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# A Retrospective Study Assessed Eosinophil Levels in Patients who Presented to the Emergency Department with Deep Vein Thrombosis

**Özge Atik[1*], Ramazan Sami Aktas[2], Çağlar Kuas[3], Ertuğ Günsoy[2]**

[1]*Division of Immunology and Allergic Diseases, Department of Internal Medicine, University of Health Sciences, Van Training and Research Hospital, Van, Türkiye*
[2]*Department of Emergency Medicine, Van Training and Research Hospital, Van, Türkiye*
[3]*Department of Emergency Medicine, Faculty of Medicine, Eskişehir Osmangazi University Hospital, Eskişehir, Türkiye*

**Abstract**

Eosinophils are increasingly recognized as active participants in thrombogenesis, yet their precise role in venous thromboembolism remains unclear. This study aimed to evaluate the association between eosinophil elevation and the presence and severity of deep vein thrombosis (DVT) in patients presenting to the emergency department.
This retrospective observational study included patients aged ≥18 years who were diagnosed with acute DVT by Doppler ultrasonography between January 2019 and December 2024 in a tertiary emergency department. Patients with malignancy, autoimmune disease, renal failure, prior venous thromboembolism, or thrombophilia were excluded. Demographic, clinical, laboratory, and imaging data were retrieved from electronic medical records. Eosinophilia was defined as an absolute eosinophil count (AEC) ≥ 500 cells/μL. Statistical analyses were performed using appropriate parametric and non-parametric tests.
A total of 283 DVT patients (mean age 56 ± 19 years; 58.3% male) were evaluated, along with 46 control patients without DVT. The median eosinophil count was higher in DVT patients than in controls (130 vs 100 cells/μL; $p = 0.044$). DVT predominantly involved the lower extremity (92.6%), with left-sided thrombosis in 56.9%. Patients with multiple-vein DVT had significantly higher age, leukocyte, neutrophil, and CRP levels compared to those with single-vein involvement ($p < 0.05$). Eosinophilia was present in 1.4% of DVT cases.
Elevated eosinophil levels may contribute to DVT pathophysiology through endothelial damage, tissue factor expression, and coagulation activation. Although eosinophilia was rare, its potential role in thrombus formation warrants further large-scale prospective studies.

**Keywords:** Eosinophilia; Deep Vein Thrombosis; Venous Thromboembolism; Coagulation; Thrombosis

## Introduction

Eosinophils are a type of granulocytic leukocyte known for their involvement in allergic responses and in defense against parasitic infections. Recent evidence indicates that eosinophils can play an active role in thrombus formation by producing tissue factor, releasing various procoagulant mediators, and forming eosinophil extracellular traps (EETs) (1-3,13-16). Degranulation of eosinophil granules releases toxic cationic proteins that may damage the vascular endothelium and trigger local inflammation (1-3,15,16). Eosinophils can also facilitate the coagulation cascade by providing surface phospholipids that activate Hageman factor (Factor XII) and by inhibiting natural anticoagulant pathways (1-3,13-16). These pathophysiological mechanisms lead to endothelial dysfunction, resulting in a pro-thrombotic environment (1-3).
Eosinophilia can occur in various conditions such as allergic diseases, infections, inflammatory processes, and neoplastic disorders (4,5). Hypereosinophilic syndrome (HES) is characterized by a persistently high eosinophil count accompanied by organ damage, which can lead to thrombotic events (4,5,12). In cases of HES with additional pro-thrombotic risk factors-such as inherited thrombophilia, active malignancy, prolonged immobilization, or significant systemic inflammation-the risk of developing DVT may be increased (4-7,12).

***Corresponding Author:** Dr. Özge Atik, Division of Immunology and Allergic Diseases, Department of Internal Medicine, University of Health Sciences, Van Training and Research Hospital, Van, Türkiye
E-mail: drozgeatik@gmail.com, Fax: +90 432 212 1954
**ORCID ID: Özge Atik:** 0000-0003-2219-0039, **Ramazan Sami Aktas:** 0000-0003-1072-382X, **Çağlar Kuas:** 0000-0003-1283-9420, **Ertuğ Günsoy:** 0000-0002-9653-3236
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The relationship between eosinophilia and venous thromboembolism (VTE) is still not completely understood. A recent cohort study and a meta-analysis have identified a significant link between high eosinophil counts and an increased risk of DVT (6,7). Furthermore, case reports in the literature have noted that certain eosinophilic disorders may initially present clinically as VTE (8,9,17). Taken together, these observations suggest a potential role for eosinophils in thrombogenesis. Our study aims to investigate the diagnostic and prognostic significance of eosinophil elevation in patients diagnosed with acute DVT in the emergency department (6,7,12).

## Materials and Methods

**Study Design and Data Collection:** This study was conducted as a single-center, retrospective observational study. We included patients diagnosed with acute DVT in our hospital's emergency department from January 2019 to December 2024. In addition to patients who tested positive for DVT, we also included patients who were evaluated for suspected DVT but tested negative as the control group. Patient data were got from the electronic medical record system. During the chart review, we recorded each patient's demographic information, presenting clinical features, laboratory values (complete blood count parameters, C-reactive protein [CRP], troponin I), and imaging findings (Doppler ultrasonography) (10–12). The location and extent of DVT involvement were noted for all cases. DVT was classified as "single vein involvement" if the thrombus was present in one vein, and "multiple vein involvement" if the thrombus spanned two or more venous segments (10,11). The DVT region was categorized as either upper or lower extremity, and the side of involvement was recorded as right, left, or bilateral (10,11). Patients with an eosinophil count ≥ 500 cells/μL were analyzed as a separate subgroup and compared with the other patients (4–7,12). Additionally, for baseline comparisons, we identified a control group of patients (n = 46) who were evaluated for suspected DVT in the emergency department during the same period but had DVT ruled out by imaging. Laboratory and demographic data from these non-DVT patients were collected to compare with the DVT group.

**Inclusion and Exclusion Criteria:** Patients aged 18 years or older, with available laboratory data at presentation and a confirmed diagnosis of acute DVT by imaging, were included in the study. The following patients were excluded: those with a history of VTE (DVT or pulmonary embolism); those with inherited or gained thrombophilia (e.g., Factor V Leiden mutation, anticardiolipin antibody positivity, etc.); patients with active malignancy, autoimmune disease, chronic renal failure, or a major transient VTE risk factor (such as recent major surgery or trauma); and patients on medications that could significantly affect eosinophil counts (e.g., systemic corticosteroids) (10,12).

The study protocol was approved by the local ethics committee (Decision No: GOKAEK-2024-02-17), and the research was conducted in accordance with the principles of the Declaration of Helsinki (12).

**Statistical Analysis:** Data analysis was performed using SPSS for Windows version 21.0 (SPSS Inc., Chicago, IL, USA). The distribution of continuous variables was examined using visual methods (histograms and probability plots) and analytical methods (Kolmogorov–Smirnov test). Normally distributed continuous variables were presented as mean ± standard deviation (SD), and non-normally distributed variables as median (interquartile range, IQR). For comparisons between two groups, Student's t-test or the Mann–Whitney U test was used for continuous variables as appropriate. Categorical variables are presented as number and percentage (%), and were compared using the chi-square test. A two-tailed p-value <0.05 was considered statistically significant for all analyses (12).

## Results

A total of 283 patients with DVT were included in the analysis. The general demographic, laboratory, and radiological characteristics of the DVT patients are summarized in Table 1. The mean age of the DVT cohort was 56 ± 19 years, and 58.3% were male. In the great majority of cases (92.6%), the thrombosis was in the deep veins of the lower extremity, with left-sided involvement being predominant (56.9% of all cases). Multiple deep vein involvement was present in 55.1% of patients, while in the remaining 44.9% the thrombus was confined to a single vein. The median absolute eosinophil count in the entire DVT cohort was 130 (70–220) cells/μL. The median CRP level was 18 (4.9–66) mg/L, the median leukocyte count was 8.0 (6–10) × $10^3/mm^3$, and the median neutrophil count was 5 (4–7) × $10^3/mm^3$. Among the 96 patients for

**Table 1:** Baseline Demographic, Laboratory, and Radiological Characteristics of Patients With DVT, Compared To Patients Without DVT (controls)

| Characteristic | DVT Patients (n = 283) | Non-DVT Patients (n = 46) | p value |
|---|---|---|---|
| Age, years (mean ± SD) | 56 ± 19 | 58±20 | 0.462 |
| Sex, male, n (%) | 165 (58.3) | 24 (52.2) | 0.291 |
| Absolute eosinophil count (cells/μL), median (IQR) | 130 (70–220) | 100 (40-190) | 0.044 |
| Leukocyte count (×10³/mm³), median (IQR) | 8.0 (6–10) | 9 (6-12) | 0.290 |
| Neutrophil count (×10³/mm³), median (IQR) | 5.0 (4–7) | 6 (4-9) | 0.138 |
| CRP (mg/L), median (IQR) | 18 (4.9–66) | 13 (2.8-39) | 0.383 |
| Troponin (ng/L), median (IQR)* | 0.1 (0.1–3) | 0.1 (0.1–0.1) | 0.072 |
| DVT region – Lower extremity, n (%) | 262 (92.6) | — | — |
| DVT region – Upper extremity, n (%) | 21 (7.4) | — | — |
| DVT side – Right / Left / Bilateral, n (%) | 107 (37.8) / 161 (56.9) / 15 (5.3) | — | — |
| DVT type – Single vein / Multiple veins, n (%) | 127 (44.9) / 156 (55.1) | — | — |

* Values are presented as mean ± standard deviation (SD), median (interquartile range, IQR), or number (%), as appropriate. *Troponin was measured in 96 patients (troponin data were not available for controls).* Abbreviations: DVT — deep vein thrombosis; CRP — C-reactive protein; IQR — interquartile range; SD — standard deviation.

whom a troponin measurement was available, the median troponin level was 0.1 (0.1–3) ng/L.

These baseline findings, along with a comparison to the non-DVT control group, are presented in Table 1. In brief, the DVT group had a higher Absolute eosinophil count compared to the non-DVT patients (130 cells/μL vs. 100 cells/μL p =0.044 respectively). In Table 1; There was no significant difference in leukocyte count, neutrophil count, CRP between the DVT and non-DVT groups. Troponin levels were also similar between the DVT patients and the controls (0.1 vs 0.1 ng/L p=0.072).

In a subgroup analysis based on the extent of DVT involvement, patients with single vein involvement were compared with those with multiple vein involvement (Table 2). The multiple-involvement group had a significantly higher mean age than the single-involvement group (59 ± 19 vs 52 ± 17 years; p = 0.004). Similarly, patients in the multiple vein involvement group exhibited higher markers of inflammation: the leukocyte count (median 9 vs 7.1 × 10^3/mm³; p = 0.023), neutrophil count (median 5.6 vs 4.3 × 10^3/mm³; p = 0.001), and CRP level (median 35 vs 7 mg/L; p = 0.001) were all significantly greater in the multiple-involvement DVT group than in the single-involvement group (12). There was also a notable difference in thrombosis localization between these groups: in patients with multiple vein involvement, thrombosis in the lower extremity—particularly left-sided DVT—was more frequent than in those with single vein involvement. Specifically, lower-extremity DVT occurred in 95.5% of patients with multiple involvement vs 89.0% of those with single involvement, and left-sided DVT was observed in 57.7% vs 55.9% of patients, respectively (p = 0.037 for upper vs lower extremity distribution; detailed in Table 2). Additionally, bilateral DVT (involvement of both right and left sides) was present in 9.0% of the multiple-involvement group compared to only 0.8% of the single-

**Table 2:** Comparison of Clinical and Laboratory Characteristics of Patients With Single-Vein DVT Versus Multiple-Vein DVT Involvement

| Characteristic | Single Vein DVT (n = 127) | Multiple Vein DVT (n = 156) | Non-DVT Controls (n = 46) | p-value (a) |
|---|---|---|---|---|
| Age (years, mean ± SD) | 52 ± 17 | 59 ± 19 | 59 ± 19 | 0.004; 0.01 (a) |
| Sex (male / female) | 74 / 53 | 91 / 51 | 24 / 22 | 0.817; 0.36 (a) |
| Eosinophil count (cells/μL, median [IQR]) | 140 (80–240) | 125 (52–197) | 125 (60–210) | 0.073; 0.80 (a) |
| Leukocyte (×10³/mm³, median [IQR]) | 7.1 (6–10) | 9.0 (6–11) | 7.0 (5–9) | 0.023; 0.02 (a) |
| Neutrophil (×10³/mm³, median [IQR]) | 4.3 (3.2–7.0) | 5.6 (4.0–8.0) | 4.5 (3.5–6.5) | 0.001; 0.01 (a) |
| CRP (mg/L, median [IQR]) | 7 (3.1–37.7) | 35 (9–89) | 5 (2–20) | 0.001; < 0.001 (a) |
| Troponin (ng/L, median [IQR])* | 0.11 (0.1–5.4) | 0.10 (0.1–0.36) | 0.10 (0.1–0.1) | 0.108; 0.40 (a) |
| DVT region – Upper / Lower, n (%) | 14 (11) / 113 (89) | 7 (4.5) / 149 (95.5) | — | 0.037 (Sv vs Mv only) |
| DVT side – Right / Left / Bilateral, n (%) | 55 (43.3) / 71 (55.9) / 1 (0.8) | 52 (33.3) / 90 (57.7) / 14 (9.0) | — | 0.005 (Sv vs Mv only) |

(a) Data are expressed as mean ± SD, median (IQR), or number (%). For each variable, the first p-value corresponds to the comparison between the single-vein and multiple-vein DVT groups; the second p-value is for the comparison between all DVT patients (combined) and the non-DVT control group.
Troponin was measured in 96 DVT patients; troponin measurements were not available for the control group. Abbreviations: DVT — deep vein thrombosis; CRP — C-reactive protein; SD — standard deviation; IQR — interquartile range

**Table 3:** Demographic and clinical data of DVT patients with eosinophilia (AEC ≥ $0.5 \times 10^9$ /L)

| Patient | Age (years) | Sex | Eosinophil count (cells/μL) | Leukocyte (×10³/mm³) | Neutrophil (×10³/mm³) | Troponin (ng/L) | CRP (mg/L) | DVT type | DVT region | DVT side |
|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 79 | Female | 1780 | 9.8 | 6.8 | – | 82 | Multiple | Upper extremity | Right |
| 2 | 79 | Female | 580 | 13.0 | 10.0 | 0.1 | 8 | Multiple | Lower extremity | Right |
| 3 | 43 | Male | 510 | 10.5 | 5.2 | 0.1 | 1.86 | Multiple | Lower extremity | Left |
| 4 | 36 | Female | 3890 | 13.8 | 7.8 | – | 9.78 | Single | Lower extremity | Left |

- Eosinophilia was found in 4 of 283 DVT patients (1.4%). Three of these cases were female, and three had thrombus involvement of multiple deep veins. DVT was localized to the lower extremity in three patients (two right, one left) and to the upper extremity in one patient. "–" indicates that the test was not performed or data were unavailable. Abbreviations: DVT — deep vein thrombosis; AEC — absolute eosinophil count; CRP — C-reactive protein

involvement group ($p = 0.005$). There were no significant differences between the single and multiple involvement groups in terms of sex distribution or troponin levels ($p > 0.05$ for both) (12).

According to our study criteria, eosinophilia (AEC

≥ 500/μL) was identified in 4 DVT patients (1.4% of the entire cohort). These four cases were evaluated separately in terms of their clinical features and thrombosis characteristics, as shown in Table 3. Three of the 4 patients with eosinophilia were female, and three had thrombosis involving multiple veins; in 1 case, the thrombus was confined to a single vein. The thrombosis was localized to the lower extremity in three eosinophilic patients (two with right-leg DVT and one with left-leg DVT), and the remaining one patient had an upper-extremity DVT. Two patients had right-sided thrombosis and two had left-sided thrombosis. The individual demographic, laboratory, and clinical data for these four eosinophilic DVT cases are presented in Table 3. Because the eosinophilia subgroup contained a very small number of cases, no statistical comparisons were performed for this subgroup versus the rest of the cohort; however, common notable features are described qualitatively in the Discussion (6,12,17).

## Discussion

This study yielded findings that support the hypothesis that an increase in eosinophil levels may play a role in the development of venous thrombosis. In our cohort, the incidence of eosinophilia was low (only 4 of 283 patients, 1.4%); however, this could be because of the selected nature of our study population and the possibility that eosinophil counts can be suppressed during acute-phase reactions (5,12). In the literature, there is a growing body of evidence that eosinophils can trigger thrombogenesis. Indeed, a recent meta-analysis reported that patients with very high eosinophil counts have a markedly increased incidence of VTE; specifically, individuals with hyper-eosinophilia in the acute phase were found to have an approximately 6-fold higher short-term risk of VTE (6,7,12). Similarly, a large-scale population study from South Korea showed that individuals with AEC ≥ 1.5 × 10^9/L have a significantly higher risk of developing DVT and pulmonary embolism compared to the general population (6). Our findings are consistent with these reports and indicate that eosinophil elevation could be an independent factor predisposing to thrombosis. An increase in eosinophils can influence not only the risk of an initial thrombosis but also the risk of recurrence (12).

Eosinophils contribute to thrombus formation via multiple pathophysiological mechanisms. For example, eosinophils are a major intravascular carrier of tissue factor in the circulation, and in conditions of hypereosinophilia the thromboplastic activity of these cells is markedly increased (13,14). Eosinophils can also trigger the intrinsic coagulation pathway; it has been reported that they initiate Factor XII activation via procoagulant phospholipids on their surface, thereby speeding up thrombin production (1–3,15,16). In animal models, eosinophil depletion leads to the formation of significantly smaller venous thrombi after vascular injury, highlighting the contribution of eosinophils to coagulation (1–3).

Cationic proteins released from eosinophil granules have important effects on the coagulation–hemostasis balance. For instance, major basic protein (MBP) binds strongly to thrombomodulin on the endothelial surface, preventing thrombin from activating protein C; in this way, it suppresses natural anticoagulant mechanisms and increases the tendency for clotting (15). Indeed, eosinophil granule proteins (particularly MBP) have been shown to contribute to the hypercoagulability observed in eosinophilic conditions (14,15). When activated, eosinophils also release cytotoxic enzymes and inflammatory mediators that cause local inflammation and endothelial damage. For example, hypochlorous acid derivatives produced by eosinophil peroxidase can increase tissue factor expression in endothelial cells, triggering thrombosis (16). This eosinophil-mediated inflammation leads to the creation of a pro-thrombotic microenvironment in the vessel wall. Eosinophils rapidly migrate to sites of thrombus formation and interact with activated platelets; as a result of this interaction, DNA-protein web structures called eosinophil extracellular traps (EETs) are generated (1–3,13–16). EETs have been demonstrated in both human and experimental thrombus specimens and have been found to contain abundant eosinophil granule proteins (especially MBP). These web-like structures can directly activate platelets and coagulation factors, thereby supporting the growth and stabilization of the thrombus. Strategies designed to inhibit eosinophil–platelet interactions and EET formation have been shown to reduce thrombus size in vivo (1–3,13–16). In summary, eosinophils can trigger the coagulation cascade and reinforce the developing thrombus through multiple mechanisms: providing tissue factor and procoagulant molecules, activating Factor XII, releasing cytotoxic granule proteins (e.g., MBP, eosinophil cationic protein, eosinophil peroxidase), and forming pro-thrombotic EETs

(1–3,13–16). Thus, eosinophilia creates a multi-faceted effect that predisposes to venous thrombus development.

Clinical observations also support the relationship between eosinophilia and thrombotic risk. As mentioned above, both a primary cohort study and a meta-analysis have shown that VTE risk is increased in individuals with hypereosinophilia (6,7,12). Moreover, eosinophil elevation affects not only the risk of a first thrombotic event but also the risk of recurrence (12). In the literature, VTE cases associated with eosinophilia have been reported with unusual and severe clinical presentations. Sometimes, DVT or pulmonary embolism can be the first clinical manifestation of an underlying, previously unrecognized eosinophilic disorder (8,9,17). For example, Mishchenko et al. reported a case of a young female patient with unexplained Budd–Chiari syndrome (hepatic vein thrombosis) accompanied by idiopathic hypereosinophilia and a JAK2 V617F mutation; the authors suggested that mediators released by eosinophils might have contributed to the development of splanchnic vein thrombosis in that case (8). Similarly, severe VTE events can occur in cases of tropical pulmonary eosinophilia. Sharma et al. described a young patient with filarial parasite–induced hypereosinophilia who presented with distal deep vein thrombosis in the absence of other predisposing risk factors; after antiparasitic therapy was added to anticoagulation and the eosinophil count returned to normal, the thrombus completely resolved (9). Particularly in clonal eosinophilia (myeloproliferative neoplasms), thrombotic complications can follow an extremely aggressive course. In a case reported by Atri et al., a 34-year-old female patient with > 60% eosinophils in peripheral blood (due to an underlying myeloid neoplasm) presented with extensive lower extremity DVT; despite initiation of standard anticoagulant therapy, she developed a massive pulmonary embolism on the 4th day and died. This tragic case illustrates that in eosinophil-associated thrombosis, anticoagulation alone may not be sufficient, and early recognition and treatment of the underlying eosinophilic disorder with targeted therapy is of vital importance (17).

Optimal management of patients presenting with VTE in eosinophilia should involve treating both the thromboembolic event and controlling the eosinophilic process. Although there are no formal treatment guidelines specific to eosinophilia-associated thromboses, certain therapeutic approaches have been suggested based on case reports and series. In the acute management of DVT or PE, standard anticoagulant therapy (e.g., low molecular weight heparin or direct oral anticoagulants) is fundamental; however, in the presence of severe or persistent eosinophilia, adjunctive eosinophil-lowering therapy may be required. For instance, in patients with HES, recurrent thromboses have been observed even when only anticoagulation is provided, so it is recommended to add immunosuppressive therapy concurrently with antithrombotic treatment (4,5,12). Corticosteroids are widely used as a first-line therapy in reactive or idiopathic eosinophilia to achieve a rapid reduction in peripheral eosinophil counts and prevent organ damage. For severe, life-threatening thrombotic presentations, an initial pulse of high-dose intravenous methylprednisolone (for example, 500–1000 mg/day for 3 days), followed by oral prednisone maintenance (≈0.5–1 mg/kg/day), has been suggested to normalize eosinophil counts quickly (4,5,12). In cases of clonal eosinophilia (e.g., FIP1L1-PDGFRA-positive myeloproliferative syndrome), targeted therapy with the tyrosine kinase inhibitor imatinib can induce hematologic remission, eliminating both the eosinophilia and the predisposition to thrombosis (4,5,12). In steroid-unresponsive or recurrent hypereosinophilia, cytoreductive agents such as hydroxyurea or biological therapies targeting the interleukin-5 pathway (e.g., mepolizumab, benralizumab) have also been used, although the direct effects of these agents on VTE outcomes have not yet been well studied (4,5,12). Considering our findings and the existing evidence, we recommend a multidisciplinary approach in DVT cases associated with eosinophilia. Besides standard anticoagulant therapy, the underlying eosinophilic condition should be promptly identified and appropriately treated to bring it under control, which is critical for preventing recurrences and complications (12,17).

Finally, some limitations of our study should be noted. This research has a retrospective design and includes a limited sample from a single center. Therefore, it is difficult clearly to establish causality for the observed associations, and the influence of possible confounding factors cannot be completely ruled out. Our original dataset did not include a formal classification of DVT cases as provoked or idiopathic; therefore, subgroup analysis based on this distinction was not performed. The low incidence of eosinophilia in our study population limited certain statistical analyses and subgroup comparisons. Larger-scale, multicenter prospective studies are needed to more definitively determine the contribution of eosinophil elevation to thrombosis risk and to evaluate the prognostic value of eosinophil counts in clinical practice (6,7,12). Nevertheless, eosinophil levels—an easily obtained parameter from routine blood counts—appear to be a factor that could be considered in the initial risk assessment of DVT patients. Future research will help to clarify the role of eosinophil-focused strategies in the prevention and treatment of VTE (6,7,12,17).

Based on data from DVT patients evaluated in the emergency department, this study suggests that eosinophil elevation may play a role in the development of venous thrombosis. Our findings are consistent with recent evidence in the literature pointing to the impact of eosinophils on thrombogenesis (1–3,6,7,12–16). Eosinophils can support thrombus formation and progression through mechanisms such as increasing tissue factor expression, triggering the coagulation cascade, and forming extracellular traps (1–3,13–16). In particular, in the presence of hypereosinophilia, persistently high eosinophil levels can increase both the risk of an initial thrombotic event and the risk of recurrence (6,12). Therefore, investigating the prognostic significance of an easily accessible parameter like eosinophil count in DVT patients would be valuable. If an eosinophil elevation is detected in a patient with thrombosis, it may be prudent to consider therapies targeting eosinophilia alongside standard anticoagulant therapy. Large-scale prospective studies are needed in the future to draw definitive conclusions on this subject (6,7,12,17).

**Ethics Committee Approval:** This study was approved by the Van Research and training hospital local Ethics Committee (Decision No: GOKAEK-2024-02-17).

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