Published online 2023 December 15 **Original Article**

Assessment of Clinical Features and Related Risk Factors in Patients with Pulmonary Embolism in a Tertiary Teaching Center

Marjan Akhavan[1], Omalbanin Paknejad[2], Seyedeh Zahra Fotook Kiaei[2] and Mahnaz Pejman Sani[3*]

1. Department of Internal Medicine Shariati Hospital, Tehran University of Medical Sciences, Tehran, Iran
2. Department of Pulmonary and Critical Care, Shariati Hospital, Tehran University of Medical Sciences, Tehran, Iran
3. Endocrinology and Metabolism Research Center, Endocrinology and Metabolism Clinical Sciences Institute, Shariati Hospital, Tehran University of Medical Sciences, Tehran, Iran

*** *Corresponding author:*** Mahnaz Pejman Sani, Endocrinology and Metabolism Research Center, Endocrinology and Metabolism Clinical Sciences Institute, Shariati Hospital, Tehran University of Medical Sciences, Fifth Floor, North Kargar Ave, Tehran, Iran. Email: mpsani@sina.tums.ac.ir

Received 2022 December 11; **Revised** 2023 October 10; **Accepted** 2023 November 28.

Abstract

Background: Pulmonary thromboembolism (PTE) is a common and potentially life-threatening disease with manifestations similar to many other diseases, including myocardial infarction, pericarditis, myocarditis, and pneumonia.

Objectives: The present study aimed to assess clinical symptoms, some paraclinical parameters, and related risk factors in patients with confirmed pulmonary embolism.

Methods: In this retrospective study, the data of 709 patients who were admitted to Dr. Shariati Research and Treatment Center with confirmed diagnosis of PTE were examined over a period of three years.

Results: The mean age of patients was 56.48±17.84 years, and 47.2% of participants were female. The most common signs and symptoms were dyspnea (87.9), tachycardia (52.6%), and cough (41.9%), respectively. The most common comorbidities in the subjects entailed malignancy (51.1%), postoperative immobility (42%), and pneumonia (41.2%), respectively. Based on logistic regression analyses, congestive heart failure (CHF) followed by diabetes mellitus (DM) were the most common comorbidities associated with various clinical features of PTE. Moreover, clinical manifestations displayed some associations with electrocardiography (EKG) changes and venous blood gas (VBG) indices.

Conclusion: Despite its highly nonspecific clinical manifestations, the diagnosis of PTE remains a challenging issue. This study presented related signs and symptoms, clinical risk factors, comorbidities, and PTE-related EKG changes, which will help physicians properly approach and diagnose this life-threatening disease by considering all aspects.

Keywords: Clinical manifestation, Comorbidity, Pulmonary thromboembolism, Risk factor

1. Background

Pulmonary thromboembolism (PTE) is a common and potentially life-threatening condition associated with a high mortality rate whenever left untreated (1, 2.) The clinical symptoms of PTE are very nonspecific, comprising a wide range of features. Therefore, it is often undiagnosed, and more than 50% of patients are diagnosed after death (3). In general, the manifestations of PTE may be overlapped with other cardiovascular diseases or systemic disorders. More importantly, several cases can be asymptomatic, which is associated with a high patient mortality rate (4,5).

The clinical manifestations of PTE range from asymptomatic to sudden death, depending on the degree of pulmonary artery occlusion and the patient's cardiovascular status. The reported clinical manifestations include shortness of breath (82%), chest pain (49%), cough (20%), syncope (14%), and hemoptysis (7%) (6). The PTE is associated with serious comorbidities, including malignancies and ischemic heart disease, which result in the development of post-thrombotic syndrome (7,8). The etiology of PTE is often multifactorial. The known risk factors for PTE include recent surgery or trauma, immobility, malignancy, a history of venous thromboembolism (VTE), chronic disorders, stroke, pregnancy, oral contraceptives, hormone replacement therapy (HRT), central venous catheterization, cardiopulmonary disease, body mass index (BMI> 30 kg/m2), and smoking (1,9,10).

2. Objectives

A thorough knowledge of related clinical manifestations and a proper approach to the disease play a major role in the diagnosis and prognosis of PTE. The present study aimed to assess the clinical symptoms, some paraclinical parameters, and related risk factors in patients with a confirmed diagnosis of PTE.

3. Methods

This retrospective cross-sectional study was conducted at Dr. Shariati Hospital, affiliated with Tehran University of Medical Sciences, from 2016 to 2019. After excluding pregnant women and patients with incomplete data, a total of 709 hospitalized patients ≥ 18 years with PTE diagnosis eligible for the

study were obtained. The PTE diagnosis was made based on computed tomography pulmonary angiography (CTPA), or ventilation-perfusion scan, and/or history of DVT with pulmonary manifestations. The CTPA was performed using a 16-detector rapid CT scanner with four detector arrays (Siemens Medical Systems) while the patient was in the supine position for less than a second. During the imaging process, the patient was asked to hold his/her breath. Scan volume contained the entire chest from the top of the lung to the base in the cranio-caudal direction. The detector scan area was 40mm. The thickness of the slice was 0.625 mm, and the tube current and voltage were 145ma and 120kv, respectively.

A low-osmolality nonionic contrast agent (135-145 ml) was injected into the brachial vein at a flow rate of 5 ml/s. All required information was registered in the prepared forms. Based on the previous literature, the most common symptoms, signs, and risk factors related to PTE are as follows (1, 9-11). The reviewed and registered symptoms were chest pain, dyspnea, hemoptysis, cough, and syncope. The registered signs were hypoxia, tachycardia, and tachypnea. The related risk factors used in the study were history of immobility, history of PTE, malignancy, diabetes mellitus, chronic obstructive disease (COPD), congestive heart failure (CHF), and stroke.

3.1. Definition of terms

Hypoxia was diagnosed at an O2 saturation of <92% when breathing ambient air (< 90% for COPD patients). Tachycardia was defined as a heart rate of 100 or more beats per minute. Tachypnea was considered in the presence of a respiratory rate of 20 or more breaths per breath. Regardless of the etiology, immobility was defined as bed rest for more than three consecutive days in the previous month. Electrocardiography (EKG) changes were considered whenever the patient did not have normal sinus rhythm on electrocardiography.

3.2. Statistical analyses

Data were analyzed using SPSS statistical software (version 26.0). The distribution of quantitative variables was investigated by the Kolmogorov-Smirnov test. Continuous variables are presented as the mean±standard deviation (SD), and the categorical data as numbers and percentages. The independent t-test was used to compare the means. To examine the association between independent variables and PTE clinical symptoms, logistic regression analyses were performed. All P-values <0.05 were considered statistically significant.

4. Results

The baseline characteristics of the study population are presented in Table 1. The mean age of the study participants was 56.48±17.84 years (range: 15-95 years), and 47.2% of participants were female. The most common signs and symptoms were shortness of breath (87.9%), tachycardia (52.6%), and cough (41.9%), respectively. The most common comorbidities in the subjects were malignancy (51.1%), postoperative immobility (42%), and pneumonia (41.2%), respectively. About half of the patients were current smokers, and 27% of them had a BMI above 30 kg / m^2.

Table 1. Baseline characteristic of the study population

Variable	Value
Age (years)	56.48±17.84
Gender (F)	335 (47.2)
PTE signs and symptoms	
Cough	297 (41.9)
Dyspnea	594 (83.8)
Tachycardia	367 (51.8)
Chest pain	270 (38.1)
Syncope	40 (5.6)
Fever	180 (25.4)
Hypoxemia	173 (24.4)
Hemoptysis	83 (11.7)
Comorbidities	
Immobility	298 (42)
History of PTE	103 (14.5)
Malignancy	362 (51.1)
DM	228 (32.2)
COPD	124 (17.5)
CHF	79 (11.1)
AF	215 (30.7)
HTN	282 (39.8)
IBD	119 (16.8)
Stroke	245 (34.6)
Sepsis	126 (17.8)
Smoking	245 (34.6)
BMI≥ 30	191 (26.9)
EKG changes	
T-Invert	298 (42)
S1Q3T3	222 (31.3)
VBG Indices	
PH	7.34±0.24
HCO3(meq/L)	22.28± 3.64
PCO2(mmHg)	37.27±5.54
Echocardiography*	
Ejection fraction (%)	49.83±8.81
PAPP (mmHg)	37.06±12.07
RV dilation	161 (22.7)

Data are presented as mean (standard deviation) or frequency (%).
* Data are available for 526 patients.
PTE: Pulmonary thromboembolism; DM: diabetes mellitus; COPD: Chronic obstructive pulmonary disease; CHF: Congestive heart failure; AF: atrial fibrillation; HTN: Hypertension; IBD: Irritable bowel syndrome; BMI: Body mass index; EKG: Electrocardiography; PAPP: Pulmonary artery pressure; RV: Right ventricle

4.1. Association of comorbidities with clinical features of Pulmonary thromboembolism

The results of the association between comorbidities and clinical features of PTE are displayed in Table 2. Patients who did not have a history of stroke (CVA) were less likely to develop a cough, tachycardia, hemoptysis, and hypoxemia than those with CVA. The presence of CHF was associated with a lower probability of developing a cough,

syncope, and hypoxemia but a higher probability of developing chest pain and fever. Patients who did not experience atrial fibrillation (AF) were less likely to develop shortness of breath, syncope, and fever.

Malignancy had a significant negative association with shortness of breath and syncope but a positive association with hypoxemia. Patients without COPD were more likely to develop a cough but less likely to develop shortness of breath. Patients without diabetes were more likely to develop a cough, shortness of breath, and fever, and improbable to develop tachycardia and hypoxemia. Moreover, patients with a body mass index of less than 30 kg/m2 were less likely to develop tachycardia and hypoxemia. Current smokers were more at risk of developing hypoxemia. Patients without IBD and HTN were hardly likely to develop chest pain and fever, respectively.

Table 2. Association between pulmonary thromboembolism clinical characteristic and comorbidities in study participants using logistic regression model

	OR (95% CI)										
	Stroke	**Sepsis**	**IBD**	**CHF**	**COPD**	**HTN**	**Malignancy**	**DM**	**AF**	**Smoking**	**BMI≥30**
Cough	0.68 (0.50, 0.94) *	0.73 (0.49, 1.10)	0.88 (0.59, 1.31)	0.58(0.36, 0.93) *	2.24(1.45,3.45) *	1.13(0.83, 1.54)	1.22(0.91, 1.65)	1.46(1.06,2.03) *	0.82(0.59,1.13)	1.30(0.96, 1.75)	1.00(0.71,1.41)
Dyspnea	0.96 (0.63, 1.47)	1.53 (0.85, 2.73)	0.77 (0.43, 1.36)	1.36(0.75, 2.45)	0.30(0.14,0.64)*	1.01(0.67,1.52)	0.63(0.42, 0.95) *	1.64(1.09,2.48) *	0.61(0.38,0.98) *	0.69(0.46, 1.03)	0.75(0.47,1.21)
Tachycardia	0.71 (0.52, 0.98) *	1.86 (1.25, 2.78) *	0.90(0.61,1.34)	0.70(0.43,1.13)	1.00(0.68,1.48)	0.97(0.72,1.31)	0.88(0.65, 1.18)	0.64(0.46, 0.88) *	0.74(0.54,1.03)	0.89(0.66,1.20)	0.64(0.42,0.89) *
Hemoptysis	0.41 (0.26, 0.66) *	1.32 (0.75, 2.32)	0.82(0.46,1.48)	0.70(0.36, 1.37)	0.95(0.52, 1.73)	0.84(0.53, 1.34)	0.82(0.51, 1.29)	0.86(0.53, 1.40)	0.80(0.49,1.29)	0.73(0.46,1.15)	1.02(0.61,1.72)
Chest pain	0.81 (0.59, 1.12)	1.81 (1.23, 2.67) *	0.64(0.43,0.95) *	2.09(1.21, 3.58) *	1.14(0.76,1.71)	0.87(0.63,1.18)	0.84(0.62,1.14)	1.20(0.87, 1.67)	1.14(0.82,1.59)	0.95(0.70,1.29)	1.16(0.79,1.57)
Syncope	0.62 (0.33, 1.19)	0.80 (0.33, 1.96)	2.58(0.78,8.53)	0.22(0.11, 0.46) *	0.53(0.26,1.10)	0.64(0.34,1.22)	0.37(0.18,0.76) *	1.96(0.88,4.32)	0.46(0.24,0.88) *	0.68(0.35,1.29)	0.59(0.3, 1.15)
Fever	0.94 (0.66, 1.34)	3.22 (2.15, 4.82) *	1.12(0.71,1.79)	2.03(1.07, 3.84) *	1.55(0.81,2.98)	0.62(0.44, 0.87) *	0.91(0.65,1.27)	1.58(1.08,2.32) *	0.60(0.42,0.86) *	0.72(0.51,1.02)	0.90(0.61,1.31)
hypoxemia	2.54 (1.68, 3.82)	1.36 (0.89, 2.09)	1.18(0.74, 1.90)	0.45(0.27,0.73) *	1.08(0.89,1.98)	1.24(0.87,1.77)	2.06(1.45,2.93) *	0.59(0.41, 0.84) *	0.79(0.54,1.14)	0.44(0.31,0.63) *	0.54(0.38,0.79)*

PTE: Pulmonary thromboembolism; DM: diabetes mellitus; COPD: Chronic obstructive pulmonary disease; CHF: Congestive heart failure; AF: atrial fibrillation; HTN: Hypertension; IBD: Irritable bowel syndrome; BMI: Body mass index
*p-value < 0.05

4.2. Association between paraclinical indices and clinical features of Pulmonary thromboembolism

The results of the association between some laboratory parameters and clinical features of PTE are illustrated in Table 3. The amount of red blood cells (RBC) was positively correlated with the incidence of dyspnea and fever. Each one-unit increase in the RBC mass was associated with increased odds of dyspnea and syncope by 2.63 and 4.21 times, respectively. On the other hand, for a one-unit increase in RBC mass, the odds of developing hypoxemia decreased by 79%.

Albumin levels were associated with a reduced risk of tachycardia and fever and an increased incidence of cough, hemoptysis, and chest pain. Furthermore, with each one-unit increase in albumin level, the odds of cough and chest pain increased by 45% and 33%, respectively. In the same way, each one-unit increase in albumin increased the odds of presenting hemoptysis by 2.45 times. On the contrary, each one-unit increase in albumin level reduced the odds of presenting tachycardia and fever by 30% and 26%, respectively.

Logistic regression analyses revealed that each one-unit increase in the hemoglobin level decreased the odds of presenting dyspnea, fever, and hypoxemia by 12%, 11%, and 24%, respectively; moreover, the chance of developing hypoxemia decreased by 24%. Based on two-sample t-tests, the mean white blood cell (WBC) level was significantly higher in patients with dyspnea or fever than those without these symptoms. In addition, the mean WBC level in patients with syncope was significantly lower than those without these symptoms (p-value < 0.05).

Based on two-sample t-test results, the mean

platelet count was significantly lower in patients with tachycardia or hypoxemia than in patients without these signs (P< 0.05). The results of the association between some paraclinical parameters and clinical features of PTE are demonstrated in Table 4. The results of logistic regression analyses revealed that after adjusting for HCO3 and PCO2, each one-unit increase in the pH level increased the probability of developing chest pain and hypoxemia by 3.19 and 2.62 times, respectively.

Table 3. Association between some laboratory parameters and clinical features of pulmonary thromboembolism in study participants

	OR (95% CI)		
	RBC	**Hgb**	**Albumin**
Cough	0.74 (0.53,1.04)	0.95 (0.88,1.03)	1.45 (1.12,1.90)*
Dyspnea	2.63 (1.68, 4.100*	1.12(1.02,1.22)*	0.88 (0.61,1.25)
Tachycardia	0.96 (0.69,1.32)	0.99 (0.92,1.07)	0.70 (0.54,0.90)*
Hemoptysis	1.25 (0.75,2.06)	0.97 (0.87,1.08)	2.45 (1.58,3.81)*
Chest pain	1.13 (0.81,1.58)	0.96 (0.89,1.03)	1.33 (1.02,1.74)*
Syncope	4.21(2.13,8.28)*	1.16 (0.95,1.41)	0.86 (0.50,1.50)
Fever	0.74 (0.55,0.99)*	1.11 (1.01,1.22)*	1.38 (0.95,2.02)
Hypoxemia	1.24 (0.91,1.70)	0.76 (0.63,0.83)*	0.21 (0.13,0.33)*

*P< 0.05

Table 4. Association between some paraclinical parameters and clinical features of pulmonary thromboembolism in study participants

	OR (95% CI)								
	PH	**HCO3**	**PCO2**	**EF (normal)**	**RV dilation**	**S1Q3T3**	**T-invert**	**PAP**	
								PAP <30	PAP (30-60)
Cough	2.17(0.74,6.38)	0.98(0.92,1.04)	0.99(0.96,1.03)	1.26(0.93,1.70)	1.44(0.98,2.12)	0.84(0.61,1.16)	0.92(0.68,1.24)	2.30(0.98,5.43)	1.79(0.77,4.16)
Dyspnea	1.83(0.77,4.34)	1.02(0.96,1.09)	0.97(0.94,1.01)	0.63(0.42,0.95)*	0.51(0.27,0.95)*	1.10(0.71,1.68)	0.69(0.45,1.05)	0.20(0.02,1.58)	0.21(0.29,1.63)
Tachycardia	0.40(0.14,1.14)	0.97 (0.92,1.03)	0.98(0.95,1.01)	1.72(1.27,2.32)*	0.56(0.38,0.82)*	0.92(0.67,1.26)	1.07(0.80,1.45)	1.20(0.55,2.59)	0.88(0.41,1.87)
Hemoptysis	0.58(0.24,1.14)	1.03(0.95,1.11)	0.98(0.93,1.03)	0.93(0.58,1.47)	0.80(0.45,1.39)	0.13(0.68,1.88)	1.24(0.77,2.00)	2.63(0.59,11.61)	1.47(0.33,6.51)
Chest pain	3.19(1.06,9.56)*	0.89(0.84,0.95)*	1.05(1.01,1.08)*	0.56(0.38,0.82)*	1.07(0.79,1.45)	1.07(0.77,1.49)	1.54(1.13,2.11)*	0.64(0.29,1.42)	1.06(0.49,2.29)
Syncope	2.66(0.19,36.31)	1.17(0.99,1.39)	1.02(0.96,1.09)	1.30(0.63,1.25)	2.69(1.02,7.08)*	1.88(0.85,4.15)	0.51(0.27,0.98)*	2.06(0.26,16.34)	2.19(0.28,16.97)
Fever	0.74(0.34,1.60)	1.05(0.99,1.12)	0.98(0.94,1.01)	0.89(0.63,1.25)	0.89(0.59,1.34)	1.12(0.77,1.62)	0.93(0.66,1.31)	0.68(0.30,1.56)	0.73(0.32,1.63)
hypoxemia	2.62(1.06,6.72)*	0.98(0.93,1.03)	1.04(1.01,1.07)	1.51(1.07,2.13)*	0.37(0.22,0.62)*	2.02(1.34,3.04)*	1.23(0.87,1.75)	0.68(0.30,1.56)	0.59(0.26,1.33)

*P< 0.05
EF: Ejection fraction; RV: right ventricle; PAP: pulmonary artery pressure

After adjusting for pH and PCO2 variables, each one-unit increase in the HCO3 level reduced the probability of chest pain incidence by 20%. Moreover, after adjusting pH and HCO3 variables, each one-unit increase in the PCO2 level reduced the probability of chest pain incidence by 5%. Logistic regression analyses demonstrated no statistically significant association between pulmonary artery pressure (PAP) and PTE clinical features (P>0.05). The results of logistic regression analyses revealed that the incidence of dyspnea in patients with normal EF was lower than that in those with abnormal EF (OR = 0.63; P=0.032). In a similar vein, the odds of having tachycardia and hypoxemia in patients with normal EF were 1.72 and 1.51 times higher than in patients with abnormal EF, respectively.

Patients who did not have RV dilatation were less likely to have dyspnea, tachycardia, chest pain, and hypoxemia compared to those with RV dilatation. On the other hand, syncope incidence was 2.69 times higher in patients who had RV dilatation compared to those who did not have RV dilatation (P=0.045). Logistic regression analyses illustrated that patients who did not have S1Q3T3 on EKG were 2.02 times more likely to develop hypoxemia. Similarly, patients who did not have T-invert status in EKG had a 54.5 times higher probability of developing chest pain. Finally, the patients who did not have T-invert status on the EKG were less likely to develop syncope.

5. Discussion

The PTE, which is a common clinical disorder usually underdiagnosed, is associated with high mortality due to nonspecific clinical manifestations. In accordance with previous studies, in the present research, the most common presentations of PTE were dyspnea, tachycardia, and cough. In a study by Bajaj et al (12). in China, 334 patients with PTE with a mean age of 65.8 years were examined. The researchers found that dyspnea, chest pain, and cough accounted for 72%, 38%, and 19% of patients' complaints, respectively. Moreover, the most common clinical sign was tachypnea (39%).

Nonetheless, some studies have pointed out that pleural pain and hemoptysis are the most common clinical manifestations in PTE patients (1).

In the present study, hemoptysis (11.7%) and syncope (5.6%) were less common. These differences can be ascribed to various distributions of age, studied population, as well as anatomical variations and emboli sizes (12,13) In the present study, more than half of patients (51%) suffered from some form of malignancy. This finding may lead physicians to look for malignancies in these patients. This finding was observed in some previous studies (12).

Based on logistic regression analyses, there were significant associations between some clinical features of PTE and comorbidities. Patients with CVA or CHF had higher odds of having cough. On the contrary, COPD and diabetes were negatively associated with cough. In their study, Ji et al (14) found that patients with concomitant COPD had 11.5-fold increased odds of having a cough. Nevertheless, in line with our study, patients with diabetes were less likely to present cough. In our study, patients who did not have CVA, body mass index ≥ 30, or diabetes had a lower chance of developing tachycardia. Cardiac arrhythmias following CVA can be caused by neurologically mediated myocytolysis of the heart.

In agreement with our study findings, it has been reported that obesity has an association with an increased risk of cardiac arrhythmia and sudden cardiac death (15,16). Obesity causes several anatomical and functional changes in the myocardium that play a major role in arrhythmogenesis (17). Another finding of the present study was the association of some laboratory parameters with several clinical manifestations of PTE. The RBC mass was positively associated with the risk of developing dyspnea and syncope, while it reduced the odds of developing hypoxemia. This finding was not consistent with the study by Ji et al (14) where an increase of hypoxemia by 1.76 times was reported by increasing the RBC mass.

There was a significant association between albumin level and some PTE clinical features. Previous population-based studies have pointed out that low serum albumin concentration is related to long-term venous thromboembolism (VTE) risk (18,19). Therefore, albumin measurement is recommended in embolic risk assessment models in hospitalized patients. In the present study, blood gas indices were associated with some clinical features. Moreover, chest pain was positively correlated with PH and PCO2 levels. Nonetheless, in the study by Ji et al (14). pH exhibited a negative association with dyspnea and syncope.

In the present study, RV dilatation was observed in 23% of PTE patients. According to some previous literature, RV dysfunction is associated with an increased risk of mortality in PTE patients (20). Therefore, it is recommended that physicians consider RV dysfunction and other parameters to assess the severity of the disease when evaluating patients with acute PTE (21). We observed that patients with normal EF were less likely to present dyspnea but more likely to have tachycardia and hypoxemia than those with abnormal EF. Congestive heart failure (CHF) followed by abnormal EF is an independent and major risk factor for the development of venous thromboembolism among hospitalized patients (22).

In the present study, the frequency of EKG changes, including T-invert and S1Q3TQ pattern, were 42% and 31%, respectively. The odds of developing hypoxemia were also higher in PTE patients who did not have the S1Q3T3 pattern on EKG. On the other hand, patients who did not have a T-invert pattern were more likely to present chest pain and less likely to present syncope. In the study by Ngahane et al (23). The frequency rates of T-invert and S1Q3TQ among 103 patients with PTE were 26% and 38%, respectively. The pattern of T-invert and S1Q3TQ in PTE is associated with a higher frequency than coronary syndromes (24).

Among the notable limitations of this study, we can refer to its retrospective nature. Moreover, due to the use of data registered in hospital charts, the bias may have been based on the physician's clinical judgment. Therefore, more studies are needed to confirm the diagnostic value of clinical features.

6. Conclusion

Despite the highly nonspecific clinical manifestations, the diagnosis of PTE remains a challenging issue. The classic findings related to PTE, such as tachycardia, dyspnea, hypoxemia, chest pain, and hemoptysis, are associated with many other disorders, resulting in delayed diagnosis of PTE. This study presented related signs and symptoms, clinical risk factors, comorbidities, and PTE-related EKG findings, which could assist physicians in properly approaching this dangerous and potentially fatal disease by considering all aspects.

Acknowledgments

None.

Footnotes

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

Funding: This article has no financial source because it did not cost so much. Ethical Approval Code: IR.TUMS.MEDICINE.REC.1400.142

References

1. Stein PD, Beemath A, Matta F, Weg JG, Yusen RD, Hales CA, Hull

RD, et al. Clinical characteristics of patients with acute pulmonary embolism: data from PIOPED II. *Am J Med*. 2007;**120**(10):871-9. doi: 10.1016/j.amjmed.2007.03.024. [PubMed: 17904458].
2. Miao J, Naik G, Muddana S, Li X, Bhimasani S, Mitchell RA, Alaie D, Petrillo RL. An uncommon case of lower limb deep vein thrombosis with multiple etiological causes. *Am J Case Rep*. 2017;**18**: 313-16. doi: 10.12659/ajcr.902391. [PubMed: 28348360].
3. Pineda LA, Hathwar VS, Grant BJ. Clinical suspicion of fatal pulmonary embolism. *Chest*. 2001;**120**(3):791-5. doi: 10.1378/chest.120.3.791. [PubMed: 11555511].
4. Cha SI, Choi KJ, Shin KM, Lim JK, Yoo SS, Lee J, et al. Clinical characteristics of pulmonary embolism with concomitant pneumonia. *Blood Coagul Fibrinolysis*. 2016;**27**(3):281-6. doi: 10.1097/MBC.0000000000000411. [PubMed: 26366829].
5. Chou DW, Wu SL, Chung KM, Han SC. Septic pulmonary embolism caused by a Klebsiella pneumoniae liver abscess: clinical characteristics, imaging findings, and clinical courses. *Clinics (Sao Paulo)*. 2015;**70**(6):400-7. doi: 10.6061/clinics/2015(06)03. [PubMed: 26106957].
6. Bělohlávek J, Dytrych V, Linhart A. Pulmonary embolism, part I: Epidemiology, risk factors and risk stratification, pathophysiology, clinical presentation, diagnosis and nonthrombotic pulmonary embolism. *Exp Clin Cardiol*. 2013;**18**(2):129-38. [PubMed: 23940438].
7. Klok FA, Zondag W, van Kralingen KW, van Dijk AP, Tamsma JT, Heyning FH, et al. Patient outcomes after acute pulmonary embolism: a pooled survival analysis of different adverse events. *Am J Respir Crit Care Med*. 2010;**181**(5):501-6. doi: 10.1164/rccm.200907-1141OC. [PubMed: 19965808].
8. Horvei LD, Brækkan SK, Mathiesen EB, Njølstad I, Wilsgaard T, Hansen JB. Obesity measures and risk of venous thromboembolism and myocardial infarction. *Eur J Epidemiol*. 2014;**29**(11):821-30. doi: 10.1007/s10654-014-9950-z. [PubMed: 25213403].
9. Chaulagai B, Acharya D, Poudel S, Puri P. Simultaneous aortic dissection and pulmonary embolism: a therapeutic dilemma. *Cureus*. 2021;**13**(1):e12952. doi: 10.7759/cureus.12952. [PubMed: 33654624].
10. Moheimani F, Jackson DE. Venous thromboembolism: classification, risk factors, diagnosis, and management. *ISRN Hematol*. 2011;**2011**:124610. doi: 10.5402/2011/124610. [PubMed: 22084692].
11. Miniati M, Cenci C, Monti S, Poli D. Clinical presentation of acute pulmonary embolism: survey of 800 cases. *PLoS One*. 2012;**7**(2): e30891. doi: 10.1371/journal.pone.0030891. [PubMed: 22383978].
12. Bajaj N, Bozarth AL, Guillot J, KojoKittah J, Appalaneni SR, Cestero C, Amankona RK, Pippim JA. Clinical features in patients with pulmonary embolism at a community hospital: analysis of 4 years of data. *J Thromb Thrombolysis*. 2014;**37**(3):287-92. doi: 10.1007/s11239-013-0942-8. [PubMed: 23681675].
13. Stein PD, Gottschalk A, Saltzman HA, Terrin ML. Diagnosis of acute pulmonary embolism in the elderly. *J Am Coll Cardiol*. 1991;**18**(6):1452-7. doi: 10.1016/0735-1097(91)90674-x. [PubMed: 1939945].
14. Ji QY, Wang MF, Su CM, Yang QF, Feng LF, Zhao LY, et al. Clinical symptoms and related risk factors in pulmonary embolism patients and cluster analysis based on these symptoms. *Sci Rep*. 2017;**7**(1):14887. doi: 10.1038/s41598-017-14888-7. [PubMed: 29097743].
15. Hubert HB, Feinleib M, McNamara PM, Castelli WP. Obesity as an independent risk factor for cardiovascular disease: a 26-year follow-up of participants in the Framingham Heart Study. *Circulation*. 1983;**67**(5):968-77. doi: 10.1161/01.cir.67.5.968. [PubMed: 6219830].
16. Wang TJ, Parise H, Levy D, D'Agostino RB, Wolf PA, Vasan RS, et al. Obesity and the risk of new-onset atrial fibrillation. *JAMA*. 2004;**292**(20):2471-7. doi: 10.1001/jama.292.20.2471. [PubMed: 15562125].
17. Csige I, Ujvárosy D, Szabó Z, Lőrincz I, Paragh G, Harangi M, Somodi S. The impact of obesity on the cardiovascular system. *J Diabetes Res*. 2018;**2018**:3407306. doi: 10.1155/2018/3407306. [PubMed: 30525052].
18. Chi G, Gibson CM, Liu Y, Hernandez AF, Hull RD, Cohen AT, et al. Inverse relationship of serum albumin to the risk of venous thromboembolism among acutely ill hospitalized patients: analysis from the APEX trial. *Am J Hematol*. 2019;**94**(1):21-28. doi: 10.1002/ajh.25296. [PubMed: 30252149].
19. Königsbrügge O, Posch F, Riedl J, Reitter EM, Zielinski C, Pabinger I, Ay C. Association between decreased serum albumin with risk of venous thromboembolism and mortality in cancer patients. *Oncologist*. 2016;**21**(2):252-7. doi: 10.1634/theoncologist.2015-0284. [PubMed: 26764252].
20. Vieillard-Baron A, Page B, Augarde R, Prin S, Qanadli S, Beauchet A, Dubourg O, Jardin F. Acute cor pulmonale in massive pulmonary embolism: incidence, echocardiographic pattern, clinical implications and recovery rate. *Intensive Care Med*. 2001;**27**(9):1481-6. doi: 10.1007/s001340101032. [PubMed: 11685341].
21. Singanayagam A, Chalmers JD, Scally C, Akram AR, Al-Khairalla MZ, Leitch L, Hill LE, Hill AT. Right ventricular dilation on CT pulmonary angiogram independently predicts mortality in pulmonary embolism. *Respir Med*. 2010;**104**(7):1057-62. doi: 10.1016/j.rmed.2010.02.004. [PubMed: 20202807].
22. Hsiao SH, Lee CY, Chang SM, Yang SH, Lin SK, Huang WC. Pulmonary embolism and right heart function: insights from myocardial Doppler tissue imaging. *J Am Soc Echocardiogr*. 2006;**19**(6):822-8. doi: 10.1016/j.echo.2006.01.011. [PubMed: 16762763].
23. Ngahane BH, Kamdem F, Njonnou SR, Chebou N, Dzudie A, Ebongue SA, et al. Epidemiology, clinical and paraclinical presentations of pulmonary embolism: A cross-sectional study in a sub-Saharan Africa setting. *Open Journal of Respiratory Diseases*. 2019;**9**(3):89. doi: 10.4236/ojrd.2019.93008.
24. Kosuge M, Kimura K, Ishikawa T, Ebina T, Hibi K, Kusama I, et al. Electrocardiographic differentiation between acute pulmonary embolism and acute coronary syndromes on the basis of negative T waves. *Am J Cardiol*. 2007;**99**(6):817-21. doi: 10.1016/j.amjcard.2006.10.043. [PubMed: 17350373].