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Comparison of pulmonary embolism in oncology patients with the general population in terms of diagnosis, clinical features and prognosis

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ABSTRACT

Aims: Pulmonary embolism (PE) is a frequent and potentially fatal complication in cancer patients. The diagnosis, treatment, and prognosis of cancer-associated PE (CA-PE) differ from those of PE in the general population. The primary aim of this study was to compare the clinical presentation and severity of PE between patients with and without malignancy; the secondary aim was to compare short- and long-term mortality.

Methods: Between 2020 and 2023, patients with PE without malignancy (PE group), patients with CA-PE (CA-PE group), and patients with suspected PE in whom PE was excluded by imaging (control group), 80 in each group, were retrospectively analyzed. Symptoms, risk factors, laboratory data, embolism localization, and clinical scores (Wells, modified Geneva, and Khorana for malignancy) were recorded. Prognostic scores (Pulmonary Embolism Severity Index [PESI], Bova, Pompe-C), mortality data (30-day, 3-month, 1-year), and modified Glasgow Prognostic Score were evaluated.

Results: The most common cancers were lung and brain tumors. In the CA-PE group, 67.5% were receiving active chemotherapy, and 50% had metastatic disease. Presenting symptoms were similar between the PE and CA-PE groups (dyspnea, p=0.10; chest pain, p=0.31), whereas both symptoms differed significantly from the control group (p<0.001). Embolism localization did not differ significantly between the groups. Inflammatory markers such as neutrophil/lymphocyte (p=0.006), monocyte/lymphocyte (p=0.01), and platelet/lymphocyte ratios (p=0.015) were higher in CA-PE. PESI score was significantly higher (p<0.01), whereas right ventricular dysfunction was not more frequent (p=0.129). Troponin-I and B-type natriuretic peptide (BNP) levels were similar. While 3-month and 1-year mortality rates were higher in CA-PE (p<0.01), 30-day mortality was similar.

Conclusion: Patients with CA-PE had higher inflammatory indices, but markers of PE severity were not worse than in patients without malignancy. Long-term all-cause mortality was higher in CA-PE, although cause-specific mortality was not assessed. Existing risk models may not be sufficient; malignancy-specific scoring systems are needed.

Keywords: Neoplasms, inflammation, mortality, prognosis, pulmonary embolism

INTRODUCTION

Acute pulmonary embolism (PE) is a common form of venous thromboembolism (VTE), presenting with a wide clinical spectrum, from no symptoms to sudden death. It is the third most common cardiovascular disorder globally, after myocardial infarction and stroke.[1] Because PE presents with nonspecific symptoms, several diagnostic algorithms and risk assessment models have been developed.[2]

In cancer patients, VTE is a major cause of death, second only to the malignancy itself. Its incidence is 4-7.5 times higher than in the general population.[3] Sorensen et al.[4] reported a one-year survival rate of 12% in cancer patients with VTE, versus 36% in those without. This increased risk arises from multiple factors: patient, tumor, and treatment-related.[5]

Cancer-associated thrombosis involves complex mechanisms; cancer cells activate coagulation via procoagulants, cytokines, and tissue factor-bearing microparticles.[6] This prothrombotic state increases both morbidity and mortality. PE symptoms may be masked by cancer-related complaints, complicating diagnosis.[7]

General risk scores such as the Wells and modified Geneva scores are less reliable in cancer patients. The Khorana model targets this group but has limited use due to cancer heterogeneity.[8] Likewise, the Pulmonary Embolism Severity Index (PESI) overestimates risk by assigning malignancy points regardless of cancer type, stage, or treatment.

Low molecular weight heparins (LMWH) are preferred in initial treatment according to current guidelines, with better outcomes than vitamin K antagonists.[9] However, the duration of therapy and the role of direct oral anticoagulants (DOAC) remain under investigation.

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Most studies compare cancer patients with and without VTE, offering little insight into how malignancy alters PE. Our study directly compares cancer-associated PE (CA-PE) with PE in patients without malignancy. The primary objective was to compare the clinical presentation (presenting symptoms and vital signs) and PE severity (embolism localization, right ventricular dysfunction on echocardiography, cardiac biomarkers and clinical severity scores) between patients with and without malignancy. The secondary objectives were to compare inflammatory indices, treatment approaches and all-cause mortality at 30 days, 3 months and 1 year. A control group of patients with suspected PE in whom PE was excluded was included to provide reference values for clinical and laboratory parameters. These data may support the development of more effective risk and treatment models.

METHODS

Ethics

This study was approved by the Non-interventional Researches Ethics Committee of Bezmialem Vakıf University (Date: 26.07.2023, Decision No: 2023/232). All procedures were carried out in accordance with the ethical rules and the principles of the Declaration of Helsinki.

The requirement for informed consent was waived due to the retrospective nature of the study. This study was conducted and reported in accordance with the STROBE (Strengthening the Reporting of Observational Studies in Epidemiology) guidelines.

Study Design and Population

This retrospective study was conducted at the department of pulmonology of a tertiary care university hospital between June 2020 and June 2023. Patients were divided into three groups: patients with PE without malignancy (PE group), patients with CA-PE (CA-PE group) and a control group. The control group consisted of adult patients who were evaluated during the same period for suspected acute PE because of a high clinical suspicion (based on presenting symptoms, clinical findings and clinical probability assessment) but in whom PE was excluded by CT pulmonary angiography (CTPA) or V/Q scintigraphy; thus, "negative diagnostic findings" refers to the absence of PE on these imaging studies. The same exclusion criteria applied to the PE groups were applied to the control group. Patients with malignancy were not included in the control group. The control group was included to provide reference values for clinical and laboratory parameters in patients with a similar clinical presentation, rather than in healthy individuals.

From the hospital database, 362 patients with PE diagnosis codes were initially identified and screened according to the predefined criteria described below. To obtain groups of equal size, 80 patients without malignancy and 80 patients with malignancy were randomly selected from the eligible patients. The control group of 80 patients was randomly selected in the same manner from patients with suspected PE and negative imaging during the study period.

Inclusion and Exclusion Criteria

Patients eligible for inclusion were those diagnosed with PE at the study center's department of pulmonology during the specified time period. We excluded patients under 18 years of age, pregnant women, and COVID-19 PCR positive patients to avoid confounding. Additionally, patients with pneumonia detected on CT were excluded due to potential overlap in clinical presentation. Other exclusion criteria included: patients diagnosed with PE at external centers (n=33), patients who refused treatment or terminated treatment early (n=26), patients with malignancy lacking pathological confirmation (n=21), and patients with insufficient laboratory data (n=4). In the CA-PE group, only patients with histopathologically confirmed malignancy were included. Patients with solid tumors and those with hematological malignancies were both eligible for inclusion in the CA-PE group.

Data Collection and Clinical Assessment

Patient medical records were comprehensively reviewed to collect demographic, clinical, and laboratory data. Demographics included age, gender, body-mass index (BMI), and smoking history quantified in pack-years. Clinical data encompassed presenting symptoms (dyspnea, chest pain, syncope, hemoptysis), symptom duration, vital signs (blood pressure, heart rate, respiratory rate, oxygen saturation), and shock index (heart rate/systolic blood pressure).

Comorbidities and risk factors for VTE were systematically documented, with particular attention to hypertension, diabetes mellitus, chronic kidney disease, coronary artery disease, chronic obstructive pulmonary disease, atrial fibrillation, chronic heart failure, immobilization, previous PE history, recent surgery, and blood transfusion. Performance status was evaluated using the Eastern Cooperative Oncology Group (ECOG) score, and comorbidity burden was assessed using the Charlson Comorbidity Index (CCI). Other recorded risk factors included central venous catheter, trauma, rheumatological disease, previous COVID-19, erythropoietin use, superficial vein thrombosis and the postpartum period. Inherited thrombophilia was not screened systematically; a known thrombophilia was recorded when documented in the medical records.

For patients with malignancy, we recorded cancer type, diagnosis date, disease stage (early, locally advanced, or metastatic), metastatic sites, and treatment modalities (chemotherapy, radiotherapy, surgery, hormonal therapy). The time between malignancy diagnosis and PE occurrence, as well as the time between most recent chemotherapy and PE occurrence, were calculated.

Laboratory Investigations

Blood samples were collected at admission for laboratory analysis. Standard laboratory parameters included complete blood count, D-dimer, C-reactive protein (CRP), albumin, creatinine, liver function tests, and cardiac biomarkers (Troponin-I, CK-MB, BNP). Inflammatory indices including neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), monocyte-to-lymphocyte ratio (MLR), and Systemic Immune-Inflammation Index (SII=platelet count×neutrophil count/lymphocyte count) were calculated from complete blood count data.

Imaging Studies and Diagnostic Procedures

PE was confirmed or excluded primarily by CTPA in 230 patients (95.8%), with ventilation/perfusion (V/Q)

lung scintigraphy used in 10 patients (4.2%) who had contraindications to CTPA. The anatomical location of embolism was categorized as pulmonary trunk, main pulmonary artery, lobar, segmental, or subsegmental. The presence of unilateral or bilateral emboli was noted, as was the occurrence of pulmonary infarction.

Echocardiography was performed in 168 patients (70%) to assess right ventricular function, pulmonary artery pressure, and signs of right heart strain. Doppler ultrasonography of the lower extremities was performed in 103 patients (42.9%) to detect deep vein thrombosis, with findings classified as unilateral or bilateral. Echocardiography and lower-extremity Doppler ultrasonography were performed at the discretion of the treating physician and were therefore not available for all patients.

Clinical Risk and Severity Assessment

All patients were evaluated using established risk stratification tools. Clinical probability of PE was assessed using the Wells score and Modified Geneva score. For cancer patients, VTE risk was additionally evaluated using the Khorana score. PE severity was determined using the PESI, with patients classified as low risk (class I-II, score ≤85) or high risk (class III-V, score >85).

Mortality risk was assessed using the Bova score for all PE patients, with additional cancer-specific scores including the Pompe-C score, 30-day mortality score, and Modified Glasgow Prognostic Score (mGPS) for the CA-PE group. Based on clinical and imaging findings, patients were further classified as high-risk (massive), intermediate-risk (submassive), or low-risk (non-massive) PE according to current guidelines.

Treatment and Follow-up

Treatment approaches were recorded, including the use of thrombolytic therapy, low molecular weight heparin (LMWH), vitamin K antagonists (VKA), and DOACs. Hospital length of stay and intensive care unit admission duration were documented. Patients were followed for mortality outcomes at three time points: 30 days, 3 months, and 1 year.

Statistical Analysis

The data were analyzed using IBM SPSS Statistics for Windows, version 21.0 (IBM Corp., Armonk, NY, USA). The normality of data distribution was assessed using the Kolmogorov-Smirnov and Shapiro-Wilk tests for each variable within each of the three groups separately. Continuous variables that were normally distributed in all three groups were presented as mean±standard deviation; those that deviated from a normal distribution in at least one group were presented as median (interquartile range, IQR). Categorical variables were presented as frequency and percentage.

For comparisons between groups, parametric tests were used for normally distributed variables, including Student's t-test for two-group comparisons and Analysis of Variance (ANOVA) for multiple group comparisons. For non-normally distributed variables, non-parametric alternatives were employed, specifically the Mann-Whitney U test for two-group comparisons and the Kruskal-Wallis test for multiple group comparisons.

Categorical variables were compared using the Chi-square test; Fisher's exact test was used instead for 2×2 tables when the expected cell count was below 5. For multiple group comparisons that showed statistical significance, post-hoc analyses were conducted with Bonferroni correction to identify specific between-group differences. The significance threshold was set at $p<0.05$ with a 95% confidence interval for all analyses.

RESULTS

Study Population

A total of 240 patients were included in this study: 80 patients with pulmonary embolism (PE group), 80 patients with cancer-associated pulmonary embolism (CA-PE group), and 80 patients with suspected PE in whom PE was excluded by imaging (control group). The demographic and clinical characteristics of the three groups are presented in **Table 1**.

Malignancy Characteristics

In the CA-PE group, the most frequent malignancies were lung cancer (22.5%) and brain tumors (15%), followed by multiple primary malignancies (11.3%). Prostate cancer (6.25%), breast cancer (5%), ovarian cancer (5%), and non-Hodgkin lymphoma (5%) were also commonly observed.

Of the patients with malignancy, 12 (15%) were in early stage, 28 (35%) in locally advanced stage, and 40 (50%) in metastatic stage. The most common metastatic sites were the liver (20.6%), bone (17.6%), and brain (14.4%). Fifty-four patients (67.5%) were receiving active chemotherapy, with platinum-based regimens being the most common (63% of chemotherapy patients). Radiotherapy had been administered to 43 patients (53.8%), and 36 patients (45%) had undergone cancer-related surgery.

The mean time between malignancy diagnosis and PE occurrence was 501.2±638.1 days. Fifty patients (62.5%) developed PE within the first year after cancer diagnosis. The mean time between the most recent chemotherapy and PE diagnosis was 93.07±272.1 days.

Demographic and Clinical Characteristics

The median age was 71.5 years (IQR 54.8-82.0) in the PE group, 69.0 years (IQR 56.8-76.0) in the CA-PE group, and 62.5 years (IQR 50.0-73.0) in the control group ($p=0.005$). The gender distribution was similar across groups ($p=0.432$). BMI was significantly higher in the control group (median 29.4 kg/m², IQR 27.1-33.5) than in the PE (26.6 kg/m², IQR 24.2-31.0) and CA-PE (25.4 kg/m², IQR 23.4-27.9) groups ($p=0.006$).

The most frequent presenting symptom in all groups was dyspnea, which was more common in the PE (97.5%) and CA-PE (90%) groups than in the control group (68.8%) ($p<0.001$). Chest pain was more prevalent in the control group (57.5%) than in the PE (36.3%) and CA-PE (27.5%) groups ($p<0.001$). No significant differences were observed in the frequency of syncope ($p=0.776$) or hemoptysis ($p=0.707$) among the three groups. The median symptom duration was similar across groups ($p=0.823$).

Table 1. Comparative demographic and general clinical characteristics of patients

General characteristics	PE (n=80)	CA-PE (n=80)	Control (n=80)	P
Age, years, median (IQR)	71.5 (54.8-82.0)	69.0 (56.8-76.0)	62.5 (50.0-73.0)	0.005***
Gender, n (%)				0.432**
Female	46 (57.5)	43 (53.8)	51 (63.8)	
Male	34 (42.5)	37 (46.2)	29 (36.2)	
BMI, kg/m^2, median (IQR)	26.6 (24.2-31.0)	25.4 (23.4-27.9)	29.4 (27.1-33.5)	0.006***
Symptoms				
Chest pain, n (%)	29 (36.3)	22 (27.5)	46 (57.5)	<0.001**
Syncope, n (%)	5 (6.3)	7 (8.8)	5 (6.3)	0.776**
Dyspnea, n (%)	78 (97.5)	72 (90)	55 (68.8)	<0.001**
Hemoptysis, n (%)	3 (3.8)	4 (5)	2 (2.5)	0.707**
Symptom duration, days, median (IQR)	4.0 (2.0-7.0)	4.0 (2.0-7.0)	3.0 (1.0-10.0)	0.823***
Vital Signs				
Systolic BP, mmHg, mean±SD	132.8±27.5	127.2±22.9	148.3±29.2	<0.001*
Diastolic BP, mmHg, Median (IQR)	70.0 (60.0-81.2)	70.0 (60.0-83.2)	79.0 (67.8-88.2)	0.004***
Shock index, median (IQR)	0.70 (0.60-0.93)	0.80 (0.70-0.93)	0.60 (0.50-0.70)	<0.001***
Shock index >0.9, n (%)	20 (25.0)	20 (25.0)	6 (7.5)	0.005**
Heart rate, /min, median (IQR)	96.0 (81.0-114.2)	105.0 (91.5-118.0)	88.5 (81.8-100.2)	<0.001***
Oxygen saturation, %, median (IQR)	94.0 (87.0-96.0)	89.0 (85.0-95.0)	97.0 (94.0-98.0)	<0.001***
ECOG, median (IQR)	1.0 (0.0-3.0)	2.0 (1.0-3.0)	1.0 (0.0-1.0)	<0.001***
CCI score, median (IQR)	4.5 (2.8-6.0)	9.0 (7.0-10.0)	2.5 (1.0-4.0)	<0.001***

p<0.05 was considered statistically significant. *: Analysis of Variance; **: Chi-square test; ***: Kruskal-Wallis test. Values are presented as mean±SD for variables analyzed with Analysis of Variance (*) and as median (interquartile range) for variables analyzed with the Kruskal-Wallis test (***). PE: Pulmonary embolism, CA-PE: Cancer-associated pulmonary embolism, BMI: Body-mass index, IQR: Interquartile range, BP: Blood pressure, SD: Standard deviation, ECOG: Eastern Cooperative Oncology Group, CCI: Charlson Comorbidity Index

Vital sign measurements showed significant differences between groups. The control group had higher systolic blood pressure (p<0.001) and a lower shock index (p<0.001) than the PE and CA-PE groups. Oxygen saturation was significantly lower in the CA-PE group (median 89.0%, IQR 85.0-95.0) than in the PE group (94.0%, IQR 87.0-96.0) and the control group (97.0%, IQR 94.0-98.0) (p<0.001).

The CA-PE group had higher ECOG performance status scores (median 2.0, IQR 1.0-3.0) and CCI scores (median 9.0, IQR 7.0-10.0) than the PE group (1.0, IQR 0.0-3.0 and 4.5, IQR 2.8-6.0, respectively) and the control group (1.0, IQR 0.0-1.0 and 2.5, IQR 1.0-4.0, respectively) (p<0.001 for both comparisons).

Comorbidities and Risk Factors

Comorbidities and risk factors for VTE in the study groups are presented in **Table 2**. Hypertension was more prevalent in the PE group (58.8%) than in the CA-PE group (37.5%) (p=0.007). Similarly, chronic kidney disease was more common in the PE group (18.8%) than in the CA-PE group (7.5%) (p=0.035). The prevalence of the other evaluated comorbidities, including diabetes mellitus, coronary artery disease, COPD, atrial fibrillation and chronic heart failure, did not differ significantly between the PE and CA-PE groups (**Table 2**).

Regarding risk factors for thromboembolism, immobilization was the most common risk factor in both the PE (52.5%) and CA-PE (57.5%) groups, with no significant difference between groups (p=0.525). History of previous PE was noted in 15% of PE patients and 6.3% of CA-PE patients (p=0.073). Recent surgery (within the last month) was equally prevalent in both groups (10%, p=1.0). Blood transfusion was significantly more

Table 2. Comorbidities and risk factors for venous thromboembolism

	PE (n=80)	CA-PE (n=80)	Control (n=80)	p (PE vs. CA-PE)
Comorbidities				
Hypertension, n (%)	47 (58.8)	30 (37.5)	41 (51.3)	0.007*
Diabetes mellitus, n (%)	18 (22.5)	14 (17.5)	20 (25.0)	0.429*
Chronic kidney disease, n (%)	15 (18.8)	6 (7.5)	5 (6.3)	0.035*
Coronary artery disease, n (%)	12 (15.0)	9 (11.3)	16 (20.0)	0.482*
COPD, n (%)	8 (10.0)	6 (7.5)	8 (10.0)	0.576*
Asthma, n (%)	10 (12.5)	5 (6.3)	13 (16.3)	0.175*
Atrial fibrillation, n (%)	8 (10.0)	3 (3.8)	4 (5.0)	0.118*
Chronic heart failure, n (%)	7 (8.8)	2 (2.5)	13 (16.3)	0.167**
Previous tuberculosis, n (%)	2 (2.5)	2 (2.5)	2 (2.5)	1.000**
Rheumatological disease, n (%)	3 (3.8)	1 (1.3)	4 (5.0)	0.620**
Risk factors for VTE				
Immobilization, n (%)	42 (52.5)	46 (57.5)	11 (13.8)	0.525*
Previous PE, n (%)	12 (15.0)	5 (6.3)	2 (2.5)	0.073*
Recent surgery (<1 month), n (%)	8 (10.0)	8 (10.0)	0 (0)	1.000*
Blood transfusion, n (%)	0 (0)	22 (27.5)	0 (0)	<0.001*
Central venous catheter, n (%)	1 (1.3)	8 (10.0)	0 (0)	0.034**
Trauma, n (%)	5 (6.3)	1 (1.3)	1 (1.3)	0.210**
Previous COVID-19, n (%)	5 (6.3)	0 (0)	0 (0)	0.059**
Erythropoietin use, n (%)	2 (2.5)	0 (0)	0 (0)	0.497**
Superficial vein thrombosis, n (%)	4 (5.0)	0 (0)	-	0.120**
Postpartum period, n (%)	2 (2.5)	0 (0)	0 (0)	0.497**
Known thrombophilia, n (%)	6 (7.5)	2 (2.5)	0 (0)	0.276**

p<0.05 was considered statistically significant. P values refer to the comparison between the PE and CA-PE groups. *: Chi-square test; **: Fisher's exact test. PE: Pulmonary embolism, CA-PE: Cancer-associated pulmonary embolism, VTE: Venous thromboembolism, COPD: Chronic obstructive pulmonary disease

common in the CA-PE group (27.5%) than in the PE group, in which no patients received transfusions (p<0.001).

Laboratory Parameters

Comparison of laboratory parameters between groups revealed significant differences (**Table 3**). D-dimer levels were significantly higher in both PE groups than in the control group (p<0.001), but did not differ significantly between the PE and CA-PE groups. Leukocyte and neutrophil counts were higher in both PE groups than in controls (p<0.001), while lymphocyte counts were lower in the PE and CA-PE groups than in controls (p<0.001).

Inflammatory indices showed marked differences between groups. The NLR was significantly higher in the CA-PE group (median 5.63, IQR 2.70-10.98) than in the PE group (3.17, IQR 2.14-5.92) and the control group (2.00, IQR 1.55-2.96) (p<0.001). Similarly, the monocyte-to-lymphocyte ratio was highest in the CA-PE group (0.47, IQR 0.29-0.78), followed by the PE group (0.36, IQR 0.23-0.52) and control group (0.29, IQR 0.20-0.39) (p<0.001). The PLR followed the same pattern, with higher values in the CA-PE group (166.4, IQR 109.3-286.8) than in the PE group (127.2, IQR 89.7-165.9) and the control group (115.3, IQR 96.5-160.6) (p=0.004) (**Figure**).

The CA-PE group had significantly lower hemoglobin levels (11.3±1.9 g/dl) than the PE group (12.3±2.2 g/dl) and the control group (12.7±1.6 g/dl) (p<0.001). Red cell distribution width (RDW) was higher in the CA-PE group (median 15.0%, IQR 14.0-17.1) than in the PE group (13.9%, IQR 12.8-15.8) and the control group (13.5%, IQR 12.8-14.8) (Kruskal-Wallis p<0.001).

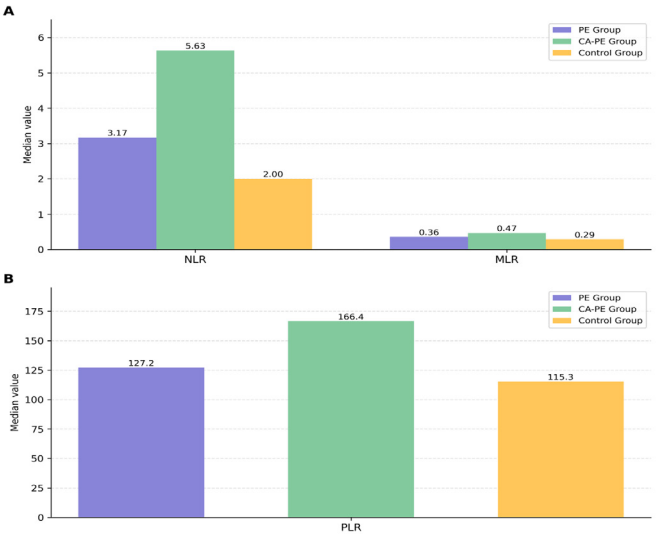


Figure. Comparison of inflammatory indices (NLR, MLR, PLR) between the PE, CA-PE, and control groups. (A) NLR and MLR; (B) PLR. Values are presented as medians. The CA-PE group showed significantly higher values for all three inflammatory indices than the PE and control groups (p<0.05)
NLR: Neutrophil-to-lymphocyte ratio, MLR: Monocyte-to-lymphocyte ratio, PLR: Platelet-to-lymphocyte ratio, PE: Pulmonary embolism, CA-PE: Cancer-associated pulmonary embolism

Lactate dehydrogenase (LDH) levels were significantly higher in the CA-PE group (median 344.5 U/L, IQR 238.8-490.0) than in the PE group (258.5 U/L, IQR 212.5-311.2) and the control group (202.5 U/L, IQR 173.0-246.8) (p<0.001). CRP levels were markedly higher in both the CA-PE (88.77 mg/L, IQR 38.46-119.75) and PE groups (30.53 mg/L, IQR 8.61-98.82) than in the control group (5.98 mg/L, IQR 0.99-9.41) (p<0.001).

Cardiac biomarkers showed that troponin-I levels were higher in both PE groups than in controls (p<0.001), but no

Table 3. Evaluation of laboratory values according to diagnostic groups

Laboratory Parameters	PE	CA-PE	Control	p
D-dimer, ng/ml	2298.5 (879.8-10557.8)	1996.5 (899.0-9954.8)	1220.0 (807.5-1647.5)	<0.001***
Leukocyte, 10^3/µL	9.70 (7.64-12.49)	9.77 (7.23-13.98)	7.68 (6.13-9.54)	<0.001***
Lymphocyte, 10^3/µL	1.86 (1.46-2.37)	1.40 (0.78-2.01)	2.09 (1.56-2.75)	<0.001***
Neutrophil, 10^3/µL	6.63 (4.84-8.97)	7.17 (5.25-10.84)	4.50 (3.59-5.96)	<0.001***
Neutrophil/Lymphocyte ratio	3.17 (2.14-5.92)	5.63 (2.70-10.98)	2.00 (1.55-2.96)	<0.001***
SII index value	839.4 (467.2-1571.0)	1214.0 (487.6-2687.7)	507.9 (351.9-827.0)	<0.001***
Monocyte, 10^3/µL	0.64 (0.51-0.96)	0.64 (0.45-0.93)	0.64 (0.52-0.77)	0.610***
Monocyte/lymphocyte ratio	0.36 (0.23-0.52)	0.47 (0.29-0.78)	0.29 (0.20-0.39)	<0.001***
Hemoglobin, g/dl, mean±SD	12.3±2.2	11.3±1.9	12.7±.6	<0.001*
RDW, %	13.9 (12.8-15.8)	15.0 (14.0-17.1)	13.5 (12.8-14.8)	<0.001***
Platelet, 10^3/µL	229.5 (178.2-294.8)	229.0 (153.2-313.0)	257.0 (216.8-308.8)	0.069***
Platelet/lymphocyte ratio	127.2 (89.7-165.9)	166.4 (109.3-286.8)	115.3 (96.5-160.6)	0.004***
LDH, U/L	258.5 (212.5-311.2)	344.5 (238.8-490.0)	202.5 (173.0-246.8)	<0.001***
CRP, mg/L	30.53 (8.61-98.82)	88.77 (38.46-119.75)	5.98 (0.99-9.41)	<0.001***
Albumin, g/dl, mean±SD	3.6±0.6	3.4±0.6	4.1±0.4	0.013*
Troponin-I, ng/L	10.6 (4.7-57.9)	14.2 (4.0-46.7)	2.5 (2.5-11.4)	<0.001***
CK-MB, µg/L	1.00 (0.40-1.77)	0.70 (0.40-1.50)	0.80 (0.30-1.80)	0.791***
BNP, ng/L	62.6 (24.4-200.0)	78.8 (26.2-177.4)	306.1 (56.6-754.6)	0.303***
Serum creatinine, mg/dl	0.93 (0.81-1.08)	0.79 (0.67-0.95)	0.84 (0.71-0.98)	0.001***
GFR, ml/min/1.73m^2, mean±SD	73.2±28.2	82.8±22.5	81.9±18.06	0.017*

p<0.05 was considered statistically significant. *: Analysis of Variance; **: Chi-square test; ***: Kruskal-Wallis test. Values are presented as mean±SD for variables analyzed with Analysis of Variance (*) and as median (interquartile range) for variables analyzed with the Kruskal-Wallis test (***). PE: Pulmonary embolism, CA-PE: Cancer-associated pulmonary embolism, SII: Systemic Immune-Inflammation Index, RDW: Red cell distribution width, LDH: Lactate dehydrogenase, CRP: C-reactive protein, CK-MB: Creatine kinase-MB, BNP: B-type natriuretic peptide, GFR: Glomerular filtration rate, SD: Standard deviation, Min: Minute

significant difference was observed between the PE and CA-PE groups. BNP and CK-MB levels did not differ significantly among the three groups.

Risk Stratification and Clinical Scores

Clinical probability, severity and prognostic scores are summarized in **Table 4**. The CA-PE group had significantly higher Wells scores (median 7.0 [IQR 5.5-7.0] vs. 6.0 [IQR 4.5-7.5], p=0.011) and Modified Geneva scores (8.0 [7.0-13.0] vs. 6.0 [5.0-10.0], p<0.001) than the PE group. When categorized by Wells score, a significantly higher proportion of CA-PE patients (56.3%) had high probability scores (>6) than PE patients (27.5%) (p<0.001).

Table 4. Clinical probability, severity and prognostic scores in the PE and CA-PE groups

	PE (n=80)	CA-PE (n=80)	P
Clinical probability			
Wells score, median (IQR)	6.0 (4.5-7.5)	7.0 (5.5-7.0)	0.011***
Wells score >6 (high probability), n (%)	22 (27.5)	45 (56.3)	<0.001*
Modified Geneva score, median (IQR)	6.0 (5.0-10.0)	8.0 (7.0-13.0)	<0.001***
Severity and prognostic scores			
PESI score, median (IQR)	100.0 (72.0-131.5)	135.5 (113.0-158.2)	<0.001***
PESI class III-V (>85), n (%)	48 (60.0)	79 (98.8)	<0.001*
Bova score, median (IQR)	1.0 (0.0-2.2)	1.0 (0.0-2.0)	0.952***
Bova score >4, n (%)	7 (8.8)	5 (6.3)	0.548*
Clinical risk class, n (%)			<0.001*
High risk (massive)	6 (7.5)	6 (7.5)	
Intermediate risk (submassive)	52 (65.0)	73 (91.3)	
Low risk (non-massive)	22 (27.5)	1 (1.3)	
Cancer-specific scores (CA-PE group only)			
Khorana score, mean±SD	-	1.4±1.0	-
Khorana score ≥3 (high risk), n (%)	-	12 (15.0)	-
Pompe-C score, %, mean±SD	-	31.5±22.4	-
30-day mortality score, mean±SD	-	5.1±2.0	-
Modified Glasgow Prognostic Score, mean±SD	-	1.4±0.7	-

p<0.05 was considered statistically significant. *: Chi-square test; ***: Mann-Whitney U test. PE: Pulmonary embolism, CA-PE: Cancer-associated pulmonary embolism, PESI: Pulmonary Embolism Severity Index, IQR: Interquartile range, SD: Standard deviation

Among cancer patients, the mean Khorana score was 1.4±1.0, with 12 patients (15%) classified as high risk (score ≥3). The PESI score was significantly higher in the CA-PE group (median 135.5, IQR 113.0-158.2) than in the PE group (100.0, IQR 72.0-131.5) (p<0.001). Additionally, a significantly higher proportion of CA-PE patients (98.8%) had high-risk PESI scores (>85) than PE patients (60%) (p<0.001). However, Bova scores were similar between the two groups (p=0.95), as was the proportion of patients with high-risk Bova scores (>4) (p=0.548). According to the guideline-based clinical classification, the proportion of high-risk PE was identical in both groups (7.5%), whereas intermediate-risk PE was more frequent (91.3% vs. 65.0%) and low-risk PE less frequent (1.3% vs. 27.5%) in the CA-PE group (p<0.001).

In the CA-PE group, the mean Pompe-C score was 31.5±22.4%, the mean 30-day mortality score was 5.1±2.01, and the mean Modified Glasgow Prognostic Score was 1.4±0.7. Interestingly, PESI scores differed across cancer stages (p=0.033), with the lowest values in patients with metastatic disease (130.7±26.6) and the highest in those with locally advanced disease (148.6±31.5).

Echocardiographic and Radiological Findings

Echocardiography was performed in 168 patients (70%): 78 in the PE group, 69 in the CA-PE group and 21 in the control group. Right ventricular dysfunction was detected in 44 of these patients (26.2%). Pulmonary artery pressure (PAP) could be measured in 103 patients (median 35.0 mmHg, IQR 25.0-49.5), 79 of whom (76.7%) had a PAP of ≥25 mmHg. Lower-extremity Doppler ultrasonography was performed in 103 patients, all of whom were in the PE groups (59 in the PE group and 44 in the CA-PE group), and deep vein thrombosis was detected in 48 of them (46.6%). Both examinations were performed in 100 patients (58 in the PE group and 42 in the CA-PE group).

Among the 150 patients diagnosed by CTPA, bilateral emboli were found in 97 patients (64.7%) and unilateral in 53 patients (35.3%). Emboli were more frequently detected in the right pulmonary arterial system (n=133, 88.7%) than in the left (n=114, 76.0%). The pulmonary trunk was numerically more commonly affected in the CA-PE group (10.5%) than in the PE group (2.7%), but the difference was not statistically significant (p=0.098). Other locations showed similar distributions between groups (**Table 5**).

Among patients who underwent echocardiography, right ventricular dysfunction was numerically more frequent in the PE group (34.6%) than in the CA-PE group (23.2%), but the difference was not statistically significant (p=0.129). Among patients with measured PAP, the proportion with a PAP of ≥25 mmHg was similar in the two groups (76.4% vs. 75.0%, p=0.878).

Treatment and Outcomes

Treatment characteristics and outcomes are summarized in **Table 6**. Low molecular weight heparin was the most common initial treatment in both the PE (95%) and CA-PE (100%) groups. For long-term anticoagulation, DOACs were more frequently used in the PE group (42.5%) than in the CA-PE group (23.8%) (p=0.012). Similarly, vitamin K antagonists were more commonly prescribed in the PE group (30%) than in the CA-PE group (5%) (p<0.001). Thrombolytic therapy was administered to similar proportions of patients in the PE (6.3%) and CA-PE (7.5%) groups (p=0.755).

Hospital length of stay did not differ significantly between the PE (median 6.0 days, IQR 4.0-8.2) and CA-PE (7.5 days, IQR 4.0-12.0) groups (p=0.105). ICU admission was required in 13 (16.3%) patients in the PE group and 10 (12.5%) in the CA-PE group (p=0.499); among these patients, ICU stay was longer in the CA-PE group (median 5.0 days, IQR 4.0-14.2) than in the PE group (3.0 days, IQR 2.0-4.0) (p=0.011).

Table 5. Echocardiographic and radiological characteristics of PE patient groups

	PE, n (%)	CA-PE, n (%)	p
DVT†	23 (39.0)	25 (56.8)	0.073*
Unilateral	19 (32.2)	19 (43.2)	0.253*
Bilateral	4 (6.8)	6 (13.6)	0.319**
Echocardiography findings			
Right ventricular dysfunction‡, n (%)	27 (34.6)	16 (23.2)	0.129*
PAP§, mmHg, median (IQR)	40.0 (26.0-53.0)	34.0 (24.5-47.2)	0.258***
PAP ≥25 mmHg§	42 (76.4)	30 (75.0)	0.878*
Embolism Location¶			
Unilateral	23 (31.1)	30 (39.5)	0.282*
Bilateral	51 (68.9)	46 (60.5)	0.282*
Right pulmonary arterial system	65 (87.8)	68 (89.5)	0.752*
Left pulmonary arterial system	60 (81.1)	54 (71.1)	0.150*
Pulmonary trunk	2 (2.7)	8 (10.5)	0.098**
Main pulmonary artery	36 (48.6)	35 (46.1)	0.750*
Lobar artery	21 (28.4)	20 (26.3)	0.777*
Segmental artery	9 (12.2)	6 (7.9)	0.384*
Subsegmental artery	6 (8.1)	7 (9.2)	0.810*
Pulmonary infarction	6 (8.1)	2 (2.6)	0.164**

p<0.05 was considered statistically significant. *: Chi-square test; **: Fisher's exact test; ***: Mann–Whitney U test. Percentages were calculated among patients with the relevant examination: †lower-extremity Doppler ultrasonography (PE, n=59; CA-PE, n=44); ‡echocardiography (n=78; n=69); §measured PAP (n=55; n=40); ¶diagnosis by CTPA (n=74; n=76). PE: Pulmonary embolism, CA-PE: Cancer-associated pulmonary embolism, DVT: Deep vein thrombosis, PAP: Pulmonary artery pressure, CTPA: Computed tomography pulmonary angiography, IQR: Interquartile range, SD: Standard deviation

Table 6. Treatment characteristics and outcomes in the PE and CA-PE groups

	PE (n=80)	CA-PE (n=80)	p
Treatment			
Initial LMWH, n (%)	76 (95.0)	80 (100)	0.120**
Thrombolytic therapy, n (%)	5 (6.3)	6 (7.5)	0.755*
Long-term DOAC, n (%)	34 (42.5)	19 (23.8)	0.012*
Long-term VKA, n (%)	24 (30.0)	4 (5.0)	<0.001*
Hospital course			
Hospital length of stay, days, median (IQR)	6.0 (4.0-8.2)	7.5 (4.0-12.0)	0.105***
ICU admission, n (%)	13 (16.3)	10 (12.5)	0.499*
ICU length of stay, days, median (IQR)†	3.0 (2.0-4.0)	5.0 (4.0-14.2)	0.011***
All-cause mortality			
30-day, n (%)	6 (7.5)	13 (16.3)	0.087*
3-month, n (%)	10 (12.5)	30 (37.5)	<0.001*
1-year, n (%)	13 (16.3)	50 (62.5)	<0.001*

p<0.05 was considered statistically significant. *: Chi-square test; **: Fisher's exact test; ***: Mann–Whitney U test. †Among patients admitted to the ICU (PE, n=13; CA-PE, n=10). PE: Pulmonary embolism, CA-PE: Cancer-associated pulmonary embolism, LMWH: Low-molecular-weight heparin, DOAC: Direct oral anticoagulant, VKA: Vitamin K antagonist, ICU: Intensive care unit, IQR: Interquartile range

Regarding mortality outcomes, 30-day mortality rates were not significantly different between the PE (7.5%) and CA-PE (16.3%) groups (p=0.087). However, both 3-month mortality (37.5% vs. 12.5%, p<0.001) and 1-year mortality (62.5% vs. 16.3%, p<0.001) were significantly higher in the CA-PE group than in the PE group.

DISCUSSION

This study compared the clinical features, inflammatory markers, severity scores, and outcomes of PE in patients with and without malignancy. Unlike previous studies comparing cancer patients with and without VTE, our direct comparison of CA-PE and PE without malignancy highlights how cancer influences PE presentation, severity and outcomes.

CA-PE patients had higher inflammatory indices and clinical risk scores, whereas clinical presentation and markers of embolism severity were similar. Although 30-day mortality was similar, long-term all-cause mortality was higher in the CA-PE group; because cause-specific mortality was not evaluated, this difference cannot be attributed to a specific cause.

Lung and brain cancers were the most common malignancies in the CA-PE group, in line with studies identifying these as high-risk malignancies.[1,10] Most CA-PE cases occurred within the first year after cancer diagnosis, consistent with the known timing of peak VTE risk.[11]

Presenting symptoms were generally similar between the two PE groups, with dyspnea being predominant. Lin et al.[12] reported that cancer patients with PE were less likely to present with chest pain; in our cohort, chest pain was numerically less frequent in the CA-PE group, although the difference did not reach statistical significance. Oxygen saturation was lower in the CA-PE group, which may reflect more pronounced respiratory compromise during the PE event; however, pulmonary involvement by the underlying malignancy may also have contributed.

The higher Wells and Modified Geneva scores in the CA-PE group are likely explained by the fact that both scoring systems assign points for malignancy, highlighting the need for cancer-specific risk assessment tools. The Khorana score classified only 15% of our CA-PE cohort as high-risk, indicating its limited utility. This is consistent with other studies reporting its low sensitivity (40%) but high specificity (88%).[8]

A notable finding of our study was that NLR, MLR and PLR values were higher in the CA-PE group than in the PE group. Several studies have shown that elevated NLR and PLR are associated with increased VTE risk in cancer patients,[13,14] and Ferroni et al.[15] found that high NLR and PLR values were associated with an approximately twofold increased risk of VTE in ambulatory cancer patients. Nevertheless, these findings should be interpreted with caution. Inflammatory indices derived from the complete blood count are not specific to thrombosis and may be influenced by the malignancy itself, tumor burden, chemotherapy- or radiotherapy-related myelosuppression, corticosteroid use, infections and other concomitant inflammatory conditions; notably, two-thirds of our CA-PE patients were receiving active chemotherapy. In addition, because these indices were measured only at the time of PE diagnosis, our data cannot determine whether elevated inflammatory indices contribute to thrombosis or merely reflect the underlying malignancy. Our findings are therefore consistent with, but do not prove, a role of inflammation in cancer-associated thrombosis.

Despite higher PESI scores in the CA-PE group, right ventricular dysfunction on echocardiography was not more frequent in these patients; it was even numerically less frequent, although this difference was not statistically significant. Similarly, the higher proportion of intermediate-risk PE in the CA-PE group probably reflects the contribution of PESI to guideline-based risk classification rather than a true difference in hemodynamic severity. This discrepancy between risk scores and cardiac findings suggests that the PESI score may overestimate PE severity in cancer patients, as it assigns points for malignancy without considering cancer type, stage or treatment status. Li et al.[16] compared different clinical prognostic scores in patients with PE and active cancer and concluded that existing scoring systems have limited value in predicting outcomes in this population.

Pulmonary trunk embolism was numerically more frequent in the CA-PE group, although the difference was not statistically significant. A higher risk of central PE in cancer patients has also been reported by Karippot et al.[17] Whether this reflects cancer-specific procoagulant mechanisms, such as the expression of tissue factor and cancer procoagulant and the release of procoagulant microparticles,[6] remains speculative and requires confirmation in larger studies.

The treatment approach differed between the groups. While low molecular weight heparin (LMWH) was the initial treatment in both groups, DOACs and VKAs were more frequently used for long-term treatment in the PE group. This practice aligns with guidelines recommending LMWH for cancer patients.[18] However, recent studies have shown the effectiveness and safety of DOACs in cancer patients, which might influence future practice.[19]

The most striking difference between the groups was in long-term outcomes. While 30-day mortality did not differ significantly between the CA-PE and PE groups, both 3-month and 1-year mortality were markedly higher in the CA-PE group. This finding is consistent with previous studies showing that cancer patients with VTE have a poorer overall prognosis; Sorensen et al.[4] reported a one-year survival rate of only 12% in cancer patients diagnosed with VTE compared with 36% in cancer patients without VTE. However, as cause-specific mortality was not evaluated in our study, we cannot determine whether the excess long-term mortality was due to cancer progression, recurrent VTE, bleeding or other causes. Our results indicate that, while short-term mortality was similar in both groups, long-term all-cause mortality was higher in patients with malignancy.

Interestingly, the PESI score was lower in patients with metastatic cancer than in those with locally advanced disease. This counterintuitive finding might be related to the age distribution in these subgroups or other factors not captured by the PESI score, further highlighting the limitations of existing risk assessment tools in cancer patients. Au et al.[20] similarly reported that cancer stage was not a significant predictor of short-term outcomes in cancer patients with PE.

Limitations

Our study has several limitations. First, its retrospective design may have led to incomplete reporting of symptoms and misclassification of outcomes. Second, the inclusion of only hospitalized patients might have introduced selection bias, as patients with milder symptoms treated in outpatient settings were not included. Third, to obtain groups of equal size, 80 patients were randomly selected for each group from the eligible population; although random selection limits deliberate selection, the equal group sizes do not reflect the true relative frequency of PE with and without malignancy at our center, and selection bias cannot be completely excluded. Fourth, the heterogeneity of malignancy types and treatments in the CA-PE group might have obscured specific associations between certain cancer types and PE characteristics. Fifth, cause-specific mortality was not evaluated; therefore, the higher long-term mortality in the CA-PE group cannot be attributed to cancer progression or to PE itself. Sixth, inflammatory indices may have been influenced by tumor burden, cancer treatment and concomitant inflammatory conditions, which could not be fully accounted for. Seventh, echocardiography, PAP measurement and lower-extremity Doppler ultrasonography were not performed in all patients, and right ventricular function was assessed qualitatively, which might have varied between observers. Eighth, inherited thrombophilia was not screened systematically, and the contribution of genetic predisposition could not be assessed. Ninth, our dataset did not distinguish between symptomatic and incidentally detected (asymptomatic) PE, which is common in oncology patients undergoing routine staging or surveillance imaging.[21] Incidentally detected PE has been reported to be associated with less severe echocardiographic findings in some studies, and its inclusion in the CA-PE group may partly explain why right ventricular dysfunction was not more frequent despite higher risk scores in this group; future studies should record and analyze this distinction separately. Finally, our sample size, although comparable to previous studies in this field, may have limited the statistical power to detect smaller differences between groups.

CONCLUSION

In this study, PE in patients with malignancy was associated with higher inflammatory indices and higher malignancy-weighted risk scores, but not with worse echocardiographic indicators of PE severity or higher 30-day mortality compared with PE in patients without malignancy. Long-term all-cause mortality, however, was substantially higher in patients with malignancy. These findings suggest that commonly used risk scores may overestimate PE severity in cancer patients, and they support the development of cancer-specific risk stratification tools and close long-term follow-up of these patients.

ETHICAL DECLARATIONS

Ethics Committee Approval

This study was approved by the Non-interventional Researches Ethics Committee of Bezmialem Vakıf University (Date: 26.07.2023, Decision No: 2023/232).

Informed Consent

The requirement for written informed consent was waived by the ethics committee because of the retrospective design and the use of anonymized data. No additional intervention was performed, and there was no direct patient contact.

Peer Review Process
This manuscript was subject to external peer review.

Conflict of Interest
The authors declare no conflicts of interest related to this study.

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Author Contributions
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REFERENCES
1. Wendelboe AM, Raskob GE. Global burden of thrombosis: epidemiologic aspects. *Circ Res*. 2016;118(9):1340-1347. doi:10.1161/CIRCRESAHA.115.306841
2. Thompson BT, Kabrhel C. Overview of acute pulmonary embolism in adults. *UpToDate* [Internet]. Waltham (MA): UpToDate; 2016.
3. Blom JW, Doggen CJ, Osanto S, Rosendaal FR. Malignancies, prothrombotic mutations, and the risk of venous thrombosis. *JAMA*. 2005;293(6):715-722. doi:10.1001/jama.293.6.715
4. Sorensen HT, Mellemkjaer L, Olsen JH, Baron JA. Prognosis of cancers associated with venous thromboembolism. *N Engl J Med*. 2000;343(25):1846-1850. doi:10.1056/NEJM200012213432504
5. Abdol Razak NB, Jones G, Bhandari M, Berndt MC, Metharom P. Cancer-associated thrombosis: an overview of mechanisms, risk factors, and treatment. *Cancers (Basel)*. 2018;10(10):380. doi:10.3390/cancers10100380
6. Falanga A, Marchetti M, Vignoli A. Coagulation and cancer: biological and clinical aspects. *J Thromb Haemost*. 2013;11(2):223-233. doi:10.1111/jth.12075
7. Streiff MB, Holmstrom B, Angelini D, et al. Cancer-associated venous thromboembolic disease, version 2.2021, NCCN clinical practice guidelines in oncology. *J Natl Compr Canc Netw*. 2021;19(10):1181-1201. doi:10.6004/jnccn.2021.0047
8. Khorana AA, Kuderer NM, Culakova E, Lyman GH, Francis CW. Development and validation of a predictive model for chemotherapy-associated thrombosis. *Blood*. 2008;111(10):4902-4907. doi:10.1182/blood-2007-10-116327
9. Key NS, Khorana AA, Kuderer NM, et al. Venous thromboembolism prophylaxis and treatment in patients with cancer: ASCO guideline update. *J Clin Oncol*. 2023;41(16):3063-3071. doi:10.1200/JCO.23.00294
10. Chew HK, Wun T, Harvey D, Zhou H, White RH. Incidence of venous thromboembolism and its effect on survival among patients with common cancers. *Arch Intern Med*. 2006;166(4):458-464. doi:10.1001/archinte.166.4.458
11. Blom JW, Vanderschoot JP, Oostindiër MJ, Osanto S, van der Meer FJ, Rosendaal FR. Incidence of venous thrombosis in a large cohort of 66,329 cancer patients: results of a record linkage study. *J Thromb Haemost*. 2006;4(3):529-535. doi:10.1111/j.1538-7836.2006.01804.x
12. Lin YC, Chen SC, Huang CM, et al. Clinical features and diagnosis of new malignancy in patients with acute pulmonary embolism and without a history of cancer. *J Chin Med Assoc*. 2020;83(3):245-250. doi:10.1097/JCMA.0000000000000259
13. Go SI, Lee A, Lee US, et al. Clinical significance of the neutrophil-lymphocyte ratio in venous thromboembolism patients with lung cancer. *Lung Cancer*. 2014;84(1):79-85. doi:10.1016/j.lungcan.2014.01.014
14. Yang W, Liu Y. Platelet-lymphocyte ratio is a predictor of venous thromboembolism in cancer patients. *Thromb Res*. 2015;136(2):212-215. doi:10.1016/j.thromres.2014.11.025
15. Ferroni P, Riondino S, Formica V, et al. Venous thromboembolism risk prediction in ambulatory cancer patients: clinical significance of neutrophil/lymphocyte ratio and platelet/lymphocyte ratio. *Int J Cancer*. 2015;136(5):1234-1240. doi:10.1002/ijc.29076
16. Li X, Hu Y, Lin P, et al. Comparison of different clinical prognostic scores in patients with pulmonary embolism and active cancer. *Thromb Haemost*. 2021;121(6):834-844. doi:10.1055/a-1355-3549
17. Karippot A, Maroules M, Debari V. Pulmonary embolism in cancer: is it different from patients without cancer? *Chest*. 2009;136(4 Suppl):147S.
18. Kearon C, Akl EA, Ornelas J, et al. Antithrombotic therapy for VTE disease: CHEST guideline and expert panel report. *Chest*. 2016;149(2):315-352. doi:10.1016/j.chest.2015.11.026
19. Raskob GE, van Es N, Verhamme P, et al. Edoxaban for the treatment of cancer-associated venous thromboembolism. *N Engl J Med*. 2018;378(7):615-624. doi:10.1056/NEJMoa1711948
20. Au C, Gupta E, Khaing P, et al. Clinical presentations and outcomes in pulmonary embolism patients with cancer. *J Thromb Thrombolysis*. 2021;51(2):430-436. doi:10.1007/s11239-020-02298-y
21. Tirandi A, Preda A, Carbone F, Montecucco F, Liberale L. Pulmonary embolism in patients with cancer: an updated and operative guide for diagnosis and management. *Int J Cardiol*. 2022;358:95-102. doi:10.1016/j.ijcard.2022.04.068