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THROMBOEMBOLISM AS A CAUSE OF SUDDEN DEATH IN IMMOBILIZED MEDICAL PATIENTS

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ABSTRACT

Background: Pulmonary embolism is a major cause of sudden clinical deterioration in immobilized medical patients and frequently remains unrecognized due to nonspecific presentation. Understanding the clinical predictors and anatomical pathway of thromboembolism is essential for early diagnosis and prevention.

Objective: To evaluate the clinical risk factors, warning signs, and anatomical distribution of pulmonary embolism in immobilized patients using clinicopathological correlation.

Methodology: This retrospective clinicopathological study was conducted at Sir Syed College of Medical Sciences (for Girls) & Khairpur Medical College, Khairpur Mir's from March 2024 to April 2025 and included 72 immobilized medical patients. Clinical records were reviewed for demographic characteristics, duration of immobilization, comorbidities, thromboprophylaxis, clinical presentation, and laboratory parameters. Postmortem examination was used for anatomical confirmation of thrombus origin and pulmonary arterial involvement. Statistical analysis was performed using chi-square and independent t-tests with $p \leq 0.05$ considered significant.

Results: Pulmonary embolism was confirmed in 48 patients (66.7%). Prolonged immobilization (>5 days), heart failure, previous deep vein thrombosis, and absence of thromboprophylaxis were significantly associated with embolism ($p<0.05$). Dyspnea, hypoxia, tachycardia, syncope, and elevated D-dimer were important clinical predictors. The femoral and popliteal veins were the most frequent thrombus sources, while the pulmonary arterial bifurcation was the most common embolic location, often accompanied by acute right ventricular dilatation.

Conclusion: Pulmonary embolism in immobilized patients follows a recognizable clinical and anatomical pattern. Early risk assessment and routine thromboprophylaxis can substantially reduce preventable deterioration and mortality.

Keywords: Pulmonary embolism, immobilization, deep vein thrombosis, clinicopathological correlation, venous anatomy

INTRODUCTION

Pulmonary embolism is a potentially fatal but frequently underdiagnosed complication in hospitalized and debilitated patients. It represents the most severe manifestation of venous thromboembolism, a condition responsible for significant morbidity and mortality worldwide [1]. Despite advances in diagnostic imaging and prophylactic strategies, pulmonary embolism continues to occur unexpectedly, particularly in patients with prolonged immobilization where symptoms may be subtle or masked by underlying disease [2-4].

Immobilization is a major contributor to thrombus formation through the mechanisms described in Virchow's triad: venous stasis, endothelial injury, and hypercoagulability. Reduced skeletal muscle contraction during bed rest diminishes venous return from the lower limbs, promoting clot formation in the deep venous system. Medical conditions such as heart failure, stroke, severe infection, and critical illness further enhance prothrombotic states. Once formed, thrombi may propagate through the iliac veins and inferior vena cava to the pulmonary arteries, producing acute cardiopulmonary compromise [5-7].

Clinical recognition of pulmonary embolism remains challenging because presenting features dyspnea, tachycardia, syncope, or unexplained hypoxia are nonspecific and often attributed to primary illness [8]. Consequently, deterioration may occur before diagnosis is suspected. Correlating premortem clinical findings with anatomical distribution of emboli provides valuable insight into disease progression and helps clinicians recognize early warning patterns in high-risk patients [9-11].

Understanding both the clinical predictors and anatomical pathway of thromboembolism is essential for prevention and timely management. However, regional clinical data integrating patient presentation with anatomical confirmation remain limited. Therefore, this study was conducted to evaluate the clinical risk factors, warning signs, and anatomical characteristics of pulmonary embolism in immobilized medical patients using a clinicopathological correlation approach.

METHODOLOGY

This retrospective clinicopathological correlation study was conducted at Sir Syed College of Medical Sciences (For Girls) & Khairpur Medical College, Khairpur Mir's over a period of one year from March 2024 to April 2025. The study included immobilized medical patients who experienced sudden unexpected clinical deterioration during hospitalization or home medical care and subsequently underwent postmortem examination for diagnostic confirmation. The purpose of including postmortem findings was to verify the anatomical source and distribution of thromboembolism and correlate them with premortem clinical features.

A total of 72 patients were selected through consecutive sampling. Immobilization was defined as restricted mobility for more than 72 hours due to acute medical illness, neurological impairment, sedation, mechanical ventilation, fracture, or severe systemic disease. Only patients with complete clinical documentation and confirmed postmortem examination were included. Traumatic deaths, poisoning, advanced decomposition, burns, and cases with incomplete clinical records were excluded.

Clinical data were retrieved from hospital medical records, emergency notes, nursing charts, and referral documentation. Demographic variables included age and gender. Clinical variables included duration and cause of immobilization, comorbid conditions (heart failure, malignancy, sepsis, dehydration, prior deep vein thrombosis), and use of thromboprophylaxis. Premortem warning signs such as dyspnea, chest pain, syncope, tachycardia, and hypoxia were recorded. Laboratory parameters included D-dimer, arterial blood gases, cardiac troponin, platelet count, prothrombin time, and activated partial thromboplastin time. When available, premortem imaging findings such as Doppler ultrasonography and CT pulmonary angiography were also documented.

Postmortem examination was performed to anatomically confirm thromboembolism and identify the venous source and pulmonary arterial distribution of emboli. The deep venous system of the lower limbs (popliteal, femoral, iliac, and pelvic veins) and inferior vena cava were examined for thrombus formation. Pulmonary arteries were assessed for location of embolus (saddle, main

branch, or segmental). Associated cardiopulmonary changes such as right ventricular dilatation and pulmonary infarction were also recorded. These findings were used for anatomical confirmation rather than legal determination.

Preventability assessment was performed by evaluating whether appropriate thromboprophylaxis (pharmacological anticoagulation or mechanical compression devices) had been administered according to standard medical guidelines.

Data were analyzed using SPSS version 26. Continuous variables were expressed as mean ± standard deviation, while categorical variables were presented as frequency and percentage. Chi-square test was used for comparison of categorical variables and independent sample t-test for quantitative variables. A p-value ≤ 0.05 was considered statistically significant.

RESULTS

Among the 72 immobilized medical patients included in the study, pulmonary embolism (PE) was confirmed in 48 patients (66.7%), while 24 patients (33.3%) had alternative causes of clinical deterioration. Patients with PE were significantly older and had a longer duration of immobilization compared to non-PE patients, highlighting the role of venous stasis in thrombus formation.

Prolonged immobilization (>5 days) showed a strong association with PE occurrence (p=0.001). Age was also significantly higher in the PE group (p=0.028), whereas gender distribution and mechanism of immobilization were not statistically different between groups.

Table 1: Demographics and Immobilization Characteristics

Variable	PE (n=48)	Non-PE (n=24)	p-value
Mean age (years)	64.8 ± 11.2	58.3 ± 12.6	**0.028**
Male	28 (58.3%)	13 (54.2%)	0.742
Female	20 (41.7%)	11 (45.8%)	—
Immobilization >5 days	37 (77.1%)	9 (37.5%)	**0.001**
Stroke with paralysis	18 (37.5%)	5 (20.8%)	0.143
ICU mechanical ventilation	16 (33.3%)	6 (25.0%)	0.461
Post-fracture immobilization	9 (18.8%)	2 (8.3%)	0.233

Heart failure, previous DVT history, and absence of thromboprophylaxis were significantly associated with PE. Other hypercoagulable or inflammatory conditions showed higher frequency but did not reach statistical significance.

Table 2: Clinical Risk Factors

Risk Factor	PE (n=48)	Non-PE (n=24)	p-value
Malignancy	14 (29.2%)	3 (12.5%)	0.118
Sepsis	19 (39.6%)	5 (20.8%)	0.104
Dehydration	22 (45.8%)	6 (25.0%)	0.082
Central venous catheter	15 (31.3%)	4 (16.7%)	0.179
Heart failure	21 (43.8%)	5 (20.8%)	**0.047**
Previous DVT history	11 (22.9%)	1 (4.2%)	**0.038**
No thromboprophylaxis given	34 (70.8%)	8 (33.3%)	**0.002**

PE patients demonstrated significantly higher frequency of respiratory compromise and circulatory instability. Elevated D-dimer, hypoxia, tachycardia, and syncope were strong clinical predictors of thromboembolism.

Table 3: Clinical and Laboratory Findings

Finding	PE (n=48)	Non-PE (n=24)	p-value
Sudden dyspnea	29 (60.4%)	6 (25.0%)	**0.004**

Chest pain	18 (37.5%)	4 (16.7%)	0.071
Syncope/collapse	23 (47.9%)	5 (20.8%)	**0.023**
Tachycardia (>110/min)	31 (64.6%)	7 (29.2%)	**0.006**
Hypoxia (SpO_2 <90%)	33 (68.8%)	8 (33.3%)	**0.005**
Elevated D-dimer	35 (72.9%)	6 (25.0%)	**<0.001**
Raised troponin	14 (29.2%)	4 (16.7%)	0.241

The lower limb deep venous system represented the principal origin of emboli, with femoral and popliteal veins accounting for the majority of thrombus formation. The most frequent embolic location was the pulmonary arterial bifurcation (saddle embolus), frequently associated with acute right ventricular dilatation.

Table 4: Anatomical Correlation of Thromboembolism (PE cases only, n=48)

Anatomical Finding	**n**	**%**
Source of thrombus		
Popliteal vein	11	22.9%
Femoral vein	17	35.4%
Iliac vein	9	18.8%
Inferior vena cava	5	10.4%
Pelvic veins	6	12.5%
Pulmonary embolus location		
Saddle embolus (bifurcation)	19	39.6%
Right pulmonary artery	11	22.9%
Left pulmonary artery	8	16.7%
Segmental branches	10	20.8%
Cardiac effects		
Acute right ventricular dilatation	32	66.7%
Pulmonary infarction	14	29.2%

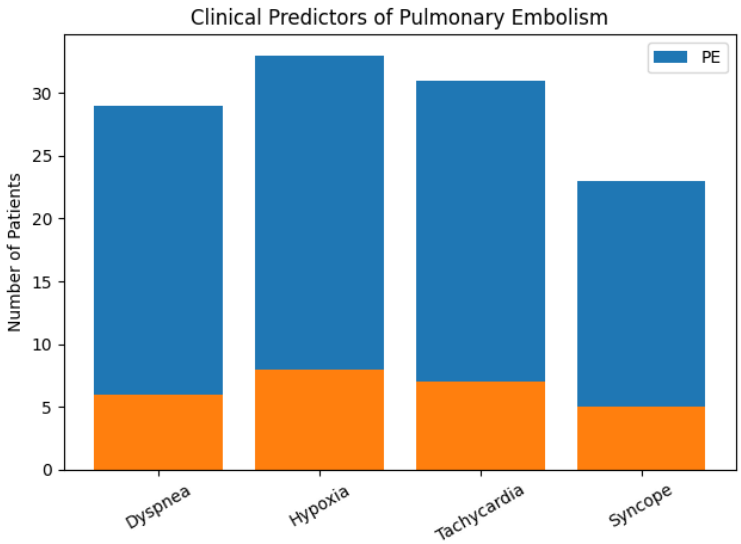

Figure 1: Frequency of major clinical warning signs in patients with pulmonary embolism compared with non-PE patients, demonstrating significantly higher occurrence of dyspnea, hypoxia, tachycardia, and syncope in the PE group, indicating recognizable pre-collapse deterioration patterns.

DISCUSSION

The present study demonstrates that pulmonary embolism represents a frequent cause of sudden clinical deterioration in immobilized medical patients and is strongly linked with prolonged immobility and absence of thromboprophylaxis. The high proportion of confirmed embolic events in the study population supports the concept that venous thromboembolism is primarily a hospital-acquired and preventable complication rather than an unpredictable event. Immobilization exceeding five days was significantly associated with pulmonary embolism, highlighting the dominant contribution of venous stasis within Virchow's triad. Reduced calf muscle pump activity leads to pooling of blood in the deep veins of the lower limb, initiating thrombus formation that may later embolize to the pulmonary circulation [12-14].

Medical comorbidities were a contributory factor in the development of thrombus. Statistically significant relationships between fatal embolism and heart failure and prior history of deep vein thrombosis showed that both circulatory stasis and pre-existing hypercoagulability are significant determinants of mortality. Even though malignancy, sepsis and dehydration were prevalent in the embryonic group, they were not significantly different as compared to their control, indicating that, immobilization itself is still the main precipitating factor. Notably, the lack of thromboprophylaxis was found to be strongly linked with the occurrence of death highlighting that a substantial number of them could have been averted [15-17].

Age and comorbid disease also contributed to risk. Patients with heart failure and previous deep vein thrombosis showed significantly higher embolic occurrence, suggesting the combined effects of circulatory stasis and pre-existing hypercoagulability. Although malignancy, dehydration, and sepsis were frequently observed in the embolism group, their statistical association was weaker, indicating that immobilization itself remains the principal triggering factor in medically ill patients. Importantly, lack of thromboprophylaxis was one of the strongest modifiable predictors, reinforcing that many of these events are clinically preventable through routine risk assessment and prophylactic measures [18-20].

The premortem clinical profile demonstrated recognizable warning signs before deterioration. Dyspnea, tachycardia, hypoxia, syncope, and elevated D-dimer were significantly more common among embolism cases. These findings indicate that pulmonary embolism rarely occurs entirely without symptoms; rather, transient cardiopulmonary instability often precedes collapse but may be overlooked in sedated or debilitated patients. Recognition of these early indicators is therefore essential for clinicians, particularly in wards and intensive care settings where baseline illness may mask acute changes [21, 22].

Anatomical findings confirmed the classical embolic pathway originating from the deep veins of the lower limbs. The femoral and popliteal veins accounted for the majority of thrombus sources, supporting the concept that calf and thigh venous stasis plays a central role in pathogenesis. The most frequent embolic location was the pulmonary arterial bifurcation (saddle embolus), commonly associated with acute right ventricular dilatation. This explains the sudden hemodynamic collapse observed clinically, as obstruction of the pulmonary trunk produces abrupt elevation of pulmonary arterial pressure and acute right heart failure. The anatomical correlation therefore provides a clear structural explanation for the rapid clinical deterioration seen in affected patients [23].

Overall, the findings emphasize that pulmonary embolism in immobilized medical patients follows a predictable sequence: venous stasis in lower limb veins, thrombus propagation through the inferior vena cava, and obstruction of the pulmonary arterial circulation leading to acute cardiopulmonary failure. Understanding this clinicopathological sequence is essential for early detection and prevention strategies in hospitalized patients.

CONCLUSION

Pulmonary embolism is a common and largely preventable cause of sudden clinical deterioration in immobilized medical patients. Prolonged immobility, absence of thromboprophylaxis, heart failure, and prior thrombotic disease significantly increase risk. Patients frequently demonstrate identifiable warning signs including dyspnea, hypoxia, tachycardia, and syncope before collapse. Anatomically,

emboli most often originate from the femoral and popliteal veins and lodge at the pulmonary arterial bifurcation, producing acute right ventricular failure.

Early risk stratification, vigilant clinical monitoring, and routine prophylaxis can substantially reduce morbidity and mortality. Integrating clinical assessment with understanding of venous anatomical pathways allows timely recognition and intervention, transforming thromboembolism from an unexpected event into a preventable medical complication.

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