# ASSOCIATION OF ASCORBIC ACID CONCENTRATION AND PRETERM PREMATURE RUPTURE OF MEMBRANE

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## Abstract

**Background:** Premature rupture of membranes (PROM) is the rupture of amniotic membranes before labor begins, and when membranes rupture occurs before 37 weeks of gestation, it is known as preterm PROM (PPROM). It is known to complicate around 3% of preterm pregnancies and is responsible for 40% to 75% of neonatal deaths. It is suggested that vitamin C status plays a role in reducing the incidence of PPROM. The study aims to establish the association between maternal plasma vitamin C concentration among women with PPROM and women without PPROM.

**Subjects and Methods:** A prospective cohort study was done at Al Basrah Teaching Hospital for maternity and children over the period from January 1st, 2023, to December 31st, 2023. The study included 34 cases of PPROM, which were matched for gestational age with 28 controls. The data were collected from women who were included in the study through a well-formed questionnaire. Then 5 cc of blood was drawn from each woman to measure the Hb, WBC, and vitamin C levels.

**Results**: The vitamin C level was lower among cases (7.22 ± 4.66) than controls (14.23±5.29) with a P-value of 0.001. Urinary tract infection was also higher among cases (p –value=0.001). Women with caesarean sections had a lower level of vitamin C (6.23 ± 2.8) in comparison to women with vaginal delivery (11.63 ± 6.05), and those with postpartum haemorrhage had a lower level of vitamin C (7.25 ± 4.91). however, only the mode of delivery had a statistically significant level (p-value of 0.001).

**Conclusion:** A lower level of vitamin C was noticed among women with PPROM in comparison to healthy women. A higher incidence of postpartum haemorrhage (PPH) and neonatal complications was noticed among those with PROM and vitamin C deficiency**.**

**Keywords:** Preterm, PROM, vitamin C, pregnancy.

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## Introduction

Psoriasis is a collection of long-lasting immune-mediated inflammatory skin illnesses that are associated with many types of metabolic and cardiovascular conditions [1]. Complex multisystemic interactions disturb the balance between anti-inflammatory and immune responses contributing to psoriasis's pathogenesis [2]. Psoriasis is characterised by an increase in the thickness of the outer layer of the skin (epidermal hyperplasia) and the presence of immune cells in the deeper layer of the skin (dermal immune cell infiltration). Psoriasis has a multifaceted origin that entails interactions among keratinocytes, immune cells, and other cells residing in the skin. Psoriasis is commonly understood as an immune cell-driven disorder, where keratinocytes play a supporting role in the activity of immune cells. [3]

It has been shown that lymphocytes and the cytokines they release, particularly interferon (IFN) and interleukin 17 (IL-17), have an impact on other cells, causing persistent inflammation and recognizable signs such as cutaneous plaques in psoriasis [4]. Current research on immunological diseases has shown that oxidative stress and altered phospholipid metabolism frequently underlie the development of these conditions suggesting that both reactive oxygen species (ROS) and lipid mediators play significant roles in the pathophysiology of psoriasis [5].

Lipids play a vital role in all biological mechanisms, including developing and maintaining the skin barrier. The metabolism of n-3 and n-6 polyunsaturated fatty acids (PUFAs) is of interest. The most dysregulated n-6 PUFA in psoriasis is arachidonic acid, and many lipid mediators derived from it are present in significantly higher concentrations in the skin [2]. Psoriatic skin was shown to have a higher level of 12- hydroxy-eicosatetraenoic acids (HETEs), a product of lipoxygenases (LOX). It was discovered that 12-HETE, a significant leukocyte chemoattractant, encouraged T cell recruitment in psoriasis [6]. Lipoxygenase action on Alpha Linolenic acid (ALA) produces hydroxy-octadecadienoic acids (HODEs). Although, 13 HODE was found to have an antiproliferative effect in normal skin. It was discovered that 9-HODE and 13-HODE were higher in psoriatic skin than in healthy skin [7].

So this study aim to explore further on the level of these mediators in psoriasis and attempt to link the level with severity of psoriasis measured by Psoriasis Area and Severity index. Emphasizing on its role as severity markers

## Materials and Methods

A case-control study was conducted between November 2019 and January 2021 on 31 patients diagnosed with psoriasis who were attending outpatient dermatology clinics of AL-Sadder Teaching Hospital and AL-Basra Teaching Hospital and patients referred from dermatologist clinics. In addition, 20 apparently healthy adults, who were of the same age and sex group as the patient cohort, were included as controls in the study. After excluding patients on topical and systemic treatment, patients on non-steroidal anti-inflammatory drugs and biological therapy. Patients with chronic medical illness and arthritis also excluded.

Age, gender, duration of psoriasis, family history of psoriasis and used of any medication obtained through a detailed interviewer-administered questionnaire. The dermatologist performs clinical and dermatological assessments using The Psoriasis Area and Severity Index (PSAI) is computed for every individual patient. The PSAI score is determined by assessing the extent of psoriasis on four specific body areas: the head and neck (H), the upper limbs (UL), the trunk (T), and the lower limbs (LL). Each location is assigned a numerical score (A) that represents the

proportion of skin afflicted. 1 represents a range of 0-9%, 2 represents a range of 10-29%, 3 represents a range of 30-49%, 4 represents a range of 50-69%, 5 represents a range of 70-89%, and 6 represents a range of 90-100%. Subsequently Within each location (H, UL, T, LL), disease severity is determined by evaluating three signs of plaque - erythema (E), thickness/induration (I), and desquamation/scaling (D) - on a 5-point scale: 0 (none), 1 (mild), 2 (moderate), 3 (severe), or 4 (extremely severe). The ultimate PASI score is computed via the subsequent formula:

PASI = 0.1 (EH+IH+ DH) AH + 0.2 (EUL+IUL+ DUL) AUL + + 0.3 (ET+IT+ DT) AT + 0.4 (ELL+ILL+ DLL) ALL

The final calculate PASI score ranges from 0 to 72, with a higher score corresponding to greater disease severity [8]. Bodyweight and height were measured. Body mass index (BMI) was calculated.

Venous blood samples, measuring five to eight millilitres, were obtained from healthy control individuals and psoriatic patients while seated, using disposable syringes. Additionally, eight-millimetre skin biopsies were extracted from the psoriatic patients' lesional and non-lesional skin. Following biopsy extraction, the skin samples were weighed and rinsed thoroughly with 500 µl of phosphate buffer saline (PBS) to remove excess blood. All tissue samples were homogenized using the Heartbreaker 2 tissue homogenizer, manufactured by Virogen Company in the United States. The homogenates were subsequently centrifuged at 5000 rpm for a period of 15 minutes, and 100 µl of the diluted tissue homogenate was analyzed for 13-HODE and 12-HETE using a kit supplied by abcam Ltd, UK. Additionally, serum samples were utilized to measure 13-HODE using a 13-hydroxyoctadecadienoic acid (HODE) ELISA kit and 12-HETE using a 12-hydroxyeicosatetraenoic acid (HETE) ELISA kit. The lipid profile, including cholesterol, triglyceride, high Density Lipoprotien Cholesterol (HDLc), and Low Density Lipoprotein Cholesterol (LDLc) measurements, was analyzed using the Roche Hitachi 400 plus autoanalyzer.

Prior to their participation in the study, all patients provided written informed consent. The study was conducted in accordance with the guidelines outlined in the 1995 Helsinki Declaration and was approved by the Scientific Council of Dermatology, Arab Board of Health Specializations.

Statistical analysis was carried out utilizing the MedCalc® version 19.7 product developed by MedCalc Software Ltd in Belgium. Numerical variables were expressed as means ± standard deviation (SD), whereas categorical variables were expressed as number (%) values. To compare the demographic and biochemical characteristics between the patient and control groups, independent t-tests and chi-squared tests were employed, respectively. One-way analysis

of variance (ANOVA) was used to compare the different severity groups, followed by post hoc analysis performed with Tukey Kramer test. Any p-value found to be less than 0.05 was deemed statistically significant. Correlation analysis between various biochemical parameters and the severity of psoriasis was performed using Pearson correlation, and the resulting correlation coefficient (r) was recorded

## Results

### Demographic and Biochemical characteristics of the study population

Table 3.1 presents the demographic characteristics of two groups: a group of 31 patients with psoriasis (19 males and 12 females) with a mean age of 29 (±14.09) years and a control group of 20 apparently healthy adults (12 males and eight females) with a mean age of 34.1 (±11.1) years. The statistical analysis found that the differences in age, weight, height, and BMI between the two groups were insignificant (P value >0.05).

Table 3.2 presents the results of the biochemical analysis conducted on the study population, which included 31 patients with psoriasis and 20 apparently healthy adults as a control group. The mean values of 12-HETE and 13-HODE, two important inflammatory markers, were found to be significantly higher in the patient group compared to the control group (p value <0.05). In contrast, although the serum levels of cholesterol, triglycerides, HDL, and LDL for the psoriatic patients differed from those of the control group, the differences did not reach statistical significance (p value >0.05).

### Characteristics of Psoriatic Population

Table 3.3 provides information on the clinical presentation of psoriasis in the study population. Of the 31 patients, 83.9% had plaque-type psoriasis, 9.7% had guttate type, 3.2% had pustular type, and 3.2% had an erythrodermic type. This indicates that plaque-type psoriasis was the most common psoriasis in the study population.

Table 3.3 also presents the mean Psoriasis Area Severity Index (PASI) score for the patient group, which was 19.6 (±12.7). Based on the PASI scoring system, the patients were classified into three groups: mild (12.3%), moderate (19.4%), and severe (67.7%) disease.

Table 3.4 further examines the relationship between psoriasis severity and the serum levels of 12-HETE and 13-HODE, two inflammatory markers previously found to be elevated in psoriatic patients. The results indicate that there were statistically significant differences in the serum levels of these markers between the three psoriatic severity groups ($P < 0.05$). Specifically, patients with more severe disease had higher levels of 12-HETE and 13-HODE, further supporting the association between psoriasis severity and inflammation.

### Correlation of the Serum 12-HETE and 13-HODE Levels and Psoriasis Severity:

The study's results also revealed a strong positive correlation between 12-HETE levels and psoriasis severity as assessed by the Psoriasis Area Severity Index (PASI) score, with a correlation coefficient (r) of 0.86 and a p-value of 0.001, as shown in Figure 3.1. Similarly, 13-HODE levels were found to be strongly correlated with psoriasis severity, with a correlation coefficient of 0.87 and a p-value of 0.001, as shown in Figure 3.2.

The results indicate that the mean levels of both 12-HETE and 13-HODE were higher in lesional skin as compared to non-lesional skin, and this difference was statistically significant (p-value < 0.05).

## Discussion

Psoriasis severity categories are essential tools for clinicians to use in treatment decisions and Establish the specific requirements for eligibility in clinical research. Nevertheless, due to the diversity of severity classifications and their failure to account for the influence of psoriasis involvement in specific areas or previous treatment history, patients may be incorrectly categorised, leading to uncertainty and unfairness in accessing systemic medication.[9,10]

The skin primarily stores bioactive lipid mediators derived from omega-6 and omega-3 polyunsaturated fatty acids (PUFAs). The human epidermis contains a high concentration of linoleic (LA) and arachidonic (AA) acids, which are the most prevalent omega-6 polyunsaturated fatty acids (PUFAs). Several studies identify different arrays of Lipid mediators and their role in the pathogenesis of the disease[11]. The most abundant oxylipins detected in the skin of psoriasis patients were mono hydroxy derivatives from AA (5-, 8-, 9-, 11-, 12-, and 15-HETE) and LA (9- and 13-HODE) but few studies identify the severity association of these lipid mediator with psoriasis[12]. So, this study aims at identifying two different form of lipid mediators, the pro-inflammatory 12 HETE derivative of Arachidonic acid and the anti-inflammatory 13 HODE with measurement of their concentration in both skin biopsy and blood level with suitable convenience methods and further correlate the level of these lipid mediators with severity of psoriasis estimated by PSAI score.

The study found that the serum level of 13 HODE was statistically significantly higher among patients with psoriasis compared to the apparently healthy control. Although 13 HODE is considered anti-inflammatory and maintains normal keratinocyte proliferation[13], this finding is consistent with Sorokin et al. [2], who found a higher level of 13 HODE in the psoriatic patient in comparison to a healthy individual. this is further supported by Markworth et al., who conclude that supplementation of patients with intermediary long chain omega-3 docosapentaenoic acid reverses the accumulation of 9- and 13- HODE and its oxidized products[14]. The study also demonstrates a significant increment in the serum level of 13 HODE with increasing severity of psoriasis measured by PSAI score. This correlation

concorded with Wojcik et al., who found a high level of 13 HODE with severe and systemic disease [15]. When comparing the skin level of 13 HODE in the lesion and non-lesion area, it was found that the 13 HODE measured from the psoriatic lesion were higher than the non-lesion area. Despite the evidence demonstrating 13 HODE as anti-inflammatory [13], The amount of 13 HODE produced by the rapidly dividing psoriatic skin is not enough to inhibit the excessive growth of psoriatic lesions. Furthermore, the levels of oxidised versions of these lipid mediators were notably increased in psoriasis-affected skin, with a prominent abundance of 13 HODE [16]. This could be attributed to the evidence found that the activation of 15 lipoxygenases is greater in psoriasis and particularly in psoriatic scale samples than non-psoriatic [17].

The arachidonic acid pathway plays the other role in psoriasis. The study found that markers of the arachidonic acid pathway also accumulate in high concentrations in lesional psoriasis. 12 HETE is a potent chemoattractant with pro-inflammatory function. In contrast, 15 HETE reduce inflammatory cell infiltration[18]. Both these derivatives are present in different concentrations. This study demonstrates a significantly high level of 12-HETE in lesional psoriatic lesion compared to the normal skin. This finding concordance other studies that found a high level of 12 in lesional psoriatic skin compared to normal skin[2][19]. This was an essential role in the pathophysiology of psoriasis where Arenberger and his colleague found a decrease in 12 HETE receptors number in psoriatic cell with consequent diminish 12 HETE uptake[20].

Similarly, our study demonstrates a highly significant level of 12 HETE compared to the apparently healthy control group. This finding is consistent with Sorokin et al., which show a similar high pro inflammatory marker in particular 12 HETE in psoriatic patient compared with normal. Furthermore, 12 HETE origin in psoriatic skin is not only dermis but also infiltrated neutrophils and this support the finding of high 12 HETE in circulation blood[21]. Although Takeichi etal no correlation with the severity of psoriasis measured by PSAI score and attributed that to the various treatment effect. Our study found a significant positive correlation between the severity of psoriasis measured by PSAI score and 12 HETE level.

It is important to note that while these findings provide important insights into the relationship between inflammation and psoriasis severity, further studies are needed to confirm these results and to explore the potential utility of these markers in clinical practice. Additionally, the study's relatively small sample size may limit the generalizability of the findings, and larger studies are needed to further validate these results.

This study also demonstrated a significant high level of 12-HETE in psoriatic lesion compared to the non lesional skin. This finding was in concordance to another study that found a high

level of 12-HETE in lesional psoriatic skin compared to normal skin[16,19]. This was found to be of an essential role in the pathophysiology of psoriasis[20].

When comparing the skin levels of 13-HODE in lesional and non-lesional areas, it was found that the levels of 13-HODE in psoriatic lesional skin were higher than in non-lesion areas. Despite the evidence that demonstrated 13-HODE as anti-inflammatory mediator [13], the high level of 13-HODE generated by psoriatic epidermis seem to be insufficient to suppress the hyperproliferation state of psoriatic lesions [22].

These findings suggest that both 12-HETE and 13-HODE may be involved in the pathogenesis of psoriasis, and that targeting these inflammatory markers may be an effective approach to treatment. Additionally, these results highlight the importance of examining both lesional and non-lesional skin samples to better understand the underlying pathophysiology of psoriasis.

In conclusion, this study reports a marked elevation in the levels of lipid mediators, specifically 12-hydroxyeicosatetraenoic acid (12-HETE) and 13-hydroxy octadecadienoic acid (13-HODE), in the serum of patients diagnosed with psoriasis. These increases were positively correlated with the severity of psoriatic disease, as determined by the Psoriasis Area and Severity Index (PASI). Additionally, the levels of 12-HETE and 13-HODE were significantly higher in lesional skin than in non-lesional skin in individuals with psoriasis. These findings suggest that lipid mediators may play a role in the pathogenesis of psoriasis and may serve as potential therapeutic targets for this condition.

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Abbreviations

| | |
|---|---|
| 12-HETE | 12-hydroxyeicosatetraenoic acid |
| 13-HODE | 13-hydroxy octadecadienoic acid |
| PASI | Psoriasis Area and Severity Index |
| IFN | Interferon |
| IL-17 | Interleukin 17 |
| ROS | Reactive Oxygen Species |
| PUFAs | Polyunsaturated Fatty Acids |
| LOX | Lipoxygenases |
| ALA | Alpha Linolenic acid |
| PBS | Phosphate Buffer Saline |
| BMI | Body mass index |
| HDLc | High Lipoprotien Cholesterol |
| LDLc | Low Density Lipoprotein Cholesterol |
| ANOVA | One-Way Analysis Of Variance |
| LA | Linoleic Acid |
| AA | Arachidonic Acid |

## Declarations

### Ethical Approval

The patients provided written informed consent to participate in this investigation. The study adheres to the 1995 Helsinki declaration and received approval from the Scientific Council of dermatology, Arab Board of Health Specialisations.

### Competing interests

The author declares that there is no conflict of interest.

### Authors' contributions

All authors participate in writing the main manuscript text, Al-Ashoor ZA, Al-Hamdi KI. actively involved in the preparation of patients, and determination of PSAI severity index. in addition, Al-Ashoor ZA, and Haddad NS engaged in the blood sample and tissue collection and tissue preparation. Haddad NS actively engage in laboratory analysis and statistical analysis of the results. All the authors reviewed the manuscript

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### Availability of data and materials

All data are available on request from the corresponding author on reasonable reasons.

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**Table 3.1** Demographic Distribution of the study population

| Parameter | | Cases number (31) | Controls number (20) | P value * |
|---|---|---|---|---|
| Age(years) | | 29 (±14.09) | 34.1 (±11.1) | 0.47 |
| Gender | Male | 19(61.3%) | 12(60%) | 0.94 |
| | Female | 12(38.7%) | 8(40%) | |
| Weight (Kg) | | 68.06 (±7.5) | 67.1 (±5.5) | 0.29 |
| Height ($m^2$) | | 1.64(±0.09) | 1.63(±0.1) | 0.74 |
| BMI (kg/ $m^2$) | | 25.1(±2.5) | 25.4(±1.8) | 0.36 |

Values are expressed as mean ± SD.
* p value <0.05 consider statistically significant.

**Table 3.2** Characteristics of Biochemical parameters in study population

| parameters | Patients Number (31) | Control Number (20) | P value* |
|---|---|---|---|
| Cholesterol (mg/dl) | 162.5 (±28.9) | 147(±23.8) | 0.547 |
| Triglyceride (mg/dl) | 71.2 (±40.4) | 62.9 (±35.8) | 0.742 |
| HDL (mg/dl) | 36.6 (±8.1) | 35.5 (±5.18) | 0.158 |
| LDL (mg/dl) | 110 (±30.2) | 88.9 (±25.1) | 0.575 |
| 12-HETE (ng/ml) | 5.9(±2.15) | 2.05(±0.56) | 0.001 |
| 13-HODE (ng/ml) | 171.4(±272.9) | 3.07(±1.13) | 0.001 |

Values are expressed as mean ± SD.
*p value <0.05 consider statistically significant.

**Table 3.3** Demographic Characteristics of psoriatic patients

| Characteristic | | Number | % | Mean ± SD |
|---|---|---|---|---|
| Duration of disease(years) | | 31 | | 6.02(±5.2) |
| Types of psoriasis | Plaque | 26 | 83.9% | -------- |
| | Pustular | 1 | 3.2% | -------- |
| | Guttate | 3 | 9.7% | -------- |
| | Erythrodermic | 1 | 3.2% | -------- |
| PASI Score | | 31 | | 19.6(±12.7) |
| PASI Score Categories | Mild (<7) | 4 | 12.3% | -------- |
| | Moderate (7-15) | 6 | 19.4% | -------- |
| | Sever (>15) | 21 | 67.7% | -------- |

Values are expressed as mean ± SD or number (%)

**Table 3.4** Comparison of Lipid Mediators Levels Between Psoriatic Severity Groups

| Psoriatic Severity / Lipid Groups Mediators Levels | Psoriatic Severity Groups according to PASI score | | | |
|---|---|---|---|---|
| | Mild (<7) Number =4 | Moderate (7-15) Number =6 | Sever (>15) Number =21 | *P value |
| 12-HETE(ng/ml) | 3.45 (±0.40) | 4.99 (±0.75) | 6.76 (±2.07) | 0.002 |
| 13-HODE(ng/ml) | 3.43 (±2.13) | 22.49 (±5.22) | 247.74 (±228.73) | 0.04 |

Values are presented as mean± standard deviation.
*p value <0.05 consider statistically significant.

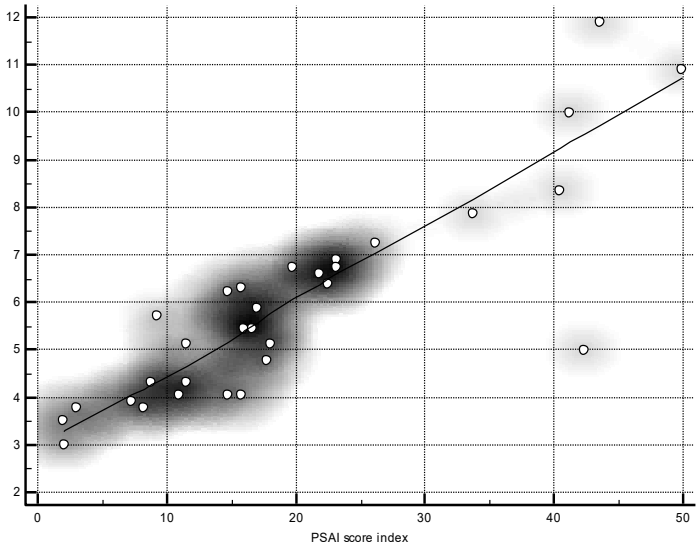


**Fig 3.1** Scatter Diagram showing the correlation between 12-HETE and PSAI score among the psoriatic patients.

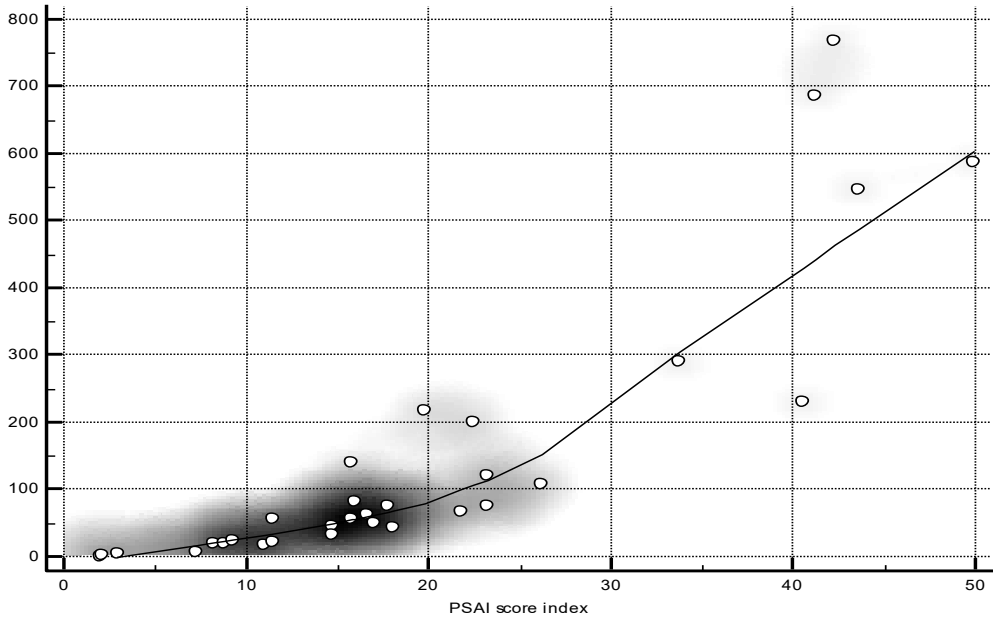


**Fig 3.2** Scatter Diagram showing the correlation between 13-HODE and PSAI score among the psoriatic patients.