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A Comparative Evaluation Of Serum Lipid Profile And Uric Acid Levels In Chronic And Aggressive Periodontitis Patients Before And After Non Surgical Periodontal Therapy .

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Abstract

Introduction: Periodontal disease is an infectious disease which results in an increase in inflammatory cytokines, leading to mobilization of lipids from the liver and adipose tissue.

Aim: Aim of the present study was to evaluate serum lipid profile and uric acid levels in patients with aggressive and chronic periodontitis before and after non-surgical periodontal therapy.

Materials and methods: Ten patients in Group A with chronic generalized gingivitis, Group B chronic periodontitis patients having a mean pocket depth of 6mm, in Group C, aggressive periodontitis patients with a mean pocket depth of 7mm. were . Blood was collected at baseline and 3 months after scaling and root planning.

Results: The results revealed that there was a reduction in PI, GI, probing depth, CAL. The lipid parameters and uric acid levels improved after non-surgical periodontal therapy in Group B and Group C patients.

Conclusion: Successful non-surgical periodontal therapy improves serum lipid concentration and increases serum uric acid levels

Key words: Serum lipid profile, Serum uric acid, Chronic periodontitis, Aggressive periodontitis, Reactive oxygen species, Anti-oxidant

Introduction

Periodontal disease refers to the inflammatory processes that occurs in the tissues surrounding the teeth in response to bacterial accumulations, or dental plaque, on the teeth. The bacterial accumulations cause an inflammatory response from the body which to alveolar bone destruction and tissue loss[1]

Reactive oxygen species (ROS) have emerged as an important signalling molecules in various cellular processes. These molecules which are originated from molecular oxygen predominantly produce cellular damage (proteins, lipids, and DNA) if not neutralized by anti-oxidant substances. More recently, it has been proposed

that ROS play a crucial role in periodontal tissue destruction, the individuals suffering from periodontitis might be at higher risk of developing other chronic systemic inflammatory diseases, like cardiovascular diseases and diabetes.[2]

Various biomarkers apart from clinical appearance have been used to evaluate periodontal destruction which includes enzymes, proteins, host cells, markers of cellular and humoral activity, ions, hormones, and markers of oxidative stress and antioxidants. Loss of balance between reactive oxygen species and antioxidant defence has also been implicated as an etiologic factor for periodontal diseases, which may manifest an increase in oxidative stress, reduction of total antioxidant capacity, or decrease in individual antioxidant level.[3]

Uric acid the most important non-enzymatic antioxidant present in saliva correlates with plasma uric acid, used to determine the effect of antioxidant therapy in the treatment of periodontal diseases.[4]

Increased serum pro-inflammatory cytokines produces alterations in lipid metabolism, such as increased LDL-C and triglycerides, which is due to increased hepatic lipogenesis, lipolysis from adipose tissue, or reduced blood clearance. Therefore these serum pro-inflammatory cytokines may play an important role in the association between periodontal disease and hyper-lipidemia. [5]

The aim of the present study was to evaluate serum lipid profiles and serum uric acid in patients with aggressive and chronic periodontitis before and after non-surgical periodontal therapy.

Materials And Methods

A total of 30 patients who attended the department of periodontics, st joseph dental college from . were categorized into 3 groups: Group A included 10 patients with gingivitis ,Group B with chronic periodontitis and Group C with aggressive periodontitis.

The patients were otherwise healthy, with no history of major illness, consumption of antioxidants, antibiotics, anti-inflammatory or any other drugs, who had not received any periodontal therapy for at least 3 months prior to the study. Ethical committee clearance and Informed consent from the patients was taken for all the patients before commencement of the study. Periodontal indices such as plaque index (PI), gingival index(GI), probing depth (PD) and clinical attachment level(CAL)was noted.5ml of blood was collected from the anti-cubital fossa by venipuncture using 20 gauge needle. The collected serum was evaluated for lipid profile and uric acid.

All the patients including Group A, Group B and Group C underwent clinical examination, serum evaluation, periodontal therapy(scaling/scaling and root planning) and are recalled after 3 months.

Statistical Analysis

Paired t-test was applied for intra group comparison , Unpaired t-test was applied for inter group comparison. Significance was tested bycalculating p value (0.005).

Results

The mean values of Plaque Index, Gingival Index, Clinical Attachment Level, Probing Pocket Depth showed significance in groups B and C as shown in table 1 and 2. The mean values of serum lipids showed significance in both the groups B,C with P values [<0.0001,0.0006] respectively and the mean values of serum uric acid showed significance in both the groups B,C with P values[<0.0001, <0.0001] respectively as shown in table 1 and 2. Group B and Group C showed slight decrease in lipid profiles and uric acid post operatively than group A which was non significant(>0.005).

Table1: Comparison of mean, standard deviation and P values of PI, GI, PPD, CAL and uric acid in group A,B and C.

Variable	Group	Base Line					At 3 months					P-value
		N	Minimum	Maximum	Mean	SD	N	Minimum	Maximum	Mean	SD	
CAL	A	10	2.0	3.0	2.20	0.42	10	2.0	3.0	2.20	0.42	1.0
	B	10	6.0	7.0	6.40	0.52	10	3.0	4.0	3.60	0.52	<0.001
	C	10	6.0	8.0	7.00	0.47	10	4.0	5.0	4.50	0.53	<0.001
GI	A	10	1.0	1.8	1.35	0.24	10	1.0	1.4	1.22	0.13	.064
	B	10	1.6	2.3	1.99	0.23	10	1.0	1.5	1.22	0.15	<0.001
	C	10	1.7	2.3	2.04	0.23	10	1.2	1.5	1.39	0.13	<0.001
PD	A	10	2.0	3.0	2.20	0.42	10	2.0	3.0	2.20	0.42	1.0
	B	10	6.0	7.0	6.20	0.42	10	3.0	4.0	3.50	0.53	<0.001
	C	10	6.0	7.0	6.70	0.48	10	4.0	5.0	4.30	0.48	<0.001
PI	A	10	1.0	1.8	1.36	0.24	10	.9	1.3	1.06	0.15	<0.001
	B	10	2.5	3.0	2.85	0.17	10	.9	1.5	1.25	0.20	<0.001
	C	10	2.5	3.0	2.87	0.17	10	1.0	1.5	1.31	0.17	<0.001
SUA	A	10	2.5	3.8	3.19	0.44	10	2.5	3.9	3.30	0.48	.09
	B	10	3.5	4.6	3.89	0.46	10	3.5	5.5	4.63	0.66	.006
	C	10	3.5	4.6	3.95	0.45	10	4.5	5.9	5.41	0.48	<0.001

Graph1: Comparison of mean, standard deviation and P values of PI, GI, PPD, CAL and uric acid in Group A, B and C.

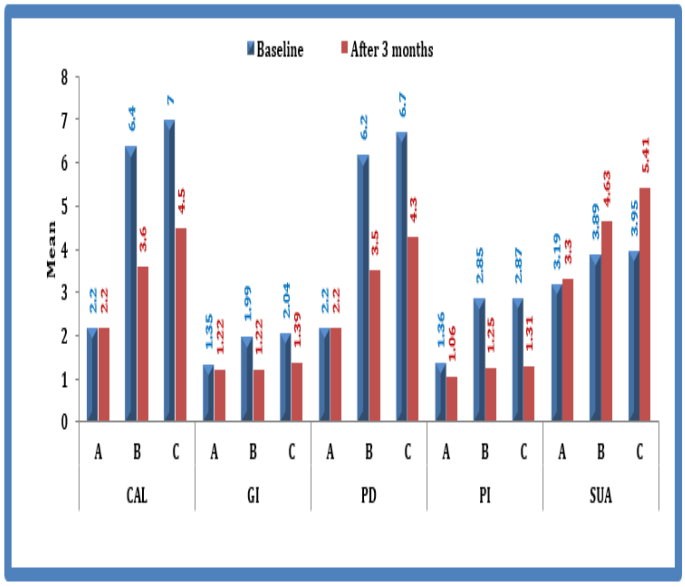

Table 1 & Graph 1 show that the clinical parameters reduced and serum uric acid levels increased after 3 months of non-surgical periodontal therapy.

Table2: Comparison of mean, standard deviation and P values of Lipid profiles in Group A, B and C

Variable	Group	Base Line					At 3 months					P-value
		N	Minimum	Maximum	Mean	SD	N	Minimum	Maximum	Mean	SD	
TC	A	10	143.0	160.0	150.80	6.56	10	140.0	157.0	148.60	5.42	0.1
	B	10	190.0	220.0	205.50	8.17	10	150.0	201.0	173.60	19.60	<0.001
	C	10	210.0	237.0	219.40	7.06	10	170.0	221.0	197.20	13.01	<0.001
TG	A	10	80.0	130.0	110.80	17.24	10	75.0	130.0	110.10	17.58	.73
	B	10	120.0	157.0	150.00	10.99	10	118.0	150.0	138.90	9.34	<0.001
	C	10	150.0	180.0	160.00	8.16	10	132.0	150.0	143.30	6.48	.001
HDL	A	10	59.0	66.0	62.60	2.55	10	55.0	68.0	63.60	3.69	.35
	B	10	42.0	57.0	51.80	5.29	10	58.0	67.0	61.50	2.76	<0.001
	C	10	32.0	56.0	43.40	7.99	10	54.0	66.0	60.40	3.31	<0.001
LDL	A	10	85.0	100.0	95.00	4.69	10	80.0	98.0	92.70	5.17	.11
	B	10	103.0	123.0	112.10	6.05	10	90.0	110.0	99.00	4.97	<0.001
	C	10	109.0	133.0	117.70	7.36	10	97.0	112.0	103.60	5.02	<0.001
VLDL	A	10	20.0	28.0	23.70	2.36	10	20.0	27.0	23.30	2.67	.72
	B	10	31.0	36.0	33.30	1.49	10	22.0	30.0	28.00	2.31	<0.001
	C	10	33.0	38.0	35.40	1.58	10	26.0	32.0	29.40	1.84	<0.001

Graph 2: Comparison of mean, standard deviation and P values of Lipid profiles in Group A, B and C.

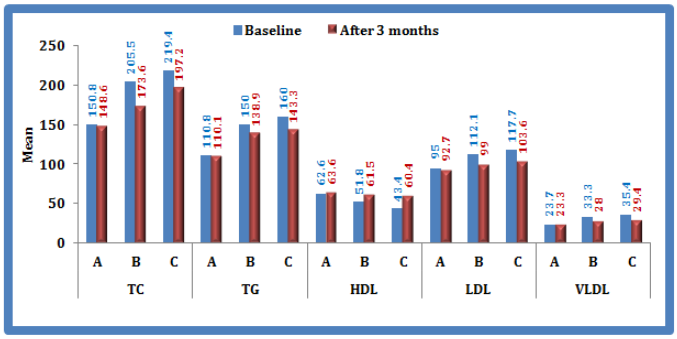

Table 2 & Graph 2 show that the serum lipid levels TC(Total cholesterol),TG(Tri glycerides),LDL(Low density lipo protein),VLDL (Very Low density lipo protein),decreased while serum HDL((High density lipo protein), increased after non-surgical periodontal therapy.

Table 3: Intergroup comparison of mean, standard deviation and P values of PI, GI, PPD, CAL and uric acid in group A,group B and group C.

Variable	Group	Mean difference from base to 3 months	SD	A vs B vs C	A vs B	A vs C	B vs C
PI	A	0.30	0.18	<0.001	<0.001	<0.001	0.92
	B	1.60	0.23				
	C	1.56	0.27				
GI	A	0.13	0.19	<0.001	<0.001	<0.001	0.53
	B	0.77	0.31				
	C	0.65	0.23				
PD	A	0.00	0.47	<0.001	<0.001	<0.001	0.47
	B	2.70	0.67				
	C	2.40	0.52				
CAL	A	0.00	0.47	<0.001	<0.001	<0.001	0.52
	B	2.80	0.63				
	C	2.50	0.71				
SUA	A	0.11	0.19	<0.001	0.02	<0.001	0.007
	B	0.74	0.65				
	C	1.46	0.49				

Graph 3:Intergroup comparison of mean, standard deviation and P values of PI, GI, PPD, CAL and uric acid in group A, group B and group C.

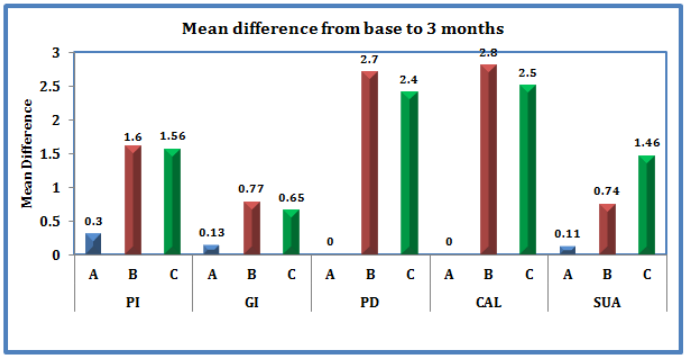

Table 3 & Graph 3 shows that the p value of clinical parameters was significant between Groups A and B, and Groups A and C, but not not between Groups B and C.

Table 4: Intergroup comparison of mean, standard deviation and P values of PI, GI, PPD, CAL and uric acid in Group A, Group B and Group C.

Variable	Group	Mean difference from base to 3 months	SD	A vs B vs C	A vs B	A vs C	B vs C
TC	A	2.20	3.79	<0.001	<0.001	0.002	0.18
	B	31.90	16.61				
	C	22.20	11.48				
TG	A	0.70	6.24	<0.001	0.02	<0.001	0.27
	B	11.10	6.06				
	C	16.70	10.57				
HDL	A	-1.00	3.23	<0.001	0.005	<0.001	0.02
	B	-9.70	5.72				
	C	-17.00	7.26				
LDL	A	2.30	4.03	<0.001	0.002	0.001	0.93
	B	13.10	6.62				
	C	14.10	7.81				
VLDL	A	0.40	3.47	<0.001	0.001	<0.001	0.84
	B	5.30	2.06				
	C	6.00	2.62				

Graph 4: Intergroup comparison of mean, standard deviation and P values of PI, GI, PPD, CAL and uric acid in Group A, Group B and Group C.

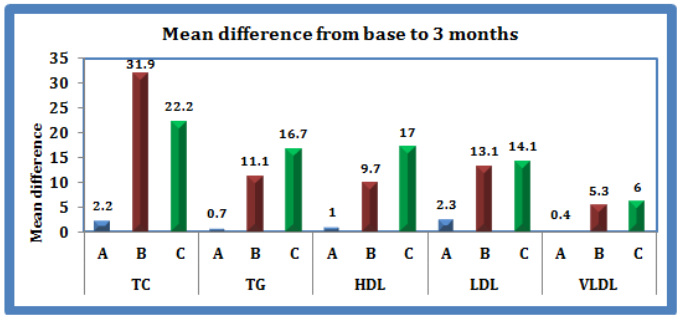

Table 4 & Graph 4 shows that the p value of lipid parameters was significant between Groups A and B, and Groups A and C, but not not between Groups B and C.

Discussion

Serum lipid profile, a major risk factor for developing risk of cardiovascular diseases ,is set to become the major causes of death worldwide. Hyperlipidemia, a condition of high levels of lipid in the blood, is considered as one of the major cardiovascular disease risk factors .[5]

Cytokines are regulators of host responses to infection, immune responses, inflammation, and trauma. Some cytokines act to make disease worse (proinflammatory), whereas others serve to reduce inflammation and promote healing (anti-inflammatory). An increase in pro-inflammatory cytokines in response to chronic periodontitis causes a rise in serum lipid levels according to study done by **Iacopino & Cutler** in 2000. They said that infection with Gram negative periodontal pathogens could prompt release of systemic IL-1β and TNF-α, causing chronic hypertriglyceridaemia.[6]

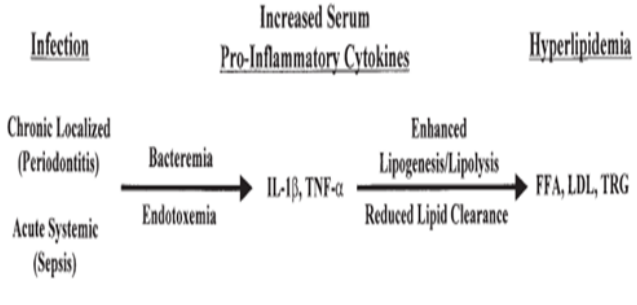

Fig. Linkage between infection and hyperlipidemia. Infection (chronic localized or acute systemic) causes bacteremia and/or endotoxemia producing a cytokine cascade that leads to increased levels of pro-inflammatory cytokines. These biological signalling molecules have physiological effects promoting increased lipogenesis, increased lipolysis and reduced lipid clearance. The end result is hyperlipidemia or an accumulation of serum FFA, LDL and TRG.

Oxidative stress lies at the heart of the periodontal tissue damage that results from host–microbial interactions, either as a direct result of excess ROS activity/antioxidant deficiency or indirectly as a result of the activation of redox-sensitive transcription

factors and the creation of a pro-inflammatory state. Hence, uric acid which is an anti-oxidant can be used as a marker for periodontitis. [7]

The aim of the present study was to evaluate serum lipid profiles and serum uric acid in patients with aggressive and chronic periodontitis before and after non-surgical periodontal therapy.

Non surgical Periodontal therapy has resulted in a significant reduction of local inflammation reduced pocket depth and reduced bleeding indices. . All lipid parameters improved after the periodontal treatmental therapy in this present study. The levels of lipoproteins after therapy seemed to be lower than those reported before treatment in pat ients with periodontitis compared with healthy ones. Lipoproteins were significantly decreased after treatment except high-density lipoprotein cholesterol which was increased.

The increase in HDL levels agree with the study conducted by Pussinen et al, in which it was considered HDL as an anti atherogenic lipoprotein because of its direct role in neutralizing LPS in circulation and protecting LDL against oxidation as well as its role in reverse cholesterol transport . Periodontitis may diminish the antiatherogenic potency of HDL, which may result in increased risk for CHD . In the present study the HDL-mediated cholesterol efflux tended to be higher after periodontal treatment; interestingly, this increase was significant among patients whose C-reactive protein decreased.[8]

There was an improvement in serum lipid levels and a decrease in serum pro-inflammatory cytokines following periodontal therapy which was in accordance by the study conducted by **Yong-Wei Fu et al.**[5] The study even supports the hypothesis that PD is significantly associated with reduction in HDL and elevation of LDL and triglyceride concentrations and hence the rationale that periodontal diseases are associated with lipid metabolic control, in accordance to the study conducted by **Rafael Nepomuceno et al**.[9]

Ovidiu Nicolaiciuc et al conducted a study, in which it was stated that chronic infections, including periodontitis, may modify the serum lipid profile in a way that increases the risk of atherosclerosis. Treatment of periodontitis may result in a potential benefic effect on the metabolic control of hyperlipidemia, reducing the risk for cardiovascular diseases.[10]

In the present study, the uric acid levels were seen to be reduced in patients with periodontitis and increased after therapy. This was in accordance to the study conducted by **Chapple** [7] who implicated oxidative stress in the pathogenesis of periodontitis i.e, the loss of balance between reactive oxygen species and antioxidant defense is an etiologic factor for periodontal diseases.

The uric acid levels were higher in aggressive periodontitis than the chronic periodontitis patients in this study. This is in accordance to the study conducted by **Moore, *et al*.**[11] who stated that uric acid is a major antioxidant in saliva & serum and contributes approximately to 70% of the total salivary and serum

antioxidant capacity and that the levels of antioxidants vary according to the severity of disease, and similarly uric acid levels also vary according to the severity of periodontal disease.

Results of the present study showed that there was a significant increase in uric acid levels in all the groups B, and C, i.e , chronic and aggressive periodontitis groups 3 months after non-surgical periodontal therapy. **DiabLadki, *et al*.**[12] in a similar study also found that there was a slight decrease in the concentration of three main antioxidants (uric acid, ascorbic acid, and albumin) in saliva with the increase in severity of periodontal disease.

Conclusion

This study shows that periodontal disease significantly affects the serum levels of lipoproteins and serum uric acid , and successful non-surgical periodontal therapy decreases serum lipid concentration and increases serum uric acid levels .However Further investigations with larger sample sizes are needed to conform or reject the above findings and for further exploration of the relationship among chronic & aggressive periodontitis, non-surgical periodontal therapy, and lipid and uric acid metabolism.

References

1. Periodontal disease and systemic conditions: a bidirectional relationship; Jemin Kim and Boston University Goldman School of Dental Medicine, Department of Periodontology and Oral Biology, Boston, MA, USA.
2. Oxidative Stress, Systemic Inflammation, and Severe Periodontitis; F. D'Aiuto*, L. Nibali, M. Parkar, K. Patel, J. Suvan, and N. Donos, J Dent Res 89(11):1241-1246, 2010.
3. Antioxidant therapy as monotherapy or as an adjunct to treatment of periodontal diseases Aditi Mathur, Lalit Mathur, Balaji Manohar, Hemant Mathur1 , Rajesh Shankarapillai, Neema Shetty, Aman Bhatia.
4. Salivary Thiocyanate, Uric Acid and pH as Biomarkers of Periodontal Disease in Tobacco Users and Non-Users- An InVitro Study; Fawaz Pullishery[1] , Ganesh Shenoy Panchmal[2] , Sabin Siddique[3]
5. Effects of periodontal therapy on serum lipid profile and proinflammatory cytokines in patients with hyperlipidemia: a randomized controlled trial; Fu YW,Li XX,Xu HZ,Gong YQ,Yng Y.
6. Pathophysiological relationships between periodontitis and systemic disease: Recent concepts involving serum lipids; Iacopino et al, J Periodontal, August 2000.
7. The role of reactive oxygen and antioxidant species in periodontal tissue destruction; Iain L. C. Chapple, Periodontology 2000, Vol. 43, 2007, 160–232
8. Periodontitis decreases the antiatherogenic potency of high density lipoprotein; Pussinen et al, Journal of Lipid Research Volume 45, 2004
9. Serum lipid levels in patients with periodontal disease. A meta-analysis and meta-regression; Rafael Nepomuceno et al, doi: 10.1111/jcpe.12792
10. Study On The Effects Of Classical Periodontal Therapy On The Lipid Profile In Hyperlipidemic And Chronic Periodontitis Patients ;Ovidiu Nicolaiciuc1, Volume 6, Issue 3 July / September 2016.
11. Antioxidant Activity Of Saliva And Periodontal Disease; Suzanne Moore et al, Free Rad. Res., Vol. 21, No. 6, pp. 417-425
12. Decrease in the total antioxidant activity of saliva in patients with periodontal diseases; Randa Diab-Ladk et al, Clin Oral Invest (2003) 7:103–107 DOI 10.1007/s00784-003-0208-5.