

Original Research Article

Lipid Levels in Patients with Oral Lichen Planus: A Case-Control Study

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ABSTRACT

Aim: The aim of this study was to analyse cardiovascular risk posed by dyslipidemia in patients with OLP.

Materials and Methods: This case-control study was based on 120 patients, visiting Department of Oral Medicine and Radiology: 60 with oral lichen planus and 60 controls which matched the cases in all demographic characteristics. The diagnosis of OLP was made on clinical examination and confirmed histopathologically by biopsy. The diagnosis was in accordance to the modified WHO criteria. Serum was analysed for: Serum Cholesterol, Triglycerides, HDL, LDL and VLDL.

Results: The Serum Cholesterol, Serum triglycerides, LDL and VLDL in OLP group were higher as compared with control whereas HDL in OLP group was low when compared with control group.

Conclusion: In conclusion, the results obtained indicate an association between OLP patients and dyslipidemia. The patients with LP therefore should be evaluated for cardiovascular risk posed by dyslipidemia.

Key words: Oral lichen planus, Lipid level.

INTRODUCTION

Oral lichen planus is a chronic immunological mucocutaneous disorder of unknown etiology with prevalence in general population ranging from 0.2 to 2%.^[1] OLP is a T-cell mediated autoimmune disease in which the auto-cytotoxic CD8 + T cells trigger apoptosis of the basal cells of the oral epithelium.^[2] In the majority of patients with oral lichen planus (OLP) there is no associated cutaneous lichen planus or lichen planus at other mucosal sites. This may be called “isolated” OLP.^[3]

OLP has six classical clinical presentations: Reticular, Erosive, Atrophic, Plaquelike, Papular, Bullous.^[4] This disease has most often been reported in middle-aged patients and is more common in females than in males.^[5] A pathogenetic link may exist between dyslipidemia and OLP.^[6]

Inflammation causes disturbances in lipid metabolism such as decrease in High Density Lipoproteins – Cholesterol (HDL-C), increase in Very Low Density Lipoprotein-Cholesterol (VLDL-C) and hypertriglyceridemia. Prolonged dyslipidemia, due to chronic inflammatory condition enhances the formation of atherosclerotic plaques and thereby augments the risk of cardiovascular disease in such patients.^[7] The aim of this study was to analyse cardiovascular risk factors in patients with OLP.

MATERIALS AND METHODS

This case-control study was based on 120 patients, visiting Department of Oral Medicine and Radiology: 60 with oral lichen planus and 60 controls which matched the cases in all demographic

characteristics. Ethical clearance was obtained from the institutional ethical committee. An informed written consent was taken from all the participants. The diagnosis of OLP was made on clinical examination and confirmed histopathologically by biopsy. The diagnosis was in accordance to the modified WHO criteria.

Clinical data were recorded including age, weight, height, body mass index (BMI = kg /m²), habits, sedentarism, hypothyroidism, personal or familiar history of cardiovascular disease.

Inclusion criteria were as follows: men and women older than 18 years, presence of oral lichen planus, and confirmation of OLP with histopathological examination.

Exclusion criteria were as follows: patients with lichenoid drug eruption, patients receiving treatment for LP such as systemic corticosteroids, retinoic acid or methotrexate, patients suffering from diabetes mellitus, uremia, nephritic syndrome, hypothyroidism,

hyperthyroidism, acromegaly and individuals on lipid-lowering drugs.

2ml of the blood was drawn from the median cubital vein under all aseptic precautions and transported in a clot activator vial. Serum was analysed for: Serum Cholesterol, Triglycerides, HDL, LDL and VLDL.

Statistical Methods: Statistical analyses were performed with the SPSS version 20.0 software programme and Microsoft Excel. Student's independent t-test was employed for parametric data and for non-parametric data, Chi-square test was employed. P-value less than 0.05 were considered statistically significant.

RESULTS

A total of 120 patients, 60 with OLP and 60 control patients were analyzed. OLP group comprised of 21 (35%) males and 39 (65%) females with a mean age of 43.7±6.07. In control group 21 males and 39 females were selected with a mean age of 44.2±6.77 (Table 1).

Table 1: Age and gender distribution of studied patients in two groups

Age and gender		OLP Group		Control Group		P-value
		No.	%age	No.	%age	
Gender	Male	21	35	21	35	1.000#
	Female	39	65	39	65	
Age	Mean±SD	43.7±6.07		44.2±6.77		0.671#
	Range	32-55		33-54		

#Statistically insignificant Difference (P-value>0.05)

Table 2: Comparison based on lipid profile in patients and controls

Lipid Profile	OLP Group		Control Group		t-value	P-value
	Mean	SD	Mean	SD		
Serum Cholesterol	223.1	29.16	195.5	23.45	5.704	<0.001*
Serum triglycerides	175.5	37.76	154.3	22.34	3.730	0.0003*
HDL	42.9	5.75	46.3	6.93	2.938	0.004*
LDL	145.1	26.16	118.4	22.65	5.988	<0.001*
VLDL	35.1	7.55	30.8	4.49	3.752	0.00027*

*Statistically Significant Difference (P-value<0.05)

Table 3: TC/HDL ratio and LDL/HDL ratio in OLP patients and Controls

Lipid Ratios	OLP Group		Control Group		t-value	P-value
	Mean	SD	Mean	SD		
TC/HDL	5.28	0.875	4.32	0.825	6.172	<0.001*
LDL/HDL	3.44	0.748	2.64	0.733	5.922	<0.001*

*Statistically Significant Difference (P-value<0.05)

Table 2 shows comparison based on lipid profile in patients and controls. The serum Cholesterol was significantly higher in OLP group with a mean of 223.1mg/dl compared with 195.5mg/dl in control group.

P-value was <0.001 which was statistically significant.

Serum triglycerides in OLP group were higher with a mean of 175.5mg/dl compared with 154.3mg/dl in control group having a significant p-value of 0.0003.

Mean LDL in OLP Group was 145.1mg/dl compared with 118.4mg/dl in control group with a significant p-value of <0.001.

VLDL in OLP group was also high with a mean value of 35.1mg/dl compared with 30.8mg/dl in control and a significant p-value of 0.00027.

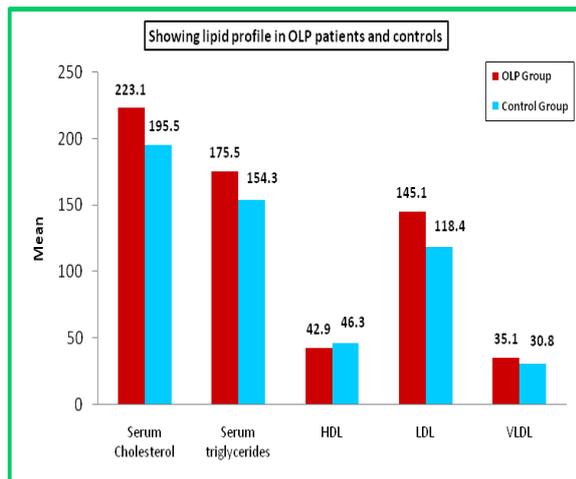


Figure 1: Comparison of lipid profile in OLP patients and control group.

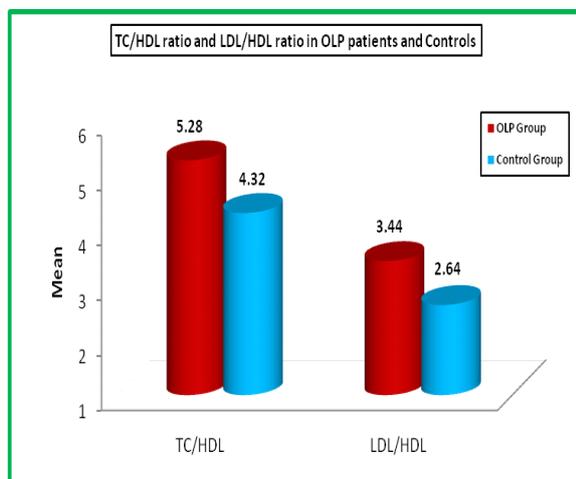


Figure 2: Comparison of TC/HDL ratio and LDL/HDL ratio in OLP patients & control group.

HDL in OLP group was low with a mean value of 42.9mg/dl compared with 30.8mg/dl in control group. p-value was 0.004 which was again statistically significant.

Table 3 shows total cholesterol /HDL-cholesterol ratio and LDL /HDL ratio both of which are used as predictor for cardiovascular risk. Higher values were seen in both ratios.

DISCUSSION

Lichen planus (LP) is a chronic inflammatory disease that affects the skin, genitalia, mucous membranes or appendages. [8]

Oral lichen planus affects women more than men (1.4:1). Oral lichen planus occurs predominantly in adults over 40, although younger adults and children may be affected. [9] In our study mean age was 43.76 and male female ratio of 1:1.85 this may be due to underreporting or small sample size of our study.

The reticular lesions, the most recognized form of OLP, encompass white lesions, which appear as a network of connecting and overlapping lines, papules or plaques. [10] Same pattern was seen in our study with reticular pattern being most common followed by erosive pattern.

The ultimate result in OLP is the destruction of the basal keratinocytes by CD8+ cytotoxic cells. This may occur either by direct activation of CD8+ cytotoxic by MHC class I molecules expressed by the epithelial cells or via the CD4+ helper cells. The Langerhans cells (LC) play a major role in the latter where they present the responsible antigen to the helper cells via the MHC class II molecules which in turn activate CD8+ cells via RCA (request cytotoxic activity) receptor (figure 1) and by the action of Interleukin-2 (IL2) and Interferon gamma (Ifn γ) [11]

Reports suggest that ROS and lipid peroxides affect the pathogenesis of LP. [12] Simark-Mattson et al found elevated concentrations of interleukin 2, IL-6, and IL-10, as well as Tumor necrosis factor- α and transforming growth factor- β within the subepithelial infiltrate in OLP patients. Dyslipidemia seen in these subjects may be attributed to these proinflammatory cytokines. [13]

In this study we found higher lipid levels in men and women with lichen planus than controls.

The results are in accordance with studies of Dreier J et al., [14] Kurgansky D

et al.,^[15] S. Arias-Santiago et al.,^[16,17] B. Krishnamoorthy et al.^[18]

Increased LDL-cholesterol /HDL-cholesterol ratio has already been considered as a sensitive predictor of cardiovascular risk and recently total cholesterol /HDL-cholesterol ratio has been found even a better predictor metabolic index for cardiovascular risk in large study. In the present study, patients with LP presented higher values of both ratios.^[19]

Clinical study of plasma lipids in patients with LP should be performed not only for the sake of diagnosis and treatment, but also for prevention given that atherosclerotic lesions start to appear at an early age and accelerate with the presence of other risk factors. In order to establish priorities with regard to intervention in patients with dyslipidemia it is necessary to stratify CV risk. Concomitant dyslipidemia and other risk factors such as arterial hypertension, diabetes, smoking, or renal disease are frequent and markedly increase CV events. Initiatives to establish evidence supporting the dyslipidemia hypothesis in LP patients could result in the possibility of assessing CV risk.^[20,21]

We have excluded patients taking drugs used for the treatment of LP such as retinoids, methotrexate or systemic corticosteroids because such drugs may cause dyslipidaemia. Patients suffering from diabetes mellitus, uremia, nephritic syndrome, hypothyroidism, hyperthyroidism, acromegaly and individuals on lipid-lowering drugs were also excluded to prevent any bias in our study.

CONCLUSION

In conclusion, the results obtained indicate an association between OLP patients and dyslipidemia. If this observation is confirmed in additional studies, the patients with LP should be evaluated for cardiovascular risk posed by dyslipidemia.

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