Original Article

Correlation of lipid profile APO A1 and APO B100 in chronic periodontitis patients leading to coronary artery disease

ABSTRACT

Background: Increased attention has been focused on the association of periodontal disease with cardio-metabolic syndrome. Although the associations are multi-factorial, very few studies have explored the role of lipoprotein Apo A1 and Apo B100 with chronic periodontitis. Additionally, obstructive sleep apnea (OSA), a chronic multi-factorial respiratory disease, consists of a temporary decrease or cessation of breath for 10 seconds and leads to a reduction in blood oxygen saturation of more than 3% to 4% and/or neurological arousal. OSA involves the upper respiratory tract, and it has been proven that snoring and OSA have systemic consequences in humans. It has been recently suggested that OSA may be related to periodontitis, another chronic multi-factorial disease.

Materials and Methods: A total of 600 participants aged between 30 and 80 years were analyzed. In this case control study, a total of 300 in the case group with chronic periodontitis and coronary artery disease (CAD) and 300 in the control group healthy population with chronic periodontitis were recruited. The following data were collected: 1) general information on socio-demographic, health-related factors, 2) periodontal status [clinical attachment loss (CAL), pocket probing depth (PPD)], and 3) a blood sample for estimation of lipoproteins and biochemical analysis.

Results: The results of the current investigation point to a potential relationship between lipid metabolism and systemic inflammation brought on by periodontitis. It was observed that there is a link between periodontal disease and CAD.

Conclusion: A significant correlation was found between the lipid profile APO A1 and APO B100 and the blood vessels involved in the case groups. Hence, cardiovascular diseases can be efficiently circumvent with a biomarker based approach to treatment, which also benefits patient's quality of life.

Keywords: Apo A1, Apo B100, chronic periodontitis, coronary artery disease, obstructive sleep apnea

INTRODUCTION

Chronic periodontitis is a complex, chronic inflammatory disease of the tooth supporting connective tissues and the alveolar bone. It is caused by an aberrant host response against oral and dental plaque bacteria.[1,2] The host response is further compromised by unfavorable lifestyle factors, such as smoking, and by systemic diseases such as diabetes. If the disease is not diagnosed and treated, the chronic periodontal infection may persist over many years. There is progression in the breakdown of tissues, and teeth may become mobile and eventually exfoliate; 8–13% of the population suffers from severe periodontitis.[3-5]

Cardiovascular diseases (CVDs) are a group of diseases that include congestive heart failure, cardiac arrhythmias,

Access this article online	
Website: www.njms.in	Quick Response Code
DOI: 10.4103/njms.njms_30_23	

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Received: 16 February 2023, **Revised:** 07 September 2023, **Accepted:** 30 October 2023, **Published:** 16 November 2024

How to cite this article: Gupta S, Lal N, Ali W, Pradhan A, Verma AK, Gupta P, *et al.* Correlation of lipid profile APO A1 and APO B100 in chronic periodontitis patients leading to coronary artery disease. Natl J Maxillofac Surg 2024;15:480-8.

coronary artery disease (CAD) (including atherosclerosis and myocardial infarction), valvular heart disease, and stroke. Among these, atherosclerosis, a major component of CVDs, is characterized by the deposition of atherosclerotic plaques on the innermost layer of walls of large- and medium-sized arteries. End-stage outcomes associated with atherosclerosis include coronary thrombosis, myocardial infarction, and stroke. CVD and periodontitis are both chronic and multi-factorial diseases and share some of their risk factors: age, male gender, lower socio-economic status, smoking, and psycho-social factors such as stress.[6] Recently, periodontal disease (PD) has been investigated as a potential factor contributing to the onset and development of CAD.

The presence of obstructive sleep apnea (OSA) is determined by the Apnea-Hypopnea Index (AHI). An AHI ≥5 is indicative of the presence of obstructive sleep apnea. OSA is a common disorder in which recurrent collapse of the upper airway during sleep results in intermittent hypoxemia and sleep fragmentation. Additional symptoms of OSA are chronic and loud snoring, mouth breathing, and interrupted sleep. Some studies have shown that OSA increased levels of systemic inflammation and could be associated with stroke, CVD, and diabetes.[7] Risk factors for periodontal disease include age, smoking, drinking, obesity, and diabetes, and both periodontal diseases and obstructive sleep apnea are associated with systemic inflammation.[8] The etiology of systemic inflammation and OSA is not clear but may be related to inflammation in the oral cavity and periodontal diseases.

AIM AND OBJECTIVES

The aim of present study is to correlate the chronic periodontitis and cardio metabolic syndrome in the North Indian population.

OBJECTIVES

1.) To evaluate chronic periodontitis in angiographically proved CAD subjects.
2.) To investigate the lipid and lipoprotein abnormalities with special reference to phospholipids and ApoA1 and ApoB100 in angiographically proven CAD subjects.
3.) To observe the OSA in chronic periodontitis with angiographically proven CAD subjects.

MATERIALS AND METHODS

Study population

This study was started in the year 2021 to identify the association of chronic periodontitis with cardio-metabolic syndrome. In this case control study, a total of 300 in the case group with chronic periodontitis and coronary heart disease (CHD) were recruited from the out-patient department (OPD) of subjects having single vessel block (n = 60), double vessel block (n = 60), triple vessel block (n = 60), stroke or myocardial infarction (n = 60), and OSA (n = 60) were recruited. The selection of patients was carried out according to the medical report of a cardiologist using angiography. Polysomnography (PSG) or sleep test was done for the patients suffering from OSA in the Department of Respiratory Medicine. Furthermore, the control group (n = 300) enrolled in the study were a healthy population with chronic periodontitis recruited from OPD, Department of Periodontology, King George's Medical University, Lucknow. All the patients enrolled under the case group were clinically checked by a periodontist and fulfilled the criteria of periodontal disease. After enrolment, detailed demographic, clinical, biochemical, smoking history, alcohol consumption, diastolic blood pressure, systolic blood pressure, and diabetes were evaluated and recorded. Informed written consent was obtained from all the patients and or their caregivers. This study protocol was approved by Institutional Ethics Committee, U.P. (Ref number: 104th ECM II B-Ph.D/PI).

Inclusion criteria

All the subjects aged between 30 and 80 years were included in the study, which were further divided into the following:

Periodontal outcome

1.) 30% of sites with clinical attachment loss
2.) Periodontal pocket depth ≥ 4 mm and presence of bleeding on probing
3.) Gingival inflammation
4.) Bleeding on probing

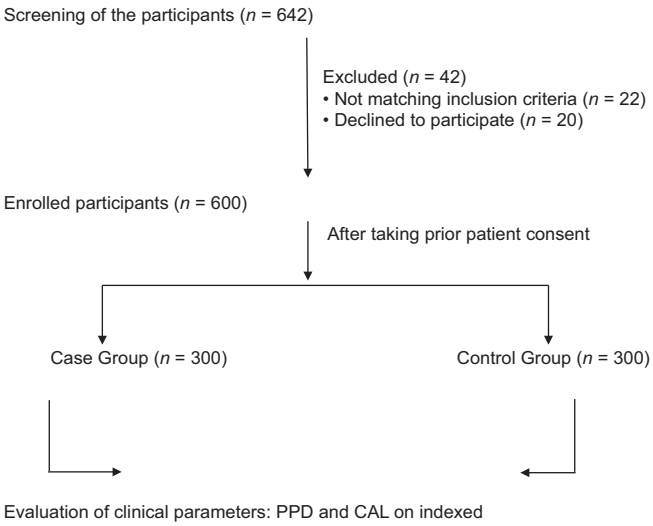
Cardio-vascular disease outcome

1.) Previous history of acute coronary syndrome which has been proven angiographically.
2.) Previous history of stroke and transient ischemic attack (TIA).

Exclusion criteria

1.) Subjects who regularly use mouth washes like chlorhexidine mouth wash.
2.) Subjects who underwent any periodontal treatment for at least 6 months prior to sampling.
3.) Lactating mothers
4.) Pregnant women

Study flow-consort chart



Evaluation of clinical parameters: PPD and CAL on indexed teeth, demographic, clinical profile, and Apo A1 and B100

Anthropometric data such as height, weight, body mass index (BMI), diastolic blood pressure, and systolic blood pressure were also documented. BMI was measured as weight/height^2 (Kg/m^2). Blood pressure was evaluated according to the standard protocol (patients examined after 15 minutes of sitting).

Laboratory analysis

Blood sample collection and storage

A total of 5 ml of the intravenous blood was collected from each participant. Of the 5 ml, 1 ml of blood was collected in a fluoride vial and stored at 4°C for the estimation of glucose. Of the 5 ml blood collected, 3 ml was taken in plain vials for serum, which was separated out by centrifugation at 3000 rpm for 5–10 minutes at room temperature within 30 minutes of blood collection for the estimation of lipid profile and inflammatory markers. Collected samples were labeled and stored at -20°C in a deep freezer.

Biochemical analysis

Lipid parameters such as low-density lipoprotein (LDL) cholesterol were calculated by using the Friedewala formula (Kannan, Mahadevan, Ramji, Jyapaul, and Kumaravel, 2014). Serum triglyceride (TG), high-density lipoprotein (HDL), and total cholesterol (TC) levels were calculated by the enzymatic method (Randox Laboratories Ltd., Antrim, UK). HbA1c was analyzed using a BIO-Rad DIO HPLC system. Lipid levels were measured in milligrams (mg) per deciliter (dL) of blood.

Biochemical estimation

Random plasma glucose

Based on the enzymatic method using the glucose oxidase peroxidase (GOD-POD) method (glucose tests: At a glance, n. d.) following the manufacturer's protocol (Randox Laboratories Ltd., Antrim, UK). The normal value of plasma glucose is <110mg/dl.

Contents	Blank	Standard	Sample
Working reagent	1.0ml	1.0ml	1.0ml
Distilled water	10.0µl	-	-
Standard	-	10.0µl	-
Sample	-	-	10.0µl

Serum lipid profile

Serum lipid profile: Total cholesterol (TC), triglyceride (TG), and high-density lipoprotein cholesterol (HDL-C) were calculated by the enzymatic method (Randox Laboratories Ltd., Antrim, UK), and low-density lipoprotein-cholesterol (LDL-C) was calculated by Friedwald's formula.

Total cholesterol

This was determined by the cholesterol oxidase-peroxidase (CHOD-POD) enzymatic method (Cholesterol: at a glance, n. d.). Cholesterol esterase hydrolyzes cholesterol ester into free cholesterol and fatty acids. In the second reaction, cholesterol oxidase converts cholesterol to cholester-4-en-3-one and hydrogen peroxide. In the presence of peroxide, hydrogen peroxide oxidatively couples with 4-aminoantipyrine and phenol to produce a red color, which is proportional to the amount of TC in specimen. Each sample was analyzed in duplicates, and for further calculation, the average of two similar readings was used. The standard cholesterol concentration was 200 mg%, which was also used with each experiment (Randox Laboratories Ltd., Antrium, UK).

Triglyceride

This was determined by the glycerol phosphate oxidase-peroxidase (GPO-POD) method (Triglyceride: at a glance, n. d.). Glycerol is released from hydrolysis of triglyceride in the presence of lipoprotein lipase. Glycerol kinase converts glycerol into glycerol-3-phosphate, which is oxidized by glycerol phosphate oxidase to dihydroxyacetone phosphate and hydrogen peroxide. In the presence of peroxide, hydrogen peroxidase oxidizes phenolic chromogen to a red-colored compound, which has maximum absorbance at 510 nm. Each sample was analyzed in duplicates, and for further calculation, the average of two similar readings was used. The standard cholesterol concentration was 200 mg%, which was also used with each experiment (Randox Laboratories Ltd., Antrium, UK).

Armamentarium used

- Disposable gloves
- Face mask
- Mouth mirror and explorer (17#23)

- Calibrated periodontal probe UNC-15 probe
- Aneroid sphygmomanometer and stethoscope
- Deep freezer for sample storage
- Disposable syringe
- Vacutainer
- Centrifugation machine
- Pipette and pipette tips
- Automatic analyzer

Lipoprotein A1 (Apo-A1) determination in serum

Principle

This was done following the manufacturer's instruction, Agappe Diagnostic Ltd. Anti-human Apo A1 antisera, when mixed with human serum containing Apo A1, react to cause an absorbance change, which is measured by the immunoturbidometric principle. The change in the absorbance can be interpolated in a calibration curve prepared with different known concentrations of the calibrator.

Lipoprotein B100 (APO-B100) determination in serum

Principle

This was done following the manufacturer's instruction, Agappe Diagnostic Ltd. The reagents containing polyclonal goat antihuman Apo-B100 antibodies, when mixed with the serum sample containing Apo-B100, causes changes in absorbance due to development of turbidity, which is directly proportional to the concentration of Apo-B100 in the sample.

Statistcal analysis

Descriptive statistics was performed by calculating the mean and standard deviation for continuous variables. The software used for the statistical analysis was SPSS (Statistical Package for Social Sciences) version 19.0.

The statistical tests used were as follows:

- Unpaired *t*-test is used for comparison of mean values between two groups when the data follows normal distribution.
- Mann–Whitney U test is used to test whether two samples are likely to derive from the same population (i.e., that the two populations have the same shape).
- Applied Kruskal–Wallis test: The Kruskal–Wallis test is a non-parametric (distribution free) test, which is used to compare three or more groups of sample data.
- The Spearman's correlation rho coefficient is used to measure the strength of association between two variables.
- *P* values < 0.05 were considered to be statistically significant.

RESULTS

The age (in years) of the case group and control group subjects was 54.22 ± 8.84 years and 53.41 ± 8.67 years, respectively [Table 1]. The mean ages of both the groups were comparable, and no statistical difference was found in the case group and control group ($P = 0.258$).

Comparison of the case and control groups for sex ratio revealed no statistically significant inter-group differences [Figure 1]. Thus, the groups were matched with male and female sexes and showed no significant inter-group differences.

Distribution of APO A1 and APO B100

Table 2 shows the APO A1 and APO B100 distribution of patients between the case and control groups. The mean (±SD) of APO A1 in case and control group patients was 240.30 (±70.34) and 310.11 (±41.46), respectively. On the other hand, the mean of APO B100 in case and control group patients was 124.81 (±83.71) and 122.44 (±29.78), respectively [Figure 2]. The Mann–Whitney U test showed a significant difference [Figure 2] between the mean of APO A1 and lipid profile APO B100 of the two groups (Z = -15.36; $P = <0.001$ and Z = -7.08; $P = <0.001$, respectively) [Table 3]. The Spearman's correlation rho coefficient showed a linear correlation between lipid profile APO A1 and different clinical parameters. In the case group, there was a significant positive correlation between lipid profile APO A1 with serum urea, serum creatinine, and plasma glucose random having a correlation coefficient of 0.115 ($P = 0.007$), 0.240 ($P = <0.001$), and 0.138 ($P = 0.017$), respectively. On the other hand, there was a significant negative correlation between lipid profile APO A1 with serum sodium and TGL, having a correlation coefficient of -0.194 ($P = 0.001$) and -0.161 ($P = 0.005$), respectively.

However, in the control group, there was a significant negative correlation between lipid profile APO A1 and

Table 1: Comparison of mean age (in years) of subjects in case group and control group

	Groups						*t*_value	*P*
	Case Group		Control Group		Total			
	Mean	SD	Mean	SD	Mean	SD		
Age (in years)	54.22	8.84	53.41	8.67	53.82	8.76	1.33	0.258

Applied unpaired *t*-test for significance

Table 2: Distribution of APO A1 and APO B100 in case and control groups

	Groups						*Z*	*P*
	Case Group		Control Group		Total			
	Mean	SD	Mean	SD	Mean	SD		
APO A1 (mg/dl)	240.30	70.34	310.11	41.46	275.20	67.44	-15.36	<0.001
APO B100 (mg/dl)	124.81	83.71	122.44	29.78	123.62	62.78	-7.08	<0.001

Applied Mann-Whitney *U* test for significance

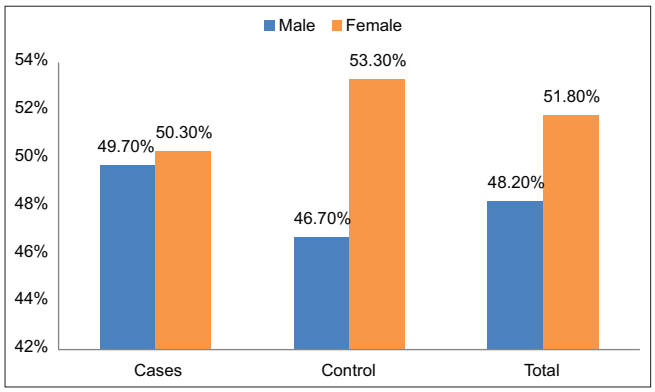


Figure 1: Gender distribution of subjects in the case group and control group

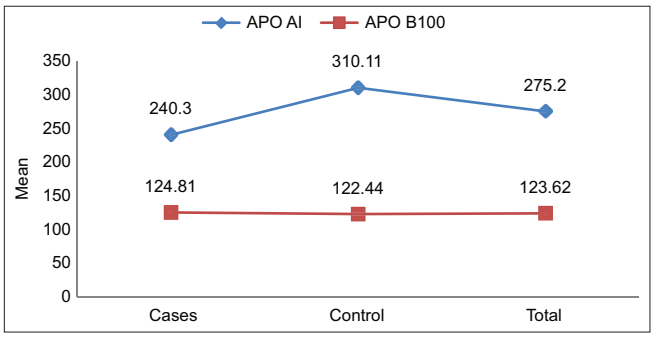


Figure 2: The Mann–Whitney U test showed a significant difference between mean of APO A1 and lipid profile APO B100

plasma glucose random, having a correlation coefficient of -0.124 ($P = 0.032$) [Table 4]. The Spearman's correlation rho coefficient showed a linear correlation in the case group and control group between lipid profile APO B100 and all other different clinical parameters. In the case group, there was a significant positive correlation between lipid profile APO B100 and serum sodium, having a correlation coefficient of 0.113 ($P = 0.050$). There was a significant negative correlation between lipid profile APO B100 and hemoglobin and serum creatinine, having a correlation coefficient of -0.117 ($P = 0.043$) and -0.167 ($P = 0.004$), respectively. However, in the control group, there was a significant negative correlation between lipid profile APO B100 and LDL, having a correlation coefficient of -0.164 ($P = 0.005$).

Correlation of APO A1 with other sub-groups

The association of lipid profile APO A1with sub-groups (single-vessel disease, double-vessel disease, triple-vessel disease, OSA, and stroke) of the case group was evaluated and is summarized in Table 5. The one-way analysis of variance showed that there was a significant difference among means of groups of lipid profile APO A1. On intra-group comparison, Tukey test showed a significant ($P < 0.001$) difference [Figure 3] of OSA with other groups (SVD, DVD, TVD, and stroke).

Correlation of lipid profile APO B100 with other sub-groups

The association of lipid profile APO B100 with sub-groups (single-vessel disease, double-vessel disease, triple-vessel disease, OSA and stroke) of cases is evaluated and is summarized in Table 6. The one-way analysis of variance showed that there was a significant difference among means of groups of lipid profile APO B100. On intra-group comparison, Tukey test showed a significant ($P < 0.001$) difference [Figure 4] of OSA with other groups (SVD, DVD, TVD, and stroke).

Correlation between lipid profile APO A1 and different clinical parameters

Table 7 shows the Spearman's correlation rho coefficient, showing a linear correlation between lipid profile APO A1 and different clinical parameters. In the case group, there was a significant positive correlation between lipid profile APO A1 and serum urea, serum creatinine, and plasma glucose random, having a correlation coefficient of 0.115 ($P = 0.007$), 0.240 ($P = <0.001$), and 0.138 ($P = 0.017$), respectively. On the other hand, there was a significant negative correlation between lipid profile APO A1 and serum sodium and TGL, having a correlation coefficient of -0.194 ($P = 0.001$) and -0.161 ($P = 0.005$), respectively.

However, in the control group, there was a significant negative correlation between lipid profile APO A1 and plasma glucose random, having a correlation coefficient of -0.124 ($P = 0.032$).

Correlation between lipid profile APO B100 and other clinical parameters

Table 8 shows the Spearman's correlation rho coefficient, showing a linear correlation in the case group and control group between lipid profile APO B100 and all other different clinical parameters. In the case group, there was a significant positive correlation between lipid

Table 3: Correlation between lipid profile APO A1 and other clinical parameters in case and control groups

Parameters	Case Group		Control Group	
	Spearman's rho	*P*	Spearman's rho	*P*
Hemoglobin	0.011	0.848	0.021	0.723
Total Leucocyte Count (TLC)	0.030	0.610	-0.040	0.491
Serum Urea	0.155	0.007	-0.021	0.717
Serum Creatinine	0.240	<0.001	-0.071	0.222
Serum Sodium	-0.194	0.001	0.060	0.296
Serum Potassium	-0.067	0.244	0.034	0.560
Serum Ionic Calcium	0.028	0.628	-0.007	0.904
Plasma Glucose Random	0.138	0.017	-0.124	0.032
Cholesterol	-0.059	0.307	-0.087	0.134
TGL	-0.161	0.005	-0.064	0.270
HDL	0.094	0.103	0.072	0.215
LDL	0.001	0.991	0.054	0.352

Table 4: Correlation between lipid profile APO B100 and other clinical parameters in case and control groups

Parameters	Case Group		Control Group	
	Spearman's rho	*P*	Spearman's rho	*P*
Hemoglobin	-0.117	0.043	0.003	0.962
Total Leucocyte Count (TLC)	-0.068	0.244	0.062	0.286
Serum Urea	-0.047	0.421	0.021	0.719
Serum Creatinine	-0.167	0.004	0.065	0.265
Serum Sodium	0.113	0.050	-0.015	0.800
Serum Potassium	-0.023	0.693	-0.024	0.685
Serum Ionic Calcium	0.037	0.525	0.052	0.370
Plasma Glucose Random	-0.056	0.335	0.004	0.943
Cholesterol	0.006	0.914	-0.062	0.287
TGL	0.028	0.624	0.010	0.857
HDL	-0.014	0.814	-0.022	0.707
LDL	-0.046	0.429	-0.164	0.005

Table 5: Association between lipid profile APO A1(mg/dl) and vessels involved in case group

Vessel Involved	Lipid Profile APO A1(mg/dl)		χ^2	*P*
	Mean	SD		
SVD	276.49	29.22	144.88	<0.001
DVD	264.33	40.83		
TVD	270.35	33.66		
Obstructive Sleep Apnea	114.81	25.76*		
Stroke	275.49	25.31		

Applied Kruskal–Wallis test for significance

Table 6: Association between lipid profile APO B100 (mg/dl) and vessels involved in Case Grouphal

Vessel Involved	Lipid Profile APO B100 (mg/dl)		χ^2	*P*
	Mean	SD		
SVD	86.63	25.73	142.08	<0.001
DVD	85.45	28.36		
TVD	85.83	25.96		
Obstructive Sleep Apnea	277.96	54.05*		
Stroke	88.18	25.07		

Applied Kruskal–Wallis test for significance

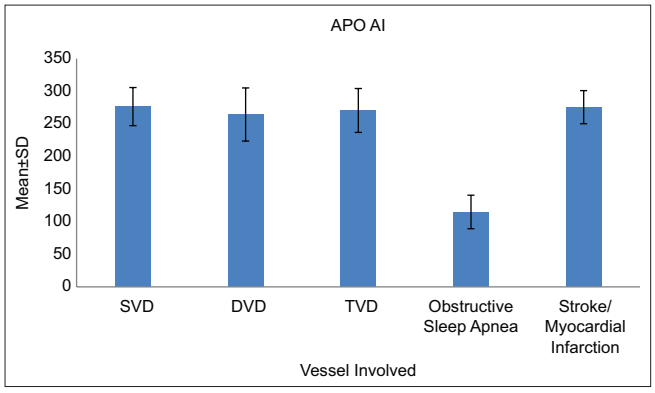


Figure 3: Tukey test showed a significant (*P* < 0.001) difference of OSA with other groups (SVD, DVD, TVD, and stroke)

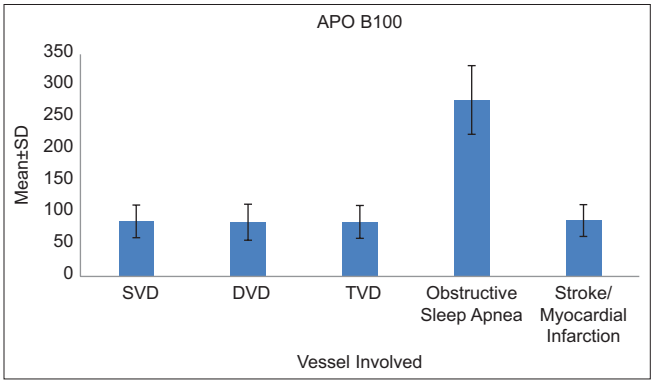


Figure 4: Tukey test showed a significant (*P* < 0.001) difference of OSA with other groups (SVD, DVD, TVD, and stroke)

profile APO B100 and serum sodium, having a correlation coefficient of 0.113 ($P = 0.050$). There was a significant negative correlation between lipid profile APO B100 and hemoglobin and serum creatinine, having a correlation coefficient of -0.117 ($P = 0.043$) and -0.167 ($P = 0.004$), respectively. However, in the control group, there was a significant negative correlation between lipid profile APO B100 and LDL, having a correlation coefficient of -0.164 ($P = 0.005$).

Correlation between pocket probing depth and lipid profile APO A1 and APO B100

Table 9 shows the Spearman's correlation rho coefficient. It shows a linear correlation in the case group and control group between PPD and different markers. In the case group, there was a significant positive correlation between PPD and lipid profile APO A1 having a correlation coefficient of 0.150 ($P = 0.009$). On the other hand, there was a significant negative correlation between PPD with lipid profile APO B100, having a correlation coefficient of -0.157 ($P = 0.007$)

Table 7: Correlation between lipid profile APO A1 and other clinical parameters in case and control groups

Parameters	Case Group		Control Group	
	Spearman's rho	*P*	Spearman's rho	*P*
Hemoglobin	0.011	0.848	0.021	0.723
Total Leucocyte Count (TLC)	0.030	0.610	-0.040	0.491
Serum Urea	0.155	0.007	-0.021	0.717
Serum Creatinine	0.240	<0.001	-0.071	0.222
Serum Sodium	-0.194	0.001	0.060	0.296
Serum Potassium	-0.067	0.244	0.034	0.560
Serum Ionic Calcium	0.028	0.628	-0.007	0.904
Plasma Glucose Random	0.138	0.017	-0.124	0.032
Cholesterol	-0.059	0.307	-0.087	0.134
TGL	-0.161	0.005	-0.064	0.270
HDL	0.094	0.103	0.072	0.215
LDL	0.001	0.991	0.054	0.352

Table 8: Correlation between lipid profile APO B100 and other clinical parameters in case and control groups

Parameters	Case Group		Control Group	
	Spearman's rho	*P*	Spearman's rho	*P*
Hemoglobin	-0.117	0.043	0.003	0.962
Total Leucocyte Count (TLC)	-0.068	0.244	0.062	0.286
Serum Urea	-0.047	0.421	0.021	0.719
Serum Creatinine	-0.167	0.004	0.065	0.265
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Cholesterol	0.006	0.914	-0.062	0.287
TGL	0.028	0.624	0.010	0.857
HDL	-0.014	0.814	-0.022	0.707
LDL	-0.046	0.429	-0.164	0.005

Table 9: Correlation between PPD and different markers in case and control groups

Parameters	Case Group		Control Group	
	Spearman's rho	*P*	Spearman's rho	*P*
Lipid Profile APO AI	0.150	0.009	0.005	0.930
Lipid Profile APO B100	-0.157	0.007	-0.100	0.084

Table 10: Correlation between CAL and different markers in case and control groups

Parameters	Case Group		Control Group	
	Spearman's rho	*P*	Spearman's rho	*P*
Lipid Profile APO AI	-0.004	0.947	0.042	0.464
Lipid Profile APO B100	-0.058	0.314	-0.014	0.811

in the case group. However, in the control group, there was no significant correlation between PPD and different markers.

Correlation between clinical attachment loss and lipid profile APO A1 and APO B100

Table 10 depicts the Spearman's correlation rho coefficient, which shows a linear correlation in the case group and control group between CAL and different markers. In the case group, there was no significant correlation between CAL and different markers.

DISCUSSION

CVD is a major contributor of morbidity and death in adults globally. The development of heart disease can be attributed to hereditary factors and several environmental risk factors such as smoking, age, abnormal serum lipids, diabetes, and hypertension. These known causal factors, individually or in combination, contribute to atherosclerosis, the cause of CVD.[9] In addition to these causal factors, viral and bacterial infections may also contribute to acute thromboembolism in susceptible individuals. Periodontal disease is a series of inflammatory diseases primarily caused by bacteria and their by-products. There is increasing evidence that poor dental health, especially the presence of periodontal disease, increases the risk of CVD.[10]

The present study was conducted with 600 subjects; 300 subjects had chronic periodontitis with patients (cases) with CAD, and 300 subjects had chronic periodontitis only (control). The mean age of the case group was 54.22 ± 8.84 years and 53.82 ± 8.76 years. The study population proved to be homogeneous with respect to gender distribution. It was found that the mean hemoglobin of the control group was significantly higher. No comparable differences were found for mean TLC counts, serum urea, and serum creatinine between the case and control groups, but serum creatinine, serum sodium, plasma glucose, random plasma glucose, and serum potassium were higher in the case group. In a study done by Bokhari *et al*.,[11] subjects with CAD had increased serum glucose, APO A1, creatinine, potassium, triglycerides, APO B100, and red blood cell count compared to controls. However, correlations were exercised through classical risk factors rather than independently.

Notably, our study is the first to illustrate the relationship of Apo A1 and B100 with number of vessels involved in CAD, MI, and OSA in case and control groups. The result of the present study showed that there is a significant difference among means of lipid profile Apo A1 and Apo B100. The intra-group comparison showed significant ($P < 0.001$) OSA with other groups (SVD, DVD, TVD, and Stroke/MI).

According to the study done by **Hua** *et al*.,[12] the increase in ApoB/ApoA1 was a risk factor for double-vessel (OR: 1.681,

95% CI: 1.230–2.296, $P = 0.001$) and triple-vessel (OR: 3.908, 95% CI: 2.900–5.268, $P < 0.001$) disease compared to single-vessel disease.

In another study done by Yaseen *et al*.,[13] Apo B is an independent risk predictor for the severity of CAD in patients with acute coronary syndromes. Moreover, the Apo B/Apo A1 ratio remains highly significant in patients with a high Gensini score.

Similarly, Tian *et al*.[14] studied 2256 patients presented with CAD, and they reported a significant association between ApoB/ApoA1 ratios and Gensini scores among these patients. Another study conducted by Song *et al*.[15] on 792 angiographically defined CAD patients argued that the ApoB/ApoA1 ratios could be a convenient predictor for the coronary stenosis severity in CAD patients.

According to the available data, changes in lipid metabolism are related to oral hygiene practices and periodontal disorders. For instance, a research in Japan involving adult participants indicated that those who self-reported having better oral hygiene practices had lower levels of triglycerides.[16] In the present research, total cholesterol (mg/dl) and triglycerides (TG) (mg/dl) were found to be more in the case group, whereas no difference was seen in HDL and LDL levels among cases and controls. A study by Morita *et al*. (2016)[17] found no significant link between dyslipidemia and periodontitis, but the 4-year cohort study by Morita *et al*. (2010)[18] found a significant relationship between the two factors. When the ORs for increased triglycerides and decreased high-density lipoprotein cholesterol were assessed separately, both were slightly greater in subjects with periodontal pockets.

In Brazil, a case-and-control study found no link between periodontitis severity and potentially associated serum lipid levels due to the limited subject size and selection method.[19] HDL lipoproteins are considered antiatherogenic because they neutralize circulating LPS,[20] prevent LDL oxidation,[21] antagonize cholesterol transport,[22] and pick up cholesterol from cell membranes during its excretion. This process is enabled by passive diffusion of cholesterol into HDL and is actively facilitated by interactions of apolipoprotein A1 (ApoA1), pre-Beta HDL, or ABCA1-poor lipoproteins to facilitate cholesterol removal.[23] In the circulation, HDL cholesterol is esterified and sent straight to the liver for excretion via LDL. Thus, HDL helps cholesterol clearance and impairment of this clearance pathway may be associated with vessel wall thickening and the development of early atherosclerotic lesions in blood vessels.[24] Triglycerides are also increased in inflammation, infection, and the formation of cholesterol-HDL-rich complexes and are substrates for hepatic lipase, which, when activated, stimulate the formation of weak lipids that accelerate catabolism by the kidneys.[25]

Other atherogenic alterations in lipoprotein profiles are also brought on by infection. These might be a component of the pathways relating persistent inflammation to the formation of atherosclerosis. Chlamydia pneumoniae, *Helicobacter pylori*, and the periodontal pathogens P. gingivalis, T. forshytia, and A. actinomycetemcomitans are only a few of the infections that can disrupt lipid metabolism, which is linked to several lipid metabolites.[26] Regarding antibody levels against periodontal pathogens, in this study, T. Forsythia was significantly associated with HDL35 in bivariate analysis, whereas serum IgG1 against P. gingivalis was linked with HDL-35 and serum IgG1 against T. forsythia was linked with TG. Furthermore, high levels of HDL and HDL-35 were associated with the existence of IgG2 antibodies against A. actinomycetemcomitans.[27]

Khader *et al*.[28] reported periodontitis severity as measured by mean pocket probing depth (PPD) and clinical attachment level (CAL), and degree of periodontitis as measured by the percentage of sites with CAL ≥3 mm and the percentage of sites with PPD more than 3 mm. A significant correlation was found between the lipid profiles APO A1 (mg/dl) and APO B100 and the blood vessels involved in the case groups.

Through literature search, we could not find similar studies; hence, many of the parameters in the present study could not be compared with those of the available literature. In the current investigation, there was a link between cardiovascular risk factors and chronic periodontitis. Therefore, those with chronic periodontitis may have a higher risk of developing cardiovascular disorders. The results of the current investigation point to a potential relationship between lipoprotein metabolism and systemic inflammation brought on by periodontitis. It was observed that there is a link between periodontal disease and CVD.

CONCLUSION

APO A1 is the main protein component of HDL. APO A1 activates lecithin cholesterol, acyltransferase which catalyzes the esterification of cholesterol; this can be transported to the liver, metabolized, and excreted. People with atherosclerotic vascular changes frequently exhibit decreased levels of APO A1. Even if the concentrations of lipoprotein B are normal, a decreased APO A1 level may be

a risk factor for atherosclerosis. Decreased levels of APO A1 also occur in dyslipoproteinemias, acutehepatic cirrhosis, and insulin-treated patients.

APO B100 is the main protein component of LDL. It is necessary for the reaction with LDL receptors in the liver and on cell walls and thus involved in transporting cholesterol from the liver to the vessel cells. Elevated levels of APO B100 are frequently found in atherosclerotic vascular changes and are risk factors for atherosclerosis.

Hence, the results of the current investigation point to a potential relationship between lipoprotein metabolism and systemic inflammation brought on by chronic periodontitis. It was observed that there is a link between periodontal disease and lipoproteins, especially APO A1 and APO B100

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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