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ORIGINAL ARTICLE

Uric acid in patients with lichen planus and healthy control

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ABSTRACT:
Background: A chronic inflammatory disease that affects the skin, mucous membranes, hair, and nails is called lichen planus. Small, irritating, flat-topped pimples or papules that may be purplish, reddish, or brownish in appearance are its defining feature. The present study was conducted to assess uric acid in patients with lichen planus. **Materials & Methods:** 38patients of lichen planus of both genderswere put in group I and 38 healthy controls in group II. Five milliliters of venous blood sample were collected in each group after 12 h of fasting. Serum UA was assayed using Coralab 3000 semiautoanalyser, by uricase method. **Results:** Group I had 12 males and 26 females and group II had 14 males and 24 females. The mean serum uric acid in group I was 3.52 mg/dl and in group II was 3.96 mg/dl. The difference was significant (P< 0.05). **Conclusion:** LP is associated with a decrease in serum UA levels. For monitoring and treatment planning, UA may prove to be a useful biomarker of LP's antioxidant status.
Keywords: Lichen planus,papules, Uric acid

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INTRODUCTION

A chronic inflammatory disease that affects the skin, mucous membranes, hair, and nails is called lichen planus. Small, irritating, flat-topped pimples or papules that may be purplish, reddish, or brownish in appearance are its defining feature. These papules can appear in groups and frequently feature delicate white lines or scales on their surface.[1] The wrists, ankles, lower back, genitalia, mouth, and nails are among the bodily parts that can be impacted by lichen planus. Although the precise cause of lichen planus is unknown, an aberrant immune response is thought to be involved. It is believed that lichen planus results from an autoimmune reaction, where the body's immune system unintentionally targets its own cells. But it's unclear exactly what set off this immunological reaction.[2]

Lichen planus can run in families, thus some people may be genetically predisposed to getting it. Lichen planus has occasionally been linked to the development of certain illnesses, such as hepatitis C virus (HCV) infection.[3] Some people may develop lichen planus as a result of taking certain medications, such as beta-blockers, nonsteroidal anti-inflammatory drugs (NSAIDs), and some antibiotics. Depending on the body part afflicted, lichen planus symptoms can differ.[4] Apart from the typical papules, other symptoms of lichen planus include burning, discomfort, itching, and soreness. Lichen planus can result in ulcers, redness, or white spots in the mouth, genitalia, or other places where the mucous membranes are affected.[5]One of the significant antioxidants in plasma is uric acid (UA). UA has the ability to bind metal ions and scavenge ROS. Therefore, keeping an eye on the serum UA level as a measure of antioxidant defense (oxidative balance) may be crucial for the treatment plan that the physicians employ.[6]The present study was conducted to assess uric acid in patients with lichen planus.

MATERIALS & METHODS

The present study consisted of 38 cases of lichen planus of both genders. All gave their written consent to participate in the study.

Data such as name, age, gender etc. was recorded. Group I consisted of patients, while group II consisted of healthy controls. Following a 12-hours fast, five milliliters of venous blood were drawn from each group. The Coralab 3000 semiautoanalyser was used to measure serum UA using the uricase technique. Data thus obtained were subjected to statistical analysis. P value < 0.05 was considered significant.

RESULTS

Table I Distribution of patients

Groups	Group I (38)	Group II(38)
Male	12	14
Female	26	24

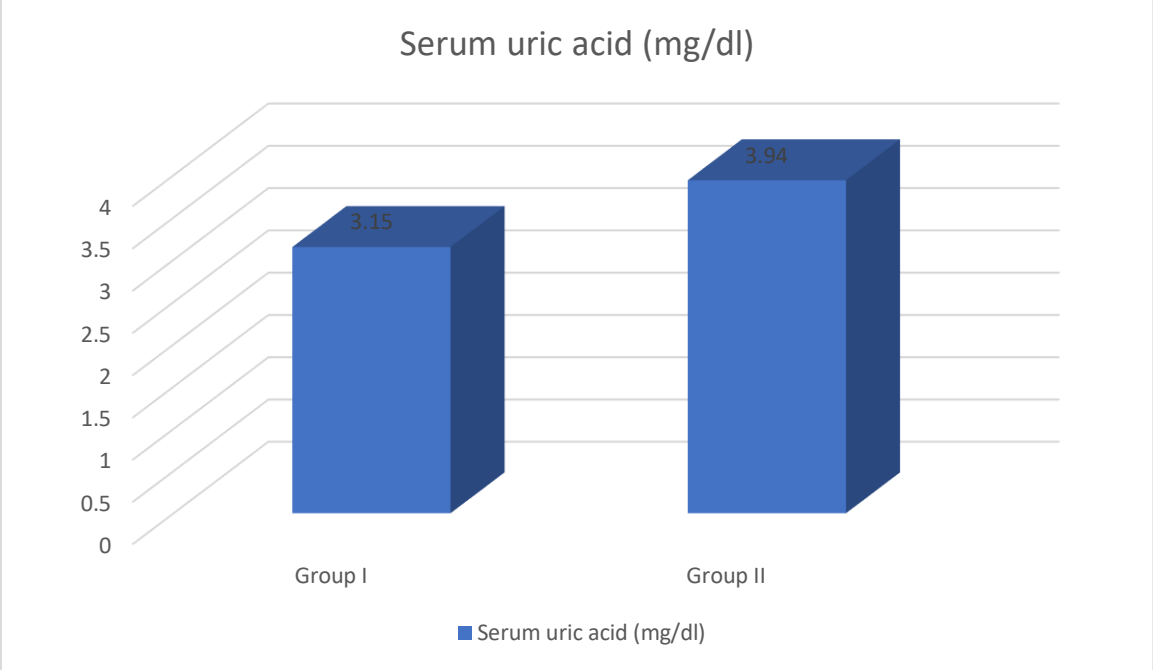
Table I shows that group I had 12 males and 26 females and group II had 14 males and 24 females.

Table II Comparison of serum uric acid in both groups

Groups	Serum uric acid (mg/dl)	P value
Group I	3.15	0.01
Group II	3.94	

Table II, graph I shows that mean serum uric acid in group I was 3.52 mg/dl and in group II was 3.96 mg/dl. The difference was significant (P< 0.05).

Graph I Comparison of serum uric acid in both groups



DISCUSSION

One of the main antioxidants found in human physiological fluids is UA, a strong scavenger of free radicals that may also work as a chelator to change metal ions like iron and copper into less reactive forms that can't catalyze free-radical reactions.[7,8] Although the primary defense mechanisms against oxidative stress include antioxidants such vitamins, certain enzymes, and others, it is thought that UA is responsible for about half of blood's antioxidant capacity.[9,10] UA prevents the production of peroxynitrite by neutralizing cellular superoxide and preventing its interaction with nitric oxide. By preventing the breakdown of superoxide dismutase, the enzyme that breaks down superoxide, it can help eliminate superoxide. It is a scavenger of free radicals, including NO2, which is created when peroxynitrite breaks down.[11,12]The present study was conducted to assess uric acid in patients with lichen planus.

We found that group I had 12 males and 26 females and group II had 14 males and 24 females. Sezer et al[13] assessed how well the antioxidant defense system and oxidative stress were functioning in LP patients. This prospective study included 40 LP patients (23 men and 17 women; mean +/- SD age 43.27 +/- 1.96 years) and 40 age and gender-matched control volunteers. Immunosuppressive medicine, a history of trauma or surgery, and a history of alcohol consumption for at least one month before the trial were among the exclusion criteria. Both groups' levels of erythrocyte catalase (CAT), serum nitric oxide (NO), malondialdehyde (MDA), and superoxide dismutase (SOD) were examined. Serum NO (74.60 +/- 17.96 micromol/L) and MDA (18.24 +/- 5.21 micromol/L) mean +/- SD levels were greater in LP patients than in the control group.Additionally, LP patients had greater serum SOD levels (18.19 +/- 3.71 U/mL) than healthy controls (P = 0.002). Erythrocyte CAT levels, on the other hand, were substantially lower in the patient group than in the control group (13 557.80 +/- 4134.42 U/kg hemoglobin; P = 0.009).

We found mean serum uric acid in group I was 3.52 mg/dl and in group II was 3.96 mg/dl. Chakraborti et al[14] evaluated serum uric acid (UA) levels as a measure of the antioxidant defense status in LP patients. Serum UA levels were determined in 58 LP patients and 61 controls. Serum UA levels were significantly decreased in patients with respect to controls. Moreover, serum UA level was decreased according to increasing duration of disease.

Ergun et al[15]assessed oxidative stress and antioxidant profile in patients with oral lichen planus (OLP) using serum and salivary samples and to compare these biomarkers in a group of healthy subjects.Twenty-one recently diagnosed patients with OLP and 20 healthy controls with matched periodontal status were recruited to the study. Total antioxidant activity (TAA) and lipid peroxidation product

malondialdehyde (MDA) in both serum and saliva were determined. In OLP patients, total antioxidant defense (TAA) was significantly lower than that in healthy subjects in their serum samples (P = 0.01). Salivary MDA levels were significantly higher in the OLP group compared with healthy subjects (P = 0.03). A significant correlation was found between serum and saliva TAA estimates in patients with OLP (r = 0.714 and P = 0.0001) and in the control group (r = 0.69 and P = 0.001). Significant correlation was also found between serum and saliva MDA values in control group (r = 0.464 and P = 0.04). A significant inverse correlation was found between salivary MDA and TAA values in the control group (r = -0.598 and P = 0.005).

The limitation of the study is the small sample size.

CONCLUSION

Authors found that LP is associated with a decrease in serum UA levels. For monitoring and treatment planning, UA may prove to be a useful biomarker of LP's antioxidant status.

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