Relationship between lichen planus and dyslipidemia: A case-control study from Southwest Iran

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Background: Lichen planus (LP) is a chronic inflammatory skin condition which leads to changes in lipid metabolism. It may cause chronic atherosclerosis and metabolic syndrome. The objective of this study is to compare lipid levels of patients with LP to healthy controls.

Methods: This case-control study recruited 100 total participants, 50 (25 male and 25 female) patients with LP and 50 healthy controls admitted to the Dermatology Clinic of Imam Hospital, Ahvaz, Iran. Serum triglyceride, total cholesterol, HDL cholesterol, and LDL cholesterol levels were measured in samples drawn after a 12-hour fasting period.

Results: Patients with LP had significantly higher triglycerides (147.7 vs. 118.4 mg/dl, $P<0.05$), total cholesterol (188 vs. 173 mg/dl, $P<0.05$), LDL (122 vs. 106 mg/dl, $P<0.05$), and lower HDL (49 vs. 57 mg/dl, $P<0.05$) levels compared to controls.

Conclusion: This study showed an association between LP and dyslipidemia. Screening of serum lipids in patients with LP might be valuable to prevent cardiovascular diseases.

Keywords: cardiovascular; cholesterol; dyslipidemia; lichen planus; triglyceride

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INTRODUCTION

Lichen planus (LP) is an idiopathic inflammatory dermatosis that affects the skin and mucous membranes [1-3]. Cutaneous LP occurs in less than 1% of the population and more common among women [4,5]. The classical manifestation of LP is flat violaceous polygonal papules or plaques over the wrists, thighs, abdomen, and extremities [6,7]. The clinical presentation of LP has several forms that include the classic plaque, oral, hypertrophic, follicular, linear, actinic, and bullous types [8].

It has been demonstrated that inflammation triggers changes known as acute–phase responses (APR). APR changes lipid metabolism, as seen with increased serum triglyceride levels and decreased HDL and LDL cholesterol levels. Despite these primary advantages, lipid disturbances linked to chronic inflammation increase the cardiovascular (CV) risk associated with hyperlipidemia [9].

LP produces disturbances of lipid metabolism such as increases in serum triglycerides or decreases in high-density lipoprotein cholesterol [10,11]. Inflammation produces disturbances of lipid metabolism such as serum increases of triglycerides or decreases of high-density lipoprotein cholesterol [1-3,6,10,11]. LP is a T-cell-mediated inflammatory disorder, wherein inflammation is assumed to be closely related to disturbances in lipid metabolism along with an increased risk of CV events [6,12-15].

Although there is a reported relationship between LP and dyslipidemia [5,10,16], few studies have investigated this relationship. Therefore, we designed this study to compare lipid levels of patients with LP to healthy controls.

PARTICIPANTS AND METHODS

This case–control study enrolled 100 subjects admitted to the Dermatology Clinic of Imam Hospital in Ahvaz, Southwest Iran, from 2014 to 2015. A total of 50 patients with LP and 50 healthy control subjects enrolled in the study. Patients with LP who were more than 18 years of age were included the study. Patients were excluded from the study if they had taken lipid lowering drugs and systemic steroids, or had lichenoid drug eruption. Diagnosis of LP was confirmed based on clinical findings or biopsy. We gathered data on age, weight, height, body mass index (BMI), smoking, alcohol consumption, hypothyroidism, and familial history of CV disease. Serum triglycerides, total cholesterol, HDL cholesterol, and LDL cholesterol were measured in samples drawn after a 12-hour fasting period.

Continuous quantitative variables were summarized using mean ± standard deviation (SD) and placed in tables. Nominal and categorical variables were presented in frequency tables. Statistical analyses were performed using PASW 18 (IBM Corp., Armonk, NY, USA). Independent *t*- and chi square tests were used. $P<0.05$ was considered statistically significant.

The Ahvaz University of Medical Sciences Ethical Committee reviewed and approved the study protocol. Participants enrolled in the study after they provided informed consent for study participation.

RESULTS

A total of 100 participants enrolled in the study, 50 patients comprised the case group (25 males and 25 females) and 50 patients were placed in the control group (25 males and 25 females). The mean age was 42±9.7 years for LP patients and 41.5±7.4 years for the controls (Table 1).

The mean serum levels for triglycerides, cholesterol, HDL, and LDL of patients with LP and the controls are shown in Tables 2 and 3, and Figure 1.

There was a significant difference in the level of triglyceride between the two groups ($P<0.05$). LP patients had higher triglyceride levels compared to healthy participants. Patients with LP had higher cholesterol levels compared to healthy individuals,

Table 1. Demographic information of the participants.

	Case (n=50)	Control (n=50)	*P*
Age (years)	42±9.7	41.5±7.4	0.77
Height (cm)	165±10.8	163±11.2	0.36
Weight (kg)	67±9.1	65±10.3	0.30
BMI (kg/m^2)	24.1±1.9	23.7±2.3	0.34

BMI: Body mass index

Table 2. Comparison of mean serum lipid levels in patients with lichen planus and controls.

	Patients (Mean±SD)	Controls (Mean±SD)
Triglycerides (mg/dl)	148.0±17.43	118.0±14.8
Cholesterol (mg/dl)	188.0± 26.5	173.0±21.6
HDL (mg/dl)	49.0±10.2	58.0±7.5
LDL (mg/dl)	122.0±12.4	106.0±11.6

*Mean serum lipid differences between patients and healthy controls showed statistical significance for all comparisons ($P<0.05$).

Table 3. Mean serum lipids levels in patients with lichen planus and healthy individuals according to gender.

Gender	Patients	Controls	*P*
Triglycerides (mg/dl)			
Male	150	117	<0.001
Female	145	119	<0.001
Cholesterol (mg/dl)			
Male	190	172	<0.001
Female	187	174	0.007
HDL (mg/dl)			
Male	48	57	<0.001
Female	51	58	<0.001
LDL (mg/dl)			
Male	121	105	<0.001
Female	124	107	<0.001

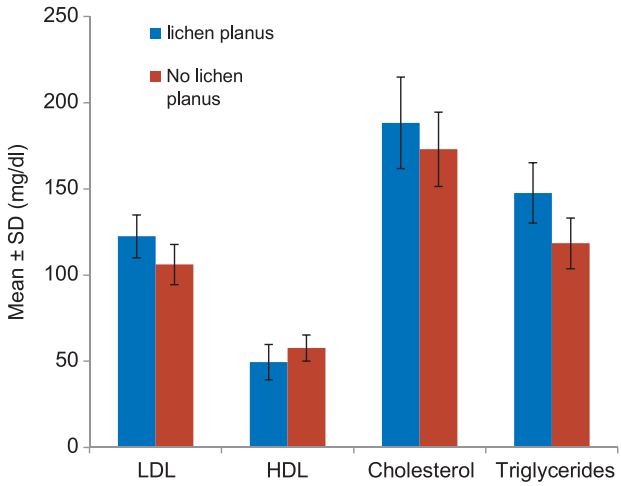

Figure 1. Comparison of lipid profiles in patients with lichen planus and healthy individuals.

which was statistically significant (P<0.05). HDL levels were less in LP patients compared to the control group (P<0.05). LDL levels in patients with LP were higher than healthy controls (P<0.05). Out of 50 patients with LP, 29 (58%) patients had dyslipidemia. In the control group of 50 subjects, 12 (24%) patients had dyslipidemia (P=0.001).

DISCUSSION

Increased serum triglyceride and decreased HDL levels occur in lipid metabolism during the APR of inflammation. Such alterations help to decrease the toxicity of the causative agent and aid in tissue repair. If the inflammation becomes chronic, then the changes in the lipid profile become sustained and thereby augment the accumulation of cholesterol in cells and formation of lipid foam cells which, in turn, produce fatty streaks in the arterial walls [6].

Arias-Santiago *et al.* conducted a case-control study to evaluate lipid levels in men and women with LP compared to healthy controls. Patients with LP showed higher triglycerides, total cholesterol, and LDL levels, along with lower HDL levels in comparison with the controls [10]. Our findings were consistent with Arias-Santiago *et al.* in which LDL value was 120 mg/dl in LP patients and 100 mg/dl in healthy individuals. Lipid screening of patients with LP might be useful to prevent CV disease. Dreiher *et al.* reported a higher frequency of dyslipidemia in the LP group compared to the control group [5].

The relationship between LP and dyslipidemia has been documented in previous studies [6]. We excluded patients with lichenoid drug eruptions and those who received systemic corticosteroid therapy to prevent bias. Chronic inflammation has been demonstrated to be essential in the progress of dyslipidemia. Accordingly, studies reported that patients with psoriasis, systemic lupus erythematous, and rheumatoid arthritis also had dyslipidemia [10,16,17].

In LP, lymphocytes infiltrate into the epidermis and attack keratinocytes. The keratinocytes produce more cytokines that attract more lymphocytes. These cytokines [tumor necrosis factor-α (TNF-α), interleukin- 6 (IL6), IL10, and IL4] are involved in the pathogenesis of LP and may explain the relationship with dyslipidemia [18]. Chronic inflammation is related to visceral obesity and increased adipocytokines such as TNF-α, IL-1, and IL-6 [18]. IL-6 plays an important role in various metabolic processes and actions of adipocytes. High levels of IL-6 have been observed in adipose tissues of obese patients [19].

The results of present study showed a significant relationship between LP and dyslipidemia. Further confirmation of these results with more extensive studies could be used as primary prevention of CV disease in patients with LP. LP might be a prognostic factor for CV disease and treatment of dyslipidemia would be critical.

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