



Evaluation of Lipid Profile and Serum Adiponectin Levels in Patients with Lichen Planus

*Ladan Dastgheib¹, Nasrin Hamidizadeh¹, Maryam Mardani², Sara Ranjbar¹, Peyman Jafari³, *Farnoosh Nozari¹*

1. *Molecular Dermatology Research Center, Shiraz University of Medical Sciences, Shiraz, Iran*
2. *Oral and Dental Disease Research Center, School of Dentistry, Shiraz University of Medical Sciences, Shiraz, Iran*
3. *Department of Biostatistics, Shiraz University of Medical Sciences, Shiraz, Iran*

***Corresponding Author:** Email: farnoosh.nozari.7391@gmail.com

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Dear Editor-in-Chief

Lichen planus (LP) is a chronic inflammatory disease that typically affects the skin and mucous membranes. Inflammation has been shown to affect lipid and carbohydrate metabolism, insulin signaling, and adipogenesis (1).

Adipose tissue is an active endocrine organ that secretes numerous factors such as adiponectin, which influence carbohydrate and lipid metabolism (2). Low plasma adiponectin levels have been reported in a variety of conditions, including obesity, type 2 diabetes, and LP (2,3). However, whether these differences in adiponectin levels in LP are due to the high prevalence of comorbidities such as diabetes and obesity or the sole effects of disease on adiponectin is not obvious.

In this study, we examined the lipid profile and serum adiponectin levels in patients with LP and healthy controls.

Thirty-nine patients diagnosed with LP and 19 controls were included. All participants signed an informed consent form before the study. This report was approved by the local ethics committee. Blood samples were taken after a 12 hour fast. An enzyme-linked immunosorbent assay (ELISA) method was applied to determine serum adiponectin levels.

Dyslipidemia was defined as total cholesterol (TC) > 200 mg / dl, low-density lipoprotein cholesterol (LDL) > 130 mg / dl, triglyceride (TG) > 150 mg / dl and high-density lipoprotein cholesterol (HDL) < 40 mg / dl in men or HDL < 50 mg / dl in women (4). Central obesity was defined as a waist circumference of at least 88 cm in women and 102 cm in men (5).

The independent *t*-test, chi-square test, correlation analysis, and regression were used to examine differences between the two groups. Statistical analysis was performed with SPSS version 19 (IBM Corp., Armonk, NY, USA).

Of 39 patients, 20 (51.3%) had oral involvement and 19 (48.7%) had cutaneous involvement. The patient and control groups showed no significant differences in terms of age and gender.

The patients had a significantly larger mean waist circumference than the controls ($P=0.013$). According to the frequency of participants with central obesity or dyslipidemia, no significant differences were found between patients and controls. Serum TG levels were significantly higher in patients than in controls ($P=0.030$). In addition, the patients had significantly lower serum adiponectin levels than the controls ($P=0.004$). There was a positive correlation between serum



adiponectin and HDL levels in patients (correlation (r) = 0.346; P = 0.031). A backward linear regression model showed that LP was associated with adiponectin levels even after adjusting for dyslipidemia, FBS, and obesity (B = 0.307; β = 0.373; t = 3.006; P = 0.004).

However, concerning the above parameters, no significant differences were observed between patients with oral and cutaneous involvement.

Various interleukins (ILs) are elevated in LP patients, which may play an essential role in the development of dyslipidemia (6). Panchal et al observed significantly higher TC, TG, and LDL levels and lower HDL levels in LP patients compared to controls (7). Conversely, Özkur et al found no significant differences in serum levels of TC, LDL, and TG between patients with LP and controls (8).

Our study showed significantly higher TG levels in patients with LP than in controls. However, no significant differences were observed between the two groups concerning other parameters of the lipid profile. The different ranges of LP duration in this study may have influenced lipid profiles of patients.

Our study also showed that patients with LP had significantly lower serum adiponectin levels than controls. Similarly, Ismail et al found significantly lower serum adiponectin levels in LP patients (3). The prevalence of comorbidities such as diabetes and dyslipidemia is higher in patients with LP (1). Patients with obesity, diabetes, and dyslipidemia often have low levels of adiponectin (9). However, we found that LP was associated with lower plasma adiponectin levels regardless of dyslipidemia, diabetes, and obesity. We suggest that lower adiponectin levels in LP patients are not entirely due to the high rate of comorbidities in LP, but rather to a unique effect of LP on adipose tissue function and adiponectin signaling. These results may be due to the complex interactions between elevated levels of various cytokines and adipose tissue in LP.

In conclusion, early lipid profile screening in LP patients may not be of great value and inflammation present in LP may be associated with adipose tissue dysfunction.

Conflict of interest

The authors declare that there is no conflict of interest.

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