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Lipids and Periodontal Disease– A Glimpse

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ABSTRACT:

Lipids within the structure of the cell membranes within the oral surroundings facilitates in elasticity permeability of the cellular membrane which helps in transduction pathways between the salivary glands and other tissues. The houses and lipid issue of the cellular range from one of a kind physiological and pathological situation in periodontal ailment. The inflammatory mediators play a main role in the oral and systemic situation. There may be a upward thrust and fall proinflammatory mediators within the systemic conditions which also play a vital function within the oral health

Keywords: Lipids, Proinflammatory Mediators, Saliva, Blood

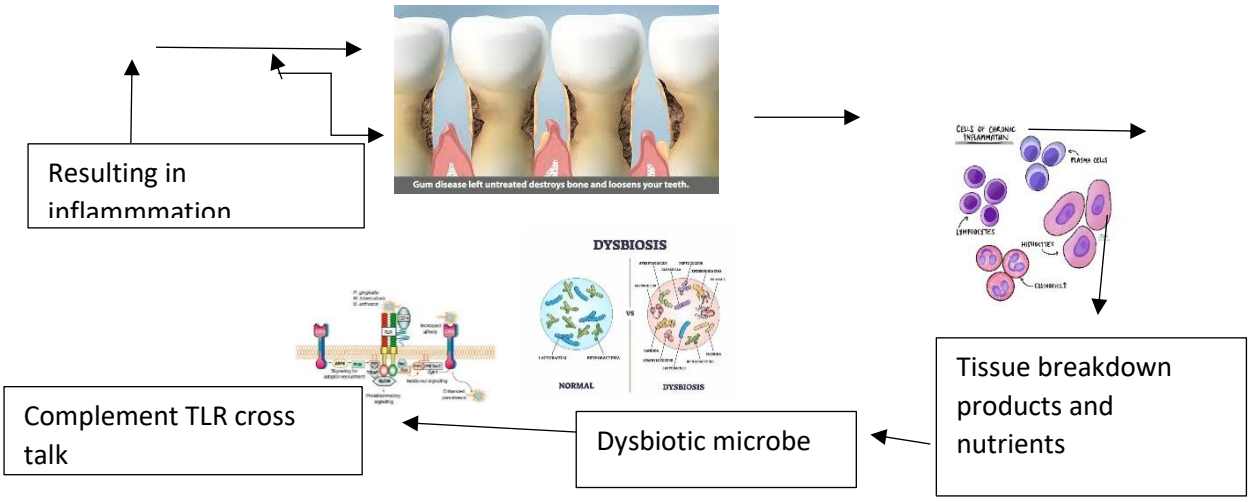
1. INTRODUCTION

Periodontal disease is a multi factorial disease that affects the gingiva and the tooth supporting structures[(1,2)]. Dyslipidemia is related to various diseases progress as it represents a substantial cardiovascular it is widely recognized that extended visceral fat generally results in abnormalities in serum lipid profiles [(3,4)] related to elevated stages of LDL cholesterol, HDL cholesterol, TG,and TCular ailment(CVD) danger component that's concerned in chronic infection[(5,6,7)].It is found that there is a bidirectional relation between elevation in serum lipids and periodontal disease, high serum blood lipid levels shows increase in proinflammatory mediators which is the major factor for periodontal disease. Studies shows that there is a proven factors for this increase[(8)].

The presence of periodontitis increase the extent of total triglyceride stages and general ldl cholesterol in the serum. when the periodontitis and triglyceride tiers had been as compared with the healthy individuals there had been no statistical importance .however some research show the opportunity on this correlation[(9)]. Acute systemic or chronic infections appear to result in changes in the plasmatic attention of cytokines and hormones, which determine changes inside the lipid metabolism[(10,11)]. In a study done by Feingold et al in 1992 he administered low doses of endotoxins to rats which showed hypertriglyceridemia which shows

similar increase in local infection such as periodontal disease in which there is a chronic exposure to microorganisms and lipopolysaccharides[12,13].

MECHANISM OF PERIODONTITIS FORMATION DUE TO PERIODONTAL MICROBIOME/MICROBIOTA AND THE INFLAMMATORY RESPONSE OF THE HOST CAN READILY EXPLAIN LOCAL TISSUE DESTRUCTION IN PERIODONTITIS



Recent studies in animal models have demonstrated biologically possible mechanisms by which periodontitis may increase a patient's susceptibility to two or more diseases [14]. Recent advances in the understanding of the epigenetic basis of innate immune memory suggest that inflammatory cells in the bone marrow may be associated with chronic inflammatory diseases .The mechanisms linking periodontitis to extra-oral comorbidities are consistent with clinical observations associating periodontitis with bacteremia's, low-grade systemic inflammation, increased myelopoietic activity and the ability of local periodontal treatment to attenuate systemic inflammatory markers and improve comorbid disease activity (surrogate markers)[15,16]

Periodontitis and systemic inflammation

Another molecular mechanism causing periodontal tissue destruction is the dysregulation of proinflammatory cytokines. Cytokines have been important for preserving tissues, hemostasis and immune response regulation and cell signaling. Gram negative bacteria causes proinflammatory cytokines to be produced during inflammatory reaction and alveolar bone loss has been observed with elevated cytokine levels [17]. The dysregulation between pro-inflammatory and anti-inflammatory cytokines is another molecular mechanism contributing to the periodontal tissue damage. It has been widely demonstrated that cytokines play a significant role in maintaining tissue homeostasis, and in regulating the immune response and cell signaling. During inflammatory reactions, LPS from Gram-negative bacteria elicit the production of pro-inflammatory cytokines. Consistently higher level of cytokines has been directly related to alveolar bone loss. Many inflammatory cytokines such as interleukin IL-1,IL-6,IL-7, and tumor necrosis factorTNF-alpha play a major role in causing pathogenesis of periodontitis. The mechanisms linked to systemic and oral health include acquired risk factors, genetic and epigenetic factors, and shared risk factors and social determinants that modify the immune response both locally and systemically. Risk factors such as financial standing, way of life, stress, high blood sugar, use of tobacco and alcohol, and high-fat and sugary foods), prescription medications, microbial dysbiosis, and bacteremia.

Inflammatory changes occurring in periodontitis

Inflammatory alterations in the tissues supporting teeth are seen in cases of periodontitis. Numerous oral pathogens, including viruses, fungi, and bacteria, are responsible for inflammation. One of the body's first defense mechanisms against any external infection is inflammation. Acute inflammation lasts a few days, but chronic inflammation is for several months or even years. Chronic inflammation, which is mediated by various inflammatory mediators, which causes periodontitis. Lipopolysaccharide (LPS), which is composed of lipid and polysaccharide molecules, is thought to be a significant virulence factor of Gram-negative bacteria.

The Glycolipid also known as an endotoxin, the glycolipid LPS interacts with the immune system of the host.

Elevations in serum lipopolysaccharide levels trigger the activation of macrophages, which function as regulatory agents within the immune system. For inflammatory conditions in periodontitis, macrophage-target therapy may therefore be helpful. In periodontitis, colonization of sub gingival anaerobic bacteria is a significant event. The microbes that cause periodontitis up regulate the expression of genes linked to the cell cycle, apoptosis, immunological and inflammatory responses. Genetic loci may be connected to bacterial colonization in periodontitis. Periodontitis-related genetic variants in the host are highlighted by genetic biomarkers. Therefore, epigenetic regulation may contribute to improved periodontitis treatment in the future.

Dysregulation of lipid metabolism and periodontitis

The activation of multiple pathways resulting in bone resorption is attributed to inflammatory mediators. One of the molecular pathways controlling bone remodeling activity is the nuclear factor-kappa B (RANK), RANK ligand (RANKL), and osteoprotegerin (OPG) axis. RANK initiates the differentiation of pre-osteoclasts into mature osteoclasts by activating its receptor on their surface; on the other hand, OPG inhibits RANK's interaction with RANKL, which in turn stops all the molecular processes that result in bone resorption. Under physiologic conditions, the remodeling process is characterized by

the coupling of degradation of the bone matrix by the osteoclasts and its reformation by osteoblasts. An excess of osteoclast activity causes a disruption in the bone modeling when the balance shifts in favor of alveolar bone resorption, as is the case with periodontitis. Another molecular mechanism contributing to periodontal tissue damage is the dysregulation between pro- and anti-inflammatory cytokines. Cytokines have been extensively shown to be important in immune response regulation, cell signaling, and tissue homeostasis maintenance. Gram-negative bacteria's LPS causes pro-inflammatory cytokines to be produced during inflammatory reactions [(18)]. Alveolar bone loss has consistently been linked to elevated cytokine levels. The pathophysiology of periodontitis involves several inflammatory cytokines, including interleukin (IL)-1, IL-6, IL-17, and tumor necrosis factor (TNF)-{. The pro-inflammatory cytokine IL-1 activates Th1 and Th2 cells, which are then involved in the host immune response that is connected to the pathogenesis of periodontitis. Additionally, IL-1 is in charge of bone resorption, and the degree of the condition may be directly correlated with its level. Following periodontal therapy, the total level of IL-1 in gingival fluid—which is correlated with the severity of periodontitis—decreases, emphasizing the significance of inflammatory indicators[(19)]. A wide range of cytokines, such as tumor necrosis factor (TNF)-{, IL-1~, IL-6, IL-12, and (IL)-1~, are involved in the process of inflammatory changes in the bone.

Obesity and periodontitis

The National Institutes of Health defines obesity as having a body mass index (BMI) of ≥30.0 kg/m2. Obesity is associated with a number of chronic disorders, including dyslipidaemia, hypertension, type II diabetes, and coronary heart disease[(20)]. Obesity may also be linked to oral illnesses, specifically periodontitis, according to recent research[(21)] Cutler et al. reported in their study that there was a direct correlation between blood lipid concentrations, Porphyromonas gingivalis antibodies, causing periodontal disease[(22)] Adult periodontitis may develop as a result of white blood cell stimulation (increased generation of oxygen radicals) brought on by hyperlipidaemia[(23)]. The second most potent risk factor for inflammatory periodontal tissue damage is obesity, followed by smoking. Further research is necessary to determine whether periodontal disease raises the risk of coronary heart disease. Furthermore, differences in the prevalence, course, and risk of coronary artery disease among ethnic groups remain unclear. Additionally, Sridhar et al. measured the serum lipid levels in four patient groups: healthy, those with chronic periodontitis, those with coronary heart disease, and those without periodontitis. They came to the conclusion that neither patients with nor without coronary heart disease (CHD) had higher lipid levels as a result of periodontitis[(24)]. Hujoel et al. assessed the impact of periodontitis on patients with pre-existing/self-reported cardiovascular disease, and concluded that periodontitis/gingivitis did not elevate CHD risk among them([25)]. That being said, Loesche, et al. found a strong correlation between blood triglyceride levels and periodontal health[(26)]. In their cohort analysis, Morrison et al. discovered a statistically significant correlation between periodontitis and fatal cardiovascular disease[(27).]
The discrepancy seen in the numerous research linking hyperlipidaemia with periodontal disease may be partially explained by

The methods of assessing periodontitis, namely, based only on - clinical measures like bleeding on probing, clinical attachment level, and probing depth;[(28)] and -non-clinical measures like systemic antibody response, alveolar bone levels, and carotid artery intimal-medial thickness[(29)], are affected by the many confounding variables involved, including diet and physical activity habits, socioeconomical conditions, obesity, age, stress, and geographical location. When examining the effects of periodontal therapy on lipid levels, Shruthi et al. discovered that phase I therapy improved all lipid values except HDL[(30)]. Ramirez et al. is expected to provide more evidence on the effects of different treatment modalities on levels of surrogate biomarkers for CVD[(31)] There is no conclusive evidence that support the hypothesis that periodontitis is a separate risk factor for atherosclerotic cardiovascular disease, despite the large number of contradictory research that have been reported in the literature.

Nonetheless, there are a few clinical guidelines to remember. Patient instruction To maximize risk reduction, the cardiologist and periodontist should work closely together. Routine assessment of heart and periodontal health. Interventional periodontal therapy to lower potential coronary heart disease risk.

Conclusion although some studies prove that there is a strong correlation between periodontitis and lipids further more studies are needed to evaluate that lipids are true surrogate biomarkers to periodontitis.

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