Dental Research Journal

Review Article

The association of periodontitis and metabolic syndrome

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Received: September 2012
Accepted: May 2013

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ABSTRACT

Metabolic syndrome (MS) is a condition, which constitutes a group of risk factors that occur together and increase the risk for Coronary Artery Disease, Stroke and type 2 diabetes mellitus. This disorder is found prevalent in the industrialized societies of the world in epidemic proportions. Periodontitis is an oral disease of microbial origin characterized by loss of attachment apparatus of tooth, resulting in edentulism if untreated. Periodontitis has been attributed to produce a low grade systemic inflammatory condition. The link of periodontitis to various systemic disorders has led to the evolution of a new branch termed as "periodontal medicine." Studies reviewed in the present paper have indicated a positive link between the MS and periodontitis and it is suggested that subjects displaying several components of MS should be submitted to periodontal examination. Present studies have displayed coherent relation between the two entities. This review will address the vicious association between MS and periodontitis, depicting the commonality of pathophysiological pathway between the two entities. Systematic reviews, meta-analysis addressing the concerned subject were screened. Whether the systematic periodontal therapy in individuals exhibiting MS has the potential to reduce the incidence of various adverse systemic complications remains a logical proposition. Further, longitudinal and controlled trials with a large population would be imperative to depict the robustness in the association between MS and periodontal disease in human subjects.

Key Words: Metabolic syndrome, periodontitis, periodontal therapy, type 2 diabetes mellitus

INTRODUCTION

The developing countries serve as a major contributor to the global increase in cardiovascular disease (CVD) through the increased mortality and prevalence of metabolic syndrome (MS). MS is a widely prevalent and multi-factorial disorder, also known by other names such as Reaven's syndrome, insulin resistance (IR) syndrome, plurimetabolic syndrome, syndrome X and the deadly quartet. MS is a configuration of multiple system abnormalities characterized by hyperglycemia, central obesity, abnormal cholesterol and triglyceride (TG) levels and hypertension (HT).[1] MS has attracted immense clinical significance since the last two decades. Prevailing data suggests that obesity and MS are immediate precursors of type 2 diabetes mellitus (T2DM) and CVD.[2] Various definitions of MS have been proposed over the years, punctuating on IR or abdominal/visceral obesity. International Diabetes Federation proposed a new definition based on clinical criteria [Tables 1 and 2].[3]

Periodontitis is a common, chronic, low-grade inflammatory disease of microbial origin, affecting humans and resulting in the destruction of tooth supporting apparatus. The signs and symptoms of periodontitis include swollen gums, deepening of the gingival crevice leading to the formation of periodontal pocket, bleeding on brushing, increased spacing between the teeth, loose teeth, teeth loss and edentulism can occur if the periodontal

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Table 1: International diabetes federation: Metabolic syndrome definition based on clinical criteria

Central obesity
Waist circumference - ethnicity specific (refer Table 2)
Plus any two
Raised triglycerides
>150 mg/dL (1.7 mmol/L)
Specific treatment for this lipid abnormality
Reduced HDL-cholesterol
<40 mg/dL (1.03 mmol/L) in men
<50 mg/dL (1.29 mmol/L) in women
Specific treatment for this lipid abnormality
Raised blood pressure
Systolic >130 mm Hg
Diastolic >85 mm Hg
Treatment of previously diagnosed hypertension
Raised fasting plasma glucose
Fasting plasma glucose >100 mg/dL (5.6 mmol/L)
Previously diagnosed type 2 diabetes
If above 5.6 mmol/L or 100 mg/dL, oral glucose tolerance test is strongly recommended, but is not necessary to define presence of syndrome

HDL: High-density lipoprotein

Table 2: Ethnic-specific values for waist circumference based on clinical criteria

Ethnic group	Waist circumference (as a measure of central obesity)
Europeans	
Men	≥94 cm
Women	≥80 cm
South Asians	
Men	≥90 cm
Women	≥80 cm
Chinese	
Men	≥90 cm
Women	≥80 cm
Japanese	
Men	≥85 cm
Women	≥90 cm
Ethnic South and Central Americans	Use South Asian recommendations until more specific data are available
Sub-saharan Africans	Use European data until more specific data are available
Eastern Mediterranean and Middle East (Arab) populations	Use European data until more specific data are available

treatment is not instituted in these subjects. Recent studies have indicated that periodontitis may have an unfathomed effect on the systemic health. A vivid exploration of the cryptic mechanisms linking periodontitis to systemic disorders has ensued into the development of a new branch termed "periodontal medicine."[4]

METHODS OF DATA COLLECTION

Studies examining the association of MS with periodontitis were identified using PubMed search with key search terms such as "MS," "obesity," "type 2 diabetes," "dyslipidemia," "periodontitis" and "periodontal therapy (PT)." Systematic reviews, meta-analysis addressing the concerned subject were screened. Only human studies published in English language were considered. The text of the manuscript has been prepared by screening PUBMED database from January 1990 to March 2012.

ETIOPATHOGENESIS OF PERIODONTITIS

Dental plaque consists of at least 800 bacterial species.[5] Subgingival biofilm houses the structured communities of microorganisms with noted heterogeneity, providing a significant impetus for progression of periodontitis. During the past two decades with extensive research, we have come to realize that, although bacterial etiology is the prime component for the occurrence of periodontitis, its mere presence is inadequate for the disease to occur. Numerous host factors such as genetics, systemic health and environmental factors such as tobacco smoking, stress and various other risk factors may even preponderate the bacterial etiology for disease occurrence and prevail the severity of clinical disease expression. Thus, the recent conceptual model of periodontitis recognizes the various risk factors, which maneuver by modifying host responses, resulting in a change of disease expression.[6] In this model, host immune-inflammatory mechanisms are triggered by bacteria and their products. Activation of the host response in turn induces the expression of antibodies and priming the polymorphonuclear neutrophils to counteract the microbial challenge in the gingival sulcus. Cytokines, prostanoids, matrix metalloproteinases expressed as a result to the host response, may actually augment the destruction of periodontal connective tissue. These observations have led to a greater scope in change of ideas and concepts about pathogenesis, prevention and treatment of periodontal diseases. The tissue destruction in periodontitis is characterized by the production of numerous cytokines that mediate inflammatory mechanisms. Various cell types in the periodontium produce chemokines, including fibroblasts, endothelial cells, macrophages, osteoclasts, epithelial cells, neutrophils, monocytes, lymphocytes and mast cells.

Neutrophils, monocytes and other cells produce innate immune cytokines such as interleukin (IL) and tumor necrosis factor-alpha (TNF-α) in the diseased periodontal site. These cytokines play an important role in bone resorption and periodontal tissue destruction.[7]

Subgingival microbiota in periodontitis subjects serves as a significant and persistent gram negative challenge to the host. These microbes and their toxic products such as Lipopolysaccharide (LPS) induce macrophages to secrete cytokines (IL-1α and 1β and TNF-α).[8] Elevated cell and cytokine-mediated markers of inflammation, including C-reactive protein (CRP),[9] fibrinogen,[10,11] matrix metalloproteinases[12] and various cytokines[13] are associated with periodontitis.

The periodontal bacteria and their noxious products gain ready access to the periodontal tissues and to the systemic circulation through the ulcerated sulcular epithelium of the gingiva in periodontitis. In untreated severe periodontitis, the accumulative surface area of ulcerated pocket epithelium liaison with the subgingival microbiota including their products, has been estimated to range from 15 cm^2 to 20 cm^2, which is approximately the size of the palm of an adult hand.[8] Thus, severe chronic periodontitis exemplifies a condition corresponding to sub-clinical septicemia.[14] Patients with periodontitis have been shown to demonstrate endotoxin activity in the serum.[15] Just as the periodontal tissues mount an immune-modulatory response to the bacteria and their products, systemic challenge with these agents also induces a major response. Some patients with periodontitis have reported to express tenfold increase in local and systemic expression of inflammatory cytokines, such as TNF-α and IL-6, by monocytes and macrophages.[16] Since periodontitis itself ensues in the expression of pro-inflammatory cytokines such as TNF-α and IL-6 it should be aptly considered as a systemic disorder.[17] The pro-inflammatory cytokines and periodontal bacteria enter the systemic circulation and produce a "low level systemic inflammation/infection" [Figure 1]. Thus, periodontitis has clinical implications reaching beyond the limits of the oral cavity and linking it to various systemic diseases.[13,18]

HYPERGLYCEMIA AND PERIODONTITIS

T2DM is the most common metabolic disorder characterized by impaired glucose homeostasis. This condition is subsequent to a persistent deterioration of β cell function and hyperglycemia. IR is the salient feature of T2DM. The β cells fail to compensate for the IR, conducing to overt T2DM.[19] Increased oxidative stress and chronic subclinical systemic inflammation is contributory to impaired glycemic control and increased IR; thus, paving a way for T2DM.[20,21] Chronic hyperglycemia facilitates the non-enzymatic glycation of proteins with the formation of advanced glycation end products (AGEs). Although, AGEs are physiologically produced, in conditions of hyperglycemia this process is appreciably enhanced. AGEs are reported to prime the macrophages to express inflammatory cytokines. These cytokines are instrumental in the release of acute phase reactants CRP from the liver, further exacerbating the existing inflammation.[22] T2DM subjects with concomitant periodontitis exhibit increased biomarkers and oxidative stress. Impaired β cell function and enhanced IR is attributed to the intensified oxidative stress as a result of hyperactivated neutrophils in periodontitis, resulting in the boosted release of reactive oxygen species. These subjects show decreased plasma antioxidant capacity.[23,24] Periodontitis and T2DM reveal a commonality in the pathogenesis process, featuring inflammatory response at the local and systemic level.[25] Various studies[26-30] have shown a bi-directional relationship between periodontal status and diabetes.

OBESITY AND PERIODONTITIS

Obesity is an excessive amount of body fat in proportion to lean body mass posing a risk to general health. Obesity is a chronic disease, with a multi-factorial etiology. The most commonly used measure of body fat is the body mass index.[31] Obesity might represent a systemic condition, cognizable of regulating the onset and progression of periodontitis. The understanding with regards to adipose tissue has undergone a sea of change. Previously, adipose tissue was only considered as an inert organ, concerned with the storage of TGs. Currently, adipose tissue is reckoned as an endocrine organ, a major depot, capable of secreting bioactive agents called adipokines.[32] Some of the important adipokines are adiponectin (ADN), leptin, resistin, visfatin, chemerin, TNF-α, IL-1, IL-6, IL-8, IL-10, plasminogen activator inhibitor type-1 (PAI-1), monocyte chemoattractant protein-1 and retinol binding protein-4. Obesity ensues in decreased uptake

of insulin by the liver, increased gluconeogenesis in the liver and dyslipidemia. There is a steep elevation in the TG level as a result of increase in free fatty acids.[33] The immunologic activity of these adipokines may play a significant role in the development of IR and in periodontitis. Recent cross-sectional studies and a meta-analysis have divulged positive associations between obesity and periodontal disease.[34-41]

Recently, Han *et al*. concluded that the visceral fat area was the most appropriate indicator of obesity in relation to periodontitis and that obesity could act as a substantial risk factor for periodontitis.[42] Although, the meta-analysis points to a positive association of obesity and periodontitis, the magnitude of the correlation is still not defined. This warrants further prospective studies to clarify the association.

DYSLIPIDEMIA AND PERIODONTITIS

Dyslipidemia is a state of abnormal lipid profile, characterized by an increase in the serum concentrations of TGs, total cholesterol and low-density lipoprotein cholesterol, accompanied by a reduction in the levels of high-density lipoprotein (HDL) cholesterol. It has been proposed that this dyslipidemia conduits to a pro-inflammatory state, further leading to an increase in the levels of pro-inflammatory cytokines and oxidative stress. The presence of systemic inflammation can lead to the down regulation of host protective mechanisms.[43] The association between altered lipid profile and periodontitis has been investigated in several studies.[44-51] Although, it is suggested that dyslipidemia could be associated with periodontitis, its role as a risk factor is still under investigation.[52] Serum pro-inflammatory cytokines may orchestrate a vital role in the association between periodontitis and dyslipidemia. It is proposed that periodontitis is not only associated with the severity of the deterioration of lipid metabolism, but also that the aggravation of hyperlipidemic state is linked with periodontal inflammation by the up-regulation of serum and gingival crevicular fluid pro-inflammatory cytokines.[53]

HT AND PERIODONTITIS

HT is a highly prevalent chronic vascular disease, which is a significant cause of cardiovascular morbidity and mortality. Current evidence implicates periodontitis as a risk factor for atherosclerotic cardiac disease and possibly peripheral arterial disease.[17] Periodontitis is a risk factor for the establishment of atherosclerosis.[54] Studies have revealed that subjects with advanced chronic periodontitis show increased left ventricular mass.[54-57] It is proposed that periodontitis induced systemic inflammation may perpetuate atherosclerosis. A state of systemic inflammation conduces to the stiffness of large arteries and increases the pulse wave velocity. This arterial stiffness as a result of impairment in elastic properties of large arteries could be a contributory mechanism to the pathogenesis of HT. Further, increased blood pressure adds to the risk of cardiovascular events. In hypertensive subjects, periodontitis may enhance the risk and degree of target organ damage.[57-60] Although, the present literature shows a possible association between periodontitis and HT, the existence of a causal relationship needs to be ascertained. The effect of periodontitis on the blood pressure of periodontitis affected subjects and the increase of blood pressure with the deterioration in the degree of periodontitis should be examined. Well-designed, prospective randomized controlled trials should be carried out henceforth.

ASSOCIATION OF PERIODONTITIS AND MS

MS as defined by Reaven consists of obesity, IR, HT, impaired glucose tolerance or diabetes, hyperinsulinemia and dyslipidemia characterized by elevated TG and low HDL concentrations. The constellation of the features mentioned above, are decipherable risk factors for atherosclerosis.[61] Fibrinolytic dysfunction, characterized by elevated levels of PAI-1 is implemental for the pathogenesis of cardiovascular events for subjects with MS.[62] Thus, MS is an established risk for coronary heart disease[63] and T2DM.[64] Various studies have demonstrated a statistically significant association between established periodontitis and CVD.[16,65] Recently, Buhlin *et al*. showed that periodontal inflammation and bone loss is related to angiographically verified coronary artery narrowing in patients with stable coronary artery disease or acute coronary syndrome.[66] Romagna *et al*., in across sectional study on 150 patients, demonstrated that bone loss in periodontitis is associated with a risk of multiple coronary lesions.[67] Studies have reported a positive association between MS and periodontitis[51,68-82] [Table 3]. Two hypotheses could be suggested to explicate the relationship between periodontitis and MS. One hypothesis is a cause-effect relationship. However, longitudinal and

Table 3: Studies with perio and MS

Author, year and reference	Sample size, age	Parameters	Results	Conclusion
Shimazaki *et al.* 2007[68]	584♀	BMI, TG, HDL, BP, FPG, PD, CAL	MS subjects > CAL, PD	MS ↑risk of perio
Nibali *et al.* 2007[51]	302 severe perio 183 non-perio	WBC, HDL, LDL, FPG, PD	Perio pts - ↑WBC, ↓HDL, ↑LDL, ↑FPG	+ve link - perio, systemic. inflammation and MS
D'Aiuto *et al.* 2008[69]	13, 994, ♂ ♀≥ 17 years	BMI, TG, HDL, LDL, IR, PD, BOP	Prevalence of MS ↑ with severity of perio (↑PD↑BOP)	Severe perio ≈ MS in middle aged
Khader *et al.* 2008[70]	78MS; 78 non MS ≥25 years	PI, GI, PD, CAL	≥3 mm PD and CAL in MS	Pts with MS showed ↑ severity of perio
Kushiyama *et al.* 2009[71]	1070 ♂ ♀ 40, 50, 60, 70 years	BMI, HT, HDL, TG, BSL, CPI	↑BP and ↓HDL=CPI code 4 ↑comp. of MS= ↑ CPI	Suspected ≈ between MS and perio
Morita *et al.* 2009[72]	2478 ♂ ♀ mean: 43.3 years	BMI, HT, FPG, TG, HbA1c, PD	↑BMI, BP, TG and HbA1c ↑ (P<0.05) with PD ≥4 mm	perio ≈ MS
Li *et al.* 2009[73]	152 MS; 56 non MS	BOP, PI, CAL	↑BOP, PI, CAL in MS	perio ≈ with MS
Timonen *et al.* 2010[74]	2050 ♂ ♀ 30-64 years	PD, IR, BMI, TG, FPG, HT	MS with PD ≥4 mm and dental caries	MS weakly ≈ MS and dental caries
Morita *et al.* 2010[75]	1023 ♂ ♀ mean: 37.3 years	BP, TG, HDL, PD	PP ≈ +ve conversion of ≥1 comp. of MS	PP ≈ +ve conversion of MS comp.
Han *et al.* 2010[76]	1046 mean: 37.3 years	CPI, FPG, TG, HDL, BP	MS strongly ≈ with perio FPG and HT ↑ ≈ perio	MS might be ≈ perio ↑ link with ↑ FPG and HT
Nesbitt *et al.* 2010[77]	112 ♂ (56.7 years) 78 ♀ (60 years)	Radiograph, FPG, TG, BP, BMI, WBC	advanced alveolar bone loss ≈ comp. of MS	perio ≈ comp. MS
Benguigui *et al.* 2010[78]	255 ♂ ♀ 35-74 years	BMI, TG, HDL, LDL, IR, CAL, PD, PI	Perio ≈ MS (P=0.050) IR ≈ severe perio	Perio ≈ MS, with a central role of IR
Adriankaja *et al.* 2011[79]	7431 ♂ ♀ ≥20 years	BMI, TG, HDL, HT, FPG, PD	~ with ≥2 comp. MS ≈ perio; ↑obesity ≈ perio	~ perio ≈ MS; obesity contributory in | ~
Kwon *et al.* 2011[80]	7178 ♂ ♀ 19 years	BMI, TG, HDL, HT, CPI, PD	MLR analysis, MS ≈ perio	MS ≈ perio
Chen *et al.* 2011[81]	253 HD pts	BMI, CRP, TG, HDL, FPG, PI, GI, PDI, HT	MS ↑ perio pts.; ↓ non perio pts	Perio ≈ MS in HD pts
Han *et al.* 2012[82]	167 MS; 166 non MS	BMI, CRP, TG, HDL, FPG, CPI	Perio ≈ MS risk as compared to non- MS	Perio ≈ MS

♂: Male; ♀: Female; BMI: Body mass index; BP: Blood pressure; TG: Triglyceride; HDL: High density lipoprotein; LDL: Low density lipoprotein; FPG: Fasting plasma glucose; HT: Hypertension; BOP: Bleeding on probing; PD: Probing depth; CAL: Clinical attachment loss; IR: Insulin resistance; ≈: Association; comp.: Components; pts: Patients; WBC: White blood cell count; CPI: Community periodontal index; HbA1c: Glycated hemoglobin assay; PP: Periodontal pockets; +ve: Positive; ↑: Increase; ↓: Decrease; MS: Metabolic syndrome; HD: Hemodialysis; MLR: Multivariate logistic regression; Perio: Periodontitis; CRP: C-reactive protein

large-sample studies are needed to corroborate, which disease is the cause. The other hypothesis proposes a commonality in risk factors (excess caloric intake, sedentary life-style and poor oral hygiene) between the two conditions.[73] It is observed that periodontitis shares some common risk factors with MS, including hyperglycemia, obesity, dyslipidemia and elevated blood pressure. Although, the causative association of periodontitis with most of the mentioned factors is yet to be emphatically proven, periodontitis can pose as a risk factor, capable of modifying the disease course. The inflamed gingival tissue in periodontitis can act as a perennial source of pro-inflammatory cytokines, bacteria and LPS furnishing the impulse for systemic inflammation and infection [Figure 1]. It has been reported that the association between periodontitis and MS could be bi-directional.[76] The inflammatory markers in various components of MS can up-regulate the periodontal inflammatory process and the persistent periodontal inflammation may worsen the inflammatory components of MS [Figure 2]. Obesity can result in exuberance of various cytokines, which can further exacerbate the periodontal inflammation. It is demonstrated that subjects with MS have elevated levels of PAI-1, compared with healthy controls.[83] Tissue plasminogen activator and PAI-1 are instrumental in the pathogenesis of periodontitis by regulation of the proteolytic events in the extracellular matrix.[84] Obesity can affect the levels of PAI-1, which can promote periodontitis. Dyslipidemia and impaired glucose homeostasis can result in endothelial dysfunction. This may interrupt the blood supply to the periodontium. Nutritional deficiency may play a role in the modulation of chronic disease

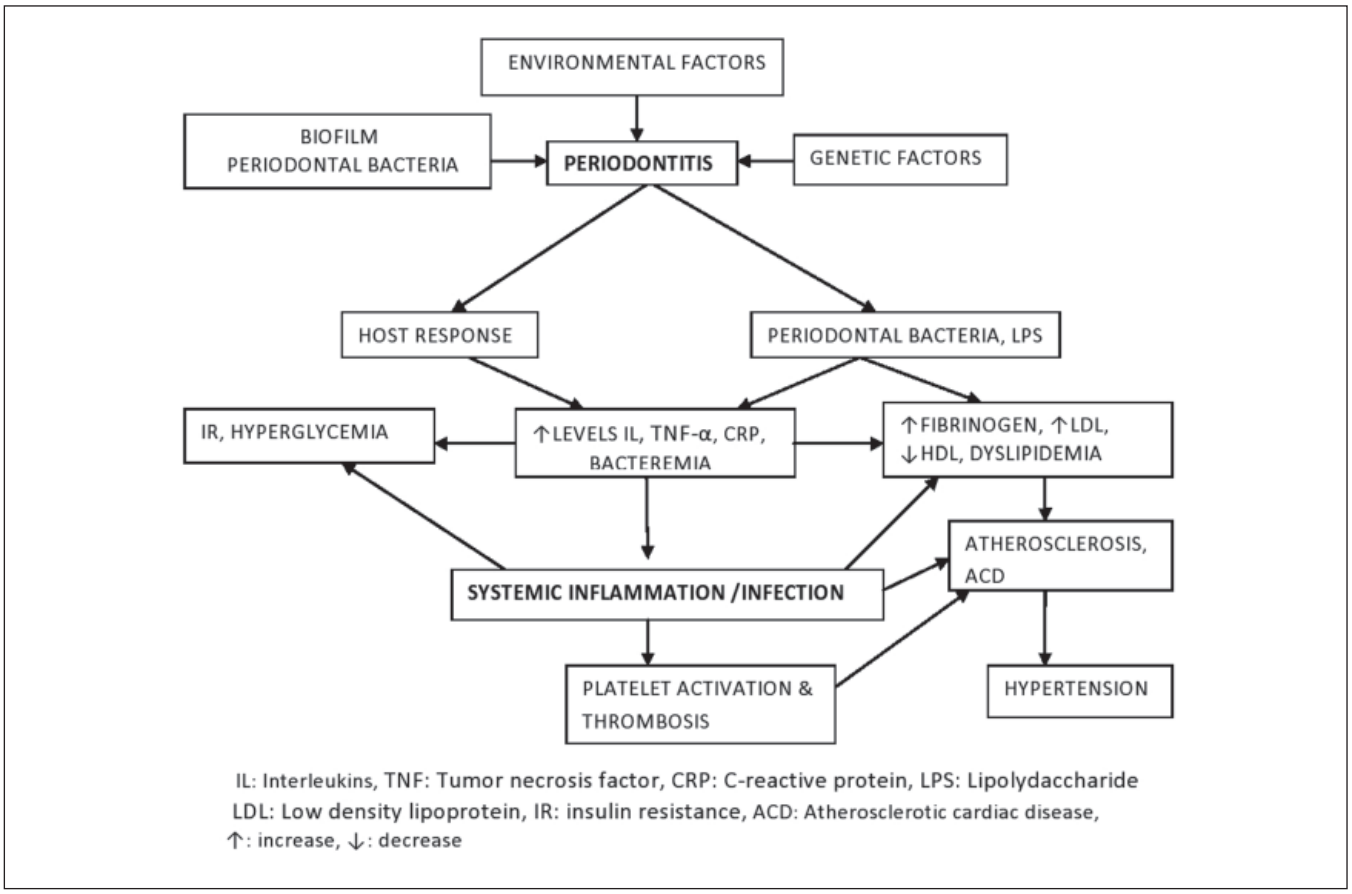

Figure 1: Systemic consequences of periodontitis and possible links

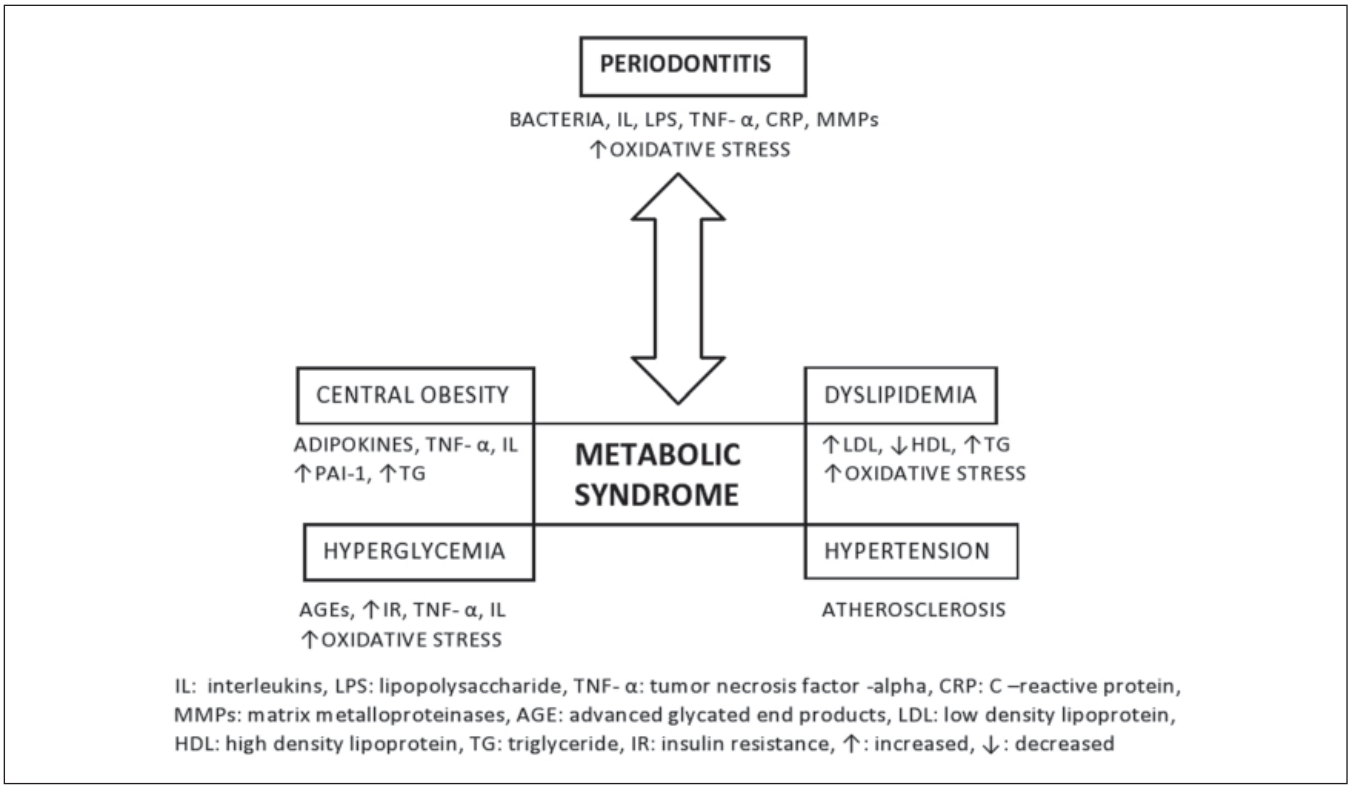

Figure 2: A possible two-way relationship between periodontitis and metabolic syndrome

process.[85] As a chronic inflammatory disease process, periodontitis can also have an adverse effect on MS. Subjects with periodontitis display an increment in the levels of various inflammatory markers in comparison to periodontal healthy controls.[86] Products of periodontal inflammation may upgrade the levels of systemic cytokines, which may further enhance lipolysis. This may result in the increase of circulating TG[87] and exacerbate IR.[23] Nishimura *et al*. aptly proposed that periodontal disease should be considered as a component of MS.[25]

Certain studies have revealed positive effects of non-surgical PT on MS patients. PT has demonstrated amelioration in the tiers of inflammatory markers, expressed by the components of MS. Shimada *et al*. investigated the results of PT on serum leptin and pro-inflammatory cytokine levels in chronic periodontitis subjects. The study sample consisted of 33 chronic periodontitis and 18 patients with healthy periodontal status. Serum samples were evaluated for leptin, ADN, TNF-α, IL-6 and CRP levels before and after non-surgical PT. Non-surgical PT ensued in a significant diminution of serum leptin, IL-6 and CRP levels. It was hence deduced that non-surgical PT is efficacious in amending the dysmetabolic status.[88]

Acharya *et al*. assessed the effect of PT in a sample of periodontitis patients with MS and other group of systemically healthy individuals (control group). The study design consisted of 31 subjects with chronic generalized periodontitis. This sample was segregated into16 subjects (Group A) diagnosed with MS and 15 subjects as healthy (Group B). Non-surgical PT was instituted in both groups. In both groups; high-sensitivity C-reactive protein (hs-CRP), total leukocyte count, parameters of lipid metabolism were evaluated at baseline and 2 months later. In the MS group, PT produced a significant improvement in the levels of inflammatory metabolic markers as compared with the baseline values. Systemically healthy group showed no statistical change in these markers. Thus, PT produced beneficial effects in patients with MS and chronic periodontitis.[89]

Sun *et al*. noted a significant decrease in the serum levels of hs-CRP, TNF-α, IL-6, fasting plasma glucose, glycated hemoglobin, fasting insulin, IR index, TG in the T2DM group receiving PT as compared with the T2DM non treated group ($P < 0.01$ or $P < 0.05$). ADN levels also escalated significantly in the treated group ($P < 0.01$).[90] Although this study did not involve MS subjects; it is notable to consider the positive influence of PT in the down regulation of various inflammatory markers, associated with components of MS.

López *et al*. conducted a parallel-arm, double blind, randomized clinical study of 1 year duration in patients with MS and periodontitis. The experimental study group ($n = 82$) received scaling and root planning (SRP) plus antimicrobial therapy (amoxicillin and metronidazole) and the control group ($n = 83$) received only supra gingival scaling with placebo. Periodontal parameters and serum markers (lipid profile, fibrinogen and hs-CRP levels) were evaluated at 3, 6, 9 and 12 months after therapy. A total of 79 patients in the study group and 81 patients in the control group finished the complete trial. Reduction in CRP levels was found to be statistically significant at 9 ($P = 0.024$) and 12 months ($P = 0.001$) in both groups, without a difference between the groups. Fibrinogen levels diminished significantly in the experimental group at 6 and 12 months, but not in the control group. Hence, it was inferred that elimination of periodontal inflammation by SRP and antimicrobials had a salutary effect on the reduction of CRP levels in MS patients.[91]

CONCLUSION

MS is likened to an epidemic gripping the modern civilization. Life-style, genetics, stress and dietary habits are the preliminary factors. Many studies point out to the positive relation of MS with periodontitis. Further longitudinal, long-term, well-designed, multi-centric studies based on a large sample sizes are mandatory to boost this relationship. With reference to the outcome of periodontal interventional studies in MS subjects there is still a vivid scope. The non-surgical PT is a relatively simple and cost-effective intervention consisting of SRP. SRP eliminates the microbial deposits favoring periodontal health. Gargantuan research in this area of periodontal medicine is anticipated. Periodontitis is a widely prevalent disease, but if diagnosed in the initial stage can be managed successfully without much morbidity. The outcome of therapy largely depends on the motivation and maintenance of patient. PT is noted to be instrumental in amelioration of the various inflammatory biomarkers associated with MS. MS subjects should be recommended to go for frequent periodontal screening and PT should be instituted at

the earliest if indicated. The respective governments should intensively focus on oral health-care programs for the treatment of periodontal diseases in developing countries. An orderly interdisciplinary approach by the physician and oral health-care professional is commended to control the severity of MS and restrict the morbidity and mortality attributed to the components of MS.

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How to cite this article: Gurav AN. The association of periodontitis and metabolic syndrome. Dent Res J 2014;11:1-10.

Source of Support: Nil **Conflict of Interest:** None declared.