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Inflammatory Markers in Supraventricular Tachycardia: Insights for Emergency Management

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Abstract

Aim: Supraventricular tachycardia (SVT) is a common reason for emergency department visits and can significantly impact patients' quality of life. Certain hematological parameters may support the diagnosis and aid in the clinical management of conditions that often occur in the absence of structural heart disease. This study aimed to evaluate hematological markers, particularly inflammatory parameters, and their potential role in SVT.

Materials and Methods: This retrospective study included 243 newly diagnosed SVT patients and 220 healthy controls. Demographic data and laboratory parameters such as white blood cell count, neutrophil count (NE), red cell distribution width (RDW), platelet count, and mean platelet volume (MPV) were analyzed. Additionally, the neutrophil-to-lymphocyte ratio (NLR) and the platelet-to-lymphocyte ratio were calculated to assess inflammatory status.

Results: The findings revealed that NLR, RDW, and NE levels were significantly higher in the SVT group, while eosinophil, hemoglobin, and hematocrit levels were significantly lower. ROC analysis identified NLR as a significant predictor of SVT, with an optimal cut-off value of 2.62 and a specificity of 72.3%. Although MPV did not reach statistical significance, a proportional increase was observed in SVT, patients.

Conclusion: This study highlights the potential role of NLR and RDW as supportive biomarkers in SVT diagnosis. Our findings indicate that NLR and RDW levels were significantly higher in SVT patients compared to controls, suggesting a link between inflammation and SVT pathogenesis. These findings suggest that inflammation may play a role in SVT and that hematological parameters could aid its evaluation.

Keywords: Supraventricular tachycardia, biomarkers, inflammation, lymphocyte counts, neutrophil-to-lymphocyte ratio, red cell distribution width

Introduction

Palpitations, defined as an irregular, rapid, or forceful sensation of the heartbeat, are among the most common complaints in patients presenting to the emergency department (ED). It is estimated that approximately 10% of ED visits are due to palpitations (1,2). Given the broad differential diagnosis ranging from benign conditions to potentially life-threatening arrhythmias accurate and efficient evaluation is essential for patient management and risk stratification. Although palpitations can originate from both cardiac and non-cardiac causes, identifying underlying arrhythmias is particularly important to guide appropriate treatment strategies.

Among cardiac arrhythmias, supraventricular tachycardia (SVT) accounts for approximately 2.25 per 1000 ED admissions for palpitations (3). SVT is an umbrella term encompassing various rhythm disturbances originating anatomically above the atrioventricular node. Although SVT has multiple subtypes, most episodes present as paroxysmal (3). Paroxysmal supraventricular tachycardia (PSVT) is characterized by the sudden onset and abrupt termination of tachycardia (4). Most patients with PSVT do not have

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structural heart disease, and the primary mechanisms include increased automaticity, triggered activity, and reentry (5). Due to these mechanisms, PSVT is more commonly observed in younger patients and is often first diagnosed in the emergency setting (6). Although electrocardiogram (ECG) remains the gold standard for diagnosing SVT, transient episodes that resolve before evaluation can complicate diagnosis. Identifying laboratory biomarkers associated with SVT could provide additional diagnostic support, particularly in patients presenting with nonspecific symptoms or an unclear arrhythmic history.

Previous studies have demonstrated that blood parameters possess predictive and prognostic value in various cardiac pathologies (7-10). In particular, hemogram parameters have been widely investigated for their diagnostic and prognostic utility (11-14). However, the relationship between PSVT and hemogram parameters remains controversial, with only a limited number of conflicting reports available in the literature (8,15). Additionally, several studies suggest that inflammation may serve as a triggering factor for SVT (1,9,16-18), acting both as an initiator and a facilitator (1). Emerging evidence suggests that inflammation may contribute to arrhythmogenesis by promoting autonomic imbalance, myocardial excitability, and atrial remodeling, which may increase susceptibility to SVT (1,9,16-18). Consequently, some evidence indicates that inflammatory markers could have a predictive role in tachyarrhythmias. Although hematological parameters have been investigated in various cardiovascular conditions, their specific role in SVT remains unclear, with conflicting findings in the literature (8,15).

In this study, we aimed to assess laboratory parameters that may support SVT diagnosis and contribute to patient evaluation in the emergency setting. By analyzing these parameters, our goal is to provide additional insights that could assist clinicians in managing SVT more effectively.

Materials and Methods

Our study was designed as a retrospective analysis and conducted on patients newly diagnosed with SVT in the emergency department of Etlik City Hospital between December 1, 2022, and December 8, 2024, based on clinical presentation and ECG findings (6,19). Since this was a retrospective study, only patient data were analyzed. Ethical approval was obtained from the Etlik City Hospital Scientific Research Evaluation and Ethics Board Commite before data collection, and the study was conducted in accordance with institutional regulations and ethical guidelines (desicion number: AESH-BADEK-2025-0021, date: 08.01.2025).

To minimize confounding factors, we evaluated patients with nearly isolated SVT by excluding various conditions that could influence the results. The control group consisted of patients who presented to the ED without chronic diseases and met none of the exclusion criteria. This approach aimed to eliminate the potential effects of comorbidities and medication use on laboratory results.

For all patients, demographic data, medical history, medication history, and laboratory blood parameters were collected and analyzed. The SVT group and the control group were compared, with a particular focus on hemogram parameters.

Inclusion criteria:

- Patients aged 18 years and older
- Patients diagnosed with SVT for the first time in the emergency department

Exclusion criteria:

- Pregnant patients
- Presence of known heart disease
- History of previously diagnosed arrhythmia
- History of major trauma or surgery within the past 3 months
- Presence of chronic inflammatory disease
- Presence of acute rheumatologic or infectious disease
- Diagnosis of malignancy
- Diagnosis of rheumatologic disease
- Presence of immunosuppressive disease

Statistical Analysis

Data were analyzed using IBM SPSS Statistics version 25.0 software (IBM Corp., Armonk, NY, USA). Descriptive statistics were presented as frequency (n), percentage (%), mean ± standard deviation, or median (Q1-Q3) values. The Pearson chi-square test was used to evaluate categorical variables. The normality of numerical variables was assessed using normality tests and Q-Q plots. For comparisons between two groups, the independent samples t-test was used for normally distributed variables, while the Mann-Whitney U test was applied for non-normally distributed variables. Additionally, ROC analysis was conducted to evaluate the predictive value of certain parameters in SVT. A p value <0.05 was considered statistically significant.

Result

The study included 243 patients diagnosed with SVT and 220 control patients. The mean age of the SVT group was 51.1±16.5 years, while the mean age of the control group was 40.4±9.6 years. Gender distribution, lymphocyte count, mean platelet volume (MPV), platelet distribution width (PDW), and platelet (PLT) values were comparable between the two groups (Table 1).

However, white blood cell (WBC) count, neutrophil count (NE), red cell distribution width (RDW), urea, creatinine, alanine aminotransferase, and aspartate aminotransferase levels were significantly higher in the SVT group. In contrast, eosinophil count (EO), hemoglobin (HGB), and hematocrit (HCT) values were significantly lower in patients with SVT (Table 1).

The neutrophil-to-lymphocyte ratio (NLR) was significantly higher in the SVT group compared to the control group [median: 2.16 (0.39-43.0) vs. 1.92 (0.60-18.60), p=0.0219] (Figure 1). In contrast, the platelet-to-lymphocyte ratio (PLR) did not differ significantly between the two groups (p=0.335).

ROC analysis demonstrated that NLR had a significant predictive value for SVT [area under the curve (AUC): 0.562, p=0.022]. The optimal cut-off value for NLR in predicting SVT was determined as 2.62, with a sensitivity of 40.0% and a specificity of 72.3% (Figure 2).

Additionally, RDW (AUC: 0.591, p=0.001), NE (AUC: 0.617, p<0.001), and EO (AUC: 0.557, p=0.036) have significant predictive values for SVT as indicated by. However, MPV (AUC: 0.552, p=0.053), PDW (AUC: 0.549, p=0.069), and PLR (AUC: 0.526, p=0.335) did not demonstrate significant predictive value (Table 2).

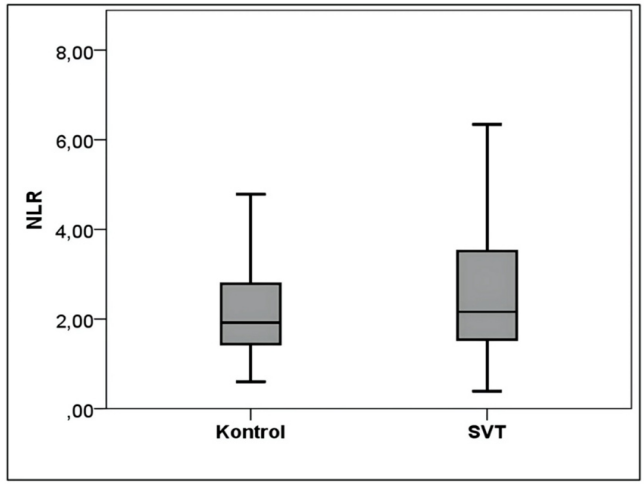

Figure 1. Comparison of NLR between groups
SVT: Supraventricular tachycardia, NLR: Neutrophil-lymphocyte ratio

Table 1. Demographic features and hematological findings in SVT and control patients

	SVT (n=243)	Control (n=220)	p value
Gender			
Male	98 (40.3%)	100 (45.5%)	0.266
Woman	145 (59.7%)	120 (54.5%)	
WBC	10.34±3.98	8.95±2.78	<0.001
EO	0.13±0.11	0.15±0.13	0.035
NE	6.56±3.02	5.51±2.43	<0.001
LY	2.74±1.25	2.59±0.96	0.287
MPV	10.60±0.88	10.45±0.91	0.053
RDW	43.44±4.62	42.10±3.69	0.001
PDW	12.49±2.01	12.19±2.06	0.069
HGB	13.70±2.00	14.14±1.94	0.017
HCT	42.20±5.27	43.24±5.06	0.040
PLT	278.35±86.23	281,43±75.45	0.622
Ure	34.42±15.49	27.11±8.68	<0.001
Creatine	0.95±0.50	0.79±0.19	<0.001
AST	32.66±35.05	21.55±21.38	<0.001
ALT	30.63±49.24	22.08±29.27	<0.001

Chi-square test, t test for independent variables, Mann-Whitey U test.
SVT: Supraventricular tachycardia, WBC: Wight blood cell, NE: Neutrophil count, EO: Eosinophil count, LY: Lymphocyte count, MPV: Mean platelet volume, RDW: Red cell distribution width, PDW: Platelet distribution width, HGB: Hemoglobin, HCT: Hematocrit, PLT: Platelet, NE: Neutrophil count, AST: Aspartate aminotransferase, ALT: Alanine aminotransferase

Discussion

SVT is a common cause of ED visits and can significantly impact patients' quality of life. It is characterized by sudden onset and termination and typically occurs without underlying structural heart disease. Given these characteristics, supportive laboratory parameters may serve as valuable diagnostic and prognostic aids. In our study, we analyzed hematological parameters and assessed their potential role in SVT diagnosis and pathogenesis. We found that inflammatory markers were consistently elevated in the SVT group, with NLR and RDW emerging as significant predictors of SVT.

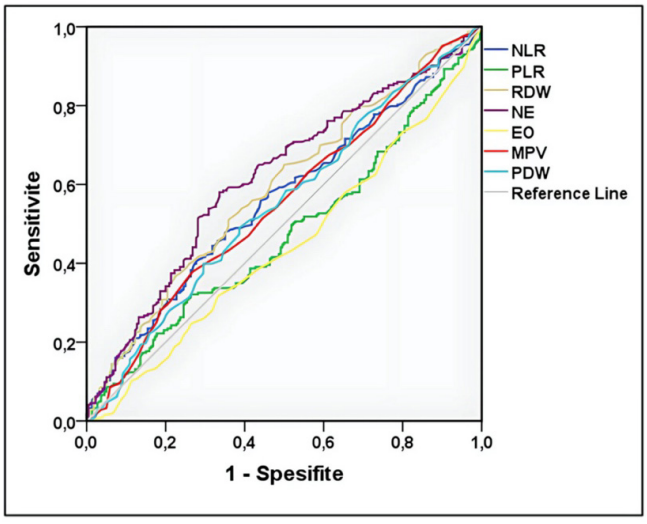

Figure 2. ROC analysis of haemogram parameters

NLR: Neutrophil-lymphocyte ratio, PLR: Platelet-lymphocyte ratio, RDW: Red cell distribution width, NE: Neutrophil count, EO: Eosinophil count, MPV: Mean platelet volume, PDW: Platelet distribution width

Inflammation has been recognized as a contributing factor in arrhythmogenesis, particularly in the development of premature cardiac beats, which can serve as triggers for SVT onset (18). Additionally, the association between inflammatory markers and premature cardiac beats has been well documented in various cardiovascular diseases (18,20). This supports the hypothesis that inflammation may increase susceptibility to SVT by promoting ectopic activity and reentry mechanisms. Several studies have further suggested that inflammation is involved in SVT etiology (1,8,15). Our findings align with the cardiovascular inflammatory hypothesis, which suggests that inflammation is a key factor in the pathogenesis of cardiologic and vascular diseases, as previously proposed by Sen et al. (21).

Multiple studies support the hypothesis that inflammation plays a role in arrhythmogenesis by evaluating conditions such as stroke, pulmonary thromboembolism (PTE), and peripheral artery disease. Yang et al. (22) investigated systemic inflammatory markers in arrhythmia, while Pektezel et al. (23) and Sadeghi et al. (24) examined NLR, PLT, and MPV values in stroke. Similarly, Gosav et al. (25) evaluated NLR in common cardiovascular diseases, and Zhang et al. (26) explored its relationship with myocardial infarction and heart failure.

In atrial fibrillation (AF), Berkovitch et al. (27) investigated NLR, while da Silva et al. (28) examined NLR and RDW in AF and rheumatic valve diseases. Further, Guan et al. (29) assessed NLR, RDW, and PLR in critically ill patients with AF (29). The diagnostic utility of hematological parameters in PTE was highlighted by Karakurt et al. (30), while Işık et al. (14) examined eosinophil counts in patients undergoing cardiopulmonary resuscitation. Teperman et al. (31) also studied NLR in lower extremity peripheral artery disease. Collectively, these studies emphasize the role of inflammatory processes in disease pathogenesis

Table 2. Effectiveness of blood parameters in predicting SVT

	AUC	SE	95% CI		p value
			Lowest	Highest	
NLR	0.562	0.027	0.510	0.614	0.022
PLR	0.526	0.027	0.473	0.579	0.335
RDW	0.591	0.026	0.539	0.642	0.001
NE	0.617	0.026	0.566	0.668	0.000
EO	0.557	0.027	0.504	0.609	0.036
MPV	0.552	0.027	0.500	0.604	0.053
PDW	0.549	0.027	0.496	0.601	0.069

SVT: Supraventricular tachycardia, NLR: Neutrophil-lymphocyte ratio, PLR: Platelet-lymphocyte ratio, RDW: Red cell distribution width, NE: Neutrophil count, EO: Eosinophil count, MPV: Mean platelet volume, PDW: Platelet distribution width, AUC: Area under the curve, SE: Standard error, CI: Confidence interval

and their potential use as biomarkers for risk stratification and clinical decision-making.

The Coumel arrhythmia development theory, as discussed by Farré and Wellens (32) and Rebecchi et al. (33), highlights three key contributors: an anatomical factor (extra/accessory pathway), a triggering factor, and a modulating factor (e.g., autonomic nervous system). Our findings suggest that inflammation serves as a key triggering factor in SVT pathogenesis, leading to premature beats and increasing SVT susceptibility. This aligns with evidence from Güngör et al. (34), who reported an association between inflammatory markers and AF recurrence. Conversely, Marcus et al. (35) found that reducing inflammation contributes to arrhythmia regression, further supporting the role of inflammation as a modifiable risk factor. Additionally, Frustaci et al. (36) provided histopathological evidence of inflammatory infiltrates in atrial biopsies of AF patients, reinforcing the mechanistic role of inflammation in arrhythmogenesis.

Among the hematological markers evaluated, NLR and RDW were found to be significant predictors of SVT in our study. We determined an optimal NLR cut-off value of 2.62, with a specificity of 72.3%, highlighting its potential diagnostic relevance. A similar study by Akpek et al. (37) in acute coronary syndrome (ACS) reported an NLR cut-off of 3.3 with a specificity of 83%, though the difference may be attributed to ACS, being a more hemodynamically disruptive pathology.

Similarly, RDW, a marker reflecting red blood cell size variation, has been linked to systemic inflammation and adverse cardiovascular events, independent of HGB and HCT levels (38-41). Our study demonstrated significantly higher RDW levels in SVT patients, consistent with findings from Güngör et al. (34) and Li et al. (42), the latter of whom identified RDW as an independent predictor of paroxysmal atrial fibrillation. However, Bassareo et al. (18) found no significant correlation between RDW and arrhythmias, possibly due to differences in patient selection criteria and study methodology.

While MPV values did not reach statistical significance in our study, a proportional increase was observed in SVT patients, aligning with previous literature. Differences in study design and sample size may explain why Ocak et al. (1) found a significant association, whereas Cosgun et al. (15) did not.

Additionally, our findings of higher WBC counts and lower EO, HGB, and HCT levels in SVT patients further support the inflammatory hypothesis. These hematological alterations have been linked to poor clinical outcomes in prior studies (14,43). Elevated liver and kidney function markers observed in our study also suggest potential hemodynamic consequences of SVT, further reinforcing the interplay between inflammation and cardiovascular pathology.

Conclusion

Our study demonstrates that NLR, RDW, and NE may serve as valuable diagnostic markers and contribute to the clinical management of SVT patients. These hematological parameters could assist in risk stratification and guide treatment decisions. However, further prospective, multicenter studies are needed to validate these findings and support their clinical implementation.

Study Limitations

This study has several limitations. Its single-center, retrospective design may limit generalizability. Consequently, we were unable to assess the effects of recurrent SVT attacks. Excluding patients with chronic thromboembolic events could have strengthened the study's findings. Moreover, we did not evaluate inflammatory markers (e.g., C-reactive protein, tumor necrosis factor, interleukin), which could have provided a more comprehensive assessment of SVT-related inflammation. Additionally, variability in findings may have been introduced due to the non-homogeneous patient population. Despite these limitations, our study provides valuable insights by analyzing a large cohort of newly diagnosed SVT patients. Future multicenter, prospective studies are needed to validate these findings and establish their clinical relevance.

Ethics

Ethics Committee Approval: Ethical approval was obtained from the Etlik City Hospital Scientific Research Evaluation and Ethics Board Commite before data collection, and the study was conducted in accordance with institutional regulations and ethical guidelines (desicion number: AESH-BADEK-2025-0021, date: 08.01.2025).

Informed Consent: Our study was designed as a retrospective analysis and conducted on patients newly diagnosed.

Footnotes

Authorship Contributions

Surgical and Medical Practices: Ö.F.T., N.İ.I., Concept: Ö.F.T., Design: Ö.F.T., Data Collection or Processing: Ö.F.T., N.İ.I., Analysis or Interpretation: Ö.F.T., Literature Search: N.İ.I., Writing: Ö.F.T.

Conflict of Interest: The authors declare that they have no conflict of interest.

Financial Disclosure: There are no financial conflicts of interest to disclose.

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