

Periodontitis And Cerebrovascular Disease

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Abstract: Systemic health is often associated with the condition of the oral cavity because many systemic diseases manifest in the mouth. Effects of systemic disorders on periodontal diseases are well established. Periodontal disease affects many systemic diseases like cardiovascular diseases, diabetes, pre-term low birth weight babies, preclampsia, respiratory infections, cancer, rheumatoid arthritis, gastrointestinal disease, osteoporosis, renal disease, prostatitis, alzheimer's disease and cerebrovascular disease. With an increasing body of epidemiological and experimental work, specific risk factors and risk indicators for periodontitis such as tobacco smoking, demographic factors, and socio-economic status have been identified and acknowledged. Any changes to the systemic health because of any of these factors can be seen as a change in periodontal health. So, any changes in the systemic health can definitely affect the periodontal health and vice versa. Periodontitis patients are susceptible to cerebrovascular diseases and many other diseases. This review article mainly highlights the connection between periodontitis and cerebrovascular disease.

Keywords: Cerebrovascular disease, Focal infection, Periodontal pockets, Periodontitis, Systemic disorders

I. Introduction

Periodontitis is a disease of inflammation and infection of the periodontal ligaments and alveolar bone that supports the tooth structure. Periodontitis results from complex interplay between chronic bacterial infections and the inflammatory host response leading to irreversible destruction of tooth structure causing loss of the tooth. [2] This disease harbors large numbers of anaerobic gram negative pathogens, including Porphyromonas Gingivalis and Aggregatibacter Actinomycetemcomitans. Periodontal disease can affect a person's susceptibility for systemic diseases in many ways, shared risk factors like smoking, stress, aging, race or ethnicity and gender. The dynamics of periodontium are a product of its circulation, hormonal changes and mechanism of immune responses. [3]

Cerebrovascular disease is a disease of blood vessels that supplies the brain. Cerebrovascular disease can mainly be divided into embolism and aneurysm. Most important risk factors that can cause cerebrovascular diseases are smoking, demographic factors, socio-economic status. Causes of cerebrovascular disease can be periodontitis, [2] obesity, diabetes, hypertension and etc.

PERIODONTIUM

Periodontium is a reservoir of cytokines like tumor necrosis factor- α , interleukin-1 β , and gamma interferon. So, a spillover of these mediators can enter the circulation and induce systemic diseases, such as cerebrovascular disease. [3] Periodontitis happens when there is inflammation and infection of the periodontal ligaments and alveolar bone that supports the tooth structure., harmful oral bacteria freely enters the blood stream and forms plaque and fatty deposits in blood vessels and travels throughout the organs of the body causing pathological manifestation. [4]

PERIODONTITIS AND CEREBROVASCULAR DISEASE

Bacteria that initiates periodontal disease are called periopathogens. most common bacteria that is seen in periodontitis are p.gingivalis and a.actinomycetemcomitans. Highly vascularized and ulcerated tissues of the oral cavity are highly susceptible to invasion of periopathogens which in order will enter the blood stream and causes a condition called bacteremia. Bacteria can induce thrombus formation by platelet aggregation degrading collagen. Bacteria can enter the blood stream and organs through a few pathways and cause focal infection. [3]

PATHWAYS CAUSING FOCAL INFECTION

A) Metastatic infection from oral cavity due to bacteremia

Often, bacteremias are short lived and transient, highest intensity would be the first 30 mins after the trigger episode. they are dealt swiftly by body defenses. This may lead to spreading of the organism to different target organs like kidney, liver and brain causing subclinical, acute and chronic infections which can cause many critical conditions.

B)Metastatic injury caused by oral microbial toxins

The pathogenicity of a bacteria depends upon the structural component of the cells for example, capsules, fimbria, lipopolysaccharides or active secretion of the substances, where the microorganism damage the host tissues or protects the bacteria against host defenses. These bacterial toxins are carried by the vascular or lymphatic system thereby causing cytotoxic effects at tissue sites causing a large number of pathological manifestation.

C)Metastatic inflammation due to immunological injury caused by microorganisms in the oral cavity

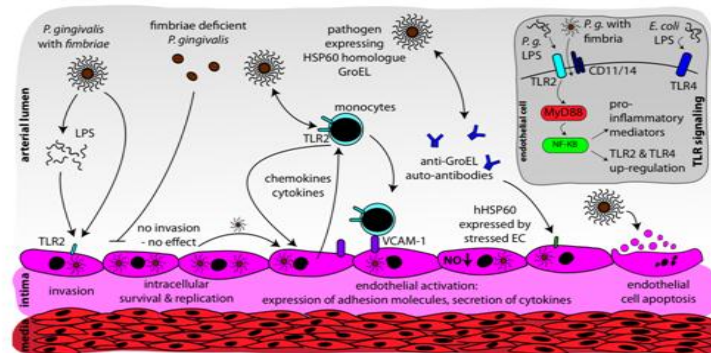
The microorganisms enters the blood instantly during bacteremia and react with the antibodies and form complexes to cause many acute and chronic inflammatory reactions where they are deposited.

PERIODONTAL POCKETS

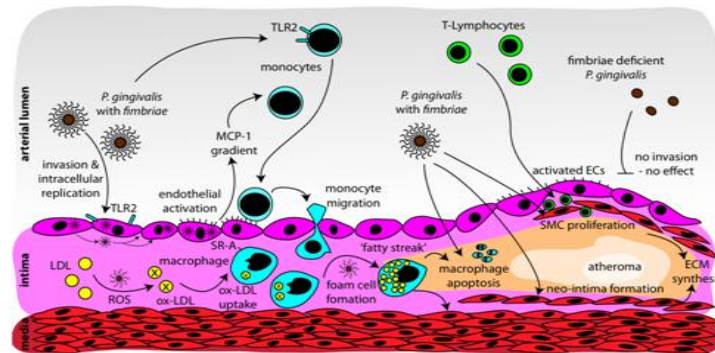
Periodontal pockets are pathologically deepened gingival sulcus. Bacteria from the periodontal pockets can enter the bloodstream during activities like mastication and brushing teeth. Infections and low grade inflammatory processes influence the development of atherosclerosis and ischaemic lesions.[5] Dental sites with deep pockets harbor a large number of bacteria with a positive correlation between pocket probing depth and bacterial levels during ischemic stroke in the present study. A greater incidence of ischemic stroke was previously observed in young patients with periodontal disease, particularly in those with decreased numbers of remaining teeth.[6]

HEMORRHAGIC CEREBROVASCULAR EVENT

Another mechanism for atherogenesis is the induction of immunological processes leading to increased levels of C-reactive proteins(CRP) and increases in CRP even within the range of normal values is considered a reliable predictor of cardiovascular disease. In present studies, 13 out of 20 patients develop ischaemic stroke linked to periodontitis developing a hemorrhagic cerebrovascular event.[3]



Schematic overview of potential mechanisms linking periodontal infections and endothelial dysfunction/incipient atherosclerosis. Vascular endothelial cells are invaded by fimbriated pathogens, e.g., *P. gingivalis*. These pathogens can persist and multiply intracellularly. Activation of Toll-like receptor 2 (TLR2) by fimbriated bacteria or LPS results in release of pro-inflammatory mediators and up-regulation of cell adhesion molecules. Monocytes are recruited by a gradient of chemotactic cytokines, such as MCP-1. Antibodies against bacterial heat-shock proteins, such as HSP60-related GroEL, auto-react with mammalian HSP60 expressed by activated endothelium, resulting in cell destruction. Further, *P. gingivalis* induces apoptosis of endothelial cells.



Potential mechanisms linking periodontal infections and fatty-streak formation/plaque maturation. Monocytes activated by periodontal pathogens chemotactically migrate into the sub-endothelial space, and transform into macrophages and, subsequently, into foam cells after uptake of oxidized LDL. Apoptosis of LDL-laden macrophages results in accumulation of lipids in the sub-endothelial space. Furthermore, periodontal pathogens induce smooth-muscle-cell proliferation in the intima and neo-intima formation. Extracellular matrix build-up and extravasation of T cells consummate the formation of a fibrous cap covering the plaque.

II. Conclusion

Periodontal disease is a paradigm of chronic infection in dental pathology which can lead to many systemic manifestation,likecerebrovascular disease. Poor oral hygiene, many other shared factors, like smoking, diabetes, genetic factors combined, can lead to systemic disorders. Oral cavity is one system which is interconnected with the whole body. A healthy oral cavity prevents from numerous systemic condition.

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