CASE REPORT

Spectrum of Acute Pulmonary Embolism: A Case Series Highlighting Diagnostic Challenges, Risk Stratification, and Thrombolysis in Complex Clinical Scenarios – Implications for Prevention and Long-Term Managements

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ABSTRACT

Background: Acute pulmonary embolism (PE) is a potentially fatal manifestation of venous thromboembolism (VTE) with highly variable presentations, from subtle mimics to life-threatening obstructive shock. Challenges intensify in atypical groups such as those with congenital heart disease or pre-existing pulmonary hypertension.
Methods: Retrospective descriptive case series of three consecutive adult patients managed in a tertiary emergency department from December 2024 to 2025. Evaluation included clinical probability scoring (Wells, Revised Geneva), biomarkers (D-dimer, troponin, NT-proBNP), point-of-care ultrasound (POCUS), echocardiography, and confirmatory CT pulmonary angiography (CTPA). Thrombolysis decisions were physiology-based, prioritizing hemodynamic impact.
Results: Three patients with distinct clinical phenotypes of acute pulmonary embolism were successfully managed. The first case involved a 40-year-old male presenting with massive PE complicated by obstructive shock, in whom administration of tenecteplase (40 mg bolus) resulted in rapid hemodynamic stabilization. The second case was a 37-year-old male with submassive PE that clinically mimicked pneumonia, presenting with hemoptysis and radiological consolidation; further evaluation revealed elevated Factor VIII levels (191%), suggesting an underlying hypercoagulable state. He was treated with alteplase (100 mg over 2 hours) in addition to antibiotics, leading to complete clinical recovery. The third case involved a 60-year-old male with an unrepaired atrial septal defect (ASD) and severe pulmonary arterial hypertension (PAH), in whom acute PE acted as a "second-hit," precipitating significant hemodynamic compromise. Despite the presence of hemoptysis, systemic thrombolysis with alteplase was administered following careful risk–benefit assessment, resulting in full recovery. All patients were subsequently transitioned to oral anticoagulation with rivaroxaban and discharged with cardiology follow-up for long-term management.
Conclusion: High-index suspicion, multimodal risk stratification, prompt CTPA, and individualized thrombolysis (even overriding relative contraindications in select high-risk cases) are essential for survival. Long-term care must emphasize guideline-directed secondary prevention with direct oral anticoagulants (DOACs), early ambulation, and lifestyle modification to minimize recurrence.

Keywords: Pulmonary embolism, thrombolysis, CTPA, Wells score, pulmonary hypertension, atrial septal defect, DOAC, VTE prevention, Factor VIII thrombophilia

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INTRODUCTION

Pulmonary embolism (PE) arises from embolization of thrombus—predominantly from lower-extremity deep vein thrombosis (DVT)—into the pulmonary arteries, resulting in ventilation–perfusion mismatch, hypoxemia, elevated pulmonary vascular resistance, and right ventricular (RV) strain. Severe cases culminate in acute RV failure, obstructive shock, and high mortality if untreated [1].Despite diagnostic advances including multidetector CTPA and biomarker integration, PE remains underdiagnosed owing to nonspecific symptoms overlapping with pneumonia, acute coronary syndrome, exacerbation of chronic lung disease, or heart failure. Risk stratification tools (Wells score, Revised Geneva score) assist probability assessment but have limitations in complex or atypical presentations[2].

This case series describes three distinct phenotypes encountered in emergency practice:

1. Massive PE with obstructive shock
2. Submassive PE mimicking community-acquired pneumonia
3. Acute PE superimposed on unrepaired adult congenital heart disease (ACHD) with severe pulmonary arterial hypertension (PAH)

The report bridges acute intervention with long-term prevention, aligning with the 2026 AHA/ACC/ACCP/ACEP/CHEST/SCAI/SHM/SIR/SVM/SVN Guideline for the Evaluation and Management of Acute Pulmonary Embolism in Adults, which introduces a new severity classification (Categories A–E) and emphasizes multidisciplinary care, prompt anticoagulation, and recurrence prevention strategies.

METHODS

This study was designed as a retrospective descriptive case series conducted in a single tertiary-care emergency department over the period from December 2024 to 2025. Adult patients (≥18 years) with a confirmed diagnosis of acute pulmonary embolism (PE) based on CT pulmonary angiography (CTPA) and managed in the emergency department with complete clinical records were included. Data were obtained from structured emergency clinical documentation, including primary and secondary survey findings and AMPLE history, along with laboratory investigations such as D-dimer, troponin-I, NT-proBNP, and coagulation profile. Imaging data included electrocardiography (ECG), chest radiography, point-of-care ultrasound (POCUS)/echocardiography, and CTPA. Treatment details and 30-day clinical outcomes were also recorded.

Diagnostic evaluation followed a standardized protocol incorporating clinical probability assessment using the Wells score, Revised Geneva score, and Pulmonary Embolism Rule-out Criteria (PERC) where applicable, in conjunction with biomarkers and bedside imaging for risk stratification, with CTPA serving as the gold-standard confirmatory modality. Severity classification and management strategies were aligned with contemporary 2026 AHA/ACC risk categories (A–E) wherever applicable, with thrombolysis decisions guided primarily by physiological parameters such as hemodynamic instability and right ventricular dysfunction rather than clot burden alone. Statistical analysis was descriptive in nature, given the case-series design, and no inferential statistics were applied. Institutional ethics committee approval was obtained, and written informed consent for publication was secured from all patients.

RESULTS

Case 1: Massive Pulmonary Embolism with Obstructive Shock

A 40-year-old previously healthy male presented with 4-day progressive dyspnea, acute worsening (onset ~6:00 am), hypotension (BP 70/50 mmHg), tachycardia (HR 130/min), and severe hypoxemia (SpO_2 75% on room air).

Key Investigations

- ABG: pH 7.30, lactate 6.1 mmol/L, PO_2 165 mmHg on 15 L NRBM
- ECG: sinus tachycardia, classic S1Q3T3 pattern
- POCUS: dilated RA/RV, IVC 2.1 cm, no DVT on screening
- D-dimer: 9.9 µg/mL; troponin-I positive; NT-proBNP 1100 pg/mL
- Wells score: 4.5 (intermediate); Revised Geneva: 5 (moderate)

Imaging Figure 1: CTPA – saddle embolus at main pulmonary trunk bifurcation with bilateral lobar, segmental, and subsegmental extensions; wedge-shaped juxta-pleural opacity in left lower lobe on CXR.

Management

- High-flow oxygen (NRBM)
- Noradrenaline infusion for shock
- Unfractionated heparin (5000 IU bolus + 1000 IU/h infusion)

- **Tenecteplase 40 mg single bolus** (weight-based)
- Cardiology consultation; transferred to CCU

Outcome Hemodynamic stabilization within 2 hours; discharged Day 5 on rivaroxaban 15 mg once daily; lower-limb Doppler normal; advised long-term cardiology follow-up.

Case 2: Submassive Pulmonary Embolism Mimicking Pneumonia

A 37-year-old male presented with 2-day left-sided chest pain, dyspnea (NYHA III), fever, and 4 episodes of blood-tinged sputum. SpO_2 97% on room air, RR 26/min, hypotension (BP 72/52 mmHg), temperature 100.9°F.

Key Investigations

- WBC 12,310/µL; D-dimer 1.28 µg/mL; troponin-I 0.08 ng/mL (mild); NT-proBNP 70 pg/mL, RA/RV dysfunction
- Factor VIII activity: 191% (identified prothrombotic trigger)
- Wells score: 1; Revised Geneva: 2; PERC positive for hemoptysis
- CXR: left lower lobe opacity

Imaging Figure 2: CTPA – filling defect in left pulmonary artery segmental branches with associated consolidation.

Management

- LMWH stat → **Alteplase 100 mg IV over 2 hours**
- Cefoperazone-sulbactam for presumed concurrent infection
- NIV support; heparin infusion (weight-based nomogram) from Day 4
- Thrombophilia work-up completed

Outcome Symptom resolution by Day 7; discharged on rivaroxaban 20 mg once daily for 3 months with cardiology follow-up.

Case 3: Acute PE in Unrepaired Atrial Septal Defect with Severe Pulmonary Hypertension

A 60-year-old male with known large ostium secundum ASD (surgery refused), severe PAH (baseline mean PAP 45 mmHg, on sildenafil + torsemide), and recurrent pneumonias since 2019, presented with cough, dyspnea, and mild hemoptysis. Vitals: HR 100/min, RR 30/min, BP 100/65 mmHg, SpO_2 69% on room air.

Key Investigations

- Echocardiography: single atrium, dilated RA/RV, estimated PAP 64 mmHg, TR gradient 35 mmHg, PAT 40 ms; 60/60 sign assisted differentiation from chronic strain alone
- CTPA: left lower lobar and segmental PE

Pathophysiology Chronic PAH caused baseline RV pressure overload; acute PE imposed a "second-hit," precipitating disproportionate PVR rise, acute RV failure, and shock.

Management

- Intubation and mechanical ventilation
- Noradrenaline 0.5 µg/kg/min
- **Alteplase 100 mg over 2 hours** (administered after multidisciplinary risk-benefit discussion despite active hemoptysis)

Outcome Rapid clinical improvement; full recovery and discharge on continued anticoagulation + PAH therapy

Table 1: Comparative Clinical Characteristics

Parameter	Case 1	Case 2	Case 3
Age/Sex	40/M	37/M	60/M
Severity (approx. AHA/ACC Category)	High (E-equivalent)	Intermediate (C/D)	High (E-equivalent)
Shock	Yes	Yes	Yes
RV Dysfunction	Yes	Yes	Yes
Troponin	Positive	Mildly elevated	Not available
Thrombolytic	Tenecteplase (bolus)	Alteplase (infusion)	Alteplase (infusion)
Unique Feature	Obstructive shock	Pneumonia mimic + Factor VIII elevation	ASD + severe PAH + hemoptysis

Table 2: Thrombolytic Agents Comparison

Parameter	Alteplase	Tenecteplase
Type	Recombinant tissue plasminogen activator (tPA)	Modified tPA
Dose	100 mg IV infusion over 2 hours	Weight-based single bolus (30–50 mg)
Half-life	Short (initial 5 min; terminal up to 72 min)	Longer (20–130 min)
Advantage	Titratable infusion; established evidence	Single bolus; rapid administration in ER

Figure 1 CT pulmonary angiography (CTPA) image from Case 1 showing saddle embolus at the bifurcation of the main pulmonary trunk with bilateral lobar, segmental, and subsegmental extensions.

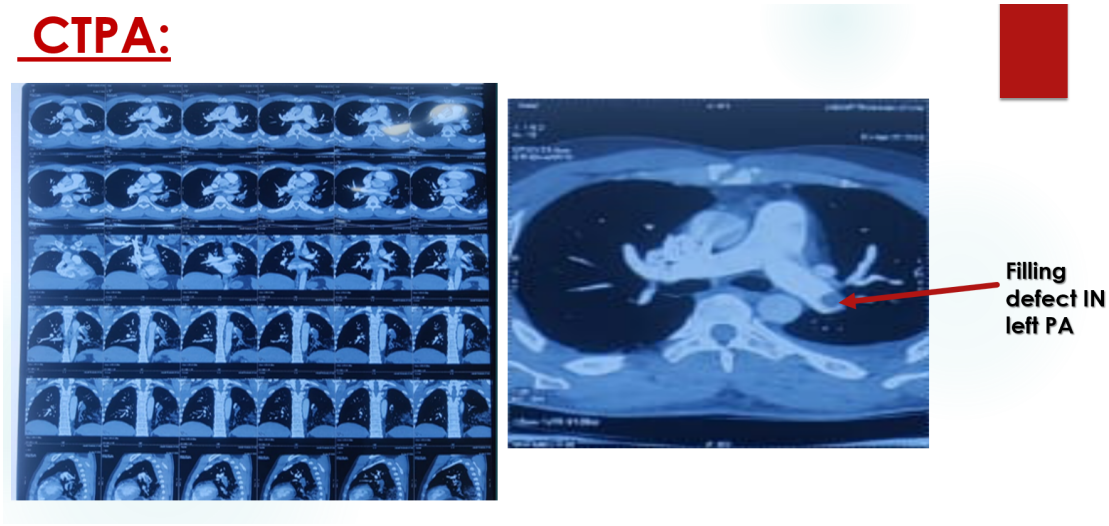


Figure 2 CT pulmonary angiography (CTPA) image from Case 2

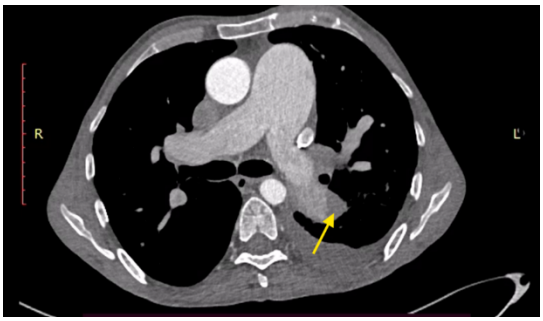


Figure 3 Schematic diagram illustrating the pathophysiology of acute pulmonary embolism.

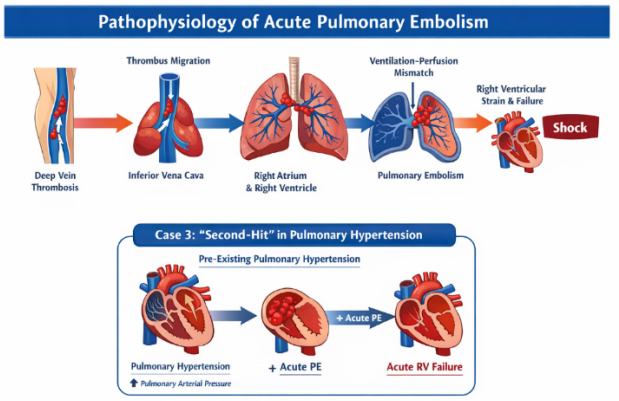
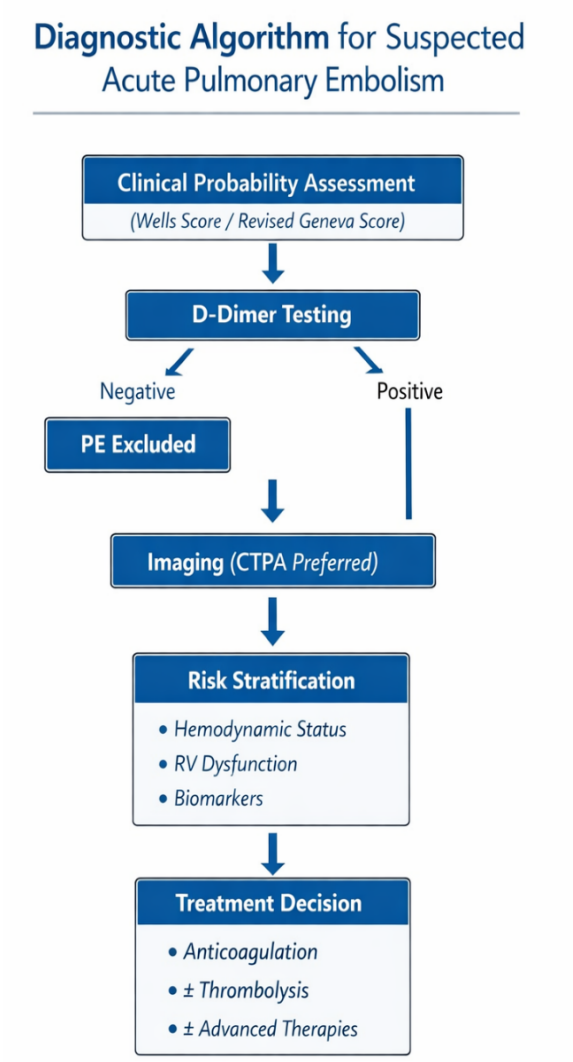


Figure 4 ***Diagnostic*** algorithm for suspected acute pulmonary embolism.



DISCUSSION

Diagnostic Challenges Case 2 exemplifies how PE can mimic pneumonia (fever, hemoptysis, lobar opacity), underscoring that scoring systems alone may underestimate probability. Integration of D-dimer, troponin, and high clinical gestalt prompted CTPA, confirming the diagnosis[4].

Role of Imaging and Bedside Tools CTPA remains the gold standard. POCUS (RV dilation, McConnell's/60/60 signs) and ECG (S1Q3T3) provided rapid bedside clues, especially valuable in resource-limited or unstable settings.

Thrombolysis Decision-Making

- Indicated (Class 1) in massive/high-risk PE with shock (Cases 1 and 3).
- Considered (Class 2a/2b per 2026 guideline) in select intermediate-risk cases with RV strain.
- Case 3 demonstrates judicious override of relative contraindication (hemoptysis) when physiology indicated imminent collapse, consistent with prior reports of life-saving thrombolysis despite bleeding risk.

Special Populations Patients with ACHD/PAH (Case 3) warrant physiology-based rather than protocol-driven care; chronic RV strain masks acute changes, necessitating multidisciplinary input (e.g., PERT activation).

Prevention and Long-Term Management Acute survival must transition to robust secondary prevention to reduce recurrence (up to 30% in unprovoked PE). Per the 2026 AHA/ACC/ACCP/ACEP/CHEST/SCAI/SHM/SIR/SVM/SVN Guideline:

- **Anticoagulation:** DOACs (rivaroxaban, apixaban, edoxaban, dabigatran) preferred over vitamin K antagonists for long-term therapy due to superior efficacy, lower bleeding risk, and no routine monitoring (Class 1 recommendation)[5].

- **Duration:** Extended/indefinite for unprovoked PE, thrombophilia (e.g., elevated Factor VIII), or persistent risks (PAH, ACHD); 3–6 months for provoked events. Reduced-dose DOACs (e.g., apixaban 2.5 mg BD) considered in lower-bleeding-risk patients on long-term therapy[6].
- **Lifestyle and Risk Reduction:** Early ambulation encouraged post-stabilization; prolonged immobility avoided; hydration maintained; smoking cessation and weight optimization advised. For long-distance travel (≥4–5 hours): frequent movement, graduated compression stockings (15–30 mmHg), and prophylactic LMWH/DOAC in high-risk individuals[7].
- **Primary Prophylaxis (in at-risk settings):** Padua/Caprini/IMPROVE score-guided LMWH or mechanical methods (IPCD) in hospitalized/surgical patients; individualized in cancer, pregnancy, or thrombophilia[7-8].

These cases reinforce that prevention bridges acute care to sustained survival, with DOAC preference and lifestyle measures central to reducing recurrence.

CONCLUSION

Acute PE demands rapid recognition, multimodal stratification (scores + biomarkers + imaging), and bold, individualized intervention—including thrombolysis in high-risk physiology despite relative contraindications. Long-term outcomes hinge on structured DOAC-based anticoagulation, early mobility, and guideline-directed prevention to avert recurrence. High clinical suspicion combined with prevention strategies saves lives acutely and sustains them long-term. Implementation of pulmonary embolism response teams (PERTs) and prospective studies in special populations are recommended.

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Data Availability: De-identified case data available from corresponding author upon reasonable request.