International Journal Of

Recent Scientific Research

ISSN: 0976-3031

Volume: 7(4) April -2016

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THE OFFICIAL PUBLICATION OF
INTERNATIONAL JOURNAL OF RECENT SCIENTIFIC RESEARCH (IJRSR)
http://www.recentscientific.com/ recentscientific@gmail.com

ISSN: 0976-3031

Available Online at http://www.recentscientific.com

International Journal of Recent Scientific Research
Vol. 7, Issue, 4, pp. 10441-10444, April, 2016

International Journal of Recent Scientific Research

Research Article

COMPARITIVE EVALUATION OF SERUM TITERS OF APOLIPOPROTEIN A1 IN CHRONIC GINGIVITIS AND CHRONIC PERIODONTITIS PATIENTS BEFORE AND AFTER SCALING AND ROOT PLANING

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ARTICLE INFO

Article History:

Received 20th January, 2016
Received in revised form
29th February, 2016
Accepted 30th March, 2016
Published online 28th April, 2016

Keywords:

Serum titers, Apolipoproteins A1, Scaling and Root planing

ABSTRACT

Background: Intact bacteria or bacterial products, including lipopolysaccharide (LPS), in the periodontal tissue may enter the bloodstream through inflamed periodontal tissue or via saliva. High-density lipoprotein (HDL) is considered as an antiatherogenic lipoprotein because of its direct role in neutralizing LPS in circulation and protecting low density lipoprotein (LDL) against oxidation as well as its role in reverse cholesterol transport. Apolipoproteins are proteins that bind to lipids to form lipoproteins; its main function is transporting lipids.

Aim: The aim of the present study is to compare and evaluated serum titers of Apolipoprotein A1 in both chronic gingivitis and chronic periodontitis patients before and after scaling and root planing.

Materials and Methods: The study included a total of 40 patients in which 20 are patients with chronic gingivitis and 20 are patients with chronic periodontitis. 5ml of non fasting blood was collected from the patients at baseline (before scaling and root planning), and after 3 months and sent to lab for evaluation of Apolipoprotein A1 (APO A1).

Results: The Plaque index, gingival indices, Probing depth and Clinical attachment level are measured. The mean value of Apolipoprotein A1 in group A and group B is increased from baseline to 3 months of follow up. The p value is statically significant.

Conclusion: The results showed that the mean value of Apolipoprotene A1 in group A and group B is increased from baseline to 3 months before and after scaling and root planing.

INTRODUCTION

Periodontitis is defined as an inflammatory disease of supporting tissues of teeth caused by specific microorganisms or groups of specific microorganisms, resulting in progressive destruction of the periodontal ligament and alveolar bone with periodontal pocket formation, gingival recession or both. Intact bacteria or bacterial products, including lipopolysaccharide (LPS), may enter the bloodstream through inflamed periodontal tissue or via saliva. Bacterial dissemination, further potentiated by gastrointestinal microbiota, may result in endotoxemia and low-grade inflammation.[1]

Lipopolysaccharide (LPS) -macrophage-inflammatory mediators plays a critical role in infection-associated atherogenesis and thromboembolism by inducing the release of cytokines, by enhancing platelet aggregation and endothelial monocyte adhesion, and by promoting the formation of lipid-laden foam cells[2]. Lipopolysaccharide (LPS) also interferes with macrophage cholesterol metabolism by down regulating both scavenger receptor B1 and ATP binding cassette transporter A1 (ABCA1) expression[3]. The functions of these two transmembrane proteins have been associated with cholesterol efflux.

High-density lipoprotein (HDL) is positively associated with a decreased risk of coronary heart disease (CHD). HDL is considered an antiatherogenic lipoprotein because of its direct role in neutralizing LPS in circulation and protecting LDL against oxidation[4] as well as its role in reverse cholesterol transport[5].

Apo lipoproteins (APO A1) are proteins that bind to lipids to form lipoproteins; its main function is transporting lipids. These proteins are important in maintaining the structural integrity and solubility of lipoproteins and play an important role in lipoprotein receptor recognition and the regulation of certain enzymes in lipoprotein metabolism. There are six major

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classes of apolipoproteins: A, B, C, D, E and H. Apo A1 is the major protein component of high-density lipoprotein (HDL). Deficiency of Apolipoproteins (APO A1) is associated with HDL deficiencies, including Tangier disease and systemic non-neuropathic amyloidosis. It may have a role in protection against Alzheimer's disease and interact to modify triglyceride levels in coronary heart disease patients.

As it is known that chronic inflammation reduces the level of high density lipoproteins (HDLs) in blood (Lippi G., *et al* 1998), serum Apolipoprotenen A1 value is measured in the present study to evaluate the level of high density lipoproteins (HDLs) in the blood of chronic gingivitis and chronic periodontitis patients as they are chronic inflammatory conditions. And Apolipoprotein A1 is selected for measuring as it is a major component of high density lipoproteins (HDL).

The aim of the present study is to evaluate the serum Apolipoprotein A1 (APO A1) level in both chronic gingivitis patients and chronic periodontitis patients before and after scaling and root planing

MATERIALS AND METHODS

Study Design

The study included a total of 40 patients in which 20 are patients with chronic gingivitis and 20 are patients with chronic periodontitis. All the patients were selected randomly who attended the outpatient department of Periodontics and implant dentistry, St. Joseph Dental College and Hospital, Eluru. The patients with chronic gingivitis are grouped as Group A and with chronic periodontitis are grouped as Group B. All the clinical parameters such as Plaque index (Loe and Silness 1964), Gingival index (Loe and Silness 1963), Probing depth (measured with Williams periodontal probe), Clinical attachment level. (Table 1)

Table 1 Mean Values of Clinical Parameters In Group A And Group B

Clinical Parameters	Group A (mean values)		Group B (mean values)	
	Baseline	3 Months	Baseline	3 Months
Plaque index(pi)	0.861±0.257	0.439±0.171	1.291±0.277	0.807±0.232
Gingival index(gi)	0.874±0.259	0.518±0.202	1.614±0.241	1.037±0.122
Probing depth(pd)	2.7±0.47	2.4±0.502	8±1.59	6.3±1.592
Clinical attachment level(cal)	2.7±0.47	2.4±0.502	8.3±2.1	6.6±2.06

Site Selection

Preoperative and postoperative Probing depth of the deepest pocket was measured.

Inclusion and Exclusion Criteria for the Study

Patients with chronic generalized periodontitis with at least eight teeth having probing depth of > 5 mm and attachment loss of > 2 mm were included. The exclusion criteria for the study is as follows 1)patients with systemic diseases,2) Patients with smoking habit, 3) pregnant patients,4) lactating mothers, and 5) diabetic patients.

Ethical committee approval was obtained from St. Joseph Dental College and Hospital Eluru and a written informed consent was taken from all the patients who were willing to participate in the study.

Study procedure

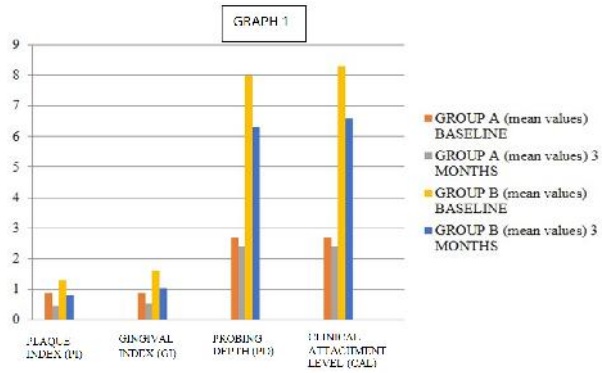
Collection of blood samples

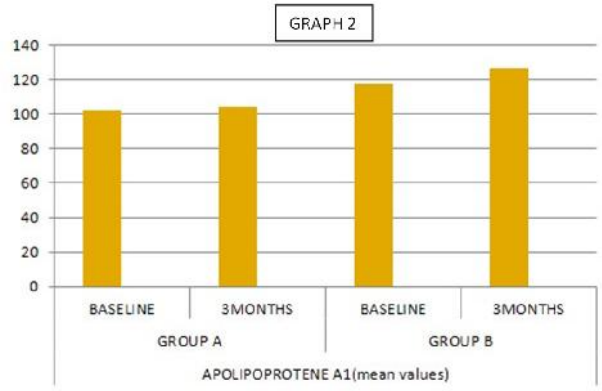
5ml of non fasting blood was collected from the antecubital fossa of all the patients at baseline (before scaling and root planning), after the collection of blood it is centrifuged and the serum was separated and refrigerated at 35^0 Fahrenheit and sent to lab for evaluation of Apolipoproteins (APO A1). Scaling is performed on the same day and after 1 week subgingival scaling and root planing is performed. The patient is recalled after 3 months. The second sample of blood was then collected in the same procedure.

The serum samples were analyzed for ApolipoproteneA1 by nephlometry.

Stastical Analysis

Stastical analysis was done using graph pad prism (version 6.1). The Plaque index, Gingival index, Probing depth, Clinical attachment level were measured at baseline and 3 months (Table1), (graph 1). Apolipoprotein A1 level are measured in both the groups using at baseline, 3 months(graph 2) and the p value is recorded using paired and unpaired t test.





RESULTS

The Plaque index, gingival indices, Probing depth and Clinical attachment levels of both the groups are measured (graph 1). The mean value of Apolipoprotein A1 in group A and group B is increased from baseline to 3 months of follow up (graph 2). The p value is statically significant in both the groups but more significant for group B than group A.

Table 2 Mean Values of Apolipoprotene A1 in Group A and Group B

Apolipoprotene A1(mean values)			
Group A		Group B	
Baseline	3Months	Bsaeline	3Months
102.1±7.51	104.5±9.37	117.8±9.63	126.2±12.8

Table 3 P Values of All the Clinical Parameters

P values	Group a(pre op and post op)	Group b(pre op and post op)
Plaque index(PI)	< 0.0001	< 0.0001
Gingival index (GI)	< 0.0001	< 0.0001
Probing depth(PD)	0.1105	< 0.0001
Clinical attachment level(CAL)	0.1105	< 0.0001
Apolipoprotene A1 (individual)	0.0038	0.0063
Apolipoprotene A1 (inter group)	Pre Op Group A AND B < 0.0001	Post Op Group A AND B < 0.0001

DISCUSSION

Apolipoprotein A1 is the major protein component of high density lipoprotein (HDL) in plasma. Apolipoprotein A-I (ApoA-I), play a role in Reverse Cholesterol Transport (RCT). The efficiency of RCT depends on the specific ability of ApoA-I to promote cellular cholesterol efflux, bind lipids, and activate lecithin: cholesterol acyltransferase (LCAT), and form matureHDL that interacts with specific receptors and lipid transfer proteins[6]. Periodontitis further causes changes in the activities of HDL conversion factors, which is paralleled by alterations in HDL composition and subclass distribution leading to an impaired capacity of HDL during reverse cholesterol transport[7].

In the present study Apolipoprotene A1 was measured in chronic gingivitis patients and chronic periodontitis patients before and after SRP (scaling and root planning).The levels of HDL cholesterol and APO-A1 are decreased during acute and chronic inflammatory conditions[8] and HDL can bind and neutralize endotoxin (lipopolysaccharide) that is directly involved in mediating the inflammatory process[9]. HDL can also inhibit the formation of foam cells by promoting cholesterol efflux from monocytes/ macrophages.

The mechanisms behind the association between periodontitis and atherosclerosis are largely unknown, but may include bacteria-derived activation of inflammatory reactions systemically and in the arterial wall, enhanced cytokine production, low-density lipoprotein (LDL) modifications, lipoprotein-derived cholesterol/cholesteryl ester (CE) enrichment of macrophages,[10] and adverse alterations in the lipoprotein profile resulting in decreased levels of anti-atherogenic, high-density lipoprotein (HDL)[7].In addition to its other anti-atherothrombotic functions, HDL can inhibit cytokine- induced expression of adhesion molecules by endothelial cells. These effects could explain, at least in part, the influence of inflammatory periodontal disease on HDL levels and the greater ability of the ‘‘two-pronged’’ treatment, i.e., bacterial ‘‘load’’ reduction by SRP[11] to reduce more effectively the severity of chronic periodontitis and the risk of low HDL levels. The increasing amounts of HDL cholesterol and APO-A in both groups are important because it has been reported that every 1 mg/dl increase in HDL cholesterol is associated with a 2–3% decrease in CAD risk, independent of LDL cholesterol and triglyceride levels (Castelli *et al*. 1986). It has also been reported that HDL cholesterol could be a therapeutic target due to this protective effect (Toth 2005).

Serum is selected as it is usually used for the detection of active phases of periodontal disease and to identify individuals at higher risk for future disease occurrence. In the current study we chose to evaluate the Apolipoprotein A1 levels in serum as it is easy to procure. With the advent of highly sensitive techniques, traces of markers can be accurately established in Serum.

Emerging evidence shows that periodontaltreatment could significantlyimprove endothelial function [12, 13, 14], but the level of improvementdoes not correlate with the changeof inflammatory biomarkers like hs-CRP and interleukin (IL)-6 [13, 15]. They are numerous studies which prove that SRP reduces the PI, GI, PD and CAL in chronic periodontitis patients[16, 17, 18].

In the present study the p values of all the clinical parameters of chronic periodntitis patients are significant (<0.001) and for chronic gingivitis patients it is significant for PI and GI but no significant for PD (0.1105), and CAL (0.1105). The p value for intra group serum ApolipoproteneA1 is significant in both chronic gingivitis and chronic periodontitis patients but it is more significant in chronic periodontitis patients than in chronic gingivitis patients.

CONCLUSION

The present study is done to evaluate the serum titers of Apolipoprotene A1 in chronic gingivitis and chronic periodontitis patients before and after scaling and root planing. Results showed that the mean value of Apolipoprotene A1 in group A and group B is increased from baseline to 3 months before and after scaling and root planning but, there is a significant increase in group B than in group A. Further long term studies with larger sample size are required for confirmation of results of the present study.

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How to cite this article:

Musalaiah S.V.V.S *et al*.2016, Comparitive Evaluation of Serum Titers of Apolipoprotein A1 In Chronic Gingivitis And Chronic Periodontitis Patients Before And After Scaling And Root Planing. *Int J Recent Sci Res.* 7(4), pp. 10441-10444.

TSSN 0976-3031