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Original Research Article

# Assessment of Biochemical Oxidative Stress Markers in Patients with Psoriasis

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**Abstract:**

**Background:** Psoriasis is a chronic immune-mediated inflammatory disorder increasingly recognized as a systemic disease associated with oxidative stress and metabolic abnormalities.

**Aim:** To assess biochemical oxidative stress markers in patients with psoriasis and evaluate their association with disease severity.

**Methodology:** This hospital-based case–control study included 90 participants (45 psoriasis patients and 45 age- and sex-matched healthy controls). Fasting blood samples were analyzed for routine biochemical parameters and oxidative stress markers, including malondialdehyde (MDA), advanced oxidation protein products (AOPP), and catalase. Disease severity was assessed using the Psoriasis Area and Severity Index (PASI). Statistical analysis was performed using SPSS version 27.0.

**Results:** Psoriasis patients showed significantly higher levels of MDA, AOPP, CRP, fasting glucose, total cholesterol, triglycerides, and LDL, with lower HDL and catalase levels ($p<0.05$). MDA and AOPP demonstrated positive correlations with PASI score, while catalase showed a negative correlation. Logistic regression identified elevated MDA, AOPP, low catalase, and high CRP as significant predictors of psoriasis.

**Conclusion:** Psoriasis is associated with increased oxidative stress, impaired antioxidant defense, and systemic inflammation, which correlate with disease severity.

**Keywords:** Psoriasis, Oxidative stress, Malondialdehyde, AOPP, Catalase, PASI.



## Introduction

Psoriasis is a chronic disease that results from immune system dysregulation and causes skin symptoms and multiple organ system effects which make doctors consider it a systemic condition that leads to various additional medical issues [1]. Psoriasis used to be classified as a skin disease that caused red scaly patches but now researchers view it as an advanced inflammatory condition that creates severe metabolic and immune system effects. The global prevalence of psoriasis shows different rates between geographic areas and different population groups which results in adult prevalence rates that range from 0.51% to 11.43% [2]. The different outcomes show how genetic factors and environmental elements and personal habits affect psoriasis which has become a serious public health issue that exists throughout the world.

Even though etiopathogenesis of psoriasis has not been fully clarified, the current evidence indicates that the central role belongs to dysregulation of immune system. The incorrect regulation and aberrant activation of the T lymphocytes, either by the cells of the innate immune system (macrophages, neutrophils, keratinocytes, and dendritic cells), or, perhaps, by an unknown autoantigen, are taken to be the key initiating events [3]. Immune dysregulation results in an auto-inflammatory cascade that is marked by exaggeration of proinflammatory mediators and prolonged immune pathway stimulation. As a result, there is an overgrowth and unreasonable differentiation of the keratinocytes leading to the development of the typical psoriatic plaques.

Besides immune dysfunction, oxidative stress has also turned out to be a key factor in pathogenesis of psoriasis. The same as the other chronic systemic diseases including diabetes mellitus, hypertension, obesity, and cardiovascular disease, oxidative stress and chronic inflammation are believed to have

central roles in the pathogenesis and evolution of psoriasis [4]. Oxidative stress is characterized by the lack of balance between the generation of pro-oxidants (reactive oxygen species (ROS) and reactive nitrogen species (RNS)) and the ability of the antioxidant defense system to counteract them. Excessive ROS and RNS are caused by the redox imbalance in the favor of pro-oxidants, which creates oxidative changes in the fundamental biomolecules, such as lipids, proteins, and DNA, to cause structural and functional damage of cells.

The inflammatory environment of psoriasis leads to increased oxidative stress in the condition. The increased secretion of proinflammatory cytokines which includes tumor necrosis factor alpha (TNF-α) together with interleukin (IL)-1β and IL-6 and IL-17 and IL-22 and IL-12/23 leads to excessive keratinocyte growth and incorrect skin cell development. These cytokines cause two effects which persist inflammation and increase production of ROS and RNS that results in more oxidative harm. The preexisting oxidative damage becomes worse because chronic inflammation inhibits the body's ability to defend itself through antioxidant systems. The interaction between oxidative stress and inflammation creates a destructive loop which results in greater intensity of psoriatic skin lesions.

Psoriasis affects the body through two different types of effects which include cutaneous symptoms and systemic metabolic disturbances that particularly impact lipid metabolism. Proinflammatory cytokines have been shown to influence cholesterol metabolism and lipid homeostasis. Cholesterol metabolites affect keratinocyte function through direct interactions which also impact inflammatory responses and immune cell activity, thus creating additional pathways which lead to disease development. The skin disorders of psoriatic patients develop from lipid metabolism disturbances which also cause the systemic complications that occur in these patients.

Psoriasis has evolved into recognition as a systemic inflammatory disease which results in changes to cholesterol metabolism biomolecules that affect multiple metabolic disorders. The metabolic disorders include obesity and dyslipidemia and hypertension and insulin resistance and non-alcoholic fatty liver disease (NAFLD) and diabetes mellitus [5]. The coexistence of these metabolic conditions significantly increases the overall morbidity and mortality among patients with psoriasis. The development of psoriasis leads to chronic low-grade systemic inflammation which results in oxidative stress that connects with these cardiometabolic disorders through shared basic mechanisms.

Psoriasis research now shows that patients with this condition face an increased risk of developing kidney complications. The research demonstrates that individuals with psoriasis face higher chances of developing chronic kidney disease (CKD) and end-stage renal disease [6]. The presence of proinflammatory cytokines results in harmful effects which disrupt kidney blood flow and cause sodium retention and the development of high blood pressure and ongoing kidney damage. The research results demonstrate that psoriasis exists as a systemic inflammatory disorder which affects multiple body systems instead of only remaining a skin disease.

Research about oxidative stress and its link to psoriasis has increased in recent years yet studies that measure oxidative stress through its by-products and antioxidant levels in psoriatic patients have produced conflicting results which sometimes show opposing outcomes together with their results [7]. The differences between studies arise from their research methods which include various factors such as study design, sample size, patient characteristics, disease severity, and laboratory procedures. Some studies report elevated levels of oxidative stress markers and reduced antioxidant capacity in psoriasis, while others fail to demonstrate significant differences compared to healthy controls [8]. The existing research results show opposing outcomes which establish the requirement for additional detailed studies that will determine the precise role of oxidative stress together with related biochemical markers in the development of psoriasis.

The assessment of oxidative stress biochemical markers gives researchers critical information about patient redox status which helps them investigate the biological processes that drive disease development. The detection of particular markers which indicate lipid peroxidation and protein oxidation and DNA damage together with antioxidant defense mechanisms enables researchers to determine the degree of oxidative damage which affects inflammatory processes and metabolic disorders. The evaluation of these markers in connection to disease severity provides both prognostic value and therapeutic implications.

The systemic characteristics of psoriasis together with the intricate relationships between its multiple components require researchers to assess oxidative stress markers through complete testing. The research findings will establish disease pathways and enable scientists to discover new biomarkers which can be used to develop innovative treatment methods.

The current research intends to study oxidative stress and inflammatory and metabolic markers in psoriasis patients while comparing them to healthy individuals to investigate their association with disease severity. The clarification of these connections will enhance our understanding of psoriasis disease mechanisms which will help develop more precise treatment methods".

## Methodology

**Study Design:** This hospital-based case–control study was conducted to assess biochemical oxidative stress markers in patients with psoriasis and to compare them with age- and sex-matched healthy controls. The study was carried out after obtaining approval from the Institutional Ethics Committee, and written informed consent was obtained from all participants prior to enrollment. The study adhered to the ethical principles of the Declaration of Helsinki.

**Study Area:** The study was conducted in the Department of Skin & VD and Biochemistry, Sri Krishna Medical College and Hospital, Muzaffarpur, Bihar, India.

**Study Duration:** The total duration of the study was six months from March 2025 to August 2025.

**Study Participants:** A total of 90 participants were included in the study, comprising clinically diagnosed cases of psoriasis and healthy controls.

### Inclusion Criteria

- Patients aged ≥18 years diagnosed clinically with psoriasis.
- Patients willing to provide written informed consent.
- Patients not on systemic antioxidant supplementation.
- Age- and sex-matched apparently healthy individuals for the control group.

### Exclusion Criteria

- Patients with known malignancies.
- History of cardiovascular diseases or stroke.
- Presence of chronic kidney or liver disease.
- Patients with autoimmune or inflammatory disorders other than psoriasis.
- Individuals with psychiatric illness.
- Pregnant or lactating women.
- Participants using antioxidant supplements or systemic steroids in the past three months.

**Sample Size:** The total sample size of the study was 90 participants.

**Procedure:** After enrollment, detailed demographic and clinical information was recorded using a structured questionnaire, including age, gender, duration of disease, medication history, lifestyle habits, and comorbidities. Anthropometric measurements such as height and weight were measured using standardized techniques, and body mass index (BMI) was calculated. The severity of psoriasis was assessed using the Psoriasis Area and Severity Index (PASI), and the impact on quality of life was evaluated using the Dermatology Life Quality Index (DLQI).

Venous blood samples were collected from all participants in the morning after an overnight fasting period of at least 8 hours. Approximately 5 ml of venous blood was drawn under aseptic precautions and transferred into serum separator and clot activator tubes. The samples were allowed to clot for 30 minutes at room temperature and were then centrifuged at 3000 rpm for 10 minutes to separate serum. The separated serum was stored at −20°C until biochemical analysis.

Routine biochemical parameters such as fasting blood glucose, total cholesterol, triglycerides, high-density lipoprotein (HDL), low-density lipoprotein (LDL), urea, uric acid, creatinine, alanine aminotransferase (ALT), aspartate aminotransferase (AST), and C-reactive protein (CRP) were measured using an automated chemistry analyzer in the central laboratory of the institution.

Oxidative stress markers were estimated spectrophotometrically. Malondialdehyde (MDA), an indicator of lipid peroxidation, was measured using the thiobarbituric acid reactive substances (TBARS) method. Advanced oxidation protein products (AOPP) were determined based on the reaction with potassium iodide in an acidic medium. Catalase (CAT) activity was assessed by measuring the decomposition rate of hydrogen peroxide. All assays were performed according to standard laboratory protocols, and quality control measures were maintained throughout the analysis.

**Statistical Analysis:** Data were entered into Microsoft Excel and analyzed using Statistical Package for the Social Sciences (SPSS) version 27.0. Continuous variables were expressed as mean ± standard deviation (SD) or median (interquartile range) depending on data distribution, while categorical variables were presented as frequencies and percentages. Normality of data was assessed using the Shapiro–Wilk test. Independent Student's t-test was used to compare normally distributed continuous variables between cases and controls, whereas the Mann–Whitney U test was applied for non-normally distributed data. The Chi-square test was used to analyze categorical variables. Correlation between oxidative stress markers and disease severity (PASI score) was assessed using Spearman's correlation coefficient. A p-value of $<0.05$ was considered statistically significant".

## Result

Table 1 shows the demographic and clinical characteristics of the study participants in both the psoriasis (n=45) and control (n=45) groups. The mean age of patients with psoriasis was 43.2 ± 10.8 years, which was comparable to the control group (42.6 ± 11.1 years), with no statistically significant difference (p=0.81). The gender distribution was also similar between the two groups, with males constituting 60% in the psoriasis group and 57.8% in the control group (p=0.83), indicating proper matching. The mean BMI was 24.7 ± 3.2 kg/m² in psoriasis patients

and 24.3 ± 3.0 kg/m² in controls, again showing no significant difference (p=0.54). Among psoriasis patients, the mean duration of disease was 6.4 ± 3.5 years. The mean PASI score was 12.8 ± 5.2, suggesting moderate disease severity, while the mean DLQI score was 13.9 ± 5.8, indicating a considerable impact on quality of life. Overall, both groups were comparable in baseline characteristics, reducing potential confounding factors.

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

| Variable | Psoriasis (n=45) | Controls (n=45) | p-value |
|---|---|---|---|
| Age (years) (Mean ± SD) | 43.2 ± 10.8 | 42.6 ± 11.1 | 0.81 |
| Male, n (%) | 27 (60%) | 26 (57.8%) | 0.83 |
| Female, n (%) | 18 (40%) | 19 (42.2%) | |
| BMI (kg/m²) (Mean ± SD) | 24.7 ± 3.2 | 24.3 ± 3.0 | 0.54 |
| Duration of disease (years) | 6.4 ± 3.5 | — | — |
| PASI score (Mean ± SD) | 12.8 ± 5.2 | — | — |
| DLQI score (Mean ± SD) | 13.9 ± 5.8 | — | — |

Table 2 shows the comparison of routine biochemical parameters between psoriasis patients (n=45) and healthy controls (n=45). The mean fasting glucose level was significantly higher in psoriasis patients (102.8 ± 16.4 mg/dL) compared to controls (95.6 ± 14.2 mg/dL) with a p-value of 0.028. Similarly, total cholesterol (206.3 ± 32.8 vs 185.9 ± 28.7 mg/dL; p=0.003) and triglycerides (170.4 ± 39.6 vs 145.2 ± 35.1 mg/dL; p=0.002) were significantly elevated in the psoriasis group. LDL levels were also higher among cases (129.7 ± 26.9 mg/dL) compared to controls (111.6 ± 23.8 mg/dL) with strong statistical significance (p=0.001), whereas HDL levels were significantly lower in psoriasis patients (39.4 ± 6.8 mg/dL) than in controls (44.1 ± 6.2 mg/dL; p=0.001). Additionally, CRP levels were markedly increased in the psoriasis group (6.5 ± 2.8 mg/L) compared to controls (3.1 ± 1.6 mg/L), showing highly significant difference (p<0.001). These findings indicate a significant alteration in glycemic status, lipid profile, and inflammatory markers among psoriasis patients.

**Table 2: Comparison of Routine Biochemical Parameters**

| Parameter | Psoriasis (n=45) Mean ± SD | Controls (n=45) Mean ± SD | p-value |
|---|---|---|---|
| Fasting Glucose (mg/dL) | 102.8 ± 16.4 | 95.6 ± 14.2 | 0.028 |
| Total Cholesterol (mg/dL) | 206.3 ± 32.8 | 185.9 ± 28.7 | 0.003 |
| Triglycerides (mg/dL) | 170.4 ± 39.6 | 145.2 ± 35.1 | 0.002 |
| HDL (mg/dL) | 39.4 ± 6.8 | 44.1 ± 6.2 | 0.001 |
| LDL (mg/dL) | 129.7 ± 26.9 | 111.6 ± 23.8 | 0.001 |
| CRP (mg/L) | 6.5 ± 2.8 | 3.1 ± 1.6 | <0.001 |

Table 3 shows the comparison of oxidative stress markers between psoriasis patients and healthy controls. The mean serum MDA level was significantly higher in psoriasis patients (5.74 ± 1.21 nmol/mL) compared to controls (3.58 ± 0.92 nmol/mL), with a highly significant p-value (<0.001), indicating increased lipid peroxidation in the disease group. Similarly, AOPP levels were markedly elevated in psoriasis patients (121.8 ± 26.4 µmol/L) as compared to controls (85.6 ± 18.9 µmol/L), also showing strong statistical significance (p < 0.001), reflecting enhanced protein oxidation. In contrast, the antioxidant enzyme catalase was significantly reduced in psoriasis patients (40.2 ± 8.7 U/mL) compared to controls (55.4 ± 10.6 U/mL), with p < 0.001, suggesting impaired antioxidant defense. Overall, the findings demonstrate significantly increased oxidative stress and decreased antioxidant activity in patients with psoriasis compared to healthy individuals.

**Table 3: Comparison of Oxidative Stress Markers**

| Oxidative Marker | Psoriasis (n=45) Mean ± SD | Controls (n=45) Mean ± SD | p-value |
|---|---|---|---|
| MDA (nmol/mL) | 5.74 ± 1.21 | 3.58 ± 0.92 | <0.001 |
| AOPP (µmol/L) | 121.8 ± 26.4 | 85.6 ± 18.9 | <0.001 |
| Catalase (U/mL) | 40.2 ± 8.7 | 55.4 ± 10.6 | <0.001 |

Table 4 shows the correlation of oxidative stress markers with PASI score among 45 cases of psoriasis. The findings indicate a statistically significant positive correlation between MDA and PASI score (Spearman's r = 0.58, p < 0.001), suggesting that higher lipid peroxidation is associated with increased disease severity. Similarly, AOPP demonstrated a moderate positive correlation with PASI score (r = 0.52, p = 0.001), indicating that elevated protein oxidation is linked with more severe

psoriasis. In contrast, catalase showed a significant negative correlation with PASI score ($r = -0.46$, $p = 0.002$), implying that antioxidant defense decreases as disease severity increases. Overall, these results highlight that increased oxidative stress and reduced antioxidant activity are significantly associated with higher PASI scores in psoriasis patients.

**Table 4: Correlation of Oxidative Stress Markers with PASI Score (n=45 cases)**

| Variable | Spearman's r | p-value |
|---|---|---|
| MDA vs PASI | 0.58 | <0.001 |
| AOPP vs PASI | 0.52 | 0.001 |
| Catalase vs PASI | −0.46 | 0.002 |

Table 5 presents the binary logistic regression analysis for predictors of psoriasis and demonstrates that oxidative stress and inflammatory markers were significantly associated with the disease. Elevated MDA (>4.5 nmol/mL) showed the strongest association, with an odds ratio (OR) of 3.42 (95% CI: 1.56–7.48; $p = 0.002$), indicating that individuals with higher MDA levels were approximately 3.4 times more likely to have psoriasis compared to those with normal levels. Similarly, elevated AOPP (>100 µmol/L) was significantly associated with psoriasis (OR = 2.98; 95% CI: 1.37–6.47; $p = 0.006$). Low catalase levels (<45 U/mL) also increased the odds of psoriasis by 2.76 times (95% CI: 1.25–6.08; $p = 0.012$), suggesting reduced antioxidant defense in affected patients. Additionally, high CRP (>5 mg/L) was identified as a significant predictor (OR = 2.39; 95% CI: 1.10–5.21; $p = 0.028$). Overall, all variables were statistically significant ($p < 0.05$), highlighting the important role of oxidative stress and systemic inflammation in the pathogenesis of psoriasis.

**Table 5: Binary Logistic Regression Analysis for Predictors of Psoriasis**

| Variable | Odds Ratio (OR) | 95% CI | p-value |
|---|---|---|---|
| Elevated MDA (>4.5 nmol/mL) | 3.42 | 1.56–7.48 | 0.002 |
| Elevated AOPP (>100 µmol/L) | 2.98 | 1.37–6.47 | 0.006 |
| Low Catalase (<45 U/mL) | 2.76 | 1.25–6.08 | 0.012 |
| High CRP (>5 mg/L) | 2.39 | 1.10–5.21 | 0.028 |

## Discussion

The present research results show that oxidative stress markers including malondialdehyde and advanced oxidation protein products show higher levels in psoriasis patients compared to healthy controls who exhibit decreased levels of catalase enzyme activity. The mean PASI score of 12.8 ± 5.2 in our cohort indicates moderate disease severity and the observed positive correlation between MDA and AOPP with PASI shows that oxidative stress increases with higher disease activity. The results confirm the findings of Dobric et al. (2022) [9] and Cannavò et al. (2019) [10] who established that oxidative stress functions as a primary factor in the development of psoriasis which correlates with the severity of the disease".

The study found that psoriasis patients showed higher serum MDA levels which indicated they experienced more severe lipid peroxidation damage to their cell membranes. Yildirim et al. (2003) [11] conducted a study which showed that psoriatic patients had higher serum MDA levels than control participants, which indicated they experienced increased oxidative damage. Sikar Aktürk et al. (2012) [12] discovered that both plasma and tissue MDA levels increased with higher PASI scores, which showed that lipid peroxidation linked to disease severity. Skoie et al. (2019) [13] discovered that psoriasis patients and control subjects showed no MDA level difference, which indicated that disease duration and treatment status and comorbid conditions all affected oxidative profiles. The difference arose from three factors: researchers used different sample sizes, they distributed disease severity differently, and they applied different research methods. The research demonstrates that psoriasis patients experience active lipid peroxidation because our data showed high MDA levels which correlated with PASI scores.

AOPP levels showed significant elevation in our psoriasis group which correlated with increased disease severity. The results of this study confirm the findings of Yazici et al. (2016) [14] who discovered that psoriasis patients exhibit AOPP levels which exceed normal ranges because protein oxidation constitutes a major aspect of oxidative stress that affects this condition (Yazici et al., 2016). Shakoei et al. (2021) [15] discovered that psoriasis patients had higher serum AOPP levels than control subjects, which indicates that their protein oxidative modification process was increased. The research conducted by Skoie et al. (2019) found no difference between AOPP levels among different patient groups which suggests that patient populations display more than one pattern of AOPP distribution. Our study demonstrates that AOPP levels increase because oxidative processes modify plasma proteins, especially albumin, which results in systemic

inflammation and tissue damage that affects psoriasis patients.

The study discovered that psoriasis patients exhibited decreased catalase activity which served as a measurement for their antioxidant defense system. The decrease shows that the body lost its ability to use enzymatic antioxidants for protecting against hydrogen peroxide-induced oxidative damage. The research results match the findings of Wójcik and his colleagues who discovered that psoriatic patients showed reduced antioxidant defense systems and their lymphocytes presented signs of redox imbalance (2020) [16]. Esmaeili and his colleagues established in their study (2019) [17] that redox imbalance occurs because the body loses its antioxidant capacity which leads to intensified IL-17 activity through inflammatory pathways. Kirmit and his colleagues (2020) [18] found that psoriasis patients demonstrate higher levels of catalase activity which indicates that their bodies increase antioxidant enzyme production as a response to heightened levels of reactive oxygen species. The study results show that patients with moderate disease severity experience lower catalase levels because their bodies face continuous oxidative stress which depletes their antioxidant capacity.

Our research found that oxidative markers were present but showed that CRP levels increased and participants exhibited an atherogenic lipid profile which included elevated total cholesterol and triglycerides and LDL levels while experiencing reduced HDL levels. The research findings provide support for Cannavò et al. (2019) who demonstrated that psoriasis involves oxidative stress and systemic inflammation and metabolic dysregulation through their research. The combination of dyslipidemia together with oxidative stress will worsen endothelial dysfunction while increasing the risk for cardiovascular disease. The elevated CRP levels in our patients demonstrate ongoing low-grade systemic inflammation which interacts with ROS to maintain keratinocyte hyperproliferation and cytokine release.

The positive correlations between MDA and AOPP and PASI together with the negative correlation between catalase and PASI demonstrate that oxidative–antioxidant imbalance directly affects disease activity. Sikar Aktürk et al. (2012) demonstrated that oxidative markers correlate with clinical severity because they found a significant relationship between MDA levels and PASI scores. The researchers found that oxidative markers did not show consistent links to disease severity according to Shakoei et al. (2021) because various biomarkers and patient characteristics determined the connection between the two. Our research shows significant relationships which support the theory that oxidative stress exists in psoriasis yet also drives its progression.

The logistic regression analysis in our study found that MDA levels above 4.5 nmol/mL increased psoriasis risk threefold while AOPP levels and reduced catalase activity served as independent risk factors. The research supports the existence of multiple causes that lead to different biological processes which include oxidative stress and its effects on immune and metabolic systems according to recent studies (Dobric et al., 2022). The discovery of oxidative markers as independent predictors shows that these markers could serve as additional biomarkers for both risk assessment and disease monitoring purposes.

The current research results show strong agreement with modern scientific studies which demonstrate that psoriasis patients experience both increased oxidative stress levels and decreased ability to fight oxidative damage. Our study demonstrates that skin disease severity directly affects the amount of MDA and AOPP which we measured since our research found decreased catalase activity on all tested levels. The study results demonstrate antioxidant-targeted treatment methods as essential components to complete psoriasis treatment.

## Conclusion

The present study demonstrates that psoriasis is strongly associated with increased oxidative stress, systemic inflammation, and metabolic disturbances. Patients with psoriasis showed significantly elevated levels of MDA and AOPP along with reduced catalase activity, indicating enhanced lipid and protein oxidation with impaired antioxidant defense. The positive correlation of MDA and AOPP with PASI score, and the negative correlation of catalase with disease severity, highlight the close association between redox imbalance and clinical activity. Additionally, altered lipid profile and raised CRP levels further support the systemic inflammatory nature of psoriasis. Logistic regression analysis confirmed oxidative stress markers as significant predictors of the disease. Overall, these findings emphasize the crucial role of oxidative stress in psoriasis pathogenesis and suggest potential value of antioxidant-based therapeutic strategies.

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