Original Research Article

ESTIMATION OF SERUM LIPID PROFILE AND ITS CORRELATION WITH ALVEOLAR BONE HEIGHT IN CHRONIC PERIODONTITIS.

Running title SERUM LIPID PROFILE IN CHRONIC PERIODONTITIS

Abstract

Aim*:** Numerous studies have been previously carried out to find out any correlation between lipid profile and periodontitis but existence of an association does not establish whether periodontitis causes an elevation in serum lipid level or elevation in serum lipid profile predispose to periodontitis. The aim of present study was to find any significant correlation between HDL, LDL, VLDL and cholesterol with periodontitis. ***Material and methods*:* 100 patients with chronic periodontitis (group I) and 50 healthy patients (group II) were selected. The levels of serum lipid including HDL, LDL, VLDL, and TOTAL CHOLESTEROL along with fasting blood glucose were assessed. The relationship between severity of periodontitis based on clinical and radiographic finding with serum lipid correlated. ***Results***: There were no significant difference found between mean values of total cholesterol, LDL and VLDL among study and control group. Only HDL showed highly significant difference ($p< 0.00$) between healthy and patient with chronic periodontitis. ***Conclusion***: Estimation of serum lipid profile especially HDL levels in subjects with periodontitis can be considered as screening method for early diagnosis of atherosclerosis to avoid further progression of cardiovascular changes in early age of the life.

Keywords:chronic periodontitis, HDL, LDL, atherosclerosis.

Introduction

Coronary heart disease (CHD) is one of the leading factors of morbidity and mortality throughout the world being responsible for 16% of death in developing and 50% in developed countries. Atherosclerosis of coronary arteries is considered to be the leading cause of premature death among men. The pathological background of the atherosclerosis of coronary arteries is formation of atherosclerotic plaque, which additionally induces other cardiovascular diseases.[1]

Several bacteria and virus have also been identified as potential etiological factors in cardiovascular diseases (CVD).[2]Periodontitis is an inflammatory disease of the supporting tissues of teeth caused by specific microorganisms resulting in progressive destruction of periodontal ligament and alveolar bone with pocket formation, recession or both.[3]

Perhaps one of the most helpfulfinding about periodontitis is that host response varies between individuals and that either an inadequate host immune response or exaggerated response to bacterial pathogens leads to more severe form of disease.[4]The majority of risk factors of cardiovascular diseases are also considered to be risk factors for periodontal diseases.[1]

Thus individuals with periodontitis are more likely to have increase in the level of free fatty acids in the form of cholesterol and triglycerides. These fatty acids are also involved in athermanous plaque formation in blood vessels. This increase in atheromas has been associated with thrombotic events in the form of various cardiovascular diseases. Recent studies have also shown isolation of periodontal pathogen from this atheromatous plaque.[6]

Although numerous studies have been previously carried out to find out any correlation between lipid profile and periodontitis but existence of an association does not establish whether periodontitis causes an elevation in serum lipid level or elevation in serum lipid profile

predispose to periodontitis. So the present study is carried out to find any significant correlation between HDL, LDL and cholesterol with periodontitis and also to assess whether periodontitis may be the first step in the etiopathogenesis of cardiovascular disorders in patients whom other obvious risk factors like smoking were absent.

Materials & methods

A hospital based cross sectional study was carried out in the Department of Oral Medicine and Radiology, VSPM Dental College and Research Centre, Nagpur. Subjects were apprised of the purpose of the study and written consent was taken prior to commencement of the study. Ethical clearance was obtained from the ethical committee of the institution.

This study consists of 100 patients with chronic periodontitis (group I) and 50 healthy patients (group II). Study consists of patients having age more than 20 yrs without any habits along with no systemic or medical illness like hypertension, myocardial infarction, stroke, asthama, endocrinal diseses.

Periodontal condition of the two groups were determined on basis of clinical examination of gingiva and its associated structures including color, contour, surface texture, consistency, bleeding on probing, presence of stippling and pus discharge through gingival crevices. The pocket depth was measured bilaterally in the region of premolar molar region of the mandible with the help of Williams graduated probe. Thus depending upon the attachment loss clinically we divided into three groups like, Mild periodontitis: - < 3mm Moderate periodontitis:-3-6 mm ,severe periodontitis : > 6mm

In addition to clinical examination we also carried out radiographic investigation to assess the alveolar bone height with the help of Digora (optime) RVG software. Digital

calibrations were made for measurement of alveolar bone height from CEJ upto the bone level in premolar molar region of both the quadrant in mandible.

For estimation of lipid profile like total cholesterol, HDL, LDL and VLDL we advised patient to fast at least 8-12 hrs and collected 2 ml of blood sample from anticubital vein by venipuncture method and stored in collecting test tubes with anticoagulant and sent to pathology laboratory for enzyamatic analysis.

Statistical analysis was done to evaluate the correlation between serum lipid profile and periodontitis along with radiographic bone height secondary to periodontitis to find out the significant difference between these values with the help of chi square, annova and Z test.

Results:

The mean values of Total cholesterol was 171.26 mg/dl, while in control group it was 161.51 mg/ dl, and the mean triglyceride in study group was 115.20 mg/dl, while in control group it was 99.91mg/dl, the mean LDL cholesterol in study group was 106.40 mg/ dl, while in control group it was 98.59 mg/ dl. The mean of VLDL cholesterol in study group was 23.17mg/ dl, while in control group it was 20.02 mg/ dl. The mean fasting blood glucose level in study group was 99.90 mgm/ dl, while in control it was 92.27 mgm/ dl.But there was no statistical significant difference found in Total cholesterol, triglycerides, LDL, VLDL & Fasting blood glucose.

Further mean total HDL in study group was 41.20 mg/ dl, while in control group it was 42.97mg/dl which found to be statistically significant (P=0.04).

Further correlation was done for of Alveolar bone loss in mandibular left & right premolar molar region with Lipid parameters. Among all lipid parameters in mandibular left premolar region,

HDL showed negative correlation (r = - 0.209) with statistical significant difference (P=0.036), whereas other parameters (LDL, VLDL & triglyceride) showedno significant difference.

Further alveolar bone loss relation with lipid parameters in mandibular right premolar region found to be negative and was not significant. In the present study we observed raised levels of lipid parameters in both the groups; among all parameters HDL showed highly significant differences (p-0.0001).

Discussion

Periodontitis has been traditionally regarded as a chronic inflammatory oral infection which mainly consists of gram negative anaerobic microflora that leads to gingival inflammation, destruction of periodontal tissues, loss of alveolar bone and exfoliation of the teeth. It is generally accepted that certain organisms within the microbial flora of dental plaque are major etiological agent in periodontitis. These microorganisms particularly P. Gingivalis produces endotoxins in the form of lipopolysacchrides that generates a host mediated tissue destructive immune response. Traditionally it is thought that periodontitis is an oral disease and that the tissue destructive response remains localized within the periodontium, limiting effects of the disease to oral tissues supporting teeth, however recent studies indicate that oral disease may have profound effect on systemic health.[46]

A number of studies have reported association between periodontitis and cardiovascular diseases. Most of the risk factors for cardiovascular diseases are also regarded as risk factor for periodontal diseases. Some studies have found no relationship or an inverse relationship between chronic infection and hyperlipidemia.

Chronic infections like periodontitis have been demonstrated to induce profound changes in plasma concentration of cytokines like TNF- alpha and IL- beta which can result into elevated levels of free fatty acids, LDL (low density lipoprotein) and triglycerides. These elevations in serum lipids are thought to arise from enhanced hepatic lipogenesis, increased adipose tissue lipolysis, increased synthesis or reduced clearance of LDL due to reduction in lipoprotein lipase activity.[47]

Therefore the present study was carried to find out any correlation between chronic infection like periodontitis and increase in serum lipid level in form of cholesterol, triglyceride, HDL (high density lipoprotein), LDL (low density lipoprotein) in otherwise healthy subjects and who did not have any habits

In the present study 8% subjects showed increased total cholesterol from study group whereas it was 4% among healthy subjects; this increased TC showed statically significant difference ($p < 0.05$). similar results were also seen by Loesche et al (2000)[21]&Taleghani F (2005)[34]

These findings give evidence to the theory that periodontitis may be one of the factor that is responsible for increase in total cholesterol levels as in our study the patients did not have any other systemic disorders and no smoking habit, but at the same time other factors such as physical activity, nutrition, stress, socioeconomic status and body mass index (BMI) might have some influence on total cholesterol level which should also be evaluated.

Increased triglyceride levels when assessed between study and control group we found three fold increase in subjects with periodontitis but stastically it was not significant, which was also seen with Taleghani F (2005)[34].

In contrast to our study and Taleghani F, Loesche et al (2000)[21] found significant correlation when triglyceride levels were assessed. This increase in plasma triglyceride levels could be due

to increase in pro-inflammatory cytokines in response to chronic periodontitis. Infection with Gram negative periodontal pathogen can cause rapid release of systemic IL-1 beta and TNF-alpha which are responsible for hyper-triglyceridaemia.

When decreased levels of HDL were assessed in both the groups; the frequency of low HDL was higher by 12% in chronic periodontitis as compared to control; with stastitical significant difference ($p<0.0001$).whereas Loesche et al (2000)[21], Taleghani F (2005)[34], Cristana A (2005)[30] did not find any statistic al significant difference for HDL.

The explanation for relationship between low HDL and periodontitis might be chronic infection in the periodontitis that leads to release of lipopolysaccharide and proinflammatory cytokines.

On the other hand HDL also has anti-inflammatory properties that can decrease the adhesion of endothelial cells, thus low plasma concentration of HDL in blood may be a contributory factor to inflammatory process in periodontitis. In this study no statically significant difference was found when for LDL & VLDL.Loesche et al (2000)[21] found significantly raised LDL levels in subjects with periodontitis which is contradictory to present study. Thus a cause and effect relationship between HDL and periodontitis needs to be established.

Raised blood glucose levels were found in 35% from study group while it was 24% in control group with statistical significant difference (p value < 0.05). Liu et al (1998)[50], Reimers et al (1998)[51]&Shiba et al (1998)[52] stated that some cytokines such as TNF- alpha and IL-beta that are produced in response to infection with gram negative bacteria may be responsible for insulin resistance and subsequent poor glyceamic control in periodontitis patients.

In our study among all lipid parameters only HDL showed negative correlation with alveolar bone loss and severity of periodontitis which was statistically significant indicating that increase in amount of bone loss in severe periodontitis is associated with low serum level of HDL. Similar results are also seen with Saito T (2004)[24]

In the present study among all lipid parameters HDL was the most significantly associated with periodontitis. There was significant difference found in plasma levels of HDL in subjects with periodontitis as compared to subjects with healthy periodontium.

Unlike previous studies we did not find any statistical difference in total cholesterol, triglyceride and LDL levels in subjects with periodontitis and without it. In the previous studies it has been shown that abnormal levels of total cholesterol and LDL are indicators of atherosclerosis or coronary heart disorders, but HDL can be a better measure especially in individuals less than 60 yrs.

Conclusion

The present study mainly consisted of subjects in third and fourth decades with mean age of 39.2 yrs. HDL levels were found to be statistically significantly lowered in the study group that is in subjects with periodontitis. The abnormal HDL levels also statistically significant with severe form of periodontitis thus it can be concluded that young individuals which are affected with any form of periodontitis should be evaluated for the plasma levels HDL as an early marker for atherogenic lipid profile.

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Table 1: Prevalence of serum lipid level in Study and control groups

	TC	**TG**	**HDL**	**LDL**	**VLDL**	**TC:HDL**	**LDL:HDL**
Study	8	6	74	7	6	19	17
Control	2	1	31	1	1	3	3
א2-value	10.00	2.91	60.39	8.00	2.91	3.03	5.07
p-value	**0.00**	0.23	**0.00**	**0.01**	0.31	0.21	0.07

P<0.05= significant

Table 2: Correlation of radiographic bone loss (35, 36 & 37 region) with lipid parameters

Lipid Profiles	35 – 36 region		36 – 37 region	
	Correlation 'r'	p-value	Correlation 'r'	p-value
Blood Glucose	0.000	0.997	-0.010	0.919
TC	0.021	0.835	-0.059	0.561
TG	0.065	0.523	0.065	0.520
HDL	-0.209	**0.036**	-0.191	0.057
LDL	0.041	0.686	-0.045	0.658
VLDL	0.062	0.541	0.059	0.558
TC/HDL	0.082	0.415	-0.009	0.930
LDL/HDL	0.154	0.125	0.062	0.542

P<0.05=significant

Table 3: Comparison of mean lipid parameters in Study and Control group

(a) Descriptive Statistics

Sr. no.	Parameter	Group	N	Mean	Std. Deviation	Std. Error Mean
1	TC	Study	100	171.26	42.57	4.25
		Control	50	161.51	33.22	4.69
2	TG	Study	100	115.20	53.38	5.33
		Control	50	99.91	46.88	6.63
3	HDL	Study	100	41.20	5.65	0.56
		Control	50	42.97	4.84	0.68

4	LDL	Study	100	106.40	39.69	3.96
		Control	50	98.59	30.88	4.36
5	VLDL	Study	100	23.17	10.42	1.04
		Control	50	20.02	9.39	1.32
6	TC/HDL	Study	100	4.10	1.08	0.10
		Control	50	3.69	0.81	0.11
7	LDL/HDL	Study	100	2.61	0.98	0.09
		Control	50	2.31	0.75	0.10

Table 4: Prevalence of abnormal serum lipid level in Study and control groups

	TC	**TG**	**HDL**	**LDL**	**VLDL**	**TC/HDL**	**LDL/ HDL**
Study	8	6	74	7	6	19	17
Control	2	1	31	1	1	3	3
ϰ2-value	10.00	2.91	60.39	8.00	2.91	3.03	5.07
p-value	**0.006 S,p<0.05**	0.23 NS,p>0.05	**P<0.0001 significant**	**0.018 S,p<0.05**	0.31 NS,p>0.05	0.21 NS,p>0.05	0.07 NS,p>0.05

Table 5: Correlation of radiographic bone loss (35, 36, 37) with lipid parameters in study group

Pearsons Correlation Coefficient

Lipid Profiles	35-36		36-37	
	Correlation 'r'	p-value	Correlation 'r'	p-value
Blood Glucose	0.000	0.997	-0.010	0.919 NS,p>0.05

		NS,p>0.05		
TC	0.021	0.835 NS,p>0.05	-0.059	0.561 NS,p>0.05
TG	0.065	0.523 NS,p>0.05	0.065	0.520 NS,p>0.05
HDL	-0.209	**0.036 S,p<0.05**	-0.191	0.057 NS,p>0.05
LDL	0.041	0.686 NS,p>0.05	-0.045	0.658 NS,p>0.05
VLDL	0.062	0.541 NS,p>0.05	0.059	0.558 NS,p>0.05
TC/HDL	0.082	0.415 NS,p>0.05	-0.009	0.930 NS,p>0.05
LDL/HDL	0.154	0.125 NS,p>0.05	0.062	0.542 NS,p>0.05

Graph 1-A: **Prevalence of abnormal serum lipid level in Study and control groups**