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Original Research

Assessment of uric acid in patients with lichen planus

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ABSTRACT:

Background: Lichen planus is a chronic inflammatory condition that affects the skin, mucous membranes, hair, and nails.The present study was conducted to assess uric acid in patients with lichen planus. **Materials & Methods:**58 cases of lichen planus of both genderswere put in group I and healthy control 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 18 males and 40 females and group II had 29 males and 29 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:** A drop in serum UA levels is linked to LP. UA has the potential to be a valuable biomarker of LP's antioxidant status for monitoring and therapy planning.

Keywords: Lichen planus, nonsteroidal anti-inflammatory drugs, Uric acid

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INTRODUCTION

Lichen planus is a chronic inflammatory condition that affects the skin, mucous membranes, hair, and nails. It is characterized by the presence of small, itchy, flat-topped bumps or papules that can be purplish, reddish, or brownish in color.[1] These papules often have fine white lines or scales on their surface and may occur in clusters. Lichen planus can affect various parts of the body, including the wrists, ankles, lower back, genitals, mouth, and nails.[2]

The exact cause of lichen planus is not fully understood, but it is believed to involve an abnormal immune response.[3]Lichen planus is thought to involve an autoimmune reaction in which the body's immune system mistakenly attacks its own cells. However, the exact trigger for this immune response is unknown.Some individuals may have a genetic predisposition to developing lichen planus, as it can run in families.Certain infections, such as hepatitis C virus (HCV) infection, have been associated with the development of lichen planus in some cases.[4]Certain medications, including nonsteroidal anti-inflammatory drugs (NSAIDs), beta-blockers, and some antibiotics, have been implicated as potential triggers for lichen planus in some individuals.The symptoms of lichen planus can vary depending on the affected area of the body. In addition to the characteristic papules, lichen planus may cause symptoms such as itching, pain, burning, or tenderness. In cases where the mucous membranes are involved, lichen planus may cause ulcers, redness, or white patches in the mouth, genitals, or other areas.[5]

Uric acid (UA) is one of the important antioxidants in plasma. UA can scavenge ROS and can chelate metal ions. Thus, monitoring UA level in serum as an indicator of the antioxidant defense (oxidative balance) could be important for the clinicians' treatment strategy.[6,7]The present study was conducted to assess uric acid in patients with lichen planus.

MATERIALS & METHODS

The present study consisted of 58 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. Patients were put in group I and healthy control 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. Data thus

obtained were subjected to statistical analysis. P value < 0.05 was considered significant.

RESULTS

Table I Distribution of patients

Groups	Group I	Group II
Status	Lichen planus	Healthy
M:F	18:40	29:29

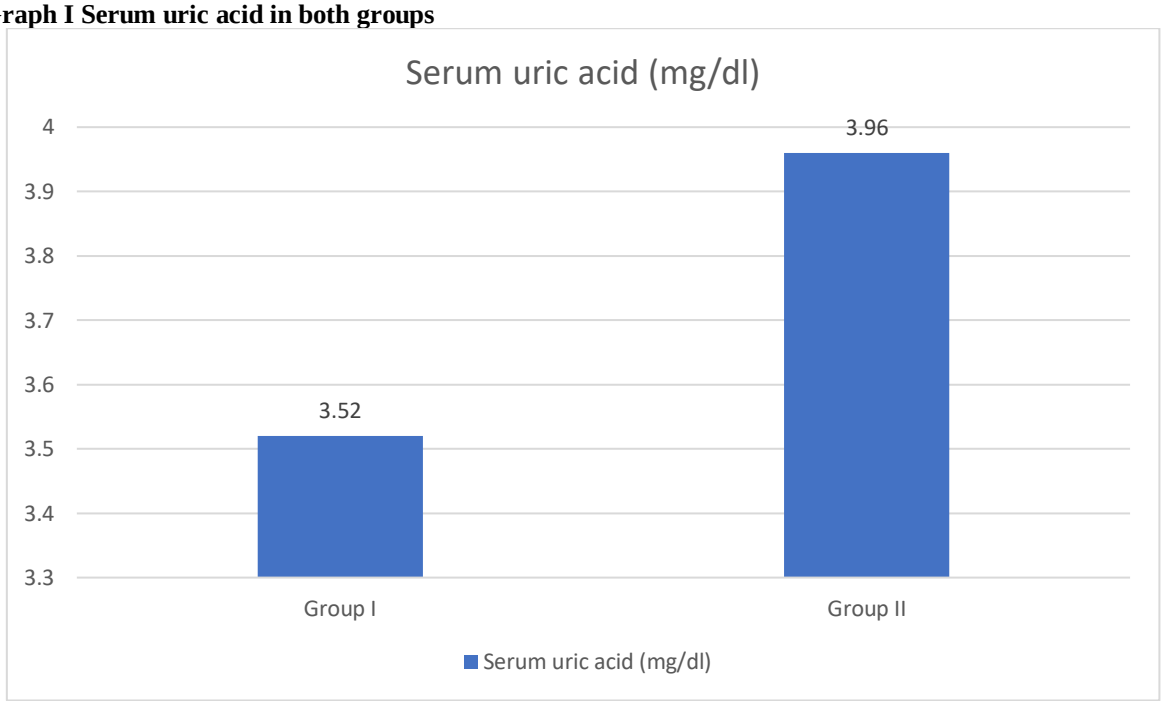
Table I shows that group I had 18 males and 40 females and group II had 29 males and 29 females.

Table II Serum uric acid in both groups

Groups	Serum uric acid (mg/dl)	P value
Group I	3.52	0.01
Group II	3.96	

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 Serum uric acid in both groups



DISCUSSION

One of the key antioxidants in human bodily fluids is UA, which is a potent free radical scavenger and may also function as a chelator of metal ions like iron and copper, transforming them into forms that are less reactive and incapable of catalyzing free-radical processes.[8] Antioxidants such as vitamins, certain enzymes, and others are the main defensive mechanisms against oxidative stress; nonetheless, it is believed that UA accounts for more than half of blood's antioxidant capacity. By neutralizing cellular superoxide and blocking its interaction with nitric oxide, UA stops the generation of peroxynitrite.[9,10] It is a scavenger of free radicals, including NO2, which is produced when peroxynitrite breaks down and could help remove superoxide by stopping the breakdown ofsuperoxide dismutase, the enzyme that is responsible for clearing superoxide from the cell.[11,12]The present study was conducted to assess uric acid in patients with lichen planus.

We found that group I had 18 males and 40 females and group II had 29 males and 29 females. Chakraborti et al[13]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.

We found that mean serum uric acid in group I was 3.52 mg/dl and in group II was 3.96 mg/dl. Sezer et al[14]evaluated the status of the oxidative stress and antioxidant defence system in patients with LP.In total, 40 patients with LP (23 men, 17 women; mean +/- SD age 43.27 +/- 1.96 years) and 40 control subjects, matched for age and gender, were enrolled in this prospective study. The exclusion criteria included medication with immunosuppressive agents, history of trauma and surgery, and history of alcohol ingestion for at least 1 month prior to the study. The

serum nitric oxide (NO), malondialdehyde (MDA) and superoxide dismutase (SOD) levels and the erythrocyte catalase (CAT) levels were investigated in both groups.Mean +/- SD levels of serum NO (74.60 +/- 17.96 micromol/L) and MDA (18.24 +/- 5.21 micromol/L) in patients with LP were higher than those of the control group (P = 0.007 and P = 0.031, respectively). Serum SOD levels (18.19 +/- 3.71 U/mL) in patients with LP were also higher than in healthy controls (P = 0.002). In contrast, erythrocyte CAT levels (13 557.80 +/- 4134.42 U/kg haemoglobin) were significantly lower in the patient group than in the control group (P = 0.009).
The limitation of the study is the small sample size.

CONCLUSION

Authors found that a drop in serum UA levels is linked to LP. UA has the potential to be a valuable biomarker of LP's antioxidant status for monitoring and therapy planning.

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