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Original Article

Oxidative stress markers and antioxidant status in erythrodermic vs chronic plaque psoriasis

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ABSTRACT

Background: "Tinea corporis is a prevalent superficial fungal infection globally and has become a notable dermatological issue in India due to rising chronicity, recurrence, and treatment resistance. Diabetes mellitus is acknowledged as a significant risk factor for fungal infections due to compromised immune responses and modified skin barrier function. Nonetheless, information concerning the clinico-mycological attributes of tinea corporis and its correlation with diabetes mellitus in Eastern India is scarce. **Materials and Methods**: In this hospital-based comparative cross-sectional study, 40 adult patients were enrolled, including 20 patients with EP and 20 with CPP. Disease severity was assessed using the Psoriasis Area and Severity Index (PASI), body surface area (BSA) involvement, erythrocyte sedimentation rate (ESR), and C-reactive protein (CRP). Circulating oxidative stress biomarkers, including malondialdehyde (MDA), lipid hydroperoxides (LOOH), advanced oxidation protein products (AOPP), protein carbonyls, nitric oxide metabolites (NOx), and 8-hydroxy-2′-deoxyguanosine (8-OHdG), together with antioxidant parameters comprising superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPx), reduced glutathione (GSH), total antioxidant capacity (TAC), vitamin C, and vitamin E, were quantified using standardized biochemical assays. Statistical analysis utilised suitable parametric and non-parametric tests, whereas multivariable logistic regression identified factors linked to chronic and recurring disease. A significant number of patients with tinea corporis exhibited diabetes mellitus. Diabetic patients exhibited markedly prolonged illness duration, extensive lesion involvement, elevated recurrence rates, and a higher incidence of multi-site involvement compared to non-diabetic patients ($p < 0.05$). Trichophyton species were the primary isolates, with the Trichophyton mentagrophytes complex being the most commonly identified dermatophyte. Inadequate glycaemic management was markedly linked to chronic and recurring infections. Multivariable analysis identified diabetes mellitus as an independent predictor of widespread and recurrent tinea corporis."
Conclusion: Diabetes mellitus is significantly linked to heightened severity, persistence, and recurrence of tinea corporis. The prevalence of Trichophyton species and the considerable influence of glycaemic state underscore the necessity for comprehensive dermatological and metabolic assessment. Timely detection and effective glycaemic management may enhance treatment results and diminish illness recurrence in affected patients.

Keywords: *Tinea corporis; Dermatophytosis; Diabetes mellitus; Clinico-mycological profiling; Trichophyton mentagrophytes complex; Glycemic dysregulation; Recurrent dermatophytic infection; Chronic superficial mycosis; Fungal epidemiology; Host–pathogen interactions.*

INTRODUCTION

Dermatophytosis is one of the most common superficial fungal infections globally and poses a considerable public health concern, especially in tropical and subtropical areas. Tinea corporis is a commonly observed clinical symptom, distinguished by annular erythematous lesions including scaling and center clearing on the glabrous skin. The prevalence of dermatophytosis has significantly escalated over the last decade, particularly in developing nations like India, where elevated temperatures and humidity, overcrowding, inadequate hygiene, and the prevalent misuse of topical corticosteroid formulations have facilitated its emergence as a chronic and recurrent infection [1,2].

Recent epidemiological research have underscored a shifting clinico-mycological spectrum of dermatophytosis in India, characterised by heightened chronicity, recurrence, widespread body surface involvement, and treatment failure [3,4]. Simultaneously, changes in the distribution of causative dermatophyte species have been observed, characterised by a predominance of Trichophyton species and the establishment of antifungal-resistant strains, which present further obstacles to disease management [5,6]. These evolving patterns require regular assessment of the clinical manifestations and mycological attributes of tinea corporis across various geographical areas.

Diabetes mellitus is a chronic metabolic condition marked by sustained hyperglycemia due to deficiencies in insulin secretion, insulin action, or both. The worldwide incidence of diabetes is increasing, with India representing one of the largest diabetic populations globally. Chronic hyperglycaemia negatively impacts both innate and adaptive immunological responses, resulting in diminished neutrophil function, decreased phagocytic activity, modified cytokine production, and weakened epidermal barrier integrity [8]. As a result, persons with diabetes exhibit increased vulnerability to many infectious illnesses, encompassing both superficial and invasive fungal infections [9].

Numerous investigations have shown an increased incidence of dermatophytosis in people with diabetes mellitus, indicating that inadequate glycaemic management may affect the severity, persistence, recurrence, and treatment efficacy of fungal infections [10,11]. Hyperglycemia fosters an environment conducive to fungal development by enhancing tissue glucose availability and compromising host defence mechanisms. Furthermore, microvascular dysfunction and impaired wound healing linked to diabetes may lead to chronic infections and recurrent relapses [12]. Global research has indicated a substantial correlation between diabetes and widespread dermatophytosis, while Indian studies have also recognised diabetes as a critical risk factor for chronic and recurrent tinea infections [13,14].

Eastern India is characterised by climatic conditions that favour dermatophyte proliferation; however, extensive data on the clinico-mycological profile of tinea corporis and its association with diabetes mellitus is scarce. The majority of existing studies have concentrated on the epidemiology of dermatophytosis or the distribution of fungal species, with relatively fewer analyses exploring the impact of diabetes on disease manifestation and mycological attributes [3,5,14]. Comprehending this correlation is crucial for formulating effective diagnostic, therapeutic, and preventive strategies, especially in areas with a significant prevalence of both dermatophytosis and diabetes.

This study aimed to assess the clinico-mycological profile of tinea corporis patients at a tertiary care center in Eastern India and to examine the relationship between diabetes mellitus and various clinical and mycological parameters of the condition. The study aims to identify patterns of dermatophyte infection, evaluate disease characteristics in diabetic and non-diabetic persons, and produce region-specific information to enhance the management of dermatophytosis in clinical practice.

MATERIALS & METHODS:

This hospital-based, comparative cross-sectional study was conducted in the Department of Dermatology in collaboration with the Department of Biochemistry T Hi-Tech Medical College & Hospital, Raurkela, Odisha. A total of 40 consecutive adult patients (≥18 years) with clinically diagnosed psoriasis were enrolled and categorized into two groups: erythrodermic psoriasis (EP; n = 20) and chronic plaque psoriasis (CPP; n = 20). The diagnosis of psoriasis subtype was established by experienced dermatologists based on established clinical criteria, with histopathological confirmation when clinically indicated.

Inclusion and Exclusion Criteria

Patients aged ≥18 years with clinically suspected chronic plaque psoriasis or erythroderma were considered for enrollment. Patients with chronic plaque psoriasis and erythroderma were included only after histopathological examination of skin biopsy specimens confirmed psoriasis or identified the underlying dermatological disorder responsible for erythroderma. Histopathological assessment was performed in conjunction with the clinical findings to ensure diagnostic accuracy. Only cases with a definitive histopathological diagnosis of psoriasis or erythroderma were included to ensure diagnostic accuracy, particularly because erythroderma has overlapping clinical features with several dermatological disorders and may be difficult to diagnose on clinical grounds alone. Patients with inconclusive or unavailable histopathological findings, other causes of erythroderma, mixed dermatological conditions, active systemic infections, malignancies, autoimmune disorders, pregnancy, lactation, or incomplete clinical records were excluded. No hematological investigations were used as diagnostic criteria for participant selection.

Clinical assessment: Demographic characteristics, including age and sex, together with disease duration, were recorded for all participants. Disease severity was evaluated using the Psoriasis Area and Severity Index (PASI), while the extent of skin involvement was determined by estimating the percentage of body surface area (BSA) affected. Systemic inflammatory status was assessed by measuring erythrocyte sedimentation rate (ESR) and serum C-reactive protein (CRP) using standard laboratory methods.

Blood sample collection: After an overnight fast of 10–12 hours, approximately 10 mL of venous blood was collected from each participant under aseptic conditions. Blood samples were divided into EDTA and plain tubes. Plasma and serum were separated by centrifugation at 3000 rpm for 10 minutes at 4°C and stored at −80°C until biochemical analyses were performed. All assays were completed within the recommended storage period to prevent degradation of analytes.

Estimation of oxidative stress biomarkers: Lipid peroxidation was assessed by measuring malondialdehyde (MDA) using the thiobarbituric acid reactive substances (TBARS) assay. Lipid hydroperoxide (LOOH) concentrations were determined by the ferrous oxidation–xylenol orange (FOX) method. Advanced oxidation protein products (AOPP) were quantified spectrophotometrically using chloramine-T as the calibration standard. Protein carbonyl content was determined by reaction with 2,4-dinitrophenylhydrazine (DNPH), and the absorbance of the resulting hydrazone derivatives was measured spectrophotometrically. Nitric oxide production was estimated indirectly by measuring total nitrate and nitrite (NOx) concentrations using the Griess reaction following enzymatic reduction of nitrate to nitrite. Oxidative DNA damage was evaluated by determining serum 8-hydroxy-2′-deoxyguanosine (8-OHdG) concentrations using a commercially available enzyme-linked immunosorbent assay (ELISA) kit according to the manufacturer's instructions.

Assessment of antioxidant status: Enzymatic antioxidant defense was evaluated by measuring superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPx) activities using standardized spectrophotometric methods. Reduced glutathione (GSH) concentration was determined using Ellman's reagent (DTNB). Total antioxidant capacity (TAC) was measured using the Trolox equivalent antioxidant capacity assay. Serum vitamin C concentrations were estimated by a colorimetric method based on reduction of ferric ions, whereas vitamin E levels were quantified using a spectrophotometric method following extraction into an organic solvent. All biochemical analyses were performed in duplicate, and internal quality control procedures were implemented throughout the study.

Ethical Considerations

The Institutional Ethics Committee evaluated and approved the study protocol prior to the initiation of the trial. Informed written consent was acquired from all subjects before enrolment. The confidentiality of patient information was upheld during the study, and all methods adhered to the ethical principles established in the Declaration of Helsinki.

Statistical analysis:

Data were input into Microsoft Excel and analysed with Statistical Package for the Social Sciences (SPSS) software version 26.0. Continuous variables were represented as mean ± standard deviation (SD), whereas categorical variables were displayed as frequencies and percentages. Comparisons between diabetes and non-diabetic cohorts were conducted utilising the independent Student's t-test or Mann–Whitney U test for continuous variables, contingent upon data distribution. Categorical variables were assessed with the Chi-square test or Fisher's exact test as deemed appropriate. A multivariable logistic regression analysis was conducted to determine independent variables of chronic or recurring tinea corporis. Odds ratios (ORs) accompanied with 95% confidence intervals (CIs) were computed. A two-tailed p-value of less than 0.05 was deemed statistically significant.

RESULTS:

Figure 1: **Demographic and Clinical Characteristics of Study Participants**

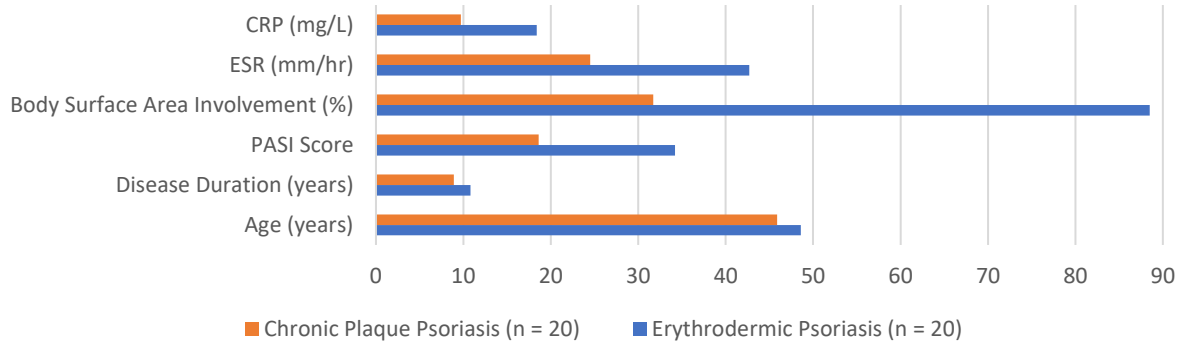


Figure 1 illustrates the demographic and clinical attributes of patients with erythrodermic psoriasis and chronic plaque psoriasis. The average age of participants in the erythrodermic psoriasis group was 48.6 ± 12.4 years, whereas in the chronic plaque psoriasis group it was 45.9 ± 11.7 years, with no statistically significant difference observed between the groups ($p = 0.312$). The distribution of males and females was analogous, signifying demographic homogeneity between the research cohorts ($p = 0.633$). The average illness duration was somewhat greater in individuals with erythrodermic psoriasis (10.8 ± 5.7 years) than in those with chronic plaque psoriasis (8.9 ± 4.8 years); yet, this difference did not reach statistical significance ($p = 0.108$). The indices of disease severity varied significantly between the two groups. Patients with erythrodermic psoriasis demonstrated markedly elevated PASI scores (34.2 ± 7.5) compared to those with chronic plaque psoriasis (18.6 ± 5.1) ($p < 0.001$). The proportion of body surface area impacted was significantly higher in the erythrodermic psoriasis cohort (88.5 ± 9.2%) than in the chronic plaque psoriasis cohort (31.7 ± 10.6%) ($p < 0.001$). Indicators of systemic inflammation were markedly increased in erythrodermic psoriasis. The average erythrocyte sedimentation rate was 42.7 ± 11.8 mm/hr in individuals with erythrodermic psoriasis, compared to 24.5 ± 8.3 mm/hr in patients with chronic plaque psoriasis ($p < 0.001$). Serum C-reactive protein levels were markedly elevated in the erythrodermic psoriasis cohort (18.4 ± 6.2 mg/L) compared to the chronic plaque psoriasis cohort (9.7 ± 4.1 mg/L) ($p < 0.001$). The findings suggest an increased inflammatory burden and heightened disease severity in people with erythrodermic psoriasis.

Table 1 delineates the oxidative stress profiles of patients with erythrodermic psoriasis and chronic plaque psoriasis. Markedly increased levels of all evaluated oxidative stress biomarkers were noted in the erythrodermic psoriasis cohort. Malondialdehyde concentrations, an indicator of lipid peroxidation, were significantly elevated in erythrodermic psoriasis patients (8.92 ± 1.64 nmol/mL) compared to chronic plaque psoriasis patients (5.87 ± 1.28 nmol/mL) ($p < 0.001$). Lipid hydroperoxide concentrations were considerably elevated in the erythrodermic psoriasis group (12.48 ± 2.73 µmol/L) compared to the chronic plaque psoriasis group (8.96 ± 2.15 µmol/L) ($p < 0.001$). Advanced oxidation protein products, signifying oxidative protein damage, were significantly higher in erythrodermic psoriasis patients (118.6 ± 24.7 µmol/L) compared to chronic plaque psoriasis patients (82.3 ± 19.5 µmol/L) ($p < 0.001$). The protein carbonyl content exhibited a same trend, showing significantly elevated levels in erythrodermic psoriasis (3.91 ± 0.86 nmol/mg protein) compared to chronic plaque psoriasis (2.57 ± 0.63 nmol/mg protein) ($p < 0.001$). Nitric oxide metabolite concentrations were markedly elevated in erythrodermic psoriasis patients (48.7 ± 10.5 µmol/L) relative to chronic plaque psoriasis patients (35.4 ± 8.1 µmol/L) ($p < 0.001$). Additionally, blood levels of 8-hydroxy-2'-deoxyguanosine, indicative of oxidative DNA damage, were significantly higher in erythrodermic psoriasis (16.8 ± 3.9 ng/mL) than in chronic plaque psoriasis (11.4 ± 2.8 ng/mL) ($p < 0.001$). The data collectively indicate a significantly increased oxidative stress burden in erythrodermic psoriasis compared to chronic plaque psoriasis, implying a greater role of oxidative damage in the pathogenesis of the erythrodermic variant of the illness.

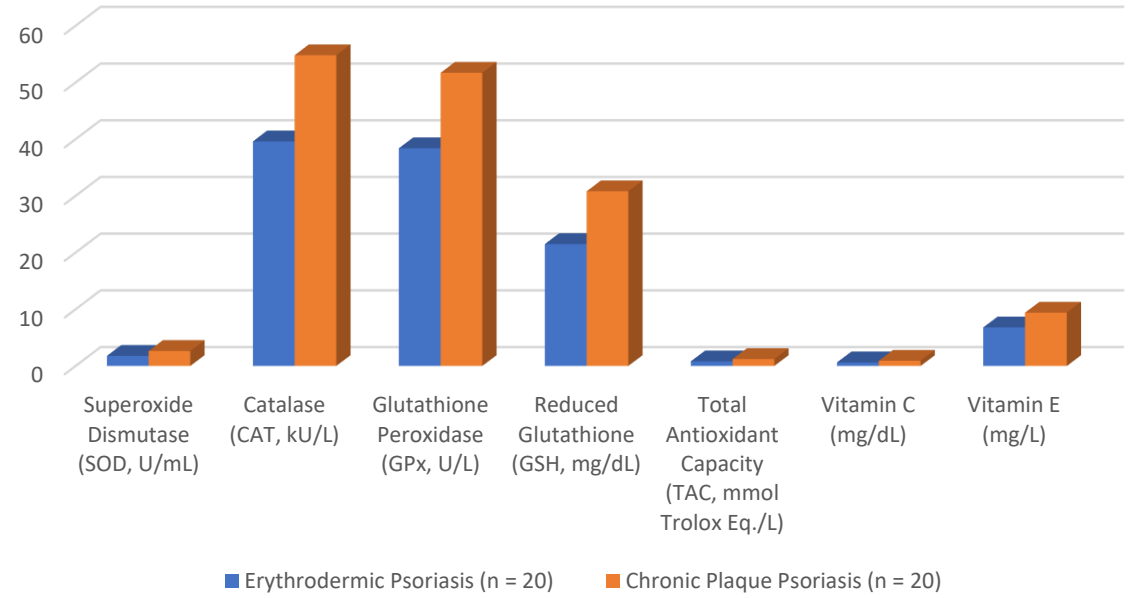
Table 2. Comparison of Oxidative Stress Markers Between Erythrodermic and Chronic Plaque Psoriasis Patients

Oxidative Stress Marker	Erythrodermic Psoriasis (n = 20)	Chronic Plaque Psoriasis (n = 20)	p-value
Malondialdehyde (MDA, nmol/mL)	8.92 ± 1.64	5.87 ± 1.28	<0.001*
Lipid Hydroperoxides (LOOH, µmol/L)	12.48 ± 2.73	8.96 ± 2.15	<0.001*
Advanced Oxidation Protein Products (AOPP, µmol/L)	118.6 ± 24.7	82.3 ± 19.5	<0.001*
Protein Carbonyl Content (nmol/mg protein)	3.91 ± 0.86	2.57 ± 0.63	<0.001*
Nitric Oxide Metabolites (NOx, µmol/L)	48.7 ± 10.5	35.4 ± 8.1	<0.001*
8-Hydroxy-2'-deoxyguanosine (8-OHdG, ng/mL)	16.8 ± 3.9	11.4 ± 2.8	<0.001*

Figure 2 delineates the antioxidant status of individuals with erythrodermic psoriasis and chronic plaque psoriasis. A notable decline in both enzymatic and non-enzymatic antioxidant defences was noted in patients with erythrodermic psoriasis. The activity of superoxide dismutase was markedly reduced in the erythrodermic psoriasis cohort (1.78 ± 0.42 U/mL) compared to the chronic plaque psoriasis cohort (2.64 ± 0.51 U/mL) ($p < 0.001$). Catalase activity was significantly decreased in erythrodermic psoriasis patients (39.6 ± 8.7 kU/L) compared to chronic plaque psoriasis patients (54.8 ± 10.2 kU/L) ($p < 0.001$). The activity of glutathione peroxidase exhibited a notable reduction in erythrodermic psoriasis (38.4 ± 7.9 U/L) compared to chronic plaque psoriasis (51.7 ± 8.6 U/L) ($p < 0.001$). Erythrodermic psoriasis patients had considerably lower quantities of reduced glutathione (21.5 ± 5.1 mg/dL) compared to chronic plaque psoriasis patients (30.8 ± 6.2 mg/dL) ($p < 0.001$). The total antioxidant capacity was significantly reduced in the erythrodermic psoriasis group (0.82 ± 0.18 mmol Trolox equivalent/L) compared to the chronic plaque psoriasis group (1.21 ± 0.24 mmol Trolox equivalent/L) ($p < 0.001$). In erythrodermic psoriasis patients, blood vitamin C levels were substantially lower (0.63 ± 0.15 mg/dL) than in chronic plaque psoriasis patients (0.92 ± 0.21 mg/dL) ($p < 0.001$). Vitamin E levels were significantly lower in erythrodermic psoriasis (6.8 ± 1.7 mg/L) compared to chronic plaque psoriasis (9.4 ± 2.1 mg/L) ($p < 0.001$). The observed

reduction in antioxidant defences in erythrodermic psoriasis, along with increased oxidative stress markers, indicates a significant redox imbalance in this severe clinical subtype of psoriasis.

Figure 2: **Comparison of Antioxidant Status Between Erythrodermic and Chronic Plaque Psoriasis Patients**



DISCUSSION:

This study examined oxidative stress indicators and antioxidant levels in patients with erythrodermic psoriasis and chronic plaque psoriasis, revealing a significant redox imbalance in the erythrodermic variant of the condition. Patients with erythrodermic psoriasis demonstrated markedly elevated levels of lipid, protein, and DNA oxidation products, such as malondialdehyde (MDA), lipid hydroperoxides (LOOH), advanced oxidation protein products (AOPP), protein carbonyls, nitric oxide metabolites, and 8-hydroxy-2′-deoxyguanosine (8-OHdG), in comparison to patients with chronic plaque psoriasis. Erythrodermic psoriasis was concurrently characterised by significant depletion of both enzymatic and non-enzymatic antioxidant defences, including superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPx), reduced glutathione (GSH), total antioxidant capacity (TAC), vitamin C, and vitamin E. The data indicate that oxidative stress may significantly influence disease severity and systemic inflammatory load in psoriasis.

Psoriasis is acknowledged as a chronic immune-mediated inflammatory condition characterised by the excessive generation of reactive oxygen species (ROS), which leads to keratinocyte hyperproliferation, immunological dysregulation, angiogenesis, and tissue damage [1,2]. A thorough systematic evaluation published in 2022 found that oxidative stress indicators are constantly heightened in psoriasis and related with disease severity and duration [1]. A recent study similarly indicated a considerable oxidative imbalance in psoriasis vulgaris, reinforcing the notion that oxidative stress plays a role in disease progression [3].

The present study's most notable finding was the markedly increased MDA levels in erythrodermic psoriasis. MDA is a recognised marker of lipid peroxidation and membrane injury. Comparable findings have been documented by Indian researchers, who revealed markedly elevated MDA levels in individuals with psoriasis relative to healthy controls [4,5]. Research has indicated heightened levels of lipid peroxidation products in severe psoriasis, proposing that augmented reactive oxygen species (ROS) production fosters chronic inflammation and cardiovascular comorbidities [6–8]. The significantly elevated MDA levels reported in erythrodermic psoriasis in this study suggest that substantial skin involvement may exacerbate lipid oxidative damage.

The current study also demonstrated markedly elevated levels of AOPP and protein carbonyl content in erythrodermic psoriasis. Protein oxidation products are regarded as dependable indicators of chronic oxidative damage and inflammation. A study indicated markedly increased AOPP levels in psoriasis patients and proposed that oxidative protein modification plays a role in disease development [6]. Comparable results have been seen in European research assessing oxidative protein damage in severe inflammatory dermatoses [8,9]. The elevated protein oxidation noted in erythrodermic psoriasis may indicate increased systemic inflammatory activity and extended oxidative damage.

The erythrodermic group had significantly higher levels of nitric oxide metabolites and 8-OHdG. Excessive generation of nitric oxide has been associated with psoriasis via the activation of inducible nitric oxide synthase, leading to increased inflammatory signalling and keratinocyte proliferation [10]. Elevated 8-OHdG levels signify oxidative DNA damage and correlate with disease severity and chronic systemic inflammation [11]. The findings indicate that oxidative damage in erythrodermic psoriasis affects not only lipids and proteins but also nucleic acids, potentially leading to disease persistence and consequences.

A significant observation was the considerable decline in antioxidant defences in people with erythrodermic psoriasis. SOD, CAT, and GPx form the principal enzymatic antioxidant system that neutralises superoxide radicals and hydrogen peroxide. Numerous Indian and worldwide investigations have documented diminished activity of these enzymes [4,5,12,13]. The current findings align with those that indicated a depletion of glutathione-related antioxidant systems in psoriasis patients, particularly in individuals with severe illness [14]. Excessive consumption during prolonged oxidative stress and inflammatory reactions may diminish antioxidant enzyme function.

Non-enzymatic antioxidants, such as GSH, vitamin C, vitamin E, and TAC, were markedly diminished in erythrodermic psoriasis. Comparable reductions have been documented in research undertaken in India, Iran, Egypt, and Eastern Europe [5,6,12,15]. Vitamin C and vitamin E are essential in neutralising free radicals and safeguarding cellular membranes from oxidative damage. Their reduction in erythrodermic psoriasis may indicate heightened consumption due to elevated reactive oxygen species production. The diminished TAC further signifies the depletion of total antioxidant reserves, underscoring a significant redox imbalance.

A striking discovery was the markedly elevated inflammatory markers, such as ESR and CRP, in erythrodermic psoriasis. Oxidative stress and inflammation are intricately linked processes. Reactive oxygen species (ROS) activate the NF-κB, MAPK, and JAK-STAT signalling pathways, leading to an elevated synthesis of pro-inflammatory cytokines including TNF-α, IL-17, and IL-23 [1,2]. The increased oxidative stress noted in erythrodermic psoriasis may directly exacerbate the pronounced inflammatory condition typical of this severe disease type.

The clinical ramifications of these discoveries are substantial. Growing data indicates that oxidative stress plays a role in both the dermatological symptoms and systemic consequences of psoriasis, such as metabolic syndrome, endothelial dysfunction, and cardiovascular disease [1,7,8]. Thus, evaluating oxidative biomarkers may yield supplementary insights on disease severity and therapy efficacy. Moreover, antioxidant supplementation alongside conventional therapy has demonstrated encouraging results in enhancing oxidative balance and clinical outcomes in individuals with psoriasis [13]. Constraints of the Research: Numerous constraints must be recognised. The cross-sectional approach prevents the determination of causal links between oxidative stress and disease progression. Secondly, the sample size was rather small and sourced from a single location, thereby constraining the generalisability of the results. Third, the absence of healthy control participants precluded direct comparison with normative oxidative and antioxidant profiles. The assessment did not thoroughly examine dietary patterns, nutritional antioxidant consumption, smoking status, and lifestyle factors that could affect oxidative stress. Cytokine profiling and molecular studies of oxidative stress-related signalling pathways were ultimately not conducted, which may have yielded further mechanistic insights.

CONCLUSION:

This study shows that erythrodermic psoriasis is linked to much higher oxidative stress and more antioxidant depletion compared to chronic plaque psoriasis. In erythrodermic psoriasis, heightened levels of lipid, protein, and DNA oxidation products, coupled with diminished enzymatic and non-enzymatic antioxidant defences, signify a significant systemic redox imbalance. These data substantiate the concept that oxidative stress plays a crucial role in disease severity and inflammatory activity. Future multicentric longitudinal studies that include molecular biomarkers and pharmacological interventions aimed at oxidative pathways are necessary to elucidate the function of redox regulation in the treatment of severe psoriasis.

Conflict of interest:

None among the present study authors.

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