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Research Article

Utilization of HALP and Systemic Immune-Inflammation Index for Thrombotic Risk in Polycythemia Vera and Essential Thrombocythemia

Cumali Yalcin[1], Aycan Acet[2], Aysun Gonderen[1], Ezgi Feyza Aydin[2], Can Ozlu[1]

[1] Department of Internal Medicine, Division of Hematology, Faculty of Medicine, Kütahya, Kütahya Health Sciences University, Türkiye
[2] Department of Internal Medicine, Faculty of Medicine, Kütahya Health Sciences University, Kütahya, Türkiye

Abstract

Aim: Thrombotic complications are a primary source of morbidity for patients with Polycythemia Vera (PV) and Essential Thrombocythemia (ET). The present study was designed to evaluate the clinical utility of the systemic immune-inflammation index (SII) and the HALP score—a composite of hemoglobin, albumin, lymphocytes, and platelets—in predicting the risk of thrombosis within this population.

Material and Method: This retrospective study included adult patients diagnosed with PV or ET between 2015 and 2024. Demographic, clinical, genetic, and laboratory data were obtained from hospital records. HALP and SII scores were calculated using laboratory parameters. Patients were categorized according to the presence or absence of thrombosis, and comparative statistical analyses were performed. The discriminatory performance of SII and HALP for thrombotic risk was evaluated using ROC curve analysis.

Results: The study included 93 patients (mean age, 62.1 ± 14.6 years; 63.4% male). PV constituted 80.6% of diagnoses. Thrombotic events occurred in 27 patients (29%). Neutrophil counts were higher in the thrombosis group (8.8 ± 3.3 vs. 7.4 ± 3.5 ×10⁹/L, p = 0.008), while lymphocyte counts were lower (2.1 ± 0.7 ×10⁹/L vs 2.5 ± 0.8, p = 0.042). HALP scores were markedly lower among individuals with thrombosis (37.5 ± 18.7 vs. 52.9 ± 25.4, p < 0.001). SII did not show a significant difference (p = 0.084). ROC analysis revealed that HALP had acceptable predictive accuracy (AUC = 0.723), with a cutoff of 32.2 yielding 70.6% sensitivity and 59.4% specificity. Overall, HALP was superior to SII for predicting thrombosis.

Conclusion: The HALP score appears to be a practical and informative biomarker for identifying PV and ET patients at elevated thrombotic risk.

Keywords: HALP, thrombosis, inflammation, myeloproliferative neoplasms

INTRODUCTION

Polycythemia Vera (PV) and Essential Thrombocythemia (ET) are clonal hematologic disorders characterized by acquired genetic alterations such as JAK2V617F mutation, CALR, or MPL, known as myeloproliferative neoplasms (MPN) (1, 2). The main contributors to morbidity and mortality in these diseases are thrombotic events, which significantly affect both arterial and venous vascular systems (3).

According to recent studies, thrombosis is largely caused by chronic systemic inflammation in PV and ET, which activates platelets and damages endothelium (4-6). Biomarkers reflecting this inflammatory state, which can be easily derived from complete blood count data, are gaining increasing attention in predicting thrombotic risk (7). In this context, this study focuses on two new combined inflammation scores: the HALP score (which includes

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Corresponding Author: Cumali Yalcin, Department of Internal Medicine, Division of Hematology, Kütahya Health Sciences University, Faculty of Medicine, Kütahya, Türkiye
E-mail: doktorcumali@hotmail.com

hemoglobin, albumin, lymphocyte, and platelet) and the systemic immune-inflammation index (SII). SII (which includes neutrophil, platelet, and lymphocyte components) and HALP are scores that show potential in predicting prognosis and thrombotic events in different hematological malignancies (8-10).

This study's main goal is to determine whether the HALP and SII inflammatory scores can predict the development of thrombosis in PV and ET patients. It aims to determine whether these scores, when used independently of or in combination with existing clinical and genetic risk factors, provide additional benefit in predicting vascular complications.

MATERIAL AND METHOD

Designed as a single-center retrospective observational study, this research enrolled adult patients diagnosed with PV or ET at Kütahya City Hospital's Hematology Clinic during the period from 2015 to 2024, with diagnoses based on the WHO (2022) guidelines. The ethics committee of the Faculty of Medicine, Kütahya Health Sciences University approved this study (decision number: 2025-07/28). Due to the retrospective design and the use of anonymized medical record data, the requirement for informed consent was waived by the ethics committee.

Patients with evidence of active acute infections, concomitant chronic autoimmune diseases, or secondary active malignancies at the time of laboratory assessment were excluded from the study.

The patients' demographic characteristics (age, gender), clinical findings (splenomegaly, B symptoms, ECOG performance score), genetic test results (JAK2V617F), and laboratory parameters were obtained retrospectively from patient files and hospital information systems at diagnosis. Thrombotic events included thrombosis in cerebral, cardiac, pulmonary, abdominal, or extremity locations confirmed by imaging methods (CT, MRI, Doppler ultrasonography) in the arterial or venous vascular system.

HALP and SII scores were used to calculate inflammation scores at the time of diagnosis. The HALP score was calculated by multiplying hemoglobin level (Hgb, g/dL) by albumin level (Alb, g/L) and lymphocyte count (L, $\times 10^9$/L), then dividing the resulting value by platelet count (P, $\times 10^9$/L): HALP = (Hgb × Alb × L) / P. The SII was calculated by multiplying the neutrophil count (N, $\times 10^9$/L) by the platelet count (P, $\times 10^9$/L) and dividing the resulting value by the lymphocyte count: SII = (N × P) / L.

Statistical Analysis

All statistical procedures were conducted using IBM SPSS Statistics for Windows, Version 25.0. Categorical variables were expressed as frequencies and percentages, whereas continuous variables were presented as mean ± standard deviation (SD). Group comparisons were conducted using the independent-samples t-test or Mann–Whitney U test for continuous variables, as appropriate, and the chi-square test for categorical variables. The receiver operating characteristic (ROC) curve analysis was used to assess the diagnostic accuracy of HALP and SII scores in predicting thrombotic events. Variables with $p<0.20$ in univariable analyses, together with clinically relevant covariates, were considered for inclusion in the multivariable model. Odds ratios (ORs) with 95% confidence intervals (CIs) were calculated. Lymphocyte and platelet counts were not included as separate variables in the multivariable analysis because they are incorporated into the calculation of HALP and SII scores, and their simultaneous inclusion could result in multicollinearity. The HALP score was categorized as low (<32.2) or high (≥32.2) based on ROC curve analysis and entered into the logistic regression model as a binary categorical variable. Statistical significance was established with a two-tailed p value of less than 0.05. Scores with an area under the curve (AUC) value between 0.7 and 0.8 were considered "acceptable," between 0.8 and 0.9 "good," and 0.9 and above "very good" in terms of discriminatory power.

RESULTS

The study included 93 patients (mean age, 62.1 ± 14.6 years; 63.4% male). When the distribution of diagnoses was examined, 75 (80.6%) patients had PV, and 18 (19.4%) patients had ET. The JAK2V617F mutation was found in 88.1% of the patients. Splenomegaly was present in half of the patients (50.5%). The mean hemoglobin level was 16.9 ± 1.7 g/dL, hematocrit was 50.3 ± 6.3%, and platelet count was 516 ± 284 × 10^9/L. The mean SII value was 1803 ± 1176, and the HALP score was 40.5 ± 22.2. Mortality had occurred in one patient (Table 1).

Table 1. Clinical and demographic characteristics of patients.

Variables	All patients n=93
	n (%)
Male gender	59 (63.4)
PV	75 (80.6)
ET	18 (19.4)
JAK2 V617F (+)	82 (88.1)
Splenomegaly (+)	47 (50.5)
B symptoms (+)	22 (23.7)
ECOG	
0	59 (63.4)
1	24 (25.8)
2	7 (7.5)
3	3 (3.2)
Myelofibrotic transformation	8 (8.6)
Mortality	1 (1.1)
	Mean ± SD
Age, years	62.1 ± 14.6
Albumin, g/L	44.2 ± 2.6
Hemoglobin, g/dL	16.9 ± 1.7
Hematocrit, %	50.3 ± 6.3
WBC, $\times 10^9$/L	11.1 ± 4.2
Neutrophils, $\times 10^9$/L	7.7 ± 3.5
Lymphocytes, $\times 10^9$/L	2.2 ± 0.8
Monocytes, $\times 10^9$/L	0.6 ± 0.3
Platelets, $\times 10^9$/L	516 ± 284
MPV	9.8 ± 1.1
LDH	250.1 ± 76.9
HALP	40.5 ± 22.2
SII	1803 ± 1176

ECOG, Eastern Cooperative Oncology Group; ET, essential thrombocythemia; HALP, hemoglobin, albumin, lymphocyte, and platelet score; LDH, lactate dehydrogenase; MPV, mean platelet volume; PV, polycythemia vera; SII, systemic immune-inflammation index; WBC, white blood cell.

Thrombosis occurred in 27 patients (29%); 8 at diagnosis, and 19 after diagnosis. The median follow-up period was 45.4 months. Twenty patients had arterial thrombosis (14 coronary artery, 5 cerebrovascular, and 1 peripheral artery), while seven patients had venous thrombosis (5 deep vein thrombosis, 1 pulmonary embolism, and 1 superficial thrombophlebitis).

Age, gender, diagnosis subtype, splenomegaly, B symptoms, and ECOG performance status did not differ statistically significantly between the two groups (p>0.05). In the thrombosis group, neutrophil count increased while lymphocyte count decreased (8.8 ± 3.3 × 10⁹/L vs. 7.4 ± 3.5 × 10⁹/L, p = 0.008 and 2.1 ± 0.7 × 10⁹/L vs 2.5 ± 0.8 × 10⁹/L, p = 0.042, respectively). A decrease in platelet count was noted in the group with thrombosis compared to the non-thrombosis group; nevertheless, the finding was not statistically significant (p = 0.052). Patients with thrombosis demonstrated markedly lower HALP scores compared with those without thrombotic events (37.5 ± 18.7 vs. 52.9 ± 25.4, p < 0.001). In contrast, SII values were higher among patients with thrombosis, though this difference was not statistically significant (1942 ± 1299 vs 1465 ± 713; p = 0.084). Additionally, no significant intergroup differences were detected for albumin, hemoglobin, LDH, or MPV (p > 0.05) (Table 2).

Table 2. Comparison of demographic and laboratory parameters in patients with or without thrombosis.

Variables	Thrombosis (+) n=27	Thrombosis (-) n=66	p
Age, years	63.5 ± 13.5	61.6 ± 15.1	0.597
Male gender, n (%)	16 (59.3)	43 (65.2)	0.592
PV, n (%)	24 (88.9)	51 (77.3)	0.298
ET, n (%)	3 (11.1)	15 (22.7)	
Splenomegaly (+), n (%)	15 (55.6)	32 (48.5)	0.536
B Symptoms (+), n (%)	9 (33.3)	13 (19.7)	0.160
Albumin, g/L	44.0 ± 3.0	44.3 ± 2.5	0.828
Hemoglobin, g/dL	17.2 ± 1.0	16.7 ± 2.0	0.421
Hematocrit, %	51.0 ± 6.6	50.0 ± 6.2	0.277
WBC, ×10⁹/L	11.5 ± 3.7	10.9 ± 2.9	0.282
Neutrophils, ×10⁹/L	8.8 ± 3.3	7.4 ±3.5	0.008
Lymphocytes, ×10⁹/L	2.1 ± 0.7	2.5 ± 0.8	0.042
Monocytes, ×10⁹/L	0.7 ± 0.5	0.6 ± 0.2	0.294
Platelets, ×10⁹/L	410 ± 178	559 ± 307	0.052
MPV	9.9 ± 1.2	9.8 ± 1.0	0.623
LDH	254.0 ± 77.0	248.0 ± 77.0	0.813
HALP	37.5 ± 18.7	52.9 ± 25.4	<0.001
SII	1942.0 ± 1299.0	1465.0 ± 713.0	0.084
ECOG, n (%)			
0	17	42	0.598
1	7	17	
2	3	4	
3	0	3	
JAK2 V617F (+), n (%)	25 (92.5)	57 (86.3)	0.548
Myelofibrotic transformation, n (%)	4 (14.8)	4 (6.1)	0.172
Mortality, n (%)	1 (3.7)	0	0.245

ECOG, Eastern Cooperative Oncology Group; ET, essential thrombocythemia; HALP, hemoglobin, albumin, lymphocyte, and platelet score; LDH, lactate dehydrogenase; MPV, mean platelet volume; PV, polycythemia vera; SII, systemic immune-inflammation index; WBC, white blood cell.

ROC curve analysis indicated that the HALP score showed acceptable discriminatory performance in predicting thrombosis (AUC = 0.723; 95% CI: 0.653–0.830; p < 0.001). For a cutoff value of 32.2, sensitivity was calculated as 70.6% and specificity as 59.4% (Figure 1) SII did not demonstrate significant discriminatory ability for thrombosis (AUC=0.614; 95% CI: 0.498–0.730; p=0.083) (Table 3).

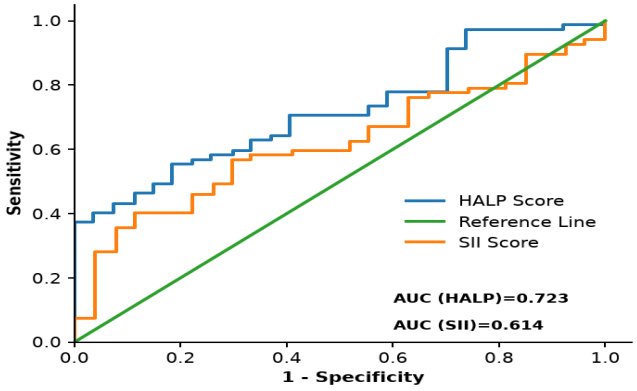

Figure 1. Diagnostic performance of HALP and SII scores on the ROC curve.

Table 3. Sensitivity and specificity of HALP and SII scores according to cutoff values.

Variables	Cut-off	Sens.	Spec.	95% CI of AUC	p
HALP	≤32.2	70.6%	59.4%	0.653 – 0.830	< 0.001
SII	≥870.8	40.3%	88.6%	0.498 – 0.730	0.083

AUC, area under the curv; CI, confidence intervals; HALP, hemoglobin, albumin, lymphocyte, and platelet score; SII, systemic immune-inflammation index; Sens, sensitivity; Spec, specificity.

A multivariable logistic regression was used to determine independent predictors of thrombotic events. Variables with possible significance in univariate analysis were included in the model: myelofibrotic transformation, JAK2 V617F mutation status, HALP score (<32.2), and the SII. HALP score less than 32.2 was found to be the only significant independent predictor of thrombosis among the variables examined (OR: 1.53; 95% CI: 1.18–2.89; p = 0.007). Conversely, myelofibrotic transformation (OR: 2.08, p=0.114), JAK2 V617F positivity (OR: 2.58, p=0.266), and the SII index (OR: 1.16, p=0.208) did not achieve statistical significance as independent predictors (Table 4).

Table 4. Multivariable logistic regression analysis of factors associated with thrombotic events.

Variables	OR	95% CI	p
Myelofibrotic Transformation	2.08	0.72 – 20.59	0.114
JAK2 V617F positivity	2.58	0.49 – 13.76	0.266
HALP score (<32.2)	1.53	1.18 – 2.89	0.007
SII index	1.16	0.67 – 6.50	0.208
	-2 Log likelihood: 94.797, Nagelkerke R^2: 0.242, p<0.001		

DISCUSSION

This study examined the predictive value of systemic inflammatory status indicators, such as SII and HALP scores, for thrombotic events in patients with PV and ET. Our results showed that patients with thrombosis had significantly lower HALP scores compared to patients without thrombosis. A HALP score <32.2 remained independently associated with thrombosis in the multivariable logistic regression model. Although SII is a well-established inflammatory marker in various malignancies, it did not emerge as an independent predictor of thrombosis in our risk model.

PV and ET are clonal hematopoietic chronic myeloproliferative neoplasms that are highly susceptible to thrombosis. The most significant factors that impact mortality and morbidity in these conditions are thrombotic events (11-13). Traditionally, the risk of thrombosis has been assessed based on age, history of previous thrombotic events, presence of JAK2 mutation, and hematological parameters (14). In our study, factors such as myelofibrotic transformation and presence of JAK2 V617F mutation did not demonstrate statistical significance in the multivariate analysis. This observation could be clarified by the elevated occurrence of JAK2 mutations in our group, which probably restricted the ability to differentiate mutation status among the comparison groups.

However, recent studies have demonstrated that persistent systemic inflammation is a significant factor in this process, impacting the hematopoietic microenvironment and causing endothelial dysfunction as well as platelet activation (15-17). The literature shows that SII and HALP scores have mostly been studied as prognostic indicators in solid tumors and hematological malignancies, but there are only a limited number of studies on thrombosis prediction in myeloproliferative neoplasms (18-21). Studies investigating the relationship between systemic inflammation and thrombotic processes in PV and ET patients have generally focused on parameters such as C-reactive protein (CRP), neutrophil/lymphocyte ratio (NLR), or platelet/lymphocyte ratio (PLR) (22, 23). From this perspective, the use of new composite indices such as HALP and SII offers a more sensitive and multidimensional inflammatory assessment.

SII is a combined inflammation marker derived from neutrophil, platelet, and lymphocyte components. Neutrophils increase the release of proinflammatory cytokines, while platelets play an essential role in endothelial activation and clot formation; a decrease in lymphocyte count reflects impaired immune regulation (24). According to previous studies, patients with PV and ET who have high SII values are more likely to develop thrombotic events (18). However, in some series, the prognostic value of SII has been found to be limited; this suggests the influence of different inflammatory profiles or accompanying comorbidities (25). In our study, the discriminatory power of SII in predicting thrombosis did not reach statistical significance.

In contrast, the HALP score is regarded as a more comprehensive indicator of systemic response because it assesses both hematological and biochemical parameters together. Solmaz et al. reported significantly longer overall survival in patients with multiple myeloma who had higher HALP scores than in those with lower HALP scores (26). Similarly, Xin et al. reported that the HALP score is an independent predictor of mortality and major thrombotic events in cardiovascular diseases (27). Yilmaz et al. evaluated the HALP score in 124 PV patients with and without a history of thrombosis and suggested that the HALP score was not associated with the risk of thrombosis (28). In our study, the significantly lower HALP score in the group that developed thrombosis suggests that this parameter may reflect a biological response associated with a thrombotic tendency. This result supports that the HALP score may be not only a prognostic biomarker but also a risk indicator reflecting hemostatic and inflammatory activity.

Although SII incorporates neutrophil, platelet, and lymphocyte counts, it does not include albumin or hemoglobin, which may provide additional information regarding systemic inflammation, nutritional status, and disease-related hematologic burden. The lack of statistical significance for SII in our study may also be related to the relatively small sample size, limited number of thrombotic events, and heterogeneity of thrombotic manifestations. Moreover, the wide variability of platelet counts in PV and ET patients may have reduced the discriminatory ability of SII. These findings suggest that HALP may capture a broader biological spectrum relevant to thrombosis than SII in this patient population.

Our study has some limitations. First, it is retrospective in design with a limited sample size. HALP scores and SII were assessed based on a single measurement at diagnosis; dynamic changes were not monitored. The number of thrombotic events was low in multivariate analysis. Therefore, the results of multivariate analysis should be interpreted carefully. Additionally, the relationship between inflammatory markers and cytokine profiles was

not investigated. Furthermore, as this is a retrospective analysis, we were unable to fully adjust for the potential confounding effects of active cytoreductive therapies (e.g., hydroxyurea) or phlebotomy on the hematological parameters comprising the HALP and SII scores at the time of measurement. Another limitation of this study is that treatment status (e.g., aspirin or hydroxyurea use) at the exact time of each thrombotic event during follow-up was not systematically assessed. Therefore, the potential impact of ongoing therapy on thrombotic risk could not be evaluated.

CONCLUSION

This study highlights the utilization of the HALP score as a biomarker for determining thrombotic risk in patients with PV and ET. HALP is an index that can be used in combination with current risk assessment models. It is based on routine, easily accessible and reasonably priced biochemical measurements. These results suggest that the HALP score may be used as a useful clinical parameter if these findings are validated by large-scale prospective studies.

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Conflict of interest: The authors declare they have no conflicts of interest.

Ethical approval: The ethics committee of the Kütahya Health Sciences University Faculty of Medicine approved this study (decision number: 2025-07/28).

Informed consent: Due to the retrospective design and the use of anonymized medical record data, the requirement for informed consent was waived by the ethics committee.

Data availability statement: The data that support the findings of this study are available on request from the corresponding author.

Author contributions: Conceptualization, C.Y.; methodology/design, C.Y.; supervision, A.A. and C.O.; materials, E.F.A.; data collection and/or processing, A.G. and E.F.A.; analysis and/or interpretation, C.Y. and A.A.; literature review, A.A., A.G., E.F.A., and C.O.; writing—original draft preparation, C.Y.; writing—review and critical revision, A.A., A.G., E.F.A., and C.O. All authors have read and agreed to the published version of the manuscript.

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