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RETROSPECTIVE STUDY OF CLINICAL PATIENTS AND MANAGEMENT OF PULMONARY THROMBOEMBOLISM IN A TERITIORY CARE CENTRE

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INTRODUCTION

Pulmonary embolism causes cardiovascular death and disability as well as psychological illness and emotional distress.[1] Acute pulmonary embolism is a life-threatening condition and the 3rd most common cause of cardiovascular disease mortality worldwide.[2] Pulmonary Embolism is the most common preventable cause of hospital death and responsible for 10% of hospital deaths.[3] The diagnosis of pulmonary embolism is often delayed due to its non-specific signs and symptoms which is the cause for mortality So high degree of clinical suspicion is needed for timely diagnosis and treatment.

Pulmonary Embolism occurs when a blood clot that has arisen from a different area obstruct the pulmonary arteries. Most often pulmonary embolism originates from a DVT in the lower limbs than from upper limbs.[5,6] Pulmonary Embolism can be classified as massive pulmonary embolism, sub-massive pulmonary embolism, small to moderate Pulmonary Embolism. Patient presents with systolic blood pressure <90mm hg, poor perfusion multiple system failure due to extensive thrombus such as saddle Pulmonary Embolism or Right or Left main pulmonary artery thrombus. In sub-massive pulmonary embolism patient is hemodynamically stable but RV dysfunction can be seen where as in small to moderate pulmonary embolism pt has normal hemodynamic with normal RV function.[7]

This classification assists in prognostication and clinical management. Pulmonary Embolism causes non-specific signs and symptoms hence clinical suspicion is utmost important in arriving at the diagnosis.as Pulmonary Embolism notoriously masquerades other illness, patient present with unexplained dyspnoea, tachypnoea or chest or present of risk factors for Pulmonary Embolism must undergo diagnostic tests for Pulmonary Embolism.[8]

Plasma d-dimer through a nonspecific test can be used as a screening tool when pulmonary embolism is not among the likely diagnosis. But when Pulmonary Embolism is strongly suspected one can directly proceed to CT chest imaging without d-dimer.[9] the upper limit of d-dimer needs to be adjusted according to age. For older patients the age adjusted d-dimer cut-off level is age multiplied by.[10] Despite recent advancements pulmonary embolism is underdiagnosed and unreported in india.[11] our study tries to shed more light regarding various clinical aspects of pulmonary embolism.

MATERIAL AND METHODS

We conducted a retrospective study of patients with acute Pulmonary Embolism at our institute between AUG 2024 to AUGUST 2025. the study included all the patients diagnosed with acute pulmonary thromboembolism. Data on clinical presentation, Investigations and management were collected and analysed. A diagnosis of pulmonary embolism was established of there was a evidence of thrombus on CT Pulmonary Angiography.

Patients with pulmonary embolism were categorised

based on clinical and echocardiography findings. Massive pulmonary thromboembolism was diagnosed by evidence of hemodynamic compromise (systolic blood pressure <90mmhg) while sub-massive pulmonary embolism was defined by right ventricular dysfunction on echocardiography in the absence of hemodynamic compromise. Patients without either of these features were classified as having minor pulmonary embolism.

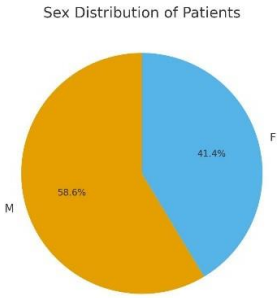
D dimer testing was performed using enzyme limed fluorescent assay with values >500ng/dl considered positive.2D ECHOCARDIOGRAPHY was performed using PHILIPS HD 7 machine, CT Pulmonary Angiography was carried by 192 slice siemens machine. Necessary biomedical investigations were done.

Patient eligible for thrombolytic therapy received Tenecteplase.

The rest of the patients were anticoagulated with low molecular weight heparin. Statistical Analysis: Data were analysed using descriptive and inferential statistics. A p-value < 0.05 was considered statistically significant.

RESULTS

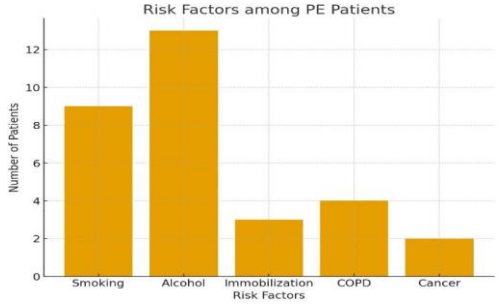
29 patients diagnosed with Pulmonary Embolism were included in the study. The mean age of patients included in the study was found to be 51.5 years.



Male Female ratio was 1.42:1

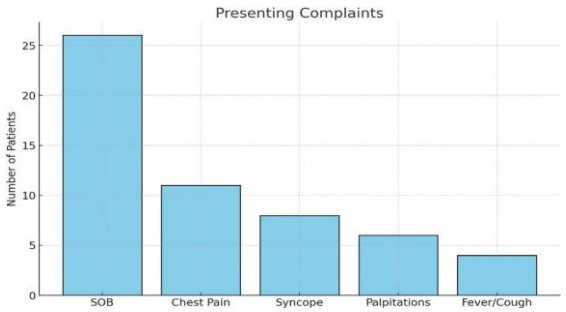
Risk factors are assessed and illustrated in the table, mc risk factor in our study was alcohol.

Risk factor	No.of patients
SMOKING	9-(31.03%)
ALCOHOL	13-(44-82%)
H/O IMMOBILITY	3-(10.34%)
H/O COPD	4-(13.79%)
H/O CARCINOMA	2-(6.8%)



Symptoms

SYMPTOMS	NO.OF PATIENTS
SHORTNESS OF BREATH	26 PATIENTS (89.6%)
CHEST PAIN	11 PATIENTS (37.9%)
SYNCOPE	8 PATIENTS (27.57%)
PALPITATIONS	6 PATIENTS (20.6%)
FEVER WITH COUGH	4 PATIENTS (13.7%)

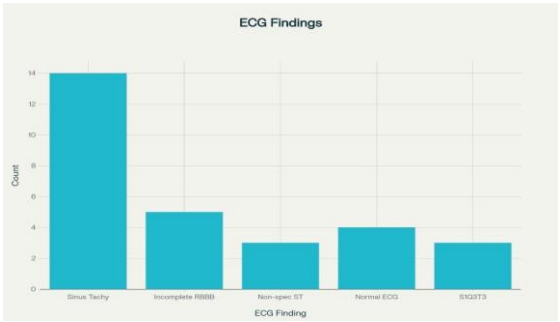


D DIMER

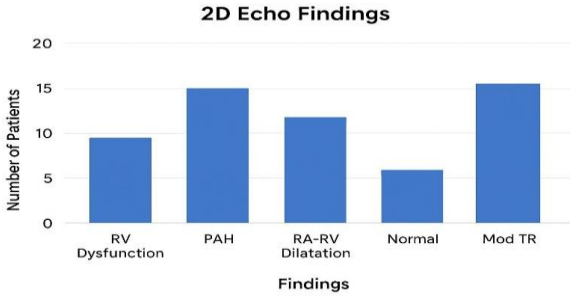
28 patients have elevated d dimer, 1 patient is negative for d dimer and is highly suspicious patient.

ECG

ECG CHANGES	NO.OF PATIENTS
SINUS TACYCARDIA	14 PATIENTS (48.2%)
RBBB	5 PATIENTS (17.2%)
ST SEGMENT CHANGES	3 PATIENTS (10.3%)
NORMAL	4 PATIENTS (13.7%)
S1Q3T3	3 PATIENTS (10.3%)



ECHO FINDINGS	NO.OF PATIENTS
RV DYSFUNCTION	9 PATIENTS (31.03%)
PAH	16 PATIENTS (55.17%)
RA -RV DILATION	11 PATIENTS (37.93%)
NORMAL	6 PATIENTS (20.68%)
TR	17 PATIENTS (58.62%)



CT PULMONARY ANGIOGRAPHY

CTPA was done for all the patients was positive for presence of pulmonary thrombus.

Based on CTPA findings and other investigations patients were classified into Massive & Sub- massive, Minimal PE 3 patients with massive pulmonary embolism-Tenecteplase was administered. 6 patients with sub massive pulmonary embolism -managed conservatively 20 patients with mild pulmonary embolism- managed conservatively.

DISCUSSION

Pulmonary embolism (PE) notoriously masquerades as other illnesses such as asthma, pneumonia, and congestive heart failure, and often occurs concomitantly with other medical conditions.[6] Hence, it remains a diagnostic challenge, while progress in diagnostic modalities and treatment alternatives has improved considerably. However, challenges remain in achieving prompt diagnosis, refining therapeutic approaches, and minimizing long-term complications.[12,13,14] The present study enhances current knowledge regarding the clinical manifestation of pulmonary embolism.

Recent surgery, malignancy, immobilization, and recent hospitalization are major risk factors for the development of venous thromboembolism (VTE). Hospitalization for surgery or acute medical illness accounts for nearly 50–66% of the total VTE burden. Estrogen- and progesterone- containing medications also increase the risk of VTE.[15] Major modifiable risk factors include obesity, cigarette smoking, and hypertension. In the present study, approximately 70–80% of patients had a definite identifiable risk factor.

Patients presenting with dyspnoea, tachycardia, and hypoxia (in about 50% of cases) should raise suspicion of pulmonary embolism. Syncope and cyanosis generally indicate massive pulmonary embolism[17]. In our series, dyspnoea was the most common presenting symptom, followed by chest pain.

The diagnostic workup of pulmonary thromboembolism (PTE) requires a battery of investigations such as electrocardiography (ECG), chest X-ray (CXR), arterial blood gas (ABG) analysis, D- dimer assay, cardiac biomarkers, and CT pulmonary angiography (CTPA).[18]

Echocardiogram findings in pulmonary thromboembolism(PTE) are diverse and non-specific and may include T-wave inversions, ST-segment abnormalities, incomplete or complete right bundle branch block, right axis deviation, and sinus tachycardia. Sinus tachycardia is the most common ECG finding. In approximately 20% of cases, ECG may be normal. The classical S1Q3T3 pattern is seen in about 10% of patients, as demonstrated by McGinn and White.[18,19] In our study, sinus tachycardia occurred frequently in patients with PTE, a finding that parallels observations made in earlier studies.

The plasma D-dimer is a specific derivative of cross-linked fibrin. When the D-dimer level is below the established cut-off value, the sensitivity and negative predictive value for venous thromboembolism (VTE) are 95% or higher.[20] Elevated D-dimer levels are non-specific for pulmonary embolism and may be seen with advancing age, infections, malignancy, and during the second and third trimesters of pregnancy. Age-adjusted cut-off values (age × 10 µg/L above 50 years) improve the specificity of D-dimer testing. In our case series, D-dimer was positive in 85– 90% of patients, which was similar to studies conducted by Hammond and Hassan, who reported that no patients with D-dimer levels <275 ng/dL had pulmonary embolism.[22]

In the majority of cases, pulmonary embolism originates from lower-extremity deep vein thrombosis (DVT). Venous ultrasonography (USG) establishes the diagnosis of DVT and is a widely available technique.[23] In the setting of suspected pulmonary embolism, USG is considered diagnostic when it demonstrates DVT in a symptomatic patient. However, in many cases of CT-confirmed pulmonary embolism, there is no evidence of peripheral venous thrombosis, probably because the entire thrombus embolized to the pulmonary arteries.[24] In our present study 30-40% had evidence of DVT.

Echocardiographic assessment of right ventricular(RV) function aids in prognostic assessment, particularly in normotensive patients. The presence of RV dysfunction represents an independent predictor of adverse outcomes.[25] Patients with severe RV dysfunction may demonstrate McConnell's sign, characterized by severe hypokinesia of the RV apex.[26] A normal echocardiogram in a hemodynamically unstable patient significantly reduces the likelihood of acute pulmonary embolism as the cause of hypotension.[27] In the present study, 45-55% of patients had RV dysfunction, and 35-45% had pulmonary hypertension.

Multidetector CT pulmonary angiography (CTPA) has become the initial imaging modality in most patients with suspected pulmonary embolism.[28] The latest generation of scanners can image thrombi in sixth-order pulmonary vessels. CT chest can also detect pulmonary diseases that coexist with pulmonary embolism. CTPA can demonstrate signs of RV dysfunction by assessing the RV-to-LV diameter ratio, RV-to-LV volume ratio, and interventricular septal bowing.[29] In the present study, CTPA was the imaging modality used for diagnosis of pulmonary thromboembolism.

Anticoagulation remains the cornerstone of treatment of acute pulmonary embolism. In addition to anticoagulation, high-risk patients may require systemic thrombolysis or Surgical embolectomy.[30]

CONCLUSION

Majority of Pulmonary Embolism patients presented with dyspnoea and had echocardiographic evidence of Pulmonary Artery Hypertension or Tricuspid Regurgitation. Early diagnosis using non-invasive methods like 2D Echo and CTPA can improve management outcomes.

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