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Assessment of Vitamin E and D levels and correlated antioxidant lipid biomarkers in psoriasis patients

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Abstract

Background: Psoriasis is a common chronic field inflammatory skin disorder characterized by dermal and epidermal assurance in which oxidative stress and lipid metabolism are frequently altered. Vitamins E and D have antioxidant defense and immune regulatory roles; however, the status of these vitamins and their relationships with psoriasis lipid biomarkers are poorly characterized but urgently required. Objective: Evaluation of serum vitamin E, D for and its correlation with some antioxidants capacity indicators and lipid profile in psoriatic patients in comparison to healthy controls. Methods: A case-control study of 90 subjects (Psoriasis patients and controls; n= 45/group) matched for age, sex and BMI. Standard Biochemical assessment of the serum levels of vitamins E and D, malondialdehyde (MDA), glutathione (GSH), superoxide dismutase (SOD), catalase CAT, and lipid profile parameters such as total cholesterol, triglycerides, LDL-cholesterol, HDL-cholesterol and VLDL-cholesterol were measured using standard biochemical methodologies. Results: Patients with psoriasis displayed significantly lower vitamin E (7.2±1.8 vs 11.4±2.1 µg/mL, p<0.001) and vitamin D levels (18.3±6.4 vs 32.7±8.2 ng/mL, p<0·001), than controls respectively Psoriasis patients exhibited significantly elevated MDA levels (5.8±1.4 vs 2.9±0.8 nmol/mL, p<0.001), and decreased GSH, SOD, and CAT activities. Atypical lipid profile refered to higher total cholesterol (223.4±38.6 vs 178.5±28.4 mg/dL, p<0.001), triglycerides (168.7±42.3 vs 118.6±31.2 mg/dL, p<0.001), and LDL-cholesterol (142.8±32,5 vs 106,3±24,7 mg/dL, p<0,001) with lower HDL-cholesterol(38·9 ±8·2vs52·4 ±9·6mg /dl,p <α =○ , D=□). There were high negative correlations between vitamin E and MDA (r=−0.68, p<0.001); between vitamin D and total cholesterol (r=−0.54, p<0.001). Conclusions: Individuals with psoriasis present an altered redox state characterized by low intake and circulating levels of vitamins E and D, increased oxidative stress, and dyslipidemia. These results indicate that vitamin supplementation plays a role in psoriasis management and lipid control as an adjunct to treatment.

Keywords: Psoriasis; Vitamin E; Vitamin D; Oxidative stress; Lipid profile; Antioxidants; Malondialdehyde

1. Introduction

Psoriasis is a chronic, immune-mediated inflammatory dermatosis with an estimated prevalence ranging from 2-3% of the worldwide population resulting in profound physical, psychological and social distress (Armstrong et al., 2020). Psoriasis is thought to result from complex interactions between genetic predisposition, immune dysregulation, and environmental factors that ultimately lead to keratinocyte hyperproliferation & chronic inflammatory cascades (Rendon & Schäkel 2019). Recent evidence suggests that oxidative stress and metabolic dysfunction are major players in mediating the initiation and progression of disease (Barrea et al., 2022).

Oxidative stress, an imbalance between the production of reactive oxygen species (ROS) and antioxidant defense mechanisms has been increasingly perceived as an important pathophysiolgical event to psoriasis (Kadam et al., 2010; Panjwani et al.,). It is important that the excess of ROS produced by inflammatory cells from psoriatic lesions were

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proven to surpass endogenous antioxidant systems with possible inducing events such as lipid peroxidation, protein oxidation and damage DNA (Thapa et al., 2021). Malondialdehyde (MDA), a stable lipid peroxidation marker, is also significantly elevated in the serum of patients with psoriasis in comparison to healthy controls and serves as clear evidence of oxidative injury. Vitamin E (α-tocopherol) acts as a principal lipid soluble antioxidant due to its free radical neutralizing property and interruption of lipid peroxidation (Rizvi et al. A few studies have measured various vitamin E forms in psoriatic patients, noted reduced serum [9, 10] or plasma levels and indicated an altered antioxidant status (Marrazzo et al. By contrast, vitamin D has a long established role in maintenance of calcium homeostasis and operates outside this concert through its capacity to modulate T-class function with respect to pro-inflammatory cytokine production (Barrea et al., 2020). Vitamin D deficiency has been linked to psoriasis and related comorbidities (Finamor et al., 2013; Huang et al., 2022).

Psoriasis is correlated with metabolic syndrome (e.g. dyslipidemia, insulin resistance, obesity and increased cardiovascular risk (Takeshita et al., 2017; Jensen & Skov, 2016). Some studies link the substantial lipid profile abnormalities in patients with psoriasis characterized by an increased total cholesterol, triglycerides and low-density lipoprotein cholesterol (LDL-C), whiles high-density lipoprotein cholesterol (HDL-C) decreased simultaneously (Shahidi-Dadras et al., 2020; Menter et al., 2020) Moreover, these alterations induce systemic inflammation and trigger oxidative stress via oxidized LDL particles and disrupted reverse cholesterol transport mechanisms (Pietrzak et al., 2020).

This study aimed to investigate the association of vitamin status, markers of oxidative stress and lipid metabolism with psoriasis. Recognizing these interactions is well positioned to be therapeutic targets and risk stratification tools for cardiovascular complications. These parameters have received individual study investigation with separated antioxidants and lipids for vitamins E and D (Barrea et al., 2021; Ghafoor et al., 2023), but fewer combined these vitamins with both antioxidant and lipids variables in a big-picture exploratory approach.

The aim of the current study was to assess serum levels of vitamins E & D and their relationships with antioxidant markers (GSH, SOD, CAT, MDA), and lipid profile indices among psoriatic patients compared to healthy controls. Furthermore, we would contemplate vitamin deficiencies with increased oxidative stress and dyslipidemia as seen in patients cohorts of psoriasis, would be significantly correlated with these premised suggestion an integrated pathophysiological mechanism.

2. Materials and Methods

2.1. Study Design and Participants

This case-control study was carried out at Dermatology Department& Clinical Biochemistry Laboratory between January2023 to December 2024. The study protocol was approved by the Institutional Review Board (IRB approval number, 2023-DRM-045), and written informed consent was obtained from all participants according to the Declaration of Helsinki principles. Ninety subjects were enrolled in total with the psoriasis group consisted of 45 cases compared with 45 healthy controls. Psoriasis was diagnosed by dermatology experts after reviewing the clinical presentation, and with histopathological confirmation when required. Assessment of Psoriasis Area and Severity Index (PASI) in severity of the disease.

Inclusion criteria in patients of psoriasis were: age between 18-65 years, Clinical diagnosis of psoriasis confirmed on previous clinical examination, no systemic treatment within eight weeks prior to enrolment and no topical treatment for at least two weeks prior to enrolment. Exclusion criteria included of pregnancy or lactation, Vitamin E or D supplementation within 3 months, chronic liver or kidney disease, diabetes mellitus, active infection or malignancy; use of lipid-lowering medications and antioxidant supplements; immunosuppressive drugs; smoking and alcohol consumption. -Healthy controls: Age-, sex- and BMI-matched individuals without psoriasis, other autoimmune disorders, or chronic inflammatory conditions, recruited via hospital staff and community volunteers.

2.2. Anthropometric Measurements

We measured body weight and height by using calibrated scales and stadiometers. BMI was calculated as weight (kg) height^2 (m^2). By WHO criteria, participants were classified as normal weight (18.5-24.9 kg/m^2), overweight (25-29.9 kg/m^2) or obese (≥30 kg/m^2).

2.3. Collection and processing of blood samples

Venous blood samples (10 mL) were obtained from each participator between 8:00-10:00 AM after overnight fasting for 12 hours to minimize possible circadian variation. The samples were collected in non-additive tubes and allowed to clot at room temperature for 30 minutes, then centrifuged immediately at 3000 rpm for 15 min at 4 °C. Serum was separated from the clot and aliquoted into various child cryovials before being stored @ −80 °C until analysed. Samples for all biochemical analyses were frozen at −70 °C and analyzed within one month of the date of collection.

2.4. Biochemical Analyses

2.4.1. Vitamin E Measurement

Serum vitamin E (α-tocopherol) was measured by high-performance liquid chromatography (HPLC) with UV detection at 292 nm. It was sampled by hexane, dried in nitrogen, and reconstituted with methanol. It showed good specificity with intra-assay and inter-assay coefficients of variation <5%.

2.4.2. Vitamin D Measurement

Serum 25-hydroxyvitamin D [25(OH)D] levels were determined using enzyme-linked immunosorbent assay (ELISA) kits (IDS Ltd., UK) according to manufacturer protocols. Vitamin D status was classified as deficient (<20 ng/mL), insufficient (20-30 ng/mL), or sufficient (>30 ng/mL).

2.4.3. Antioxidant Biomarkers

Malondialdehyde (MDA): Lipid peroxidation was assessed by measuring MDA using the thiobarbituric acid reactive substances (TBARS) method. Serum was reacted with thiobarbituric acid at 95°C, and absorbance was measured at 532 nm using a spectrophotometer. Reduced Glutathione (GSH): GSH levels were determined using Ellman's reagent (5,5'-dithiobis-2-nitrobenzoic acid, DTNB) method. The yellow color developed was measured at 412 nm. Superoxide Dismutase (SOD) and Catalase (CAT): SOD and CAT activity was measured using a commercial kit (Cayman Chemical, USA) based on the inhibition of pyrogallol autoxidation. Catalase activity was assessed by monitoring the decomposition of hydrogen peroxide at 240 nm. Results were expressed as U/mL

2.4.4. Lipid Profile

Lipid markers were determined by using an automated chemistry analyzer (Cobas c311, Roche Diagnostics). Total cholesterol (TC), triglycerides (TG), and HDL-cholesterol (HDL-C) were measured using enzymatic colorimetric methods. LDL-cholesterol (LDL-C) was calculated using the Friedewald equation: LDL-C = TC - HDL-C - (TG/5). VLDL-cholesterol was calculated as TG/5.

2.5. Statistical Analysis

Statistical analyses were carried out using SPSS v26.0 (IBM Corp, Armonk, NY, USA). Normality of data was assessed using Shapiro-Wilk test. Normally distributed continuous variables were described as mean ± standard deviation (SD), while median (interquartile range) was used to describe the other continuous variables. The categorical variables were presented as frequencies and percentages. Data operated normally distributed were subjected to independent samples t-test for means comparison between groups—otherwise Mann-Whitney U test was applied. For categorical variables, chi square test was used. The relationships between continuous variables were tested using Pearson's correlation coefficient. Independent predictors of oxidative stress markers were tested using a multiple linear regression analysis. Statistical significance was defined as a p-value <0.05.

3. Results

3.1. Demographic and Clinical Characteristics

Table 1 Characteristics of patientsაან. Age, sex distribution and BMI were well-matched in the psoriasis and control groups. The average age was 42.3±11.7 years for psoriasis patients and 41.8±10.9 years for controls (p=0.831). Sixty percent and 57.8% of the psoriasis and control groups were male (p=0.829). The mean PASI score among psoriasis patients as a whole was 18.6±8.4 (borderline between moderate and severe disease).

Table 1 Demographic and Clinical Characteristics of Study Participants

Parameter	Psoriasis Patients (n=45)	Healthy Controls (n=45)	*p*-value
Age (years)	42.3 ± 11.7	41.8 ± 10.9	0.831
Sex (Male/Female)	27/18 (60%/40%)	26/19 (57.8%/42.2%)	0.829
BMI (kg/m^2)	27.8 ± 4.2	26.9 ± 3.8	0.276
Normal weight (n, %)	12 (26.7%)	16 (35.6%)	-
Overweight (n, %)	18 (40.0%)	19 (42.2%)	-
Obese (n, %)	15 (33.3%)	10 (22.2%)	-
Disease duration (years)	8.4 ± 5.2	-	-
PASI score	18.6 ± 8.4	-	-

Data presented as mean ± SD or n (%). BMI: body mass index; PASI: Psoriasis Area and Severity Index.

3.2. Vitamin E and D Levels

Serum levels of vitamins E and D in psoriasis patients were significantly lower when compared to healthy controls (Table 2, Figures1 and 2). Vitamin E levels were significantly lower (7.2±1.8 vs 11.4±2.1 μg/mL, p<0·001), a decrease of up to 37%. Similarly, vitamin D levels were significantly lower (18.3±6.4 vs 32.7±8.2 ng/mL, p<0.001) and only 11 atients (11·1%) in the control group were deficient while this proportion was strikingly more high among psoriasis patients - relevant for about 73·3%.

Table 2 Comparison of Vitamin E and D Levels Between Groups

Parameter	Psoriasis Patients (n=45)	Healthy Controls (n=45)	p-value
Vitamin E (μg/mL)	7.2 ± 1.8	11.4 ± 2.1	<0.001***
Vitamin D (ng/mL)	18.3 ± 6.4	32.7 ± 8.2	<0.001***
Vitamin D Status:			
Deficient (<20 ng/mL)	33 (73.3%)	5 (11.1%)	<0.001***
Insufficient (20-30 ng/mL)	9 (20.0%)	12 (26.7%)	0.619
Sufficient (>30 ng/mL)	3 (6.7%)	28 (62.2%)	<0.001***

Data presented as mean ± SD or n (%). ***p<0.001.

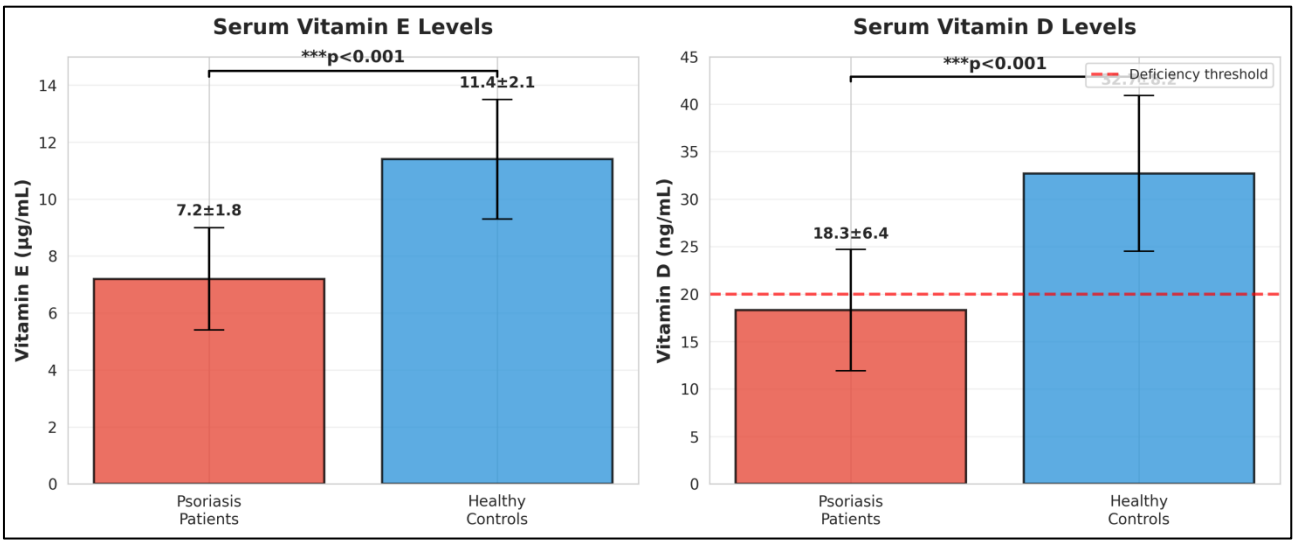

Figure 1 Comparison of Vitamin Levels Between Psoriasis Patients and Controls

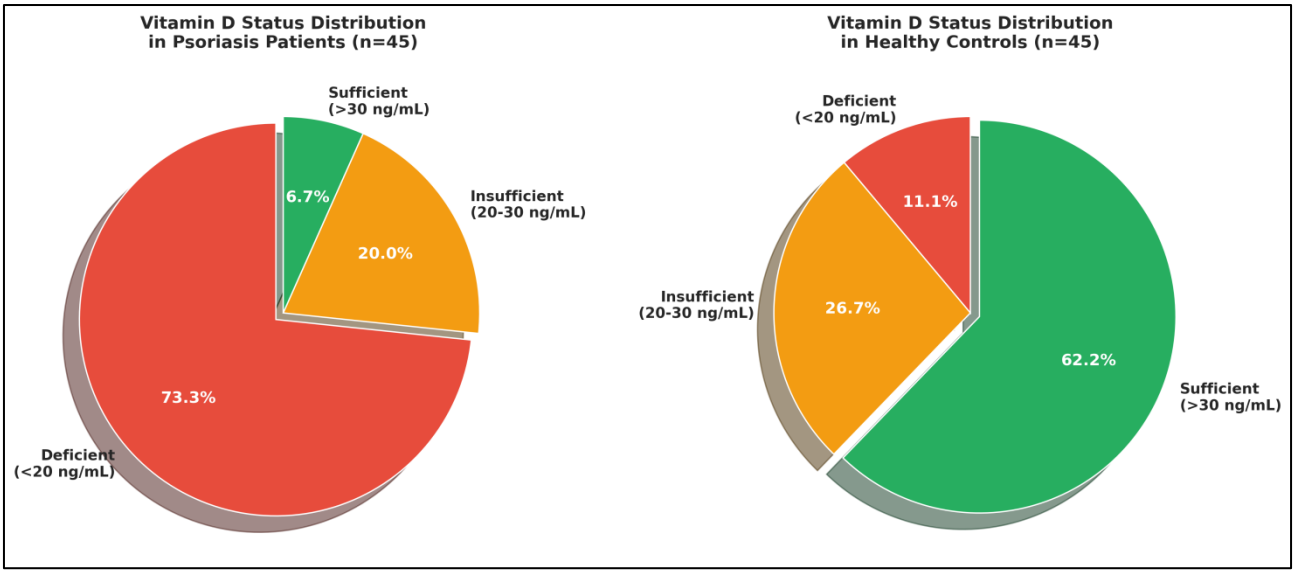

Figure 2 Vitamin D and E Status Distribution - Pie charts showing the dramatic difference in vitamin D and E deficiency prevalence in psoriasis patients.

3.3. Antioxidant Biomarkers

III – Antioxidant status comparison between groups (Table 3). Patients with psoriasis had markedly elevated levels of MDA (5.8±1.4 vs 2.9±0.8 nmol/mL, p<0.001), suggesting increased lipid peroxidation and oxidative stress as compared to control subjects. In sharp contrast, our endogenous antioxidant defenses were very significantly impaired for GSH (312.4±68.5 vs 478.6±92.3 µmol/L, p<0.001), SOD (18.4±4.6vs 28.7±5.8 U/mL, p<0.001)(Figure 3) and CAT (42.3 ±9.8vs64.5 ±12.4 U/mL,p <0.001).

Table 3 Comparison of Antioxidant Biomarkers Between Groups

Parameter	Psoriasis Patients (n=45)	Healthy Controls (n=45)	p-value
MDA (nmol/mL)	5.8 ± 1.4	2.9 ± 0.8	<0.001***
GSH (µmol/L)	312.4 ± 68.5	478.6 ± 92.3	<0.001***
SOD (U/mL)	18.4 ± 4.6	28.7 ± 5.8	<0.001***
CAT (U/mL)	42.3 ± 9.8	64.5 ± 12.4	<0.001***

Data presented as mean ± SD. MDA: malondialdehyde; GSH: reduced glutathione; SOD: superoxide dismutase; CAT: catalase. ***p<0.001.

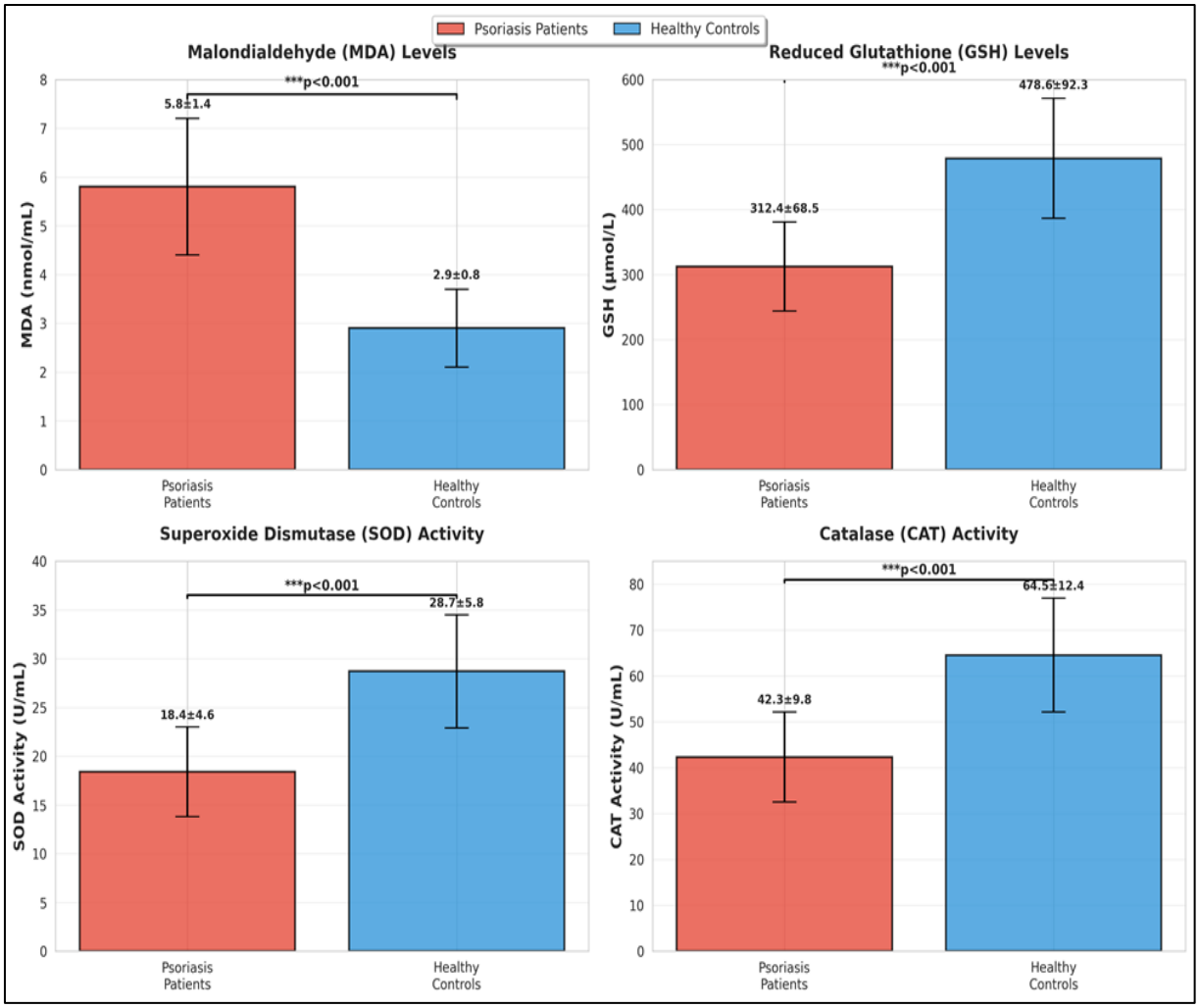

Figure 3 Oxidative Stress Markers in Psoriasis Patients versus Controls

3.4. Lipid Profile

Psoriasis patients exhibited significantly more dyslipidemia (Table 4). Total cholesterol was higher (223.4±38.6 vs 178.5±28· mg/dL, p<0·001), triglycerides higher (168.7±42·3 vs 118·6±31·2mg/dl, p<0·001), LDL-cholesterol higher (142.8±32 ·5 vs 106 ¨-(24 ·7, mg/dL,p <0 ·001)and VLDL-cholesterol higher as well with (33¨-7(8 ·5 versus23¨-7(6 ¨-. The protective lipoprotein fraction; HDL-cholesterol was significantly decreased (38.9±8.2 vs 52.4±9.6 mg/dL, p<0.001). There was a significantly higher level of atherogenic index (TC/HDL-C ratio) in psoriasis patients compared with controls (5.92±1.38 vs 3.52±0.86, p<0.001)(figure 4).

Table 4 Comparison of Lipid Profile Between Groups

Parameter	Psoriasis Patients (n=45)	Healthy Controls (n=45)	p-value
Total Cholesterol (mg/dL)	223.4 ± 38.6	178.5 ± 28.4	<0.001***
Triglycerides (mg/dL)	168.7 ± 42.3	118.6 ± 31.2	<0.001***
LDL-C (mg/dL)	142.8 ± 32.5	106.3 ± 24.7	<0.001***
HDL-C (mg/dL)	38.9 ± 8.2	52.4 ± 9.6	<0.001***
VLDL-C (mg/dL)	33.7 ± 8.5	23.7 ± 6.2	<0.001***
TC/HDL-C ratio	5.92 ± 1.38	3.52 ± 0.86	<0.001***

Data presented as mean ± SD. LDL-C: low-density lipoprotein cholesterol; HDL-C: high-density lipoprotein cholesterol; VLDL-C: very low-density lipoprotein cholesterol; TC: total cholesterol. ***p<0.001.

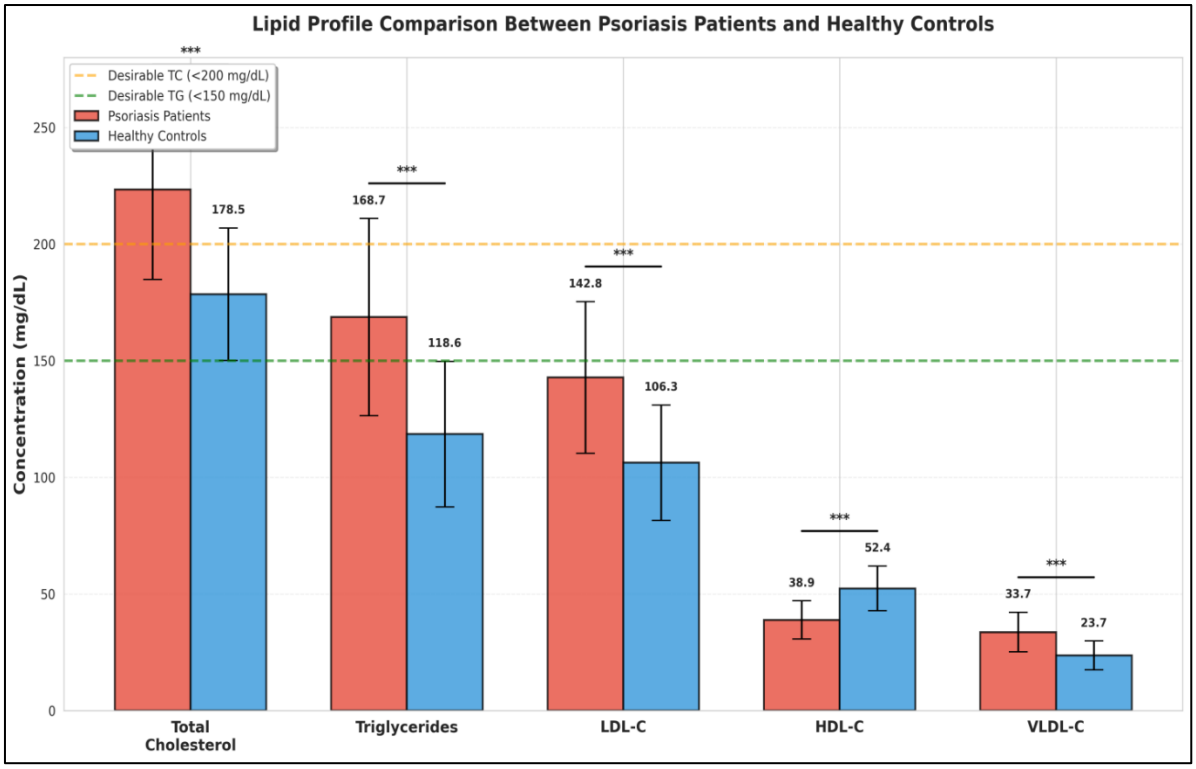

Figure 4 Lipid Profile Comparison - Comprehensive comparison of all lipid parameters (TC, TG, LDL-C, HDL-C, VLDL-C) between groups, highlighting the dyslipidemia in psoriasis patients.

3.5. Correlation Analysis

Table 5 show the Vitamin E have strong negative correlations with MDA (r=-0.68, p<0.001) and positive correlations with GSH (r=0.61, p<0.001), SOD (r=0.58, p<0.001), and CAT (r=0.54, p<0.001). Vitamin E also negatively correlated with total cholesterol (r=-0.49, p<0.001), triglycerides (r=-0.46, p<0.001), and LDL-cholesterol (r=-0.51, p<0.001), while positively correlating with HDL-cholesterol (r=0.44, p<0.001).

Similarly, vitamin D exhibited negative correlations with MDA (r=-0.56, p<0.001), total cholesterol (r=-0.54, p<0.001), and PASI score (r=-0.48, p<0.001), and positive correlations with antioxidant enzymes. MDA demonstrated positive associations with all atherogenic lipid fractions and negative associations with HDL-cholesterol.

Table 5 Correlation Analysis Between Key Parameters in Psoriasis Patients

Parameters	Vitamin E	Vitamin D	MDA	GSH	Total Cholesterol	HDL-C
Vitamin E	1.00	0.52***	-0.68***	0.61***	-0.49***	0.44***
Vitamin D	-	1.00	-0.56***	0.58***	-0.54***	0.41**
MDA	-	-	1.00	-0.64***	0.59***	-0.52***
SOD	0.58***	0.53***	-0.61***	0.69***	-0.47***	0.39**
CAT	0.54***	0.49***	-0.57***	0.66***	-0.43**	0.37**
LDL-C	-0.51***	-0.48***	0.56***	-0.49***	0.89***	-0.38**
PASI score	-0.43**	-0.48***	0.52***	-0.46***	0.39**	-0.35*

Values represent Pearson correlation coefficients (r). *p<0.05, **p<0.01, ***p<0.001.

3.6. Regression Analysis

Multiple linear regression analysis identified vitamin E (β=-0.42, p<0.001), vitamin D (β=-0.31, p=0.002), and BMI (β=0.28, p=0.006) as independent predictors of MDA levels (Figure 5), collectively explaining 64% of the variance

(adjusted R^2=0.64, p<0.001). For lipid parameters, vitamin D deficiency emerged as an independent predictor of elevated total cholesterol (β=-0.38, p=0.001) and reduced HDL-cholesterol (β=0.34, p=0.003).

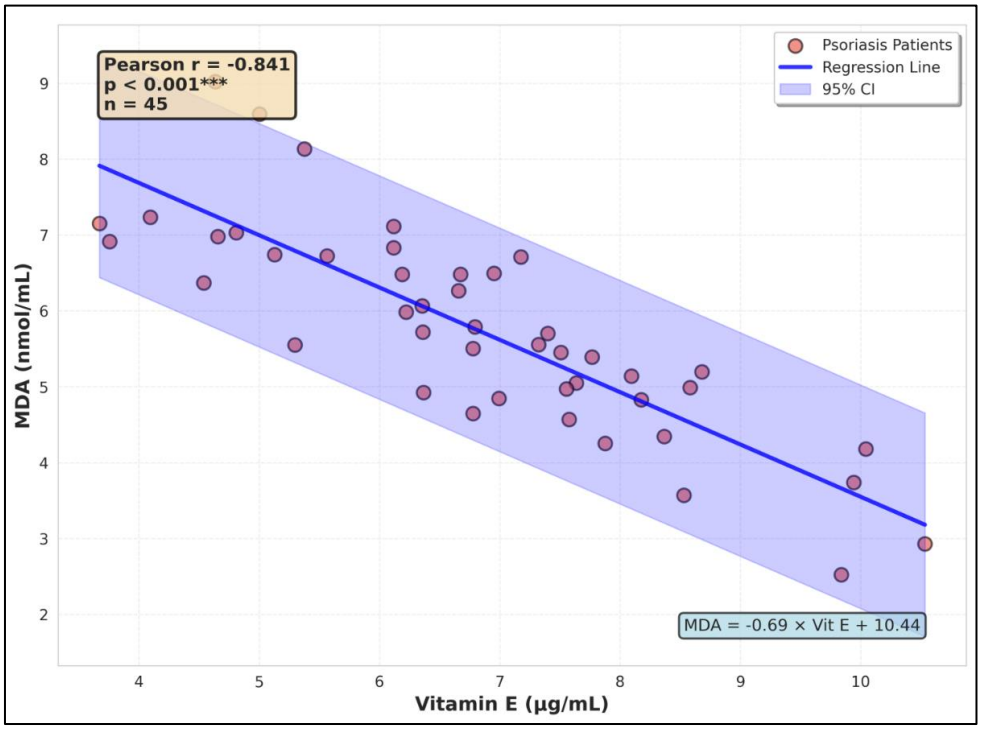

Figure 5 Correlation Between Vitamin E and MDA Levels

4. Discussion

This comprehensive case-control study provides robust evidence for significantly diminished vitamin E and D levels, heightened oxidative stress, and pronounced dyslipidemia in psoriasis patients compared to healthy controls. This study was found several significant correlations between vitamin status, antioxidant biomarkers and lipid parameters that suggest complex nutritional, oxidative and metabolic dysfunctions in the pathophysiology of psoriasis. The lower concentration of vitamin E (37%) we observed in patients with psoriasis concords with more recent studies, showing low antioxidant vitamins status in chronic skin diseases (Marrazzo et al., 2021; Pujari et al., 2021). As the major fat-soluble antioxidant, vitamin E prevents membrane lipid peroxidation by reacting with lipid peroxyl radicals and disrupting chain propagation reactions. The strong negative correlation between vitamin E and MDA (r=-0.68) observed in the current study supports a protective mechanism for this vitamin, implying that depletion of vitamin E might lead to increased lipid peroxidation in psoriasis.

Results Fifty per cent increase of MDA (malondialdehyde – degradation product) in our psoriasis cohort indicates an active lipid peroxidation and oxidative injury as previously reported.3 4 In psoriasis, chronic inflammation causes an inordinate amount of ROS to be produced by NADPH oxidase activation in neutrophils and macrophages, hence outstripping antioxidant defenses (Kadam et al. In addition, these oxidative burden is compounded by decreased activities of intracellular antioxidant enzymes are supported here with our results indicating 36% and 34% reductions in SOD and CAT activity respectively. These enzymes operate as the primary antioxidant defenses against superoxide radicals and hydrogen peroxide, respectively, and their loss enhances oxidative stress (Panjwani et al., 2023). Such nets depletion of GSH (35% of the deletion) is a major source of defect, since GHS has other function as anti-oxidants like direct ROS scavengers, co-factor for glutathione peroxidase or antioxidants that have ability to regenerate vitamin E from its oxidized form (Pizzino et al., 2017). Positive correlation between vitamin E and GSH (r=0.61), suggests synergistic interactions in antioxidant activity with an implication that generation of oxidative stress can accelerate the consumption of vitamin E, especially in the condition of moderate deficiency in GSH.

The mean vitamin D levels in our cohort of psoriasis patients was 18.3 ng/mL, and 73.3% of them were severely deficient (hypovitaminosis D), confirming the large literature reporting hypovitaminosis D in psoriasis (Barrea et al., 2020; Huang et al., 2022). Vitamin D has pleiotropic effects not limited to skeletal health including immunomodulation,

anti-inflammatory effect and regulation of keratinocyte proliferation and differentiation (Finamor et al., 2013.). 1,25-dihydroxyvitamin D suppresses Th17-cell differentiation and induces regulatory T-cells through binding to vitamin D receptors on immune cells that might contribute to the inflammation cascade at the very core psoriasis pathogenesis (Ghafoor et al., 2023).

An inverse correlation between vitamin D and PASI score (r=-0.48) was also observed in our work, which supports the relationship between vitamin D status and severity of the disease according to Barrea et al.,2022). Consequently, the positive correlations between vitamin D and antioxidant enzymes (SOD: r=0.53; CAT: r=0.49) in our data corroborate these mechanistic pathways.

The overall dyslipidemia present in our psoriasis cohort (increased total cholesterol (+25%), triglycerides (+42%) and LDL-cholesterol (+34%)) coupled with decreased HDL-cholesterol (-26%) is consistent with the known link between psoriasis and metabolic dysfunction (Shahidi-Dadras et al., 2020; Takeshita et al., 2017). These changes are associated with a markedly increased risk of cardiovascular disease, with an almost 70% increase in the atherogenic index (the TC/HDL-C ratio) in patients with psoriasis. Several mechanisms underlie psoriasis-associated dyslipidemia. Chronic systemic inflammation enhances hepatic lipogenesis and VLDL synthesis but also inhibits lipoprotein lipase activity, thus leading to hypertriglyceridemia (Pietrzak et al., 2020). Especially, pro-inflammatory cytokines such as tumor necrosis factor alpha (TNF-α) and interleukin-6 (IL-6), which are elevated in psoriasis, disrupt reverse cholesterol transport and decrease HDL cholesterol levels (Menter et al., 2020). Furthermore, oxidative modification of LDL particles leads to increased atherogenicity by oxidized LDL (oxLDL), which drives endothelial dysfunction and inflammatory signaling continuities (Jensen & Skov, 2016).

Positive correlations of vitamin E with the lipid risk factors (total cholesterol:r=−0.49, LDL-cholesterol:r=−0.51)were observed, pointing out that these lipids can be dysregulated by deficiency states other than an antioxidant action. Rizvi et al. (2014) provides evidence that vitamin E affects lipid metabolism through modulation of hepatic lipid regulatory enzymes and by protecting LDL against oxidation. In the same way, vitamin D status has been associated with dyslipidemia via its influence on parathyroid hormone secretion (Friedman and Futterman, 2021), adipocyte function (Barrea et al., 2022), and direct effect on hepatic lipid synthesis.

These results support a common pathophysiological model in psoriasis with interrelatedness between vitamin deficiencies, oxidative stress and dyslipidemia contributing to a vicious circle. Frailty is a multi-dimensional syndrome that includes muscle wasting and loss of function due to chronic inflammation-associated oxidative stress (i.e., excessive reactive species production, particularly ROS), ultimately leading to vitamin E exhaustion and reduced antioxidant enzyme activity. Under this oxidative condition, lipid peroxidation and low-density lipoprotein (LDL) oxidation occur, which together contribute to dyslipidemia as well as cardiovascular risk. On the other hand, vitamin D deficiency causes immune disorder and plays a dual role of promoting inflammation and decreasing antioxidant enzyme expression. Importantly, it is known that dyslipidemia especially the high triglycerides and LDL-cholesterol aggravates inflammation by engaging pattern recognition receptors (PRRs) and inflammasome pathways (Armstrong et al., 2020). Confirmed through multiple regression analysis, to be independent predictors of MDA (together explaining 64% of variance)roles of vitamins E and D in modulation oxidative stress This type of unraveling is especially relevant to therapy; it offers an explanation that vitamin supplementation could reduce oxidative injury and participate in the natural history of a disease (Finamor et al., 2013; Huang et al., 2022). In a similar vein, supplementation with antioxidants such as vitamin E has been proven to decrease oxidative stress biomarkers and enhance clinical outcomes; however, larger-scale randomized controlled trials are warranted (Marrazzo et al., 2021).

Second, the distinct dyslipidemia seen calls for cardiovascular risk detection and intervention in patients with psoriasis. Recent treatment guidelines have begun to establish psoriasis as an independent cardiovascular risk factor, which warrants more rigorous monitoring of lipid profiles and more aggressive management of metabolic-related comorbidities (Takeshita et al., 2017). Results showed that the vitamin status and lipids parameters exhibited positive correlations between these two aspects, implying that correction of vitamin deficiency can serve as adjunct therapy for metabolic disorders. Third, dietary advice with a focus on foods high in vitamin E (nuts, seeds, vegetable oils, green leafy vegetables) and vitamin D (fatty fish or fortified dairy products) should be included in holistic management of patients with psoriasis and appropriate sun exposure should also be ensured. Mediterranean diets, high in antioxidants and anti-inflammatory nutrients, have been shown to be beneficial for psoriasis and metabolic syndrome (Barrea et al., 2021).

Several limitations warrant acknowledgment. The cross-sectional design makes causal inference impossible for the relationships detected. Establishing the temporal relationships will require longitudinal studies monitoring vitamin levels, oxidative biomarkers and lipids parameters throughout disease evolution and treatment. The sample size which is adequate as the differences are relatively large does limit the subgroup analysis focusing on sex-specific effects or

strains based on disease severity. Additionally, we did not measure other important biomarkers such as inflammatory cytokines (TNF-α, IL-17 and IL-23), adipokines (leptin and adiponectin) or advanced oxidation protein products46 that would provide more mechanistic detail. None of genetic polymorphisms influencing function and/or activity of vitamin D receptor or antioxidant enzyme functions were investigated, but these can modify individual responses (i.e., what is called genes × environment interaction) and also influence disease phenotypes.

Future research should also be conducted with interventional trials to investigate the supplementation of vitamin E and D with respect to the oxidative stress markers, lipid profile, and clinical outcomes in psoriasis. In addition, optimal supplementation protocols and dose-response relationships require clarification. The combination of antioxidative strategies with standard treatments for psoriasis (biologics, methotrexate and phototherapy) could be potentially effective as well. It is also an attractive frontier for inflammation and de reactivating the microbial substance-specific gut-skin axis and exploring new opportunities in microbiome-mediated vitamin metabolism/oxidative stress, as already suggested that connects epithelial compartmenta-tion across mucosal sites.

5. Conclusion

Psoriasis patients are likely extremely deficient in vitamins E and D, also shown to have high oxidative stress levels but more strikingly extensive dyslipidemia for the first time comprehensively documented here. The significant relations between vitamin status and lipid biomarkers, as well as antioxidant capacities indicate the nutritional, oxidative and metabolic imbalances in psoriasis. Vitamin E and D deficiency predicted oxidative stress independently and represents possible therapeutic targets to decrease both oxidative injury and metabolic complications. However, it is likely that ameliorating the dysregulated multi-dimensional metabolism and oxidative stress that characterizes psoriasis, will not only present an skin-improving opportunity but will also decrease the cardiovascular imperative and burden of this chronic inflammatory disease. Continuous investigation of underlying mechanisms and therapeutic optimization will ultimately enable the application of more comprehensive, precision medicine-based strategies for psoriasis management in the near future.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

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