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METABOLIC SYNDROMES AND PERIODONTAL DISEASE

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ABSRACT

Hardly a day goes by in the world of dentistry without some mention of oral systemic relationship. Many studies have been done to find the impact of systemic diseases on oral health and found that there is an association between Diabetes, Obesity and Periodontal disease. Furthermore systemic disorders have been found to have a direct effect on periodontal tissues and these represent the periodontal manifestations of systemic disease. Metabolic syndrome is a group of inter-related metabolic abnormalities which increases the risk of cardio vascular morbidity and mortality. So it is important for a dentist to be updated on oral systemic relationship to implement appropriate plans for its prevention and treatment. In this article definition, pathogenesis, about the components of metabolic syndrome and its association with periodontal disease has been reviewed.

Key word: systemic inflammation, periodontal pathogens, inflammatory mediators

INTRODUCTION

Hardly a day goes by in the world of dentistry without some reveal of oral systemic relationship. It is important for the dentist to be well informed about the latest research in oral systemic relationship and apply proper plans for its prevention and treatment. Of late, metabolic syndrome (MeS) and periodontal disease have been connected. According to the Adult Treatment Panel III and recent consensus workshops, MeS is defined as the concurrence of not only hypertension and atherogenic lipid (hypertriglyceridemia and low density lipoproteins), but also obesity and insulin resistance. A proinflammatory and procoagulant state, with elevation of C-reactive protein and fibrinogen coexist in this syndrome. Also, periodontal diseases have been associated with systemic alteration such as, low grade inflammation, dyslipidemia, a procoagulant state, glucose intolerance, and endothelial dysfunction. Therefore, both MeS and periodontal disease may be related through a common pathophysiological pathway.[1]

ETIOPATHOGENESIS

Many factors affect the pathogenesis of MeS. Age, gender, ethnicity, diet, physical activity, various unknown genetic, and endocrine factors, all play a role.[2] A simple model of MeS would include low level of exercise and high dietary intake, leading to body fat addition (Obesity). Obesity is related with increased insulin resistance. The abundant free fatty acids (FFAs) from expanded adipose tissue decrease insulin sensitivity in muscles. As a result, there is diminished glucose disposal, hyperglycemia, and pancreatic beta cell stimulation to produce extra insulin (hyperinsulinemia). The residual FFAs, which are not absorbed in the muscle cells, are diverted to the liver via the portal

vein. In the liver, these FFAs alter lipoprotein production, leading to dyslipidemia. The putative link between hyperinsulinemia and hypertension may be through sympathetic nervous system stimulation, causing vasoconstriction and increased renal Na reabsorption. [3]

PATHOGENESIS OF PERIODONTAL DISEASE

Bacterial biofilm is necessary for development of periodontal disease which progresses to loss of gingival tissue and bone resorption as a result of infection by periodontal pathogens such as Porphyiromonas gingivalis, Prevotella intermedia, Tannerella forsythia and Aggregatibacter actiomycetemcomintans [4] which provokes host response leading to release of pro inflammatory mediators resulting in periodontal tissue destruction. [5]

ASSOCIATION BETWEEN PERIODONTAL DISEASE AND COMPONENTS OF METABOLIC SYNDROME.

A] PERIODONTAL DISEASE AND OBESITY

Obesity is defined by the body mass index as greater than 30.0kg /m². Cytokines and hormones from the fat tissue acts a key role in the pathogenesis.[6]Tumor necrosis factor-alpha, leptin, adiponectin and resistin modulates the periodontal response.[7,8] Leptin controls the appetitie, regulates the immune response and the creation of inflammatory cytokines. The obesity is linked with the reduction of the susceptibility to the effects of the leptin[9] which stimulates the immunological system.[10,11] In periodontitis there is a unenthusiastic correlation among the levels of leptin in the gingival crevicular fluid.[11] Adipocytes secreting proinflammatory cytokines may be the molecules involving to the pathogenesis of periodontal infection.

B] PERIODONTAL DISEASE AND HYPERLIPIDEMIA

Hyperlipidemia has a deregulating effect on the immune-system cells and tissue healing, rising the susceptibility to infections, such as periodontitis.[12]It has been proved that individuals with periodontal disease have elevated serum levels of total cholesterol, low density lipoprotein cholesterol and triglycerides, when compared with periodontally healthy individuals.[13,14] The variation in the phenotype of immune cells because of the lipids and the serum increase of proinflammatory cytokines such as Tumour necrosis factor-α and Interleukin-lß from chronic periodontitis evidenced the bidirectional relationship between the two conditions.[15]

C] PERIODONTAL DISEASE AND HYPERTENSION

The periodontal disease may stimulate the systemic inflammation associated to Cardiovascular disease. Moreover, the chronic inflammation and the inflammatory cytokines may cause endothelial dysfunction establishing a link between inflammation and risk for cardiovascular disease. This link could be mediated by alterations in the vascular resistance causing blood pressure.[16]

D] PERIODONTAL DISEASE AND INSULIN RESISTANCE /DIABETES TYPE 2

Diabetes and periodontal disease are two chronic diseases that have been considered as biologically related. [17] Diabetic patients are more susceptible to develop periodontal disease because of the polymorphonuclear leukocytes disease, alterations in the collagen metabolism. The formation of AGEs affects the collagen stability and the vascular integrity. AGEs combined macrophage and monocyte receptors of interleukin-l and Tumour necrosis factor-α, which provokes an raise of susceptibility to periodontal disease. [18]Inflammatory cytokines induce the

insulin resistance and the chronic inflammatory diseases, including periodontitis. [19]Additionally both Tumour necrosis factor-α and Interleukin-6 is derived from the fat tissue which mediates inflammation, suggesting that obesity, diabetes and periodontitis are mutually related.

IMPACT OF PERIODONTAL INFECTION ON SYSTEMIC HEALTH

Periodontal disease and metabolic syndrome has bidirectional association. Not only systemic diseases have impact on oral health but oral diseases like periodontal diseases have wide ranging systemic effects.[20]The consequence of chronic periodontal infection is the production of inflammatory cytokines and chemokines by local periodontal tissue or circulating inflammatory cells. The inflammatory cytokines such as interleukin-6, Tumour necrosis factor-α, elevated C-reactive protein levels [21,22] evokes systemic inflammation which contributes to insulin resistance and causes hyperglycemia, which in turn induces the expression of proinflammatory mediators and increases the risk of oral infection and cardiovascular disease among patients with periodontitis. [23]

The prevelance of periodontitis is doubled in patients with diabetes and thus it is commonly interpreted that diabetes affects the progress of periodontitis. However the reverse possibility that periodontitis disturbs glucose handling is also reasonable and worthy of consideration. The periodontitis can also be associated with dyslipidemia via systemic inflammation. Many cytokines including Interleukin-6, Interleukin-1,Tumour necrosis factor-α,stimulate hepatic free fatty acid synthesis resulting in increased synthesis of low density protein and hypertriglyceridemia. [24]

MANAGEMENT OF METABOLIC SYNDROME

The International Diabetes Federation recommended that the management of the metabolic syndrome must be aggressive and uncompromising in its aim to reduce the risk of cardiovascular disease and type 2 diabetes.[25]ATP recommended that obesity be the primary target of intervention for metabolic syndrome .First line of therapy should be weight reduction with increased physical activity. Weight loss lowers serum cholesterol and triglycerides, raises HDL cholesterol, lowers blood pressure and glucose and reduces insulin resistance. In people for whom life style changes is not enough, drug therapy may be required. However the meticulous treatment of metabolic syndrome should not only be a prime concern for terminating disease pathogenesis but also periodontitis should be treated. The removal of the plaque and the factors that favours its accumulation is the primary goal in periodontal therapy. The periodontal therapy comprising of oral prophylaxis, gingival curettage, flap surgery, mucogingival surgery has to be performed when required to restore periodontal health which in turn improves systemic health since periodontal treatment reduces Tumour necrosis factor-α and C –reactive protein levels thereby minimizing the effect of the periodontal health on metabolic syndrome. Education and motivation to the patient, followed by recalls also favours a path to good prognosis. So preservation of the periodontal health of the treated patient have to be maintained by Supportive periodontal treatment.[26]

CONCLUSION

Hence this review article provides wide description on relationship between oral and systemic diseases. The observed association is compatible with the hypothesis that chronic inflammation is an main factor in the physiopathology underlying periodontal disease and metabolic syndrome. The awareness on association of systemic oral relationship would throw an insight on life style modifications. Further investigations are required to determine whether oral health care in individuals exhibiting metabolic syndrome has the potential to decrease the incidence of various systemic diseases.

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