

Research Article

A Comparison of the Serum Lipid Level between Patients with Periodontitis and Healthy Individuals

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Abstract

Background and aims. A link between the occurrence of severe periodontitis and several systematic health changes including an altered lipid metabolism has been suggested. The aim of this study was to investigate the relationship between chronic periodontitis and serum lipid levels.

Materials and methods. Thirty patients with chronic periodontitis (the community periodontal index of treatment needs – CPITN scores III & IV; age range 30-40 years old) as test group and 30 healthy individuals as control group were selected. The levels of serum lipids including total cholesterol and triglyceride as well as high- and low-density lipoproteins (HDL and LDL) were assessed. The relationship between serum lipids and periodontal disease was tested using Student's *t*-test.

Results. There were no significant differences in any criteria between case and control groups. The presence of periodontal disease was significantly related with higher total cholesterol in the case group ($p < 0.05$). Triglycerides, HDL, and LDL did not show any difference between case and control groups.

Conclusion. Chronic periodontitis raises the chance of occurrence of hyperlipidemia in healthy people. The findings of this study support the reports linking increased prevalence of changing serum lipids among patients with periodontal disease.

Key words: Cholesterol, chronic periodontitis, HDL, hyperlipidemia, LDL.

Introduction

Today, most populations suffer from elevated blood lipids.¹ Studies show prevalence of elevated lipid

levels in Iran, and indicate that 23.6% percent of adults aged twenty and above have cholesterol total values

above the normal limit.² Hyperlipidemia is considered as one of the main risk factors of cardiovascular diseases.^{3,4} Treatment of hyperlipidemia consists of administering drugs responsible for decreasing serum lipids in the blood stream. Hyperlipidemia can lead to development of atherosclerosis, cardiac ischemic disorders, myocardial infarctions, and strokes and in case of high triglyceride levels, it can also result in pancreatitis.⁵ Among the factors involved in increased levels of blood lipids, genetics, high-fat diet and lack of physical exercise are the main factors. One question raised recently is whether periodontal diseases could be a risk factor for development of hyperlipidemia. Recent studies suggest a relationship between periodontal diseases and hyperlipidemia, which introduces the idea of periodontal diseases being a risk factor for hyperlipidemia. Many studies support this relationship,⁶⁻¹⁰ though two studies have shown opposite results.^{1,11}

Periodontitis is a common oral chronic infection leading to gingival inflammation, destruction of periodontal tissues and deterioration of alveolar bones and finally loss of teeth.^{12,13} In Iran prevalence of periodontitis in age group 40-69 amounts to 70% of the common population.¹¹ Considering the wide range of microbial plaques associated with this disease, its chronic nature and local and systemic immunological responses of the host, it is reasonable that periodontal infection affects overall health and could be involved in the development of systemic diseases such as hyperlipoproteinemia and hypertriglyceridemia.¹⁴ Furthermore, several studies have found an association between periodontitis and increased relative risk for cardiovascular disease,¹⁵ coronary heart disease^{16,17} and ischemic stroke.¹⁵ A probable mechanism by which periodontitis might effect cardiovascular health is chronic oral inflammation may lead to increase of the blood cholesterol level. Total cholesterol and low-density lipoprotein (LDL) in patients affected by these diseases were shown to be higher than those in the control group in one study,⁸ although later research on this issue did not yield similar results. Therefore, the aim of the present study was to examine the relationship between chronic periodontitis and serum lipid levels in an Iranian population.

Materials and Methods

This case-control study consisted of 60 patients, 30 with chronic periodontitis (case group) and 30 healthy individuals (control group). These two groups were paired with respect to gender and age; in addition, they were similar in relation to weight, height, diet, health conditions, and teeth number. The study procedure was

approved by the Ethics Committee of Qazvin University of Medical Sciences. The patients were informed about the purpose and the methods of study.

Periodontal condition of the two groups were determined on the basis of these indices: presence of subgingival and supragingival plaques and germs, presence of at least one periodontal pocket with a depth equal to or more than 4 mm in each quadrant and evidence of alveolar bone destruction, correlating with plaques and calculus, determined on panoramic radiograph.

In addition, subjects had no history of periodontal treatment 6 months prior to the study, were not afflicted with any systemic diseases, were not taking any medications for reduction of serum lipids and were also nonsmokers. Total cholesterol and triglyceride as well as low- and high-density lipoproteins (LDL and HDL) levels were measured in both groups using enzymatic-calorimetric method or by single-point measurements via photometry method in terms of mg/dl. In addition, the community periodontal index of treatment needs (CPITN) index was also determined using WHO probe.

Obtained data was analyzed with Student *t*-test using STATA Version 8.

Results

Mean age was 35.93 ± 5.68 years old in the case and 34.7 ± 5.59 years old in the control group.

Levels of total cholesterol, triglyceride, HDL and LDL in blood serum were measured in both groups and assuming a normal distribution curve, average level for two groups were calculated. Odds ratios were calculated for cholesterol and triglyceride.

Mean total cholesterol of case and control groups (202.8 ± 33.49 mg/dl and 193.46 ± 26.4 mg/dl, respectively) did not show any significant difference ($p=0.24$; odds ratio (OR)=3.76, CI [95%]:0.91-18.35). (Table1)

Triglyceride level of case and control groups

Table1. Frequency distribution of cholesterol and triglyceride levels in subjects according to chronic periodontitis state

	Cholesterol level		Triglyceride level	
	Normal	Abnormal	Normal	Abnormal
Case group	19 63.3%	11 36.7%	23 76.7%	7 23.3%
Control group	26 86.7%	4 13.3%	25 83.3%	5 16.7%
Total	45 75%	15 25%	48 80%	12 20%

Table 2. CPITN index in 2 studied groups

Group	CPITN					Total
	0	1	2	3	4	
Case	0	0	0	9	21	30
Control	17	10	3	0	0	30
Total	17	10	3	9	21	60

(175.5±94.63 mg/dl in case and 149.5± 54.86 in control group) also did not show any significant difference (T=0.3019, p=0.198; OR=1.52, CI [95%]:0.35-6.95).

Comparing mean HDL between the two groups (46.4±7.76 in the case and 44.12±9.24 mg/dl in the control group) showed no significant difference (T=1.035, p=0.305); HDL levels in all 60 studied subjects were within the normal range. Regarding mean LDL levels (126.53±31.48 mg/dl in the case and 119.16±25.83 in the control group), the difference was not statistically significant (T=0.9873, p=0.3276). Only one subject in the case group had an LDL level above the normal range.

Table 2 shows CPITN index state in 2 groups of the present study.

Discussion

This case-control study was designed and performed in order to explore the relationship between presence of periodontal disease and level of serum lipids. We compared serum lipid levels between two groups, one with and one without periodontal disease. According to the results, levels of total cholesterol, triglyceride, LDL and HDL had no correlation with periodontal disease. This is in contrast with reports indicating altered lipid profile could be a risk factor for various disease such as atherosclerosis and cardiac ischemic disease.

In line with our results, a case control study of 60 patients found no relationship between lipid profile and periodontal disease.¹ Another study performed on 56 individuals also was not able to demonstrate a significant relationship between periodontal diseases and serum levels of HDL, LDL, total cholesterol, or triglyceride.¹⁸ Heinrich et al¹⁹ studying 45 patients and 31 healthy controls, showed that periodontal disease is related to total cholesterol and HDL levels, but no other relationship was found. Other studies have also shown a relationship between lipid profile and periodontal disease, including a research on 26 patients and 26 healthy individuals, which showed periodontitis was related to increased levels of cholesterol and triglyceride.²⁰ Lösche et al⁸ in a case-control study on 39 patients with periodontitis and 40 healthy individuals showed that serum levels of total cholesterol, triglyceride and LDL were higher in patients compared with

those in the control group. Other studies have found a positive correlation between periodontitis and increased serum lipid profile.^{8,23-25} The majority of these studies, however, were unable to demonstrate a positive correlation between periodontal disease and all parameters of lipid profile, i.e. total cholesterol, LDL, HDL and triglyceride.

Furthermore, in an animal study, a positive correlation between the extent of lipid deposition and the severity of periodontal disease was detected.²¹

Pussien et al²¹ reported that patients with periodontitis had higher levels of lipopolysaccharides and cholesterol compared with control patients; in addition, the case group had higher levels of HDL and also had a higher HDL/LDL ratio. It was suggested that periodontal disease had caused activation of macrophages through increase of lipopolysaccharides and decrease of LDL levels.²² Another study with 40 case and 40 control subjects concluded that the average cholesterol levels were different between healthy subjects and those with periodontal disease.⁵

It seems that one important factor contributing to different results are different and, in some cases, inadequate sample size. Our study was composed of 60 patients separated into two groups with a statistical first-order error of 0.05 for the two groups. A statistical difference of 0.17 and 0.16 was exhibited in the HDL and LDL levels between the two groups, respectively. Our results indicate no significant statistical difference between case and control groups, similar to the findings of the majority of studies on the subject. Another reason for the discrepancy among the results of these studies is the severity of periodontitis in individuals assigned to case groups. Another flaw among the existing research is due to the case-control design in which reproducibility is difficult and also determining the order in which periodontal disease and hyperlipidemia appear in the individual, i.e., it is difficult to determine if periodontal disease precedes hyperlipidemia or vice-versa. Therefore, even in studies leading to a significant result, determining if hyperlipidemia has been a risk factor or an outcome is difficult. It seems other study designs, such as cohort studies with a larger sample size, may yield more reliable results.

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References

1. Machado AN, Quirino MR, Nas Cimento LF. Relation between chronic periodontal disease and plasmatic levels of triglycerides, total cholesterol. *Clin Infect Dis* 2005;38:88-92.
2. Azizi F. Investigation of blood glucose and lipid levels in patients of Tehran city, Endocrine and Metabolism Research Center. *Dental Journal of Shahid Beheshti University of Medical Sciences* 2001: 97-9.
3. Stamler J, Stamler R, Neaton JD, Wentworth D, Daviglius ML, Garside D, et al. Low risk-factor profile and long-term cardiovascular and non cardiovascular mortality and life expectancy. *JAMA* 1999; 282:2012-8.
4. Glick M. Screening for traditional risk factors for cardiovascular disease. *J Am Dent Assoc* 2002; 133:291-300.
5. Moeintaghavi A, Haerian-Ardakani A, Talebi-Ardakani M, Tabatabaie I. Hyperlipidemia in patients with periodontitis. *J Contemp Dent Pract* 2005;6:78-85.
6. Katz J, Chaushu G, Sharabi Y. On the association between hypercholesterolemia, cardiovascular disease and severe periodontal disease. *J Clin Periodontol* 2001;28:856-68.
7. Lindhe J, Lang NP, Karring T. *Clinical Periodontology and Implant Dentistry*, 5th ed. UK: Wiley-Blackwell; 2008:1340.
8. Lösche W, Karapetow F, Pohl A, Pohl C, Kocher T. Plasma Lipid and blood glucose levels in patients with destructive periodontal disease. *J Periodontol* 2000; 27: 537-41.
9. Mattila K, Nieminen MS, Valtonen VV, Rasi VP, Kesaniemi YA, Syrjala SL, et al. Association between dental health and acute myocardial infarction. *Br Med J* 1989; 298: 778-82.
10. Persson GR, Ohlsson O, Pettersson T, Renvert S. Chronic periodontitis, a significant relationship with acute myocardial infarction. *Eur Heart J* 2003; 24:2108-15.
11. Khoshkhounejad AA, Shayesteh Y, Khoshkhounejad G. The relationship between periodontal disease and blood lipid levels in cardiovascular patients. A thesis for doctorate degree in dentistry no. 4229. Tehran University of Medical Sciences; 2003.
12. Lösche W. Periodontitis and cardiovascular disease: periodontal treatment lowers plasma cholesterol. 2007. [Retrieved 24 October 2009]; Available from: [http://www.thefreelibrary.com/Periodontitis and cardiovascular disease: periodontal treatment...-a0168070769](http://www.thefreelibrary.com/Periodontitis+and+cardiovascular+disease:+periodontal+treatment...-a0168070769).
13. Černochová P, Augustin P, Fassmann A, Izakovičová-Hollá L. Occurrence of periodontal pathogens in patients treated with fixed orthodontic appliances. *Scripta Medica (brno)* 2008; 81:85-96.
14. The Research, Science and Therapy Committee of the American Academy of Periodontology. Periodontal disease as a potential risk factor for systemic disease. *J Periodontol* 1998; 69:841-50.
15. Wright HJ, Matthews JB, Chapple IL, Ling-Mountford N, Cooper PR. Periodontitis associates with a type 1 IFN signature in peripheral blood neutrophils. *J Immunol* 2008;181:5775-84.
16. Beck J, Garcia R, Heiss G, Vokonas PS, Offenbacher S. Periodontal disease and cardiovascular disease. *J Periodontol* 1996; 67:1123-37.
17. Dietrich T, Jimenez M, Krall Kaye EA, Vokonas PS, Garcia RI. Age-dependent associations between chronic periodontitis/edentulism and risk of coronary heart disease. *Circulation* 2008;117:1668-74.
18. Daneshmand M.R, Shayesteh Y. The relationship between periodontal disease and blood lipid levels, A thesis for doctorate degree in dentistry, no. 1403. Tehran University of Medical Sciences; 2002.
19. Heinrichs J, Desvarioux M. Serum total cholesterol and high density lipoprotein cholesterol related to periodontitis. *American Dental Annual Meeting 2001 Abstracts* 27: 1269.
20. Cutler CW, Shinedling EA, Nunn M, Jotwani R, Kim BO, Nares S, et al. Association between periodontitis and hyperlipidemia: cause or effect? *J Periodont* 1999; 70:1429-34.
21. Jain A, Batista EL Jr, Serhan C, Stahl GL, Van Dyke TE. Role for periodontitis in the progression of lipid deposition in an animal model. *Infect Immun* 2003;71:6012-8.
22. Pussinen p.J, Vilkkuna-Rautiainen T, Alftan G, Palosuo T, Jauhiainen M, Sundvall J, et al. Severe periodontitis enhances macrophage activation via increased serum lipopolysaccharide. *Arterioscler Thromb Vasc Biol* 2004; 24:2174-80.
23. Lösche W, Marshal GJ, Apatzidou DA, Krause S, Kocher T, Kinane DF. Lipoprotein-associated phospholipase A2 and plasma lipids in patients with destructive periodontal disease. *J Clin Periodontol* 2005; 32: 640-4.
24. Katz J, Flugelman MY, Goldberg A, Heft M. Association between periodontal pockets and elevated cholesterol and low density lipoprotein cholesterol levels. *J Periodontol* 2002;73: 494-500.
25. Uchiumi D, Kobayashi M, Tachikawa T, Hasegawa K. Subcutaneous and continuous administration of lipopolysaccharide increases serum levels of triglyceride and monocyte chemoattractant protein-1 in rats. *J Periodontal Res* 2004; 39: 120-8.