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Role of Periodontal Disease in Coronary Heart Disease and Atherosclerosis- A Review

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ABSTRACT:CHD-related events are a major cause of death. MI has beenassociated with acute systemic bacterial and viral infections and issometimes preceded by influenza-like symptoms.[1]Traditional riskfactors such as smoking, hypertension, and diabetesmellitus do not explain the presence of coronary atherosclerosis in alarge number of patients.[2] Localized infection that results in a chronicinflammatory reaction has been suggested as a mechanism underlyingCHD in these individuals.

Keywords: Hypertension, Diabetes mellitus, Blood vessels, Microorganisms

I. INTRODUCTION: -

In cross-sectional studies of patients with acute MI or confirmedCHD who were compared with age- and gender-matched controlpatients, patients with MI had significantly worse dental health (e.g.,periodontitis, periapical lesions, caries, pericoronitis) than controls.[3]This association between poor dental health and MI was independentof known risk factors for heart disease, such as age, cholesterol levels,hypertension, diabetes, and smoking.[4] Because atherosclerosis is amajor determinant of CHD-related events, dental health has also beenrelated to coronary atheromatosis. There is evidence that the extent ofperiodontal disease may be associated with CHD.[5] For example, theremay be a greater risk for CHD-related events, such as MI, in subjectswho have periodontitis affecting a greater number of teeth in themouth compared with those who have periodontitis involving fewerteeth.

II. MATERIAL AND METHODS:

Effects of periodontal infection

There are numerous mechanisms—both direct and indirect—throughwhich periodontal infection may affect the onset or progression ofatherosclerosis and CHD.[6] Periodontitis and atherosclerosis both havecomplex etiologic factors that combine genetic and environmentalinfluences. In addition to smoking, the diseases share many riskfactors and have distinct similarities with regard to their basicpathogenic mechanisms.

III. DISCUSSION:-

Ischemic heart disease

Ischemic heart disease is associated with the processes ofatherogenesis and thrombogenesis. Damage to the vascularendothelium, with a subsequent inflammatory reaction, plays a major role in atherosclerosis and ischemic organ damage.[7] Increased viscosityof blood may promote major ischemic heart disease andcerebrovascular accident (stroke) by increasing the risk of thrombus formation. Fibrinogen is a major factor in the promotion of thishypercoagulable state. Fibrinogen is the precursor to fibrin, andincreased fibrinogen levels increase blood viscosity. Increased plasma fibrinogen is a recognized risk factor for cardiovascular events andperipheral vascular disease.[8] An elevated white blood cell count is alsoa predictor of heart disease and stroke, and circulating leukocytes maypromote the occlusion of blood vessels. Coagulation factor VIII (Von Will brand factor) has likewise been associated with a risk of ischemicheart disease.

Systemic infections

Systemic infections are known to induce a hypercoagulable state andincrease blood viscosity (Fig. 24.1).[9] Fibrinogen levels and white bloodcell counts are often increased in patients with periodontal disease.[10]Individuals with poor oral health may also have significant elevationsin coagulation factor VIII/Von Will brand factor antigen, therebyincreasing the risk of thrombus formation.[11] Thus, periodontal infectionmay also promote increased blood viscosity and thrombogenesis,which leads to an increased risk for central and peripheral vasculardisease.

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Atherosclerosis

Atherosclerosis is a focal thickening of the arterial intima, theinnermost layer lining the vessel lumen, and the media, the thick layerunder the intima that consists of smooth muscle, collagen, and elastic fiber.[12] The formation of atherosclerotic plaques isprecipitated by damage to vascular endothelium that results in aninflammatory response in which circulating monocytes adhere to thevascular endothelium.[13] Damage to vascular endothelium can occurbecause of the presence of intravascular microorganisms and theirproducts; chemical damage, often resulting from elements of tobaccoand other exogenous toxins; and increased shear force along thevascular lining, such as that occurring in hypertension.[14] The adherenceof monocytes to the damaged vascular endothelium is mediated byseveral adhesion molecules on the endothelial cell surface, includingintercellular adhesion molecule-1 (ICAM-1), endothelial leukocyteadhesion molecule-1 (ELAM-1), and vascular cell adhesion molecule-1(VCAM-1). These adhesion molecules are upregulated by a number offactors, including bacterial LPSs, prostaglandins, andproinflammatory cytokines.[15] After binding to the endothelial celllining, monocytes penetrate the endothelium and migrate under thearterial intima.[16] The monocytes ingest circulating low-densitylipoprotein in its oxidized state and become engorged, therebyforming the foam cells that are characteristic of athermanous plaque.

IV. CONCLUSION:

Athermanous plaque formation and thickening of the vessel wallnarrow the lumen and dramatically decrease blood flow through thevessel again leading to an acute event such as MI or cerebralinfarction (stroke).

Ethical clearance – Not required since it is a review article

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Conflict of interest – nil

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