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Assessing the severity of acute pulmonary embolism: A review of epidemiological, clinical, biological, and CT angiography parameters

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ABSTRACT

Pulmonary embolism (PE) presents with diverse clinical manifestations, which often do not correlate with the size of the clot. Common symptoms such as dyspnea and pleuritic chest pain are frequent but nonspecific, complicating clinical diagnosis. This review aims to assess the severity of acute pulmonary embolism by examining epidemiological, clinical, biological, and computed tomography (CT) angiography parameters to improve diagnostic accuracy and prognostic evaluation. Clinical diagnostic scores and electrocardiogram (ECG) findings are supplemented by D-Dimer assays in cases with low or intermediate clinical probability. Chest X-rays are recommended urgently at the bedside to exclude alternative causes of dyspnea and to assess suitability for ventilation/perfusion scans. Transthoracic cardiac Doppler ultrasound evaluates hemodynamic impact. Definitive diagnosis is established by chest CT angiography, which has largely replaced traditional angiography and lung scintigraphy. D-Dimer testing demonstrates a high negative predictive value (~95%) when clinical probability is low or intermediate, effectively ruling out venous thromboembolism (VTE) when negative. Chest X-rays may appear normal within 24 hours or show signs such as moderate pleural effusion, band atelectasis, or pulmonary infarction evidenced by Hampton's hump. Cardiac Doppler ultrasound reveals signs indicative of PE including right ventricular dilation (>25 mm), interventricular septum displacement, and pulmonary artery trunk enlargement. CT angiography identifies acute PE by detecting central intravascular hypodensities with iodinated contrast, often forming acute angles with vessel walls. The integration of clinical, biological, and imaging parameters, particularly CT angiography, enhances the accuracy of acute PE diagnosis and assessment of severity. Prompt bedside imaging and ultrasound provide critical information to differentiate PE from other severe conditions. CT angiography remains the gold standard for confirming clinical suspicion and guiding urgent management.

INTRODUCTION

Pulmonary embolism (PE) is a serious and potentially life-threatening condition characterized by the obstruction of one or more pulmonary arteries. While most commonly caused by a fibrinocruoric thrombus, rarer etiologies include tumor cells, septic emboli, fat, amniotic fluid, parasites, or foreign bodies. PE is a critical component of venous thromboembolism (VTE), a broader clinical entity that also encompasses deep vein thrombosis (DVT).

The global burden of acute pulmonary embolism is substantial. In the United States, the annual incidence is estimated at 69 per 100,000 individuals, with an associated in-hospital mortality rate ranging from 8% to 10% (Sanchez et al., 2011). This mortality escalates significantly with increasing disease severity: it reaches 16% to 22% when vascular obstruction exceeds 50% or is accompanied by pulmonary arterial hypertension, 25% to 32% in cases of cardiogenic shock, and a dire 65% when circulatory arrest occurs (Sanchez et al., 2011). While clinically severe forms, characterized by hypotension or signs of peripheral shock, have a lower incidence (around 4.2%), their rapid identification is crucial for immediate diagnostic workup and aggressive management, often involving thrombolysis or, less commonly, surgical embolectomy.

For patients without overt shock, the presence of right ventricular dysfunction and/or elevated biomarkers, such as brain natriuretic peptide (BNP), indicates a higher risk of clinical deterioration. Consequently, comprehensive risk stratification and severity assessment are paramount in acute PE (Torbicki et al., 2008). This stratification aims to distinguish high-risk patients who warrant close monitoring and potentially thrombolytic therapy from low-risk individuals who might be safely managed in an outpatient setting (Torbicki et al., 2008). Therefore, establishing a definitive diagnosis is a prerequisite for initiating appropriate treatment and risk-adapted management.

Several studies, both from Western countries (Alotaibi et al., 2016; Elias et al., 2015; Jiménez et al., 2010) and the sub-region (Ceriani et al., 2010; Doolittle et al., 2019; Jiménez et al., 2016), consistently report that multi-slice thoracic CT angiography (CTPA) is the preferred imaging modality for confirming the diagnosis of PE. Given the importance of accurate severity assessment in guiding clinical decisions and improving patient outcomes, this article aims to systematically review the current literature on the epidemiological, clinical, biological, and CT angiography parameters used for assessing the severity of acute pulmonary embolism.

DIAGNOSIS OF PULMONARY EMBOLISM

Clinical Diagnosis of Pulmonary Embolism

Pulmonary embolism has many clinical presentations, and its symptoms do not correlate with the size of the clot. In addition, they are not very specific: dyspnea and pleural-type chest pain are the most common manifestations of PE. However, cough, hemoptysis, and pain and/or edema of the lower limbs may also occur when PE is associated with DVT. The physical signs observed are also nonspecific: tachycardia, rales, gallop rhythm, accentuation of the pulmonary component of the second heart sound (B2), and signs of deep vein thrombosis. Signs of right heart failure such as jugular venous distension, tachycardia, and hepatojugular reflux can also be present. Occasionally, PE can be asymptomatic. For these reasons, incidental pulmonary embolism is sometimes diagnosed on CT scans (Douma et al., 2011; Kohn et al., 2012; Thieme et al., 2012).

To assess for massive PE, blood pressure (systolic and diastolic) is measured, and clinical signs of peripheral shock—such as mottling, cold extremities, and signs of low cerebral perfusion (confusion, impaired alertness)—are sought. Unfortunately, deaths from PE often occur in patients with atypical clinical presentations.

The combination of clinical features allows the identification of three presentations highly suggestive of PE:

- Acute dyspnea of sudden onset with a normal chest X-ray, a gas exchange abnormality (hypoxia and hypocapnia), and possible signs of right heart failure;
- Pulmonary infarction, characterized by lower pleuritic chest pain, possibly accompanied by hemoptysis, gas exchange abnormalities, and radiological abnormalities such as pleural effusion, band atelectasis, or diaphragmatic elevation;
- A state of shock with acute right heart failure, indicative of massive PE.

Serious signs of PE essentially correspond to clinical signs of acute cor pulmonale and indicate massive pulmonary embolism, including tachycardia, hepatojugular reflux, jugular venous distension, arterial hypotension, peripheral shock signs, repolarization disorders, right bundle branch block, and P wave abnormalities. Recognizing these signs enables emergency diagnostic and therapeutic management to exclude massive PE.

Due to the low specificity of symptoms, many differential diagnoses exist initially: asthma attack, pneumonia, bronchial cancer, esophageal spasm, anxiety attack, heart failure, myocardial infarction, aortic dissection, among others. This complicates the clinical diagnosis of PE. Hence, diagnostic strategies and clinical scores such as the revised Geneva score and the Wells score have been developed to establish clinical probability and facilitate diagnosis (Wells et al., 2000).

Table 1:
Comparison of Major Clinical Prediction Rules for Acute Pulmonary Embolism

Score	Parameters Included	Risk Categories	Clinical Use
Wells Score	Clinical signs of DVT, PE most likely diagnosis, HR >100 bpm, immobilization/surgery, previous DVT/PE, hemoptysis, malignancy	High (≥7), Intermediate (2–6), Low (≤1)	Widely used for initial risk estimation and pre-test probability assessment
Revised Geneva Score	Age >65, previous DVT/PE, surgery/fracture, active cancer, unilateral leg pain, hemoptysis, HR, pain on lower limb palpation	High (>11), Intermediate (4–10), Low (0–3)	Objective, no physician judgment required
PESI	Age, male sex, cancer, heart failure, chronic lung disease, vital signs (BP, HR, RR, Temp, SpO_2), altered mental status	Class I–V (I = lowest, V = highest mortality)	Stratifies 30-day mortality risk; validated for inpatient/outpatient decisions
sPESI	Age >80, cancer, chronic heart/lung disease, HR ≥110 bpm, SBP <100 mmHg, SpO_2 <90%	0 = low risk, ≥1 = higher risk	Simpler version; predicts short-term mortality; useful for outpatient selection

Biological Diagnosis of Pulmonary Embolism

The accuracy of clinical diagnostic scores and ECG is improved by D-Dimer assays in cases of low or intermediate probability, as shown in Carrier's meta-analysis of 5,000 patients (Jardin et al., 1987). This study found a PE prevalence of 0.14% (95% confidence interval [CI], 0.05–0.41) in patients with low clinical probability and normal D-Dimer levels.

D-Dimers have a high negative predictive value (~95%) when clinical probability is low or intermediate, effectively ruling out VTE when negative. However, their specificity is low, as levels can increase in inflammatory syndromes, progressive cancer, and with age. Thus, their utility is limited in patients with inflammatory conditions, cancer, and the elderly. Troponin may be positive in PE due to right ventricular ischemia, and brain natriuretic peptide (BNP) may rise due to right ventricular dilation. Arterial blood gas analysis often shows hypoxia and hypocapnia but may be normal, especially in young patients without cardiopulmonary history (Victor et al., 2008).

Radiographic Diagnosis

Chest X-rays should be performed urgently, preferably at the patient's bedside. Their primary role is to exclude other causes of dyspnea (e.g., infectious pneumonia, pneumothorax, pulmonary edema) and to assess suitability for ventilation/perfusion scans. Chest X-rays are normal in approximately 20% of PE cases.

Within the first 24 hours, chest X-rays may reveal right heart enlargement with dilation of the right atrium and ventricle. Signs of the rare Westermark triad may appear: dilation of the pulmonary artery proximal to the obstruction with large, dense unilateral or bilateral hila, and decreased distal pulmonary vascularity causing hyperlucency, along with elevation of the ipsilateral diaphragm (Fraser et al., 1999).

After 24 hours, chest X-rays may remain normal or show moderate pleural effusion such as blunting of a pleural recess, band atelectasis, or pulmonary infarction characterized by a triangular opacity with a pleural base and a hilar apex (Hampton's hump). This lesion typically occurs in the lower lobes, especially on the right, near the costodiaphragmatic angle. The opacity varies in size and shape, may be excavated (with or without infection), and may be single or multiple, unilateral or bilateral. Its evolution often involves concentric regression, sometimes leaving linear scarring 4–6 cm long. Pleural sequelae such as sinus filling or dome attraction may also occur. Rarely, arterial thrombosis may calcify, resulting in calcifications molding the lumen of a pulmonary artery or bifurcation.

Ultrasound Diagnosis

Transthoracic cardiac Doppler ultrasound is a simple examination that can easily be performed at the bedside of an intensive care patient. This non-invasive examination allows estimation of the hemodynamic impact of pulmonary embolism. Signs suggestive of pulmonary embolism include dilation of the right ventricle with a diameter greater than 25 mm, displacement of the interventricular septum towards the left ventricle, and dilation of the pulmonary artery trunk. These signs correspond to acute cor pulmonale, observed with 40 to 50% obstruction. A right ventricle/left ventricle ratio greater than 0.6 is associated with massive pulmonary embolism in almost all cases (Jardin et al., 1987).

The advantage of transthoracic ultrasound is its rapid ability to differentiate pulmonary embolism from other serious conditions such as cardiac tamponade, aortic dissection, and cardiogenic shock. It can exceptionally visualize thrombi, particularly in the right heart chambers, in approximately 10% of cases. However, a normal ultrasound result does not exclude pulmonary embolism. Transesophageal ultrasound allows direct visualization of thrombi within the pulmonary arteries (Fraser et al., 1999). However, the sensitivity of transthoracic ultrasound is limited to 50–60%, with a specificity of 88–100%. Additionally, the risk of clinical deterioration in patients with severe pulmonary embolism limits its routine use in current practice.

CT Diagnosis

The definitive diagnosis of pulmonary embolism currently relies on chest CT angiography, which is the first imaging examination performed in hemodynamically stable patients with suspected PE (Stein et al., 2006; Nonent et al., 2009). This examination has largely replaced angiography and ventilation/perfusion lung scintigraphy (Ferretti et al., 2005). It is rapid, sensitive, specific, and widely accessible.

CT angiography allows confirmation or exclusion of clinical suspicion in an emergency, with even faster results in cases of pregnancy, contraindication to anticoagulants, heparin-induced thrombocytopenia, or massive PE. CT also benefits from a high negative predictive value, particularly when clinical suspicion is low or intermediate.

The validation of CT angiography for PE diagnosis was demonstrated in 2009 by the PIOPED II study, which established the test's intrinsic characteristics: sensitivity of 83% and specificity of 96% (Maitre et al., 2008). Signs of acute pulmonary embolism on CT include central intravascular hypodensities centered by iodinated contrast material or forming an acute angle with the vessel wall.

The affected vessel may be partially or completely obstructed and may or may not be enlarged. Diagnosis is aided by the presence of complications directly related to obstruction, such as pulmonary infarction. The impact of CT in PE management, introduced by PIOPED II, has been confirmed by several studies, including those by Perrier et al. (2005) and Righini et al. (2008). The latter randomized study showed that performing venous Doppler ultrasound in addition to chest CT angiography did not improve diagnostic performance for PE.

CT not only allows positive diagnosis of PE through direct visualization of the clot but also enables evaluation of severity, assessment of underlying pulmonary parenchymal quality, and identification of differential diagnoses responsible for acute chest pain (Ghaye et al., 2002).

PROGNOSTIC CRITERIA FOR PULMONARY EMBOLISM

Why Stratify the Prognostic Risk of Pulmonary Embolism?

Estimating prognosis using objective criteria allows assessment of the progressive risk for a given patient. Indeed, once the diagnosis has been made, not all patients have the same risk of mortality and complications. It is therefore recommended to stratify the prognostic risk of PE for each patient. This aims to guide patients towards the most appropriate therapeutic strategies within an optimally organised care framework, improving quality of life and life expectancy while conserving healthcare resources.

The benefits of severity markers are twofold: selecting patients whose low risk of complications allows outpatient treatment, and selecting patients whose high progressive risk justifies more aggressive therapy or close monitoring. The therapeutic decision must also consider comorbidities, bleeding risk, social circumstances, the patient's degree of autonomy and understanding, geographical distance from

healthcare facilities, the feasibility of monitoring coagulation during the transition to vitamin K antagonists, possible psychiatric conditions, and patient compliance.

Numerous prognostic scores have been developed in recent years—clinical, biological, radiological, or combined—to stratify prognostic risk. However, none of these scores is yet sufficiently specific or sensitive, although some have been validated by large studies.

CLINICAL CRITERIA

Prognosis Linked to Hemodynamic Status

Simple clinical data analysis currently distinguishes low-risk pulmonary embolisms (non-massive PE) from massive embolisms complicated by shock, which have a much poorer prognosis and require more aggressive treatment than anticoagulation alone. Massive PE or high mortality risk is defined by shock characterized by arterial hypotension below 100 mmHg persisting for more than 15 minutes or by signs of peripheral hypoperfusion requiring catecholamine administration (Torbicki et al., 2008).

Prognosis Linked to the Underlying Condition

The underlying condition has also been implicated in early PE mortality, as it causes difficulties adapting to sudden increases in right ventricular (RV) afterload. Mortality increases with the presence of right ventricular dysfunction and is much higher in patients with massive PE than in those with non-massive PE (Kasper et al., 1997; Ribeiro et al., 1997). The underlying pathologies most correlated with PE mortality include chronic respiratory failure, congestive heart failure, cancer, and age over 70 years (Kline et al., 2003).

Other Clinical Prognostic Criteria

Other studies show the prognostic value of measuring arterial oxygen saturation (SaO2). Among 207 patients without arterial hypotension, mortality was 20% when SaO2 was less than 95%, compared to only 2% when SaO2 was 95% or higher.

Clinical or Combined Prognostic Scores

Table 2:
Key Biomarkers and Their Prognostic Role in Acute Pulmonary Embolism

Biomarker	Function	Prognostic Significance	Limitations
D-dimer	Fibrin degradation product	High sensitivity; useful for ruling out PE in low-risk patients	Low specificity, especially in elderly or inflammatory states
Troponins (I/T)	Marker of myocardial injury	Elevated levels associated with RV dysfunction and higher mortality	Not PE-specific; may be elevated in MI, CHF, or renal failure
BNP / NT-proBNP	Indicates right ventricular strain	Elevated levels correlate with increased short-term mortality and ICU need	Influenced by age, renal function, and heart failure
H-FABP	Cytosolic protein from heart muscle	Early predictor of RV dysfunction and poor outcome	Limited availability; less studied in PE context
Lactate	Marker of tissue hypoperfusion	High levels associated with shock and poor prognosis	Non-specific; elevated in sepsis, trauma, etc.

The Geneva Prognostic Score

The Geneva Prognostic Score (GPS) stratifies patients according to their risk of adverse events, with a 3-month composite endpoint including death, major rebleeding, and symptomatic thromboembolic recurrence (Carson et al., 1992; Wicki et al., 2000). This score combines clinical, biological, and radiological criteria to assign each patient a severity score from 0 to 8.

Beer et al. (2003) demonstrated that patients with a GPS less than 3 could be treated as outpatients without loss of safety. Indeed, patients scoring less than 3 (66% of PE cases) had a 3-month adverse event risk of 2.2% compared to 26.1% in patients scoring 3 or higher. A score above 2 points had 85% sensitivity, 73% specificity, and a 98% negative predictive value for complications within three months (Ribeiro et al., 1997). External validation studies have confirmed the GPS's performance in different populations.

The PESI Scores

Another severity score based exclusively on clinical elements was developed by Aujesky, with 30-day mortality as the endpoint: the Pulmonary Embolism Severity Index (PESI). It was developed retrospectively from a large North American hospital registry and underwent internal validation as well as external validations in various independent cohorts, particularly in Europe (Donzé et al., 2008; Aujesky et al., 2005). The score increases with age,

male gender, number of comorbidities, and clinical abnormalities. Patients are divided into five classes, with mortality increasing from class I to V: approximately 2.5% in classes I and II, 7% in class III, 11% in class IV, and 24% in class V. Notably, low mortality is associated with groups I and II, which account for about 40% of patients. Patients at low risk of mortality are those without significant comorbidity or right heart dysfunction. These patients are effectively identified using the PESI score (class I or II, score ≤ 85) and could be candidates for outpatient care. The PESI score is therefore a useful tool to identify patients at lower risk of mortality who may be treated as outpatients and to make appropriate and objective hospitalization decisions (Donzé et al., 2008).

Aujesky et al. (2006) identified 11 characteristics independently associated with the risk of 30-day mortality from PE: two general characteristics (age and male sex), three clinical comorbidities (presence of cancer, chronic respiratory failure, or chronic heart failure), and six clinical factors (heart rate ≥110/min, systolic blood pressure <100 mmHg, respiratory rate ≥30/min, temperature <36°C, oxygen saturation <90%, altered mental status). These characteristics were combined to form the PESI score (Aujesky et al., 2006).

Scannographic Prognostic Criteria

The latest European recommendations highlight spiral pulmonary angiography not only for the diagnosis but also for the prognostic assessment of PE (Torbicki et al., 2008). Advances in imaging now allow visualization of anomalies such as dilatation of the right heart chambers and paradoxical septal motion on multi-slice CT scanners. These findings have prognostic value.

Valvular flows, typically evaluated by ultrasound and not directly studied on CT angiography (especially tricuspid insufficiency), currently have limited prognostic use in PE. Thus, ultrasound parameters for PE prognosis can technically be replaced by CT studies, which indirectly evaluate right ventricular dysfunction by measuring the right ventricle to left ventricle (RV/LV) ratio, presence or absence of paradoxical septal motion, and reflux of contrast into the inferior vena cava (IVC) and superior hepatic veins (SHV). This prognostic evaluation is particularly interesting because CT angiography is the reference examination for PE diagnosis, performed on all patients except those with contraindications.

Schoepf et al. (2004) confirmed the value of the RV/LV ratio measured on CT as a predictor of mortality.

Table 3:
Key Imaging Signs of Severe Pulmonary Embolism on CTPA

CT Finding	Description	Associated Risk/Significance	Notes
Right-to-left ventricular ratio >1	RV diameter > LV diameter on axial or 4-chamber views	Marker of RV strain; associated with higher mortality	Most widely used radiologic severity indicator
Contrast reflux into IVC/hepatic veins	Retrograde opacification during pulmonary contrast injection	Suggestive of increased RV pressure and acute cor pulmonale	Supportive but not specific
Septal bowing toward LV	Interventricular septum shifts toward the left ventricle	Indicates elevated RV pressure	Also seen in chronic pulmonary hypertension
Pulmonary artery obstruction index (Qanadli)	Quantifies clot burden by segmental obstruction score	Correlates with clinical severity and mortality in some studies	Mostly research use
Main pulmonary artery enlargement	Diameter >29 mm	May reflect chronic hypertension or acute overload	Non-specific
Saddle embolus	Thrombus straddling main pulmonary artery bifurcation	Often associated with large clot burden and RV dysfunction	Not always clinically severe

The Degree of Vascular Obstruction as a Prognostic Marker

Qanadli et al. (2001) created a CT-adapted score where a PE was considered severe when the arterial obstruction index exceeded 40%. This index is based on clot location and the degree of arterial blockage. The score was shown to be reproducible and highly correlated with the angiographic obstruction score used before CT became the diagnostic gold standard for PE (Qanadli et al., 2001).

Signs of Right Ventricular Dysfunction on CT as Prognostic Markers

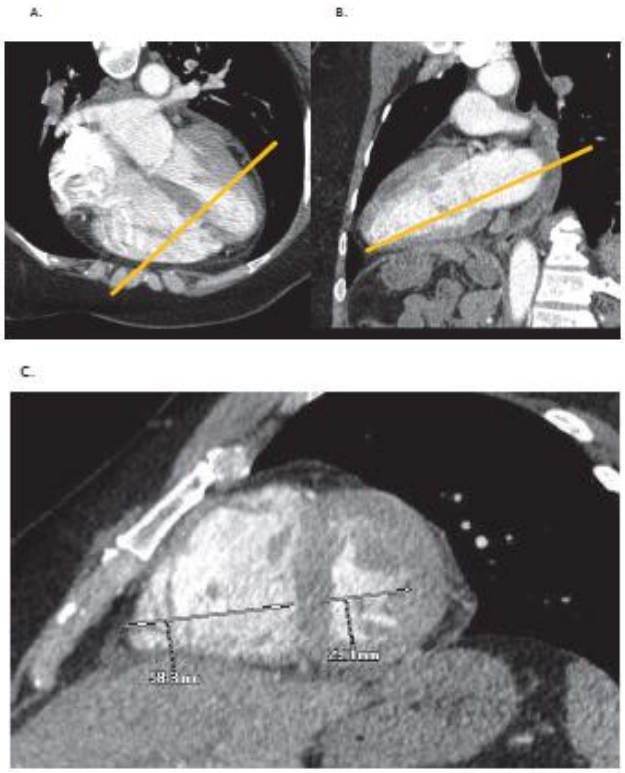
Signs of right ventricular dysfunction validated on echocardiography are also prognostically relevant on CT imaging. Since CT is the gold standard for PE diagnosis, a single examination can simultaneously diagnose and stratify prognostic risk. Indirect evaluation of right heart function includes measuring the RV/LV diameter ratio on short-axis views, assessing the interventricular septum position, detecting contrast reflux into the IVC or SHV, and

measuring pulmonary artery size. Direct signs include pulmonary infarction and the vascular obstruction index, all of which can be readily assessed on CT angiography.

Key signs indicating acute cor pulmonale include:

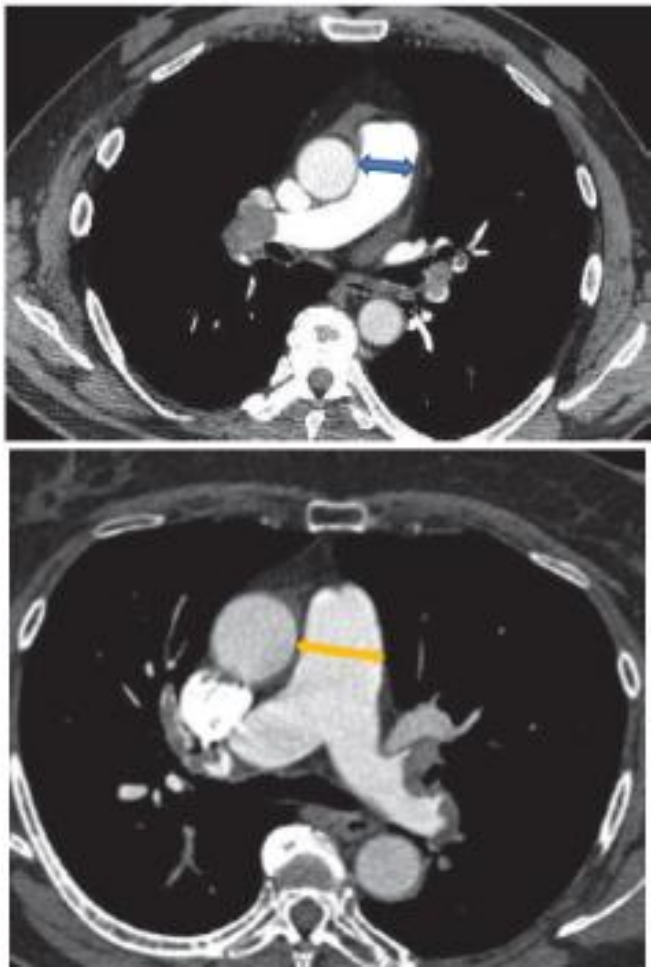
- **RV/LV ratio:** Measured between the internal wall of the free RV edge and the interventricular septum at the distal two-thirds junction on short-axis multiplanar reconstructions. An RV/LV ratio >1 suggests right ventricular dysfunction.

Figure 1:
CT Scan Measurement of the Small Diameters of the Right and Left Ventricles



- **Pulmonary artery trunk diameter:** Measured axially just before bifurcation. A diameter >29 mm is considered pathological.

Figure 2:
Pulmonary Artery Diameter on an Axial View of a CT Angiogram



- **Interventricular septum shape:** Classified as normal (not deviated) or deviated. A deviated septum may be rectilinear or bulging into the left ventricle (paradoxical septal motion).
- **Contrast reflux into the IVC or SHV:** A qualitative sign indicating right ventricular overload.

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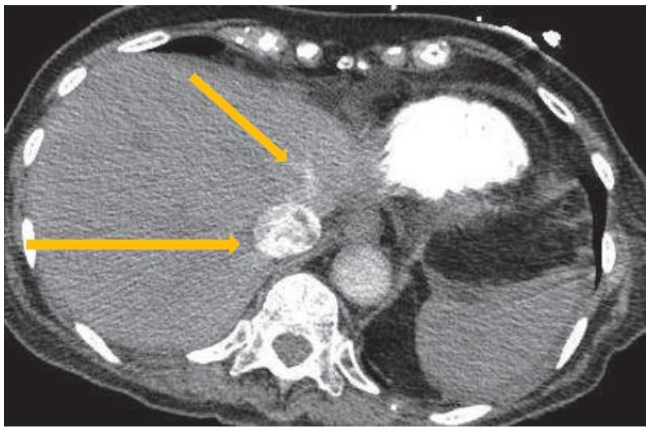
Figure 3:
Reflux of Contrast Agent into the IVC and SVC (the left SVC is visualised here)



- **The embolic load**: The embolic load is measured using the Qanadli index, expressed as a percentage. The pulmonary arterial tree of each lung consists of 10 segmental arteries (3 upper lobe, 2 middle lobe or lingula, 5 lower lobe). The presence of an embolus in a segmental artery scores 1 point; emboli in proximal arteries score according to the number of downstream segmental arteries affected. A weighting factor is applied: 0 for no defect, 1 for partial occlusion, and 2 for complete occlusion.

  The formula:

  $$Score = \frac{(2xn) + (1xn) + (0xn)x100}{40}$$

  Where

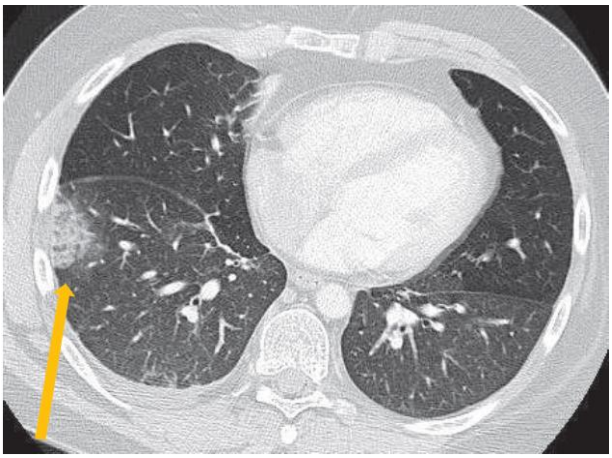
  n = number of segmental branches concerned
  2 = complete occlusion
  1 = partial occlusion
  0 = no occlusion

  Presence of pulmonary infarction is noted when pulmonary consolidation with a pleural base occurs in a vascular territory affected by PE.

  The maximum score is 40 (2 points per segment in cases of complete occlusion, with a total of 20 segments), which corresponds to an obstruction index of 100%.

- **Pulmonary infarction (presence or absence):** When pulmonary consolidation with a pleural base was present in a vascular territory affected by pulmonary embolism, the scan was considered positive for infarction **(Figure 4)**.

Figure 4:
A 23-year-old patient with right lower lobar pulmonary embolism (Qanadli score: 5%) and pulmonary infarction in the corresponding territory (arrow).



Evaluation of image quality (according to Nazaroglu criteria): Density measurement of the pulmonary artery trunk was classified into one of three groups:

- **≤199 HU**: suboptimal enhancement; limited image quality
- **200–249 HU**: acceptable enhancement; good image quality
- **≥250 HU**: excellent enhancement; excellent image quality

CONCLUSION

Thoracic CT angiography is a comprehensive examination enabling both diagnostic and prognostic assessment in a single step. It effectively distinguishes hemodynamically severe from non-severe PE, correlating well with clinical findings. In our centre, three markers of hemodynamic severity stand out: the Qanadli vascular obstruction score, the RV/LV ratio, and interventricular septal deviation. In contrast, pulmonary artery diameter, coexistence of DVT, reflux of contrast in the IVC, and presence of pulmonary infarction were not correlated with hemodynamic severity of PE.

Abbreviations

ACFA: Cardiac arrhythmia due to atrial fibrillation
AP: Pulmonary artery

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AVC: Cerebrovascular accident
AVK: Anti-vitamin K
BB: Branch block
COPD: Chronic obstructive pulmonary disease
CPC: Chronic cor pulmonale
CRP: C-reactive protein
DRA: Acute respiratory distress
ECG: Electrocardiogram
ESC: European Society of Cardiology
PE: Pulmonary embolism
ETT: Transthoracic ultrasound
HR: Heart rate
FDR: Risk factor
EN: Respiratory rate
LMWH: Low molecular weight heparin
UFH: Unfractionated heparin
PH: Pulmonary hypertension
HVD: Right ventricular hypertrophy
MI: Myocardial infarction
IC: Right heart failure
INR: International Normalized Ratio
IR: Renal failure
MI: Lower limb
VTE: Venous thromboembolism
CBC: Blood count
SBP: Systolic blood pressure
PEC: Support
PESI: Pulmonary Embolism Severity Index
ILD: Diffuse interstitial pneumonia
SAO2: Arterial oxygen saturation
TBK: Tuberculosis
APTT: Activated partial thromboplastin time
HIT: Heparin-induced thrombocytopenia
PT: Prothrombin level
DVT: Deep vein thrombosis
IVC: Inferior vena cava
RV: Right ventricle
LV: Left ventricle

Ethical Approval: Nil applicable.

Conflicts of Interest: None declared.

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