REVIEW ARTICLE

Lipid Profile and Periodontitis

CS Prabhahar [1] Ramya Kumari [2]

1. Prof and HOD, Dept of Periodontics, Ultra's Best Dental College, Madurai.
2. Post Graduate, Dept of Periodontics, Ultra's Best Dental College, Madurai.

Abstract

Several decades it has been proposed that infections may be responsible for the accelerated development of atherosclerosis. The initiation of the atherosclerotic plaque is described as focal accumulation of lipids. This explains the importance of plasma lipids in the development of atherosclerosis. Recent reports point towards a possible association between periodontal disease and increased risk for cardiovascular disease. Thus, periodontitis and cardiovascular disease may share common risk factors, and association between periodontitis and coronary heart disease may be due to the elevated levels of plasma lipids. Epidemiological and clinical studies have also suggested that there is a relationship between periodontal disease and impaired lipid metabolism. In this review, we summarized the potential link mechanisms in the association between periodontal infection and serum lipids.

***Keywords**: Chronic periodontitis, Serum lipid profile, Scaling and Root planing, Cardio vascular disease*

Address for correspondence: Dr CS Prabhahar, 9-3, Vallalar Street, Alagappapuram, Karaikudi, Sivagangai District, Tamil Nadu-630002. Email: cyespee@gmail.com

Received on: 05/05/2017 Revised: 10/05/2017 Accepted : 15/07/2017

Introduction

Since the last five decades, the view among dentists and other physicians was that periodontal infections were localized only in the periodontium and that they rarely had systemic implications in healthy individuals. More recently, however, evidence indicated that patients with periodontitis present with increased systemic complication and has proved the association with cardiovascular disorders, endocrine disorders, respiratory disorders, and an adverse relationship in the outcome of pregnancy[1].

Periodontal disease is one of the most frequent chronic infections in humans[2]. Periodontitis is a result of interaction between inflammatory cells and gram negative bacteria. The response is generated by cell wall components, including lipopolysaccharide (LPS)[3]. In addition, their products can invade the periodontal tissue and gain access to systemic circulation[4]. These activate the immune system, potentially deregulating lipid metabolic and increasing the expression of cytokine inflammation[5].

Hyperlipidemia is considered to be one of the major risk factors for cardiovascular disease (CVD). According to reports of the World Health Organization (WHO), CVD is one of the major causes of mortality worldwide. Atherosclerotic vascular disease encompasses coronary heart disease, cerebrovascular disease, and peripheral artery disease that are responsible for the majority of cases of CVD and stroke. Various risk factors such as age, smoking habit, diet, obesity, lack of physical exercise, and modern living lifestyle have been confirmed to be the predisposing factors for hyperlipidemia. Of late, a number of common infectious inflammatory conditions, including periodontitis, have been hypothesized to promote atherogenesis and increase the risk for cardiovascular and cerebrovascular events by continuous release of chronic inflammatory markers to systemic circulation[1]. This review gives the relationship between periodontitis and lipid profile and its change in the nonsurgical therapy.

Lipid Profile & Periodontitis

Over the last 15 years, studies have pointed out a possible association between periodontal disease and an increased risk for cardiovascular disease. Thus, periodontitis and cardiovascular disease may share common risk factors, such as smoking, diabetes, behavioural factors, aging, and male gender.

Cardiovascular disease, which is primarily associated with atherosclerosis, remains one of the primary causes of death worldwide. Age, male gender, smoking, systemic hypertension, plasma fibrinogen, white blood cell count, diabetes mellitus, and hypercholesterolemia are the main risk factors for atherosclerosis. The initiation of atherosclerotic plaque is ascribed to the focal accumulation of lipids. This explains the importance of plasma lipids in the development of atherosclerosis[8].

One of the risk factors strongly related to the pathophysiology of CVD is disturbance of serum lipid profile, that is, elevated blood concentrations of triglycerides (TG), total cholesterol (TC), and low-density lipoprotein-cholesterol (LDL-C), and decreased levels of high-density lipoprotein cholesterol (HDL-C)[7]. Lipids may interact directly with the macrophage cell membrane, interfering with membrane-bound receptors and enzyme systems and altering macrophage gene expression for essential polypeptide growth factors and proinflammatory cytokines, such as tumour necrosis factor-alpha (TNF-a) and interleukin (IL)-1b, which are believed to be associated with periodontal disease[8].

Periodontitis is associated with increased levels of low-density lipoprotein- cholesterol (LDL-C), triglycerides (TG) and total cholesterol (TC). It was shown that periodontitis decreases the level and anti- atherogenic potency of high density lipoprotein (HDL)[6]. Tumour necrosis factor (TNF) induces a rapid increase in serum triglyceride, VLDL and cholesterol levels. Although the mechanism by which TNF increases serum cholesterol levels is unknown, the increase in hepatic cholesterol synthesis may be due to an increase in the activity of 3-hydroxy-3-methyl glutaryl coenzyme A (HMG-CoA) reductase[9].

Non-Surgical Therapy of Periodontitis and Lipid Profile

Periodontal treatment traditionally comprises initial nonsurgical debridement followed by a re-evaluation, at which stage the need for further treatment, usually surgical in nature, is established.

Conventional nonsurgical periodontal therapy consists of mechanical supra- and subgingival tooth debridement and instruction in self-administered oral health care measures. Hand instrumentation, ultrasonic, and sonic instrumentation seem to lead to similar clinical improvements in patients with advanced periodontitis. Periodontal instrumentation is aimed at effectively removing plaque and calculus without excessively instrumenting the tooth surfaces[10].

Chronic destructive periodontal disease is increasingly being recognized not only as a localized inflammatory disease but also as a most common condition with an impact on a variety of medical diseases as well. In this regard, CP can be a risk factor for CVD, stroke, bacterial pneumonia and less well-regulated diabetes mellitus (Ryan 2006). Thus, the question has been asked: can periodontal therapy that effectively reduces local inflammation reduce the risk for CVD? In this regard, several studies have found that periodontal therapy,

i.e., SRP, can reduce the level of circulating biomarkers of systemic inflammation. For example, periodontal therapy was found to reduce significantly plasma levels of CRP after 6 weeks (Mattilla et al. 2002) and after 6 months (D'Aiuto et al. 2004a, b). However, studies by other groups (Ide et al. 2003, Yamazaki et al. 2005) did not observe significant changes in these systemic biomarkers after periodontal therapy consisting of SRP[11].

Vijay Lal et al in 2015 studied the relation between serum lipid profile and periodontitis. The levels of serum lipid profile in 60 subjects, 30 with chronic generalized periodontitis based on clinical attachment loss constituting the test group and 30 without periodontitis constituting the control group, were measured and compared with each other. Results showed that high serum cholesterol and total cholesterol may be

associated with periodontitis in otherwise healthy people[1].

Aditi Sangwan et al in 2013 evaluated the association between serum lipids and periodontal disease in patients with chronic periodontitis with limited data available regarding periodontal status of patients with hyperlipidemia. In this cross-sectional study, 94 patients with hyperlipidemia (50 receiving statins and 44 receiving nonpharmacologic therapy) and 46 control individuals who were normolipidemic underwent periodontal examination. Biochemical parameters measured included serum triglyceride, total cholesterol, low-density lipoprotein cholesterol, and high-density lipoprotein cholesterol levels. The results showed Probing Depth and Gingival Index were significantly higher in patients with hyperlipidemia who were non-statin users compared with the normolipidemic individuals[7].

Andrea M. Monterio et al in 2009 evaluated forty patients with periodontitis and forty healthy individuals by measuring total cholesterol, high density lipoprotein, low density lipoprotein, levels of cytokines, antibodies against oxidized low density lipoprotein, thiobarbituric acid reactive substances, total and differential white blood cell counts. The levels of triglyceride and high density lipoprotein in periodontitis patients were significantly higher and lower respectively compared to controls[6].

Ozlem Fentoglu et al (2009) in a study evaluated fifty-one subjects with hyperlipidemia and 47 normolipidemic subjects. Biochemical parameters, including plasma triglyceride, total cholesterol, low-density lipoprotein cholesterol, and high-density lipoprotein cholesterol levels, and periodontal parameters, were assessed. Results of study showed that patients with mild or moderate hyperlipidemia manifested higher values of periodontal parameters compared to normolipidemic individuals[8].

Tuter G et al (2007) studied thirty-six patients randomly distributed into two groups (Placebo or sub-antimicrobial dose doxycycline for 6 weeks) who also received two regimens of scaling and root planing. At baseline and 6 weeks, gingival crevicular fluid and blood were collected and clinical indices were recorded. Between groups, there were statistically significant greater improvements in pocket depth, gingival index , apolipoprotein-A and and high-density lipoprotein cholesterol levels , favouring the group receiving sub-antimicrobial dose doxycycline adjunctive to scaling and root planing[11].

Conclusion

However, it is unclear whether the association of periodontal disease and impaired lipid metabolism is a cause-effect interrelationship, namely whether periodontitis induces higher serum lipid levels or higher serum lipid levels are predisposing factors for periodontitis, although the associations of these two phenomenons are widely discussed in the periodontal literature.

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

References

1. Lal V, Dubey D, Rath SK, Lohra P. Effect of chronic periodontal infection on systemic lipid profile: A clinical and biochemical study. Journal of the International Clinical Dental Research Organization. 2015 Jul 1;7(2):106.
2. Pussinen PJ, Paju S, Mantyla P, Sorsa T. Serum microbial-and host-derived markers of periodontal diseases: a review. Current medicinal chemistry. 2007 Sep 1;14(22):2402-12.
3. Hettne KM, Weeber M, Laine ML, Cate HT, Boyer S, Kors JA, Loos BG. Automatic mining of the literature to generate new hypotheses for the possible link between periodontitis and atherosclerosis: lipopolysaccharide as a case study. Journal of clinical periodontology. 2007 Dec 1;34(12):1016-24.
4. Lösche W, Marshal GJ, Krause S, Kocher T, Kinane DF. Lipoprotein- associated phospholipase A2 and plasma lipids in patients with destructive periodontal disease. Journal of clinical periodontology. 2005 Jun 1;32(6):640-4.
5. Wendel M, Paul R, Heller AR. Lipoproteins in inflammation and sepsis. II. Clinical aspects. Intensive care medicine. 2007 Jan 1;33(1):25-35.
6. Monteiro AM, Jardini MA, Alves S, Giampaoli V, Aubin EC, Figueiredo Neto AM, Gidlund M. Cardiovascular disease parameters in periodontitis. Journal of periodontology. 2009 Mar;80(3):378-88.
7. Sangwan A, Tewari S, Singh H, Sharma RK, Narula SC. Periodontal status and hyperlipidemia: statin users versus non-users. Journal of periodontology. 2013 Jan;84(1):3-12.
8. Fentoğlu Ö, Öz G, Taşdelen P, Uskun E, Aykaç Y, Bozkurt FY. Periodontal status in subjects with hyperlipidemia. Journal of periodontology. 2009 Feb;80(2):267-73.
9. Fentoglu O, Bozkurt FY. The bi-directional relationship between periodontal disease and hyperlipidemia. Eur J Dent. 2008 Apr 1;2(2):142-6.
10. Claffey N, Polyzois I, Ziaka P. An overview of nonsurgical and surgical therapy. Periodontology 2000. 2004 Oct 1;36(1):35-44.
11. Tüter G, Kurtiş B, Serdar M, Aykan T, Okyay K, Yücel A, Toyman U, Pınar S, Cemri M, Çengel A, Walker SG. Effects of scaling and root planing and sub-antimicrobial dose doxycycline on oral and systemic biomarkers of disease in patients with both chronic periodontitis and coronary artery disease. Journal of clinical periodontology. 2007 Aug 1;34(8):673-81.