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Periodontal Disease And Metabolic Syndrome

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Abstract

Metabolic syndrome (MetS) is characterized by multiple disorders. MetS is a group of disorders that raise the risk of cardiovascular disease and diabetes type 2. Obesity, lack of physical activity and insulin resistance are the most prominent risk factors for MetS, but ageing, hormonal imbalance and genetic susceptibility also play a role. Periodontitis is a complex chronic inflammatory condition characterised by gradual loss of the tooth-supporting system and associated with dysbiotic plaque biofilms. It has been suggested that periodontitis is associated to metabolic syndrome, caused by increase in oxidative stress and an overactive inflammatory response. Periodontitis can cause increased cytokine levels and oxidative stress, which can contribute to decreased insulin sensitivity. Reduced insulin sensitivity is seen as a critical event in the progression of MetS. The presence of MetS or one of its components may promote a pro-oxidant state, which may reduce periodontal tissues' antioxidant capacity, affecting the normal physiological reaction to bacterial stimulus and raising the risk of periodontal disease. Chronic systemic inflammation may predispose a person with periodontitis to acquire MetS components, or vice versa. It's unknown whether MetS and periodontal disease have a one-way or two-way link. However, the link between MetS and periodontitis suggests that a better knowledge of this link could lead to more interprofessional collaboration where the physicians can refer patients to dentists to ensure that they obtain a dental evaluation and any necessary treatment. These clinical practises would have a positive impact on both oral and overall health.

Keywords: Periodontal disease, Metabolic syndrome, Obesity, Diabetes mellitus, Cardiovascular disease

Introduction

Periodontal disease (PD) is a group of conditions affecting the supporting tissues of the teeth – the gingiva, periodontal ligament, cementum and alveolar bone. It is most often the result of persistent infection and inflammation in response to the presence of periodontal pathogens (e.g. *Aggregatibacter actinomycetemcomitans, Porphyromonas gingivalis, Prevotella intermedia and Fusobacterium nucleatum*).[1] Although bacterial biofilms are necessary for disease development, they are not sufficient to produce the disease. A susceptible host is required, and the host response, through release of a broad spectrum of proinflammatory mediators, is responsible for much of the periodontal tissue destruction observed in the disease. Several other factors have been reported to contribute to disease development. Specifically, independent of any other known risk factors, obesity and hypertension are suggested to be risk factors for periodontal disease and tooth loss, as reported in recent epidemiological studies.[2,3,4,5]

Gingivitis and periodontitis are the most common forms of periodontal disease. Gingivitis is an inflammation of the gingiva without loss of alveolar bone. It is plaque induced and can be reversed with improved oral hygiene. Gingivitis may develop into periodontitis, which is an inflammatory condition that results in loss of support for the dentition. Periodontitis is characterised by progressive and irreversible alveolar bone loss, and, ultimately, loosening and loss of teeth. Signs and symptoms

include erythema, oedema and haemorrhage, deepening of the gingival crevice and periodontal pocket formation. Severe periodontitis is the sixth most prevalent disease in the world and the main cause of disability-adjusted life-years among oral conditions.[6] Periodontitis being a chronic oral infection is associated with numerous bacterial species organized in biofilms posing a perpetual threat to the defence mechanisms triggering inflammatory and immune responses with release of inflammatory mediators as an elevated C-reactive protein, prostaglandin E2 (PGE2) with an increase in cytokines with proinflammatory action such as interleukin (IL-1β), and tumor necrosis factor-alpha (TNF-α). These responses are not only limited to periodontitis but also can be observed in systemic conditions such as diabetes, obesity and arthritis.[7] Chronic periodontitis is diagnosed according to the clinical signs of increased periodontal probing depth (PPD), clinical attachment loss (CAL), gingival inflammation and alveolar bone loss detectable on radiographs.[9] The low-grade inflammatory status induced by untreated PD creates a systemic inflammatory phenotype that has been associated with several other systemic diseases/disorders, including cardiovascular diseases,[7] obesity,[10] insulin resistance[11] and metabolic syndrome (MetS).[12]

The metabolic syndrome (MetS) is a spectrum of conditions that place an individual at increased risk for cardiovascular disease (CVD) and type 2 diabetes mellitus (T2DM). These conditions include dysglycemia, visceral obesity, atherogenic dyslipidemia [elevated triglycerides (TG) and low levels of high-density lipoprotein (HDL)] and hypertension. Periodontal disease is a recognised risk factor for the complications of T2DM.[13,14] as well as poor metabolic control,[15] and has also been associated with increased risk for CVD in cross-sectional studies.[16.17]

As noted, MetS is a spectrum of conditions that increase the risk of CVD and T2DM. The most important risk factors for MetS are obesity, physical inactivity and insulin resistance, but aging, hormonal imbalance and genetic predisposition also have a contributing role.[18,19] Similarly to obesity, the prevalence of MetS has increased over the last decade.[20] The development of insulin resistance, a potential consequence of obesity, is a major event in MetS aetiology and has been hypothesised as a link between MetS components.[21] Abdominal obesity, hypertension and hyperglycaemia are the most frequently occurring components of MetS. MetS seems to be a graded condition, with the likelihood of sequelae, such as CVD and T2DM, increasing as the number of components of MetS increases.[22]

The focus more recently has been on identifying possible mechanisms that underlie these associations and whether treating oral diseases leads to an improvement in markers of systemic disease. Cardiovascular disease, obesity and diabetes, in particular, are significant public health problems worldwide and governments are well aware that, unless action is taken, the cost of managing these diseases is capable of bankrupting health budgets in the not-too-distant future. As a result, public awareness has been raised quite dramatically by encouraging individuals to make lifestyle changes regarding diet and exercise. Poor oral health is also a public health problem, with gingivitis and chronic periodontitis being among the most common human infections. Current evidence suggests that improved oral health should be encouraged as part of the healthy lifestyle message to reduce the burden of chronic disease. The objective of this review, therefore, is to provide an update on current understanding of the relationship of periodontal disease and metabolic syndrome, the possible mechanisms involved and the relevance of this for general dental practitioners.

Cardiovascular disease

Cardiovascular disease (CVD) is the leading cause of death in many societies. However, up to 50 per cent of patients with cardiovascular disease have none of the traditional risk factors. Yet, if morbidity and mortality from this disease are to be reduced, it is necessary to understand all possible risk factors. After two decades of research, it has been firmly established that an association exists between periodontal disease and CVD. The pertinent question, however, is about the nature and relevance of this association. Specifically, does the infectious and inflammatory periodontal disease process contribute causally to heart attacks and strokes, or are these two conditions coincidentally associated. An association exists between periodontal disease and CVD. It is unknown, however, whether this relationship is causal or coincidental. The advent of the

Volume 5, Issue 3; May-June 2022; Page No 1169-1175

inflammation paradigm in coronary pathogenesis stimulated research in chronic infections caused by a variety of micro-organisms—such as Chlamydia pneumoniae, Helicobacter pylori, and cytomegalovirus—as well as dental pathogens, since these chronic infections are thought to be involved in the etiopathogenesis of CVD by releasing cytokines and other pro-inflammatory mediators (e.g., CRP,TNF-a) that may initiate a cascade of biochemical reactions and cause endothelial damage and facilitate cholesterol plaque attachment. Yet, due to the multi-factorial nature of dental infection and CVD, confirming a causal association is difficult, and the published results are conflicting. The main deficit in the majority of these studies has been the inadequate control of numerous confounding factors, leading to an overestimation and the imprecise measurement of the predictor or overadjustment of the confounding variables, resulting in underestimation of the risks. Early studies predominantly used nonspecific clinical and radiographic definitions of periodontal disease as surrogates for infectious exposure. While most studies demonstrated positive associations between periodontal disease and CVD, not all studies were positive, and substantial variations in results were evident. More recent studies have enhanced the specificity of infectious exposure definitions by measuring systemic antibodies to selected periodontal pathogens or by directly measuring and quantifying oral microbiota from subgingival dental plaque. Results from these studies have shown positive associations between periodontal disease and CVD. Evidence continues to support an association among periodontal infections, atherosclerosis and vascular disease.[23] It is now widely accepted that infection and inflammation play an important role in the initiation and progression of atherosclerosis and there are several studies supporting a role for oral infections in this regard. Oral bacteria have been identified in atherosclerotic plaques, demonstrating that they invade blood vessel walls. Porphyromonas gingivalis was found in 100 per cent of atherosclerotic plaques from carotid endarterectomy samples, with Fusobacterium nucleatum and Tannerella forsythia found in up to 80 per cent.[24] Other oral organisms have also been detected. In a prospective cohort study investigated a reported association between dental disease and risk of coronary heart disease. American adults participated in a health examination survey in the early 1970s, participants underwent a standard dental examination at baseline and were followed up to 1987. The results showed that among all 9760 subjects included in the analysis those with periodontitis had a 25% increased risk of coronary heart disease relative to those with minimal periodontal disease. Poor oral hygiene, determined by the extent of dental debris and calculus, was also associated with an increased incidence of coronary heart disease.[25] A recent study has shown that increasing bacterial load of oral bacteria, rather than any specific bacterial species, was associated with myocardial infarction and this was independent of other cardiovascular risk factors.[26] Similarly, a mixed infection with high levels of P. gingivalis, T. forsythia and F. nucleatum was found in the majority of a group of cardiovascular patients in the ongoing longitudinal Brisbane Cardiovascular and Periodontal Study (CAPS).[27] The fact that this association with periodontopathic bacteria was independent of actual periodontal disease status emphasizes the importance of the role dentists play in assisting and encouraging their patients to achieve good plaque control, irrespective of their periodontal status.

Diabetes

Diabetes Mellitus (DM) is a group of metabolic diseases characterized by hyperglycemia due to defects of insulin secretion and/or insulin action. Chronic hyperglycemia is associated with the deterioration, dysfunction, and failure of various organs, including eyes, kidney, nerves, heart, and blood vessels. According to multiple studies, dysglycemia shows the most well-established relationship to periodontal disease. Inflammation is the central feature of the pathogenesis of both DM and periodontal disease. DM1 and DM2 are associated with high levels of systemic inflammation markers. This elevated inflammatory state contributes to macro- and micro-vascular complications; moreover, hyperglycemia can activate pathways that increase inflammation, oxidative stress, and apoptosis. Elevated IL-6 and TNF-α levels have been observed in diabetes and in obesity, and the onset of DM2 can be predicted by increased serum levels of IL-6 and CRP. Besides DM2, elevated CRP levels are also associated with insulin resistance and cardiovascular disease (CVD). TNF-α and IL-6 are the main inducers of acute phase proteins (e.g., CRP)

Volume 5, Issue 3; May-June 2022; Page No 1169-1175

that also impair the intracellular signalling of insulin, potentially contributing to insulin resistance. Serum IL-6 and CRP levels are also elevated in patients with periodontitis, and a correlation has been observed between IL-6 levels and the extent of periodontal disease. Therefore, the systemic inflammation associated with periodontal disease can promote the development of a diabetic state. It has been found that adipokines can also contribute to the susceptibility to both periodontitis and DM, and the proinflammatory properties of leptin may be especially important in the overexpression of periodontal tissue inflammation in individuals with obesity and/or DM2. Diabetes is known to increase inflammation in periodontal tissues. Thus, gingival crevicular fluid (GCF) levels of PGE2 and IL-1β are higher in patients with DM1 and gingivitis or periodontitis than in non-diabetic individuals with the same degree of periodontal disease. One study found significantly higher GCF IL-1β levels in DM2 patients with HbA1c > 8% than in those with HbA1c < 8%, finding that HbA1c and glucose levels were independent predictors of elevated GCF IL1β levels. After the damage caused by the lipopolysaccharide, monocytes in DM1 patients produce significantly higher concentrations of TNF-α, IL-1β, and PGE2 in comparison to those in non-diabetics. Various studies have consistently documented the consequences of defects in PMN leukocyte activity in diabetic patients, including the alteration of chemotaxis, phagocytosis, and bactericide functions. PMNs require energy for these functions, and these defects may be related to the metabolic changes that take place in diabetes. Chemotaxis was found to be lesser in diabetic patients with advanced periodontitis than in those with mild periodontitis.[28] A study investigated the association between glycemic control of type 2 diabetes mellitus (type 2 DM) and severe periodontal disease in the US adult population ages 45 years and older. They found that individuals with poorly controlled diabetes had a significantly higher prevalence of severe periodontitis than those without diabetes after controlling for age, education, smoking status, and calculus. For better controlled diabetes mellitus individuals had a tendency for a higher prevalence of severe periodontitis.[29]

Obesity

Obesity is an excess amount of body fat in proportion to lean body mass, to the extent that health is impaired. The most commonly used measure of body fat is the body mass index, which is defined as a person's weight, in kilograms, divided by the square of his/her height in meters.[30] Obesity is also a risk factor for hypertension, dyslipidaemia, CHD and stroke, and there is now evidence that obesity is also associated with periodontitis.[31] The most commonly used indicator of obesity is body mass index (BMI) (weight in kg ⁄(height in m2)), with a BMI 25 kg ⁄ m2 considered overweight and 30 kg ⁄ m2 considered obese. Indeed, BMI together with extensive periodontal disease has been related to increased CRP levels in otherwise healthy middle-aged adults.[32] Obesity is associated with reduced sensitivity to the appetite suppressing effects of leptin, leading in turn to higher levels. Infection and inflammation can also lead to higher leptin levels.[31] As raised serum leptin concentration can enhance atherosclerosis it is, therefore, a potential risk factor for CVD. However, whether periodontal inflammation has any effect on leptin levels, or vice versa, remains to be determined, especially in view of the epidemiological association between obesity and periodontitis. Nevertheless, a recent study suggests that overweight and obese individuals who are periodontally healthy may be at risk for initiation and progression of periodontal disease due to an overgrowth of Tannerella forsythia in subgingival plaque.[33] A study summarised that, periodontists must be aware of the increasing numbers of obese persons and of the significance of obesity as a multiple-risk factor syndrome for overall and oral health.[34]

Metabolic Syndrome

Studies support a bidirectional relationship between periodontal disease and MetS. In addition to sharing risk factors, it is believed they also share a pathologic role in the development or progression of CVD. CVD includes a range of disorders that affect the body's blood vessels and the heart's structure and function. Risk factors for CVD include hypertension, abnormal cholesterol levels, and elevated blood glucose levels. One clearly identified cluster of conditions that increases the risk of CVD is called metabolic syndrome (MetS). MetS includes five core components, all of which are known metabolic risk factors for CVD. These include: abdominal obesity, elevated triglycerides, reduced HDL cholesterol, hypertension, and elevated fasting blood glucose.[35] A study assessed the relationship between periodontitis

Volume 5, Issue 3; May-June 2022; Page No 1169-1175

and 5 components of metabolic syndrome abdominal obesity, triglyceride level, high-density lipoprotein cholesterol level, blood pressure, and fasting blood sugar level in 584 Japanese women reported that each component was analysed separately, waist, HDL cholesterol, and fasting plasma glucose had significant relationships with periodontal disease. If the participants had more of the components of metabolic syndrome, the risk of periodontal disease tended to increase according to the number of the components.[36] A study on Indian population to assess the association between metabolic syndrome and periodontal disease suggested that the association between metabolic syndrome and periodontal disease was significant, and abdominal obesity appeared to be the most important contributing metabolic factor to periodontal disease. In addition to the relationship between periodontal disease and MetS, research also indicates that there are relationships between periodontal disease and the individual metabolic risk factors that make up MetS. A study investigated whether periodontal therapy may reduce systemic inflammation in patients with MetS and reduce cardiovascular risk, the results showed that reduction of periodontal inflammation either with root planing and systemic antibiotics or with plaque control and subgingival scaling significantly reduces CRP levels after 9 months in patients with MetS.[37]

Conclusion

Periodontitis is a multifactorial chronic inflammatory disease associated with dysbiotic plaque biofilms and characterized by progressive destruction of the tooth-supporting apparatus. Globally, it is estimated that 740 million people are affected by its severe form. Periodontitis has been suggested to be linked to obesity and metabolic syndrome. Obesity, defined as excessive fat accumulation, is a complex multifactorial chronic inflammatory disease, with a high and increasing prevalence. Metabolic syndrome is defined as a cluster of obesity, dyslipidemia, hypertension, and dysglycemia. Obesity, metabolic syndrome and periodontitis are among the most common non-communicable diseases and a large body of evidence from epidemiologic studies supports the association between these conditions. At present there is only limited evidence available from a few intervention studies. Nevertheless, the global burden of periodontitis combined with the obesity epidemic has important clinical and public health implications for the dental team. In accordance with the common risk factor approach for tackling non-communicable diseases, it has been proposed that oral healthcare professionals have an important role in the promotion of periodontal health and general well-being through facilitation of healthy lifestyle behaviours.

In 2000, former Surgeon General David Satcher released *Oral Health in America: A Report of the Surgeon General*, which highlighted the importance of oral health as the gateway to general health and well-being. The report revealed how oral disease is a silent problem. Proper oral health is vital to a productive and healthy life. By understanding the risk factors associated with systemic diseases we are able to improve the quality of our increasing longevity and reduce the burden of disease for current and future generations. The prevalence of metabolic syndrome due to the urbanization, inactivity and excessive consumption of energy is increasing, and since this disease is the source of many other diseases including diabetes, atherosclerosis, heart attacks and so forth, seeking therapies for this is vital.

Based on literature, a strong association was found between metabolic syndrome and periodontal health. Extensive research has established plausible mechanisms to explain how these conditions can negatively impact each other. Various pathologies associated with MetS can be related to and favour the onset of CVD and, especially, periodontitis. Oxidative stress seems to be the chief suspect in ethiopathogenesis of periodontal disease. However, due to the heterogeneity of criteria to assess periodontitis and MetS and also paucity of longitudinal studies, it is difficult to determine the relative contribution of periodontitis to MetS. Age and the number of positive components of MetS appear to strengthen the relationship, however, incidence of each disease entity increases with ageing. Consequently, it is critical to determine the relationship between metabolic disorders and periodontal disease, as well as the mechanisms that may underlie such association. Such knowledge may ultimately help to confirm the precise pathways that account for the otherwise uncertain observed association between periodontal disease and systemic conditions, such as type 2 diabetes or CVD. On the other hand, it cannot be ruled out that periodontal disease may just be an intermediate, non-contributory

Volume 5, Issue 3; May-June 2022; Page No 1169-1175

factor between metabolic syndrome and many systemic diseases. As such, it may only be a marker of the presence of a chronic systemic inflammatory condition rather than a risk factor for it.

As a conclusion, both METs and periodontal diseases are worldwide significant community problem. Several clinical and laboratory studies have indicated an association between these common problems. However, the results of studies are variable or changeable. Existing cofound factors, such as the standardization of the study groups, may alter the methodology of the previous and ongoing studies. However, for practical purposes one should be aware the importance of oral hygiene and care in the patients with METs. Routine oral examination should take place in the management of the patients with METs with appropriated and early dental interventions whenever required.

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Volume 5, Issue 3; May-June 2022; Page No 1169-1175

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Volume 5, Issue 3; May-June 2022; Page No 1169-1175