Oxidative Stress in Classic Type Lichen Planus

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ABSTRACT

Background: *Lichen planus (LP) is an autoimmune inflammatory disease of the mucocutaneous tissue, whose exact pathological course is not yet understood. Many studies have implicated the role of reactive oxygen species (ROS) and the protective role of antioxidants in several autoimmune skin disorders.* ***Aim of study:*** *is to evaluate the role of oxidant/antioxidant status in patients with classic type lichen planus.* ***Patients and Methods:*** *The study included 30 patients of classic type lichen planus attending Dermatology, Venereology and Andrology Outpatient Clinic in Sohag University Hospital, and 20 healthy controls. Serum and tissue levels of nitric oxide(NO),malondialdehyde (MDA), superoxide dismutase(SOD), and catalase (CAT) were measured in patients with classic type lichen planus.* ***Results:*** *The LP patients showed statistically significant higher level of serum and tissue NO, MDA and SOD compared to that of controls. On the other hand, patients showed statistically significant lower level of serum and tissue CAT. Male patients showed statistically significant higher levels of serum NO, MDA and SOD than that of female patients. On the other hand, male patients had statistically significant lower level of serum and tissue CAT when compared with female patients. On the other hand male patients showed statistically significant lower levels of serum and tissue CAT than that of male controls.* ***Conclusions:*** *The imbalance between oxidant/ antioxidant mechanisms may be a primary etiological factor or a secondary possible etiological factor in pathogenesis of classic LP.*

INTRODUCTION

Lichen planus (LP) is defined as subacute, chronic dermatosis characterized by small, flat-topped, shiny, polygonal, violaceous papules that may coalesce into plaques. It is commonly seen in the skin, mucous membrane, genitalia, nails, scalp and hair follicles [(1)], and its etiology is unknown until now[(2)].

Lichen planus occurs throughout the world, in all races. It may be familial in about 1-2% of cases. It appears in men at a constant rate from the early twenties through sixties, whereas in women, the rate of new cases continue to increase with increasing age, reaching a peak in the sixties [(3)]. Both oral and cutaneous LP has rarely been reported in childhood[(4)].

The clinical presentation of the disease is variable, with several forms involving generalized (classic) type, lesion affecting mucous membrane, hypertrophic, follicular, linear**,** actinic, pigmentosus, atrophic, annular and lesions affecting palm and sole [(5)].

The precise pathological course of LP is not yet understood. Cytokine-mediated apoptosis of basal keratinocytes as a result of the accumulation of activated T-cells in the dermoepidermal junction had been proposed as one mechanism, but the initial antigen which triggers that process has not yet been identified[6-8]. Antioxidants (both enzymatic and nonenzymatic) act as a physiological protective means against reactive oxygen species (ROS) and cytokine-associated toxicity in dermatological disorders[9,10]. Reduced antioxidant levels as a result of extended release of ROS, such as in chronic inflammation, increase oxidative damage[11]. Oxidative stress is an imbalance in oxidant and antioxidant levels. If an overproduction of oxidants overwhelms the antioxidant defenses, oxidative damage of cells, tissues and organs ensues. In some cases, oxidative stress is assigned as a causal role in disease pathogenesis, whereas in others, the link is less certain [12].

Aim of the Work:

The aim of the present study is to evaluate the role of oxidant/ antioxidant status by comparing serum and tissue level of nitric oxide (NO), malondialdehyde (MDA), superoxide dismutase (SOD), and catalase (CAT) in patients with classic type lichen planus and controls.

PATIENTS & METHODS

The study was conducted on 30 patients of classic type lichen planus attending Dermatology, Venereology and Andrology Outpatient Clinic in Sohag University Hospital, compared with 20 healthy controls.

Prior to initiation of the study, a master sheet including personal history, disease history (onset, duration and distribution), drug history and clinical examination was obtained from all patients and every subject was informed about the aim of the study and a written informed consent was obtained from them.

Exclusion criteria:

Included those who received any systemic treatment suppressing the immune system for the last 4 weeks, patients who received topical medications for the last 2 weeks before sample collection, patients with history of trauma or any surgery 4 weeks prior to sampling, patients suffering from any systemic or dermatological disease affecting the immune system or any malignancy and those with specific habits as smoking or alcohol drinking were also excluded.

Methods:

Both patients and controls had been subjected to blood sampling and skin biopsy for assessment of oxidant/ antioxidant status in blood and skin respectively.

Skin biopsy:

Four mm punch biopsy was taken from one of the active skin lesions of all patients and from the skin of controls. Biopsy samples were transferred into aliquots and stored at -40°C until use. Tissue samples were weighed and homogenized using a glass homogenizer under cold phosphate buffer. The homogenate was then centrifuged and the supernatant was used for the determination of tissue SOD, CAT

activity and NO as well as MDA levels.

Blood samples:

After 12 hours of fasting, 5 ml venous blood had been withdrawn in a dry clean tube. Blood was centrifuged and the serum was aspirated and transferred into aliquots and stored at -40°C till assay.

1. **Measurement of CAT activities:** Catalase activities were measured by colorimetric method using commercial kit (Biodiagnostic; Pharmaceutical Industries, CAT. No. CA 25 16, Egypt) [(13)].
2. **Measurement of Lipid peroxide (MDA) levels:** MDA levels were measured by colorimetric method using commercial kit (Biodiagnostic; Pharmaceutical Industries, CAT. No. MD 25 28, Egypt) [(14)].
3. **Measurement of SOD activities:** Superoxide dismutase activities were measured by colorimetric method using commercial kit (Biodiagnostic; Pharmaceutical Industries, CAT. No. SD 25 20, Egypt)[(15)].
4. **Measurement of $NO^{\cdot}$ Levels:** Nitric oxide levels were measured by colorimetric method using commercial kit (Biodiagnostic; Pharmaceutical Industries, CAT. No. NO 25 32, Egypt) [(16)].

Statistical Analysis:

SPSS software version 9 was used for statistical analysis. LP patients were compared with controls using unpaired students t- test for comparison of means. Chi- square test was used for comparing categorical data. Person correlation was used for correlation study. Data were presented either as mean ± SD or numbers and percentage as appropriate.

RESULTS

The present study included 30 patients with classic type lichen planus [20 females (66.7%) and10 males (33.3%)] .Their ages ranged 23 to 65 years, with a mean of 41.66± 10.46 years. Their ages and sex were matched with 20 healthy controls. There were no significant differences in demographic data (table 1).

Table (2) presents the clinical data of the patient`s criteria. The LP patients showed statistically significant higher level of serum and tissue NO, MDA and SOD compared to that of controls. On the other hand, patients showed statistically significant lower level of serum and tissue CAT (table 3).

Male patients showed statistically significant higher levels of serum and tissue NO, MDA and SOD than that of female patients. On the other hand, male patients had statistically significant lower level of serum and tissue CAT when compared with female patients.

On the other hand male patients showed statistically significant lower levels of serum and tissue CAT than that of male controls (tables 4,5) .

The serum and tissue oxidants/antioxidants including NO, MDA and SOD showed statistically significant positive correlation when compared with each other and with duration of illness. On the other hand, these parameters showed statistically significant negative correlation with each of serum and tissue CAT. The serum and tissue CAT showed statistically significant negative correlation with duration of illness (table 6).

Table 1: Demographic data

Variable	Patients (N=30).	Controls (N= 20).	P- value
Age (years) Range Mean ± SD	 26 – 62 41.66 ± 10.46	 26- 48 38 ± 7.22	0.14
Sex [No. (%)] Male Female	 10 (33.3 %) 20 (66.7%)	 8 (40%) 12 (60%)	0. 765
Residence [No. (%)] Rural Semi urban Urban	 14 (46.7 %) 10 (33.3 %) 6 (20 %)	 10 (50%) 5 (25%) 5 (25%)	0.8
Marital status [No.(%)] Single Married	 9 (30%) 21 (70 %)	 5(25%) 15(75%)	0.758

Table (2): Clinical data of the patients criteria

Clinical information	Data
Duration (years) Mean ± SD Range	 1.91±3.84 0.02- 18.00
Course [No.(%)] Fluctuating Progressive Stationary	 15 (50%) 10 (33.3%) 5 (16.7%)
Family history [No.(%)] Positive Negative	 3 (10%) 27 (90 %)
Residual hyperpigmentation [No.(%)] Positive Negative	 19 (63.3 %) 11 (36.7 %)
Kobner´s phenomena [No.(%)] Positive Negative	 5 (16.7 %) 25 (83.3 %)
Affected site ** [No.(%)] Chest Abdomen Back Upper limb Lower limb Both limbs	 9 (30 %) 17 (56.7%) 11 (36.7 %) 6 (20%) 1 (3.3 %) 23 (76.7 %)

Table (3): comparison between serum and tissue levels of NO, MDA, SOD and CAT in patients and controls

Biochemical item	**Patients(n=30)**		**Controls(n=20)**		**P-value**	
	Mean± SD (serum)	**Mean± SD (tissue)**	**Mean± SD (serum)**	**Mean± SD (tissue)**	**(serum)**	**(tissue)**
NO (µmol/L)	68.07± 34.41	248.29± 174.2	29.86± 14.47	59.11± 60.94	<0.001	< 0.001
MDA (nmol/ml)	42.36± 36.18	2327.46± 552.57	16.34± 8.81	313.42± 349.65	0.001	< 0.001
SOD (U/ml)	67.83± 35.50	1189.34± 1017.34	37.62± 33.44	363.24± 290.3	0.004	< 0.001
CAT (U/L)	853.08± 510.9	118.02± 131.44	1461.61± 856.5	241.48± 136.76	0.008	0.002

Table (4): Comparison of serum levels of oxidants/antioxidants between male and female patients

	Male patients (n= 10)	**Female patients (n = 20)**	**Male controls (n= 8)**	**Female controls (n=12)**	**P_1**	**P_2**	**P_3**
NO (µmol/L)	104.02± 12.86	50.1± 26.62	25.44 ± 14.54	32.82± 14.26	<0.001	<0.001	0.02
MDA (nmol/ml)	80.87± 37.45	23.1± 12.78	13.02± 5.06	18.56± 10.22	<0.001	<0.001	0.3
SOD (U/ml)	108.26± 22.6	47.61± 19.78	31.79± 26.28	41.50± 38.09	<0.001	<0.001	0.61
CAT (U/L)	443.2± 60.9	1057.97± 513.87	1247.61± 721.76	1604.28± 938.15	0.016	0.02	0.08

Data is presented as mean ±SD

P_1: P-value of comparison of the means between the male and female patients.

P_2: P-value of comparison of the means between male patients and male controls.

P_3: P-value of comparison of the means of the female patients and female controls.

Table (5): Comparison of tissue levels of oxidants/antioxidants between male and female patients

	Male patients (n= 10)	**Female patients (n = 20)**	**Male Controls (n=8)**	**Female controls (n=12)**	**P_1**	**P_2**	**P_3**
NO (umol/L)	462.16± 117.82	141.36± 60.35	57.24± 50.67	60.36± 69.11	<0.001	<0.001	0.002
MDA (nmol/g tissue)	5141.3± 2671.05	920.53± 560.79	290.80± 191.75	328.50± 432.61	<0.001	<0.001	0.002
SOD (U/g tissue)	2264.02± 1138.11	652± 232.15	427.95± 204.13	320.10± 337.62	<0.001	<0.001	0.003
CAT (U/g tissue)	21.87± 14.2	166.09±. 137.76	245.09± 159.83	239.07± 126.62	0.004	<0.001	0.14

Data is presented as mean ±SD

P_1: P-value of comparison of the means between the male and female patients.

P_2: P-value of comparison of the means between male patients and male controls.

P_3: P-value of comparison of the means of the female patients and female controls.

Table (6): correlation studies

	Duration	**Serum NO**	**Serum MDA**	**Serum SOD**	**Serum CAT**	**Tissue NO**	**Tissue MDA**	**Tissue SOD**	**Tissue CAT**
Serum NO									
.r*	0.625		0.870	0.931	-0.889	0.899	0.795	0.787	-0.831
.p	<0.001		<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
Serum MDA									
.r	0.911			0.901	-0.676	0.940	0.919	0.937	-0.616
.p	< 0.001			<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
Serum SOD									
.r	0.684				-0.820	0.969	0.919	0.895	-0.784
.p	<0.001				<0.001	<0.001	<0.001	<0.001	<0.001
Serum CAT									
.r	-0.410					-0.739	-0.616	-0.617	-0.970
.p	0.024					<0.001	<0.001	<0.001	<0.001
Tissue NO									
.r	0.735						0.961	0.943	-0.686
.p	<0.001						<0.001	<0.001	<0.001
Tissue MDA									
.r	0.790							0.979	-0.566
.p	<0.001							<0.001	0.001
Tissue SOD									
.r	0.844								-0.568
.p	<0.001								0.001
Tissue CAT									
.r	-0.366								
.p	0.04								

r*: Person correlation.

DISCUSSION

Lichen planus (LP) is an autoimmune inflammatory disease of the mucocutaneous tissue [17].

In general, oxidative stress is caused by an imbalance between the production of ROS and a biological system's ability to detoxify the reactive intermediates or easily repair the resulting damage [18,19]. Oxidative stress leads to excessive accumulation of free radicals, which damage cellular compounds such as proteins, carbohydrates, DNA and lipids [20].

NO is a gaseous free radical that is released by the family of No synthetase enzymes. It is a potent vasodilator, thus contributing considerably to the cardinal signs of inflammation .It is also known to exhibit cytotoxic effects in human skin [21]. In the present study, serum and tissue levels of NO were significantly higher in LP patients compared to controls. Also, there was a significant higher serum and tissue levels of MDA in LP patients than in controls.

Such finding agrees with those of **Sezer et al.,** and **Aly and Shahin,** [22,23] in their study of 40 LP patients, who reported significant increase of serum NO in LP patients compared to controls, and suggested that oxidative stress resulting in generation of free radicals may play a role in pathogenesis of LP.

The skin possesses an array of defense mechanisms that interact with ROS to obviate their deleterious effect. SOD is considered the first line defense against oxygen-derived free radicals, converting the superoxide anion ($O_2^{\bullet-}$) into H_20_2. H_2O_2 is dangerous in the cell because it can be easily converted into hydroxyl radicals, one of the most destructive free radicals [24]. There are also other enzymes such as CAT, which decomposes peroxides. CAT is considered the main enzyme involved in removing H_20_2 [25]. In the present study, there were significant higher levels of tissue and serum SOD in LP patients in comparison to controls. On the other hand, there were significant lower levels of serum and tissue CAT in LP patients in comparison to controls. In agreement with these results, **Sezer et al.,** and **Aly and Shahin,** [22,23] reported significant higher level of serum SOD and significant lower level of serum CAT in their LP patients.

Although, in the present study, there were significant higher levels of tissue and serum NO, MDA and SOD and significant lower levels of tissue and serum CAT in LP patients in comparison to controls, the changed oxidant/ antioxidant levels in the serum of LP patients might be a peripheral response to oxidative stress. So. these findings may indicate that measurement of tissue oxidant/ antioxidant levels may be more acceptable than the serum measurement as the latter may not be able to determine the origin of these oxidant and antioxidant enzymes.

In the present study, there were significant higher levels of serum and tissue NO, MDA, SOD in male patients compared to those of females. On the other hand, there were significant lower levels of serum and tissue CAT in male patients than in females. That finding supports the hypothesis that oxidative stress is

greater in men than in women [26] which may be related to female protection from the damaging effect of oxidative stress by the antioxidant properties of estrogens. Estradiol has been documented as having antioxidant effects [27].

This finding is in accordance with that of **Aly and Shahin,** [23] who reported that male LP patients had significant higher level of serum MDA and significant lower level of serum CAT when compared to female patients. However, contrary to our results, the previous authors in this study reported no significant difference between male and female LP patients regarding NO and SOD serum levels.

In the present study, the parameters of oxidant and antioxidant levels including serum and tissue NO, MDA and SOD showed statistically significant positive correlations when every two of these parameters correlated with each other and with the duration of illness (Table 6). On the other hand, each of the serum and tissue NO, MDA and SOD showed significant negative correlations when everyone correlated with serum and tissue CAT. Also, there was a statistically significant negative correlation between serum and tissue CAT and the duration of illness. In agreement with our results, **Aly and Shahin,**[23] reported significant positive correlation between serum NO, MDA and SOD and duration of illness and negative correlation between serum CAT and duration of illness. Also, the authors reported significant positive correlation between serum NO and SOD and significant negative correlation between serum CAT and MDA, but contrary to our results, these authors, reported no significant correlation between serum NO and MDA, serum NO and CAT, serum CAT and SOD and between serum SOD and MDA.

From these results, it could be concluded that free radicals and the resulting oxidative damage might be important in the pathogenesis of LP lesions. Further longitudinal studies are required to investigate the potential role of oxidative stress in classic type LP patients.

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تأثير جهد الأكسدة فى النوع الكلاسيكى للحزاز المنبسط

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الحزاز المنبسط هو التهاب مزمن يصيب الجلد والأغشية المخاطية، وما زالت أسباب المرض غير واضحة، ولكنه ثبت أنه نتيجة لخلل مناعى ذاتى. وهناك دراسات عديدة على دور الفصائل الاكسجينية الحرة النشطة ومضادات الاكسدة .

أهداف البحث: تحديد تأثير جهد الأكسدة على حدوث الحزاز المنبسط كلاسيكي النوع و ذلك بواسطة قياس نسبة كل من أكسيد النيتريك ، وثنائي الدهيد المالون ، وإنزيمات سوبر أكسيد ديسميوتاز، و كتالاز في أنسجة و دم مجموعة من مرضى الحزاز المنبسط كلاسيكي النوع و مقارنتها بمجموعة من الأشخاص الأصحاء.

طريقة البحث: أجرى البحث على ٣٠ مريضا من مرضى الحزاز المنبسط كلاسيكي النوع الذين يترددون على عيادة الأمراض الجلدية بمستشفى سوهاج الجامعي ومقارنتهم بالمجموعة الضابطة و عددهم ٢٠ شخص سليم روعي في هذه الدراسة أن يكون كل من المرضى والأصحاء غير مدخنين أو يخضعون لأي علاج خلال فترة ٢-٤ أسابيع أو أي عمليات جراحية خلال فترة ٤ أسابيع على الأقل قبل إجراء الدراسة و لا يعانون من أية أمراض أخرى ظاهرة .

تم التعامل مع المرضى و المجموعة الضابط بأخذ عينة من أنسجة الجلد (٤مللتر) وأخرى من الدم،و قد خضعت هذه العينات لمجموعة من العمليات الكيميائية و ذلك لتحديد نسبة المواد المؤكسدة و مضادات الأكسدة.

نتائج البحث: أظهرت نتائج البحث أن هناك زيادة في أكسيد النيتريك، ثنائي الدهيد المالون ،و سوبر أكسيد ديسميوتاز بأنسجة و دم المرضى بالمقارنة بنتائج أنسجة و دم الأصحاء. وأن هناك نقص في نشاط إنزيم الكتالاز بأنسجة و دم المرضى مقارنة بنتائج أنسجة و دم الأصحاء. وأن هناك زيادة في أكسيد النيتريك، ثنائي الدهيد المالون ، وسوبر أكسيد ديسميوتاز بأنسجة و دم المرضى الذكور مقارنة بنتائج أنسجة و دم المرضى الإناث. كما وجد أن هناك نقص في نشاط إنزيم الكتالاز بأنسجة و دم المرضى الذكور مقارنة بنتائج أنسجة و دم المرضى الإناث.

الاستنتاج: تشير نتائج هذه الدراسة إلى أن جهد التأكسد له دور في حدوث مرض الحزاز، وقد يكون جهد التأكسد عاملا أساسيا مسببا للمرض أو عاملا ثانويا نتيجة لعوامل أخرى مسببة للمرض. كذلك تشيرنتائج هذه الدراسة إلى أن الذكور المصابين بمرض الحزاز أكثر عرضة لجهد التأكسد من المرضى الإناث.