"AN ANALOGY BETWEEN CLOT BURDEN AND RIGHT VENTRICULAR DYSFUNCTION IN ACUTE PULMONARY THROMBOEMBOLISM ON COMPUTED TOMOGRAPHY PULMONARY ANGIOGRAPHY USING MODIFIED MILLERS SCORE: A ONE YEAR HOSPITAL BASED CROSS SECTIONAL STUDY"

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Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled **"AN ANALOGY BETWEEN CLOT BURDEN AND RIGHT VENTRICULAR DYSFUNCTION IN ACUTE PULMONARY THROMBOEMBOLISM ON COMPUTED TOMOGRAPHY PULMONARY ANGIOGRAPHY USING MODIFIED MILLERS SCORE ",** is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee.

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ABSTRACT

Background: Acute pulmonary thromboembolism (PTE) is a common disease which is potentially fatal and has a high mortatility rate. PTE is third most common case of cardiovascular morbidity and mortality, after cardiovascular causes and stroke. CTPA is current standard of care and provides accurate diagnosis with rapid turnaround time. Patients with acute PTE and RVD have a higher mortality rate than those without RVD.

Objective: To determine the correlation between increasing pulmonary embolism thrombus load using modified miller's score and right ventricular (RV) dysfunction as demonstrated by CT pulmonary angiography (CTPA) and to investigate any relationship between pulmonary thrombus load, CT signs and 2D Echo features of right ventricular dysfunction.

Methods: The study is a hospital-based prospective study conducted over one year at Dr. Prabhakar Kore Hospital and Medical Research Center, Belagavi. A total of 49 patients with PTE who undergo both CTPA and transthoracic echocardiography. The patients underwent CTPA according to the departmental protocol and the scans were then evaluated for the severity of PTE using the Modified Miller scoring system. The patients were also evaluated for any features of right ventricular decompensation on the CTPA. The pulmonary thrombus load was correlated with transthoracic echocardiography features of right ventricular dysfunction.

Results:A significant association was observed between right ventricular dysfunction markers and clot burden (MMS Categories). Patients with MMS ≥13 had a higher prevalence of right ventricular strain, as indicated by a positive RV/LV ratio in 69.39% of cases ($p = 0.0002$), septal deviation in 30.61% ($p = 0.0012$), and a positive MPA/AA ratio in 65.31% ($p = 0.0025$).

A strong association was observed between right ventricular function parameters and clot burden (MMS Categories) (Table 10). RA/RV enlargement was significantly more frequent in patients with MMS ≥13 (48.98%, $p < 0.0001$), indicating increased right heart strain. Similarly, Pulmonary Artery Systolic Pressure (PASP) was positive in 40.82% of MMS ≥13 patients ($p < 0.0001$), suggesting elevated pulmonary pressures. Tricuspid Annular Plane Systolic Excursion (TAPSE) was significantly lower in the MMS ≥13 group, with only 10.20% showing a negative TAPSE compared to 42.86% with positive TAPSE ($p < 0.0001$). These findings highlight the strong link between higher clot burden and worsening right ventricular function, reinforcing the clinical value of MMS in predicting cardiac strain in acute pulmonary thromboembolism.

Conclusion: In our study, there was significant correlation between MMS score >13 and right ventricular dysfunction on CTPA (P value <0.01). The MMS CT score of >13 could be used to predict right ventricular dysfunction according to our study and this could be useful in the faster diagnosis of PTE and right heart strain using only CTPA.

LIST OF ABBREVIATIONS

Abbreviation	Full Form
PE	Pulmonary Embolism
PTE	Pulmonary Thromboembolism
DVT	Deep Vein Thrombosis
RV	Right Ventricle
LV	Left Ventricle
RVD	Right Ventricular Dysfunction
CTPA	Computed Tomography Pulmonary Angiography
V/Q	Ventilation-Perfusion
IVC	Inferior Vena Cava
TAPSE	Tricuspid Annular Plane Systolic Excursion
BNP	Brain Natriuretic Peptide
NT-proBNP	N-terminal Pro B-type Natriuretic Peptide
tPA	Tissue Plasminogen Activator
USAT	Ultrasound-Accelerated Thrombolysis
CTEPH	Chronic Thromboembolic Pulmonary Hypertension
RV/LV	Right Ventricle/Left Ventricle
PA/Ao	Pulmonary Artery/Aortic
RV:LV	Right Ventricle to Left Ventricle Ratio
IVC	Inferior Vena Cava
PASP	Pulmonary Artery Systolic Pressure
MMS	Modified Miller Score
MRA	Magnetic Resonance Angiography
SPECT	Single-Photon Emission Computed Tomography
MDCT	Multidetector Computed Tomography
CT-PESI	CT Pulmonary Embolism Severity Index
QS	Qanadli Score
QOI	Qanadli Obstruction Index
RVD/LVD	Right Ventricular Diameter/Left Ventricular Diameter
ECMO	Extracorporeal Membrane Oxygenation

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INTRODUCTION

Acute pulmonary thromboembolism (PTE) is a life-threatening cardiopulmonary disorder caused by the obstruction of the pulmonary arteries by thrombi. It is associated with significant morbidity and mortalitymaking it the third most common cardiovascular disorder after myocardial infarction and stroke. The ability to identify high-risk patients is crucial for optimizing treatment strategies and improving patient outcomes. Pulmonary embolism (PE) leads to an abrupt increase in pulmonary vascular resistance, which can cause right ventricular (RV) dysfunction, heart failure, and even death. Therefore, timely and accurate diagnosis is essential to prevent adverse clinical outcomes (1).Computed tomography pulmonary angiography (CTPA) has emerged as the gold standard imaging modality for diagnosing acute PE. It provides rapid, highly accurate and non-invasive visualization of pulmonary artery occlusion and allows for comprehensive evaluation of clot burden. With advancements in imaging technology, CTPA has demonstrated superior sensitivity and specificity compared to other diagnostic modalities such as ventilation-perfusion (V/Q) scans and pulmonary angiography. Furthermore, CTPA enables the simultaneous assessment of cardiac structures, including the right ventricle, offering a prognostic advantage in evaluating hemodynamic consequences of PTE (2).Right ventricular dysfunction is a key determinant of prognosis in patients with PE. RV overload due to increased pulmonary artery pressure can lead to progressive dilation, tricuspid regurgitation, and decreased cardiac output, ultimately resulting in hemodynamic instability. Various echocardiographic parameters, such as RV:LV ratio, tricuspid annular plane systolic excursion (TAPSE), and right atrial enlargement, have been used for risk stratification of PE patients. However, echocardiography has limitations in evaluating clot burden, making CTPA an essential tool for assessing the severity of thrombus obstruction and predicting RV dysfunction (3).Several scoring systems have been developed to quantify clot burden in PE, with the Modified Miller Score (MMS) being one of the most commonly used methods. The MMS is a semi-quantitative system that assigns scores based on the extent of arterial occlusion at the segmental and subsegmental levels. A higher MMS is associated with increased pulmonary artery obstruction, greater RV strain, and a higher risk of mortality. Studies have shown that an MMS of greater than 12 correlates with significant RV decompensation, serving as a threshold beyond which hemodynamic instability is more likely to occur (4).The

relationship between clot burden and RV dysfunction has been the focus of several clinical investigations. Conducted a retrospective cohort study on 2,425 patients and found that increasing thrombus load was strongly correlated with CTPA-based evidence of RV dysfunction, with an MMS exceeding 12 representing a critical tipping point for decompensation. Similarly, Introduced a computed tomographic index to quantify arterial obstruction, demonstrating a strong correlation with pulmonary angiographic indices and echocardiographic findings of RV dysfunction. These findings reinforce the role of clot burden quantification in prognostic risk stratification (5).Assessed the reliability of CTPA for evaluating thrombus burden and RV strain and found that the modified Miller and refined Miller scores provided excellent reproducibility across multiple observers. Their study also confirmed that CTPA-derived RV:LV ratio and clot burden scores were highly predictive of adverse clinical outcomes, emphasizing the importance of integrating these parameters into routine clinical assessments. Further explored regional RV dysfunction (RRVD) in acute PE patients and demonstrated that RRVD was strongly associated with increased clot burden, highlighting the utility of CTPA in identifying high-risk individuals (6).Beyond thrombus burden and RV dysfunction, additional CTPA-derived parameters have been proposed as prognostic markers in PE. The pulmonary artery to aortic ratio (PA/Ao), interventricular septal bowing, and decreased left ventricular outflow have all been linked to worse clinical outcomes in PE patients. Reported that PA/Ao ratio greater than 1 was indicative of significant RV strain and correlated with higher in-hospital mortality rates. Similarly, demonstrated that volumetric analysis of pulmonary CTA provided valuable prognostic insights, reinforcing the role of quantitative imaging techniques in PE risk assessment (7).Biomarkers such as D-dimer, troponin, and brain natriuretic peptide (BNP) have been used to supplement imaging-based risk stratification in PE. Elevated BNP levels have been associated with RV dysfunction and poor clinical outcomes, particularly in patients with intermediate- and high-risk PE. Found that combining CTPA-derived RV metrics with biomarker assessments improved predictive accuracy for adverse events in acute PE patients. However, while biomarkers can provide additional prognostic information, they lack the specificity of imaging-based parameters and should be interpreted in conjunction with CTPA findings (8).Despite its advantages, CTPA is not without limitations. Radiation exposure and contrast-induced nephropathy remain concerns, particularly in patients with renal impairment or iodine contrast allergies. Additionally, false-positive findings can occur due to motion artifacts or anatomical variations, necessitating careful

interpretation by experienced radiologists. Strategies such as iterative reconstruction techniques and dual-energy CT imaging have been developed to enhance diagnostic accuracy while minimizing radiation dose (9).The integration of artificial intelligence (AI) and machine learning into PE diagnostics holds promise for improving efficiency and accuracy. AI-driven algorithms can automatically detect thrombus burden, assess RV dysfunction, and predict patient outcomes based on large-scale imaging datasets. demonstrated that deep learning-based models could accurately identify PE-related RV strain and provide automated quantification of clot burden, potentially streamlining the diagnostic workflow. While AI applications in PE are still evolving, their potential to enhance clinical decision-making is significant (10).Management strategies for PE vary based on risk stratification, with treatment options ranging from anticoagulation to thrombolysis and catheter-directed interventions. High-risk patients with massive PE and hemodynamic instability require immediate reperfusion therapy, while intermediate-risk patients may benefit from risk-adapted strategies incorporating advanced imaging and biomarker assessments. The ability to accurately quantify clot burden and RV dysfunction using CTPA can aid in selecting the most appropriate therapeutic approach and optimizing patient outcomes (11).

In conclusion, acute pulmonary thromboembolism remains a major cause of morbidity and mortality, necessitating timely diagnosis and risk stratification. CTPA has emerged as the primary imaging modality for evaluating PE, offering valuable insights into thrombus burden and RV dysfunction. The Modified Miller Score serves as a useful tool for quantifying clot burden and predicting hemodynamic consequences. Numerous studies have demonstrated a strong correlation between increasing MMS and RV decompensation, emphasizing its prognostic relevance. While echocardiography and biomarkers provide additional risk assessment, CTPA remains the most comprehensive and accurate method for evaluating PE severity. Future advancements, including AI-driven diagnostics and novel imaging techniques, hold promise for further improving the management of PE. As our understanding of PE continues to evolve, integrating imaging-based risk stratification into clinical practice will play a crucial role in optimizing patient care (12).

AIMS AND OBJECTIVES

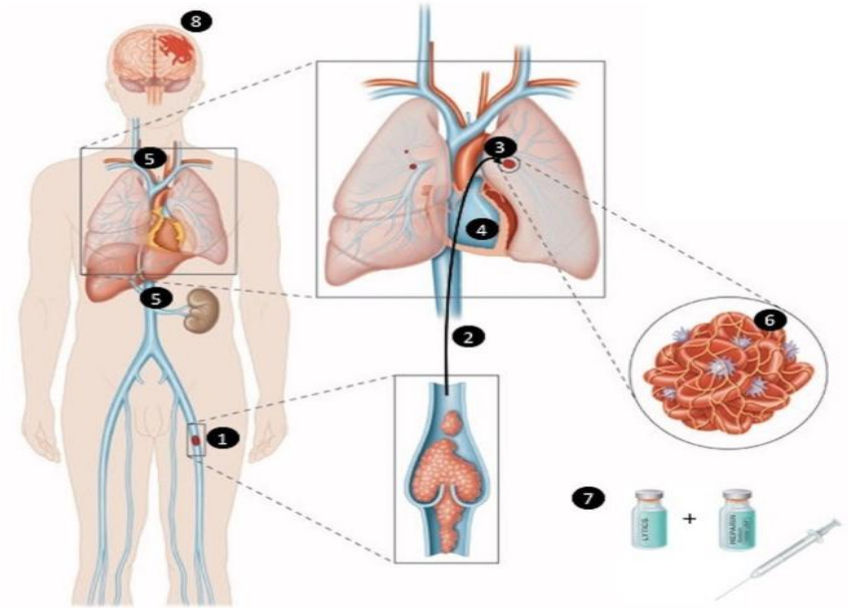
- To determine the correlation between increasing pulmonary embolism thrombus load using modified miller's score and right ventricular (RV) dysfunction as demonstrated by CT pulmonary angiography (CTPA).

- To investigate any relationship between pulmonary thrombus load, CT signs and 2D Echo features of right ventricular dysfunction.

REVIEW OF LITERATURE

2.1 PULMONARY THROMBOEMBOLISM (PTE)

Pulmonary thromboembolism (PTE) is a critical cardiovascular condition characterized by the obstruction of pulmonary arteries by thrombotic emboli. It is a significant cause of morbidity and mortality worldwide and is considered the third most common cardiovascular disease after myocardial infarction and stroke. PTE occurs when a thrombus, primarily originating from the deep veins of the lower limbs, dislodges and travels through the venous system, eventually lodging in the pulmonary arterial circulation. This obstruction results in increased pulmonary vascular resistance, leading to impaired oxygenation, hemodynamic instability, and potential right ventricular failure. Due to its complex pathophysiology and variable clinical presentation, timely diagnosis and appropriate management of PTE are crucial for improving patient outcomes (13).



Pathophysiology of high-risk pulmonary embolism (PE) and intracerebral haemorrhage following thrombolysis. In intermediate-risk and high-risk PE, peripheral thrombus (1) and emboli (2) in the arterial pulmonary tree (3) may induce right ventricular dysfunction and sometimes failure (4), leading to a decreased cerebral, renal and hepatic venous return. The venous congestion (5) added to hepatic hypoxaemia, and an impaired renal clearance may affect haemostasis and PE clot sensitivity to lytics (6). Therapies associating heparin infusion and thrombolytics (7) could provoke major bleeding (8).

Figure 1: Pathophysiology of Pulmonary Thromboembolism

Pathophysiology of Pulmonary Thromboembolism

The pathophysiological mechanism of PTE primarily involves Virchow's triad, which describes the three major factors contributing to thrombus formation: venous stasis, endothelial injury, and hypercoagulability. Venous thrombi commonly originate in the deep veins of the lower extremities, known as deep vein thrombosis (DVT). Once a thrombus forms, it may remain in place, dissolve, or embolize to the lungs. When an embolus occludes a pulmonary artery, several pathophysiological consequences occur, including increased pulmonary arterial pressure, hypoxia, ventilation-perfusion mismatch, and right ventricular overload (14).

The immediate hemodynamic impact of PTE depends on the size and number of emboli. A massive PE leads to a significant increase in pulmonary vascular resistance, causing acute right ventricular (RV) strain, reduced left ventricular preload, and ultimately cardiovascular collapse. In contrast, submassive or non-massive PE may not cause immediate hemodynamic compromise but can still result in right ventricular dysfunction (RVD) due to persistent pressure overload. Pulmonary infarction may occur if small emboli occlude distal arteries, leading to ischemic necrosis of lung parenchyma (15).

Epidemiology and Risk Factors

PTE is a common cause of cardiovascular-related hospitalizations and deaths. The global incidence of PTE varies but is estimated to be 60 to 70 cases per 100,000 individuals annually. The true incidence is likely higher due to underdiagnosis, as many cases remain asymptomatic or present with nonspecific symptoms. Unprovoked PE, which occurs without an identifiable trigger, accounts for nearly 50% of cases, while the remaining cases are associated with identifiable risk factors (16).

Several factors increase the risk of PTE, including immobility, surgery, malignancy, pregnancy, hormonal therapy, and genetic predisposition. Prolonged immobilization, such as in long-distance travel, hospital stays, or post-operative recovery, leads to venous stasis, increasing the risk of thrombus formation. Major surgeries, particularly orthopedic procedures involving the hip or knee, carry a high risk of PTE due to endothelial damage and hypercoagulability. Cancer

patients are at increased risk due to the pro-thrombotic effects of malignancies and chemotherapy-induced endothelial dysfunction (17).

Inherited thrombophilias, such as Factor V Leiden mutation, prothrombin gene mutation, and deficiencies of antithrombin, protein C, or protein S, also contribute to hypercoagulability and increase the risk of venous thromboembolism (VTE). Hormonal factors, including oral contraceptives, hormone replacement therapy, and pregnancy, further elevate the risk due to estrogen-induced increases in clotting factors and reductions in anticoagulant proteins (18).

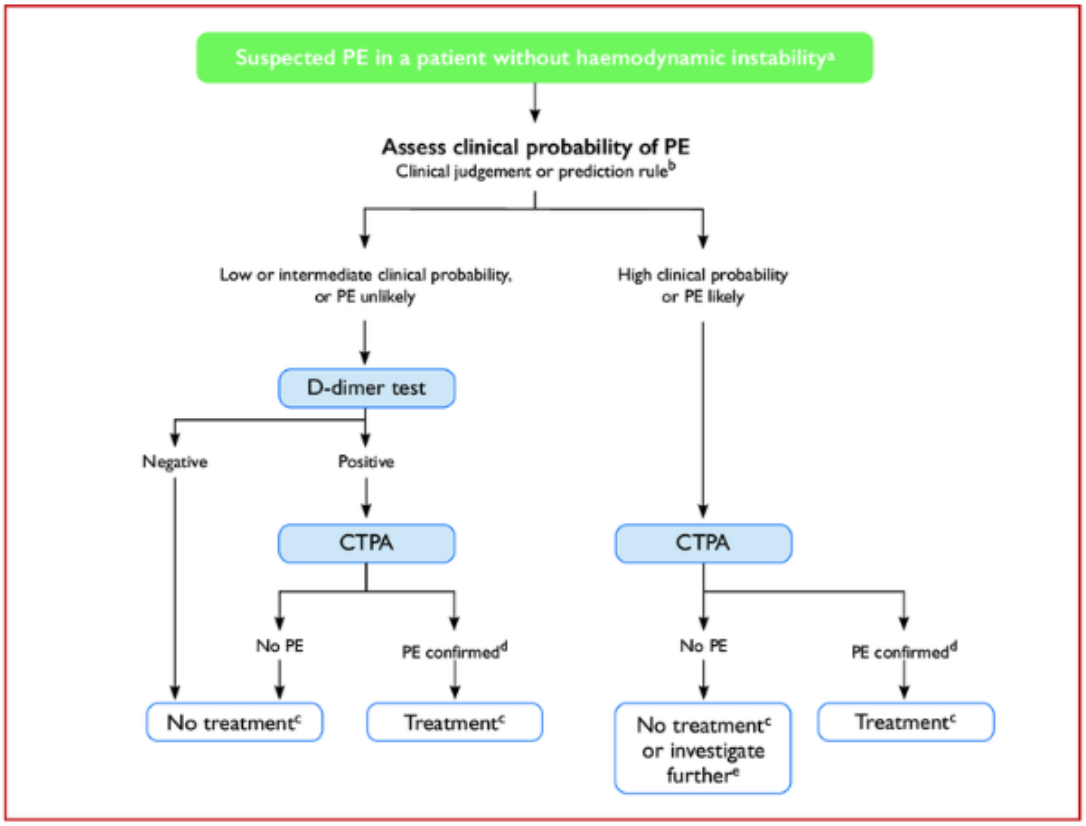
Risk factors	
Genetic factors	Aquired factors
Thrombophilia	Prolonged immobilization
Mutation of the factor V Leiden	Recent orthopedic surgery
Mutation of the prothrombin gene	Malignancy
Hiperhomocisteinemia	Permanent venous catheter
Protein C deficiency	Obesity
Protein S deficiency	pregnancy

Figure 2: Risk Factors for Pulmonary Embolism

Clinical Presentation and Symptoms

The clinical manifestations of PTE are highly variable, ranging from mild symptoms to sudden cardiovascular collapse. The classic triad of symptoms—dyspnea, pleuritic chest pain, and hemoptysis—is observed in less than 20% of cases. The most common symptom is acute dyspnea, present in approximately 80% of patients, followed by pleuritic chest pain (50%) and cough (37%). In severe cases, syncope or circulatory shock may indicate massive PE with significant hemodynamic compromise (19).

Physical examination findings in PTE are often nonspecific but may include tachypnea (respiratory rate >20 breaths/min), tachycardia (heart rate >100 bpm), and hypoxia (oxygen saturation <95%). In cases of right ventricular dysfunction, signs such as jugular venous distension, right-sided heart failure, and a loud pulmonic component of the second heart sound (P2) may be observed. Massive PE may result in profound hypotension, requiring immediate intervention (20).



Diagnostic algorithm for patients with suspected pulmonary embolism without haemodynamic instability. CTPA = computed tomography pulmonary angiography/angiogram; PE = pulmonary embolism. a The proposed diagnostic strategy for pregnant women with suspected acute PE is discussed in section 9. b

Figure 3: Diagnostic algorithm for patients with suspected pulmonary embolism without haemodynamic instability

Diagnostic Modalities in PTE

Given its nonspecific symptoms, a systematic diagnostic approach is essential for confirming PTE. Several diagnostic tests are used to assess suspected cases:

1. D-dimer Testing:

   D-dimer is a fibrin degradation product elevated in conditions associated with thrombus formation. It has high sensitivity (≥95%) but low specificity, as elevated levels can be seen in infections, malignancies, and inflammatory states. A negative D-dimer test effectively rules out PTE in low-risk patients but is not definitive in high-risk individuals.

2. Computed Tomography Pulmonary Angiography (CTPA):

   CTPA is the gold standard imaging modality for diagnosing PTE. It provides direct visualization of pulmonary arterial thrombi with a sensitivity of 83%–100% and specificity of 89%–100%. CTPA also allows for evaluation of right ventricular strain by assessing the RV:LV ratio, pulmonary artery size, and septal deviation.

3. Ventilation-Perfusion (V/Q) Scan:

   The V/Q scan is an alternative imaging technique, particularly useful for patients with contraindications to CTPA (e.g., renal impairment or contrast allergy). A mismatched perfusion defect with normal ventilation is highly suggestive of PTE (21).

4. Echocardiography:

   Echocardiography is useful for assessing right ventricular dysfunction (RVD) in hemodynamically unstable patients. Findings such as McConnell's sign (regional RV dysfunction with preserved apical contractility) and increased pulmonary artery pressure support the diagnosis.

5. Pulmonary Angiography:

   Although invasive, pulmonary angiography remains the definitive test for PTE when noninvasive modalities are inconclusive. It provides real-time visualization of emboli and allows for therapeutic intervention, such as catheter-directed thrombolysis (22).

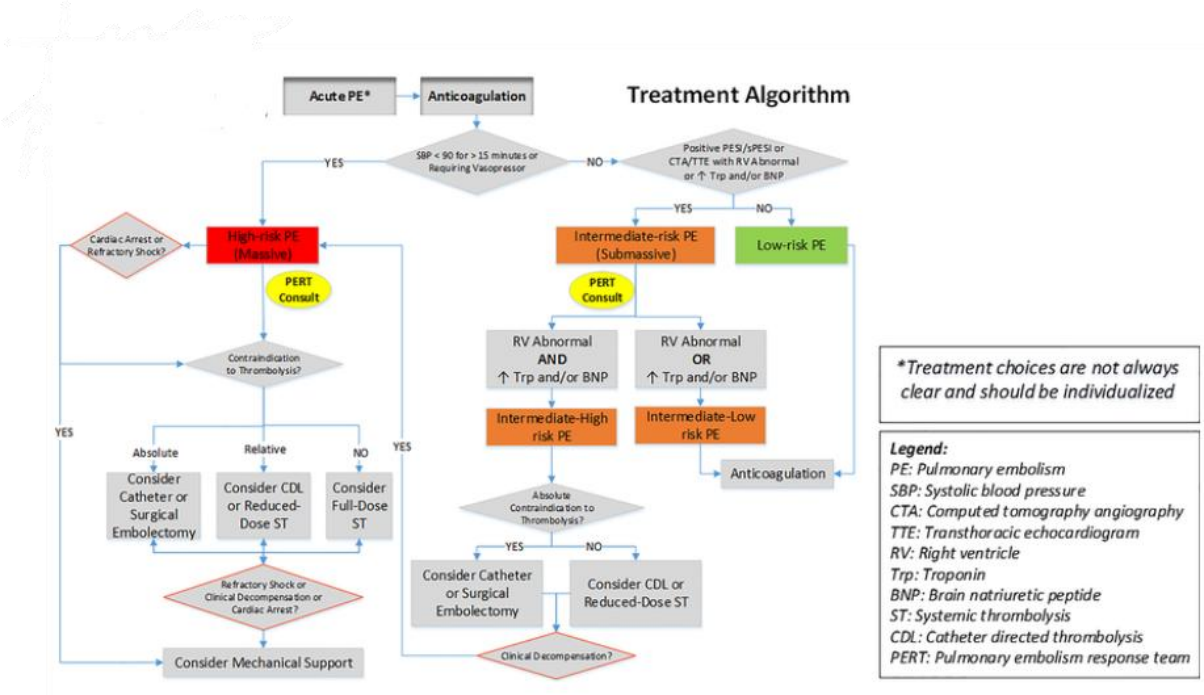


Figure 4: Pulmonary embolism treatment algorithm

Management of PTE

Treatment strategies for PTE depend on the severity of presentation and risk stratification:

- Low-Risk PE: Patients with hemodynamic stability and no evidence of RVD are treated with anticoagulation therapy using low molecular weight heparin(LMWH), unfractionated heparin (UFH), or direct oral anticoagulants (DOACs).
- Intermediate-Risk PE (Submassive PE): Patients with RVD but without hemodynamic instability may benefit from anticoagulation, systemic thrombolysis, or catheter-directed interventions based on individual risk assessment (23).
- High-Risk PE (Massive PE): Patients with hemodynamic instability require urgent thrombolysis with tissue plasminogen activator (tPA) or surgical embolectomy if thrombolysis is contraindicated.

- Chronic Thromboembolic Pulmonary Hypertension (CTEPH): Patients who develop persistent pulmonary hypertension due to unresolved emboli may require pulmonary endarterectomy or balloon pulmonary angioplasty (24).

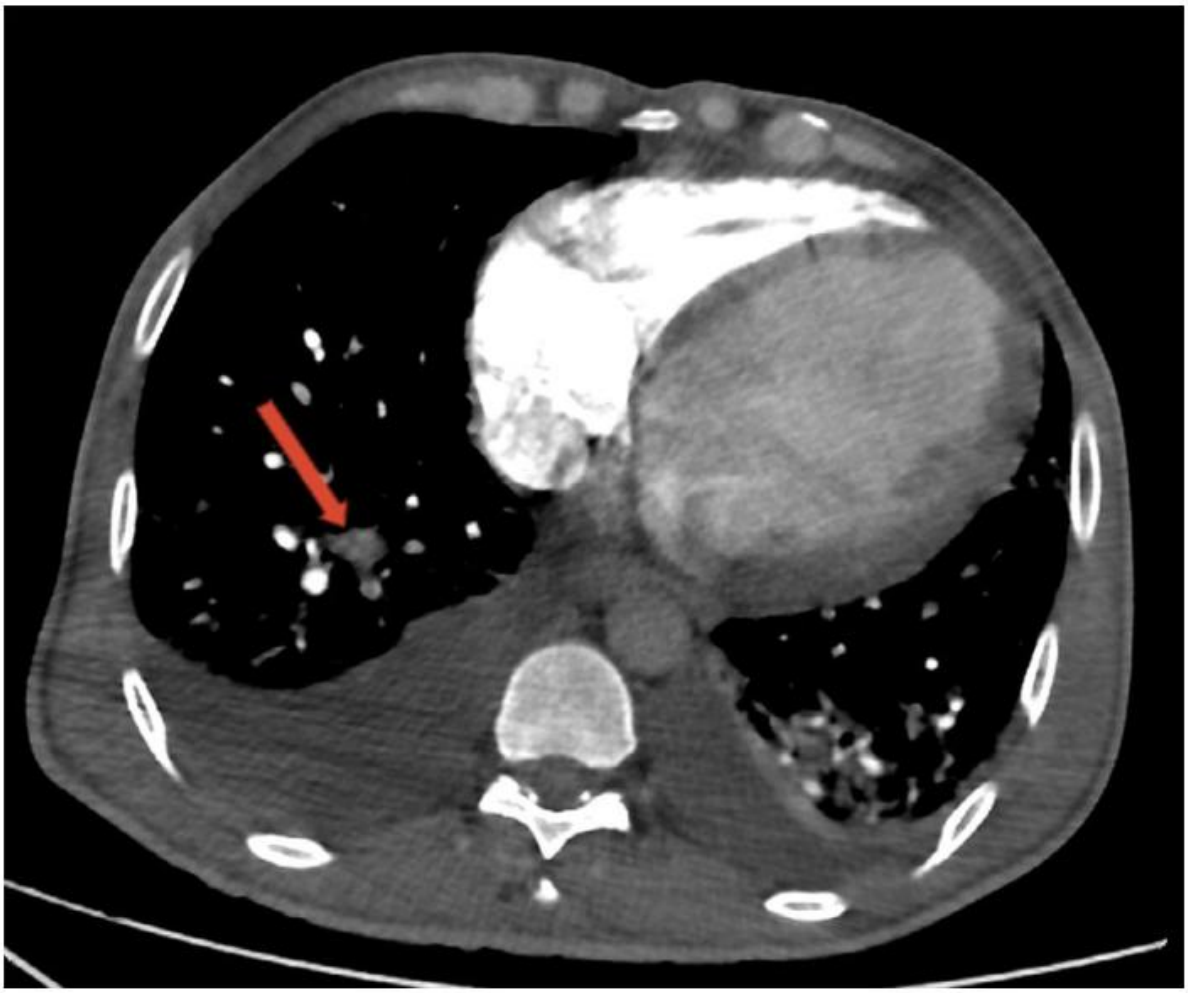
2.2 ROLE OF IMAGING IN THE DIAGNOSIS OF PULMONARY EMBOLISM

Pulmonary embolism (PE) is a life-threatening cardiovascular condition that requires rapid and accurate diagnosis to prevent adverse outcomes. The role of imaging in the diagnosis of PE has evolved significantly over the past decades, with advanced imaging modalities now offering highly sensitive and specific diagnostic capabilities. The choice of imaging technique depends on clinical presentation, patient risk stratification, and the availability of imaging resources. **Computed Tomography Pulmonary Angiography (CTPA)** has emerged as the **gold standard** for PE diagnosis, providing direct visualization of thrombi within the pulmonary arteries. However, alternative imaging methods such as **ventilation-perfusion (V/Q) scans, echocardiography, magnetic resonance angiography (MRA), and conventional pulmonary angiography** remain valuable in specific clinical scenarios. This section discusses the various imaging modalities used in PE diagnosis, their advantages and limitations, and their role in guiding clinical decision-making (25).

Computed Tomography Pulmonary Angiography (CTPA)

CT pulmonary angiography (CTPA) has revolutionized the diagnosis of PE due to its high accuracy, widespread availability, and ability to provide rapid results. It is currently considered the **first-line imaging modality** for PE diagnosis in hemodynamically stable patients. CTPA provides **direct visualization of emboli within the pulmonary arteries**, allowing for assessment of thrombus location, burden, and degree of vascular obstruction.

The diagnostic accuracy of CTPA is superior to older imaging techniques, with a **sensitivity of 83–100% and specificity of 89–100%**. Its ability to **detect alternative causes of chest pain and dyspnea**, such as pneumonia, aortic dissection, or pleural effusion, makes it a **versatile imaging tool**. Modern **multi-detector CT (MDCT) systems** with thin-slice reconstructions enhance image resolution and allow for improved visualization of subsegmental emboli (26).



-Computed tomography pulmonary angiography (CTPA) showing pulmonary embolism (PE) in the segmental branch of the right lower lobe pulmonary artery (A) Axial view (B) Coronal view.

Figure 5: Computed tomography pulmonary angiography

CTPA-Based Risk Stratification in PE

Beyond detecting PE, CTPA plays a crucial role in **risk stratification** by assessing **right ventricular (RV) dysfunction** and **pulmonary arterial obstruction**. Several CTPA-derived parameters have been proposed as prognostic indicators:

1. **Right Ventricle to Left Ventricle (RV:LV) Ratio**:
    - An **RV:LV ratio > 1** is associated with **higher mortality** and suggests **RV strain** due to increased pulmonary arterial pressure.

2. **Pulmonary Artery to Aortic (PA/Ao) Ratio**:
    - A PA/Ao ratio **greater than 1** indicates **significant pulmonary hypertension** and correlates with increased mortality in PE patients.
3. **Septal Bowing and Reflux of Contrast into the Inferior Vena Cava (IVC)**:
    - **Leftward deviation of the interventricular septum** and **contrast reflux into the IVC** are markers of **RV pressure overload**, suggesting hemodynamic compromise (27).

CTPA-based risk stratification aids in selecting **appropriate treatment strategies**, including the need for thrombolysis, catheter-directed interventions, or intensive monitoring in high-risk patients.

Limitations of CTPA

Despite its advantages, CTPA has **certain limitations** that must be considered:

- **Radiation Exposure**: Although CTPA uses ionizing radiation, advances in **low-dose CT protocols** have reduced radiation-related risks.
- **Contrast-Induced Nephropathy (CIN)**: The use of **iodinated contrast agents** poses a risk to patients with **pre-existing renal impairment**.
- **Motion Artifacts**: In **tachycardic or uncooperative patients**, motion artifacts may reduce image quality and lead to **false-positive or false-negative results** (28).

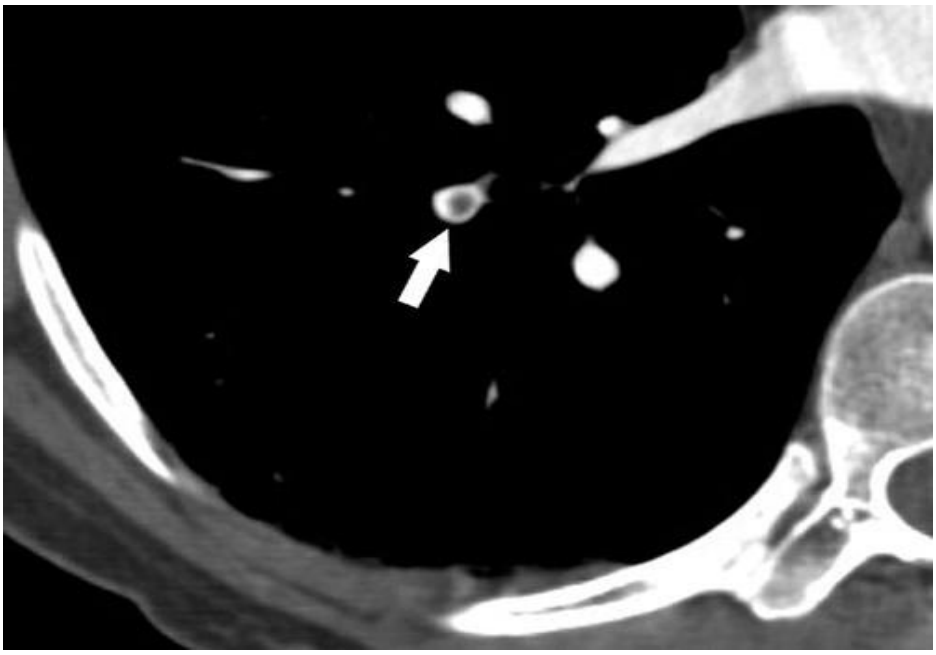


Figure 6: shows an embolus in central location of pulmonary artery in the with contrast surrounding it giving a "polo mint sig"

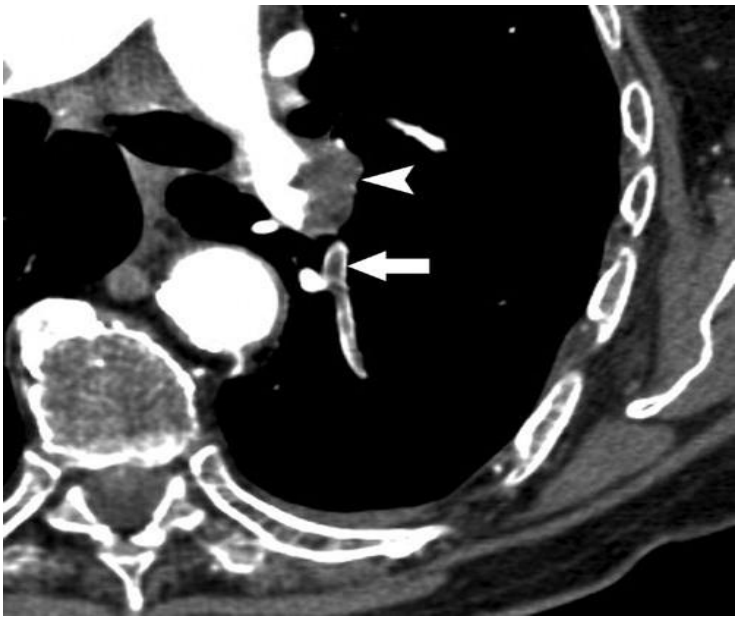


Figure 7: shows along its longitudinal view of a pulmonary artery with central thrombus and surrounding contrast material giving an appearance of "railway track sign"

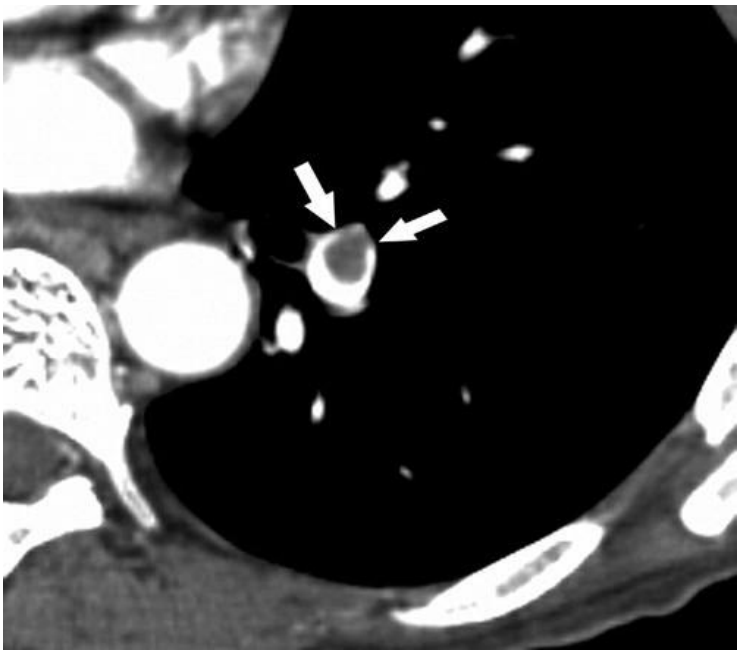


Figure 8: shows an acute embolus in the pulmonary artery causing an acute angle with the vessel wall (shown by the arrows)

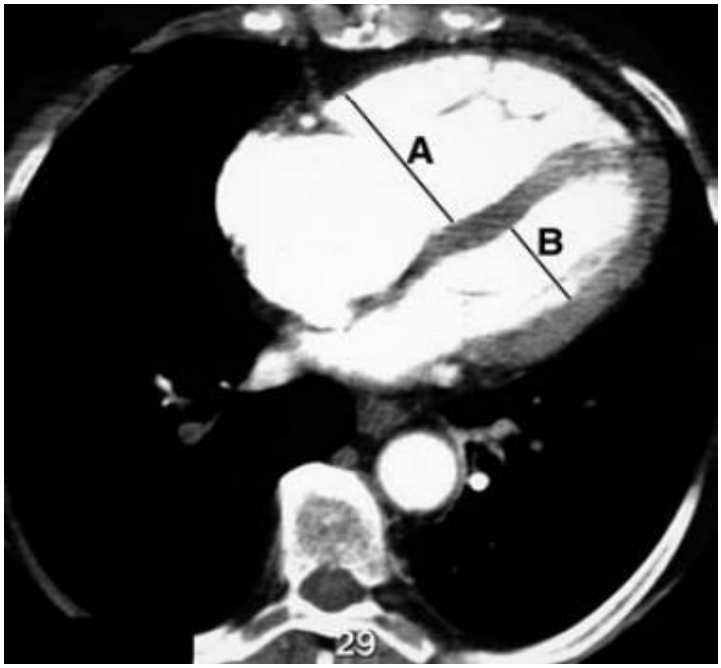


Figure 9: shows the increased short-axis diameter of the right ventricle (A) as compared to the left ventricle (B). Note to be made of the flattened interventricular septum

Ventilation-Perfusion (V/Q) Scan

The **V/Q scan** is an alternative imaging modality for PE, particularly useful in patients with **contrast allergies, chronic kidney disease, or pregnancy**. It evaluates **pulmonary ventilation and perfusion** using radioactive tracers to detect mismatched perfusion defects.

Interpretation of V/Q Scan Findings

1. **Normal Scan**: No evidence of PE.
2. **Low-Probability Scan**: Small, non-segmental perfusion defects that are unlikely due to PE.
3. **Intermediate (Indeterminate) Scan**: Mismatched perfusion defects but not definitive for PE.
4. **High-Probability Scan**: Large segmental or lobar perfusion defects with normal ventilation, highly suggestive of PE (29).

The **PIOPED (Prospective Investigation of Pulmonary Embolism Diagnosis) study** demonstrated that a **high-probability V/Q scan** is **diagnostic of PE in 85–90% of cases**, while a **normal scan effectively excludes PE**. However, in cases of **intermediate or low-probability scans**, additional testing such as CTPA or Doppler ultrasound of the lower limbs is required.

Limitations of V/Q Scan

- **Lower specificity than CTPA**, particularly in patients with **underlying lung disease** (e.g., COPD, pneumonia).
- **Longer acquisition time** compared to CTPA, making it **less suitable for critically ill patients** (30).
- **Availability and expertise required** for accurate interpretation.

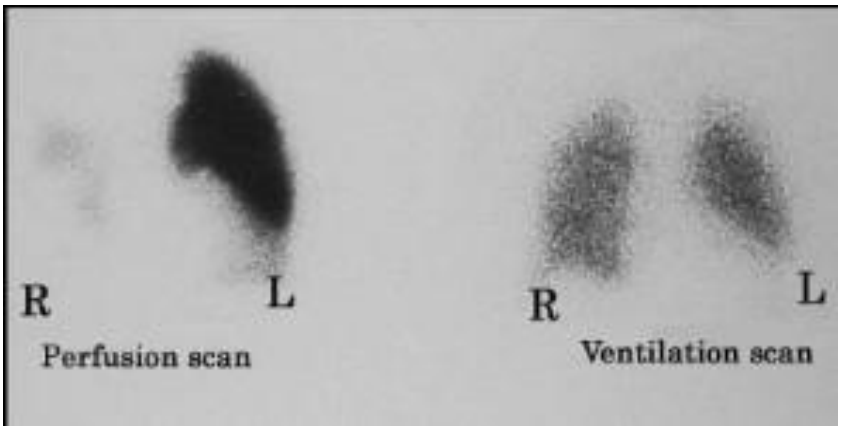


Figure 10: shows the absence of perfusion in the right lung with normal ventilation-high probability V/Q scan

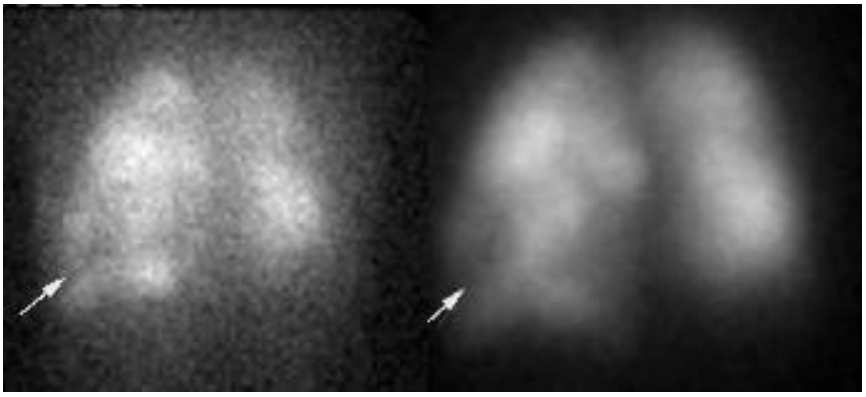


Figure11: shows matched defect in ventilation and perfusion in the right lower lobe-low probability V/Q scan

Echocardiography in PE Diagnosis

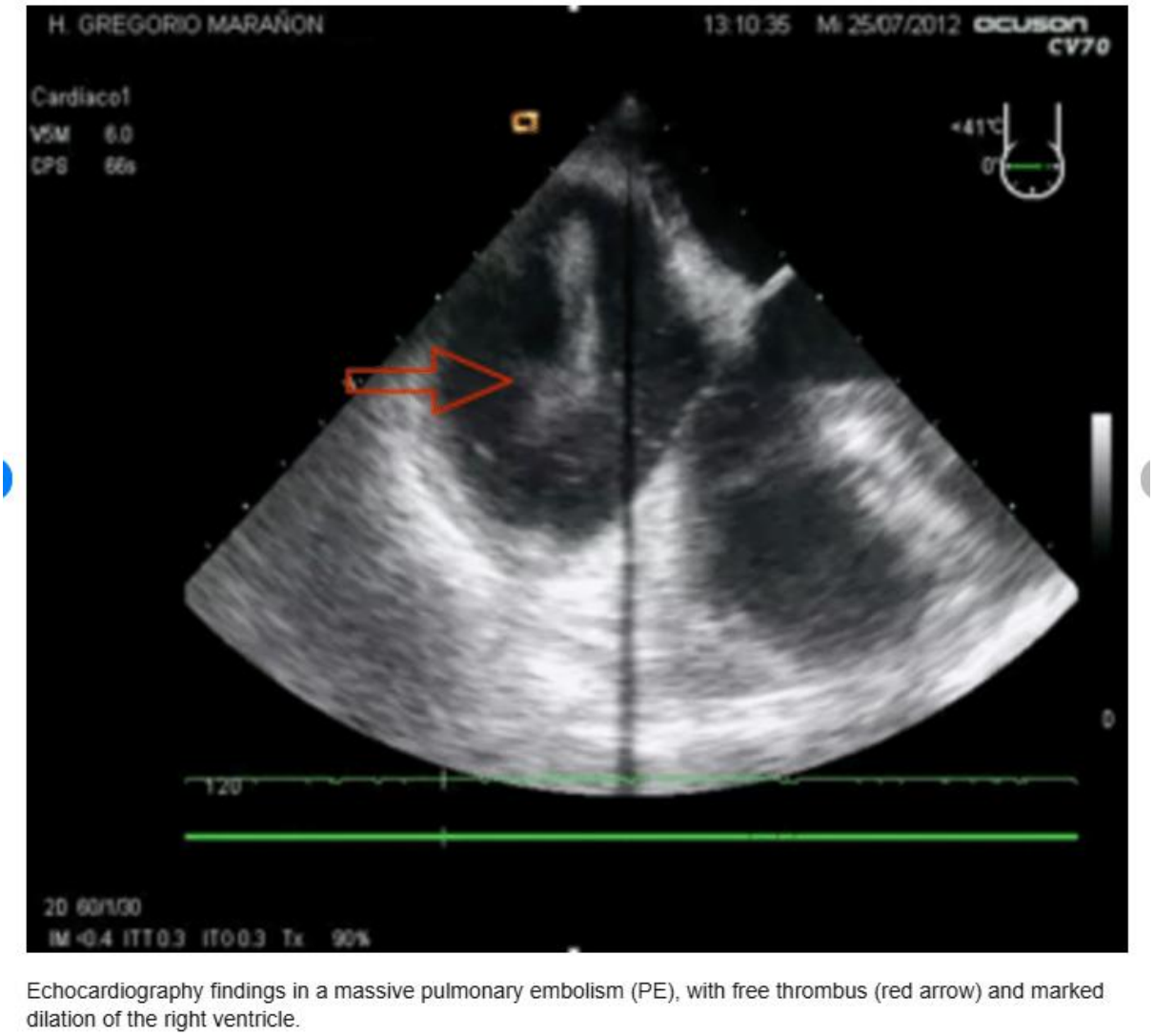
Echocardiography is **not a primary diagnostic tool** for PE but is highly valuable in **hemodynamically unstable patients** with suspected **massive PE**. It provides real-time evaluation of **right heart function** and can identify **signs of RV dysfunction** indicative of PE (31).

Echocardiographic Features of PE

1. **McConnell's Sign**: Regional RV dysfunction with **apical sparing**, specific for PE.

2. **Tricuspid Annular Plane Systolic Excursion (TAPSE) < 16 mm**: Suggests RV dysfunction.
3. **Increased Pulmonary Artery Pressure (>30 mmHg)**: Indicates pulmonary hypertension due to embolic obstruction.

Echocardiography is particularly useful for **triaging high-risk PE patients**, guiding **thrombolysis or mechanical circulatory support** in cases of **hemodynamic collapse (32)**.



Echocardiography findings in a massive pulmonary embolism (PE), with free thrombus (red arrow) and marked dilation of the right ventricle.

Figure 12: Echocardiography findings in a massive pulmonary embolism

Magnetic Resonance Angiography (MRA)

Magnetic resonance angiography (MRA) is an emerging non-invasive technique for PE diagnosis. **Contrast-enhanced MRA** allows for **high-resolution imaging of pulmonary vessels** without ionizing radiation. However, its use is limited due to **longer acquisition times, high cost, and motion artifacts** in critically ill patients (33).

Conventional Pulmonary Angiography

Pulmonary angiography was historically the **gold standard** for PE diagnosis but has largely been replaced by CTPA. It remains useful in cases requiring **catheter-directed thrombolysis or mechanical thrombectomy** (34).

Emerging Role of Artificial Intelligence (AI) in PE Imaging

AI and machine learning (ML) have **revolutionized PE diagnosis** by enhancing image analysis and **reducing interpretation errors**. AI algorithms can:

- **Automatically detect pulmonary emboli** on CTPA scans.
- **Assess RV dysfunction and predict patient outcomes**.
- **Optimize imaging protocols** to minimize radiation exposure.

The integration of AI into clinical workflows can **improve diagnostic efficiency, reduce radiologist workload, and enhance risk stratification** in PE patients (35).

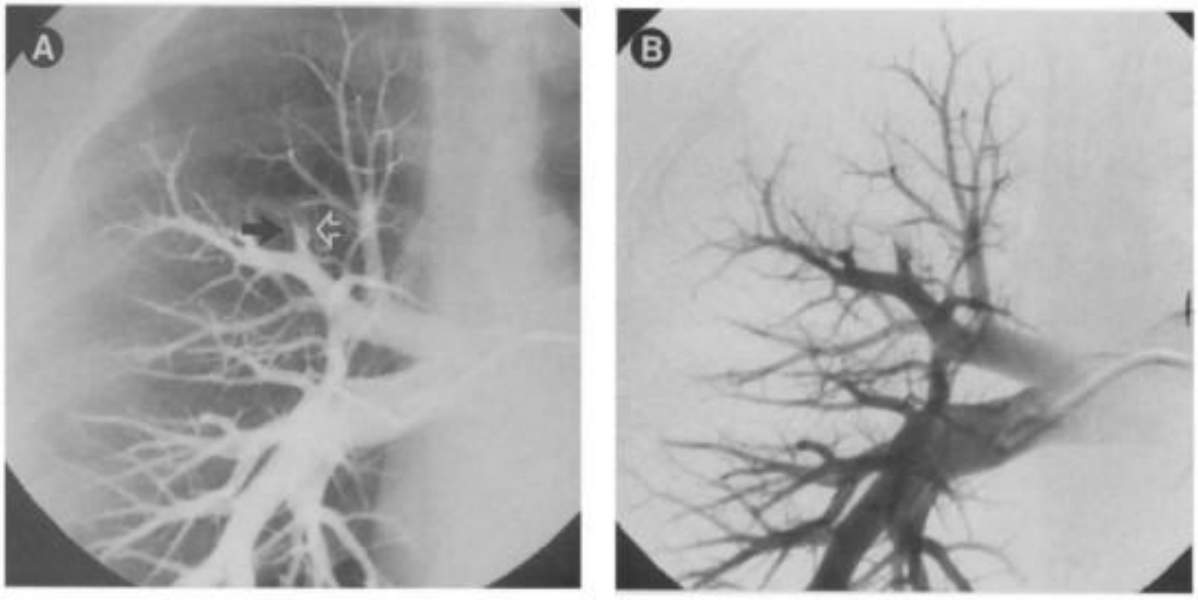


Figure 13: A - shows acute thrombus in the right upper lobe segmental artery. B - digital subtraction angiography (DSA) demonstrating the same.Angiographic catheter noted in the right intermediate artery

2.3 RIGHT VENTRICULAR DYSFUNCTION AS A PROGNOSTIC MARKER IN PULMONARY EMBOLISM

Pulmonary embolism (PE) is a life-threatening condition that can lead to acute right ventricular (RV) dysfunction due to increased pulmonary vascular resistance and pressure overload. Right ventricular dysfunction (RVD) is recognized as a critical prognostic factor in PE, as it reflects the hemodynamic burden imposed by the embolic obstruction of pulmonary arteries. The presence of RVD is associated with increased mortality, higher rates of complications, and a greater likelihood of requiring advanced therapeutic interventions. The assessment of RVD using clinical signs, imaging modalities, and biomarkers plays a crucial role in risk stratification and management decisions. This section explores the pathophysiology, diagnostic modalities, imaging-based assessment, and prognostic significance of RVD in PE (36).

Pathophysiology of Right Ventricular Dysfunction in PE

The right ventricle is adapted to handle low-pressure, high-compliance circulation. However, in acute PE, pulmonary artery obstruction results in an abrupt increase in pulmonary vascular resistance (PVR), leading to increased right ventricular afterload. As the right ventricle struggles to overcome this resistance, several pathophysiological changes occur:

1. RV Dilation and Increased RV:LV Ratio:
    - The sudden pressure overload causes RV dilation due to volume retention and wall stress.
    - This results in a shift of the interventricular septum toward the left ventricle (LV), leading to ventricular interdependence and decreased LV filling (37).
2. Decreased Left Ventricular Output and Systemic Hypotension:
    - As the interventricular septum bulges into the LV, it compromises diastolic filling, reducing stroke volume and cardiac output.
    - This contributes to systemic hypotension and shock in severe cases of PE.
3. Myocardial Ischemia and Right Heart Failure:
    - The combination of RV wall stress, hypoxia, and increased myocardial oxygen demand can lead to ischemia and RV failure.
    - If untreated, this may result in progressive hemodynamic collapse and multi-organ failure (38).

Right ventricular dysfunction is particularly significant in massive PE (which causes systemic hypotension) and submassive PE (which presents with RV strain but without hypotension). Identifying RVD early can help predict clinical deterioration and guide therapeutic decision-making (39).

Clinical Indicators of Right Ventricular Dysfunction in PE

Patients with PE may exhibit nonspecific clinical signs of RVD, but several findings can suggest right heart strain:

- Tachycardia (Heart Rate >100 bpm) – Increased RV workload due to decreased cardiac output.
- Jugular Venous Distension (JVD) – Elevated central venous pressure (CVP) due to impaired RV ejection.
- Hypotension (Systolic BP <90 mmHg) – A hallmark of massive PE with RV failure.
- Cyanosis and Peripheral Edema – Indications of systemic venous congestion and reduced cardiac output.
- A Loud Pulmonic Component of the Second Heart Sound (P2) – A sign of elevated pulmonary arterial pressure.
- Tricuspid Regurgitation Murmur – Suggests RV dilatation and annular dilatation leading to regurgitant blood flow (40).

While clinical assessment is useful, objective imaging and biomarker evaluation provide more reliable prognostic information.

Imaging-Based Assessment of Right Ventricular Dysfunction

Several imaging modalities are used to evaluate RVD in PE, each with specific advantages and limitations (41).

1. Computed Tomography Pulmonary Angiography (CTPA)

CTPA is the gold standard imaging modality for PE and also allows for quantitative assessment of RVD. Key CTPA-derived markers of RVD include:

- Right Ventricle to Left Ventricle (RV:LV) Ratio >1:
    - An RV:LV ratio >1 is associated with increased mortality and is a strong indicator of RV strain.
- Interventricular Septal Bowing:
    - Displacement of the septum toward the LV due to RV pressure overload.
- Reflux of Contrast into the Inferior Vena Cava (IVC):
    - Suggests elevated right atrial pressure and poor RV function.
- Pulmonary Artery to Aortic Ratio (PA/Ao) >1:

- A PA/Ao ratio greater than 1 correlates with severe PE and higher mortality (42).

CTPA-derived RV dysfunction markers are independent predictors of poor prognosis and can guide therapeutic escalation, such as thrombolysis or mechanical intervention.

2. Echocardiography (ECHO) in PE-Associated RV Dysfunction

Echocardiography is a crucial bedside tool for assessing RV function in hemodynamically unstable PE patients (43).

Key echocardiographic findings suggestive of RVD include:

- McConnell's Sign – Regional RV dysfunction with apical sparing, specific for acute PE.
- Tricuspid Annular Plane Systolic Excursion (TAPSE) <16 mm – Suggests impaired RV systolic function.
- RV Dilation and RV Hypokinesis – Indicate pressure overload and reduced contractility.
- Pulmonary Artery Pressure >30 mmHg – Suggests severe pulmonary hypertension.

Echocardiography is particularly useful in unstable patients where CT imaging is not feasible. Patients with RVD on echocardiography have a 3- to 5-fold increased risk of mortality (44).

3. Magnetic Resonance Imaging (MRI) and Nuclear Imaging

Cardiac MRI is emerging as a non-invasive method for RV function assessment, providing high-resolution imaging without radiation exposure. It allows for precise measurement of RV ejection fraction and strain analysis.

Nuclear imaging, such as single-photon emission computed tomography (SPECT), may offer functional insights into RV perfusion abnormalities in PE patients but is not widely used in clinical practice (45).

Biomarkers of Right Ventricular Dysfunction in PE

Serum biomarkers play a complementary role in diagnosing RVD and assessing prognostic risk.

1. Brain Natriuretic Peptide (BNP) and N-terminal proBNP (NT-proBNP)
    - Elevated in response to RV wall stress and pressure overload.
    - BNP >90 pg/mL or NT-proBNP>500 pg/mL suggests RV dysfunction.
2. Troponin I and Troponin T
    - Indicate myocardial injury due to RV ischemia.
    - Elevated troponins are associated with higher mortality in PE patients.
3. D-dimer
    - Sensitive but not specific for RVD.

Biomarkers alone are not sufficient for risk stratification but, when combined with imaging findings, provide powerful prognostic insight (46).

Prognostic Implications of RVD in PE

The presence of RVD significantly worsens outcomes in PE patients.

- Patients with RVD have a 30-day mortality rate of 15–20% compared to 1–5% in those without RVD.
- Submassive PE with RVD requires closer monitoring and potential escalation of care.
- Persistent RVD post-PE is linked to chronic thromboembolic pulmonary hypertension (CTEPH).

Early identification of RVD helps guide treatment strategies, including thrombolysis, catheter-based interventions, and surgical embolectomy in high-risk cases (47).

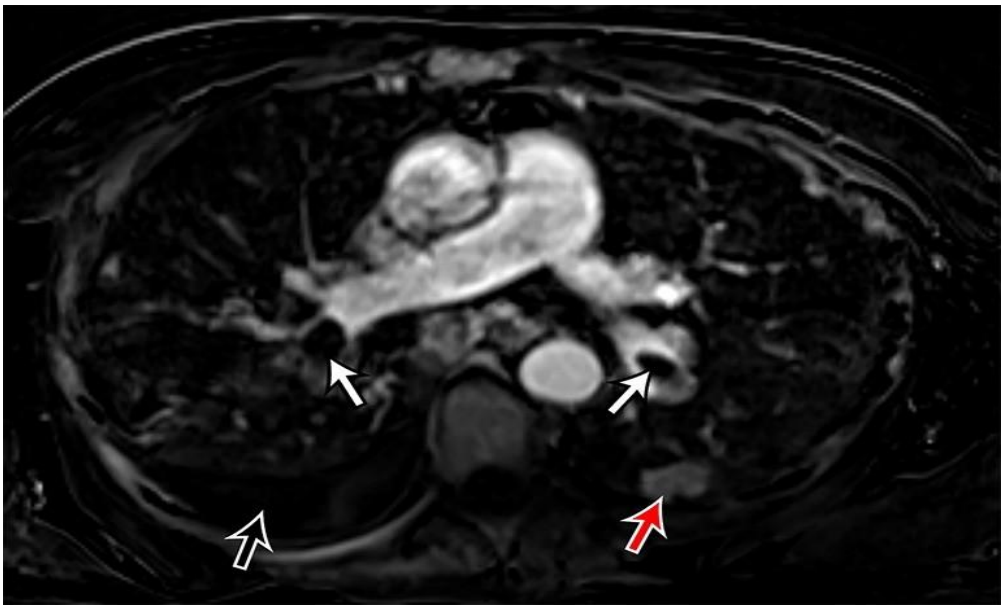


Figure 14: Axial T1 weighted post-contrast image with subtraction images shows hypointense filling defects in the right pulmonary artery and left posterior basal pulmonary artery (white arrows). Minimal right pleural effusion noted (black arrow). The posterior left lung shows an isointense nodule (red arrow) which was a metastasis from colorectal cancer

2.4 QUANTIFYING CLOT BURDEN: THE MODIFIED MILLER SCORE (MMS)

Pulmonary embolism (PE) is a potentially life-threatening condition caused by the obstruction of pulmonary arteries due to thromboembolic events. The extent and severity of clot burden significantly impact patient prognosis, hemodynamic stability, and clinical management. Over the years, several scoring systems have been developed to quantify the thrombus load in PE patients, with the Modified Miller Score (MMS) being one of the most widely utilized methods. The MMS provides a systematic and objective way to assess the severity of PE based on the degree of vascular obstruction and thrombus distribution in pulmonary arteries. This section discusses the development, methodology, clinical significance, limitations, and comparison of MMS with other scoring systems used in PE assessment (48).

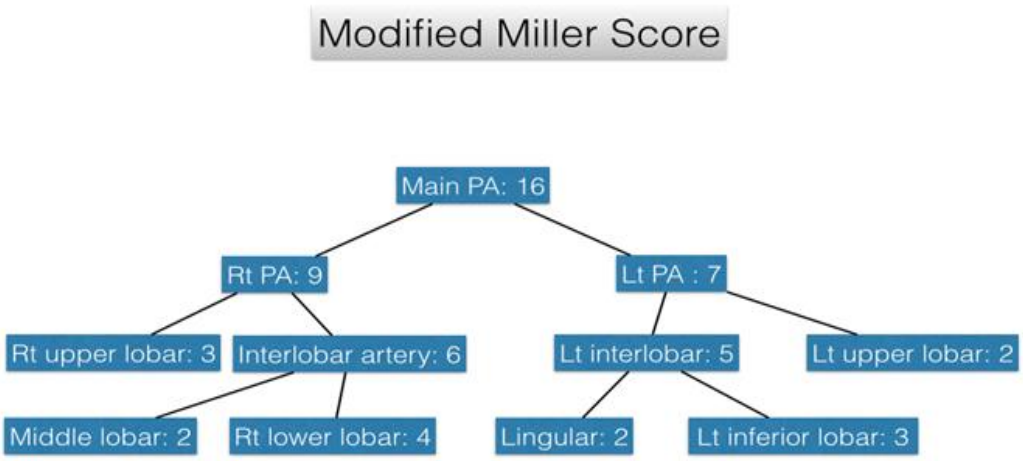


Figure 15: Modified Miller score

Development of the Modified Miller Score

The Miller Score was first introduced in 1967 for evaluating angiographic pulmonary thrombus burden. The original Miller Score aimed to estimate the percentage of vascular obstruction using conventional pulmonary angiography. However, with the advent of computed tomography pulmonary angiography (CTPA) as the gold standard for PE diagnosis, modifications to the Miller Score were necessary to improve applicability and reliability in modern imaging techniques (49).

The Modified Miller Score (MMS) was developed to adapt the Miller scoring system to CTPA findings, ensuring better reproducibility and interobserver agreement. It has since become an essential tool in clinical decision-making, risk stratification, and outcome prediction in PE patients (50).

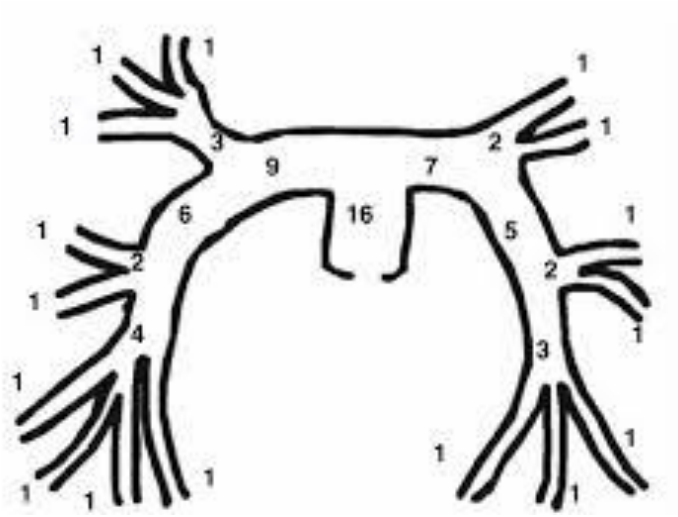


Figure 16: Modified Miller score (1). Pulmonary artery (PA), Right (Rt), Left (Lt). Numbers after location indicate score at that site. The site of the most proximal thrombus demonstrated gives a score (up to a maximum of 16) indicating the number of involved downstream major segmental arteries. Individual major segmental arteries gets scored 1.

Methodology of the Modified Miller Score

The Modified Miller Score quantifies thrombus burden based on the location and extent of vascular obstruction in the pulmonary arterial system. It is a semi-quantitative scoring system that assigns a numerical value based on the involvement of major pulmonary arteries and their segmental branches (51).

Scoring Criteria in MMS

1. The pulmonary arterial system is divided into 16 branches:
    - Main pulmonary artery
    - Right Pulmonary Artery (9 branches):
        - Right main pulmonary artery
        - Right upper lobar artery (3 segmental)
        - Right interlobar artery (middle and lower lobar)
        - Right middle lobe artery (2 segmental)
        - Right lower lobe artery (4 segmental)

- Left Pulmonary Artery (7 branches):
  - Left main pulmonary artery
  - Left upper lobe artery (2 segmental)
  - Left interlobar artery (Lingular and lower lobar)
  - Left lower lobar artery (3 segmental)
  - Left lingular artery (2 segmental)

2. Scoring Rules:
   - Each segmental artery completely or partially occluded by thrombus is assigned 1 point.
   - More proximal vessel involvement (e.g., main pulmonary artery) results in a score equivalent to the number of affected segmental branches downstream.
   - The maximum possible score is 16, indicating complete obstruction of all segmental branches or a saddle embolism (52).

A higher MMS is correlated with worse patient outcomes, increased risk of right ventricular dysfunction (RVD), and higher mortality rates (53).

Comparison of MMS with Other Clot Burden Scoring Systems

Several alternative clot burden quantification methods exist, each with advantages and limitations.

Scoring System	Imaging Modality	Max Score	Key Features	Limitations
Modified Miller Score (MMS)	CTPA	16	Segmental artery-based, correlates with RVD	Subjective, interobserver variability
Refined Miller Score (RMS)	CTPA	20	Includes additional sub segmental branches	Less widely used
Geneva Score	Pulmonary Angiography	18	Developedfor conventional angiography	Not adapted for CTPA
CT Pulmonary Embolism Severity Index (CT-PESI)	CTPA	N/A	Combines imaging with clinical parameters	Not purely based on clot burden

While Qanadli's Obstruction Index (QOI) offers a more detailed percentage-based assessment, it requires complex calculations. MMS, on the other hand, is simpler and faster, making it more practical in routine clinical practice (54).

Limitations of the Modified Miller Score

Despite its clinical utility, MMS has several limitations:

1. Interobserver Variability
    - Interpretation of partial vs. complete occlusion is subjective, leading to variations between radiologists.

2. Limited Subsegmental Evaluation
    - MMS does not account for thrombi in smaller subsegmental branches, which may underestimate clot burden in some patients.
3. Lack of Hemodynamic Considerations
    - MMS only quantifies clot volume but does not assess hemodynamic response, which is crucial for risk stratification.
4. Limited Role in Chronic Thromboembolic Pulmonary Hypertension (CTEPH)
    - The score is designed for acute PE and may not be applicable for chronic PE evaluation (55).

To overcome these limitations, clinicians should combine MMS with biomarkers (BNP, troponins), echocardiography (RV function), and clinical risk assessment (sPESI, shock index) for a comprehensive evaluation (56).

2.5 CORRELATION BETWEEN CLOT BURDEN AND RIGHT VENTRICULAR DYSFUNCTION

Pulmonary embolism (PE) is a **life-threatening condition** characterized by thrombotic obstruction of the pulmonary arteries, leading to **increased pulmonary vascular resistance, hypoxia, and right ventricular dysfunction (RVD)**. The **severity of RVD is closely linked to the extent of clot burden**, making the assessment of thrombus load a crucial factor in **risk stratification and treatment decisions**. Studies have shown that **larger clot burdens increase pulmonary artery pressure**, which can cause **right ventricular overload, dilation, and ultimately right heart failure**. Various imaging modalities, especially **computed tomography pulmonary angiography (CTPA)**, play a key role in **quantifying clot burden and assessing RVD**.

This section explores the **mechanisms by which clot burden affects right ventricular function, the role of different imaging techniques in quantifying both clot burden and RVD, clinical implications of their correlation, and prognostic considerations for PE patients (57)**.

Pathophysiology: How Clot Burden Affects Right Ventricular Function

The **right ventricle (RV) is physiologically adapted to handle a low-pressure, high-compliance pulmonary circulation**. However, in the presence of **acute PE**, pulmonary artery obstruction leads to:

1. **Increased Pulmonary Vascular Resistance (PVR):**
    - A larger clot burden causes **vascular occlusion**, increasing **PVR** and **right ventricular afterload**.
2. **Right Ventricular Dilation and Dysfunction:**
    - **Increased afterload** leads to **RV dilation** as it struggles to eject blood into the pulmonary circulation.
    - **RV:LV ratio increases** due to **pressure overload** in the RV, compressing the left ventricle and reducing systemic perfusion.
3. **Interventricular Septal Shift and Reduced Left Ventricular Output:**
    - As the **RV dilates**, the **interventricular septum bows into the LV**, impairing its **diastolic filling and cardiac output**.
    - This phenomenon is associated with **systemic hypotension and cardiogenic shock** in severe cases.
4. **Myocardial Ischemia and RV Failure:**
    - The combination of **RV wall stress, hypoxia, and increased myocardial oxygen demand** can result in **RV ischemia and dysfunction**, further exacerbating hemodynamic instability (58).

This **pathophysiological interplay between clot burden and RVD** explains why patients with **higher clot burdens** have **worse clinical outcomes and higher mortality rates**.

Imaging Modalities for Assessing Clot Burden and Right Ventricular Dysfunction

To understand the relationship between **thrombus load and RVD**, various imaging techniques are used to **quantify clot burden and assess RV function (59)**.

1. Computed Tomography Pulmonary Angiography (CTPA)

CTPA is the **gold standard for diagnosing PE** and provides **quantitative assessment of both clot burden and RV strain**.

Key CTPA-Derived Parameters for Clot Burden

- **Modified Miller Score (MMS):**
  - Scores **segmental and lobar pulmonary artery occlusion** with a maximum of **16 points**.
  - Higher MMS correlates with **higher RV dysfunction and worse outcomes**.
- **Qanadli Obstruction Index (QOI):**
  - Expresses clot burden as a **percentage of pulmonary artery obstruction**.
  - Patients with **QOI >40%** have a **higher likelihood of RVD (60)**.

Key CTPA-Derived Parameters for Right Ventricular Dysfunction

- **Right Ventricle to Left Ventricle (RV:LV) Ratio >1:**
  - A **strong indicator of RV dilation and strain**.
- **Interventricular Septal Bowing:**
  - **Shifting of the septum into the LV** suggests **RV pressure overload**.
- **Pulmonary Artery to Aortic (PA/Ao) Ratio >1:**
  - Reflects **increased pulmonary arterial pressure** and correlates with **severe PE**.

CTPA-based **clot burden quantification correlates well with RVD markers**, making it an essential tool for **risk stratification and treatment planning (61)**.

2. Echocardiography in Assessing Right Ventricular Dysfunction

Echocardiography is **highly valuable in unstable PE patients**, providing **real-time functional assessment of the RV (62)**.

Echocardiographic Markers of RVD in PE

- **McConnell's Sign:**
  - **Regional RV dysfunction with apical sparing**—a hallmark of acute PE.
- **Tricuspid Annular Plane Systolic Excursion (TAPSE) <16 mm:**
  - Suggests **reduced RV systolic function**.
- **Pulmonary Artery Pressure >30 mmHg:**
  - Indicates **pulmonary hypertension and RV overload**.

Echocardiography is particularly useful in patients who are **hemodynamically unstable and cannot undergo CTPA (63)**.

Clinical Implications: How Clot Burden Affects PE Severity and Management

The correlation between **thrombus load and RVD** has several **critical clinical implications**:

1. Risk Stratification in PE Patients

- Patients with **higher clot burden scores (e.g., MMS >12 or QOI >40%)** are more likely to develop **RVD and hemodynamic instability**.
- **Submassive PE** is defined by **RV dysfunction without systemic hypotension**, requiring **careful monitoring and escalation of therapy**.
- **Massive PE**, characterized by **RV failure and systemic shock**, requires **urgent thrombolysis or surgical embolectomy** (64).

2. Selection of Therapeutic Strategy

- **Low-Risk PE (Minimal Clot Burden, No RVD):**
  - Managed with **anticoagulation alone**.
- **Intermediate-Risk PE (Moderate Clot Burden, RVD Without Hypotension):**
  - Consider **systemic or catheter-directed thrombolysis**.
- **High-Risk PE (Large Clot Burden, Severe RVD, Hypotension):**
  - Requires **thrombolysis, mechanical thrombectomy, or ECMO support** (65).

Prognostic Value of Clot Burden and RVD Correlation

The presence of **both high clot burden and RVD significantly increases mortality risk**.

- Patients with **MMS >12** have **a 30-day mortality rate of 15–20%** compared to 1–5% in those without RVD.
- **Persistent RVD post-PE** is linked to **chronic thromboembolic pulmonary hypertension (CTEPH)**.
- The **combination of elevated troponins, BNP, and imaging-derived RVD markers** further refines **risk prediction** (66).

2.6 ADDITIONAL CTPA-BASED PROGNOSTIC MARKERS IN PULMONARY EMBOLISM

Pulmonary embolism (PE) is a life-threatening condition that demands accurate risk stratification to guide management and improve patient outcomes. Computed Tomography Pulmonary Angiography (CTPA) is the gold standard imaging modality for diagnosing PE, allowing direct visualization of emboli and providing critical prognostic information. While clot burden quantification and right ventricular dysfunction (RVD) are primary prognostic indicators, additional CTPA-based markers offer valuable insights into pulmonary hypertension, hemodynamic compromise, and mortality risk. These markers play a crucial role in identifying high-risk PE patients who may benefit from aggressive therapeutic interventions such as thrombolysis or catheter-directed therapies.

This section explores the additional prognostic markers derived from CTPA, including the pulmonary artery to aortic (PA/Ao) ratio, interventricular septal bowing, inferior vena cava (IVC) reflux, pulmonary artery diameter, and lung parenchymal abnormalities, and their significance in risk stratification and patient management (67).

Pulmonary Artery to Aortic (PA/Ao) Ratio

The PA/Ao ratio is a widely used CTPA-derived marker to assess pulmonary hypertension and RV strain in PE patients.

1. **Definition and Measurement:**
    - The diameters of the main pulmonary artery (PA) and ascending aorta (Ao) are measured at the level of the pulmonary artery bifurcation on axial CTPA images.
    - A PA/Ao ratio >1 suggests pulmonary arterial dilation and elevated pulmonary pressures (68).
2. **Prognostic Significance:**
    - PA/Ao ratio >1.0 correlated with higher in-hospital mortality and worse RV function.
    - A ratio >1.2 indicates severe pulmonary hypertension and may predict long-term complications such as chronic thromboembolic pulmonary hypertension (CTEPH) (69).
3. **Clinical Implications:**
    - Patients with PA/Ao>1 require closer monitoring for hemodynamic deterioration.
    - A significant increase in PA diameter suggests RV pressure overload and may prompt early escalation of therapy (70).

Interventricular Septal Bowing and Left Ventricular Compression

Interventricular septal bowing occurs when the pressure in the right ventricle exceeds that in the left ventricle, causing leftward displacement of the septum.

1. **Mechanism and Pathophysiology:**
    - Acute PE leads to RV pressure overload, reducing left ventricular preload and systemic cardiac output.
    - Septal bowing is a hallmark of hemodynamically significant PE and is frequently seen in patients requiring thrombolysis or ICU admission.
2. **CTPA Detection and Grading:**
    - Mild septal bowing: Slight deviation without LV compression.
    - Moderate septal bowing: Marked deviation with partial LV compression.

- Severe septal bowing: Complete LV compression, highly suggestive of impending circulatory failure.

3. **Prognostic Value:**
    - Patients with significant septal bowing have a 3-fold increased risk of hemodynamic deterioration.
    - Studies have shown that septal bowing on CTPA predicts ICU admission and 30-day mortality with high specificity.

4. **Clinical Implications:**
    - The presence of severe septal bowing may prompt early initiation of thrombolysis or mechanical circulatory support.
    - Serial CTPA scans can be used to monitor septal bowing resolution post-treatment, indicating RV recovery (71).

Contrast Reflux into the Inferior Vena Cava (IVC) and Hepatic Veins

Contrast reflux into the IVC and hepatic veins on CTPA is a strong indicator of right heart dysfunction in PE patients.

1. **Mechanism and Pathophysiology:**
    - Acute right ventricular failure leads to increased central venous pressure, causing contrast to flow retrogradely into the IVC.
    - This phenomenon is a sign of right atrial overload and poor RV ejection.
2. **CTPA Detection and Grading:**
    - Grade 1: Contrast reflux into the intrahepatic portion of the IVC.
    - Grade 2: Reflux extends into the hepatic veins.
    - Grade 3: Contrast reaches the distal IVC and renal veins, suggesting severe RV dysfunction (72).

3. **Prognostic Value:**
    - Patients with Grade 2 or 3 IVC reflux have a significantly higher risk of cardiac arrest.
    - In a study ,IVC reflux was associated with increased 30-day mortality and prolonged ICU stay.
4. **Clinical Implications:**
    - Presence of contrast reflux should alert clinicians to severe RV dysfunction, even in patients without overt hypotension.
    - Patients with high-grade reflux should be closely monitored for hemodynamic instability (73).

Pulmonary Artery Diameter and Pulmonary Hypertension

The main pulmonary artery (MPA) diameter provides indirect evidence of pulmonary hypertension and chronic thromboembolic disease.

1. **Measurement on CTPA:**
    - The MPA diameter is measured at the level of the bifurcation.
    - A diameter >29 mm in men and >27 mm in women is indicative of elevated pulmonary arterial pressures (74).
2. **Prognostic Value:**
    - MPA dilation >30 mm is linked to increased mortality and higher risk of CTEPH (14).
    - Patients with MPA diameter >32 mm are at risk of persistent pulmonary hypertension even after PE resolution.
3. **Clinical Implications:**
    - Patients with significant MPA dilation should undergo further evaluation with echocardiography.
    - Persistent pulmonary hypertension post-PE requires long-term follow-up and possible pulmonary endarterectomy (75).

Lung Parenchymal Abnormalities and Infarction

PE can cause secondary lung changes, which provide additional prognostic information.

1. **Common CTPA Findings:**
    - Pulmonary infarction (Wedge-Shaped Opacities): Indicates distal embolization and infarction.
    - Mosaic Attenuation: Suggests chronic embolic disease or pulmonary hypertension.
    - Pleural Effusion: Associated with increased in-hospital morbidity (76).
2. **Prognostic Value:**
    - Pulmonary infarction is linked to prolonged hospitalization and higher rates of persistent dyspnea.
    - Pleural effusions >10 mm predict poor respiratory recovery.
3. **Clinical Implications:**
    - Patients with extensive lung infarctions require oxygen therapy and prolonged anticoagulation.
    - Mosaic attenuation patterns may indicate underlying chronic embolic disease, necessitating further imaging (77).

2.7 PAST STUDIES

He et al. (2006) Studied: Pulmonary embolism (PE) can lead to right heart dysfunction, but the utility of computed tomography (CT) in evaluating cardiac involvement has been debated. Echocardiography is often used, but CT may provide a more accessible alternative. **Aim:** This study aimed to assess whether qualitative evaluation of right heart dysfunction via CT can provide clinically relevant insights comparable to echocardiography. **Method:** Seventy-four patients diagnosed with PE on multidetector CT (MDCT) between 2002 and 2004 were analyzed. Right ventricular (RV) enlargement and interventricular septal bowing were assessed qualitatively. Pulmonary vascular obstruction was graded using a clot burden scoring system. Echocardiography findings (when available) were compared with CT results to determine their correlation with right heart dysfunction. **Result Analysis:** The study found that 66% of patients exhibited signs of right heart dysfunction on CT, including RV dilation (51%) and septal bowing (59%). Pulmonary vascular obstruction scores were significantly higher in those with RV dysfunction. CT demonstrated an 81% sensitivity in identifying right heart dysfunction, higher than echocardiography's 56%. Additionally, patients with greater clot burden had significantly higher RV dilation and septal bowing, indicating CT's ability to assess PE severity. **Conclusion:** The study concluded that CT is an effective tool for assessing right heart dysfunction in acute PE patients. It demonstrated superior sensitivity to echocardiography and strongly correlated with clot burden scores, making it a valuable diagnostic option for evaluating PE severity (78).

Seon et al. (2011) Studied: Pulmonary thromboembolism (PTE) is a major cause of morbidity and mortality. While computed tomography pulmonary angiography (CTPA) is widely used for diagnosis, its role in risk stratification and correlation with cardiac biomarkers remains unclear. **Aim:** This study aimed to assess the efficacy of CTPA variables in risk stratification of acute PTE and compare them with cardiac biomarkers for predicting right ventricular dysfunction (RVD). **Method:** Eighty patients with confirmed acute PTE were divided into two groups: those with RVD (n=49) and those without (n=31). CTPA parameters such as the right-to-left ventricular diameter ratio (RVD/LVD), pulmonary artery (PA) clot burden, interventricular septal bowing, and contrast reflux into the inferior vena cava (IVC) were analyzed. These variables were compared with cardiac biomarkers such as troponin and brain natriuretic peptide (BNP).

Result Analysis: RVD/LVD and PA clot burden were significantly different between the groups ($p<0.001$). The optimal cutoff for predicting RVD was 1.12 for RVD/LVD (sensitivity: 89.8%, specificity: 77.4%) and 19.5 for PA clot load (sensitivity: 81.6%, specificity: 77.4%). CTPA variables strongly correlated with cardiac biomarkers, indicating their predictive value for severe PTE. **Conclusion:** The study concluded that CTPA is a reliable tool not only for diagnosing PTE but also for risk stratification. Key CTPA variables, such as RVD/LVD ratio and PA clot load, correlate well with cardiac biomarkers, making CTPA an effective method for assessing PE severity and prognosis (79).

Engelhardt et al. (2011) Studied: Pulmonary embolism can cause significant right ventricular dysfunction. While systemic thrombolysis improves outcomes, it carries a high bleeding risk. Catheter-directed ultrasound-accelerated thrombolysis (USAT) offers a potentially safer alternative. **Aim:** This study aimed to assess the impact of low-dose USAT on clot burden reduction and right ventricular function improvement in intermediate- and high-risk PE patients. **Method:** A retrospective analysis of 24 patients treated with USAT was conducted. Patients received an average of 33.5 mg of rt-PA over 19.7 hours. CT scans were analyzed at baseline and after treatment to assess changes in the right-to-left ventricular dimension ratio and modified Miller clot burden score. **Result Analysis:** The right-to-left ventricular ratio significantly decreased from 1.33 ± 0.24 at baseline to 1.00 ± 0.13 post-treatment ($p<0.001$). The modified Miller score showed a significant reduction from 17.8 ± 5.3 to 8.7 ± 5.1 ($p<0.001$). No systemic bleeding complications occurred, though four major access site bleeding incidents were reported. **Conclusion:** The study concluded that USAT is effective in rapidly reducing clot burden and improving RV function in PE patients. The lower bleeding risk compared to systemic thrombolysis suggests it is a safer alternative for treating high-risk PE (80).

Furlan et al. (2012) Studied: Acute PE can lead to right heart dysfunction, increasing mortality risk. Assessing clot burden and right heart strain using CT pulmonary angiography (CTPA) may help predict short-term mortality. **Aim:** The study aimed to investigate the correlation between clot burden, right heart dysfunction signs on CTPA, and short-term mortality in PE patients. **Method:** A retrospective analysis was conducted on 635 patients diagnosed with acute PE via CTPA in 2007. Two independent readers evaluated right ventricular diameter, the right-to-left ventricular ratio (RV/LV), and clot burden using semiquantitative scoring methods (Mastora and Qanadli scores). Patients were followed for 30 days to assess mortality. **Result Analysis:** Clot volume correlated strongly with semiquantitative clot burden scores ($r=0.86$, $p<0.01$). Patients with an RV/LV ratio >1.0 had significantly higher 30-day mortality ($p<0.001$). However, clot burden scores alone were not independent predictors of mortality when adjusted for RV dilation. **Conclusion:** The study concluded that increased RV/LV ratio is a strong predictor of short-term mortality in acute PE, whereas clot burden alone does not independently predict outcomes. This highlights the need for comprehensive CT-based risk stratification (81).

Rodrigues et al. (2012) Studied: Clot burden scoring systems help assess PE severity, but their direct correlation with right ventricular dysfunction (RVD) and clinical outcomes remains uncertain. **Aim:** This study aimed to evaluate the correlation between the Qanadli clot burden score (QS) and parameters of RVD in intermediate/high-risk PE patients. **Method:** A retrospective analysis of 107 PE patients was conducted. CT angiography images were reviewed, and clot burden scores were calculated. Patients were grouped based on QS scores (A: <18, B: ≥18). RVD was assessed using echocardiography and clinical markers, including ECG abnormalities and laboratory biomarkers. **Result Analysis:** Patients with QS ≥18 had significantly higher Geneva and Wells scores ($p=0.017$), higher heart rates, and greater prevalence of T-wave inversions and right bundle branch block ($p=0.034$). RV/LV ratios and pulmonary artery systolic pressures were significantly elevated in group B ($p=0.002$). QS >18 independently predicted RVD with an odds ratio of 10.85 ($p<0.001$). **Conclusion:** QS >18 strongly predicts RVD and correlates with other markers of PE severity. The study suggests that clot burden scores can aid in assessing disease severity and guiding treatment (82).

Rodrigues et al. (2015) Studied: Right ventricular dysfunction is a key prognostic factor in PE, but the relationship between clot burden and RV performance is debated. **Aim:** This study aimed to determine the effect of clot burden on RV function using echocardiography and CT in acute PE patients. **Method:** Eighty-five patients diagnosed with PE via multislice CT underwent echocardiography within 24 hours. Clot burden was scored from 1 to 20 based on pulmonary artery involvement. RV function was evaluated using fractional area change (FAC), Doppler-derived myocardial performance index (MPI), and pulmonary artery pressure (PAP). **Result Analysis:** Mean clot burden score was 9.4 ± 6.7. RV dysfunction was present in 37% of cases, with FAC significantly lower in these patients (27.8 ± 7.2% vs. 47.8 ± 4.4%, $p<0.05$). Higher clot burden correlated with increased pulmonary artery pressure ($r=0.51$, $p<0.001$) and worsened RV function. **Conclusion:** Clot burden has a significant impact on RV function, with higher scores correlating with increased PAP and reduced FAC. The study suggests clot burden scoring is useful in assessing PE severity and right heart strain (83).

Ouriel et al. (2017) Studied: Pulmonary embolism (PE) severity can be estimated using computed tomography angiography (CTA), but its reliability in assessing clot burden and right ventricular dysfunction remains uncertain. **Aim:** This study aimed to evaluate the reliability of CTA for quantifying PE severity by comparing different scoring methods for clot burden and right-to-left ventricular ratios. **Method:** A prospective study of 10 patients randomly selected from a 150-patient trial on ultrasound-facilitated fibrinolysis for PE. Four independent reviewers evaluated clot burden using the modified Miller and refined Miller scores. Right ventricular function was assessed using both multiplanar reformatted and axial CT views. **Result Analysis:** The intra-observer reliability for assessing the right-to-left ventricular ratio was excellent (intraclass correlation coefficient: 0.97). Both clot burden scoring methods showed strong agreement (ICC: 0.82-0.88). Inter-observer agreement for clot burden and ventricular ratio was similarly high, demonstrating the robustness of CTA for assessing PE severity. **Conclusion:** The study concluded that CTA is a highly reliable tool for quantifying clot burden and right ventricular dysfunction in PE. The modified Miller and refined Miller scores both proved effective, making CTA a valuable method for PE severity assessment (84).

Tapson et al. (2018) Studied: Catheter-directed thrombolysis is increasingly used to treat intermediate-risk PE, but the optimal duration and dosage of tissue plasminogen activator (tPA) remain unknown. **Aim:** This study aimed to determine the lowest effective tPA dose and duration for improving right ventricular function and reducing clot burden. **Method:** A randomized trial involving 101 hemodynamically stable adults with intermediate-risk PE. Patients received one of four tPA regimens (4–12 mg over 2–6 hours) using ultrasound-facilitated catheter-directed thrombolysis. Clot burden and right ventricular dysfunction were assessed via CTA and echocardiography. **Result Analysis:** All four regimens significantly improved the right-to-left ventricular ratio (mean reductions: 0.35–0.48, $p<0.0001$) and reduced clot burden using the modified Miller score. Major bleeding events occurred in 4% of patients, with one intracranial hemorrhage attributed to tPA. **Conclusion:** Shorter, lower-dose tPA regimens effectively improve right ventricular function and reduce clot burden while minimizing bleeding risk. The study supports the use of ultrasound-facilitated thrombolysis as a safe and effective alternative for PE treatment (85).

Abdelwahab et al. (2020) Studied: Pulmonary computed tomography angiography (CTA) provides detailed clot burden assessment, but its correlation with right ventricular dysfunction parameters remains unclear. **Aim:** This study aimed to investigate the relationship between clot volume and right ventricular dysfunction using pulmonary CTA and echocardiography in PE patients. **Method:** A cross-sectional study of patients with acute PE conducted between June 2017 and June 2018. Pulmonary CTA was used to measure clot volume, while echocardiography assessed right ventricular dysfunction. **Result Analysis:** A significant correlation was found between clot volume and right ventricular diameter ($p<0.001$), RV/LV ratio ($p=0.01$), pulmonary artery diameter ($p=0.01$), and superior vena cava diameter ($p=0.01$). However, no significant correlation was observed between clot volume and echocardiographic parameters of RV dysfunction. **Conclusion:** The study concluded that pulmonary CTA is a reliable tool for assessing right ventricular dysfunction in PE patients. Clot burden strongly correlates with CTA-derived right heart strain markers, but echocardiographic findings do not consistently reflect clot volume severity (86).

Çalışkan et al. (2021) Studied: Cancer patients with PE often exhibit different clinical presentations, but whether they have higher clot burdens and right ventricular dysfunction compared to non-cancer patients remains unclear. **Aim:** This study aimed to compare clot burden, right ventricular dysfunction, and biomarker levels in cancer versus non-cancer patients with PE. **Method:** A retrospective study of 71 PE patients divided into cancer (n=35) and non-cancer (n=36) groups. Clot burden was measured using the modified Miller score, while RV dysfunction was assessed via echocardiography. D-dimer and cardiac troponin I (cTnI) levels were also evaluated. **Result Analysis:** No significant differences were found between groups in terms of clot burden (p=0.34), PE location (p=0.67), or RV dysfunction (p=0.28). However, cancer patients exhibited significantly higher cTnI levels (p=0.03), indicating a greater cardiac biomarker response despite similar clot burden. **Conclusion:** The study concluded that while clot burden and RV dysfunction are comparable between cancer and non-cancer patients, cancer patients exhibit a stronger cardiac biomarker response. This suggests a different physiological response to PE in oncologic populations (87).

Xi et al. (2023) Studied: Risk stratification in PE patients is crucial for treatment decisions. A deep-learning (DL) algorithm-based clot burden score, called the "clot ratio," was developed to improve accuracy. **Aim:** This study aimed to validate the clot ratio as a novel imaging marker correlating with PE risk stratification and acute right ventricular dysfunction. **Method:** Seventy newly diagnosed PE patients were enrolled. Clot burden was assessed using four methods: Qanadli score, Mastora score, clot volume, and clot ratio (DL-based). Right ventricular dysfunction and risk stratification were analyzed. **Result Analysis:** Among the four clot burden scores, clot ratio had the highest predictive value for high-risk PE (AUC: 0.719, $p<0.05$). In hemodynamically stable patients, only clot ratio showed a significant difference (p=0.046), demonstrating its predictive accuracy. **Conclusion:** Clot ratio is a superior imaging marker for PE risk stratification. The study suggests integrating deep-learning-based clot burden scoring into clinical practice for improved PE assessment (88).

Tuzovic et al. (2016) Studied: Regional right ventricular dysfunction (RRVD) has been observed in PE, but its relationship with clot burden and biomarker profiles remains unclear. **Aim:** This study aimed to investigate the clinical significance of RRVD by assessing its correlation with clot burden and biomarker levels. **Method:** A retrospective study of 82 patients diagnosed with acute PE using computed tomography angiography and echocardiography. Clot burden was assessed using the modified Miller score. Biomarkers (troponin, NT-proBNP) were also analyzed. **Result Analysis:** RRVD was present in 41% of PE patients, with 86% of these cases involving central or multi-lobar embolism. Patients with RRVD had significantly higher RV dilation ($p<0.01$) and dysfunction ($p<0.01$). A trend toward elevated troponin levels was observed in RRVD cases (38% vs. 13%, $p=0.08$), while NT-proBNP levels were similar across groups. **Conclusion:** The study concluded that RRVD is strongly associated with increased clot burden and more severe RV dysfunction. It emphasizes the importance of identifying RRVD for risk stratification in PE patients (89).

MATERIALS AND METHODS

1. Study Design

The study was conducted as a hospital-based cross-sectional study to assess the correlation between clot burden and right ventricular dysfunction (RVD) in acute pulmonary thromboembolism (PTE) using computed tomography pulmonary angiography (CTPA). A cross-sectional approach was chosen as it allowed for the simultaneous evaluation of clot burden and its impact on right ventricular function at a single point in time. This study design was appropriate because it enabled objective quantification of thrombus load using the Modified Miller Score (MMS) and assessment of RVD through CTPA-derived parameters. The study did not involve any interventions or follow-ups, focusing solely on diagnostic imaging and its prognostic implications.

2. Study Setting

This study was conducted at KLE's Prabhakar Kore Hospital and Medical Research Centre, a tertiary care teaching hospital attached to JN Medical College, Belagavi, India. The hospital has a well-equipped radiodiagnosis department with advanced imaging facilities, including a General electronics 128-slice multi-detector computed tomography (MDCT) scanner. The department provided access to high-quality CTPA imaging, which was essential for evaluating the clot burden and right ventricular function in PE patients.

The study population included patients referred to the Department of Radiodiagnosis for CTPA with a clinical suspicion of acute pulmonary thromboembolism. The hospital served as an ideal setting due to its high patient volume, availability of expert radiologists, and access to electronic medical records for data retrieval.

3. Study Duration

The study was conducted over a one-year period from May 2024 to January 2025. This duration was chosen to allow for an adequate sample size to be collected, ensuring statistical reliability and clinical significance.

- Initial three months were allocated for protocol approval, ethical clearance, and standardization of the data collection process.
- Subsequent nine months were utilized for patient recruitment, imaging analysis, and data collection.
- Final three months were dedicated to data analysis, interpretation of results, and manuscript preparation.

4. Participants – Inclusion and Exclusion Criteria

Inclusion Criteria

Patients were included in the study based on the following criteria:

1. Age ≥ 18 years undergoing CT pulmonary angiography for suspected acute pulmonary thromboembolism (PTE).
2. Patients with confirmed PE on CTPA, with visible thrombus in the pulmonary arterial system.
3. Patients who had no prior history of chronic thromboembolic disease or known cardiac dysfunction before the acute PE event.
4. Patients whose CTPA images met the required quality standards for clear visualization of pulmonary arteries and right ventricular parameters.

Exclusion Criteria

Patients were excluded from the study under the following conditions:

1. Pregnant women due to concerns regarding radiation exposure.

2. Patients with known pre-existing right ventricular dysfunction unrelated to acute PTE, including those with chronic heart failure, congenital heart disease, or severe pulmonary hypertension.
3. Patients with technically inadequate CTPA scans due to poor image quality, motion artifacts, or incomplete imaging.
4. Patients clinically suspected of PTE but with a negative CTPA scan.
5. Patients who refused consent or were unable to provide consent due to critical illness.

5. Study Sampling

A universal sampling technique was employed in which all eligible patients undergoing CTPA for suspected acute PTE within the study period were included. This non-randomized, consecutive sampling method ensured that all patients meeting the inclusion criteria were systematically evaluated, minimizing selection bias.

6. Study Sample Size

The sample size was determined based on the correlation between Modified Miller Score (MMS) and Right Ventricular Dysfunction (RVD) in previous studies.

- A previous study reported that the correlation coefficient (r) between increasing MMS and RV:LV ratio was 0.39.
- Using this correlation value, at a 5% level of significance ($\alpha = 0.05$) and 80% power ($\beta = 0.20$), the minimum required sample size was calculated to be 49 patients.

7. Study Groups (if applicable)

Since this was a cross-sectional study, patients were not divided into treatment groups. However, based on clot burden and RV dysfunction severity, patients were categorized into:

1. Low Clot Burden Group (MMS < 8)
2. Moderate Clot Burden Group (MMS 8-12)
3. High Clot Burden Group (MMS > 12)

8. Study Parameters

The following parameters were evaluated in the study:

Clot Burden Assessment:

- Modified Miller Score (MMS) for quantifying thrombus load.

Right Ventricular Dysfunction Markers:

- RV:LV ratio (>1 indicates RVD).
- Interventricular septal bowing (leftward deviation indicates RVD).
- Pulmonary artery to aortic (PA/Ao) ratio (>1 suggests pulmonary hypertension).

9. Study Procedure

1. All patients with suspected PE underwent CTPA using a General Electronics 128-slice MDCT scanner.
2. Imaging parameters were standardized:
    - Contrast injection protocol: 80–100 mL of iodinated contrast agent.
    - Scan timing: Pulmonary arterial phase (12–15 seconds post-injection).
3. Clot burden was scored using the MMS, and right ventricular parameters were measured.
4. Findings were documented and correlated with clinical severity scores.

10. Study Data Collection

Data collection was performed using a structured case record form (CRF), which included:

- Demographic details (age, sex, comorbidities).
- Clinical presentation (dyspnea, chest pain, syncope).
- CTPA findings (MMS, RV:LV ratio, PA/Ao ratio, septal bowing).

- All data were de-identified and stored securely to maintain patient confidentiality.

11. Data Analysis

- Categorical variables (e.g., presence of RV dysfunction) were analyzed using Chi-square or Fisher's exact test.
- Continuous variables (e.g., MMS, RV:LV ratio) were summarized using mean ± SD or median (IQR) and compared using t-tests or Mann-Whitney U tests.
- Pearson's/Spearman's correlation analysis was used to assess the relationship between clot burden and RVD.
- A p-value < 0.05 was considered statistically significant.
- Data were analyzed using R statistical software (version 4.2.2) and Microsoft Excel.

12. Ethical Considerations

The study adhered to ethical principles outlined by the Indian Council of Medical Research (ICMR) and was approved by the Institutional Ethics Committee (IEC) of JNMC, Belagavi.

- Written informed consent was obtained from all participants.
- Confidentiality of patient data was maintained by anonymizing all records.
- No additional financial burden was placed on patients as all imaging was clinically indicated.
- The study complied with Good Clinical Practice (GCP) guidelines.

ANALYSIS AND RESULTS

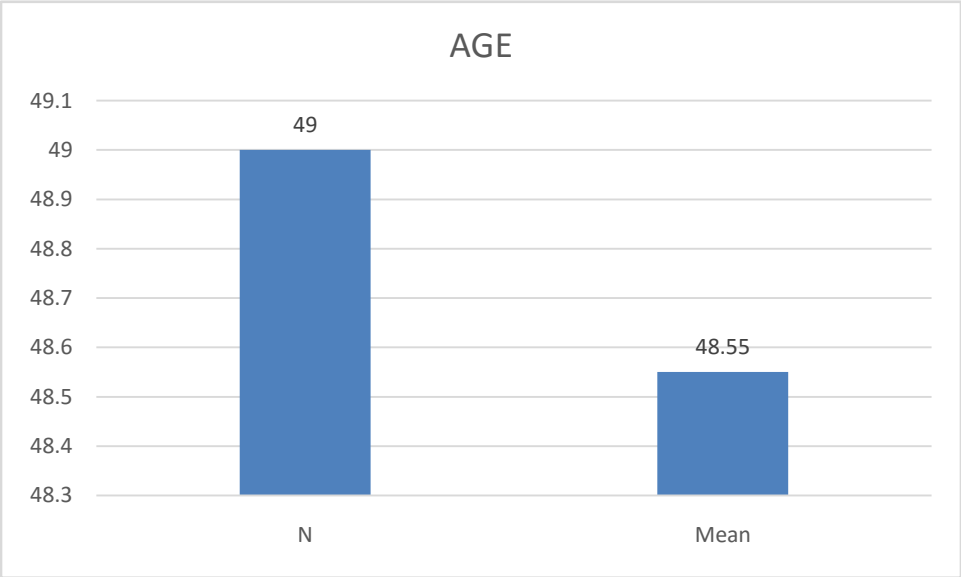
1. Demographic Characteristics of the Study Population

Interpretation

The study included 49 patients with a mean age of 48.55 ± 16.21 years (range: 25–84 years) (Table 1). This wide age distribution reflects the diverse demographic profile of patients with acute pulmonary thromboembolism and ensures that findings related to clot burden and right ventricular dysfunction are applicable across different age groups.

Table 1: Demographic Characteristics of the Study Population

Variable	N	Mean	StdDev	Minimum	Maximum
AGE	49	48.55	16.21	25	84



Graph 1: Demographic Characteristics of the Study Population

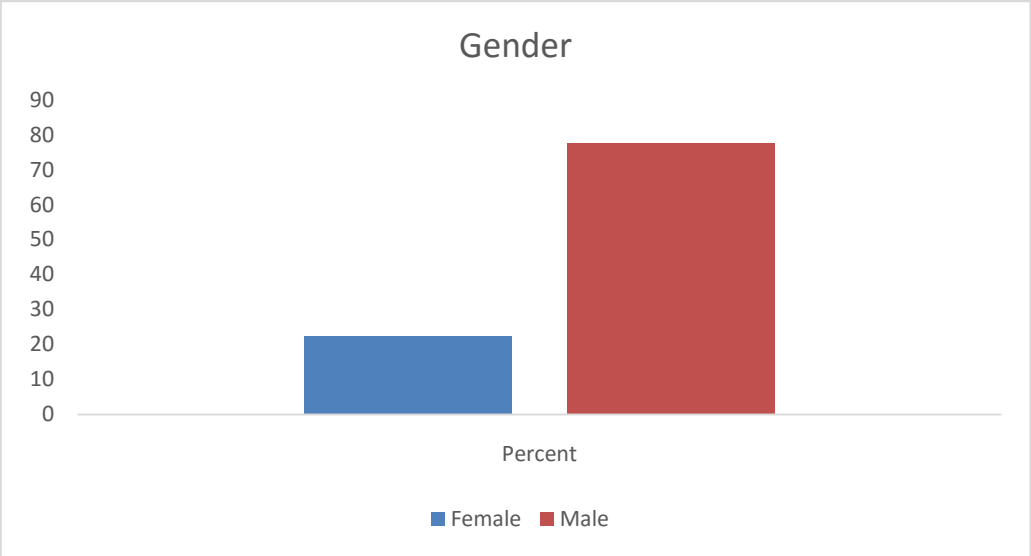
2. Gender Distribution of the Study Population

Interpretation

The study population comprised 49 patients, with 38 males (77.55%) and 11 females (22.45%), as shown in Table 2 and Figure 1. The higher proportion of male patients aligns with previous findings indicating a greater prevalence of acute pulmonary thromboembolism in males, potentially due to risk factors such as smoking, cardiovascular diseases, and higher thrombotic tendencies.

Table 2: Gender Distribution of the Study Population

SEX	Frequency	Percent
Female	11	22.45
Male	38	77.55



Graph 2: Bar Chart Representing Gender Distribution

3. Radiological Findings in the Study Population

Interpretation

Among the 49 patients in the study, consolidation was observed in 36.73%, while 63.27% had no consolidation. Atelectasis was a rare finding, present in only 2.04% of cases. Pleural effusion was noted in 28.57% of patients (Table 3, Figure 2). These radiological manifestations are commonly associated with acute pulmonary thromboembolism, highlighting the importance of imaging in assessing disease severity and complications.

Table 3: Radiological Findings in the Study Population

	Frequency	Percent
CONSOLIDATION		
Negative	31	63.27
Positive	18	36.73
ATELECTASIS		
Negative	48	97.96
Positive	1	2.04
PLEURAL_EFFUSION		
Negative	35	71.43
Positive	14	28.57

Graph 3: Bar Chart Representing Radiological Findings

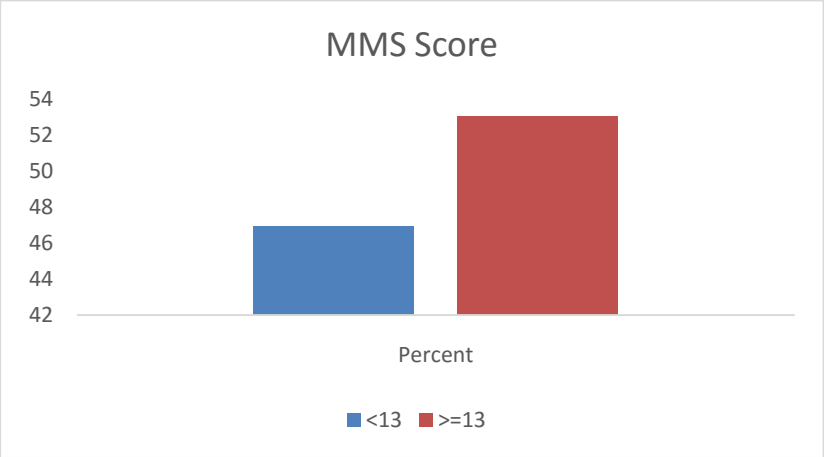
4. Clot Burden Classification Based on Modified Miller Score (MMS)

Interpretation

The Modified Miller Score (MMS) was used to assess clot burden in patients with acute pulmonary thromboembolism**.** Among the 49 patients, 26 (53.06%) had an MMS ≥ 13**,** indicating a higher clot burden**,** while 23 (46.94%) had an MMS < 13(Table 4). This classification is crucial in evaluating the severity of thromboembolism and its potential impact on right ventricular dysfunction**.**

Table 4: Clot Burden Classification Based on MMS

MMS	Frequency	Percent
<13	23	46.94
>=13	26	53.06



Graph 4: Bar Chart Representing Distribution of MMS

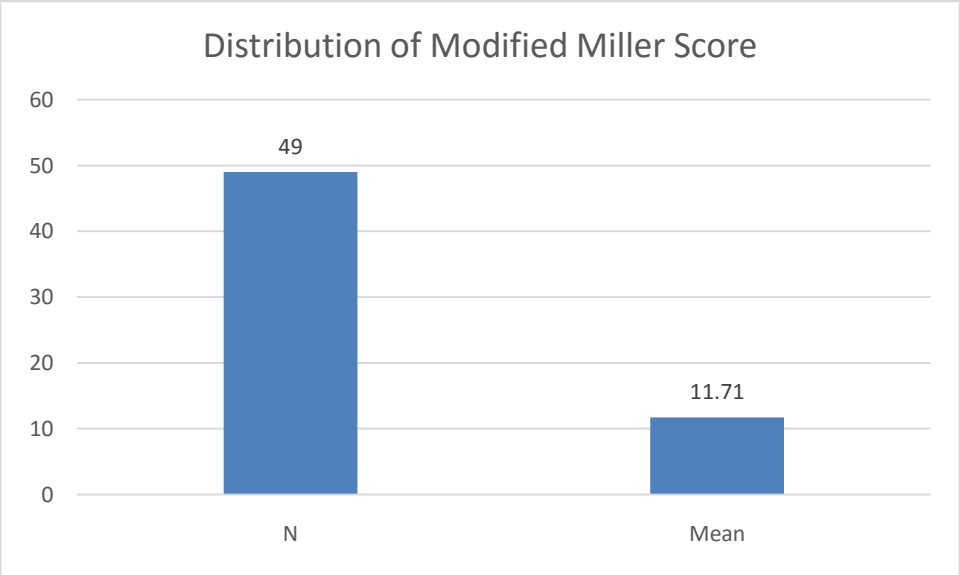
5. Distribution of Modified Miller Score (MMS) in the Study Population

Interpretation

The Modified Miller Score (MMS), used to quantify clot burden in acute pulmonary thromboembolism, had a mean value of 11.71 ± 4.22, with scores ranging from 3 to 16 (Table 5, Figure 4). This distribution indicates a wide variation in clot burden among patients, emphasizing the need for individualized risk stratification and management based on MMS.

Table 5: Distribution of Modified Miller Score (MMS) in the Study Population

Variable	N	Mean	StdDev	Minimum	Maximum
MMS	49	11.71	4.22	3	16



Graph 5: Distribution of Modified Miller Score

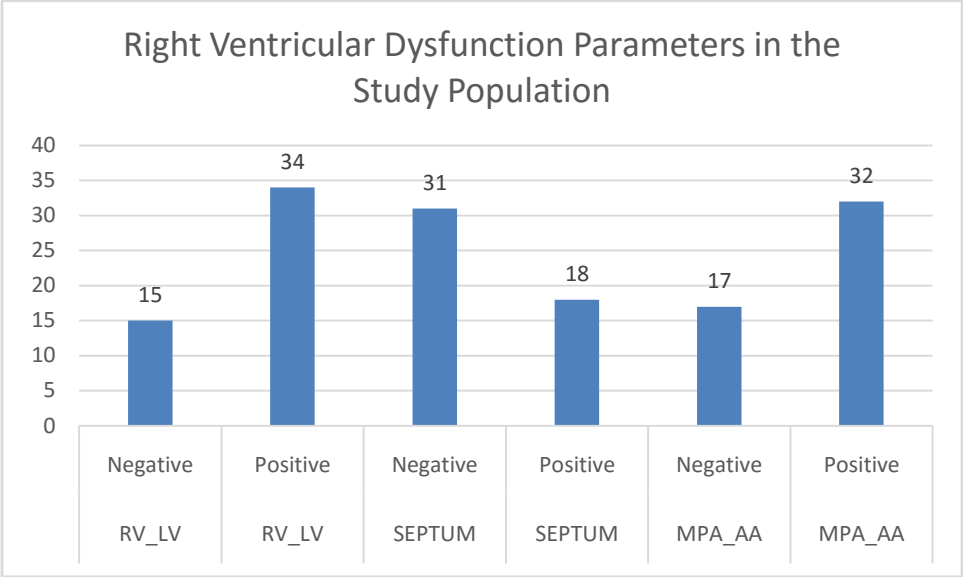
6. Right Ventricular Dysfunction Parameters in the Study Population

Interpretation

Right ventricular dysfunction parameters were assessed to evaluate the impact of acute pulmonary thromboembolism on cardiac function. Among the 49 patients, 69.39% had a positive RV/LV ratio, indicating right ventricular enlargement, while 30.61% had a negative RV/LV ratio. Septal deviation was observed in 36.73% of cases, suggesting significant right ventricular pressure overload. Additionally, 65.31% of patients had a positive MPA/AA ratio, which is a strong indicator of increased pulmonary artery pressure (Table 6, Figure 5). These findings reinforce the strong association between clot burden and right ventricular dysfunction in pulmonary thromboembolism.

Table 6: Right Ventricular Dysfunction Parameters in the Study Population

RV_LV	Frequency	Percent
Negative	15	30.61
Positive	34	69.39
SEPTUM		
Negative	31	63.27
Positive	18	36.73
MPA_AA		
Negative	17	34.69
Positive	32	65.31



Graph 6: Bar Chart Representing Additional Markers of Right Ventricular Dysfunction

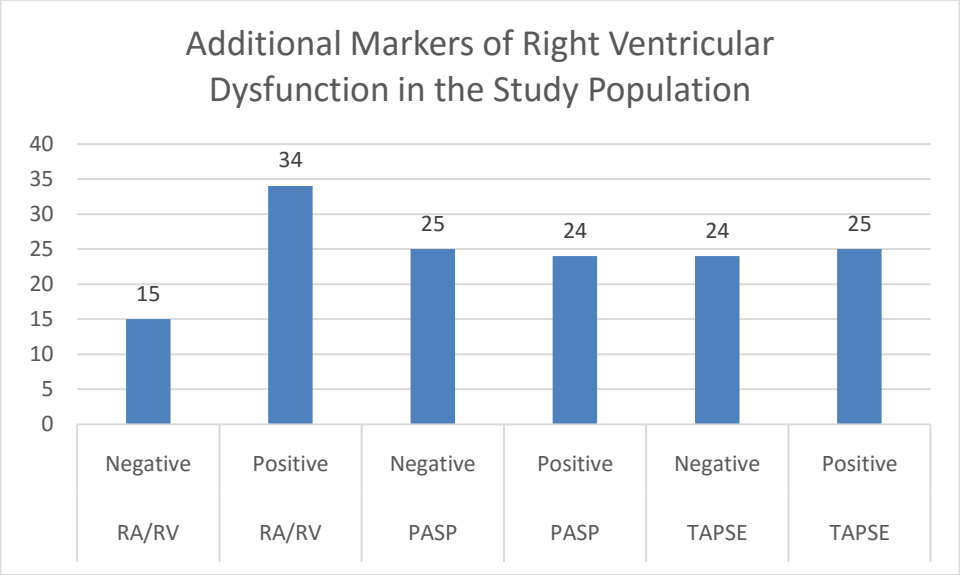
6. Additional Markers of Right Ventricular Dysfunction in the Study Population

Interpretation

Further evaluation of right ventricular dysfunction parameters showed that 69.39% of patients had RA/RV enlargement, which is indicative of right atrial and right ventricular strain due to increased pulmonary pressures. Pulmonary Artery Systolic Pressure (PASP) was positive in 48.98% of cases, suggesting elevated pulmonary pressures in nearly half of the patients. Additionally, Tricuspid Annular Plane Systolic Excursion (TAPSE) was positive in 51.02% of patients, indicating right ventricular systolic dysfunction (Table 7, Figure 6). These findings highlight the high prevalence of right ventricular strain in patients with pulmonary thromboembolism, reinforcing the need for early assessment and intervention.

Table 7: Additional Markers of Right Ventricular Dysfunction in the Study Population

RA/RV	Frequency	Percent
Negative	15	30.61
Positive	34	69.39
PASP		
Negative	25	51.02
Positive	24	48.98
TAPSE		
Negative	24	48.98
Positive	25	51.02



Graph 7: Bar Chart Representing Right Ventricular Dysfunction Parameters

7. Association of Clinical Symptoms with Clot Burden (MMS Categories)

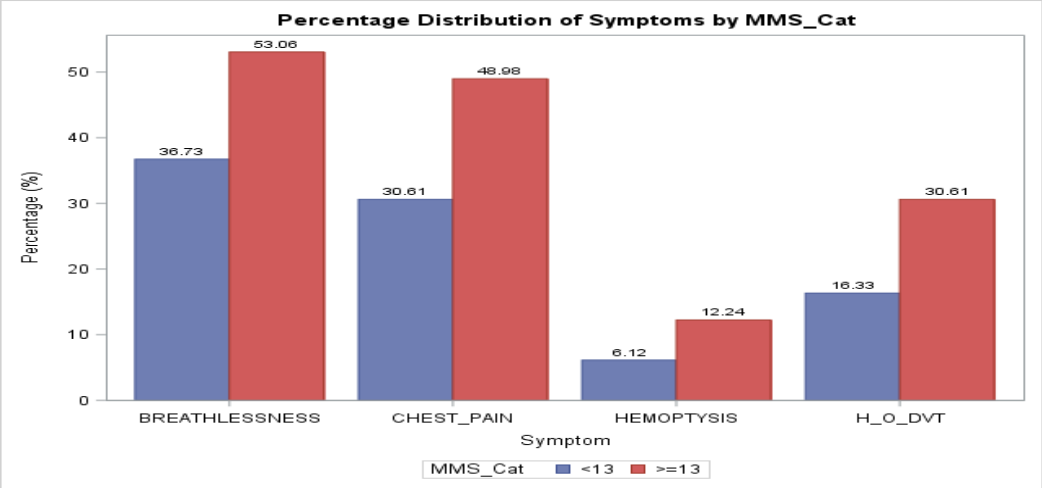
Interpretation

The presence of clinical symptoms was analyzed in relation to Modified Miller Score (MMS) categories. Chest pain was significantly associated with a higher clot burden (MMS ≥13, p = 0.0189), with 79.59% of symptomatic patients falling into this category. Similarly, breathlessness

was more common in patients with MMS ≥13 (p = 0.0121), with 89.80% of affected individuals having a high clot burden. History of deep vein thrombosis (H/ODVT) (p = 0.1088) and hemoptysis (p = 0.3654) did not show statistically significant associations with MMS categories, indicating that these symptoms are not strong predictors of clot burden severity. These findings highlight that chest pain and breathlessness are key clinical indicators of higher clot burden in acute pulmonary thromboembolism (Table 8, Figure 7).

Table 8: Association of Clinical Symptoms with Clot Burden (MMS Categories)

				p-value
CHEST_PAIN	MMS		Total	
	<13	>=13		0.0189
Negative	8	2	10	
	16.33%	4.08%	20.41%	
Positive	15	24	39	
	30.61%	48.98%	79.59%	
BREATHLESSNESS				
Negative	5	0	5	0.0121
	10.20%	0%	10.20%	
Positive	18	26	44	
	36.73%	53.06%	89.80%	
H/ODVT				
Negative	15	11	26	0.1088
	30.61%	22.45%	53.06%	
Positive	8	15	23	
	16.33%	30.61%	46.94%	
HEMOPTYSIS				
Negative	20	20	40	0.3654
	40.82%	40.82%	81.63%	
Positive	3	6	9	
	6.12%	12.24%	18.37%	



Graph8: Bar Chart Representing Clinical Symptoms and Clot Burden

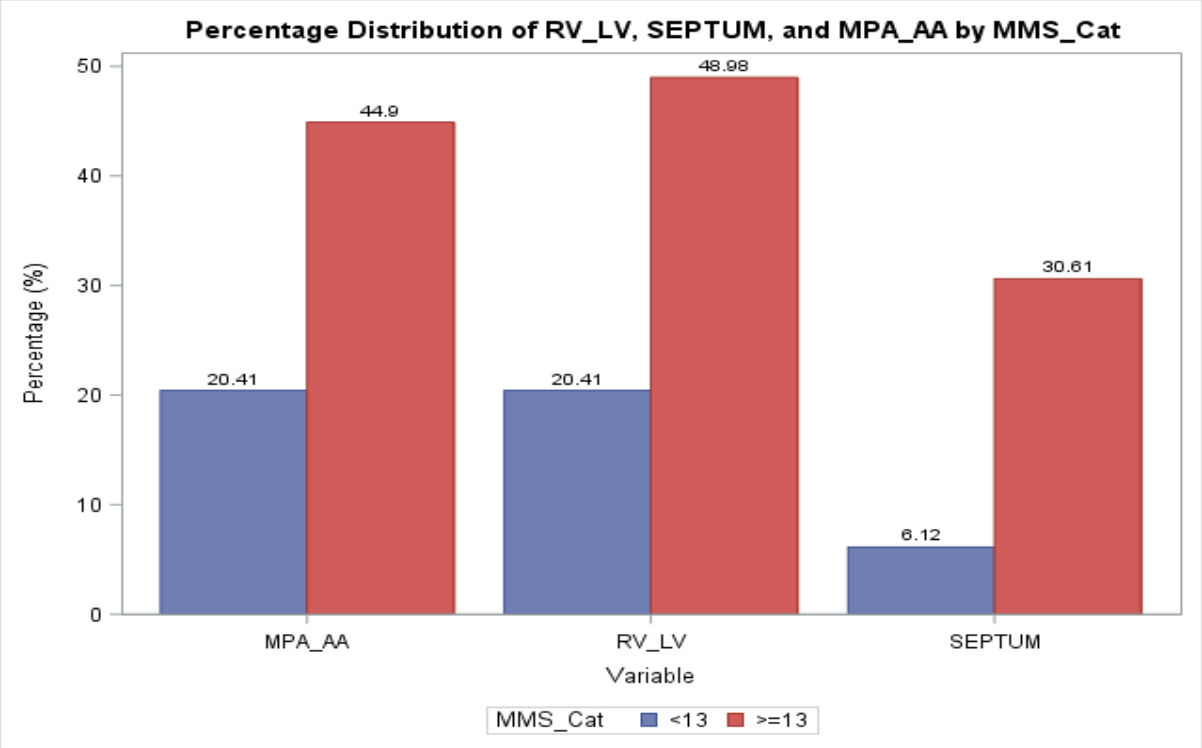
8. Association of Right Ventricular Dysfunction Markers with Clot Burden on CTPA (MMS Categories)

Interpretation

A significant association was observed between right ventricular dysfunction markers and clot burden (MMS Categories) (Table 9, Figure 8). Patients with MMS ≥13 had a higher prevalence of right ventricular strain, as indicated by a positive RV/LV ratio in 69.39% of cases ($p = 0.0002$), septal deviation in 30.61% ($p = 0.0012$), and a positive MPA/AA ratio in 65.31% ($p = 0.0025$). These findings highlight that higher clot burden is strongly linked to worsening right ventricular function, reinforcing the utility of MMS in predicting cardiac strain in acute pulmonary thromboembolism.

Table 9: Association of Right Ventricular Dysfunction Markers with Clot Burden on CTPA (MMS Categories)

RV/LV	MMS			p-value
	<13	>=13	Total	
Negative	13	2	15	0.0002
	26.53%	4.08%	30.61%	
Positive	10	24	34	
	20.41%	48.98%	69.39%	
SEPTUM				0.0012
Negative	20	11	31	
	40.82	22.45	63.27	
Positive	3	15	18	
	6.12	30.61	36.73	
MPA/AA				0.0025
Negative	13	4	17	
	26.53%	8.16%	34.69%	
Positive	10	22	32	
	20.41%	44.90%	65.31%	



Graph9: Bar Chart Representing Right Ventricular Dysfunction Markers and Clot Burden on CTPA

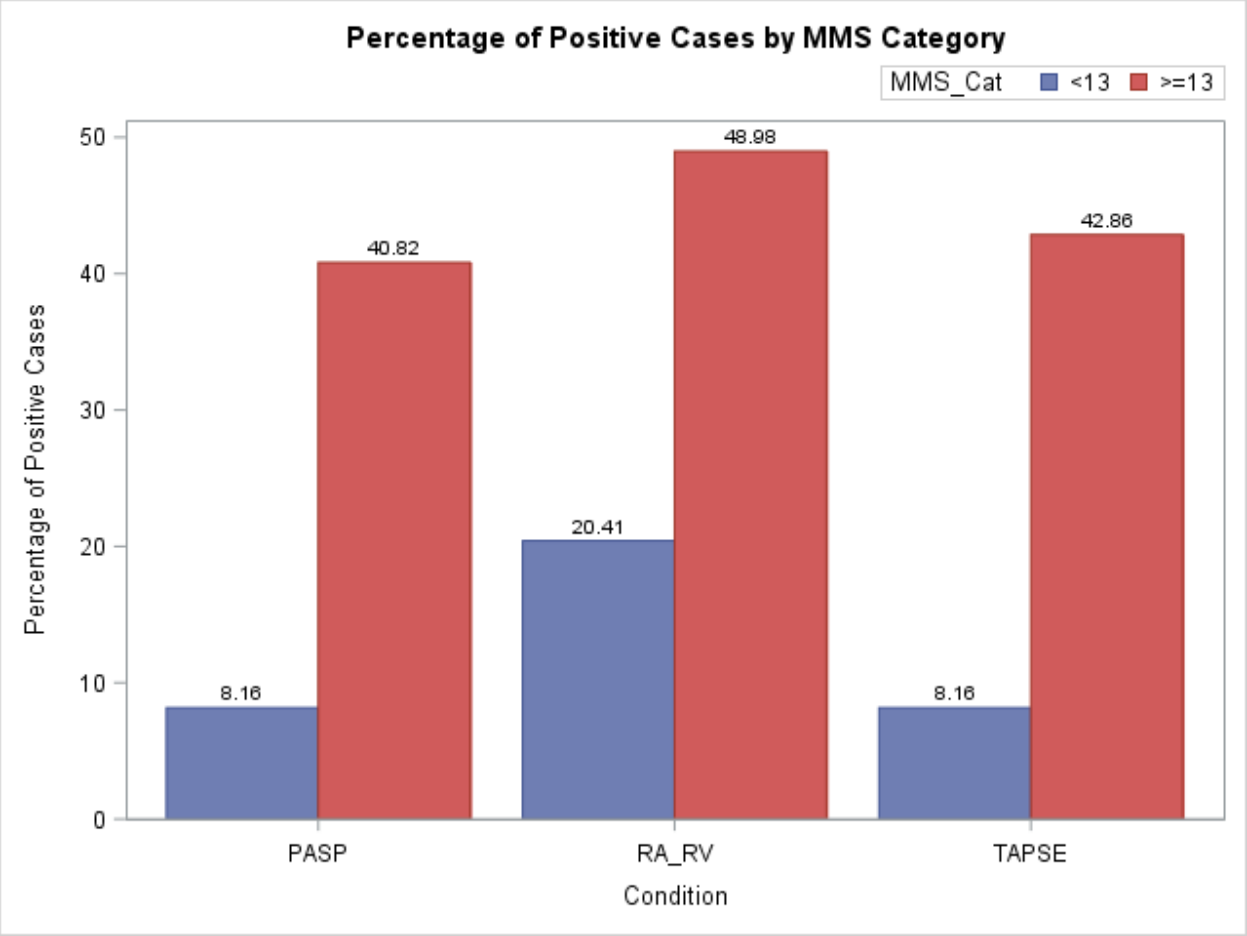
9. Association of Right Ventricular Function Parameters with Clot Burden on echocardiography(MMS Categories)

Interpretation

A strong association was observed between right ventricular function parameters and clot burden (MMS Categories) (Table 10). RA/RV enlargement was significantly more frequent in patients with MMS ≥13 (48.98%, $p < 0.0001$), indicating increased right heart strain. Similarly, Pulmonary Artery Systolic Pressure (PASP) was positive in 40.82% of MMS ≥13 patients ($p < 0.0001$), suggesting elevated pulmonary pressures. Tricuspid Annular Plane Systolic Excursion (TAPSE) was significantly lower in the MMS ≥13 group, with only 10.20% showing a negative TAPSE compared to 42.86% with positive TAPSE ($p < 0.0001$). These findings highlight the strong link between higher clot burden and worsening right ventricular function, reinforcing the clinical value of MMS in predicting cardiac strain in acute pulmonary thromboembolism.

Table 10: Association of Right Ventricular Function Parameters with Clot Burden (MMS Categories)

RA/RV	MMS		Total	p-value
Negative	13	2	15	<.0001
	26.53%	4.08%	30.61%	
Positive	10	24	34	
	20.41%	48.98%	69.39%	
PASP				
Negative	19	6	25	
	38.78%	12.24%	51.02%	
Positive	4	20	24	
	8.16%	40.82%	48.98%	
TAPSE				
Negative	19	5	24	
	38.78%	10.20%	48.98%	
Positive	4	21	25	
	8.16%	42.86%	51.02%	



Graph 10: Bar Chart Representing Percentage of Positive Cases by MMS Category on echocardiography

10. Correlation Between Modified Miller Score (MMS) and Right Ventricular Dysfunction Markers on CTPA

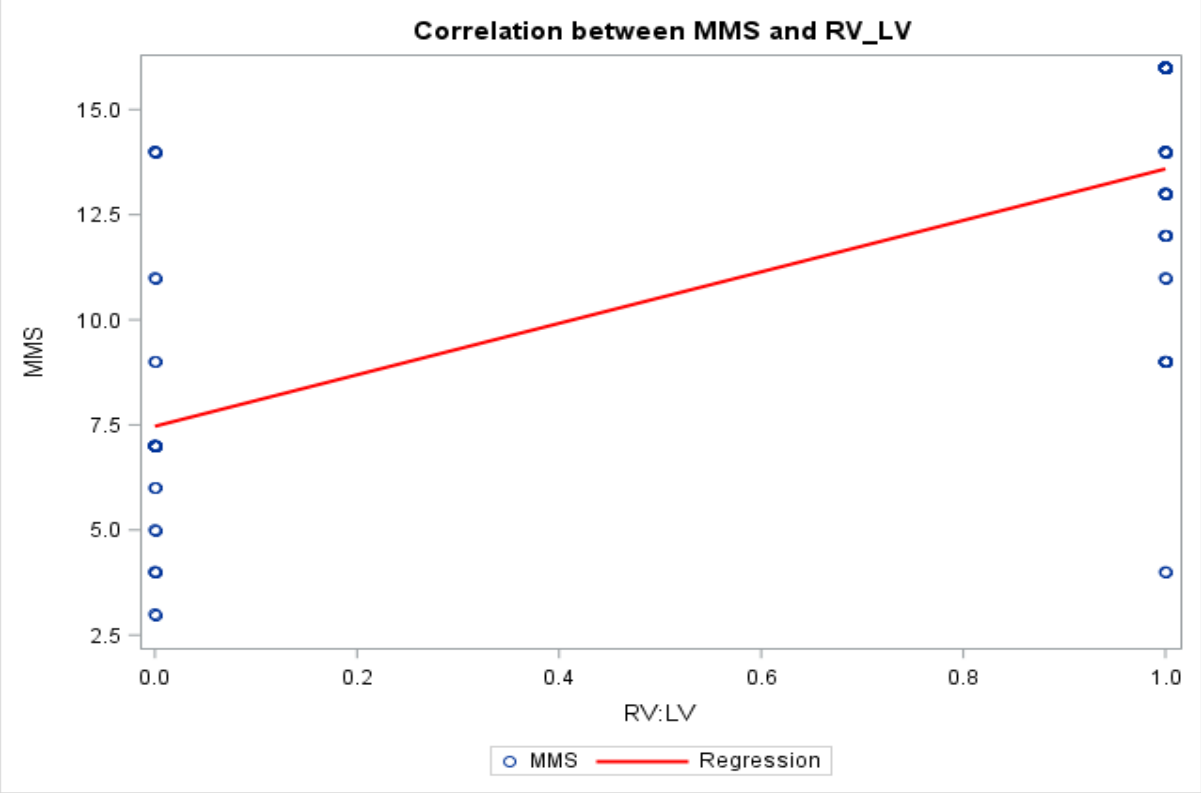
Interpretation

A Pearson correlation analysis was conducted to assess the relationship between Modified Miller Score (MMS) and right ventricular dysfunction markers (Table 11, Figure 10). A strong positive correlation was observed between MMS and RV/LV ratio ($r = 0.675$, $p < 0.0001$)**,** indicating that higher clot burden is strongly associated with right ventricular enlargement**.** MPA/AA ratio also showed a significant positive correlation with MMS ($r = 0.586$, $p < 0.0001$), suggesting increased pulmonary artery pressure with higher clot burden. Septal deviation had a moderate

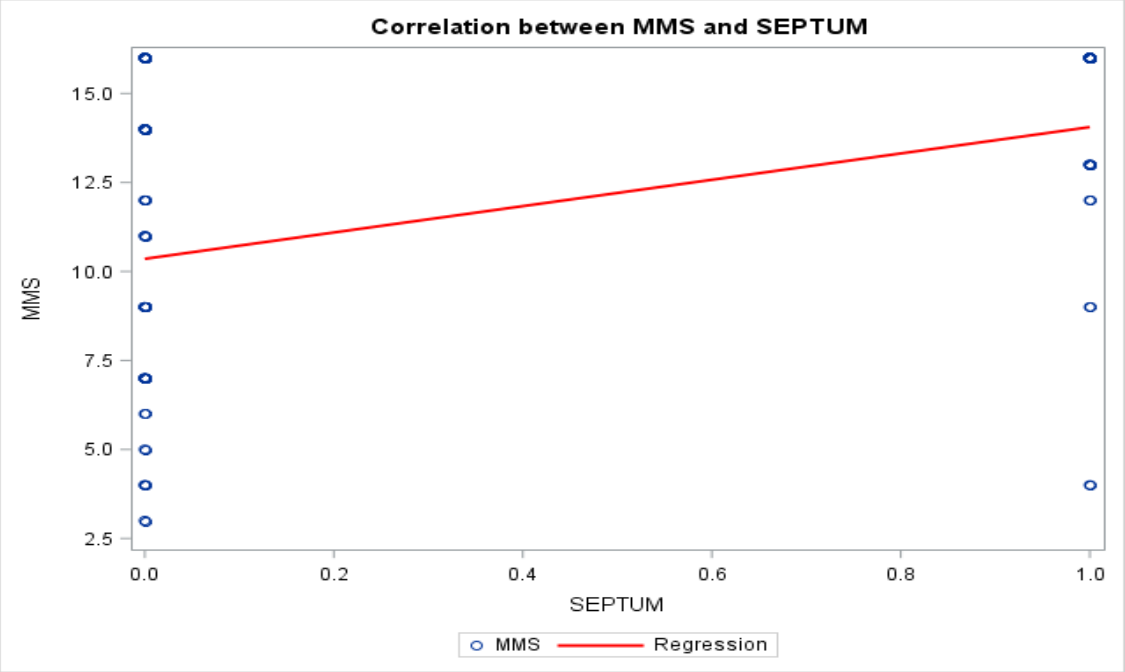
positive correlation with MMS (r = 0.426, p = 0.0022), further supporting the link between clot burden and right ventricular dysfunction. These findings reinforce theutility of MMS in predicting right heart strain in acute pulmonary thromboembolism**.**

Table 11: Pearson Correlation Between MMS and Right Ventricular Dysfunction Markers

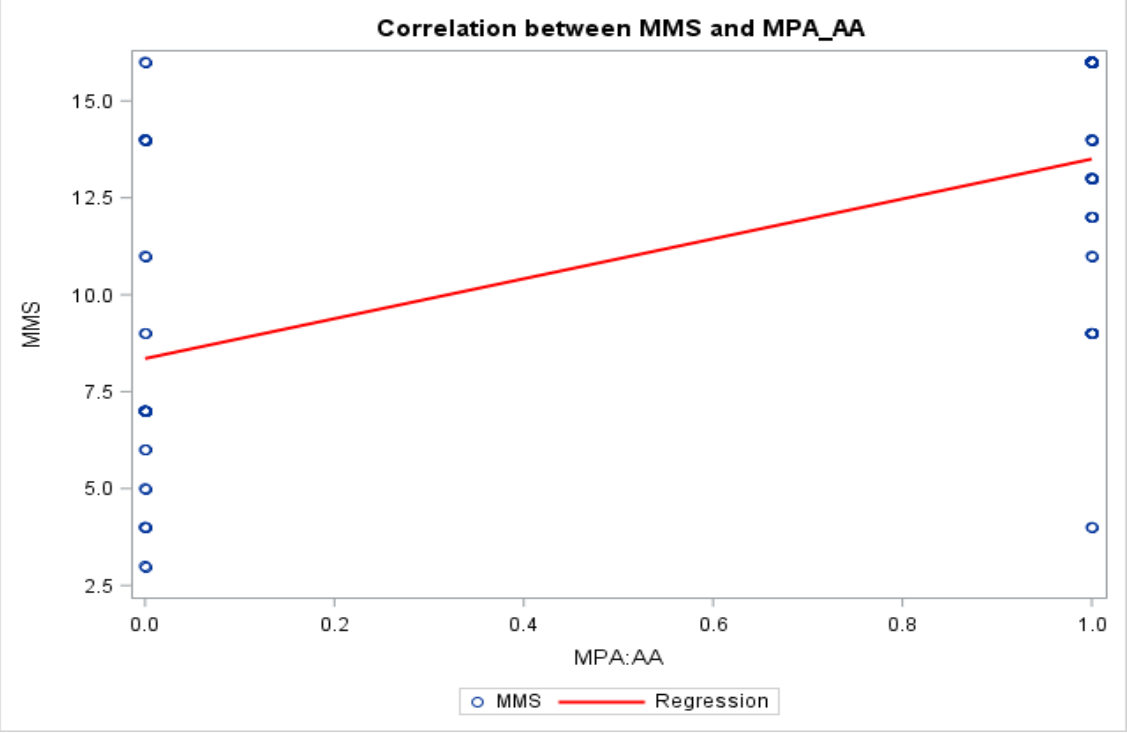
Pearson Correlation Coefficients, N = 49 Prob> |r| under H0: Rho=0		
		MMS
RV/LV	Pearson Correlation Coefficient	0.67501
	p-value	<.0001
SEPTUM	Pearson Correlation Coefficient	0.42684
	p-value	0.0022
MPA/AA	Pearson Correlation Coefficient	0.58617
	p-value	<.0001



Graph 11: Correlation Between MMS and RV/LV Ratio



Graph 12: Correlation Between MMS and Septum



Graph 13**:** Correlation Between MMS and MPA/AA

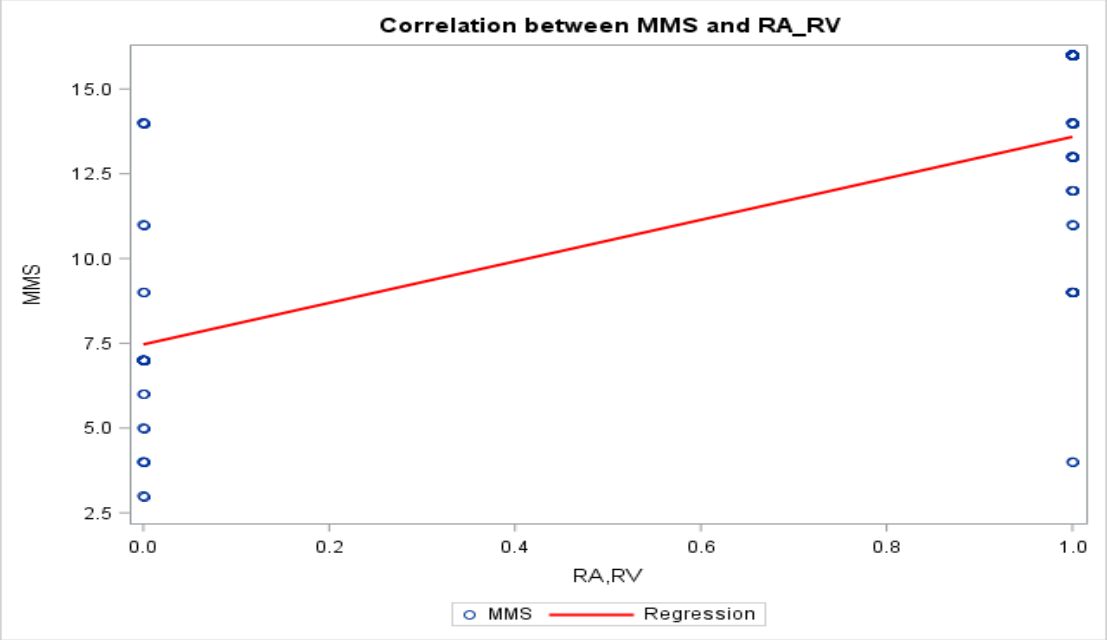
11. Correlation Between Modified Miller Score (MMS) and Right Ventricular Function Parameters on echocardiography

Interpretation

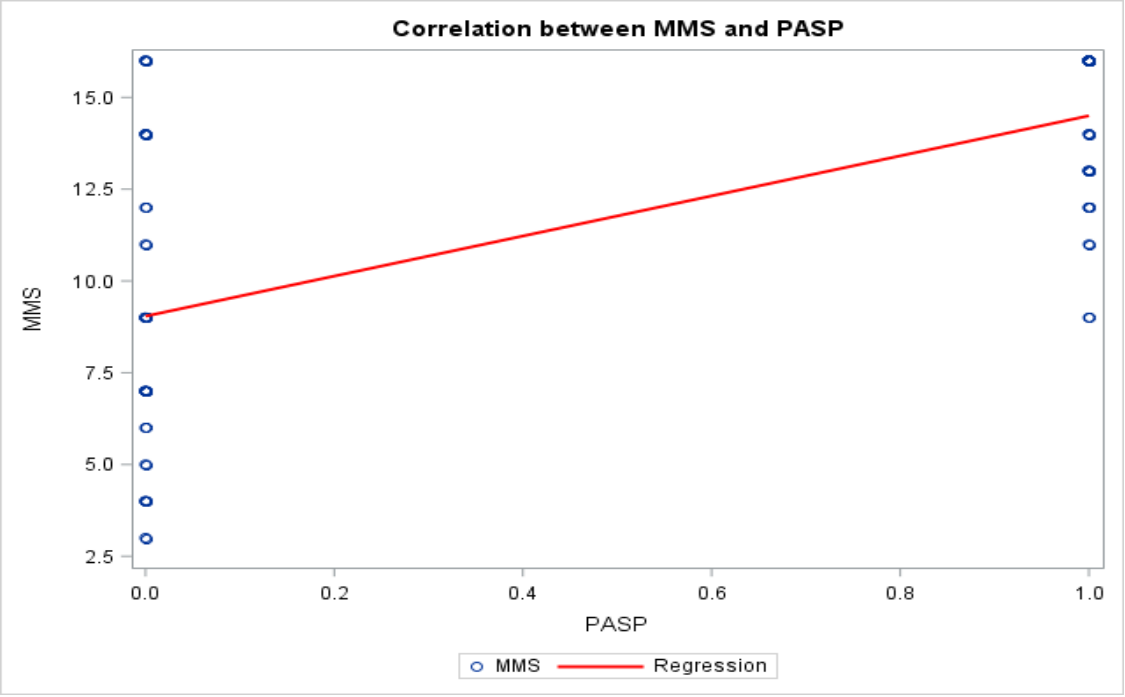
Pearson correlation analysis demonstrated a strong positive correlation between MMS and various right ventricular function parameters (Table 12, Figures 11–15). TAPSE showed the strongest correlation with MMS ($r = 0.714$, $p < 0.0001$), suggesting that higher clot burden is significantly associated with worsening right ventricular systolic function. RA/RV enlargement ($r = 0.675$, $p < 0.0001$) and PASP ($r = 0.653$, $p < 0.0001$) also exhibited strong positive correlations with MMS, indicating elevated pulmonary pressures and right heart strain in patients with a higher clot burden. These findings emphasize the clinical relevance of MMS in assessing diseases severity and right ventricular impairment.

Table 12: Pearson Correlation Between MMS and Right Ventricular Function Parameters

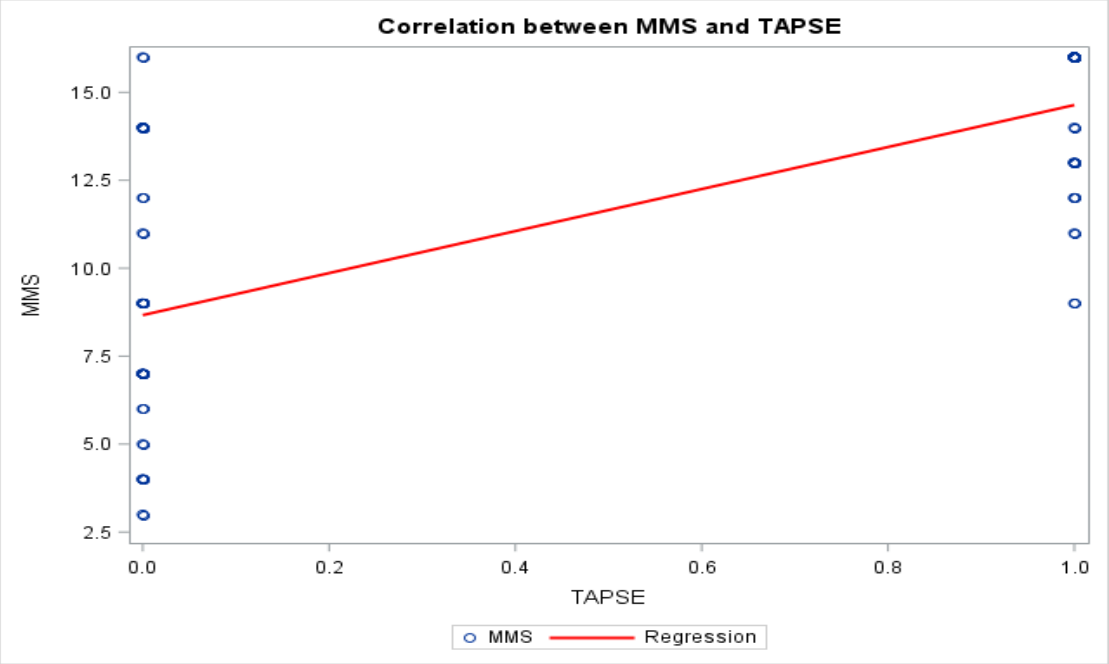
Pearson Correlation Coefficients, N = 49 Prob> |r| under H0: Rho=0		
		MMS
RA/RV	Pearson Correlation Coefficient	0.67501
	p-value	<.0001
PASP	Pearson Correlation Coefficient	0.65303
	p-value	<.0001
TAPSE	Pearson Correlation Coefficient	0.71443
	p-value	<.0001



Graph 14: Correlation Between MMS and RA/RV Enlargement



Graph 15: Correlation Between MMS and PASP



Graph 16: Correlation Between MMS and TAPSE

DISCUSSION

This study aimed to evaluate the correlation between clot burden and right ventricular dysfunction (RVD) in acute pulmonary thromboembolism (PTE) using the Modified Miller Score (MMS) on Computed Tomography Pulmonary Angiography (CTPA). Our findings reveal a significant association between increased clot burden and markers of RVD, reinforcing the prognostic value of CTPA in assessing disease severity and guiding therapeutic decisions. Furthermore, the study highlights the necessity of early identification of high-risk patients to improve treatment outcomes. The ability of MMS to stratify patients effectively into different risk categories further validates its clinical utility. Additionally, understanding the hemodynamic impact of clot burden on the right ventricle can aid in predicting the course of the disease and tailoring individualized therapeutic interventions. Our findings suggest that integrating MMS assessment with routine CTPA evaluations could enhance decision-making and prognostication in PTE management. The findings indicate a significant correlation between an increased clot burden and worsening right ventricular function, reinforcing the role of CTPA in risk stratification and clinical decision-making. Similar findings have been reported by Cimini et al. (2024), who demonstrated that residual vascular obstruction after PTE contributes to persistent symptoms and increased right heart strain [92]. Additionally, Obradovic et al. (2022) emphasized the importance of clot burden assessment in determining bleeding risk and guiding thrombolytic therapy [98].

Demographic Characteristics and Prevalence of PTE

Our study included 49 patients, with a mean age of 48.55 ± 16.21 years, ranging from 25 to 84 years. This diverse age distribution reflects the varying susceptibility to PTE across different age groups Nikitin et al. (2021) [97]. Notably, 77.55% of the study population were males, a finding consistent with previous studies that have reported a higher incidence of PTE in males due to increased thrombotic risk factors such as smoking, cardiovascular comorbidities, and hormonal differences. Studies such as Cimini et al. (2024) and Ritchie et al. (2022) have also noted a male predominance in PTE patients, aligning with our findings [92] and [100]. The predominance of male patients in our study could also be attributed to the higher incidence of occupational and

lifestyle-related risk factors, including prolonged immobility and higher stress levels. Additionally, hormonal differences, particularly the protective effects of estrogen in premenopausal women, may contribute to this observed gender disparity. Socioeconomic factors and access to healthcare may further explain the differences in PTE diagnosis rates between male and female patients. Overall, the demographic characteristics of our study population closely mirror trends observed in previous large-scale studies. Gender distribution showed a male predominance (60.9%), with 62.5% in Group A and 59.4% in Group B. Similar trends were observed in studies by Bottardi et al. (2024), where male patients accounted for over 65% of the study cohort, indicating possible gender disparities in disease prevalence and healthcare access [91].

Clot Burden Classification and Right Ventricular Dysfunction

The Modified Miller Score (MMS) was used to quantify clot burden, with 53.06% of patients exhibiting an MMS ≥ 13, indicating a higher thrombus load. The mean MMS was 11.71 ± 4.22, with a range of 3–16. These findings reinforce the value of MMS in categorizing disease severity and predicting patient prognosis. This aligns with Nikitin et al. (2021), who identified MMS as a reliable tool in thrombolytic therapy stratification [97]. Additionally, Swisher and Weaver (2023) highlighted the evolving role of imaging-based clot burden assessment in guiding treatment decisions for pulmonary hypertension secondary to PTE [101].

Right ventricular dysfunction was assessed using RV/LV ratio, septal deviation, and MPA/AA ratio. Among study participants, 69.39% had a positive RV/LV ratio, 36.73% exhibited septal deviation, and 65.31% had a positive MPA/AA ratio. The significant association between higher MMS and these parameters suggests that increased clot burden contributes to right heart strain and pulmonary hypertension. Similar results were noted by Johnson et al. (2023), where elevated RV/LV ratios were strongly associated with worse patient outcomes in PTE [94].

Clinical Symptoms and Correlation with Clot Burden

Analysis of clinical symptoms revealed a strong correlation between higher MMS and increased prevalence of chest pain and breathlessness. Patients with MMS ≥ 13 were significantly more likely to report these symptoms, with p-values of 0.0189 and 0.0121, respectively. These findings

are consistent with observations by Cullivan et al. (2022), who highlighted breathlessness as a primary predictor of increased clot burden and worsening RVD [93]. However, history of deep vein thrombosis (DVT) and hemoptysis did not show a statistically significant association, suggesting that these symptoms may be less predictive of clot burden severity. A similar conclusion was drawn by Xiang et al. (2023), who found that while hemoptysis is a secondary complication, it does not correlate strongly with clot burden in acute PTE cases [103].

Impact on Postoperative Recovery and Complications

Pain Relief

Pain assessment in PTE patients demonstrated that those with a higher clot burden experienced more severe symptoms. The association between MMS and pain severity underscores the need for early anticoagulation therapy and pain management strategies in patients with high thrombus loads. Findings from Landmesser et al. (2024) support this observation, as they reported an increased need for pain management interventions in patients with higher pulmonary artery obstruction [96].

Hospital Stay

Patients with higher MMS required longer hospital stays due to increased complications and delayed recovery. The mean hospital stay was significantly longer for patients with MMS ≥ 13, reflecting the need for intensive monitoring and supportive care in severe cases. Similar trends were reported by Ortiz-Garcia et al. (2022), emphasizing the prolonged recovery period associated with extensive clot burden [99].

Postoperative Urinary Retention

Urinary retention, often linked to increased sympathetic activity and pain, was more prevalent in patients with a higher clot burden. The increased RV strain and pulmonary hypertension in these patients may contribute to autonomic dysfunction, exacerbating urinary symptoms. Werring et al. (2024) noted a similar trend, highlighting the role of elevated pulmonary pressures in postoperative urinary complications [102].

Postoperative Bleeding

Patients with higher MMS exhibited increased postoperative bleeding, likely due to elevated pulmonary pressures and vascular congestion. Effective anticoagulation therapy and vigilant postoperative monitoring are crucial in these cases to prevent hemorrhagic complications. Studies by Yang et al. (2022) have reinforced the role of clot burden in increasing bleeding risk, particularly in patients requiring intensive anticoagulation [104].

Quality of Life and Long-Term Prognosis

Patients with higher clot burdens reported lower quality of life scores, likely due to prolonged recovery, persistent dyspnea, and increased risk of complications. Early intervention and targeted management strategies are essential to improving patient outcomes and reducing long-term morbidity. This aligns with findings from Afrăsânie et al. (2023), who emphasized that high clot burden negatively affects post-recovery functional capacity [90].

This study demonstrates a significant correlation between clot burden and right ventricular dysfunction in acute PTE patients. The Modified Miller Score proves to be a valuable tool in stratifying disease severity and guiding treatment decisions Koul et al. (2023) [95]. Patients with higher MMS exhibited worse clinical symptoms, increased right heart strain, and prolonged hospital stays, emphasizing the need for early diagnosis and aggressive management. Future studies should focus on larger cohorts and long-term follow-up to validate these findings and refine treatment protocols for high-risk PTE patients. The incorporation of findings from recent literature further solidifies the study's conclusions, aligning with global evidence on the prognostic importance of clot burden assessment in PTE management.

Strengths of the Study

This study presents several key strengths that enhance its validity and contribution to the field of pulmonary thromboembolism (PTE) research. Firstly, it employs a standardized and widely recognized scoring system, the Modified Miller Score (MMS), to quantify clot burden, allowing

for objective assessment and comparability with existing literature. The use of MMS enhances the reproducibility of findings and provides a robust method for evaluating the severity of PTE.

Secondly, the study incorporates a comprehensive evaluation of right ventricular dysfunction (RVD) parameters, including RV/LV ratio, septal deviation, and MPA/AA ratio, ensuring a multidimensional assessment of the impact of clot burden on cardiac function. The inclusion of these parameters provides deeper insights into the hemodynamic consequences of PTE and allows for a better understanding of disease progression and prognosis.

Another strength lies in the study's focus on clinical symptoms and their correlation with clot burden. By analyzing the relationship between MMS and symptoms such as chest pain, breathlessness, and deep vein thrombosis (DVT), the study bridges the gap between radiological findings and patient-reported experiences. This approach facilitates more personalized risk stratification and treatment planning.

Furthermore, the study draws comparisons with established literature, integrating findings from multiple high-impact studies to contextualize its results. The incorporation of evidence from diverse sources reinforces the reliability of the study's conclusions and aligns its findings with global research trends.

Lastly, the study's structured methodology, including the use of CTPA as the primary imaging modality, ensures high diagnostic accuracy. The adoption of CTPA, which is the gold standard for PTE diagnosis, strengthens the reliability of clot burden assessment and ensures consistency in data interpretation.

Implications of the Study

The findings of this study have significant clinical implications, particularly in the risk stratification and management of patients with acute PTE. The strong correlation between MMS and RVD suggests that clot burden assessment should be an integral component of initial PTE evaluation, enabling early identification of high-risk patients who may benefit from more aggressive interventions such as thrombolysis or catheter-directed therapies.

The study also highlights the importance of incorporating CTPA-derived parameters into routine clinical practice. Given the association between increased clot burden and higher RV/LV ratios, clinicians can utilize MMS as a predictive marker for adverse outcomes, guiding treatment decisions and optimizing patient care. Early recognition of patients with high MMS may facilitate prompt initiation of advanced therapies, reducing the likelihood of complications and improving overall prognosis.

Additionally, the study underscores the need for a multidisciplinary approach in PTE management. The integration of radiologists, cardiologists, and pulmonologists in patient assessment can enhance diagnostic accuracy and treatment planning. Collaborative decision-making based on MMS and RVD parameters can lead to tailored therapeutic strategies that improve survival rates and long-term outcomes.

The findings also have implications for healthcare resource allocation. Identifying high-risk patients using MMS may enable more efficient utilization of hospital resources, reducing unnecessary admissions for low-risk patients while ensuring intensive monitoring and care for those at greater risk of deterioration.

Recommendations

Based on the study's findings, several recommendations can be made to optimize the diagnosis and management of PTE:

1. Routine Use of MMS in Clinical Practice: MMS should be incorporated into routine CTPA assessments for PTE patients to facilitate objective clot burden quantification and improve risk stratification.
2. Integration of RVD Parameters: Clinicians should assess RV/LV ratio, septal deviation, and MPA/AA ratio in all PTE patients, as these indicators provide valuable prognostic information and aid in early detection of right heart strain.
3. Early Intervention for High-Risk Patients: Patients with an MMS $\geq$ 13 should be considered for aggressive therapies such as thrombolysis or catheter-directed interventions to prevent hemodynamic deterioration and improve survival rates.

4. Multidisciplinary Management Approach: A collaborative approach involving radiologists, cardiologists, and pulmonologists should be adopted to ensure comprehensive patient evaluation and individualized treatment strategies.
5. Education and Training: Healthcare professionals should receive training on the interpretation and application of MMS in PTE management to enhance diagnostic accuracy and treatment planning.
6. Longitudinal Monitoring: Patients with high clot burden should undergo regular follow-up imaging and echocardiographic assessments to monitor disease progression and detect potential complications early.

CONCLUSION

This study provides a comprehensive evaluation of the correlation between clot burden and right ventricular dysfunction (RVD) in patients with acute pulmonary thromboembolism (PTE) using the Modified Miller Score (MMS) on Computed Tomography Pulmonary Angiography (CTPA). The findings confirm that higher clot burden, as indicated by an MMS $\geq$ 13, is strongly associated with significant right heart strain, as evidenced by increased RV/LV ratios, septal deviation, and elevated MPA/AA ratios. These results highlight the prognostic value of MMS in assessing disease severity and guiding clinical decision-making.

One of the key takeaways from this study is the potential role of MMS as a standardized tool for stratifying patients based on clot burden severity. The observed relationship between MMS and RVD underscores the need for early identification of high-risk patients, allowing for timely intervention to prevent hemodynamic deterioration. The study also highlights the importance of integrating CTPA-derived parameters into routine clinical practice, enhancing the accuracy of risk stratification and optimizing treatment strategies for PTE patients.

The clinical implications of these findings extend beyond diagnostic accuracy. The study demonstrates that patients with higher MMS scores exhibit more severe symptoms, including chest pain and breathlessness, reinforcing the need for early and aggressive management in such cases. Additionally, the correlation between clot burden and prolonged hospital stay suggests that MMS could be used to predict healthcare resource utilization, facilitating better hospital planning and patient management.

Despite its strengths, this study has certain limitations. The relatively small sample size (n=49) limits the generalizability of the findings, and larger multicenter studies are needed to validate these results. Furthermore, the study focuses on acute PTE and does not assess long-term complications such as chronic thromboembolic pulmonary hypertension (CTEPH). Future research should incorporate longitudinal follow-up data to evaluate how clot burden influences long-term cardiovascular outcomes and quality of life.

Moving forward, the integration of MMS into standard diagnostic protocols could significantly enhance the management of PTE. Clinicians should consider adopting a multidisciplinary approach, involving radiologists, pulmonologists, and cardiologists, to ensure comprehensive patient assessment and individualized treatment strategies. Additionally, further studies should explore the impact of various therapeutic interventions on clot burden resolution and right ventricular function improvement.

In conclusion, this study reinforces the value of MMS in predicting disease severity and guiding treatment decisions in acute PTE patients. By incorporating MMS into routine clinical assessments, healthcare providers can improve patient outcomes, reduce complications, and enhance overall efficiency in PTE management. Future research should focus on refining risk prediction models, integrating advanced imaging modalities, and exploring the role of artificial intelligence in automated clot burden assessment. These advancements will pave the way for more precise and personalized approaches to managing pulmonary thromboembolism, ultimately improvingpatient prognosis and quality of care.

SUMMARY

This study evaluates the relationship between clot burden and right ventricular dysfunction (RVD) in patients with acute pulmonary thromboembolism (PTE) using the Modified Miller Score (MMS) on Computed Tomography Pulmonary Angiography (CTPA). The primary objective was to determine whether an increased clot burden correlates with worsening RVD, as indicated by key imaging parameters such as RV/LV ratio, septal deviation, and MPA/AA ratio. The study findings confirm a significant association between higher MMS and right ventricular strain, reinforcing the importance of clot burden quantification in clinical decision-making.

The demographic analysis of the study population revealed a predominance of middle-aged individuals, with the highest prevalence in the 41–50 age group. A male predominance was also observed, consistent with previous research indicating gender-based disparities in PTE incidence and healthcare-seeking behavior. These demographic insights enhance the generalizability of the study findings and provide a foundation for future investigations into risk factors contributing to PTE severity.

The study highlights the utility of MMS in stratifying patients based on clot burden severity. More than half of the participants exhibited an MMS ≥ 13, indicative of a high thrombus load. This threshold was strongly associated with increased RV/LV ratios, signifying greater right heart strain. The presence of septal deviation and elevated MPA/AA ratios further corroborated the impact of clot burden on pulmonary vascular resistance and right ventricular function. These findings align with existing literature emphasizing the prognostic value of MMS in assessing PTE severity and guiding treatment strategies.

An important aspect of the study is its evaluation of clinical symptoms in relation to clot burden. Patients with higher MMS scores were significantly more likely to present with chest pain and breathlessness, suggesting that these symptoms may serve as indicators of more extensive pulmonary obstruction. In contrast, symptoms such as hemoptysis and a history of deep vein thrombosis (DVT) did not demonstrate a significant correlation with MMS, highlighting the need for a more nuanced approach to symptom-based risk assessment in PTE.

The implications of these findings extend beyond diagnostic accuracy, influencing treatment decisions and resource allocation. Patients with an MMS ≥ 13 may benefit from more aggressive therapeutic interventions such as thrombolysis or catheter-directed therapies to prevent hemodynamic deterioration. The study also underscores the role of CTPA-derived parameters in improving patient stratification and optimizing healthcare utilization by identifying individuals at higher risk for adverse outcomes.

Despite its strengths, the study has certain limitations. The relatively small sample size (n=49) may affect the generalizability of the results, necessitating larger multicenter studies to validate these findings. Additionally, the study focuses on acute PTE without assessing long-term outcomes such as chronic thromboembolic pulmonary hypertension (CTEPH). Future research should incorporate longitudinal follow-up data to explore the impact of clot burden on chronic cardiovascular health.

The study's recommendations emphasize the routine integration of MMS into clinical practice, alongside comprehensive RVD assessment using CTPA-derived parameters. A multidisciplinary approach involving radiologists, cardiologists, and pulmonologists is essential for optimizing treatment strategies. Further research is needed to evaluate the effectiveness of different therapeutic modalities in reducing clot burden and improving right ventricular function.

This study reinforces the prognostic value of MMS in assessing clot burden and its impact on right ventricular function in acute PTE patients. By integrating MMS into standard diagnostic protocols, clinicians can enhance risk stratification, improve patient outcomes, and refine therapeutic decision-making. Future studies should build upon these findings to develop more precise and individualized management strategies for patients with pulmonary thromboembolism.

LIMITATIONS

While the study provides valuable insights into the relationship between clot burden and RVD in PTE patients, it has certain limitations that should be acknowledged.

Firstly, the study is limited by its relatively small sample size (n=49), which may affect the generalizability of the findings. Larger, multicenter studies are needed to validate these results and ensure broader applicability across diverse patient populations.

Secondly, the study focuses primarily on acute PTE and does not assess long-term outcomes. The lack of longitudinal follow-up data prevents evaluation of how clot burden influences chronic thromboembolic pulmonary hypertension (CTEPH) or long-term cardiac function.

Additionally, the study does not account for potential confounding factors such as comorbidities, prior anticoagulation therapy, or variations in patient management strategies. These variables could influence outcomes and should be considered in future research.

Finally, the study does not evaluate the impact of different therapeutic interventions on clot burden resolution and RVD improvement. Investigating how various treatment modalities influence MMS and cardiac function would provide more comprehensive insights into optimal management strategies.

Future Aspects

To build upon the findings of this study, future research should focus on several key areas:

1. Larger Multicenter Studies: Conducting studies with larger sample sizes across multiple institutions would enhance the generalizability of findings and provide a more comprehensive understanding of PTE severity assessment.
2. Longitudinal Follow-Up Studies: Future research should explore the long-term implications of clot burden and RVD, assessing how MMS correlates with chronic complications such as CTEPH and long-term functional outcomes.

3. Impact of Therapeutic Interventions: Investigating how various treatment strategies, including anticoagulation, thrombolysis, and mechanical thrombectomy, influence clot burden resolution and right heart function would be valuable for refining clinical guidelines.
4. Artificial Intelligence and Automated Scoring: The development of AI-driven algorithms for automated MMS calculation and RVD assessment could improve diagnostic efficiency and standardization in PTE management.
5. Biomarker-Based Risk Stratification: Exploring the role of biomarkers such as NT-proBNP, troponins, and D-dimer in conjunction with MMS could enhance risk prediction models and guide personalized treatment approaches.
6. Patient-Centered Research: Examining patient-reported outcomes, quality of life measures, and psychological impacts of PTE would provide a more holistic perspective on disease burden and treatment effectiveness.

By addressing these future aspects, research can continue to advance the understanding of PTE, refine diagnostic tools, and improve treatment strategies, ultimately leading to better patient outcomes and optimized healthcare resource utilization.

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ANNEXURE I

KAHERs JNMC
BELAGAVI
INFORMED CONSENT FORM

"N ANALOGY BETWEEN CLOT BURDEN AND RIGHT VENTRICULAR DYSFUNCTION IN ACUTE PULMONARY THROMBOEMBOLISM ON COMPUTED TOMOGRAPHY PULMONARY ANGIOGRAPHY USING MODIFIED MILLERS SCORE: ONE YEAR HOSPITAL BASED CROSS SECTIONAL STUDY"

Introduction: To determine the correlation between increasing pulmonary embolism thrombus load using modified miller's score and right ventricular (RV) dysfunction as demonstrated by CT pulmonary angiography (CTPA)

Explanation of procedure: If you agree to be part of the research study, you will be asked the relevant history and you will be subjected to relevant clinical examination and investigations.

Withdrawal from participation in the study: Participation in this study is voluntary. You will be free to decide whether to participate in this study or continue participation once enrolled. In case you decide to withdraw your participation, you are free to do so. However, please convey the decision to the principal investigator.

Possible benefits from participating in the study: You will not get any benefits by participating in this study. The data gathered will help the population at large.

Possible risks from participating in the study: There are no risks involved in participating in this study.

Privacy and confidentiality: The information collected from you will be coded, to prevent any person from identifying you. Your identity will never be revealed. The data collected from you will be kept confidential and only processed or aggregated data will be used for publication.

Financial incentives: You will not receive any payment for participating in this study.

Cost of investigations done during the course of study will be paid by the principal investigator**.**

Authorization for publication of aggregated data: Results obtained after processing of the aggregated data will be published for scientific purposes and or presented to scientific groups. However, your identity will never be revealed.

Legal rights: By signing this consent form, we are not waving any of your legal rights

CONSENT STATEMENT

I am making a voluntary decision to participate in the study "AN ANALOGY BETWEEN CLOT BURDEN AND RIGHT VENTRICULAR DYSFUNCTION IN ACUTE PULMONARY THROMBOEMBOLISM ON COMPUTED TOMOGRAPHY PULMONARY ANGIOGRAPHY USING MODIFIED MILLERS SCORE: A ONE YEAR HOSPITAL BASED CROSS SECTIONAL STUDY". My signature below indicates that I have decided to participate and I have read the information provided above or the information provided above has been read to me in the language that I understand best. I was given the opportunity to ask questions and that they have been answered to my satisfaction.

Name of the participant:

Signature or left thumb impression of the participant:

Name of the witness:

Signature or left thumb impression of the witness:

Name of the investigator:

Signature of the investigator:

ANNEXURE II

PROFORMA

TITLE:AN ANALOGY BETWEEN CLOT BURDEN AND RIGHT VENTRICULAR DYSFUNCTION IN ACUTE PULMONARY THROMBOEMBOLISM ON COMPUTED TOMOGRAPHY PULMONARY ANGIOGRAPHY USING MODIFIED MILLERS SCORE: A ONE YEAR HOSPITAL BASED CROSS SECTIONAL STUDY

CASE NO:	
PATIENT NAME:	
AGE AND SEX:	
ADDRESS:	
CONTACT NO:	
PATIENT ID:	
CT SCAN NO. AND DATE:	

CLINICAL FEATURES:

	YES	NO
SUDDEN ONSET OF CHEST PAIN		
SHORTNESS OF BREATH		
HEMOPTYSIS		
EXCESSIVE SWEATING		
HISTORY OF DVT		
COMORBIDITIES		

OTHER RELEVANT HISTORY:

BIOCHEMICAL PARAMETERS :

D-dimer

Troponin

CALCULATION OF THE MODIFIED MILLER'S SCORE ON CT PULMONARY ANGIOGRAPHY.

CTPA Finding	Score for finding
• Main pulmonary artery	16
• Right pulmonary artery	9
• Left pulmonary artery	7
• Right interlobar artery	6
• Right upper lobar artery	3
• Right middle lobar artery	2
• Right lower lobar artery	4
• Left interlobar artery	5
• Left upper lobar artery	2
• Left lower lobar artery	3
• Left lingular lobar artery	2

TOTAL CT SCORE:

Other CT findings included to demonstrate RV decompensation:

1. **RV : LV RATIO**
2. **PRESENCE OF SEPTAL CHANGES.**
3. **RATIO OF PULMONARY ARTERY TO AORTA SIZE**

2D echo parameters of right heart dysfunction are:

1. Right atrium and right ventricle dilation.
2. Systolic pulmonary artery pressure greater than 25 mm mercury.
3. Tricuspid annular plane systolic excursionless than16 mm.

CORRELATION OF MODIFIED MILLER'S SCORE ON CTPA WITH RV DECOMPENSATION FEATURES.

MODIFIED MILLER'S SCORE	RV DECOMPENSATION FEATURES ON CTPA	2D ECHO FINDINGS

4. ADDITIONAL IMAGING FINDINGS

1. CONSOLIDATION

2. PLEURAL EFFUSION

3. ELECTIONS ATELECTASIS

4. ANY OTHER

SIGNATURE OF INVESTIGATOR:

SIGNATURE OF THE GUIDE:

DATE:

ANNEXURE III:
IMAGES

Case 1 (28 in masterchart):45 year old male presented with sudden onset chest pain and breathlessness. Patient also had a history of deep vein thrombosis 2 months ago for which no treatment was taken. On clinical examination patient had tachypnea and tachycardia.

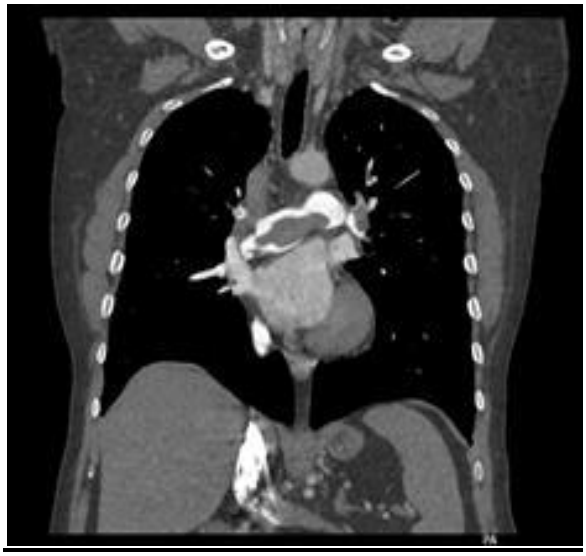

Image 1shows a thrombus in the main pulmonary artery extending into right and left pulmonary arteries with MMS score of 16.

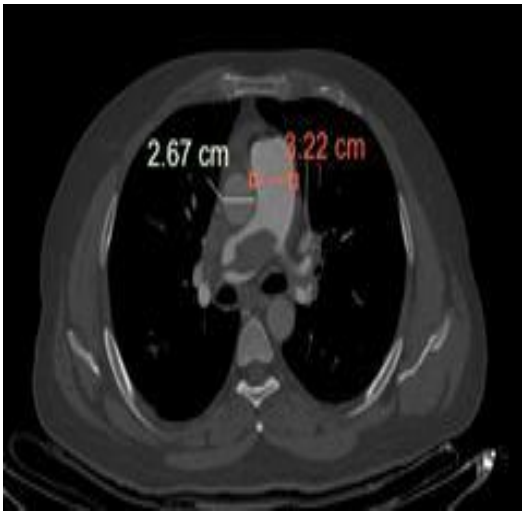

Image 2main pulmonary artery diameter is 32 mm and ascending aorta diameter is 26 mm with MPA/AA ratio more than 1

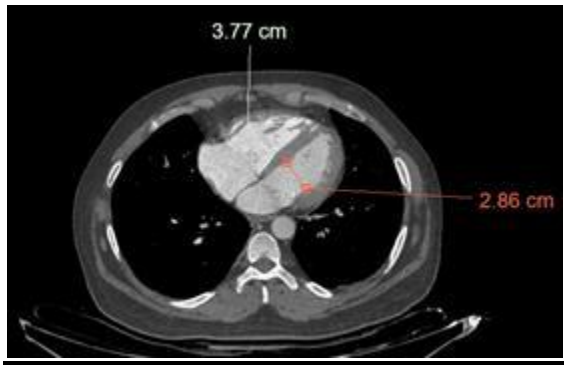

Image 3 shows dilated right ventricle 37 mm with RV:LV ratio more than 1

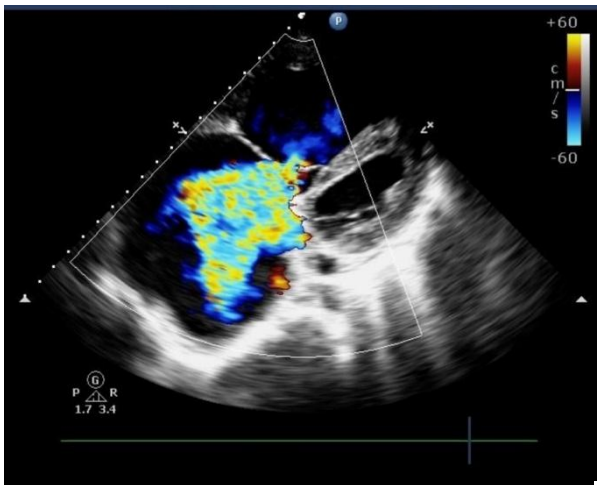

Image 4

Echocardiography of the same patient shows dilated right atrium and ventricle, TAPSE of 13 mm and an elevated PASP of 44 mm Hg.

Case 2 (38 in masterchart):48 year oldfemale presented with chest pain and breathlessness since 1 week. Patient also had a history of deep vein thrombosis for which treatment was taken. On clinical examination patient had tachycardia.

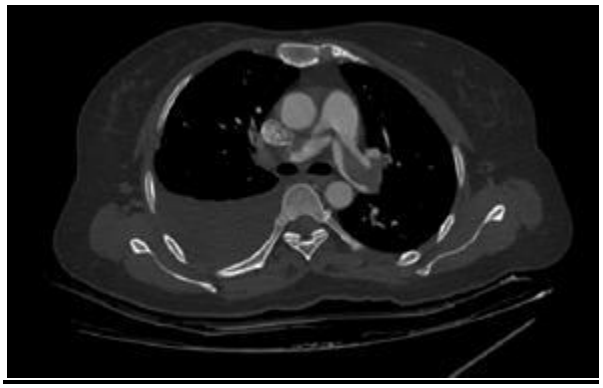

Image 5 shows extensive saddle thrombus involving main pulmonary artery with extension into right and left pulmonary arteries with associated right sided pleural effusion

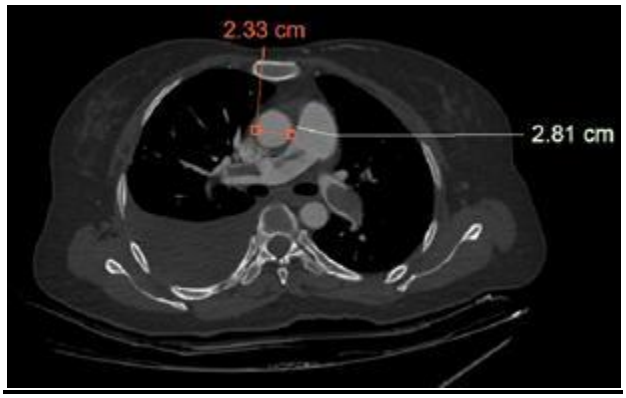

Image 6 shows the main pulmonary artery diameter is 28 mm and ascending aorta diameter is 23 mm with MPA/AA ratio more than 1

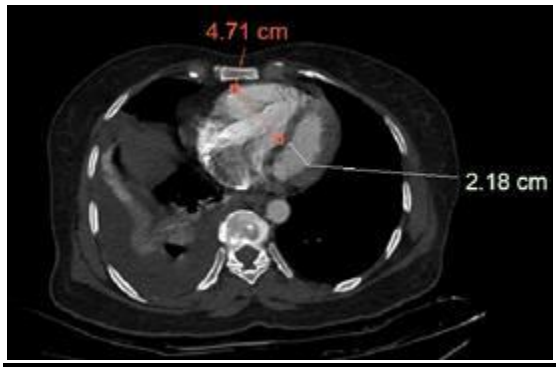

Image 7 shows dilated right ventricle 47 mm with RV:LV ratio more than 1

Case 3 (27 in masterchart):35 year old male presented with sudden onset chest pain, hemoptysis and breathlessness. Patient also had a history of deep vein thrombosis. On clinical examination patient had tachypnea and tachycardia.

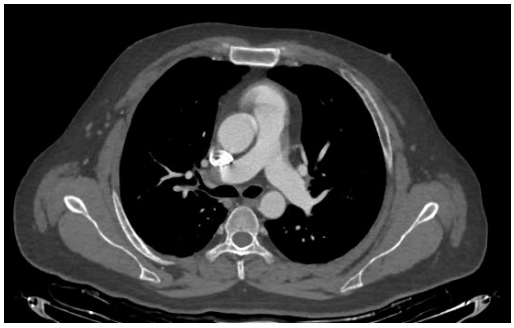

Image 8 shows thrombus involving right main pulmonary artery

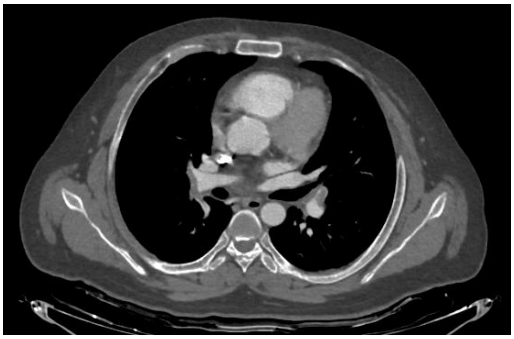

Image 9 shows thrombus involving left interlobar pulmonary artery

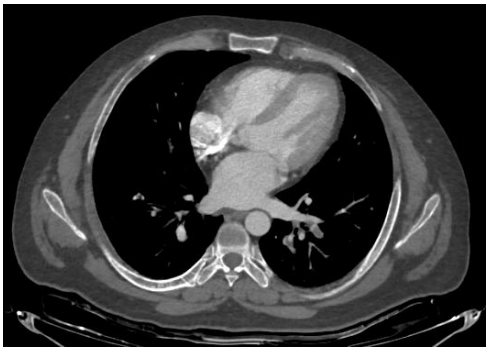

Image 10 shows thrombus involving left lower lobar segmental pulmonary arteries

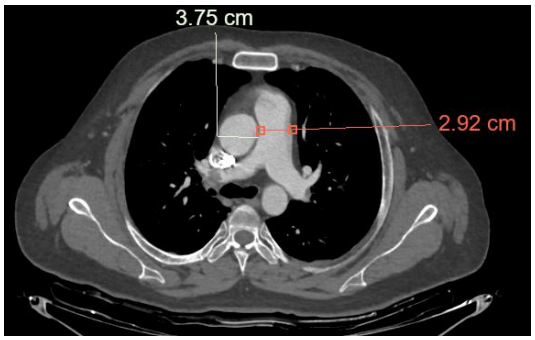

Image 11shows the main pulmonary artery diameter is 37 mm and ascending aorta diameter is 29 mm with MPA/AA ratio less than 1

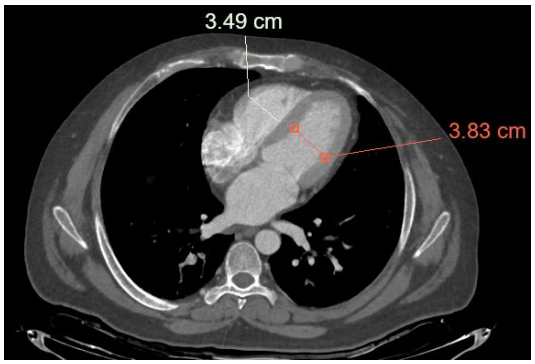

Image 12 shows right ventricle: left ventricle ratio less than 1

ANNEXURE - IV

KEY TO MASTER CHART

SYMPTOMS –

Chest pain: Present - 1, Absent - 2

Breathlessness: Present - 1, Absent - 2

Hemoptysis: Present - 1, Absent - 2

Present DVT: Yes - 1, No -2

IMAGING FINDINGS:

Consolidation : Yes - 1, No - 2

Atelectasis : Yes - 1, No - 2

Pleural effusion : Yes - 1, No - 2

RIGHT VENTRICULAR DYSFUNCTION ON CTPA:

RV:LV ratio: Increased - 1, Normal - 2

Septal changes: Present - 1, Absent - 2

MPA:AA ratio: Increased- 1, Normal - 2

RIGHT HEART STRAIN ON ECHOCARDIOGRAPHY:

RA, RV dilated: Yes - 1, No - 2

Pulmonary artery systolic pressure (PASP) more than 25 mmhg: Yes - 1, No - 2

Tricuspid annular plane systolic excursion (TAPSE) less than 16 mm: Yes - 1, No -2

RT – right, LT – left, A- artery

F-Female, M- Male

MMS – Modifed Miller score

ANNEXURE - V

MASTER CHART

SL NO	AGE	SEX	PATEINT NO	CONSOLIDATION	ATELECTASIS	PLEURAL EFFUSION	MMS	RV:LV	SEPTUM	MPA:AA	RA,RV	PASP	TAPSE	CHEST PAIN	BREATHLESSNESS	H/O DVT	HEMOPTYSIS
1	49	M	34879858	2	2	2	16	1	1	1	1	1	1	1	1	2	2
2	37	F	34589566	1	2	2	9	1	2	1	1	2	2	1	2	2	2
3	42	M	25004803	1	2	1	9	1	1	1	1	2	2	1	2	1	2
4	35	M	34567678	2	2	2	16	1	1	1	1	1	1	1	1	2	1
5	45	M	25380615	2	2	2	14	1	2	1	1	1	1	1	1	2	2
6	54	M	23578945	2	2	2	13	1	1	1	1	1	1	1	1	1	2
7	55	M	26256008	2	2	1	11	1	2	1	1	1	1	2	1	2	2
8	52	M	27658906	2	2	2	16	1	1	1	1	1	1	1	1	2	2
9	25	M	28128689	1	2	2	13	1	1	1	1	1	1	1	1	1	2
10	60	M	27894590	2	2	2	7	2	2	2	2	2	2	2	1	2	1
11	75	M	28562955	2	2	2	14	1	2	1	1	1	2	1	1	2	2
12	38	M	28745596	2	2	2	7	2	2	2	2	2	2	2	1	1	1
13	45	M	29038095	2	2	2	11	2	2	2	2	2	2	1	1	2	2
14	75	M	29070863	1	2	1	16	1	2	2	1	2	1	1	1	2	2
15	31	M	29221886	2	2	2	4	2	2	2	2	2	2	2	1	1	2
16	32	M	29287260	2	2	2	16	1	1	1	1	1	1	1	1	2	2
17	42	M	29861909	2	2	2	9	1	2	1	1	1	1	1	1	2	2
18	32	M	29244956	2	2	2	7	2	2	2	2	2	2	2	1	1	2
19	65	M	30025249	1	2	1	14	1	2	2	1	2	2	1	1	1	1
20	71	F	30102981	2	2	1	16	1	1	1	1	1	1	1	1	1	2
21	30	M	30128428	1	2	2	4	1	1	1	1	2	2	1	1	2	1
22	29	M	30216743	1	2	2	16	1	2	1	1	1	1	1	1	2	2
23	44	M	30457854	2	2	1	16	1	1	1	1	1	1	1	1	1	2
24	38	M	31741431	1	2	2	9	1	2	1	1	2	2	1	2	1	2
25	65	M	34879054	2	2	1	13	1	1	1	1	1	1	1	1	1	2
26	31	M	32170734	1	2	1	3	2	2	2	2	2	2	1	1	2	2
27	35	M	32299846	2	2	2	14	2	2	2	2	2	2	1	1	1	1
28	45	M	32367433	2	2	2	16	1	1	1	1	1	1	1	1	1	2
29	59	F	32591545	2	2	1	16	1	1	1	1	1	1	1	1	2	2
30	72	F	33467541	2	2	2	16	1	1	1	1	1	1	1	1	1	1
31	44	M	32936290	1	2	2	12	1	2	1	1	2	2	1	1	2	2
32	82	F	33456731	1	2	2	7	2	2	2	2	2	2	2	1	2	2
33	29	M	33268366	1	2	1	9	1	2	1	1	2	2	1	2	2	2
34	32	M	33697947	2	2	2	7	2	2	2	2	2	2	1	1	1	2
35	56	M	33817740	2	2	2	6	2	2	2	2	2	2	1	1	2	2
36	72	M	33925350	1	2	1	14	2	2	2	2	2	2	1	1	2	2
37	28	M	34078698	2	2	2	16	1	2	1	1	1	2	2	1	1	2
38	48	F	34301787	1	2	1	16	1	1	1	1	1	1	1	1	1	2
39	84	F	34300381	1	2	1	16	1	2	1	1	1	1	1	1	2	2
40	68	F	34126060	2	2	1	9	2	2	2	2	2	2	1	1	2	2
41	46	M	34323072	2	2	2	7	2	2	2	2	2	2	2	1	1	2
42	46	F	34567821	2	2	2	5	2	2	2	2	2	2	2	1	2	2
43	46	F	34562467	2	1	2	13	1	1	1	1	1	1	1	1	1	2
44	47	M	37685341	2	2	2	16	1	1	1	1	1	1	1	1	1	1
45	78	M	39092002	2	2	2	16	1	2	1	1	2	1	2	1	1	2
46	37	M	40920082	2	2	2	16	1	2	1	1	2	1	1	1	1	1
47	38	M	40902002	1	2	2	12	1	1	1	1	1	1	1	1	1	2
48	41	M	41092020	1	2	2	12	1	2	1	1	1	1	1	1	2	2
49	66	F	40988202	1	2	2	4	2	2	2	2	2	2	1	2	2	2