SSR Journal of Medical Sciences (SSRJMS)

OPEN ACCESS

Volume 2, Issue 2, 2025 Journal homepage: https://ssrpublisher.com/ssrjms/ ISSN: 3049-0375
Email: office.ssrpublisher@gmail.com

Hemodynamic Deterioration in Acute Pulmonary Embolism: A Clinical Case Report

Cookey SN, Asikimabo-Ofori D

Department of Internal Medicine, Rivers State University / Rivers State University Teaching Hospital, Port Harcourt, Nigeria

Received: 14.05.2025 | **Accepted:** 23.06.2025 | **Published:** 26.06.2025

***Corresponding author:** Cookey SN

DOI: *10.5281/zenodo.19034220*

Abstract **Case Studies**

Background: Acute pulmonary embolism (PE) is a common medical emergency that can rapidly become fatal. In massive PE, occlusion of the pulmonary vasculature precipitates systemic hemodynamic instability and, without intervention, cardiovascular collapse. Timely systemic thrombolysis markedly improves survival in these high-risk cases; however, in many low-resource environments, diagnostic bottlenecks—limited imaging, delayed laboratory results, and logistical constraints—frequently postpone therapy. Clinicians in such settings must weigh the bleeding risk of fibrinolytics against the imminent threat of circulatory failure, often relying on bedside assessment and expedited protocols to deliver life-saving treatment. This study aims to delineate these challenges, present real-world outcomes of prompt thrombolytic intervention under resource constraints, and propose pragmatic strategies for accelerating reperfusion therapy in similar settings.

Case Summary: We present a 42-year-old chronic smoker with acute PE who deteriorated hemodynamically (blood pressure drop from 130/70 mmHg to 86/67 mmHg; SpO_2 76 %) while awaiting advanced imaging. Bedside transthoracic echocardiography (TTE) revealed multiple echogenic masses in the main pulmonary artery and right ventricle with severe tricuspid regurgitation (Vmax 3.8 m/s, estimated PASP 56 mmHg). Systemic alteplase (100 mg over 2 h) produced rapid clinical recovery and oxygenation (SpO_2 92–95 % within 6 h). This case underscores the utility of bedside echo for rapid risk stratification and the feasibility of guideline-directed systemic thrombolysis in a resource-poor setting when computed tomography pulmonary angiography (CTPA) is either unavailable or delayed.

Conclusion: Training frontline physicians in point-of-care echocardiography and ensuring ready access to thrombolytics can be lifesaving in sub-Saharan Africa.

Keywords: Pulmonary Embolism, Thrombolysis, Bedside Echocardiography, Resource-Limited Settings, Hemodynamic Instability, Nigeria.

Citation: Cookey, S. N., Asikimabo-Ofori, D. (2025). Hemodynamic deterioration in acute pulmonary embolism: A clinical case report. *SSR Journal of Medical Sciences (SSRJMS)*, *2*(2), 59-62.

BACKGROUND

Acute pulmonary embolism (PE) remains a critical and often life-threatening cardiovascular emergency. Among affected patients, a subset may present with massive pulmonary embolism, defined by hemodynamic instability, right ventricular dysfunction, and a significantly elevated risk of mortality. In such cases, prompt diagnosis and immediate intervention are imperative to prevent cardiovascular collapse. The degree of vascular obstruction in the pulmonary circulation directly correlates with morbidity and mortality, underscoring the necessity for swift therapeutic decisions.

However, the cornerstone of emergency treatment—systemic thrombolysis—poses its own clinical conundrums. While thrombolytics can be life-saving, they also carry a substantial risk of bleeding, particularly in patients with co-morbidities or recent surgeries. The attending physician is thus placed at the precipice of a delicate balancing act: weighing the benefits of immediate clot dissolution against the catastrophic potential for hemorrhagic complications. In resource-limited settings like many Nigerian tertiary hospitals, this decision is further complicated by limited access to advanced imaging, delayed referrals, inadequate intensive care infrastructure, and often, the absence of multidisciplinary rapid response teams.

In clinical practice—especially in post-surgical or postpartum contexts—we are frequently faced with the

challenge of adhering to established international guidelines while also tailoring care to fit local realities and limitations. There is an urgent need for context-specific protocols that harmonize global evidence-based practices with pragmatic, environment-sensitive approaches. Physicians must operate at the intersection of medical science and human intuition, striving not only to follow algorithms, but also to "read the room" of each patient's complex narrative—sometimes acting boldly on clinical grounds to save a life.

Pulmonary embolism is, in fact, a cross-cutting diagnosis that sits at the convergence of multiple specialties. It is encountered not only in internal medicine and cardiology but also in obstetrics and gynecology, orthopedic surgery, oncology, and even psychiatry—especially among long-stay or immobilized patients. Its ubiquity demands heightened clinical vigilance and unified institutional readiness across all departments. Therefore, every health facility, particularly in resource-constrained settings, must develop and continually refine a locally-adapted PE protocol—one that reflects its own diagnostic capacity, patient demographics, and therapeutic resources.

Globally, the prevalence of pulmonary embolism varies by region and risk factors. In developed settings, increased imaging accessibility has contributed to rising detection rates, while in Sub-Saharan Africa, it remains underreported and frequently diagnosed late, if at all[1–2]. Common clinical contexts include post-partum states, major orthopedic surgeries (particularly lower limb fractures), prolonged bed rest, active malignancies, chronic heart failure, and inherited or acquired thrombophilias[2–3].

Case Presentation

Mr. U.C., a 42-year-old Nigerian man, was transferred from a peripheral clinic to the emergency department with a five-day history of pleuritic cough, scant hemoptysis, and left-sided chest pain, followed by three days of progressively worsening dyspnea. He reported no orthopnea, paroxysmal nocturnal dyspnea, limb swelling, or syncope, and had no personal history of hypertension, diabetes mellitus, thrombophilia, recent long-haul travel, or oestrogen use. Five months earlier he had sustained a left-calf fracture in a road-traffic accident, managed with plaster immobilization; full ambulation had resumed only recently. His social history was notable for chronic cigarette smoking of approximately 20 pack-years, with no other substance use. Prior to transfer, he had been started on therapeutic enoxaparin 80 mg subcutaneously every 12 hours for presumed acute pulmonary embolism.

On arrival at our institution, he was alert, markedly dyspneic and centrally cyanosed but afebrile (T 36.8 °C), not pale and not icteric. Anthropometry showed a height of 182 cm and weight of 102 kg (BMI ≈ 30.8 kg m^{-2}). Vital signs were: blood pressure 130/70 mmHg, heart rate 118 min^{-1}, respiratory rate 32 min^{-1} and oxygen saturation 76 % on room air (improving to 92 % with supplemental oxygen). Chest auscultation was clear; the apex beat was displaced; heart sounds were (S_1, S_2) with a prominent pulmonary component (loud P_2); parasternal pan systolic murmur non radiating, no gallops or pericardial rubs were heard. There were no clinical features of deep-vein thrombosis, heart failure, calf tenderness, limb erythema or peripheral oedema, and jugular venous pressure was not elevated.

Within 24 hours of admission (sixth day of illness) his blood pressure fell to 86/67 mmHg and SpO_2 deteriorated further, A markedly elevated D-dimer (3256 ng mL^{-1} FEU), sinus tachycardia on ECG, and bedside transthoracic echocardiography demonstrating multiple mobile thrombi in the main pulmonary-artery trunk and s sessile hyperechoic masses—approximately 15 mm in diameter—obstructing the main pulmonary-artery trunk and extending into its bifurcation; concomitant compromised LV RV relationship with interventricular-septal flattening yielded a D-shaped left ventricle, the right ventricle measured at the upper limit of normal, and severe tricuspid regurgitation (Vmax 3.8 m s^{-1}) fixed and dilated IVC produced an estimated pulmonary-artery systolic pressure of 56 mmHg, while left-ventricular structure and biplane LVEF remained intact at 65 %.

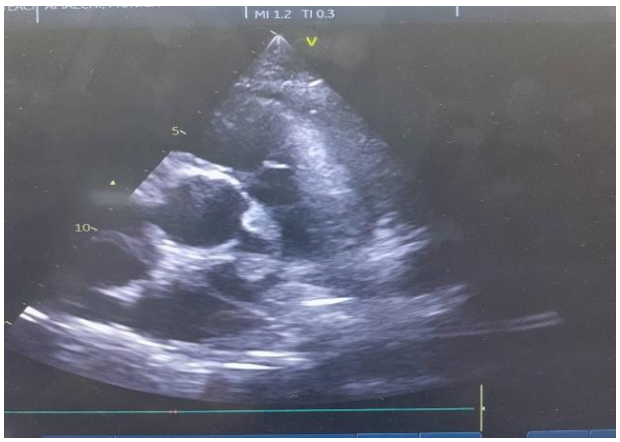
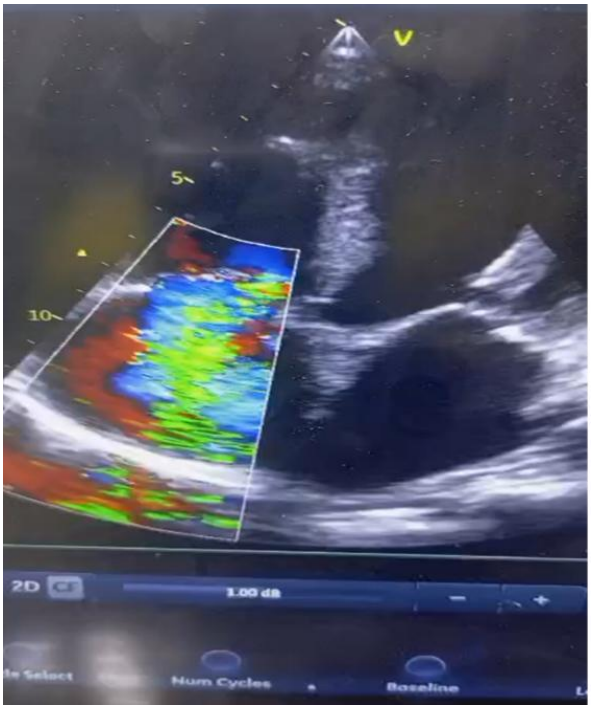


Figure 1Figure 1: Parasternal short-axis (PSAX) view demonstrating echogenic thrombi (arrow) within the main pulmonary artery. extending beyond the bifurcation of the trunck.



Computed-tomography pulmonary angiography was postponed owing to hemodynamic instability and ultimately rendered redundant after successful thrombolytic therapy. Routine biochemistry, complete blood count, and coagulation studies (INR 1.0, APTT 32 s) were otherwise within reference ranges, with a thrombophilia screen pending.

Because sustained hypotension was accompanied by transthoracic-echocardiographic evidence of large, mobile thrombi in the main pulmonary-artery trunk, the episode fulfilled European Society of Cardiology (ESC 2025) criteria for *high-risk (massive) pulmonary embolism.* Before reperfusion therapy, the patient was evaluated with a bedside bleeding-risk algorithm (absolute and relative contra-indications checklist plus BACS score = 1), categorizing him as *low risk* for hemorrhage; therefore, full-dose systemic thrombolysis was chosen. Alteplase 100 mg was infused intravenously over two hours in the intensive-care unit. Immediately after lysis, a weight-adjusted unfractionated-heparin infusion was started and titrated to maintain an activated partial-thromboplastin time of 60–80 s Six hours after thrombolysis the blood pressure had improved to 110/70 mmHg, oxygen saturation stabilized at 92–95 % on nasal oxygen, and respiratory distress lessened. Anticoagulation was switched to rivaroxaban on day 5. Placement of an inferior vena cava filter was not possible because the device was unavailable. By the fifth post-lysis day the patient was ambulant, and anticoagulation was transitioned to rivaroxaban 15 mg twice daily for 21 days, with a planned reduction to 20 mg once daily thereafter.

The patient remained hemodynamically stable, mobilized without desaturation, and was discharged on day 7 with scheduled cardiology review. At the three-month follow-up he was asymptomatic, in New York Heart Association functional class I, and repeat transthoracic echocardiography confirmed complete resolution of pulmonary-artery thrombi with a pulmonary-artery systolic pressure of 33 mmHg.

At the 30-day outpatient review he remained asymptomatic, maintained a resting SpO_2 of 97 %, and transthoracic echocardiography Rivaroxaban 20 mg daily was continued, with a minimum three-month anticoagulation plan.

DISCUSSION

The prevalence of pulmonary embolism (PE) worldwide is shaped by regional infrastructure and access to advanced diagnostics. While CTPA has become the cornerstone for PE diagnosis in developed countries, Sub-Saharan Africa continues to grapple with underdiagnosis due to limited resources[1,4]. Risk factors such as lower-limb orthopedic surgery, malignancy, postpartum states, and prolonged immobilization are common in LMICs, yet many cases remain unconfirmed until late or post-mortem[4].

In this case, the patient's steep blood pressure decline and worsening oxygenation signaled a massive PE. Per ESC guidelines, high-risk PE with sustained hypotension demands immediate reperfusion therapy to reduce mortality[5]. Given the absence of ready access to CTPA(weekend logistics challenge) and coupled with the worsening clinical state we relied on bedside echocardiography. The identification of echogenic

thrombi in the right ventricle and pulmonary artery—although rare—proved crucial. Visualized thrombi within the RV or main pulmonary artery are associated with a significantly higher risk of early death, reported in less than 4% of PE cases[6]. Moreover, the right ventricular/left ventricular ratio exceeding 1, a flattened septum with D-shaped LV, and elevated PASP provided conclusive evidence of RV strain and pressure overload.

Systemic thrombolysis with alteplase remains a mainstay for massive PE. The regimen of 100 mg over 2 hours is endorsed by both ESC and ACC as a Class I, Level B recommendation[5,7]. While catheter-directed thrombolysis and surgical embolectomy offer targeted therapy with reduced bleeding risks, these are not routinely available in low-resource settings. Our favorable outcome—marked by hemodynamic stabilization and echocardiographic resolution—aligns with other African case reports highlighting the viability of guideline-directed thrombolysis using standard protocols[4,8].

From this case, several clinical insights emerge. Early recognition of tachycardia, hypoxia, and hypotension as signs of decompensating PE must prompt urgent escalation. Point-of-care ultrasound, especially echocardiography, is a pragmatic and highly informative diagnostic alternative in unstable patients where CTPA is not feasible[6]. Systemic thrombolysis—when performed with appropriate bleeding risk assessment—can be life-saving, particularly where interventional options are lacking. Finally, structured follow-up remains essential to detect and manage chronic thromboembolic pulmonary hypertension (CTEPH), a significant long-term complication of PE.*9-11*

CONCLUSION

This case gives credence to the critical value of prompt, bedside echocardiography-guided thrombolysis in managing acute, high-risk pulmonary embolism within a resource-limited tertiary care setting. Despite infrastructural challenges, a timely clinical decision anchored in available diagnostic modalities facilitated a positive outcome. Strengthening point-of-care ultrasound capacity and maintaining an accessible stock of thrombolytic agents should be prioritized in LMIC cardiopulmonary centers. Adapting international guidelines to local realities not only enhances clinical outcomes but also ensures that life-saving interventions remain within reach for all patients, regardless of geography.

DECLARATIONS

Ethical Approval: Ethical approval and was sought from the Ethical Committee and consent obtained from patient.

Authors' contributions: The lead author conceptualized the study; the remaining authors contributed to funding and manuscript development. All authors critically reviewed the draft and approved the final version for submission.

Conflict of interest: The authors declare no conflicts of interest.

Funding: Research activities and publication costs were entirely self-funded.

REFERENCES

1. Konstantinides SV, Meyer G, Becattini C, et al. 2019 ESC Guidelines for the diagnosis and management of acute pulmonary embolism. Eur Heart J. 2020;41(4):543–603.

2. Danwang C, Tagny CT, Tochie JN, Tankeu R, Robert A. Prevalence and outcomes of venous thromboembolism in Africa: a systematic review and meta-analysis. BMJ Open. 2020;10(9):e037873.

3. Menezes RG, et al. Management of high-risk pulmonary embolism in the emergency setting. Int J Cardiol. 2024;384:115–123.

4. Oluwole JG, et al. Pulmonary embolism in a Nigerian tertiary hospital: lessons for resource-poor settings. Medicine (Baltimore). 2024;103:e34987.

5. Khemasuwan D, Yingchoncharoen T, Tunsupon P, et al. Role of echocardiography in acute pulmonary embolism. Cardiovasc Ultrasound. 2023;21:14.

6. Li XF, Zhang YQ, Huang JY, et al. Diagnostic value of bedside echocardiography in acute pulmonary embolism. J Ultrasound Med. 2022;41:973–982.

7. Sharifi M, Meeran K, Mehra A. Accelerated treatment with rtPA for pulmonary embolism. J Vasc Surg. 2021;73:213–219.

8. Goldhaber SZ, Visani L, De Rosa M. Acute pulmonary embolism: clinical outcomes in the International Cooperative Pulmonary Embolism Registry (ICOPER). Lancet. 9. 1999;353:1386–1389.

9. Meyer G, Vicaut E, Danays T, et al. Fibrinolysis for patients with intermediate-risk pulmonary embolism. N Engl J Med. 2014;370(15):1402–1411.

10. Kearon C, Akl EA, Ornelas J, et al. Antithrombotic therapy for VTE disease: CHEST guideline and expert panel report. Chest. 2016;149(2):315–352.

11. Hoeper MM, Madani MM, Nakanishi N, et al. Chronic thromboembolic pulmonary hypertension. Lancet Respir Med. 2024;12(3):208–222.