Psoriasis: Targets and Therapy

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ORIGINAL RESEARCH

Bidirectional Inflammatory Mechanisms and Hematological/Coagulation Dysregulation in Psoriasis Complicated with Atherosclerosis: A Retrospective Hospital-Based Case-Control Study Leveraging the SH-YIMED Clinical Database

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Background: Psoriasis is a chronic inflammatory skin disease associated with systemic inflammation, abnormal hemorheology, and coagulation dysfunction, all of which contribute to the development of atherosclerosis (AS). Inflammation and coagulation are closely intertwined processes; however, studies on the specific profiles of hemorheological and coagulation indices and their synergistic mechanisms remain limited.

Objective: To compare differences in hemorheological and coagulation parameters and preliminarily elucidate the regulatory mechanism of the *inflammation-hemorheology-coagulation axis*.

Methods: A total of 439 patients with psoriasis and 198 controls were enrolled; after PSM, 206 cases and 126 controls were included. All participants underwent vascular ultrasonography between May 2014 and February 2025. Patients were divided into the case group (psoriasis with AS) and control group (psoriasis without AS) based on vascular ultrasound and clinical assessment by specialized clinicians. PSM was used to balance baseline confounders, and sample sizes varied due to missing data. Statistical analyses included Spearman correlation and multivariate logistic regression.

Results: The case group had higher fibrinogen (FIB) levels (3.54 vs. 3.11, $P = 0.005$, Cohen's d = 0.163) and shorter activated partial thromboplastin time (APTT) (26.40 vs. 27.60 s, $P = 0.002$, Cohen's d = 0.181). FIB showed the strongest association, with a correlation coefficient of 0.56 ($P < 0.001$) for systemic immune inflammation index (SII) and 0.39 ($P < 0.001$) for neutrophil-to-lymphocyte ratio (NLR) in the case group, and there was a strong positive correlation between SII and FIB ($r = 0.56$, $P < 0.001$). Psoriasis inflammation directly promoted vascular inflammation with a large effect size ($\beta = 0.45$, $P < 0.001$). Multivariate logistic regression analysis showed WBV (whole blood viscosity) (1 mPa·s) was the strongest predictor, with an OR of 1.752 (95% confidence interval [CI]: 1.314–2.651, $P < 0.001$).

Limitations: Selection and recall biases of retrospective single-center studies.

Conclusion: Patients with psoriasis, particularly those with severe disease, long disease duration, or complications from other thrombotic factors, should be closely monitored for hemorheological and coagulation parameters to prevent cardiovascular disease occurrence.

Keywords: psoriasis, atherosclerosis, hematological, coagulation, inflammation-hemorheology-coagulation axis

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Introduction

Psoriasis is a common immune-mediated skin disorder that affects approximately 2–4% of the global population.[1] Cumulative evidence has confirmed that psoriasis is associated with a higher prevalence of cardiovascular risk factors such as atherosclerosis (AS), hypertension, dyslipidemia, obesity, and metabolic syndrome.[2–5]

Numerous large-sample epidemiological studies have confirmed that blood fluidity, plasma viscosity (PV), and hematocrit are independent risk factors for cardiovascular events.[6–9] Hypercoagulability is a potential mechanistic link underlying the association between cardiovascular disease (CVD) and psoriasis. Notably, there is growing recognition that inflammation and coagulation are interconnected biological processes.[10,11]

Persistent inflammation in psoriasis may induce hemorheological abnormalities and inflammatory pathway activation, while concurrently activating the coagulation system and suppressing fibrinolytic function, which ultimately synergistically accelerates the occurrence and progression of AS.[12,13] Systemic immune inflammation index (SII) and neutrophil-to-lymphocyte ratio (NLR) are well-established markers of systemic inflammation. However, research on the specific characteristics of hemorheological and coagulation indices and their synergistic mechanisms of action remains limited.

This study focused on patients with psoriasis complicated by AS, compared the differences in hemorheological and coagulation parameters, and preliminarily elucidated the regulatory mechanism of the *inflammation-hemorheology-coagulation axis*.

Methods

In accordance with the STROBE Statement for Observational Study Reporting, this study followed all the recommended guidelines, and the associated case–control study checklist is provided in Table S1.

Study Design

This study was approved by the Institutional Review Board of Yueyang Hospital of Integrated Traditional Chinese and Western Medicine, Shanghai University of Traditional Chinese Medicine (Approval No. 2024–034). All procedures were performed in accordance with the principles of the Declaration of Helsinki. The requirement for informed consent was waived due to the retrospective nature of the study. All collected datasets encompassing the vascular ultrasound findings were anonymized according to institutional medical record regulations.

This was a single-center, retrospective, case-control study that incorporated propensity score matching (PSM). A retrospective extraction and review of medical records was performed for inpatients with plaque psoriasis at our tertiary referral hospital from May 2014 to February 2025 using a standardized Clinical Data Retrieval System. The enrolled patients were divided into a case group (psoriasis combined with AS, defined as the comorbid group) and a control group (psoriasis without AS, defined as the simple psoriasis group) according to the vascular ultrasound results and evaluation by specialized clinicians.

Study Inclusion and Exclusion Criteria

For the case group, the enrollment criteria were defined as concurrent adherence to the diagnostic standards outlined in the *2023 Full Version of Chinese Guidelines for Psoriasis Diagnosis and Treatment*[14] and the *Chinese Guidelines for Cardiovascular Disease Prevention*,[15] corroborated by comprehensive clinical, pathological, and imaging evidence. For the control group, eligible patients were those who met only the diagnostic criteria for psoriasis, were free of AS and other CVDs, and had unremarkable findings on corresponding diagnostic tests. Additionally, patients in both groups were required to satisfy the following inclusion criteria: (1) availability of complete, retrievable routine blood test results obtained within 3 months before enrollment; (2) age ranging from 18 to 75 years; and (3) completion of a vascular ultrasound examination at our hospital.

The exclusion criteria were as follows: (1) comorbidity with malignant tumors, decompensated liver cirrhosis, uremia, other autoimmune diseases, or a history of acute infection/trauma within one month before enrollment; (2) administration of glucocorticoids for >2 weeks or non-psoriasis immunosuppressants within 3 months before enrollment, including

severe malnutrition, metabolic disorders, and heavy drinking history; and (3) missing key case data, refusal to provide informed consent, or poor treatment compliance.

Collection of Relevant Data

Demographic, clinical, and laboratory data of the enrolled patients were retrieved from the hospital's electronic database. The evaluated laboratory parameters included whole blood viscosity (WBV) (200), WBV (50), WBV (1), PV, hematocrit, red blood cell (RBC) aggregation index, prothrombin time (PT), activated partial thromboplastin time (APTT), fibrinogen (FIB), D-dimer, neutrophils, lymphocytes, and platelets (PLT). For patients who visited multiple hospitals during the study period, the data from their first visit were extracted and included in the dataset.

Statistical Analysis

PSM was conducted in R v.3.3.3 (R Foundation for Statistical Computing) using the MatchIt package v.3.0.2. Baseline characteristics are summarized in Table 1, using the variables included in the propensity score model to assess covariate balance before and after matching. Subsequent analyses were performed in the matched cohort using all available data for each outcome of interest. Therefore, the sample size varied slightly across the analyses depending on data completeness. To secure an adequate sample size and optimal group balance, iterative testing of multiple calipers was conducted with the final number of calipers fixed at 0.15. The baseline characteristics of the two matched groups were compared, and standardized mean differences (SMDs) were calculated to verify the balance of the pre-specified confounders.

The Shapiro–Wilk test was used to assess the data distribution normality. Categorical variables were presented as frequencies (n) and percentages (%), whereas continuous variables were expressed as mean ± standard deviation (SD) if normally distributed or median (interquartile range, [IQR]) if non-normally distributed. Intergroup comparisons for continuous variables were performed using the Mann–Whitney *U*-test, those for categorical variables were performed using the chi-square test, and Spearman correlation coefficients were calculated to assess the correlations between variables. A multivariate logistic regression model was used to calculate the odds ratio (OR), 95% confidence interval (95% CI), and *P* value for each indicator.

Results

Characteristics of the Study Population

After PSM, the matched cohort comprised 206 patients and 126 controls, with all baseline covariates achieving statistical balance (Table 1). All P-values exceeded 0.05, and the SMDs for all variables were < 0.1, a threshold indicative of a negligible imbalance. As key examples, the SMD for age decreased from 1.050 to 0.050, and the SMD for the PASI score dropped from 0.309 to 0.003. The improvement in covariate balance is clearly visualized in Figures S1, and S2 further supports the effectiveness of the PSM.

Comparison of Hemorheological Indicators and Coagulation Levels Between Groups

Furthermore, the hemorheological and coagulation parameters were compared between the case and control groups, with the key findings summarized in Table 2, Figures S3 and S4. The case group exhibited substantially elevated WBV across all shear rates: at 200 s^{-1}, the median WBV was 7.75 mPa·s (IQR: 5.26–9.43) in cases versus 5.31 mPa·s (IQR: 4.88–5.68) in controls. The PV, Hct, and RBC aggregation indices were also significantly higher in the case group, with the RBC aggregation index showing the largest effect size (Cohen's d = 0.699). Statistically significant differences were also observed for both FIB and APTT: the case group had higher FIB levels (3.54 vs. 3.11, $P = 0.005$, Cohen's d = 0.163) and a shorter APTT (26.40 vs. 27.60 s, $P = 0.002$, Cohen's d = 0.181), with both effects being small in magnitude.

Associations of SII and NLR with Key Hemorheological and Coagulation Parameters

First, as shown in Figure S5, the case group exhibited significantly higher levels of the inflammatory markers SII and NLR than the control group. The case group exhibited markedly higher SII values (median: 1,245 vs. 123, $P < 0.001$) and significantly elevated NLR (median: 4.2 vs. 1.8, $P < 0.001$) than the controls.

Table 1 Baseline Characteristics of Case and Control Groups Before and After PSM

Variables	Before PSM				After PSM			
	Case Group (n=439)	Control Group (n=198)	P-value	SMD	Case Group (n=206)	Control Group (n=126)	P-value	SMD
Age (years) Mean ±SD	65.65 ± 11.05	51.86 ± 14.94	<0.001	1.050	60.87 ± 10.67	58.18 ± 13.70	0.062	0.050
Gender: Male n (%)	304 (69.2%)	126 (63.6%)	0.162	0.119	141 (68.4%)	86 (68.3%)	0.971	0.009
Gender: Female n (%)	135 (30.8%)	72 (36.4%)			65 (31.6%)	40 (31.7%)		
BMI (kg/m^2) Mean ±SD	26.18 ± 2.93	24.99 ± 4.51	0.001	0.315	25.96 ± 2.95	25.49 ± 5.03	0.345	0.091
Psoriasis Duration Median (Q1, Q3)	15.00 (8.50–21.00)	14.00 (10.00–18.00)	0.032	0.212	15.00 (7.00–21.00)	15.00 (10.00–18.75)	0.912	0.041
PASI Score Median (Q1, Q3)	29.40 (19.00–41.90)	24.05 (10.88–40.03)	<0.001	0.309	27.80 (17.57–36.88)	25.90 (12.98–42.70)	0.499	0.003
Medication History (Yes, n %)								
Biologics	164 (37.4%)	93 (47.0%)	0.022	0.195	87 (42.2%)	58 (46.0%)	0.498	0.077
Traditional immunosuppressants	174 (39.6%)	99 (50.0%)	0.014	0.208	81 (39.3%)	61 (48.4%)	0.104	0.183
Antiplatelet drugs	167 (38.0%)	75 (37.9%)	0.969	0.003	70 (34.0%)	51 (40.5%)	0.233	0.134
Statin drugs	174 (39.6%)	75 (37.9%)	0.674	0.036	83 (40.3%)	49 (38.9%)	0.800	0.029
Topical drugs	173 (39.4%)	86 (43.4%)	0.338	0.082	85 (41.3%)	58 (46.0%)	0.394	0.096

Abbreviations: PSM, propensity score matching; BMI, body mass index; PASI, psoriasis area and severity index; SMD, standardized mean difference.

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Table 2 Comparison of Hemorheological and Coagulation Indicators Between Case Group and Control Group

Category	Indicator	Case Group	Control Group	Z	P-value	Cohen's d/r
Hemorheological	Whole blood viscosity (200) [mPa·s]	7.75 (5.26–9.43) [n=168]	5.31 (4.88–5.68) [n=105]	−7.40	<0.001	0.448
Median [Q1, Q3]	Whole blood viscosity (50) [mPa·s]	10.72 (6.71–12.60) [n=168]	6.90 (6.31–7.46) [n=105]	−7.27	<0.001	0.440
	Whole blood viscosity (1) [mPa·s]	28.48 (17.65–33.92) [n=168]	15.77 (14.08–17.58) [n=105]	−8.36	<0.001	0.506
	Plasma viscosity [mPa·s]	2.09 (1.51–2.45) [n=168]	1.62 (1.47–1.81) [n=105]	−5.89	<0.001	0.356
	Hematocrit [%]	56.70 (52.50–63.00) [n=145]	46.35 (43.25–48.73) [n=92]	−8.43	<0.001	0.548
	RBC aggregation index	7.37 (5.39–8.60) [n=168]	3.26 (2.83–3.95) [n=105]	−11.56	<0.001	0.699
Coagulation	PT [s]	11.10 (10.50–11.60) [n=181]	10.90 (10.50–11.50) [n=114]	−0.61	0.541	0.036
Median [Q1, Q3]	APTT [s]	26.40 (24.50–28.50) [n=180]	27.60 (25.90–29.35) [n=114]	3.11	0.002	0.181
	FIB [g/L]	3.54 (2.74–4.56) [n=184]	3.11 (2.67–3.85) [n=117]	−2.83	0.005	0.163
	D-dimer (ln)	−0.52 (−1.34–0.60) [n=174]	−0.83 (−1.31–-0.08) [n=106]	−1.67	0.095	0.100
	PLT [×10^9/L]	244.00 (195.00–310.00) [n=185]	227.50 (179.00–266.50) [n=124]	−2.64	0.008	0.150

Notes: Analyses were conducted in the propensity score–matched cohort using available data for each variable.
Abbreviations: RBC, red blood cell; PT, prothrombin time; APTT, activated partial thromboplastin time; FIB, fibrinogen; PLT, platelet.

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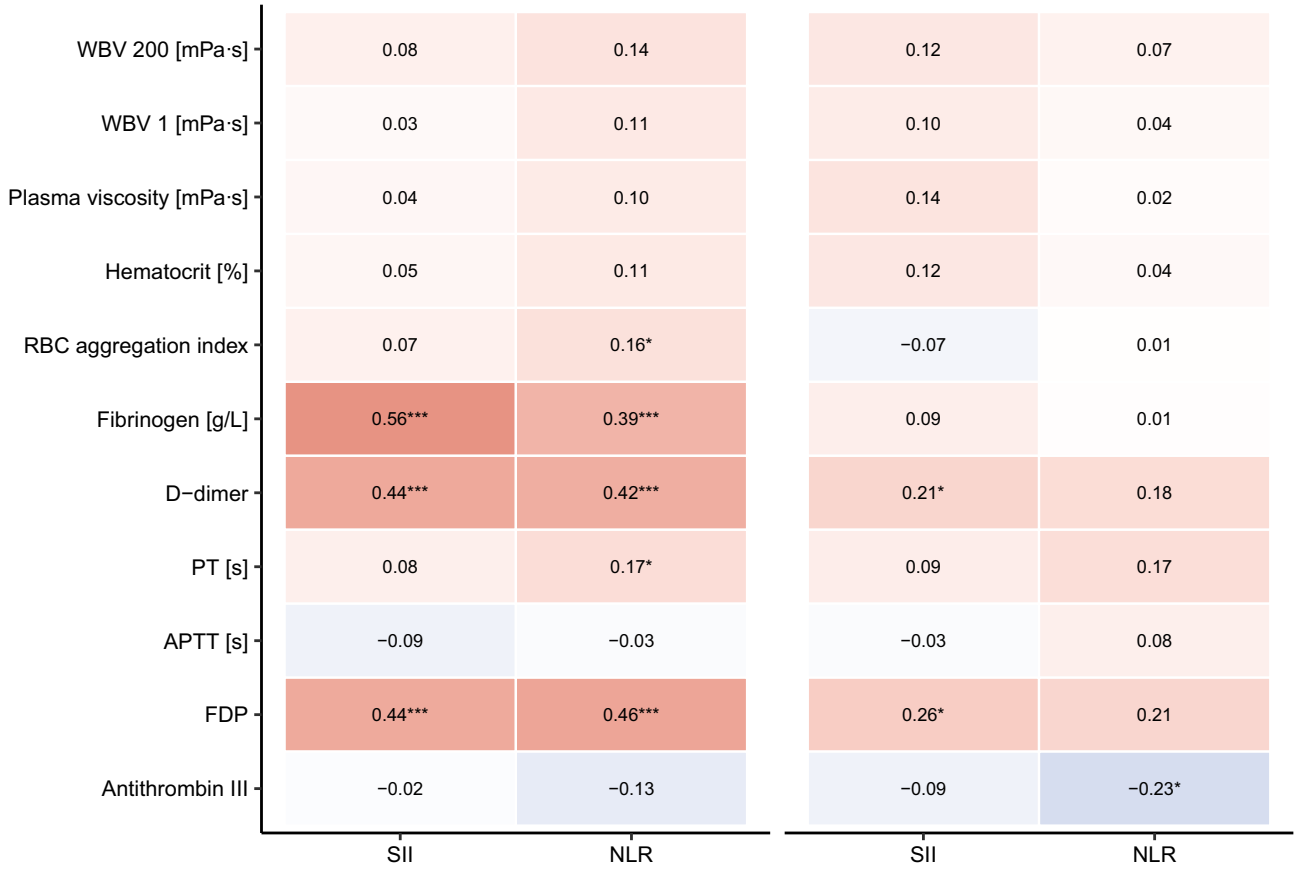

Figure 1 Spearman correlation heatmap of hemorheological/coagulation indicators with SII and NLR in case vs. control groups. Spearman correlation heatmap showing the correlation between hemorheological/coagulation parameters, SII and NLR. The heatmap is divided into two panels: left for the case group and right for the control group. Columns represent SII and NLR, and rows represent hemorheological and coagulation indicators as labeled on the left axis. The color gradient indicates the magnitude of Spearman correlation coefficient r, with red representing positive correlation and blue representing negative correlation. Significance levels: *$P < 0.05$, ***$P < 0.001$. Analyses were conducted in a propensity score-matched cohort.

Abbreviations: SII, systemic immune inflammation index; NLR, neutrophil-to-lymphocyte ratio; WBV, whole blood viscosity; RBC, red blood cell; FIB, fibrinogen; PT, prothrombin time; APTT, activated partial thromboplastin time; FDP, fibrin/fibrinogen degradation product.

Following these observations, the relationships between SII, NLR, and key hemorheological and coagulation parameters were further examined (Figure 1). Specifically, FIB showed the strongest association, with a correlation coefficient of 0.56 ($P < 0.001$) for the SII and 0.39 ($P < 0.001$) for NLR in the case group, respectively (Figure 2). In contrast, hemorheological parameters, including WBV, PV, and hematocrit, showed only weak or insignificant correlations with SII and NLR, with correlation coefficients generally < 0.15.

There was a strong positive correlation between the SII and FIB ($r = 0.56$, $P < 0.001$), with the red scatter points and fitted line clearly demonstrating that the FIB levels increased significantly as the SII increased. No statistically significant correlations were observed between the SII and hemorheological parameters in either group (Figure S6).

Mediation Pathway of Psoriasis Inflammation Promoting AS via Vascular Inflammation and Coagulation Dysfunction

Figure S7 illustrates the mediation pathway through which psoriatic inflammation contributes to AS, involving vascular inflammation and rheology/coagulation dysfunction. Psoriasis inflammation directly promoted vascular inflammation with a large effect size ($\beta = 0.45$, $P < 0.001$). Vascular inflammation also exerted a direct positive effect on AS ($\beta = 0.28$, $P = 0.010$). AS was also found to exacerbate vascular inflammation via a feedback mechanism ($\beta = 0.33$, $P = 0.002$), pointing to a self-reinforcing vicious cycle that may accelerate disease progression.

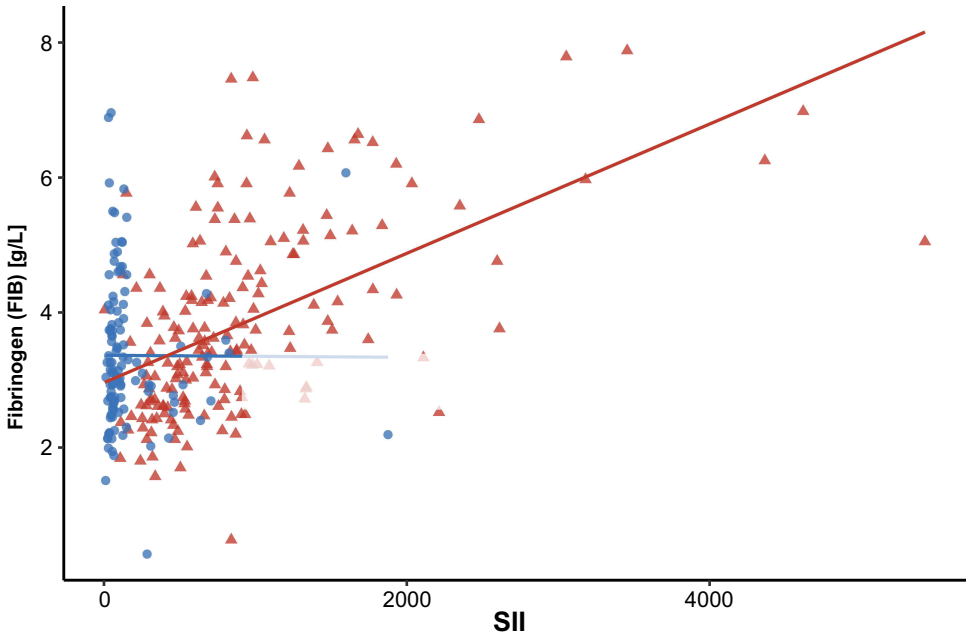

Figure 2 Correlation between SII and FIB in case vs. control groups. Scatter plot showing the correlation between SII (x-axis) and FIB (y-axis) levels. Red triangles and red linear regression line represent the case group (n = 180), blue circles and blue linear regression line represent the control group (n = 117). Spearman correlation analysis showed a strong positive correlation between SII and FIB in the case group (r = 0.56, *da* 0.001) but no significant correlation in the control group (r = 0.09, *P* = 0.356). Analyses were conducted in a propensity score-matched cohort using the available data for each variable.
Abbreviations: SII, systemic immune-inflammation index; FIB, fibrinogen.

As demonstrated in the clustering heatmap (Figure 3), the case group displayed a distinct systemic disorder profile, progressing sequentially from skin inflammation to systemic inflammation, coagulation dysfunction, and vascular injury.

Disease Duration-Dependent Differences in FIB Levels Between Groups

The subgroup analysis showed that FIB elevation was significantly correlated with the duration of psoriasis (Figure S8). The case group exhibited significantly higher FIB levels (3.82 ± 1.40) compared with the control group (3.19 ± 0.90) ($P < 0.001$).

Multivariate Logistic Regression Analysis of Independent Risk Factors for AS in Patients with Psoriasis

Multivariate logistic regression analysis showed WBV (1 mPa·s) was the strongest predictor, with an OR of 1.752 (95% CI: 1.314–2.651, $P < 0.001$). Per 1-SD increase, the OR rose to 424.060 (95% CI: 18.942–36,850.113, $P < 0.001$). The RBC aggregation index was also a significant independent risk factor, with an OR of 3.546 (95% CI: 2.105–7.275, $P < 0.001$). Per 1-SD increase, the OR was 65.052 (95% CI: 11.648–696.234, $P < 0.001$) (Table S2).

Discussion

Our case-control analysis revealed that patients with psoriasis complicated by AS had significantly higher WBV, PV, and RBC aggregation indices than those with psoriasis alone ($P < 0.05$). This comorbid group also presented with markedly elevated FIB and platelet counts, coupled with a significant reduction in APTT ($P < 0.05$). In contrast, the psoriasis-only group exhibited only mild FIB elevation and abnormal whole-blood viscosity. These findings suggest that hemorheological disorders and a hypercoagulable state play crucial roles in the development and progression of psoriasis complicated by AS.

Given the paucity of hemorheological and coagulation studies focusing on patients with psoriasis complicated by AS, it is difficult to compare our findings with those of other relevant studies. Alpsoy et al demonstrated that FIB levels were elevated in patients with psoriasis, which were negatively correlated with aortic strain and aortic compliance but positively correlated with the β-stiffness index.[16] This finding is consistent with the conclusions of our study. Increased FIB synthesis induced by inflammatory stimulation is causally associated with thrombosis.[17,18]

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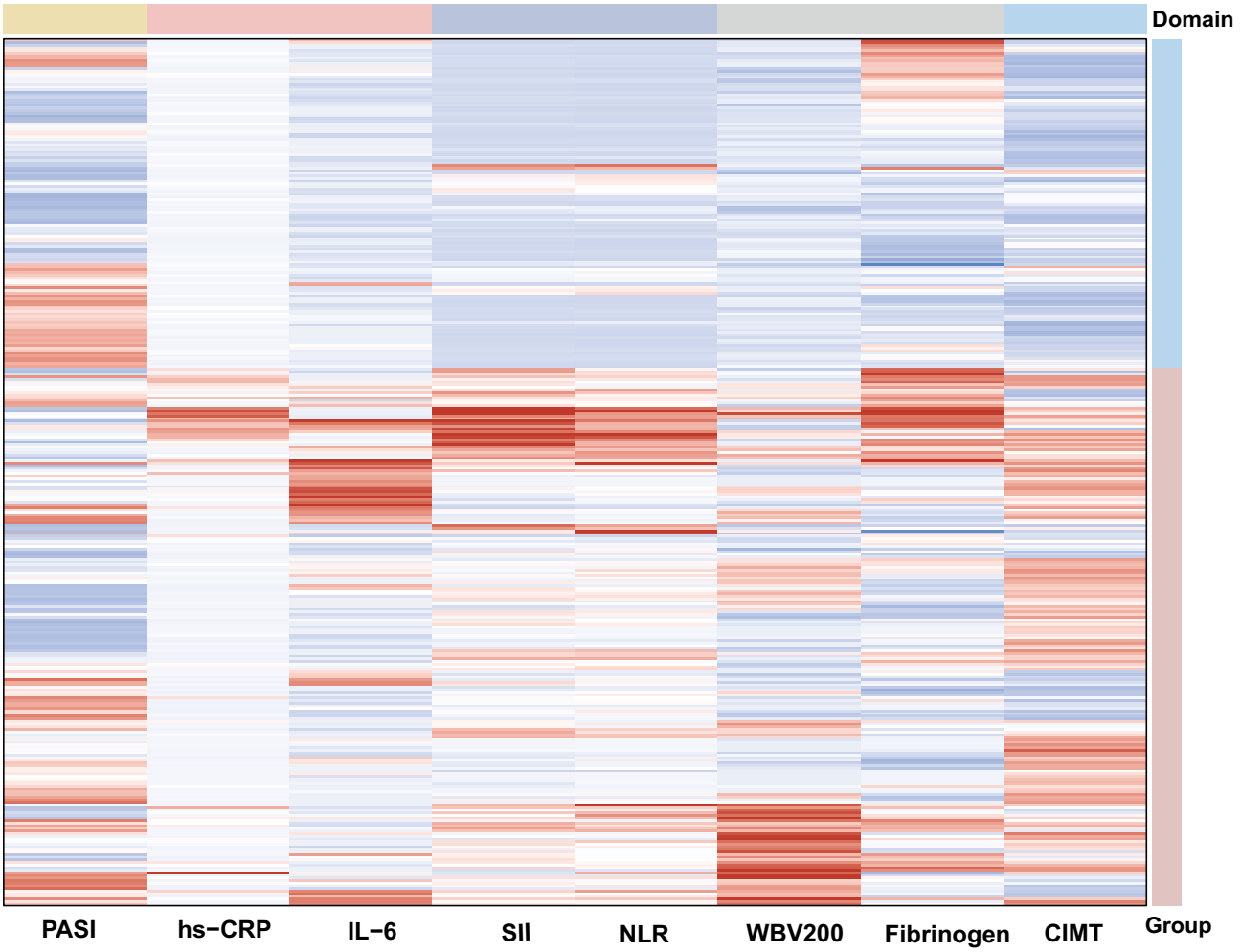

Figure 3 Integrated heatmap of inflammation–coagulation–vascular injury profiles in case vs. control groups. Heatmap of normalized and integrated Z-score showing the expression profiles of inflammation, hemorheological–coagulation, and vascular injury indicators. Columns represent detected indicators including PASI, hs-CRP, IL-6, SII, NLR, WBV200, fibrinogen, and CIMT as labeled on the bottom axis. Rows represent individual subjects. The color gradient represents Z-score values, ranging from negative (blue) to positive (red). Vertical color bars on the right indicate sample groups: pink for case group (n = 206), light blue for control group (n = 126). Top horizontal color bars indicate indicator domains: yellow for skin inflammation, blue for systemic inflammation, pink for vascular inflammation, grey for rheology/coagulation, light blue for vascular injury.
Abbreviations: SII, systemic immune inflammation index; NLR, neutrophil-to-lymphocyte ratio; WBV, whole blood viscosity; hs-CRP, high-sensitivity C-reactive protein; IL-6, interleukin-6; CIMT, carotid intima-media thickness.

Patients with psoriasis exhibit a persistent inflammatory state.[19] Inflammatory cytokines such as tumor necrosis factor – α and interleukin - 6 can damage the structure of RBC membranes, leading to decreased RBC deformability and increased aggregation.[20–22] AS directly induces vascular wall thickening and luminal stenosis, while elevated blood viscosity further exacerbates blood stasis and tissue perfusion defects, continuously aggravating vascular endothelial injury.[23,24]

Admittedly, inflammation and coagulation are interconnected processes.[10,25] The vascular endothelium also plays a critical role in clot formation, as it acts as the interface linking inflammation and coagulation.[26,27] Chronic inflammation in psoriasis activates the extrinsic coagulation pathway, prompting vascular endothelial cells to release tissue factors and leading to hypercoagulability.[28,29] Meanwhile, inflammation suppresses the expression of anticoagulant factors, including proteins C and S, thereby exacerbating the hypercoagulable state.[30]

The study's results identified striking associations between inflammatory indices and key hemorheological and coagulation parameters, with FIB increasing in parallel with increasing SII values. Heatmaps revealed a unique systemic disorder profile in the case group, featuring a sequential pathological cascade from cutaneous to systemic inflammation, coagulation dysfunction, and vascular injury. These observations underscore a pivotal *inflammation-vascular-coagulation axis*. Psoriasis is associated with increased circulating inflammatory cytokines that promote endothelial inflammation and dysfunction, which further induce platelet activation and coagulation abnormalities, thereby increasing the risk of cardiovascular events in affected patients.

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This study has several notable limitations. First, reliance on a single-center patient cohort with a relatively small sample size may have compromised the statistical power, hindering the ability to validate potential causal associations. Second, this study was restricted to a retrospective review of routine clinical records from our institution, which may have omitted critical confounding variables and detailed anamnesis information, leading to potential retrospective biases. Moreover, recall bias may have introduced inaccuracies into the data reported by clinicians or patients. Large-scale, multicenter, prospective cohort studies are needed to determine the predictive role of hemorheological and coagulation parameters for AS risk in patients with psoriasis.

Conclusion

Patients with psoriasis, especially those with severe disease, long disease duration, or other thrombotic predisposing factors, should be closely monitored for hemorheological and coagulation parameters to prevent CVD occurrence. Further studies are warranted to comprehensively elucidate the roles of hemorheological and coagulation parameters, including their potential diagnostic and prognostic utility.

Role of Funder/Sponsor Statement

The National Natural Science Foundation of China had no role in the design or conduct of the study; the collection, management, analysis, and interpretation of the data; the preparation, review, or approval of the manuscript; or the decision to submit the manuscript for publication.

Access To Data And Data Analysis

The corresponding author of this article (Xin Li) had full access to all study data and took responsibility for the integrity of the data and the accuracy of the data analysis.

Abbreviation

APTT, Activated partial thromboplastin time; AS, Atherosclerosis; CI, Confidence interval; CVD, Cardiovascular disease; FIB, Fibrinogen; IQR, Interquartile range; OR, Odds ratio; PLT, Platelet; PT, Prothrombin time; PV, Plasma viscosity; PSM, Propensity score-matched; RBC, Red blood cell; SD, Standard deviations; SMDs, Standardized mean differences; WBV, Whole blood viscosity.

Data Sharing Statement

Data supporting the findings of this study are available from the corresponding author (Xin Li) upon request.

Ethical Approval

This study was approved by the Institutional Ethics Committee of Yueyang Hospital of Integrated Traditional Chinese and Western Medicine, Shanghai University of Traditional Chinese Medicine (#2024-034). All procedures were performed in accordance with the Declaration of Helsinki. Informed consent was waived due to the retrospective and anonymous nature of the study. The authors are accountable for all aspects of this study and ensure that questions related to the accuracy or integrity of any part of the study are investigated and resolved appropriately.

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Author Contributions

Miao Zhang, Munsoon Hong, and Ruiqing Cao share first authorship. All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors declare no conflict of interest.

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