection. They were then able to collect P. gingivalis DNA from the aorta tissue of infected animals exclusively and saw evidence of accelerated early atherosclerosis in infected mice[12]. Giacona et al. discovered that some strains of Porphyromonas gingivalis can infect macrophages and promote foam cell production in the arterial wall, supporting the idea that P. gingivalis can either start or worsen the atherosclerotic process[13].

Indirect pathways: Strong inflammatory factors play a role in atherosclerosis, and epidemiologic data indicates that elevated systemic inflammatory levels may be a predictor of cardiovascular events[14]. Individuals suffering from periodontal disease exhibit increased levels of inflammatory markers in their systems, including C-Reactive Protein. Research indicates that addressing periodontal disease can lead to a reduction in systemic inflammation levels[15]. A variety of factors may contribute to this increased inflammatory response throughout the body, including temporary bacteremias and the localized synthesis of bacterial components such as lipopolysaccharide [16]. In studies, the relationship between P. gingivalis and A. actinomycetemcomitans was found[17]. A correlation between Aggregatibacter actinomycetemcomitans and periodontal pathogen density was found by Spahr et al. and Andriankaja et al[18,19]. According to this study, people with acute coronary disease had greater IgA levels against A. actinomycetemcomitans than people without cardiovascular disease. A. actinomycetemcomitans's surface materials increase the release of proinflammatory cytokines like TNF-α and IL-6 when it is incubated with whole blood cells . This shows that, in addition to allowing bacteria to survive in a variety of human tissues, broken bacterial fragments can also have proinflammatory effects[20].

HEAT SHOCK PROTEIN IN PERIODONTAL INFLAMMATION: A group of related proteins, known as heat shock proteins (HSPs) is Produced by cells in reaction to stressful situations. These proteins' expression is swiftly increased during stressful situations to protect the cell from many forms of harm. Periodontal pockets' basal layer demonstrates Affirmative representation of Highly Sensitive Persons (HSPs), greater mononuclear inflammatory cell infiltration in periodontal pockets under the basal layer. Consequently, bacteria associated with periodontal disease stimulate the production of heat shock proteins (HSPs) in periodontal cells, leading to the activation of macrophages and other inflammatory cells that generate pro-inflammatory cytokines [21]. The periodontal tissue expresses a range of proteins and responds to different stimuli, such as inflammation and mechanical stress, to maintain homeostasis. This leads to the dynamic restructuring of the periodontium.

A traumatic occlusal stress fracture caused the periodontal connective tissue to change. Many systems and organizations are known to exhibit heat-shock proteins (HSPs) as their main protein. HSPs react to force applied mechanically. HSP is influenced by a number of factors, including inflammation, chemical stress, physical stress, and a pathological change in heat shock. Still, it's unclear what each HSP accomplishes. HSP is essential for periodontium remodeling and cellular defense[22]. It is believed that host and bacterial HSPs play a role in the pathophysiology of periodontal disease. The possible connection between periodontitis and circulating HSP levels has not been thoroughly studied. Pro- and anti-inflammatory cytokines are comparable in their qualities to HSP, which regulates intercellular signaling activities[23]. HSP are implicated in periodontal disease. Future research should be directed to estimate the levels of HSP in health and in periodontal disease

CONCLUSION

Research indicates a connection between cardiovascular disease and periodontal disease. Both conditions share common increases in inflammatory mediators, lipids, hemostatic and thrombotic factors, as well as other systemic inflammatory pathways. Additionally, they exhibit several shared risk factors, such as genetic predisposition and tobacco use . It is yet unclear how much PD affects the development and course of ACVD, and more research is necessary to determine this. Periodontal pathogens have the ability to invade distant tissues and induce bacteremia, according to studies conducted on microbes. Epidemiological studies indicate that the odds ratio for atherosclerotic disease is higher in patients with Parkinson's disease compared to those without the condition . Alongside longitudinal studies and prolonged follow-up, additional research should be conducted both in vivo and in vitro to determine the causal relationship between Parkinson's Disease (PD) and Atherosclerotic Cardiovascular Disease (ACVD). This would help to give strong confirmation for the relationship and elucidate its connection.

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