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Original Research Article

ETIOLOGICAL AND CLINICAL PROFILE OF PULMONARY THROMBOEMBOLISM IN KASHMIRI POPULATION

Article History:

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Abstract: ***Background***: Pulmonary thromboembolism (PTE) is a potentially life-threatening condition resulting from embolization of thrombi, most commonly originating from deep venous thrombosis (DVT). Early recognition and management are essential to reduce mortality. ***Objective***: To study the etiological and clinical profile of pulmonary thromboembolism in the Kashmiri population. ***Methods:*** This prospective observational study was conducted over a period of two years (December 2019–December 2021) in the Postgraduate Department of Medicine, Government Medical College, Srinagar. Fifty patients aged >18 years with CT pulmonary angiography (CTPA) confirmed PTE were included. Detailed clinical history, examination, laboratory investigations including D-dimer, ECG, 2D echocardiography, and CTPA findings were analyzed. ***Results***: Mean age was 52.7 ± 16.74 years, with 60% females. Major risk factors included reduced mobility (56%), DVT (52%), smoking (42%), prior surgery (30%), fracture (26%), malignancy (10%), COVID-19 (14%), and APLA positivity (6%). Hypertension (60%) and diabetes mellitus (30%) were common comorbidities. Cough (74%) and breathlessness (70%) were predominant symptoms. Tachypnea (94%) and tachycardia (90%) were the most common signs. D-dimer was elevated (>500) in 92%. Sinus tachycardia (72%) was the most common ECG finding. Echocardiography showed RA/RV dilation (78%) and RV hypokinesia (82%). CTPA revealed subsegmental PTE in 36%, small subsegmental PTE in 22%, and massive PTE in 8%. ***Conclusion:*** Pulmonary thromboembolism in the Kashmiri population predominantly affects individuals above 50 years, with significant association with immobility and DVT. Clinical suspicion combined with D-dimer assessment and imaging remains crucial for early diagnosis and improved outcomes.

Keywords: Digital CBT, adolescent depression, randomized controlled trial, PHQ-9, mental health intervention, mobile therapy, school counselling.

INTRODUCTION

Pulmonary embolism (PE) is a life-threatening condition resulting from dislodged thrombi occluding the pulmonary vasculature; right heart failure and cardiac arrest may ensue if not aggressively treated. Pulmonary embolism (PE) is a consequence of thrombus formation within a deep vein of the body, most frequently in the lower extremities. Thrombus formation in the venous system occurs as a result of venous stasis, trauma, and hypercoagulability. These factors are collectively known as Virchow's triad.[1] Approximately 51% of deep venous thrombi will embolise to the pulmonary vasculature, resulting in a PE.[2]The annual incidence of pulmonary embolism in the population is 1 per 1000 people, but this increases sharply with age, from 1.4 per 1000 people aged 40–49 to 11.3 per 1000 aged 80 years or over.[3-5] Recurrent venous thromboembolism occurs in 30% of people, making the attack rate (including incident and recurrent venous thromboembolism) higher, estimated as up to 30 per 1000 person years.[5] The influence of race on venous incidence of thromboembolism is uncertain, but incidence may be higher in white and African-American populations and lower in Asians and Native Americans.[5]

Among women under 45 years or over 80 years, the incidence of venous thromboembolism is higher than in men. This interaction with age and sex is likely

related to estrogen and pregnancy related risk factors at a young age and longer life expectancy of women at advanced ages. Vital registration data indicate that women aged 15–55 and over 80 years have an excess pulmonary embolism related mortality compared with men.[6] Most clinically significant PEs originate as VTEs in the lower extremities or pelvic veins. Less frequently, upper extremity thromboembolic events lead to PE. Various conditions lead to the generation of VTE. Virchow's triad of hypercoagulability, venous stasis, and vessel wall injury provides a model for understanding many of the risk factors.[7-9] Overall, major risk factors for thromboembolic events include recent immobilization, myocardial infarction, cerebrovascular accident, surgery, and recent trauma. Additional major risk factors include prior VTE, advanced age, malignancy, known thrombophilia, and indwelling venous catheter. [7-9]

Prompt recognition of a constellation of nonspecific signs and symptoms is needed for diagnosis of pulmonary embolism. Prompt initiation of anticoagulation while awaiting investigations is prudent because of the high risk of early mortality with untreated pulmonary embolism.[10–12] Clinical probability scores can be used to assign a pre-test probability for pulmonary embolism. The Geneva and Wells rules are among the most commonly cited clinical probability scores.[13-16] The D-dimer is a degradation product of fibrinolysis and is increased in patients with acute venous thromboembolism as well as other non-thrombotic disorders.[17] A negative D-dimer value in combination with a low clinical probability score is useful for excluding a diagnosis of venous thromboembolism.[13,17]

The gold standard diagnostic test for pulmonary embolism has historically been interventional pulmonary angiography. Planar ventilation-perfusion lung scans and computed tomography pulmonary angiography (CTPA) are validated imaging tests.[18-20] A ventilation-perfusion lung scan in a validated diagnostic algorithm performs equally well as CTPA in the diagnosis of pulmonary embolism.[18-20] In hemodynamically stable patients without contraindications, parenteral anticoagulation with subsequent conversion to vitamin K antagonists is the mainstay of therapy. Early initiation is paramount as patients may quickly decompensate.[21] Low molecular weight heparin is advantageous in ease of administration and lower potential for heparin-induced thrombocytopenia.[22] Oral rivaroxaban has been shown to be non-inferior to standard therapy for the treatment of symptomatic pulmonary embolism.[23,24]

Despite advances in diagnostic algorithms and management strategies, pulmonary thromboembolism remains a significant cause of morbidity and mortality worldwide.[3,5] Regional epidemiological data, particularly from the Kashmiri population, remain limited. Therefore, the present study was undertaken to evaluate the etiological and clinical profile of pulmonary thromboembolism in the Kashmiri population in a tertiary care setting.

AIMS AND OBJECTIVES

Aim

To study the etiological and clinical profile of pulmonary thromboembolism in the Kashmiri population.

Objectives

1. To study the etiological profile of pulmonary thromboembolism in the Kashmiri population.
2. To study the clinical profile of pulmonary thromboembolism in the Kashmiri population.

MATERIALS AND METHODS

This prospective, observational study was carried out over a period of two years in the Postgraduate Department of Medicine, Government Medical College, Srinagar, from December 2019 to December 2021, after approval from the Institutional Ethical Committee. It was conducted according to the principles expressed in the Declaration of Helsinki. All subjects were provided written informed consent in the local commonly used language. The study included participants who were older than 18 years, of either gender, belonging to Kashmiri ethnicity, and had CT pulmonary angiography documented pulmonary thromboembolism (PTE).

Participants younger than 18 years, those on immunosuppressant or anticoagulant therapy, and those not belonging to the Kashmiri population were excluded from the study. All admitted patients were subjected to detailed clinical history with emphasis on risk factors of PTE and thorough clinical examination. All enrolled patients were subjected to laboratory and radiological investigations as part of the diagnostic work-up. Baseline hematological parameters were assessed by performing a complete blood count (CBC). Renal function was evaluated using kidney function tests (KFT), and liver function tests (LFT) were carried out to assess hepatic status. Serum D-dimer levels were measured in all patients as part of the evaluation for suspected pulmonary thromboembolism. Electrocardiography (ECG) was performed to detect rhythm abnormalities, sinus tachycardia, right bundle branch block, or other changes suggestive of right heart strain. Two-dimensional echocardiography (2D ECHO) was conducted in all patients to evaluate right atrial (RA) and right ventricular (RV) size, right ventricular free wall motion, and evidence of pulmonary arterial hypertension or right ventricular dysfunction. Computed tomography pulmonary angiography (CTPA) was performed in all cases and served as the

definitive diagnostic modality to confirm pulmonary thromboembolism and determine the anatomical location and extent of thrombus.

The diagnosis of pulmonary thromboembolism in the present study was based on clinical suspicion supported by positive imaging findings on CT pulmonary angiography. Patients presenting with symptoms such as cough, breathlessness, chest pain, hemoptysis, or syncope, along with clinical signs including tachypnea, tachycardia, hypoxia, or hypotension, were evaluated for suspected PTE. Upon clinical suspicion, initial laboratory investigations including CBC, KFT, LFT, and D-dimer were performed. Electrocardiography was used to assess cardiac rhythm and signs of right heart strain. Two-dimensional echocardiography was undertaken to evaluate right heart morphology and function, particularly right ventricular dilation and hypokinesia.

All patients underwent CT pulmonary angiography, which confirmed the diagnosis of pulmonary thromboembolism and helped categorize the embolism according to location and severity. Only those patients with imaging-confirmed PTE were included in the final analysis. Data regarding demographic characteristics, risk factors, comorbidities, clinical presentation, laboratory findings, ECG changes, echocardiographic parameters, and CTPA findings were systematically recorded using a structured proforma. The collected data were compiled and entered into Microsoft Excel and subsequently exported to SPSS Version 20.0 (SPSS Inc., Chicago, Illinois, USA) for statistical analysis. Continuous variables were expressed as Mean±Standard Deviation (SD), while categorical variables were presented as frequencies and percentages. Graphical representation of data was performed using bar and pie diagrams.

RESULTS

A total of 50 patients with CT pulmonary angiography confirmed pulmonary thromboembolism (PTE) were included in the study conducted at Government Medical College, Srinagar (COMPLETE THESIS).

Table 1: Age and Gender Distribution

Variables	Categories	No. of Patients	Percentage
Age in Years	25–39	14	28
	40–54	8	16
	55–69	19	38
	≥70	9	18
	Mean ± SD (Range): 52.7 ± 16.74 years (25–80 years)		
Gender	Male	20	40
	Female	30	60

The mean age of the study population was 52.7 ± 16.74 years. The majority of patients (38%) were in the age group of 55–69 years, indicating that pulmonary thromboembolism predominantly affected individuals above 50 years of age. Females constituted 60% of the study population, whereas males accounted for 40%, demonstrating a female predominance among patients diagnosed with pulmonary thromboembolism.

Table 2: Risk factors, comorbidities and symptoms at presentation

Variables	Categories	No. of Patients	Percentage
Risk Factors	Reduced mobility	28	56
	Deep vein thrombosis (DVT)	26	52
	Smoking	21	42
	Prior surgery	15	30
	Fracture	13	26
	Malignancy	5	10
	COVID-19	7	14
	APLA positivity	3	6
	Varicose veins	1	2
Comorbidities	Hypertension	30	60
	Diabetes Mellitus	15	30
Symptoms	Cough	37	74
	Breathlessness	35	70
	Hemoptysis	21	42

Reduced mobility (56%) and deep vein thrombosis (52%) were the most common etiological factors associated with pulmonary thromboembolism in this cohort. Smoking was observed in 42% of patients. A significant proportion had prior surgical history (30%) and fracture (26%), highlighting immobilization-related risk. Malignancy (10%), COVID-19 infection (14%), and APLA positivity (6%) were also notable contributors. Hypertension was the most prevalent comorbidity (60%), followed by diabetes mellitus (30%). This indicates a high

burden of cardiometabolic risk factors among patients with pulmonary thromboembolism. Cough (74%) and breathlessness (70%) were the predominant presenting symptoms, followed by hemoptysis (42%). These findings emphasize the nonspecific respiratory presentation of pulmonary thromboembolism.

Table 3: Distribution as per clinical signs, D-Dimer and ECG findings

Variables	Categories	No. of Patients	Percentage
Clinical signs	Tachypnea	47	94
	Tachycardia	45	90
	Hypoxia	42	84
	Hypotension	21	42
D-Dimer	<500	4	8
	≥500	46	92
ECG findings	Sinus tachycardia	36	72
	Right bundle branch block	5	10
	Normal ECG	9	18

Tachypnea (94%) was the most common clinical sign, followed by tachycardia (90%) and hypoxia (84%). Hypotension was observed in 42% of patients, suggesting a substantial proportion had hemodynamic compromise at presentation. In the present study, serum D-dimer levels were elevated (≥500 ng/mL) in 46 out of 50 patients (92%), while only 4 patients (8%) had D-dimer levels below 500 ng/Ml. Electrocardiographic evaluation revealed sinus tachycardia in 36 patients (72%), right bundle branch block (RBBB) in 5 patients (10%), while 9 patients (18%) had a normal ECG. Sinus tachycardia emerged as the most common ECG abnormality in this study. This finding reflects the physiological response to hypoxemia and increased sympathetic drive secondary to acute pulmonary vascular obstruction. The high frequency of tachycardia (72%) correlates with the clinical observation of tachycardia in 90% of patients at presentation, emphasizing the hemodynamic stress associated with pulmonary embolism. Right bundle branch block, observed in 10% of cases, suggests acute right ventricular strain caused by sudden elevation in pulmonary arterial pressure. Although less common than sinus tachycardia, the presence of RBBB may indicate more significant right heart involvement. Importantly, 18% of patients demonstrated a normal ECG despite confirmed pulmonary thromboembolism. This finding highlights the limited sensitivity and specificity of ECG in diagnosing pulmonary embolism. While ECG abnormalities may support clinical suspicion and help in risk stratification, a normal ECG does not exclude pulmonary embolism.

Table 4: Distribution as per echocardiographic and CTPA Findings

Variables	Categories	No. of Patients	Percentage
Echocardiographic findings	RA/RV dilation	39	78
	RV free wall hypokinesia	41	82
CT Pulmonary Angiography (CTPA) Findings	Subsegmental PTE	18	36
	Small subsegmental PTE	11	22
	Massive PTE (including saddle thrombus cases)	4	8
	MPA/RPA thrombus	5	10
	RPA & LPA thrombosis	3	6
	Others (isolated thrombi)	9	18

Two-dimensional echocardiography demonstrated right atrial (RA) and right ventricular (RV) dilation in 39 patients (78%), while right ventricular free wall hypokinesia was present in 41 patients (82%). The observation that 82% of patients had RV free wall hypokinesia suggests that right ventricular dysfunction was a prominent feature in this cohort. Similarly, RA/RV dilation in 78% of cases indicates significant hemodynamic impact of embolic obstruction. The high prevalence of right ventricular dysfunction in this study aligns with the presence of hypoxia (84%) and hypotension (42%) observed clinically, indicating that many patients presented with moderate to severe physiological compromise.

CT pulmonary angiography (CTPA), which served as the definitive diagnostic modality in this study, revealed a spectrum of thrombus locations and severities. Subsegmental pulmonary thromboembolism was the most common finding, observed in 18 patients (36%). Small subsegmental PTE was noted in 11 patients (22%). Together, peripheral emboli accounted for more than half of the cases, suggesting that distal pulmonary arterial branches were frequently involved in this population. Central thrombi were also observed. Thrombus involving the main pulmonary artery (MPA) and right pulmonary artery (RPA) was seen in 5 patients (10%). RPA and LPA thrombosis and saddle thrombus were each observed in 3 patients (6%). Massive PTE with saddle thrombus was present in 2 patients (4%), and saddle thrombus extending into RPA and LPA was also noted in 2 patients (4%). Isolated thrombi involving MPA, LPA, or RPA were identified in a smaller proportion (2% each).

DISCUSSION

Pulmonary thromboembolism (PTE) remains a significant cause of morbidity and mortality worldwide, with increasing incidence in advancing age.3,4,5 The present study was conducted to evaluate the etiological and clinical profile of pulmonary thromboembolism in the Kashmiri population, and the findings are discussed in light of available literature.

Age Distribution

In our study, the mean age of patients was 52.7 ± 16.74 years, and the majority (38%) belonged to the 55–69 years age group. These findings are consistent with the epidemiological trend described by Stein PD et al., who reported that venous thromboembolism increases significantly with advancing age.3,4 Similarly, Heit JA reported that the incidence of venous thromboembolism rises sharply in older populations.5 The predominance of cases above 50 years in our study aligns with these established observations.

Gender Distribution

In the present study, females constituted 60% of cases (COMPLETE THESIS). Barco S et al. reported that women aged 15–55 and over 80 years have excess pulmonary embolism–related mortality compared with men.6 Furthermore, the interaction between age and sex in venous thromboembolism has been linked to estrogen exposure and pregnancy-related risk factors in younger women.5 The female predominance observed in our study may be explained by similar hormonal and demographic influences.

Etiological Profile

Reduced mobility (56%) and deep vein thrombosis (52%) were the most common risk factors identified in our study. Virchow's triad venous stasis, endothelial injury, and hypercoagulability remains central to the pathogenesis of thrombus formation.1 Most clinically significant PEs originate from deep veins of the lower extremities.7-9

The high prevalence of DVT in our cohort supports findings by Stein PD et al., who demonstrated that a substantial proportion of patients with deep venous thrombosis have silent pulmonary embolism.25

Smoking was observed in 42% of patients in our study. Smoking has been associated with an increased risk of venous thromboembolism, likely through endothelial dysfunction and prothrombotic effects.3

A history of surgery (30%) and fracture (26%) was common in our patients. Surgical procedures and trauma are well-established strong risk factors for venous thromboembolism.8,9 Immobilization following these conditions contributes significantly to venous stasis.

Malignancy was present in 10% of patients. Malignancy is a recognized major risk factor for thromboembolic events due to tumor-associated procoagulant activity.7,8

COVID-19 infection was documented in 14% of patients. Trimaille A et al. reported that pulmonary embolism in COVID-19 patients is associated with a marked inflammatory and prothrombotic state and poorer outcomes.26 The prothrombotic milieu observed in COVID-19 likely contributed to thrombus formation in our patients.

Clinical Presentation

In our study, cough (74%) and breathlessness (70%) were the predominant symptoms, while hemoptysis was present in 42% of cases. Pulmonary embolism often presents with nonspecific respiratory symptoms, making clinical diagnosis challenging.10 Tachypnea (94%) and tachycardia (90%) were the most frequent clinical signs in our cohort. The Prospective Investigation of Pulmonary Embolism Diagnosis II (PIOPED II) trial demonstrated that tachypnea and tachycardia are among the most common clinical findings in PE patients.20 The high frequency of these signs in our study correlates with established literature.

Hypotension was observed in 42% of patients, indicating that a considerable proportion presented with hemodynamic compromise. Massive pulmonary embolism can lead to acute right ventricular failure and cardiogenic shock.10

D-Dimer Levels

D-dimer levels ≥500 were observed in 92% of patients in our study. D-dimer is a fibrin degradation product and is typically elevated in acute venous thromboembolism.17 Although highly sensitive, D-dimer lacks specificity and may be elevated in other inflammatory and prothrombotic conditions.17 The high prevalence of elevated D-dimer in our cohort reinforces its value as an important screening tool in suspected PTE, particularly when combined with clinical probability assessment as recommended by Wells et al.27 and Lucassen W et al.16.

Electrocardiographic Findings

Sinus tachycardia was the most common ECG abnormality (72%) in our study. ECG changes in pulmonary embolism are often nonspecific and reflect right ventricular strain.28
Right bundle branch block was observed in 10% of patients, suggesting acute right ventricular pressure

overload. However, 18% of patients had a normal ECG, highlighting the limited diagnostic sensitivity of ECG in pulmonary embolism.28

Echocardiographic Findings

Echocardiography demonstrated RA/RV dilation in 78% and RV free wall hypokinesia in 82% of patients. Acute pulmonary embolism results in increased pulmonary vascular resistance, leading to right ventricular pressure overload and dysfunction.10

Right ventricular enlargement and hypokinesia are established indicators of right heart strain in acute PE.8 The high proportion of RV dysfunction in our study corresponds with the significant number of patients presenting with hypoxia and hypotension.

CT Pulmonary Angiography Findings

CT pulmonary angiography (CTPA) confirmed pulmonary thromboembolism in all cases. Subsegmental PTE was the most common finding (36%), followed by small subsegmental PTE (22%). Massive PTE accounted for 8% of cases.

CTPA is a validated imaging modality for diagnosing pulmonary embolism and has largely replaced conventional pulmonary angiography.18 Ventilation-perfusion scanning and CTPA perform equally well when integrated into validated diagnostic algorithms.18-20

The presence of central thrombi, including saddle thrombus, in a subset of patients indicates severe disease and correlates with hemodynamic instability observed clinically.

The findings of the present study demonstrate that pulmonary thromboembolism in the Kashmiri population predominantly affects individuals above 50 years of age, with female predominance. Reduced mobility and deep vein thrombosis were the most important etiological factors. Clinically, cough and breathlessness were common presenting symptoms, while tachypnea and tachycardia were the most frequent signs. Elevated D-dimer levels and right ventricular dysfunction were observed in the majority of patients, and CTPA confirmed a wide spectrum of embolic involvement.

CONCLUSION

The present study demonstrates that pulmonary thromboembolism in the Kashmiri population predominantly affects individuals above 50 years of age, with a female predominance. Reduced mobility and deep vein thrombosis were the most important etiological factors identified. Cough and breathlessness were the most common presenting symptoms, while tachypnea and tachycardia were the predominant clinical signs. Elevated D-dimer levels were observed in the majority of patients, and echocardiographic findings frequently revealed right ventricular dysfunction. CT pulmonary angiography confirmed a broad spectrum of embolic involvement, ranging from subsegmental to massive pulmonary embolism. Early clinical suspicion, timely diagnostic evaluation, and prompt management are essential to improve outcomes in patients with pulmonary thromboembolism.

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