Original Article

Alterations of Serum Lipid Profile Patterns in Oral Lichen Planus Patients: A Case–Control Study

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Abstract

Background: Oral lichen planus (OLP) is a chronic disease of established immune-mediated pathogenesis. It most commonly, protractedly, and persistently involves the mucosa of the oral cavity. Antigen-specific and nonspecific mechanisms play a role in its pathogenesis, leading to T-cell accumulation in superficial lamina propria, intraepithelial T-cell migration, and keratinocyte apoptosis in OLP. Previous studies have indicated the possibility of serum lipid derangement in chronic inflammatory diseases such as systemic lupus erythematosus and psoriasis, which in turn results in elevated cardiovascular disease risk. Inflammation causes disturbances in lipid metabolism such as decrease in high-density lipoprotein-cholesterol (HDL-C) and increase in very low-density lipoprotein (VLDL)-cholesterol and hypertriglyceridemia due to direct effect on T-cell responses. Prolonged dyslipidemia, due to chronic inflammatory condition, enhances the formation of atherosclerotic plaques and thereby augments the risk of cardiovascular disease in such patients. With this background, a possible correlation between OLP and serum lipid level derangement can be anticipated. Hence, this study was taken up to probe into an association between the two. **Aims:** To determine and compare the serum lipid levels in OLP patients and healthy controls, to inquire into the possible association of OLP with alterations in serum lipid profile patterns, and to determine if the clinical characteristics of OLP differed with alterations in serum lipid profile patterns. **Subjects and Methods:** Sixty patients comprising 30 cases and 30 controls were enrolled for the study. Thirty cases of clinically and pathologically diagnosed OLP and 30 age- and sex-matched controls were subjected to blood examination for the assessment of serum lipid level, i.e., HDL, LDL, VLDL, and triglyceride. The obtained data were compared with standard values to assess any alterations of the serum lipid levels. **Statistical Analysis Used:** Cramer's V-test was performed for all the tests to measure association between two nominal variables. A $P \leq 0.05$ was considered statistically significant. **Results:** Dyslipidemia was observed in 13 (46.67%) cases as against 7 (23.33%) controls. Thus, a significant number of cases were found to have an associated serum dyslipidemia. However, pertaining to individual serum lipid levels in cases and controls, the association was found to be statistically insignificant. **Conclusions:** The current study suggested an evident association between dyslipidemia and OLP. We recommend imminent studies on a larger population to additionally substantiate a positive association between the two.

Keywords: *Cardiovascular disease risk, dyslipidemia, high-density lipoproteins, lichen planus, low-density lipoprotein, oral lichen planus, serum lipid derangement, triglyceride, very low-density lipoprotein*

Introduction

Lichen planus (LP) is a chronic, commonplace immunological disease that affects the skin, mucous membranes, nails, and hair. Oral LP (OLP) is the oral manifestation of the same which protractedly and persistently involves the mucosa of the oral cavity. It commonly affects middle-aged females, and the frequency of malignant transformation ranges from 0% to 5.3%. The buccal mucosa followed by labial mucosa and sulci forms the most frequent intraoral sites of involvement.[1]

The cause of the OLP is not well understood and is fraught with a multitude of hypothesis. Cell-mediated immunity is credited with a major aspect in the pathogenesis of OLP, and it may be initiated in individuals with a genetic predilection by various endogenous and exogenous factors. The various mechanisms hypothesized to be involved in the immune pathogenesis are antigen-specific, nonspecific mechanisms, autoimmune, and humoral immune responses. These mechanisms may combine

How to cite this article: Aniyan KY, Guledgud MV, Patil K. Alterations of serum lipid profile patterns in oral lichen planus patients: A case–control study. Contemp Clin Dent 2018;9:S112-21.

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Access this article online

Website: www.contempclindent.org

DOI: 10.4103/ccd.ccd_111_18

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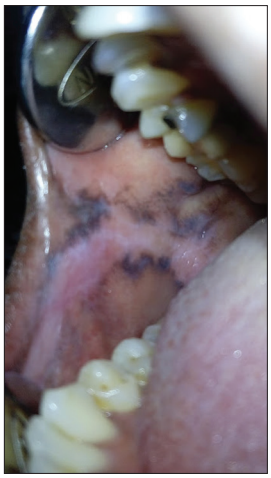

Figure 1: Reticular type of oral lichen planus characterized by white lacy striae

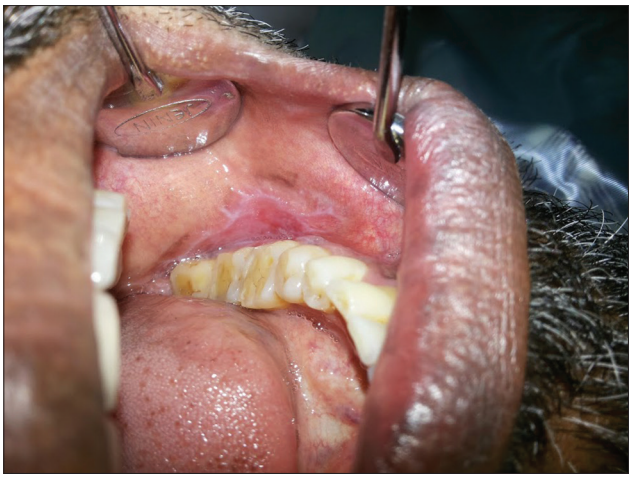

Figure 2: Atrophic type of oral lichen planus characterized by erythematous areas with radiating white striae along the margins

to cause the accumulation of T-cells in superficial lamina propria, causing disruption of basement membrane, intraepithelial T-cell migration, and keratinocyte apoptosis in OLP.[2]

Lipids are a group of fats and fat-like substances. They are essential biomolecules for maintenance of various biological functions, including stabilization of deoxyribonucleic acid helix, cell growth, and division in normal as well as in malignant tissues. The serum lipids are measured through their carrier lipoproteins, namely high-density lipoprotein (HDL), very low-density lipoprotein (VLDL), low-density lipoprotein (LDL), and triglyceride. The concentration of blood lipids depends on intake and excretion from the intestine and uptake and secretion from cells. The usefulness of variations in blood cholesterol levels in diagnosis and treatment of various diseases has been studied. An increase in the level of cholesterol is a major risk factor for coronary heart diseases; on the other hand, a decrease in the level of cholesterol has been associated with an increased risk of cancer.[3,4]

Absorbed fatty products in lieu of triglyceride enter the circulation as chylomicrons which are then broken down by lipoprotein lipase into HDL, LDL, and VLDL, respectively. The lipid–protein complex called lipoproteins enables the transport through bloodstream and is a reliable indicator for the respective lipids in the blood. Chronic inflammation induced in immune-mediated diseases instigates discrepancies in lipid metabolism as it endeavors to dilute the destruction and repair tissue by redistributing nutrients to cells involved in the host defense. The inflammatory cascade activation induces a decrease in HDL-cholesterol (HDL-C), with impairment in reverse cholesterol transport, and parallel changes in apolipoproteins, enzymes, antioxidant capacity, and ATP-binding cassette A1-dependent efflux. This decrease in HDL-C and phospholipids could stimulate compensatory changes such as synthesis and accumulation of phospholipid-rich VLDL, which binds bacterial products and other toxic substances, resulting in hypertriglyceridemia. The final consequence is an increased accumulation of cholesterol in the cells. Thus, the classical lipid changes associated with the metabolic syndrome (increased triglycerides and decreased HDL-C) may be envisioned as a highly conserved evolutionary response aimed at tissue repair, resulting in cardiovascular disease risk.[5]

In this vein, the study was taken up to probe into the possible correlation between OLP and serum lipid-level derangements and to detect the difference in clinical characteristics of OLP in association with altered serum lipid profile patterns.

Subjects and Methods

The study was carried out to determine the association of altered serum lipid profile patterns in OLP patients. Ethical clearance was obtained from the Institutional Ethical Committee before conducting the study.

The study group comprised 60 patients, irrespective of age, presenting to the institution as outpatients who were examined and selected by three investigators. Using purposive sampling method, the study was conducted from January 2016 to July 2017. Based on the prevalence of OLP (0.1%–2.2%), keeping confidence limit (Z) at 95% and allowable error (d) at 5%, the sample size for the study was fixed at 30 cases and 30 controls.

They were classified into two groups:

1. Cases - 30 individuals with clinically and histopathologically diagnosed OLP
2. Controls - 30 age- and sex-matched individuals with apparently healthy oral mucosa.

Figures 1 and 2 were the clinical photographs of the cases selected. The selected participants were explained in detail about the procedures involved and written informed consent was obtained from them. A detailed history was recorded

and a thorough general physical examination was performed wherein the relevant history (age, gender) and clinical and histopathological details were noted in an especially prepared pro forma. This was followed by a detailed examination of the OLP lesions. Clinically diagnosed OLP lesions were then subjected to histopathological evaluation for confirmation following which symptomatic cases were managed by conventional therapy. The criteria for the case and control group selection were as follows.

Inclusion criteria

- Patients with clinically and histopathologically diagnosed OLP – modified WHO criteria 2003 (cases)
- Patients with apparently healthy oral mucosa on complete oral examination (controls)
- Patients not known to be suffering from any other endocrine or metabolic disorders
- Patients not on any medications that are known to alter serum lipid levels in the body.

Exclusion criteria

- Pregnant patients
- Patients known to be suffering from any medical condition that precludes them from undergoing an oral biopsy procedure
- Patients on dyslipidemia therapy and on therapy for OLP
- Patients with any other coexisting oral lesions.

Venous blood samples were collected from the patients in the case and control group for the assessment of individual serum lipid levels. It was assessed using Chemistry Analyzer-Beckman Coulter AU480 (Beckman Coulter Diagnostics Model: February 2016). The values were subsequently recorded in the respective pro forma. The obtained data were compared to standard values to assess for alteration in serum lipid levels. The standard values to assess serum lipid levels (American Endocrine Society clinical practice guidelines, 2011) are as follows:[6]

- Triglyceride <150 mg/dl
- LDL-cholesterol (LDL-C) <100 mg/dl
- HDL-C 40–60 mg/dl
- VLDL-cholesterol 2–30 mg/dl.

The data were tabulated and subjected to statistical analysis. Descriptive statistical procedures such as means, standard deviations, medians, minimum, maximum, and percentages were used to summarize all variables. Cramer's V-test was procured to measure the association between two nominal variables. $P \leq 0.05$ was considered statistically significant. Microsoft Excel was used for data registration, and IBM SPSS Statistics (version 20.0, SPSS Inc., IBM) was used for statistical analyses.

Results

In the study group of 60 participants, the average individual serum lipid levels, HDL, and triglyceride were confined to normal limits. However, LDL was of a normal average among cases and elevated in average among controls. Of interest was the elevated average VLDL among cases and the normal average in controls [Table 1].

Of the 30 cases, 46.67% of patients had dyslipidemia, and among the 30 controls, 23.33% of patients had dyslipidemia. A significant number of cases were found to have associated serum dyslipidemia in comparison to the controls. However, pertaining to individual serum lipid levels in cases and controls, the association was found to be statistically insignificant [Table 2].

In the age range of 18–30 years among cases, dyslipidemia was noted only in HDL levels where 33.3% had decreased HDL levels and 33.33% had elevated HDL levels. In the age range of 31–45 years among cases, 18.33% had elevated LDL levels, 8.33% had elevated VLDL levels, and 16.6% had elevated triglyceride levels. In the age range of 46–60 years among cases, 7.69% demonstrated elevated HDL levels, 15.38% had elevated LDL levels, 30.76% had elevated VLDL levels, and 23.07% had elevated triglyceride levels. In the age range of >60 years among cases, 50.0% had elevated VLDL levels and 50.0% patients had elevated triglyceride levels. However, the age-wise distribution pertaining to dyslipidemia was statistically insignificant [Table 3].

Among the 30 cases, 43.33% of patients demonstrated dyslipidemia, of which 23.33% of patients were female and 20% of patients were male. It was determined that association between serum lipid levels and gender-wise distribution was insignificant [Table 4].

In the case group of 30 patients, 36.67% of patients were asymptomatic and 63.33% of patients were symptomatic.

Table 1: Distribution of average individual serum lipid levels among cases and controls

Serum lipid	Range in study (mg/ml)	Case average (mg/ml)	Control average (mg/ml)
HDL	20-77.4	43.92	45.48
VLDL	12.66-105.04	38.55	27.26
LDL	36-150	99	101.18
Triglyceride	20-525.5	152.39	129.40

HDL: High-density lipoprotein; LDL: Low-density lipoprotein; VLDL: Very LDL

Table 2: Distribution of individual deranged serum lipid levels (dyslipidemia) among cases and controls

Serum lipid levels	Cases (30)		Controls (30)	
	Normal	Deranged	Normal	Deranged
HDL	27	3	24	6
VLDL	22	8	27	3
LDL	28	2	30	0
Triglyceride	23	7	26	4

HDL: High-density lipoprotein; LDL: Low-density lipoprotein; VLDL: Very LDL

Table 3: Age wise distribution of cases based on serum lipid levels

Serum lipid	Value	Ages				Total
		19-30	31-45	46-60	60+	
HDL levels						
High	Count	1	0	1	1	3
	Percentage within lipid levels	33.33	0.0	7.69	50.0	10.0
Normal	Count	1	12	12	1	26
	Percentage within lipid levels	33.33	100	92.30	50.0	90.0
Low	Count	1	0	0	0	1
	Percentage within lipid levels	33.33	0.0	0.0	0.0	100.0
Total	Count	3	12	13	2	30
	Percentage within lipid levels	100.0	100.0	100.0	100.0	100.0
LDL levels						
High	Count	0	1	2	0	3
	Percentage within lipid levels	0.0	8.33	15.38	0.0	10.0
Normal	Count	3	11	11	2	27
	Percentage within lipid levels	100	91.6	84.6	100	90.0
Total	Count	3	12	13	2	30
	Percentage within lipid levels	100	100	100	100	100.0
VLDL levels						
High	Count	0	0	0	0	0
	Percentage within lipid levels	0.0	0.0	0.0	0.0	100.0
Normal	Count	3	12	9	6	30
	Percentage within lipid levels	100.0	100.0	100.0	100.0	100.0
Total	Count	3	12	9	6	30
	Percentage within lipid levels	100	100	100	100	100.0
Triglyceride levels						
High	Count	0	2	1	1	5
	Percentage within lipid levels	0.0	16.66	11.11	16.66	16.66
Normal	Count	3	10	8	5	25
	Percentage within lipid levels	100	83.33	88.88	83.33	83.33
Total	Count	3	12	9	6	30
	Percentage within lipid levels	100	100	100	100	100.0

HDL: High-density lipoprotein; LDL: Low-density lipoprotein; VLDL: Very LDL

However, the association between dyslipidemia and symptoms in OLP was found to be statistically insignificant [Table 5].

Pertaining to OLP, dyslipidemia, and its association to the site of presentation, 17 patients had presentation on bilateral buccal mucosa, seven patients on bilateral buccal mucosa and gingiva, and six patients on the bilateral buccal mucosa and tongue. However, the association of OLP patients with dyslipidemia according to the site of presentation was deemed statistically insignificant [Table 6].

On consideration OLP subtypes and dyslipidemia association, it was noted that 22 cases had reticular-type OLP, eight cases had atrophic-type OLP, and 14 patients demonstrated dyslipidemia. Inasmuch, the association was statistically insignificant [Table 7].

Discussion

OLP is a T-cell-mediated, chronic inflammatory oral mucosal disease of unknown etiology. Several factors have been proposed contributing to etiology including genetic background, dental materials, drugs, infectious agents, autoimmunity, immunodeficiency, food allergies, stress, habits, trauma, diabetes, hypertension, malignant neoplasm, and bowel disease.[7] It is interesting to note that quite a few skin diseases such as androgenic alopecia and psoriasis have been inseverably linked to cardiovascular risk factors.[5] The current interest is the associated serum lipid derangement of note in OLP patients, which in turn has been linked to increased cardiovascular risk.

Furthermore, among other studies, the term "dyslipidemia" when scrutinized was categorized as a broader terminology, i.e., elevation in total cholesterol and triglyceride. The terminology in actuality encompasses all the lipid levels, i.e. HDL, LDL, VLDL, and triglycerides. Thus, ideally, any elevation or depression of the aforementioned levels would umbrella under the terminology "dyslipidemia." To be explicit and eradicate the probable disadvantage imposed by these factors, individual serum lipid levels were recorded and compared among the two groups in our study.

Table 4: Gender-wise distribution of serum lipid profile in cases

Sex	Serum lipid	Value	Group		Total
			Cases	Controls	
Male	HDL-C				
	Low	Count	0	1	1
		Percentage within group	0.0	7.1	3.8
	Normal	Count	12	10	22
		Percentage within group	100.0	71.4	84.6
	High	Count	0	3	3
		Percentage within group	0.0	21.4	11.5
	Total	Count	12	14	26
		Percentage within group	100.0	100.0	100.0
Female	HDL-C				
	Low	Count	2	5	7
		Percentage within group	11.1	31.2	20.6
	Normal	Count	15	11	26
		Percentage within group	83.3	68.8	76.5
	High	Count	1	0	1
		Percentage within group	5.6	0.0	2.9
	Total	Count	18	16	34
		Percentage within group	100.0	100.0	100.0
Total	HDL-C				
	Low	Count	2	6	8
		Percentage within group	6.7	20.0	13.3
	Normal	Count	27	21	48
		Percentage within group	90.0	70.0	80.0
	High	Count	1	3	4
		Percentage within group	3.3	10.0	6.7
	Total	Count	30	30	60
		Percentage within group	100.0	100.0	100.0
Male	LDL-C				
	<150	Count	11	14	25
		Percentage within group	91.7	100.0	96.2
	>150	Count	1	0	1
		Percentage within group	8.3	0.0	3.8
	Total	Count	12	14	26
		Percentage within group	100.0	100.0	100.0
Female	LDL-C				
	<150	Count	17	16	33
		Percentage within group	94.4	100.0	97.1
	>150	Count	1	0	1
		Percentage within group	5.6	0.0	2.9
	Total	Count	18	16	34
		Percentage within group	100.0	100.0	100.0
Total	LDL-C				
	<150	Count	28	30	58
		Percentage within group	93.3	100.0	96.7
	>150	Count	2	0	2
		Percentage within group	6.7	0.0	3.3
	Total	Count	30	30	60
		Percentage within group	100.0	100.0	100.0

Contd...

Table 4: Contd...

Sex	Serum lipid	Value	Group		Total
			Cases	Controls	
Male	VLDL-C				
	2-38	Count	7	12	19
		Percentage within group	58.3	85.7	73.1
	>38	Count	5	2	7
		Percentage within group	41.7	14.3	26.9
	Total	Count	12	14	26
		Percentage within group	100.0	100.0	100.0
Female	VLDL-C				
	2-38	Count	15	15	30
		Percentage within group	83.3	93.8	88.2
	>38	Count	3	1	4
		Percentage within group	16.7	6.2	11.8
	Total	Count	18	16	34
		Percentage within group	100.0	100.0	100.0
Total	VLDL-C				
	2-38	Count	22	27	49
		Percentage within group	73.3	90.0	81.7
	>38	Count	8	3	11
		Percentage within group	26.7	10.0	18.3
	Total	Count	30	30	60
		Percentage within group	100.0	100.0	100.0
Male	Triglyceride-cholesterol				
	10-190	Count	7	12	19
		Percentage within group	58.3	85.7	73.1
	>190	Count	5	2	7
		Percentage within group	41.7	14.3	26.9
	Total	Count	12	14	26
		Percentage within group	100.0	100.0	100.0
Female	Triglyceride-cholesterol				
	10-190	Count	16	14	30
		Percentage within group	88.9	87.5	88.2
	>190	Count	2	2	4
		Percentage within group	11.1	12.5	11.8
	Total	Count	18	16	34
		Percentage within group	100.0	100.0	100.0
Total	Triglyceride-cholesterol				
	10-190	Count	23	26	49
		Percentage within group	76.7	86.7	81.7
	>190	Count	7	4	11
		Percentage within group	23.3	13.3	18.3
	Total	Count	30	30	60
		Percentage within group	100.0	100.0	100.0

HDL-C: High-density lipoprotein-cholesterol; LDL-C: Low-density lipoprotein-cholesterol; VLDL-C: Very low-density lipoprotein-cholesterol

A case–control study by Dreiher *et al*. discovered that LP was irrefutably associated with dyslipidemia in the patient series. The authors implied that LP was previously reported to be associated with abnormal carbohydrate metabolism in epidermal cells and that the chronic inflammation associated with the condition conducive to dyslipidemia.[8]

Thereafter, in another study by Arias-Santiago *et al*., comprising 200 patients, a higher significant prevalence of dyslipidemia was revealed in patients with LP (cutaneous and oral). Authors suggested the possible association of dyslipidemia with the pathogenesis of OLP, concluding that the chronic inflammation induced as the reason, thus increasing the cardiovascular risk in such subjects.[9]

None of the studies elucidated the association of individual serum lipid level alteration with OLP lesions in a specific age and gender, its type, site, and associated symptoms. We hypothesized that all these could be a variable in association of serum lipid level alteration in OLP. In our study, of the

Table 5: Symptom-wise and serum lipid profile distribution of cases

Serum lipid	Value	Symptom		Total
		Present	Absent	
HDL-C				
Low	Count	2	0	2
	Percentage within symptom	10.5	0.0	6.7
Normal	Count	16	11	27
	Percentage within symptom	84.2	100.0	90.0
High	Count	1	0	1
	Percentage within symptom	5.3	0.0	3.3
Total	Count	19	11	30
	Percentage within symptom	100.0	100.0	100.0
LDL-C				
<150	Count	17	11	28
	Percentage within symptom	89.5	100.0	93.3
>150	Count	2	0	2
	Percentage within symptom	10.5	0.0	6.7
Total	count	19	11	30
	Percentage within symptom	100.0	100.0	100.0
VLDL-C				
2-38	Count	13	9	22
	Percentage within symptom	68.4	81.8	73.3
>38	Count	6	2	8
	Percentage within symptom	31.6	18.2	26.7
Total	Count	19	11	30
	Percentage within symptom	100.0	100.0	
Triglyceride-cholesterol				
10-190	Count	14	9	23
	Percentage within symptom	73.7	81.8	76.7
>190	Count	5	2	7
	Percentage within symptom	26.3	18.2	23.3
Total	Count	19	11	30
	Percentage within symptom	100.0	100.0	100.0

HDL-C: High-density lipoprotein-cholesterol; LDL-C: Low-density lipoprotein-cholesterol; VLDL-C: Very low-density lipoprotein-cholesterol

30 cases, 13 (46.67%) patients had deranged serum lipid levels, of which two subjects had depressed HDL, two subjects had elevated LDL level, eight subjects had elevated VLDL level, and seven subjects had elevated triglyceride levels. The results of the present study are consistent with those of a study by Dreiher *et al*.[8] and Arias *et al*.[9] It was to be emphasized that the derangement was not confined to total cholesterol and triglyceride alone but included HDL, LDL, and VLDL as well. The gravity of this finding can be substantiated by the findings of Krishnamoorthy *et al*., wherein it is elaborated that chronic inflammation induced in immune-mediated diseases instigates disturbances in lipid metabolism as it aims at decreasing the toxicity of the harmful agents and tissue repair by redistributing nutrients to cells involved in the host defense. The activation of the inflammatory cascade will induce a decrease in HDL-C that could stimulate compensatory changes such as synthesis and accumulation of phospholipid-rich VLDL, which binds bacterial products and other toxic substances, resulting in hypertriglyceridemia. The final consequence is an increased accumulation of cholesterol in cells.[10] When the compensatory response (inflammation) is not able to repair injury, it turns into a harmful reaction, and the lipid changes will become chronic, either by repeated or overwhelming stimulus, enhancing the formation of atherosclerotic lesions. Thus, the classical lipid changes associated may be envisioned as a highly conserved evolutionary response aimed at tissue repair.[5]

The subjects were grouped into six categories based on the age ranges, viz., 18–30 years, 31–40 years, 41–50 years, 51–60 years, and >60 years. Although it is an established fact that dyslipidemia has an inclination toward the aging population, in the present study, it was centered on the diseased patients. Among the cases that demonstrated dyslipidemia, 37% of patients were in the middle age, i.e. 31–40 years and 41–50 years of age group. This was in standing with the fact that OLP is a disease of middle age. The association of dyslipidemia and age-wise distribution of patients with OLP, however, was found to be statistically insignificant.

In addition, the association between dyslipidemia and different site involvements in patients with OLP

Table 6: Distribution of cases pertaining to site and serum lipid levels

Serum lipid	Value	Area			Total
		BM	BM and G	BM and T	
HDL-C					
Low	Count	1	0	1	2
	Percentage within area	5.9	0.0	16.7	6.7
Normal	Count	15	7	5	27
	Percentage within area	88.2	100.0	83.3	90.0
High	Count	1	0	0	1
	Percentage within area	5.9	0.0	0.0	3.3
Total	Count	17	7	6	30
	Percentage within area	100.0	100.0	100.0	100.0
LDL-C					
<150	Count	17	6	5	28
	Percentage within area	100.0	85.7	83.3	93.3
>150	Count	0	1	1	2
	Percentage within area	0.0	14.3	16.7	6.7
Total	Count	17	7	6	30
	Percentage within area	100.0	100.0	100.0	100.0
VLDL-C					
2-38	Count	12	5	5	22
	Percentage within area	70.6	71.4	83.3	73.3
>38	Count	5	2	1	8
	Percentage within area	29.4	28.6	16.7	26.7
Total	Count	17	7	6	30
	Percentage within area	100.0	100.0	100.0	100.0
Triglyceride-cholesterol					
10-190	Count	12	5	6	23
	Percentage within area	70.6	71.4	100.0	76.7
>190	Count	5	2	0	7
	Percentage within area	29.4	28.6	0.0	23.3
Total	Count	17	7	6	30
	Percentage within area	100.0	100.0	100.0	100.0

HDL-C: High-density lipoprotein-cholesterol; LDL-C: Low-density lipoprotein-cholesterol; VLDL-C: Very low-density lipoprotein-cholesterol; BM: Buccal mucosa; G: Gingiva; T: Tongue

was assessed. Of the 17 patients among case group demonstrating dyslipidemia, 12 patients had only on bilateral buccal mucosa, five patients had on bilateral buccal mucosa and gingival, whereas three patients had on bilateral buccal mucosa and tongue. A significant preponderance to a site in association with dyslipidemia could neither be drawn out nor could an affiliation be drawn between the severity of OLP and the sites of appearance. The results of the present study demonstrated that patients with OLP unequivocally had a significant lipid alteration. The lipid profile, including HDL, VLDL, LDL, and triglyceride levels, was deranged with no significant differences between the various subtypes of OLP. The subtypes or the severity of OLP was not a confounding factor as clarified subsequently and found to be statistically insignificant.

In an interesting study by Shaynam *et al*., high-stress levels in individuals correlated with high TC, high LDL-C, and low HDL-C compared to individuals with normal lipid profile. It was postulated that stress increased blood lipids through increasing hepatic lipoprotein lipase activity caused by a heightened sympathetic neuronal response. The association of dyslipidemia and stress level of the patient could positively correlate with the occurrence of OLP. However, this aspect was above and beyond the scope of this study and perhaps a limitation as well. Further, during the course of our study, it was of notice that social standing and lifestyle would factor in the disease process. Of consideration was that a majority of diseased patients were in low or lower middle economic classification. This in turn affects the stress levels pertaining to finance and even more importantly determines the nutrition status by cause and effect. We recommend further studies with larger samples to probe this possible association.

Conclusions

The denouement was that an association between OLP and dyslipidemia is not one that has been thoroughly explored, and considering the results of the current study, we attain a conclusion that a link is certain. Hence, prowling

Table 7: Oral lichen planus subtypes distribution in dyslipidemia

Serum lipid	Value	OLP_type				Total
		Reticular	Atrophic	Papular	Erosive	
HDL-C						
Low	Count	2	0	0	0	2
	Percentage within OLP_type	9.1	0.0	0.0	0.0	6.7
Normal	Count	19	8	0	0	27
	Percentage within OLP_type	86.4	100.0	0	0	90.0
High	Count	1	0	0	0	1
	Percentage within OLP_type	4.5	0.0	0	0	3.3
Total	Count	22	8	0	0	30
	Percentage within OLP_type	100.0	100.0	0.0	0.0	100.0
LDL-C						
<150	Count	21	7	0	0	28
	Percentage within OLP_type	95.5	87.5	0.0	0.0	93.3
>150	Count	1	1	0	0	2
	Percentage within OLP_type	4.5	12.5	0.0	0.0	6.7
Total	Count	22	8	0	0	30
	Percentage within OLP_type	100.0	100.0	0.0	0.0	100.0
VLDL-C						
2-38	Count	16	6	0	0	22
	Percentage within OLP_type	72.7	75.0	0.0	0.0	73.3
>38	Count	6	2	0	0	8
	Percentage within OLP_type	27.3	25.0	0.0	0.0	26.7
Total	Count	22	8	0	0	30
	Percentage within OLP_type	100.0	100.0	0.0	0.0	100.0
Triglyceride-cholesterol						
10-190	Count	17	6	0	0	23
	Percentage within OLP_type	77.3	75.0	0.0	0.0	76.7
>190	Count	5	2	0	0	7
	Percentage within OLP_type	22.7	25.0	0.0	0.0	23.3
Total	Count	22	8	0	0	30
	Percentage within OLP_type	100.0	100.0	0.0	0.0	100.0

OLP: Oral lichen planus; HDL-C: High-density lipoprotein-cholesterol; LDL-C: Low-density lipoprotein-cholesterol; VLDL-C: Very low-density lipoprotein-cholesterol

dyslipidemia must be investigated for, when evaluating a patient with OLP for it signals a much dangerous cardiovascular disease. Conversely, a medical professional evaluating dyslipidemia must be vigilant of an enshrouded OLP or perhaps even susceptibility to developing it. Although, in association with OLP, the serum lipids are likely to be elevated, depression of serum lipid levels could be an ill-omened directive of malignant change that needs to be explored further. Further, the lesions of OLP in dyslipidemia subjects were surveyed, and we explicated no typical characteristics of OLP in dyslipidemia.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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