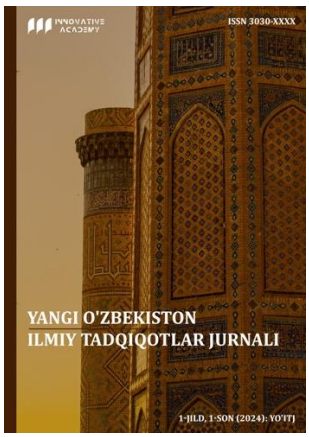

AUTOIMMUNE INFLAMMATION – AS A CAUSE-AND-EFFECT RELATIONSHIP BETWEEN PERIODONTAL DISEASE AND ATHEROSCLEROSIS.

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ABSTRACT

Over the past few years, a sufficient number of randomized clinical studies have been aimed at searching for connections between periodontal diseases, in particular, periodontitis of varying severity with chronic coronary heart disease, acute coronary syndrome, arterial hypertension and metabolic syndrome. Almost all of these cardiac diseases develop against the background of atherosclerosis. The commonality between periodontitis and atherosclerosis of various localizations remains to be elucidated, and this review is dedicated to summarizing the research to date and that still to come in the future.

The main cause of almost all cardiovascular diseases is atherosclerosis. Atherosclerosis is a chronic inflammatory vascular disease that leads to the deposition of lipids in the intima and the formation of an atherosclerotic plaque. The increase and proliferation of plaques leads to a decrease in the lumen of the vessel, and hence the blood flow, which leads to ischemia of almost all organs and tissues. Ruptures or tears of plaques can lead to vascular thrombosis and irreversible processes in the body. The main heart diseases associated with atherosclerosis include myocardial infarction (fatal and non-fatal), angina pectoris, acute coronary syndrome, arrhythmias, chronic heart failure, heart valve disease and cardiomyopathy. The main vessels affected by atherosclerosis are the coronary, carotid, peripheral arteries , abdominal aneurysm aorta and stroke, that is, cerebral vessels.

Periodontal disease (including gingivitis), resulting from various types of plaque, is estimated to affect 47.2% of adults in the United States aged 35 years and older. This figure increases to 69 % after 63 years. In addition to oral care habits, factors contributing to periodontal disease include socioeconomic status, gender (men > women), education, diet and smoking. Based on data from 2009 and 2010, it is estimated that severe periodontal disease affects 11% of adults worldwide and is the sixth most common disease (number of cases at a given time). Periodontal disease and CVD (the likelihood of new cases of the disease) increases with age.

Numerous epidemiological studies have shown an association between periodontitis (excluding gingivitis) and cardiovascular disease . In 2012, after more than 20 years of epidemiological evidence supporting a link between periodontitis and cardiovascular disease, the American Heart Association published a scientific statement confirming the link between the two diseases but emphasizing the absence of cause and effect .

Cumulative data from the literature over recent decades confirm the role of periodontitis as an independent risk factor for atherosclerosis. The presence of certain periodontal pathogens, - particularly gram-negative anaerobes, in subgingival biofilm has been associated with an increased risk of myocardial infarction; the odds ranged from 2.52 to 2.99 in the presence of T . forsythia and P. _ gingivalis respectively, compared to the control group. A hallmark of periodontitis is the increased levels of gram-negative bacteria, which are characterized by their ability to induce an intense immune response through a pathogenic mechanism such as lipopolysaccharide (LPS). Moreover, some of these bacterial species have the ability to penetrate deeper tissues, reaching the circulation and causing a systemic immune response away from their original habitat Results from several studies in vivo and in vitro showed that periodontal bacteria associated with chronic inflammation can disrupt epithelial barrier function through epithelial- mesenchymal transition

Indeed, current literature has provided valuable information on common biomarkers for PD and AC SZ that may offer prognostic and diagnostic potential to significantly reduce the risk of adverse cardiac and vascular events at earlier stages. However, further research is needed in this regard, since precise signaling other than biomarkers A CVD and PD have not yet been fully clarified.

Among the various drugs used for the treatment and prevention of A CVD, statins have demonstrated therapeutic potential in the treatment of periodontal disease. Statins are 3-hydroxymethyl inhibitors. glutaryl coenzyme A reductase (HMG- CoA reductase). These drugs have different ring structures and are known to lower LDL and cholesterol levels in the blood to prevent ASZ. In addition to their primary lipid-lowering effects, statins have several pleiotropic effects, including anti-inflammatory, antioxidant, antibacterial and immunoregulatory functions

The anti-inflammatory effect of statins is due to their ability to inhibit pro-inflammatory cytokines and increase the activity of anti-inflammatory ones. This effect is primarily associated with the activation of extracellular signal-regulated protein kinases (ERK), mitogen -activated protein kinase (MAPK), and the protein kinase (PI 3- Akt) signaling pathway . In addition, statins are able to modulate the host response to bacterial challenge; thereby preventing inflammation-mediated bone resorption and promoting new bone formation. Local administration of statins using experimental animal models promoted the prevention of alveolar bone resorption as a result of their anti-inflammatory, antimicrobial and bone remodeling properties, in addition to their inhibitory effects on metalloproteinases

A 5-year population-based competitive follow-up study examined the effect of systemically taken statins on the rate of tooth loss compared with participants not taking statins . The study reported a reduced incidence of tooth loss in patients receiving statins compared to controls . In addition, a significant improvement in the clinical signs of periodontitis leads to prolongation

of the preclinical course of ASCVD and the prevention of fatal outcomes of heart attack, stroke, and thrombosis. Further interventional studies are needed to further elucidate the relationship between PD and ACVD , especially in terms of the biological impact of PD on the atherogenic cascade through its effect on the vascular endothelium. In general, further research will undoubtedly be needed on the effect of statins on the course of periodontitis in complex standard treatment, especially its local use. Despite the promising results of statins , their effects on various aspects of soft and hard tissue healing need further study, especially in relation to wound healing and regeneration.

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