

## Detection of Gelsolin in Serum in Patients with Psoriatic Arthritis and its Correlation with Disease Activity

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### Abstract:

**Background:** Psoriatic arthritis (PsA) is an inflammatory disorder that has effects on the joints of psoriatic patients. Plasma gelsolin levels were studied in many inflammatory and autoimmune disorders. The aim of this study was to investigate the potential role of gelsolin in PsA and to determine the relation of gelsolin to PsA disease activity. **Methods:** This case control study was carried out on 60 subjects, both sexes, aged above 16 years old who were classified into Group (1) which included 30 patients diagnosed with PsA according to CASPER criteria, and Group (2) which included 30 healthy matched controls in age and sex. Examination of the skin and all body joints was performed on all psoriatic patients. Gelsolin serum level was also assessed. **Results:** Gelsolin levels show significant negative correlations with CRP, ESR, disease duration, NTJ, NSJ, pain, disease activity, DAPSA score ( $p < 0.05$ ). In univariable analysis, high CRP, ESR, duration, NTJ, NSJ, pain, activity and lower gelsolin are associated with higher DAPSA. In multivariate analysis, several variables show significant associations with DAPSA. CRP, NTJ, NSJ, pain score, and disease activity in the last week have positive coefficients, indicating that higher values in these variables are associated with higher DAPSA scores. **Conclusions:** PsA patients have a reduction in the levels of plasma gelsolin. This suggests that gelsolin may be implicated in the chronic joints inflammation process of PsA. Plasma gelsolin seems to be a useful predictive biomarker for diagnosing PsA and monitoring the disease activity.

**Keywords:** Gelsolin, Psoriatic Arthritis, Disease Activity

## Introduction

Psoriatic arthritis (PsA) is an inflammatory disorder that has effects on the joints of psoriatic patients with underlying immunological mechanism. PsA has a global prevalence of about 3%. PsA affects nearly 30% of psoriatic patients. It is characterized by a wide assortment of joints manifestations, including peripheral arthritis, axial arthritis, dactylitis, and enthesitis. PsA is generally associated with nail involvement manifestations. PsA has negative effects on patient's life quality and survival <sup>(1)</sup>.

Gelsolin is a protein of gelsolin superfamily, encoded on human chromosome 9. The gelsolin molecular weight is nearly 82–84 kDa. It has 6 homologous domains. Gelsolin regulated the actin assembly and disassembly by calcium dependent manner. Two isoforms of gelsolin were detected (cytoplasmic and plasma isoforms). Plasma gelsolin scavenges the circulating actin, while the intracellular gelsolin has a role in maintaining the cellular shape and motility besides a role in the apoptosis process <sup>(2)</sup>.

Plasma gelsolin prevents the polymerization of actin to be easily removed by the liver. Many factors including pH, calcium levels, phosphoinositides concentration, and temperature regulate the gelsolin action. Gelsolin is involved in the immune response and considered an anti-inflammatory modulator. The depletion of gelsolin causes damage in the immune cells with subsequent inflammatory mediator release <sup>(3)</sup>.

Plasma gelsolin levels' reduction was first detected in tissues damaged by traumas. Then, its reduction has been associated with sepsis, organs failure, chronic inflammatory disorders, and cancers. This reduction is explained by many theories as "lower production, redistribution to the inflammation site, combined with other plasma proteins, or higher degradation". As gelsolin can counteract the sequel of actin release during tissue injury,

extracellular recombinant gelsolin seems to be a possible therapeutic utility <sup>(4)</sup>.

Plasma gelsolin levels were studied in many inflammatory and autoimmune disorders (like rheumatoid arthritis). So, this study aims to investigate the potential role of gelsolin in PsA and to determine the association between gelsolin and the disease activity.

The aim of this work was to investigate the potential role of gelsolin in PsA and to determine the relation of gelsolin to PsA disease activity.

## Patients and Methods:

This case control study was carried out on 60 subjects, both sexes, aged above 16 years old who were classified into Group (1) which included 30 patients diagnosed with PsA according to CASPER criteria <sup>(34)</sup> recruited from inpatients and outpatient's clinics of rheumatology department of Benha University Hospitals, and Group (2) which included 30 healthy matched controls in age and sex. Patients were recruited from inpatients and outpatient's clinics of rheumatology department of Benha University Hospitals in period between September 2023 and May 2024.

An informed written consent was obtained from the patients. The study was done after approval from the Ethical Committee Benha University Hospitals

### Approval Code: MD 8-5-2022

**CASPER criteria** include evidence of psoriasis (current, past, or family) (two points if current history of psoriasis, one point others), psoriatic nail dystrophy (one point), negative rheumatoid factor (one point), dactylitis (current or past history) (one point), and radiographic evidence of juxta articular new bone formation (one point). Three or more points have 99% specificity and 92% sensitivity for diagnosis of PsA <sup>(34)</sup>.

**Exclusion criteria** were Age < 16 years, pregnant patients, other systemic autoimmune disorders, traumatic arthritis, septic arthritis, viral arthritis, gouty

arthritis, serious active medical illness, a previous diagnosis of inflammatory arthritis, and fibromyalgia (in whom tender points could be misdiagnosed as enthesitis).

All psoriatic patients were subjected to: Personal history, Complaint and its duration, and present history [Onset, course, duration of complaint, Constitutional symptoms (fatigue, weight loss, fever, anorexia), symptoms suggestive other autoimmune disease and review of other systems, musculoskeletal manifestations: pain, swelling, joint involved, muscle weakness], present medications, family history and obstetric history. Symptoms suggestive of other rheumatological diseases, full dermatological history, and symptoms suggestive of other system involvement were also assessed.

Clinical examination included general examination such as vital signs, head and neck, mucocutaneous, examination of the eyes, cardiac examination, pulmonary examination, complete abdominal examination, and neurological examination.

**Examination of the skin (for psoriasis):**

For presence of psoriasis lesions, distribution

**All body joints were examined** thoroughly according to inspection, palpation, movements, and special tests such as local examination to detect psoriatic plaques, dactylitis, enthesitis, uveitis, tender or swollen joints count and clinical assessment of disease activity in PSA patients. Disease Activity Index for PSA (DAPSA) score of 5-14 represents low disease activity, score 15-28 represent moderate disease activity, score > 28 represent high disease activity and a score of < 4 represents remission.

**Routine laboratory investigations** including CBC, UREA, creatinine, ESR and CRP were also performed.

**Assessment of Gelsolin serum level**

Gelsolin serum level was measured in plasma by a sandwich enzyme linked

immunosorbant assay (ELIZA) kit. Catalogue No. 201-12-1234.

**Assay range:** 3µg/ml→700µg/ml

**Sample Size Calculation:**

The sample size and power analysis were calculated using Epi-Info software statistical package created by World Health organization and center for Disease Control and Prevention, Atlanta, Georgia, USA version 2002. At 95% confidence limit. The sensitivity of gelsolin in differentiating between high/very high disease activity grade from low/moderate grades RA is 78.1% with a margin of error of 10% (60-80%). The sample size was found at N=60.

**Statistical analysis**

The collected data was analyzed using Statistical package for Social Science (IBM Corp. Released 2017. IBM SPSS Statistics for Windows, Version 25.0. Armonk, NY: IBM Corp.). Shapiro-Wilk test was done to test the normality of data distribution. Mean, standard deviation ( $\pm$  SD), median, and range were used for numerical data. Frequency and percentage were used for non-numerical data. Student T Test was used to assess the statistical significance of the difference between two study group means. Mann Whitney Test (U test) was used to assess the statistical significance of the difference of a non-parametric variable between two study groups. The Kruskal Wallis test was used to assess the statistical significance of the difference between more than two study group nonparametric variables. Chi-Square test was used to examine the relationship between two qualitative variables. Fisher Exact test was used to examine the relationship between two qualitative variables when the expected count is less than 5 in more than 20% of cells. Correlation analysis was performed to assess the strength of association between two quantitative variables. The ROC Curve (receiver operating characteristic) provides a useful way to evaluate the sensitivity and specificity for quantitative diagnostic measures that categorize cases

into one of two groups. Logistic regression analysis was used for the prediction of risk factors when the outcome is binary. An odds ratio (OR) is a measure of association between an exposure and an outcome. Linear regression analysis was used for the prediction of risk factors when the outcome is linear. If the p-value for a variable is  $<0.05$ , data provide enough evidence of correlation, and changes in the independent variable are associated with changes in the response. A p value is considered significant if  $<0.05$  at confidence interval 95%

### Results:

The average disease duration among these patients is 8.40 years, and ranged from 2 to 20 years

Patients with PsA have median of 6 tender joints and 2 swollen joints. Patients with PsA report a mean pain score of 6.2 and a mean disease activity score of 6.57 in the last week. The average disease activity, measured by the DAPSA score, is 33.21, with median of 31, and ranged from 6 to 81.7. The majority of patients (56.7%) have a high disease activity level, while 23.3% have medium disease activity and 20.0% have low disease activity.

The mean gelsolin level in the PsA group is significantly lower than in the control group ( $p < 0.001$ ). The average gelsolin level in the PsA group is 54.88 mg/L, while in the control group it is 118.0 mg/L (median=48.3, 117.7, respectively). **Table 1**

Gelsolin levels show significant negative correlations with CRP ( $p < 0.001$ ), ESR ( $p = 0.002$ ), disease duration ( $p < 0.001$ ), NTJ ( $p < 0.001$ ), NSJ ( $p < 0.001$ ), pain ( $p < 0.001$ ), disease activity ( $p < 0.001$ ), DAPSA score ( $p < 0.001$ ). However, there is no significant correlation between gelsolin levels with age and RF levels ( $p > 0.05$ ). The negative correlation

coefficients indicate that higher gelsolin levels are associated with lower values in the mentioned parameters. **Table 2**

Regarding the validity of gelsolin in predicting high DAPSA from low and medium, the AUC is 0.869, indicating good predictive accuracy. The cutoff value of  $\leq 52.4$  mg/L yields a sensitivity of 94.12%, specificity of 76.92%, PPV of 84.21%, NPV of 90.91%, and accuracy of 86.67%. **Figure 1A**

Regarding the validity of gelsolin in predicting medium and high DAPSA from low DAPSA among patients with PsA, the AUC is 0.993, indicating excellent predictive ability. The cutoff value of  $\leq 60.1$  mg/L provides a sensitivity of 95.83%, specificity of 100.0%, PPV of 100.0%, NPV of 85.70%, and accuracy of 96.66%. **Figure 1B**

High ESR, CRP, RF and low Gelsolin were associated with risk of PsA in univariable analysis. However, in the multivariate analysis, only lower levels of gelsolin is a significant predictor of PsA susceptibility.

### Table 3

In univariable analysis, high CRP, ESR, duration, NTJ, NSJ, pain, activity and lower gelsolin are associated with higher DAPSA. While in the multivariate analysis, several variables show significant associations with DAPSA. CRP, NTJ, NSJ, pain score, and disease activity in the last week have positive coefficients, indicating that higher values in these variables are associated with higher DAPSA scores. On the other hand, gelsolin has a negative coefficient of -0.027 ( $p < 0.001$ ), suggesting that lower gelsolin levels are associated with higher DAPSA scores. ESR and disease duration show significant associations with DAPSA in the univariate analysis, but their associations become non-significant in the multivariate analysis. **Table 4**

**Table 1:** Comparison between patients with PsA and control group regarding gelsolin

	<b>PsA n = 30</b>	<b>Control n = 30</b>	<b>Test</b>	<b>p</b>
<b>Gelsolin (mg/L)</b>				
Mean $\pm$ SD.	54.88 $\pm$ 22.17	118.0 $\pm$ 23.50	U=	<0.001*
Median	48.30	117.7	861.0*	
Min. – Max.	30.50 – 120.4	59.70 – 152.4		

Data are presented as mean  $\pm$  SD, median, Min. and Max. Min.: Minimum, Max.: Maximum, U: Mann Whitney test. p: Comparing patients and control, \*: Significant when p value <0.05

**Table 2:** Correlation between gelsolin and different parameters among patients with PsA

	Gelsolin	
	Correlation Coefficient	p
Age	-0.202	0.228
CRP	-0.679*	<0.001*
ESR	-0.546*	0.002*
RF	0.147	0.438
Disease duration	-0.739*	<0.001*
NTJ	-0.762*	<0.001*
NSJ	-0.794*	<0.001*
Pain in the last week	-0.747*	<0.001*
Disease activity in the last week	-0.626*	<0.001*
DAPSA	-0.802*	<0.001*

CRP: C-reactive protein, ESR: Erythrocyte sedimentation rate, RF: , NTJ: , NSJ: , DAPSA: , r: Spearman's rho, \*: Significant when p value <0.05.

**Table 3:** Logistic regression analysis for prediction of PsA susceptibility.

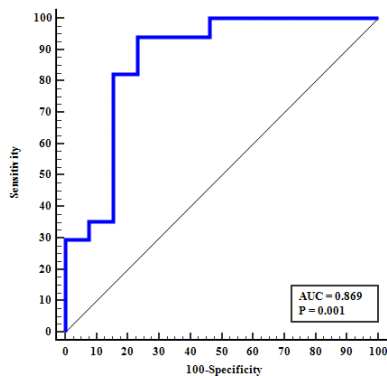
	<b>Univariate</b>			<b>Multivariate</b>		
	<b>P</b>	<b>OR</b>	<b>95% C.I</b>	<b>P</b>	<b>OR</b>	<b>95% C.I</b>
<b>Gender</b>	0.542	1.264	0.595–2.685			
<b>Age</b>	0.248	1.017	0.988–1.047			
<b>ESR</b>	<0.001*	1.022	1.010-1.034	0.166	1.016	0.993-1.039
<b>CRP</b>	0.002*	1.219	1.073-1.384	0.758	1.989	1.916-2.058
<b>RF</b>	0.012*	1.108	1.023–1.200	0.443	1.018	0.967-1.059
<b>Gelsolin</b>	<0.001*	0.956	0.940–0.973	0.038*	0.981	0.959-0.997

CRP: C-reactive protein, ESR: Erythrocyte sedimentation rate, RF: OR: Odd Ratio, CI: confidence interval. OR<1, protective; OR>1, risky; \*: Significant when p value <0.05.

**Table 4:** Linear regression analysis for prediction of DAPSA among patients with PsA.

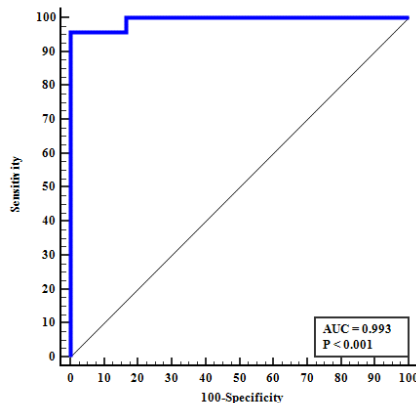
	<b>Univariate</b>		<b>Multivariate</b>	
	<b>B</b>	<b>p</b>	<b>B</b>	<b>p</b>
<b>Gender</b>	2.796	0.350		
<b>Age</b>	1.094	0.263		
<b>ESR</b>	0.730	<0.001*	0.002	0.929
<b>CRP</b>	2.665	<0.001*	1.014	<0.001*
<b>RF</b>	0.178	0.751		
<b>Disease duration</b>	2.053	<0.001*	0.001	0.987
<b>NTJ</b>	2.661	<0.001*	1.043	<0.001*
<b>NSJ</b>	6.081	<0.001*	0.986	<0.001*
<b>Pain score</b>	7.076	<0.001*	1.814	<0.001*
<b>Disease activity in the last week</b>	3.069	0.001*	0.100	<0.001*
<b>Gelsolin</b>	-0.648	<0.001*	-0.027-	<0.001*

CRP: C-reactive protein, ESR: Erythrocyte sedimentation rate, RF: , NTJ: , NSJ: , B, regression coefficient; \*: Significant when p value <0.05.



**Figure 1:** ROC Curve for gelsolin for prediction (A) high DAPSA from low and medium DAPSA, (B) medium and high DAPSA from low DAPSA among patients with PsA

(A)



(B)

## Discussion

About 20% of people with psoriatic arthritis have psoriatic arthritis (PsA), a chronic inflammatory arthritis linked to psoriasis that has many clinical similarities to rheumatoid arthritis and other spondyloarthropathies <sup>(6)</sup>.

Uncertain genetic and environmental variables combine in a complicated way to cause PsA <sup>(7)</sup>.

To measure the severity of psoriasis and the efficacy of treatment, practitioners have mostly employed and depended upon clinical evaluation methods such as the Physician Global evaluation (PGA) and the Psoriasis Area and Severity Index (PASI) <sup>(8)</sup>.

Nonetheless, the degree to which the biomarkers identified thus far are indicative of the disease's activity or severity varies greatly in their usefulness <sup>(9)</sup>.

A member of the gelsolin superfamily is the protein gelsolin. The gelsolin gene,

which is found on human chromosome 9, encodes it. It is a multifunctional, highly conserved actin-binding protein that was initially discovered in the cytosol of macrophages and subsequently found in several other cells. The extracellular secretory isoform of it, known as plasma gelsolin, is found in a number of bodily fluids, including milk, urine, blood, and cerebrospinal fluid <sup>(10)</sup>.

Gelsolin was found in two isoforms: the cytoplasmic and plasma isoforms. While intracellular gelsolin is involved in apoptosis and also maintains cellular shape and motility, plasma gelsolin scavenges circulating actin. Gelsolin is regarded as an anti-inflammatory modulator. The first indication of a drop in plasma gelsolin levels was seen in traumatised tissues. Subsequently, its decrease has been linked to malignancies, organ failure, sepsis, and chronic inflammatory conditions <sup>(11)</sup>.

So, the current study aimed to investigate the potential role of gelsolin in PsA and to

determine the association between gelsolin and the disease activity.

The current study was case control study conducted on 30 patients with PsA recruited from clinics of Banha University Hospitals, Rheumatology and Rehabilitation department, and 30 healthy matched controls.

Regarding the demographics of the current study, there was no significant difference in gender or age between the PsA group and the control group with mean age of psoriatic cases was 41.33 years old.

Which agreed with Menis et al. <sup>(12)</sup>, who examined total of 132 patients diagnosed with PsA according to CASPER criteria and found that they were included 66 men and 66 women. The average age of their population was 54.9 years (54.3 years in men and 55.3 years in women).

The current study revealed that PsA disease duration was 8.40 years ranged from 2 to 20 years. in addition, PsA patients had median of 6 tender joints (NTJ) and 2 swollen joints (NSJ).

In agreement with Gialouri et al. <sup>(13)</sup>, who had examined 281 PsA patients, showed that patients with earlier-onset PsA had median disease duration in months was 54 months ranged from 18–142 months, in addition they had 5 TJ, ranged from 3.0–7.0 and 1 SJ ranged from 2.0–4.0.

The present study found that PsA cases had a median pain score of 6 and a median disease activity score of 6 in the last week. The average disease activity, measured by the DAPSA score, was 33.21. The majority of patients (56.7%) have a high disease activity level, while 23.3% have medium disease activity and 20.0% have low disease activity.

While Becciolini et al. <sup>(14)</sup> had found that the median DAPSA score at baseline in PsA cases was 24.4. In addition, Esawy et al. <sup>(15)</sup> found that PsA patients were grouped according to their PASI scores into mild and moderate to severe categories, it turned out that nearly 76% of patients had mild psoriasis, and 24% of them had moderate to severe psoriasis.

Regarding the gelsolin level in the current study; in the PsA group, it was significantly lower than in the control group. The average gelsolin level in the PsA group was 54.88 mg/L, while in the control group it was 118.0 mg/L.

In same line with Esawy et al. <sup>(15)</sup>, who found that, compared to controls ( $184.9 \pm 18.5$ ), PsA patients had a substantially reduced plasma level of gelsolin ( $148.2 \pm 19.2$ ). According to this study, PsA patients' plasma gelsolin levels were lower than those of psoriasis and healthy controls.

This was in accordance with the study of Osborn et al. <sup>(16,17)</sup>, which found that, in comparison to healthy controls, rheumatoid arthritis patients had lower levels of gelsolin. This is consistent with a prior study on inflammatory joint illnesses (such as SLE and RA), which claims that decreased plasma gelsolin levels play a part <sup>(17)</sup>.

Another study performed on ankylosing spondylitis patients demonstrates that gelsolin levels decreased in those patients <sup>(18)</sup>.

The distribution of plasma gelsolin in inflammatory areas, where it bonded to macromolecules in the inflamed joint and was consumed, may help to explain the decrease in gelsolin levels. Also, this is in keeping with Chong et al. <sup>(19)</sup>, who demonstrated that in comparison to normal skin, the level of Flightless I, another member of the gelsolin superfamily, was higher in psoriatic skin lesions. Thus, the current findings are consistent with earlier research showing that a decrease in plasma gelsolin levels in both acute injury and chronic illnesses <sup>(20, 21)</sup>.

Many disorders may cause the biomarker to be compromised. Therefore, the clinical symptoms must be considered in relation to the marker results. It is challenging to identify subclinical PsA using the conventional methods. This leads to a postponed diagnosis of PsA <sup>(22)</sup>.

In agreement with all previous results, Lee et al. <sup>(23)</sup> who examined serum gelsoline

level in psoriasis vulgaris patients, revealed that serum gelsolin level was significantly lower in the psoriasis group ( $49.03 \pm 27.66$  ng/mL) than in the control group ( $84.21 \pm 40.78$  ng/mL). In those with psoriasis, baseline gelsolin level was significantly lower than that at 16 weeks' post-treatment ( $57.28 \pm 33.27$  ng/mL) and 52 weeks' post-treatment. But the disagreement in same report by Lee et al. <sup>(23,24)</sup> was that there were no significant differences in gelsolin level according to the presence or absence of PsA or nail changes.

Other researchers found that the gelsolin level was significantly lower in RA when compared to healthy subjects <sup>(25, 26)</sup>. While a study found that, in RA patients, there was no significant reduction in gelsolin compared to healthy controls. While in patients with OA, the gelsolin concentration in the synovial fluid was significantly reduced <sup>(27)</sup>.

Furthermore, the serum gelsolin level in patients with ulcerative colitis was found to be significantly lower than that in healthy subjects <sup>(28)</sup>. Moreover, the serum gelsolin levels were found to be decreased in atopic dermatitis <sup>(29)</sup>, primary Sjogren's syndrome <sup>(30)</sup>, acute liver failure and myonecrosis <sup>(31)</sup>.

While the current study revealed a significant difference in gelsolin levels among different grades of DAPSA, that gelsolin levels showed significantly descending level associated with ascending severity grades.

Which in disagreement with, Esawy et al. <sup>(15)</sup>, who revealed that PsA patients were classified according to the psoriasis severity, the plasma gelsolin levels showed no significant difference between mild and moderate to severe patients.

On other side, Gelsolin levels in the current study showed significant negative correlations with CRP, ESR, disease duration, NTJ, NSJ, pain, disease activity and DAPSA score.

In agreement with Esawy et al. <sup>(15)</sup>, who found that Gelsolin was significantly

inversely correlated with both the duration and severity of PsA. In a similar vein, there was a noteworthy inverse relationship between plasma gelsolin and inflammatory markers (CRP and ESR). There was shown to be a substantial inverse relationship between PsA activity and plasma gelsolin. The DAPSA and CPDAS scores served as the activity's definition.

Gama et al. <sup>(32)</sup> also, declared that gelsolin and CRP showed a negative connection in chronic hemodialysis patients.

The current study demonstrated that gelsoline level had excellent discriminative ability. The cutoff value of 65.8 mg/L provided a sensitivity of 83.33%, specificity of 96.67%, positive predictive value (PPV) of 96.16%, negative predictive value (NPV) of 85.29%, and overall accuracy of 90.0% in differentiated between cases with PsA and controls.

ROC curve analysis in Esawy et al. <sup>(15)</sup> study, showed that plasma gelsolin had a strong predictive value for detecting PsA and a significant predictive value for separating PsA from psoriasis. Gelsolin demonstrated 92.1% sensitivity, 95% specificity, 97.2% PPV, and 86.4% NPV at a threshold of 172.5 mg/L in the detection of PsA. However, when it came to separating psoriasis from PsA, it showed 92.1% sensitivity, 80% specificity, 89.7% PPV, and 84.2% NPV.

The current study found that gelsolin had good predictive accuracy in predicting high DAPSA from low and medium DAPSA among patients with PsA. While it had excellent predictive ability in predicting medium and high DAPSA from low DAPSA among patients with PsA.

The current study revealed that the lower levels of gelsolin were considered as a significant predictor of PsA susceptibility. In addition, high CRP, ESR, duration, NTJ, NSJ, pain, activity and lower gelsolin were associated with higher DAPSA.



In agreement with Esawy et al. <sup>(15)</sup> study, who showed a significant predictive value of low plasma gelsolin in PsA detection. While Mulder et al. <sup>(33)</sup> in a systematic review, indicated that a greater level of CRP, which matched ours, was a marker with a strong level of evidence for a favorable correlation with the existence of PsA in Pso.

## Conclusions:

PsA patients have a reduction in the levels of plasma gelsolin. This suggests that gelsolin may be implicated in the chronic joints inflammation process of PsA. Plasma gelsolin seems to be a useful predictive biomarker for diagnosing PsA and monitoring the disease activity.

## References:

- Liu JT, Yeh HM, Liu SY, Chen KT. Psoriatic arthritis: Epidemiology, diagnosis, and treatment. *World J Orthop*. 2014;5:537-43.
- Kolliker Frers RA, Cosentino V, Tau J, Kerzberg EM, Urdapilleta A, Chioconci M, et al. Immune-Mediated Inflammation Promotes Subclinical Atherosclerosis in Recent-Onset Psoriatic Arthritis Patients without Conventional Cardiovascular Risk Factors. *Front Immunol*. 2018;9:139.
- Bagel J, Schwartzman S. Enthesitis and Dactylitis in Psoriatic Disease: A Guide for Dermatologists. *Am J Clin Dermatol*. 2018;19:839-52.
- Ritchlin CT, Colbert RA, Gladman DD. Psoriatic Arthritis. *N Engl J Med*. 2017;376:957-70.
- Fredriksson T, Pettersson U. Oral treatment of pustulosis palmo-plantaris with a new retinoid, Ro 10-9359. *Dermatologica*. 1979;158:60-4.
- Tiwari V, Brent LH. Psoriatic Arthritis. StatPearls. Treasure Island (FL) ineligible companies. Disclosure: Lawrence Brent declares no relevant financial relationships with ineligible companies.: StatPearls Publishing
- Eder L, Haddad A, Rosen CF, Lee KA, Chandran V, Cook R, et al. The Incidence and Risk Factors for Psoriatic Arthritis in Patients With Psoriasis: A Prospective Cohort Study. *Arthritis Rheumatol*. 2016;68:915-23.
- Pourani MR, Abdollahimajd F, Zargari O, Shahidi Dadras M. Soluble biomarkers for diagnosis, monitoring, and therapeutic response assessment in psoriasis. *J Dermatolog Treat*. 2022;33:1967-74.
- Morita A, Tani Y, Matsumoto K, Yamaguchi M, Teshima R, Ohtsuki M. Assessment of serum biomarkers in patients with plaque psoriasis on secukinumab. *J Dermatol*. 2020;47:452-7.
- Sadzyński A, Kurek K, Konończuk T, Zendzian-Piotrowska M. [Gelsolin - variety of structure and functions]. *Postepy Hig Med Dosw (Online)*. 2010;64:303-9.
- Feldt J, Schicht M, Garreis F, Welss J, Schneider UW, Paulsen F. Structure, regulation and related diseases of the actin-binding protein gelsolin. *Expert Rev Mol Med*. 2019;20:e7.
- Menis J, Doussiere M, Touboul E, Barbier V, Sobhy-Danial JM, Fardellone P, et al. Current characteristics of a population of psoriatic arthritis and gender disparities. *J Clin Transl Res*. 2023;9:84-92.
- Gialouri CG, Evangelatos G, Iliopoulos A, Tektonidou MG, Sfrikakis PP, Fragoulis GE, et al. Late-Onset Psoriatic Arthritis: Are There Any Distinct Characteristics? A Retrospective Cohort Data Analysis. *Life (Basel)*. 2023;13.
- Becciolini A, Parisi S, Del Medico P, Farina A, Visalli E, Molica Colella AB, et al. Predictors of DAPSA Response in Psoriatic Arthritis Patients Treated with Apremilast in a Retrospective Observational Multi-Centric Study. *Biomedicine*. 2023;11:433.
- Esawy MM, Makram WK, Albalat W, Shabana MA. Plasma gelsolin levels in patients with psoriatic arthritis: a possible novel marker. *Clin Rheumatol*. 2020;39:1881-8.
- Osborn TM, Verdrengh M, Stossel TP, Tarkowski A, Bokarewa M. Decreased levels of the gelsolin plasma isoform in patients with rheumatoid arthritis. *Arthritis Res Ther*. 2008;10:R117.
- Hu Y, Li H, Li WH, Meng HX, Fan YZ, Li WJ, et al. The value of decreased plasma gelsolin levels in patients with systemic lupus erythematosus and rheumatoid arthritis in diagnosis and disease activity evaluation. *Lupus*. 2013;22:1455-61.
- Genre F, López-Mejías R, Miranda-Fillooy JA, Ubilla B, Carnero-López B, Gómez-Acebo I, et al. Gelsolin levels are decreased in ankylosing spondylitis patients undergoing anti-TNF-alpha therapy. *Clin Exp Rheumatol*. 2014;32:218-24.
- Chong HT, Yang GN, Sidhu S, Ibbetson J, Kopecki Z, Cowin AJ. Reducing Flightless I expression decreases severity of psoriasis in an imiquimod-induced murine model of psoriasiform dermatitis. *Br J Dermatol*. 2017;176:705-12.
- Holm FS, Sivapalan P, Seersholm N, Itenov TS, Christensen PH, Jensen JS. Acute Lung

- Injury in Critically Ill Patients: Actin-Scavenger Gelsolin Signals Prolonged Respiratory Failure. *Shock*. 2019;52:370-7.
21. Chen Z, Li K, Yin X, Li H, Li Y, Zhang Q, et al. Lower Expression of Gelsolin in Colon Cancer and Its Diagnostic Value in Colon Cancer Patients. *J Cancer*. 2019;10:1288-96.
  22. Diani M, Perego S, Sansoni V, Bertino L, Gomarasca M, Faraldi M, et al. Differences in Osteoimmunological Biomarkers Predictive of Psoriatic Arthritis among a Large Italian Cohort of Psoriatic Patients. *Int J Mol Sci*. 2019;20.
  23. Lee SH, Park YL, Bae Y. Gelsolin as a Potential Clinical Biomarker in Psoriasis Vulgaris. *J Clin Med*. 2023;12.
  24. Mosaad GM, Abdel moneam SM, Soliman AF, Ameen SG, Amer AS. Implication of plasma gelsolin in systemic lupus erythematosus patients. *Egypt Rheumatol Rehabil*. 2022;49:1.
  25. Mun S, Lee J, Lim M-K, Lee Y-R, Ihm C, Lee SH, et al. Development of a Novel Diagnostic Biomarker Set for Rheumatoid Arthritis Using a Proteomics Approach. *Biomed Res Int*. 2018;2018:7490723.
  26. Abd MY, Al-Sharifi ZAAR, Jasim NAL. Role of Gelsolin serum levels in Rheumatoid arthritis in Iraqi patients. *Int J Spec Educ*. 2022;37:5580-5.
  27. Feldt J, Schicht M, Welss J, Gelse K, Sesselmann S, Tsokos M, et al. Production and Secretion of Gelsolin by Both Human Macrophage- and Fibroblast-like Synoviocytes and GSN Modulation in the Synovial Fluid of Patients with Various Forms of Arthritis. *Biomedicines*. 2022;10.
  28. Maeda K, Nakamura M, Yamamura T, Sawada T, Ishikawa E, Oishi A, et al. Gelsolin as a Potential Biomarker for Endoscopic Activity and Mucosal Healing in Ulcerative Colitis. *Biomedicines*. 2022;10:872.
  29. Eke Gungor H, Sahiner UM, Karakukcu C, Sahiner N, Altuner Torun Y. The plasma gelsolin levels in atopic dermatitis: Effect of atopy and disease severity. *Allergol Immunopathol (Madr)*. 2016;44:221-5.
  30. Huang H, Song WQ, Li Y. The gelsolin level in patients with primary Sjogren's syndrome. *Eur Rev Med Pharmacol Sci*. 2021;25:2072-8.
  31. Suhler E, Lin W, Yin HL, Lee WM. Decreased plasma gelsolin concentrations in acute liver failure, myocardial infarction, septic shock, and myonecrosis. *Crit Care Med*. 1997;25:594-8.
  32. Flores Gama C, Rosales LM, Ouellet G, Dou Y, Thijssen S, Usvyat L, et al. Plasma gelsolin and its association with mortality and hospitalization in chronic hemodialysis patients. *Blood Purification*. 2017;43:210-7.
  33. Mulder MLM, van Hal TW, Wenink MH, Koenen H, van den Hoogen FHJ, de Jong E, et al. Clinical, laboratory, and genetic markers for the development or presence of psoriatic arthritis in psoriasis patients: a systematic review. *Arthritis Res Ther*. 2021;23:168.
  34. Taylor W, Gladman D, Helliwell P, et al. Classification Criteria for psoriatic Arthritis. development of new Criteria from a large in ternatural study. *Arthritis Rheum* 2006: 2665-73

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