International Journal of Research in Dermatology
Sree JV et al. Int J Res Dermatol. 2018 Mar;4(1):38-40
http://www.ijord.com

DOI: http://dx.doi.org/10.18203/issn.2455-4529.IntJResDermatol20175701

Original Research Article

Study of serum uric acid levels in lichen planus

J. Vijaya Sree, V. Anand Kumar*

Department of Dermatology, Venereology & Leprosy, Great Eastern Medical School and Hospital, Srikakulam, Andhra Pradesh, India

Received: 22 November 2017
Revised: 18 December 2017
Accepted: 20 December 2017

***Correspondence:**
Dr. V. Anand Kumar,
E-mail: anand2derma@gmail.com

ABSTRACT

Background: Free radicals have an important involvement in the underlying mechanism of lichen planus (LP) and also various skin diseases. The aim of the study was to evaluate serum uric acid (UA) levels as a measure of the antioxidant defense status in lichen planus patients.
Methods: The duration of the present study was from September 2016 to October 2017 which includes 30 lichen planus patients and 30 other patients whose age and sex matched with the subjects served as controls.
Results: Most common type of lichen planus is classical lichen planus. In our study, serum UA levels were significantly decreased in patients with respect to controls 3.1±0.91 and 4.9±1.08 mg/dl respectively. The difference of means is 1.8 mg/dl and was found to be significant ($p<0.001$).
Conclusions: UA may be a potential, useful biomarker of antioxidant status in LP and a rational strengthening of the antioxidant defenses should be part of an optimal treatment strategy.

Keywords: Lichen planus, ROS, Lymphocytic infiltration

INTRODUCTION

Lichen planus (LP) is a chronic, mucocutaneous, papulo-squamous disorder consisting of small, shiny, flat-topped, polygonal, violaceous papules that may coalesce into plaques involving the skin, mucous membrane, scalp and nail. It can present in various forms including classical, hypertrophic, actinic, annular, follicular, eruptive, linear, pigmented and bullous types. It affects all races and occur usually from 30 to 70 years of age.

Lymphocytic infiltration and keratinocyte apoptosis has been observed to promote the activation of a cell-mediated immune response.[1] It has been suggested that the occurrence of LP could be triggered by imbalances among the antioxidant stress markers, and thus could play an important role in the pathogenesis of LP transformation.[2] An oxidative metabolism can lead to oxidative stress as a harmful byproduct and molecular destruction in living systems, which are subsequently involved in numerous processes, such as aging, mutagenesis, and a series of pathological events.

Oxidative stress in LP releases molecules consisting of granzymes that may result in local tissue damage in the effectors.[3] Antioxidants can defend against oxidative stress and are present in mammalian cells, enzymes such as superoxide dismutase, catalase, and glutathione peroxidase, as well as nonenzymatic antioxidants, including melatonin, uric acid (UA), and vitamins A and E.[4] UA can scavenge ROS (reactive oxygen species) and can chelate metal ions.[5] Thus, monitoring UA level in serum as an indicator of the antioxidant defense (oxidative balance) could be an important strategy for treatment. There is limited data available in the literature with conflicting reports of association of UA levels among LP patients: Some showed no difference, but others found decreased levels of UA. So, the present

study was undertaken to establish whether LP was accompanied by change in serum UA levels.

METHODS

This study was a case-control study conducted in the Department of Dermatology, Great eastern medical college and Hospital. The study was approved by the hospital ethical committee. All patients and control subjects consented for the study. This study was conducted from September-2016 to October-2017 and totally 30 LP patients with disease duration ranges from 1 month to 12 months were included. In addition, age-and sex- matched 30 patients who presented with minor ailments like pyoderma, tinea etc. were selected for controls.

Complete history and physical examination of all cases and controls were undertaken, and wherever necessary, skin biopsy was taken to confirm the diagnosis.

Exclusion criteria were the presence of any systemic disease (gout, diabetes, hypertension, thyroid disease, heart disease, kidney disease, hepatitis C), alcohol consumption, smoking, the consumption of drugs that increase UA (e.g. antidiuretics), immunosuppressive drugs, nonsteroid anti-inflammatory drugs, systemic, vitamin supplements over the past month, topical steroids for past 2 weeks, a history of surgery or trauma in the past month, and having been treated for lichen planus.

Five milliliter of venous blood sample was collected from each case and control after 12 h of fasting. All samples were coded and assayed in a blind fashion by an investigator who was unaware of the subjects' clinical status. Serum UA was assayed using semiautoanalyser, by uricase method. The data was analyzed by using statistical package for social sciences (SPSS version 18). P<0.05 was considered to be significant.

RESULTS

30 male patients and 30 female patients were included. The age of patients ranged from 25 to 70 years.

Table 1: Age distribution in study.

Age intervals in years	Males (%)	Females (%)
20-29	9 (15)	11 (18.3)
30-39	6 (10)	7 (11.6)
40-49	7 (11.6)	6 (10)
50-59	5 (8.3)	4 (6.6)
60-70	3 (5)	2 (3.3)
Total	30 (50)	30 (50)

Patients aged 20-29 years are more common in study.

Table 2: Types of lichen planus in study.

Types	Number of patients	Percentage (%)
Classical	35	58
Hypertrophic	10	16.7
Eruptive	6	10
Linear	5	8.3
Actinic	2	3.3
Annular	1	1.7
Follicular	1	1.7

Most common type of LP is classical lichen planus.

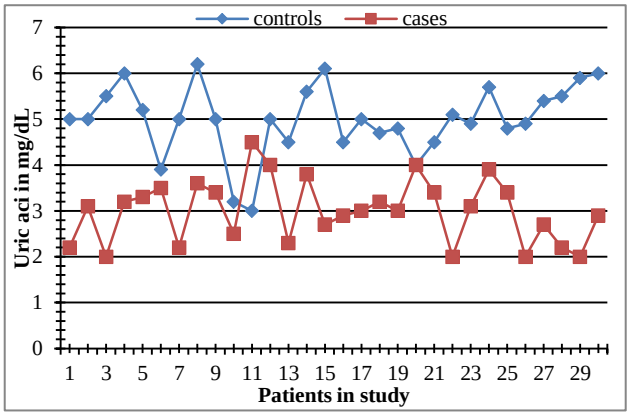

Figure 1: Uric acid levels in cases and controls.

Mean serum UA levels in patients and controls were 3.1±0.91 and 4.9±1.08 mg/dl, respectively and the difference was found to be significant (p<0.001).

DISCUSSION

Lichen planus is a chronic inflammatory disease. The pathophysiology of LP is multifarious and in the microscopic evaluation, its features are associated with pathognomonic characteristics of interphase dermatitis with degeneration of cells which is due to epithelial permeation of T-lymphocytes leading to local production of cytokines.[6] Recently, it has been stated that the imbalance in levels of free radical and ROS with antioxidants may play an important role in the start of several inflammatory disease.[7] ROS and tissues oxidative damage, following in extend a lack of antioxidants may result in appearance of this disease

The skin possesses an array of defense mechanisms that interact with ROS to obviate their deleterious effect. UA is an end product of purine metabolism and is produced in mammalian systems. The final two reactions of its production catalyzing the conversion of hypoxanthine to xanthine and the latter to uric acid are catalysed by the enzyme xanthine oxidoreductase, which may attain two inter-convertible forms, namely xanthine dehydrogenase or xanthine oxidase. The latter uses molecular oxygen as electron acceptor and generates superoxide anion and other reactive oxygen products. Evidence mainly based

on epidemiological studies suggests that increased serum levels of uric acid are a risk factor for cardiovascular disease where oxidative stress plays an important pathophysiological role. Also, allopurinol, a xanthine oxidoreductase inhibitor that lowers serum levels of uric acid exerts protective effects in situations associated with oxidative stress. It contaminates free radical substances through the inhibition of endothelial function under conditions of oxidative stress inside the cell in which glutathione is discharged. However, there is growing evidence indicating that the action of UA as an antioxidant *in vivo* and the administration of UA increase plasma antioxidant capacity. [8]

In our study, serum UA levels were significantly decreased in patients with respect to controls 3.1±0.91 and 4.9±1.08 mg/dl respectively. The difference of means is 1.8 mg/dl. This study is in correlation with Chakraborti with mean serum UA levels in patients and controls were 3.6 and 3.94 mg/dl, respectively.[9] The difference of means is 0.34 mg/dl. A significant decrease of UA levels was also observed in study of LP patients from Italy.[10] On the contrary, a report from Israel found higher prevalence of hyperuricemia than that of general population, though LP was not considered as a cause of overproduction of uric acid.[11]

Anshumalee et al reported that oxidative stress may play a role in oral LP.[12] Meanwhile, in another study, the potent antioxidant lycopene was found effective in the management of oral LP. This therapeutic effect indirectly points to the role of oxidative stress in the pathogenesis of LP.[13]

As free radical-induced damage is thought to be one of the important factors in the etiopathogenesis of LP, in our opinion, treatment guidelines should include optimal strengthening of antioxidant defense. Serum UA is a potent free radical scavenger, and it has been demonstrated, using two methodologically distinct assays, that systemic administration of UA increases *ex vivo* serum free radical scavenging capacity to a significantly greater extent than vitamin C, another important aqueous physiologic antioxidant.[13] Also, antioxidant administration increases UA levels.[14] Studies prove that oxidative stress is the initial event in the pathogenesis of LP. We suggest that antioxidant therapy may also help in clinical improvement.[15]

CONCLUSION

The results of our study suggest that LP may be related to depletion of UA levels in serum. UA may be considered as a useful biomarker of antioxidant status in LP for elaboration of treatment strategy and monitoring.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the institutional ethics committee

REFERENCES

1. Ergun S, Troşala SC, Warnakulasuriya S, Özel S, Önal AE, Ofluoğlu D, et al. Evaluation of oxidative stress and antioxidant profile in patients with oral lichen planus. J Oral Pathol Med. 2011;40:286–93.
2. Aly DG, Shahin RS. Oxidative stress in lichen planus. Acta Dermatovenerol Alp Pannonica Adriat. 2010;19:3–11.
3. ScrobotăI I, Mocan T, Cătoi C, Bolfă P, Mureşan A, Băciuţ G. Histopathological aspects and local implications of oxidative stress in patients with oral lichenplanus. Rom J Morphol Embryol. 2011;52:1305–9.
4. Nagler RM, Klein I, Zarzhevsky N, Drigues N, Reznick AZ. Characterization of the differentiated antioxidant profile of human saliva. Free Radic Biol Med. 2002;32:268–77.
5. Momen-Beitollahi J, Mansourian A, MomenHeravi F, Amanlou M, Obradov S, Sahebjamee M. Assessment of salivary and serum antioxidant status in patients with recurrent aphthous stomatitis. Med Oral Patol Oral Cir Bucal. 2010;15:557–61.
6. Khan A, Farah CS, Savage NW, Walsh LJ, Harbrow DJ, Sugerman PB. Th1 cytokines in oral lichen planus. J Oral Pathol. 2003;32:77–83.
7. Lobo V, Patil A, Phatak A, Chandra N. Free radicals, antioxidants and functional foods: Impact on human health. Pharmacogn Rev. 2010;4(8):118–26.
8. Glantzounis GK, Tsimoyiannis EC, Kappas AM, Galaris DA. Uric acid and oxidative stress. Curr Pharm Des. 2005;11:4145–51.
9. Chakraborti G, Biswas R, Chakraborti S, Sen PK. Altered Serum Uric Acid Level in Lichen Planus Patients: Indian J Dermatol. 2014;59(6):558–61.
10. Battino M, Greabu M, Totan A, Bullon P, Bucur A, Tovaru S, et al. Oxidative stress markers in oral lichen planus. Biofactors. 2008;33:301–10.
11. Shai A, Halevy S. Lichen planus and lichen planus-like eruptions: Pathogenesis and associated diseases. Int J Dermatol. 1992;31:379–84.
12. Anshumalee N, Shashikanth MC, Sharma S. Oxidative stress and oral lichen planus: A possible association? Cusp. 2007;4(2):31–4.
13. Anshumalee N and Shashikanth MC. Efficacy of oral lycopene in management of lichen planus [dissertation]. [Bangalore]: Rajv Ghandi University of Health Sciences, Bangalore; 2007: 91-119.
14. Mathur A, Mathur L, Manohar B, Mathur H, Shankarapillai R, Shetty N, et al. Antioxidant therapy as monotherapy or as an adjunct to treatment of periodontal diseases. J Indian Soc Periodontol. 2013;17:21–4.
15. Sezer E, Ozugurlu F, Ozyurt H, Sahin S, Etikan I. Lipid peroxidation and antioxidant status in lichen planus. Clin Exp Dermatol. 2007;32:430–4.

Cite this article as: Sree JV, Kumar VA. Study of serum uric acid levels in lichen planus. Int J Res Dermatol 2018;4:38-40.