

ORIGINAL ARTICLE**EVALUATION OF ROLE OF ANTIOXIDANTS IN THE TREATMENT OF PHOTO DERMATITIS**Deepti Jain¹, Harvir Singh Sodhi²

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ABSTRACT:

Background: many diseases are provoked by sunlight directly or indirectly. Skin damage occurs due to prolonged and high doses of UV on prematurely age the skin. These changes are predominantly caused by medium wavelengths and can occur in anyone with sufficiently high levels of UV exposure. Abnormal reactions to UV, however, are predominantly triggered by UV-A radiation and do not affect everyone. These diseases are considered to be true photodermatoses, the subject of this article. Hence we aim to impact of Vitamin E supplements in patients with photodermatitis. **Materials & Methods:** 40 patients diagnosed with photodermatitis were included in the study. The mean age of the patients was 35 years. Chemicals used in the present study were: NADPH, oxidized and reduced glutathione, 1, chloro, 2,4, dinitrobenzene (CDNB), glutathione reductase and bovine serum albumin (Fraction V) were obtained from Sigma Chemical Company (St. Louis, Mo, USA). 40 subjects with same age group were taken as control. All the patients were preinformed about the study and written consent was obtained from them. Blood samples were collected from each case on day one and after one week of treatment, and from control subjects once by venepuncture. Haemoglobin levels were measured in each patients followed by isolation of erythrocytes and their subsequent haemolysis. Lowery et al's method was used for estimation of protein content.⁴ The data were analysed by using SPSS PC (version 5.0) software. Paired 't' test was employed for the comparison of data between the groups. **Results:** Serum Malondialdehyde showed an approximate 35% rise in concentration in photodermatitis cases as compared to the control group. Superoxide dismutase activity and blood GSH level were decreased by approximately 12 % and 29 % respectively while activities of Catalase were increased in photodermatitis cases patients as compared to control. **Conclusion:** Oxidative stress might be a responsible cause in the pathogenesis of photodermatitis. Administration of oral Vitamin E modulates the antioxidant enzymes providing relief clinically. Future research advocated.

Key words: Photodermatitis, Sunlight, Vitamin E.

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INTRODUCTION

Sunlight provokes many diseases directly or indirectly. Acute reactions, such as sunburn, which are induced by excessive UV radiation must be differentiated from abnormal reactions to sunlight. However, prolonged and therefore cumulative high doses of UV also prematurely age the skin and lead to damage such as skin cancer. These changes are predominantly caused by medium wavelengths and can occur in anyone with sufficiently high levels of UV exposure. Abnormal reactions to UV, however, are predominantly triggered by UV-A radiation and do

not affect everyone. These diseases are considered to be true photodermatoses, the subject of this article. Photodermatoses are classified into primary and secondary types. Primary photodermatoses are induced by photosensitizing substances. They are referred to as idiopathic primary photodermatoses if the etiology is unknown.^{1, 2} While electromagnetic radiation is the critical pathogenic factor with primary photodermatoses, secondary heliotropic diseases have another genesis altogether, although they are also induced by sunlight. Secondary photodermatoses are frequently a feature of systemic diseases such as lupus erythematosus,

metabolic disorders such as porphyrias, or disorders of DNA repair such as xeroderma pigmentosum.³ Hence we aim to impact of Vitamin E supplements in patients with photodermatitis.

MATERIALS & METHODS

The study included a total of 40 patients with mean age of 35 years (22years to 44 years) reporting in the institute with the complaint of photodermatitis were included in the study. Chemicals used in the present study were: NADPH, oxidized and reduced glutathione, 1, chloro, 2,4, dinitrobenzene (CDNB), glutathione reductase and bovine serum albumin (Fraction V) were obtained from Sigma Chemical Company (St. Louis, Mo, USA). Pyrogallol and 2-thiobarbituric acid (TBA) were obtained from E. Merck (Mumbai, India). Patients who were fulfilled the following criteria were considered as patients of photodermatitis: (i) recurrent pruritic eruption clearly precipitated/ aggravated by sun exposure; (ii) eruption predominantly confined to sun exposed area; (iii) monomorphic/uniform type of lesion(s) in each patient; (iv) history of relief over time following sun protection/sun screening in earlier episodes; and (v) skin biopsy consistent with PMLE. Patients with active infection, eczema or history of any other photo aggravated dermatoses, or with any other known cause or modifier of oxidative stress parameters like diabetes, hypertension, etc or on drugs known to cause photodermatitis, eruptions/oxidative stress e.g.,

tetracycline, quinolones, corticosteroids, were excluded. Ethical approval was taken from the institution for the present study. All the patients were advised to use topical sunscreen observe strict photoprotection and were given oral vitamin E supplementation (400 mg α - tocopherol acetate OD) for 7 days. 40 subjects with same age group were taken as control. All the patients were preinformed about the study and written consent was obtained from them. Blood samples were collected from each case on day one and after one week of treatment, and from control subjects once by venepuncture. Haemoglobin levels were measured in each patients followed by isolation of erythrocytes and their subsequent haemolysis. Lowery et al’s method was used for estimation of protein content.⁴ The data were analysed by using SPSS PC (version 5.0) software. Paired ‘t’ test was employed for the comparison of data between the groups.

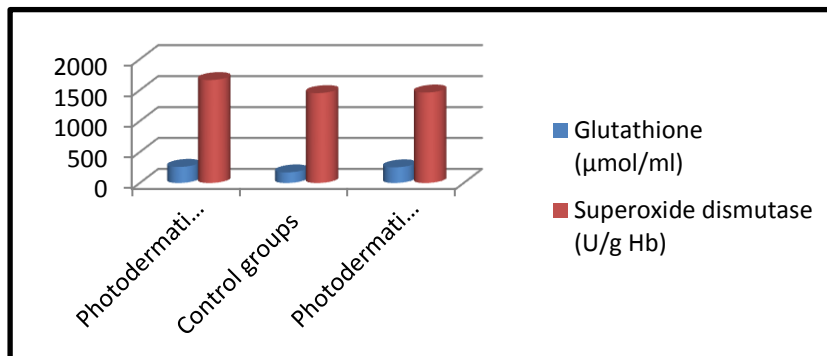
RESULTS

Approximate 35% rise in concentration of serum Malondialdehyde was found in photodermatitis cases as compared to the control group (P<0.001, Table 1, Graph 1). Superoxide dismutase activity and blood GSH level were decreased by approximately 12 % and 29 % respectively while activities of Catalase were increased in photodermatitis cases patients as compared to control (Graph 2).

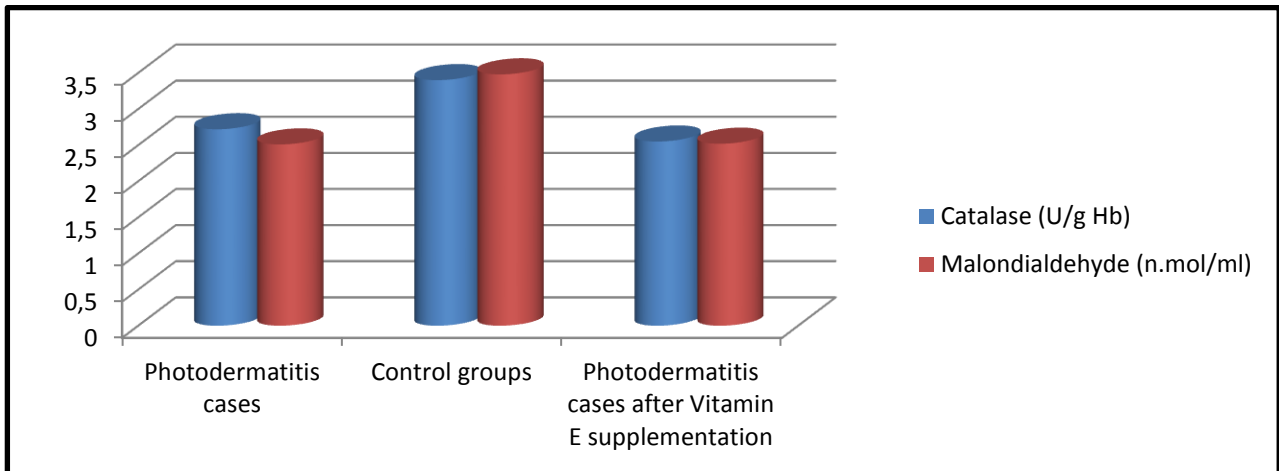
Table 1: Malondialdehyde level (in serum) and activity of antioxidant enzymes (in RBC) and glutathione level (in blood) of control subjects and photodermatitis cases

Variable	Photodermatitis cases	Control groups	Photodermatitis cases after Vitamin E supplementation	p-value
Catalase (U/g Hb)	2.72 + 0.51	3.40 + 0.42	2.55 + 0.71	0.002 (s)
Superoxide dismutase (U/g Hb)	1664 + 188.4	1453 + 110.2	1462 + 130.2	0.012 (s)
Malondialdehyde (n.mol/ml)	2.51 + 0.41	3.48 + 0.34	2.52 + 0.57	0.031 (s)
Glutathione (μ mol/ml)	260.4 + 22.12	171.5 + 16.52	252.8 + 15.42	0.001 (s)

Graph 1: Mean Activity of antioxidant enzymes (in RBC) and glutathione level (in blood) of control subjects and photodermatitis cases



Graph 2: Mean Activity of other antioxidant enzymes (in RBC) of control subjects and photodermatitis cases



DISCUSSION

Dermatosis associated with cutaneous photosensitivity is a group of photo distributed cutaneous eruptions caused or exacerbated by sunlight. Multiple clinical variants of photosensitive dermatosis have been characterized including solar urticaria, phototoxic, photo allergic dermatitis etc.⁵ Photodermatitis manifests as pruritus, erythema, macules, papules, or vesicles on sun-exposed skin arising 1-2 days after exposure and resolving spontaneously over the next 7-10 days. It is most common with initial sun exposures during spring or early summer; “hardening” of the skin may occur with subsequent exposures. Various morphological variants like the micropapular, lichen nitidus like and lichen planus like lesions have been reported.⁶ The treatment consists of avoidance of sun exposure, use of protective clothing, and regular application of broad-spectrum sunscreens, Psoralen UltraViolet A and Narrow Band –Ultra Violet B. Sharma and Basnet in their study observed that irrespective of the type of clothing or weave, the covered areas were unaffected which suggests that any type of protective clothing can prevent PMLE.⁷ Hence we evaluated the effect of Vitamin E supplements in patients with photodermatitis. We observed a high levels of serum MDA in Photodermatitis cases (Table 1) which indicates that in this UV induced photodermatitis lipid peroxidation occurs as a result of oxidative stress (OFR generation) like in other such environmental stressors.⁸ Further, serum MDA levels were found to be associated with the extent and severity of the disease (P<0.001). Persistence of inhibition activity of SOD may be due to continuous oxidative stress

induced by photodermatitis. A negative association of SOD and MDA further supported this. Further, we found a significant increase in CAT levels in PMLE cases as compared to normal subjects and the extent of elevation of CAT correlate positively with the extent of PMLE (P<0.001). This was based on comparison between CAT activity and clinical diagnosis of the patients observed by the dermatologist based on criteria mentioned.⁹ Srinivas et al reviewed the data on photodermatitis in India. They emphasized that photodermatoses are a group of disorders resulting from abnormal cutaneous reactions to solar radiation. They include idiopathic photosensitive disorders, drug or chemical induced photosensitivity reactions, DNA repair-deficiency photodermatoses and photoaggravated dermatoses.¹⁰ The pathophysiology differs in these disorders but photoprotection is the most integral part of their management. Photoprotection includes wearing photoprotective clothing, applying broad spectrum sunscreens and avoiding photosensitizing drugs and chemicals.¹¹ Lehmann et al reviewed the literature of the past 20 years and describe the modern nosology of photodermatoses and their clinical features, diagnosis, and treatment. From the results, they concluded that Photodermatoses are not life-threatening but can cause considerable suffering. Prevention is just as important as treatment.¹² The incidence of photodermatoses is common in conditions were tropical weather favours its development. Knowledge regarding sun protection, and inadvertent consumption of phototoxic drugs further predisposed for the development of the disease. Identification of the

cause and avoidance of triggering factors will help in reducing the incidence of photodermatoses.^{10, 13}

CONCLUSION

From the above results, we conclude that oxidative stress might be a responsible cause in the pathogenesis of photodermatitis. Administration of oral Vitamin E modulates the antioxidant enzymes providing relief clinically. Hence, we support inclusion of antioxidants in a diet of susceptible persons prophylactically. Future research with higher study group is advocated.

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