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CASE REPORT **Open Access**

Serial Case of Acute Pulmonary Embolism: Different Therapies Based on Different Patient Profiles

Ilzy Jum Ahmad[1,*], Idar Mappangara[1], Akhtar Fajar Muzakkir[1]
[1]Department of Cardiology and Vascular Medicine, Faculty of Medicine, Universitas Hasanuddin, Makassar, Indonesia

*Corresponding Author. Email: ilzyjum@gmail.com, Telp: +6281245402415

ABSTRACT

Pulmonary embolism (PE) is the third leading cause of cardiovascular death. The high mortality rate prompted an analysis of these three-case series to evaluate various treatment options. Case I, a 66-year-old woman with acute shortness of breath and a history of EVLA because of chronic venous insufficiency in the left leg. She had comorbid of hypertension. She was diagnosed with massive PE with unstable hemodynamics. Catheter-directed thrombolysis (CDT) using Alteplase showed significant improvement within 24 hours. Case II, a 45-year-old woman with dyspnea de effort for one month and a 5-year history of hypertension. MSCT angiography showed a significant PE with partial stenosis, pulmonary hypertension, and a bidirectional atrial septal defect. Combination therapy with heparin, sildenafil, and furosemide had been provided but the patient had sudden cardiac death. Case III, a 36-year-old woman with dyspnea on effort after the delivery. Initial echocardiography showed right atrial and left ventricular thrombi. On the second day of treatment, clinical deterioration occurred due to thrombus migration to the pulmonary artery, confirming the diagnosis of acute PE. CDT therapy was performed. A 24-hour evaluation revealed persistent occlusion of pulmonary artery, leading to percutaneous transluminal angioplasty, which successfully restored blood flow. This case series report emphasizes the importance of risk-based therapy, including CDT for high-risk PE, anticoagulation for intermediate- to low-risk PE, and hemodynamic support in cases of shock.

Keywords: Clinical deterioration, heparin, pulmonary embolism, thrombolytic therapy, tissue plasminogen activator

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Email:
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Introduction

Venous thromboembolism (VTE) represents a significant global health issue, with approximately 10 million cases documented annually, contributing to considerable morbidity and mortality. It ranks as the third leading cause of cardiovascular death and is linked to various inherited and acquired risk factors, alongside aging. While the precise incidence of pulmonary embolism (PE), a serious complication of VTE, remains unclear, it is estimated that nearly one-third of hospitalized patients in the United States are at risk for VTE. The diagnosed incidence of VTE stands at roughly 117 cases per 100,000 people, though the actual prevalence is likely higher due to underdiagnosis, often revealed only at autopsy. Moreover, the number of hospital admissions for PE surged from about 60,000 in 1993 (23 per 100,000) to over 202,000 in 2012 (65 per 100,000), with in-hospital mortality rates ranging from 5% to 10%. The clinical outcomes of PE are influenced by the extent of vascular obstruction and its hemodynamic consequences. Understanding the underlying pathophysiology is crucial for effective patient risk stratification and guiding therapeutic interventions.[1]

Between the years 1997 and 2013, global data indicated a rise in the incidence of diagnosed pulmonary embolism, coinciding with a decline in associated mortality rates. This increase in diagnoses is attributed to the greater utilization of advanced imaging techniques, particularly computed tomography pulmonary angiography (CTPA).[2] Despite the reduction in mortality rates observed until 2013, VTE, a precursor to pulmonary embolism, remains the third leading cause of death within cardiovascular disease, following myocardial infarction and stroke.[3] The significant mortality associated with PE has led to the publication of the present three case series that evaluate variations in therapeutic approaches for patients with this condition. These findings aim to serve as a reference for optimizing treatment strategies for PE in the future.

Case

Case I illustration

A 66-year-old female presented with the acute onset of shortness of breath (SOB) 15 hours before admission, with no prior history of respiratory distress. Notably, she reported lower left leg swelling persisting for three weeks, following intermittent episodes over the preceding two months, particularly while sitting with her legs dangling. This swelling was not associated with pain or erythema. Her medical history included cardiovascular risk factors such as hypertension, for which she was prescribed Candesartan 8 mg daily. The patient had a prior hospitalization due to chronic venous insufficiency demonstrated by Doppler examination revealing multiple venous dilations in her left leg. She underwent Endovenous Laser Ablation

(EVLA) and experience acute heart failure.

The patient was admitted in a conscious state, presenting with a blood pressure of 104/78 mmHg, a pulse rate of 108 beats per minute, a respiratory rate of 30 breaths per minute, a temperature of 36.6°C, and an oxygen saturation of 88% on room air. Conventional respiratory support was initiated using a non-rebreathing mask at 10 Liters per minute, which elevated the oxygen saturation to 98%. Significant findings on physical examination included anemic conjunctivae, elevated jugular venous pressure, and edema of the left lower extremity (with stockings) that was warm to the touch. The patient subsequently developed hemodynamic instability characterized by hypotension (60/40 mmHg). Initial management involved the intravenous administration of 200 cc of fluids, leading to a modest increase in blood pressure to 81/55 mmHg. Inotropic therapy was initiated with dobutamine at 3 mcg/kg body weight/minute and norepinephrine at 0.02 mcg/kg body weight/minute, which resulted in an improved blood pressure reading of 131/80 mmHg. Additionally, the patient received an 80 IU/kg body weight bolus of Heparin followed by a maintenance infusion of 18 IU/kg body weight/hour via a syringe pump.

During the supportive evaluation through electrocardiography (ECG), the patient exhibited sinus tachycardia alongside an S1Q3T3 pattern, indicative of PE. Laboratory findings showed leukocytosis (12,900) and elevated high-sensitivity Troponin I levels. Echocardiography on Figure 1 demonstrated preserved left ventricular (LV) systolic function at 56% (biplane), reduced right ventricular (RV) systolic function with a TAPSE of 1.4 cm, and evidence of diastolic dysfunction. There was dilation of the right atrium (RA) and right ventricle (RV) along with a positive McConnell sign. The LV exhibited a D-shaped morphology, accompanied by massive tricuspid regurgitation and right pulmonary artery (RPA) dilation. A thrombus measuring 3.4 cm × 2.1 cm was observed in the RPA. To corroborate the diagnosis, a contrast-enhanced CTPA was performed and revealed cardiomegaly (left ventricular hypertrophy, LVH) along with evidence of pulmonary hypertension (Figure 2). The scan identified a pulmonary embolism affecting the RPA, specifically in the superior arterial segment, the interlobar pulmonary artery, the lateral basal segment, and the posterior basal segment. Additionally, the left pulmonary artery exhibited emboli in the apicoposterior, anterior, inferior lingula, posterior basal, and lateral basal segments.

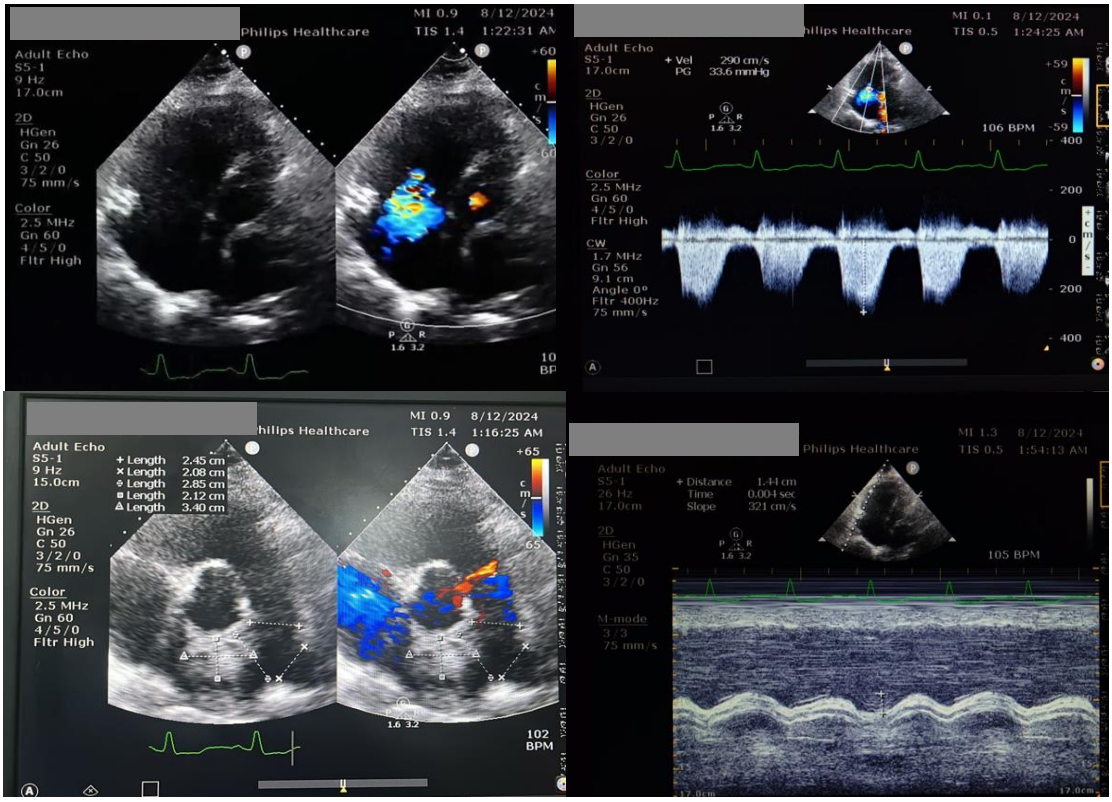

Figure 1. Initial echocardiogram shows evidence of right ventricular systolic dysfunction along with positive McConnell sign and observed thrombus in the right pulmonary artery.

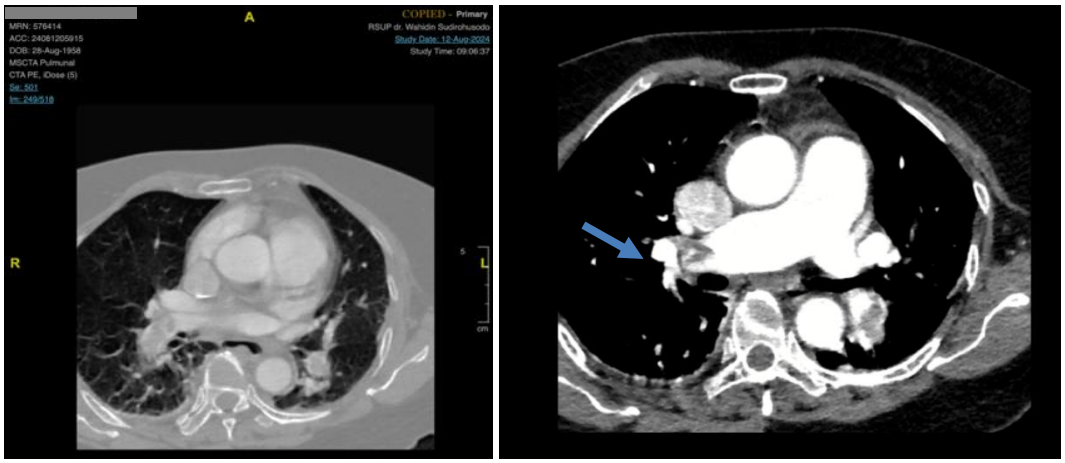

Figure 2. A contrast-enhanced CT pulmonary angiogram was performed, which identified thrombi present in both the right and left pulmonary arteries.

The patient was diagnosed with acute PE accompanied by unstable hemodynamic, prompting the initiation of catheter-directed thrombolysis (CDT) with Alteplase administered at a rate of 0.5 mg/hour via syringe pump. CDT was performed utilizing a 6F pigtail catheter accessed via the femoral vein. Due to facility constraints, the EKOS system was not employed.

The infusion lasted for 24 hours, administering a total of 12 mg of tPA. Post-infusion monitoring of RV systolic function was conducted via echocardiography. To mitigate bleeding risk in this case—given the patient's ARC-HBR 1 Major classification (high bleeding risk)—CDT was selected as the initial approach to minimize bleeding complications. This choice was particularly relevant considering the mortality rate associated with PE post-systemic thrombolysis remains significant (40-50%). Additionally, the thrombus in this patient was confined to the proximal region, further supporting the preference for CDT. Despite a low risk for intracranial bleeding linked to systemic thrombolysis, as indicated by both a PE-CH score of 0 and a BACS score of 0, which suggest a low likelihood of post-thrombolysis intracranial hemorrhage, CDT was deemed the most suitable intervention.

Prior to intervention, pulmonary angiography revealed filling defects suggestive of thrombus at the base of the main pulmonary artery (MPA) and both the left pulmonary artery (LPA) and RPA (Figure 3). A follow-up pulmonary arteriography conducted 24 hours post-CDT indicated a reduction in the size of the filling defects in both the RPA and LPA. Additionally, echocardiographic assessment of right ventricular systolic function, measured by the TAPSE parameter, demonstrated an improvement in right ventricular performance (Initial 1.4 cm to 1.5 cm on Day 1 and 1.7 cm on Day 2). Oxygen saturation increased to 97% on room air. Measurement of the RV/LV ratio and pulmonary artery pressure (PAP) was not conducted in this instance. Additionally, target aPTT monitoring (1.5-2 times the baseline) was omitted due to the prompt decision to proceed with CDT, resulting in no adjustments before the procedure.

Case II illustration

A 45-year-old female was admitted with a one-month history of progressive SOB and dyspnea de effort, accompanied by fatigue during light activities that has intensified over the past month. The patient was referred from an external hospital with a suspected diagnosis of PE and had been started on anticoagulant therapy with Fondaparinux and Dorner. Her cardiovascular history included hypertension for five years, and she was not on any regular medication. Upon examination, her vital signs revealed a blood pressure of 137/67 mmHg, a pulse rate of 125 beats per minute, a respiratory rate of 26 breaths per minute, a temperature of 36.6°C, and an oxygen saturation of 76% on room air. Physical findings included elevated jugular venous pressure (JVP), minimal bilateral leg edema, finger clubbing, and cyanosis.

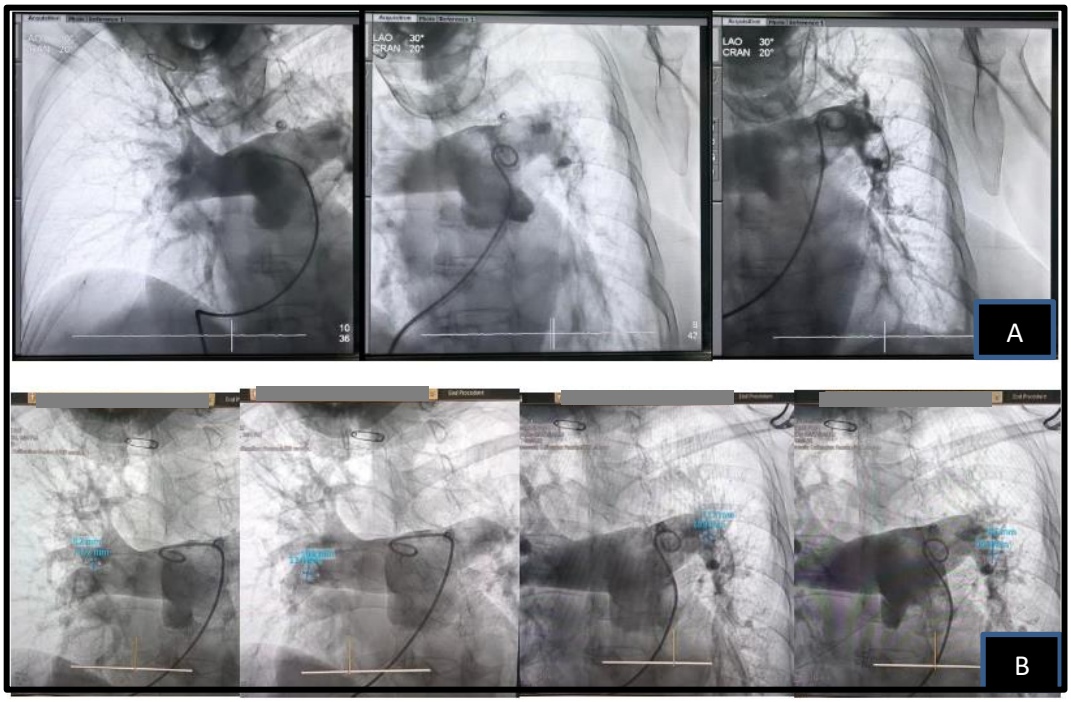

Figure 3. Pulmonary arteriography before (A) and after (B) catheter-directed thrombolysis.

On ECG, the patient exhibited sinus tachycardia and LVH, findings that correlated with the chest radiograph, which demonstrated cardiomegaly with a cardiothoracic ratio of 0.56. Laboratory assessments conducted at the referring hospital revealed leukocytosis (10,600/mcl) and an elevated D-dimer level of 585.46 ng/mL. Subsequently, the patient underwent a chest MSCT scan, which identified filling defects throughout the luminal pathway, extending from the pulmonary trunk to both the RPA and LPA. Notably, these defects remained evident distal to the right pulmonary artery emboli and slightly beyond the left pulmonary artery (Figure 4). An echocardiogram showed dilation of the MPA along with a thrombus within it, a secundum atrial septal defect (ASD) with a bidirectional shunt, as well as RA and RV dilation, while LV and RV systolic function remained normal. A CTPA further confirmed PE in the MPA and both RPA and LPA, resulting in partial stenosis, bilateral pneumonia, and cardiomegaly associated with the ASD (Figure 5).

Upon examination, the patient was diagnosed with intermediate-risk acute PE (Geneva score 5, Wells score 1.5, PESI score 95 points, moderate mortality risk: 3.2-7.1%), pulmonary hypertension type 1 dd/type 4, and a bidirectional shunt due to ASD. A patient with an unknown previous bolus dose of unfractionated heparin (UFH) received continuous heparin infusion at a rate of 18 IU/kg/hour via a syringe pump, along with oral sildenafil 20 mg every 8 hours and intravenous furosemide 40 mg every 24 hours. The patient subsequently suffered a sudden hemodynamic decline following a Valsalva maneuver during defecation. At that time,

peripheral oxygen saturation was observed to be 42-50% in all four extremities, without further diagnostic evaluations, such as echocardiography, ECG, or CT scan. Despite efforts at cardiopulmonary resuscitation, the patient experienced cardiac arrest and was pronounced deceased. The definitive cause of death remained uncertain.

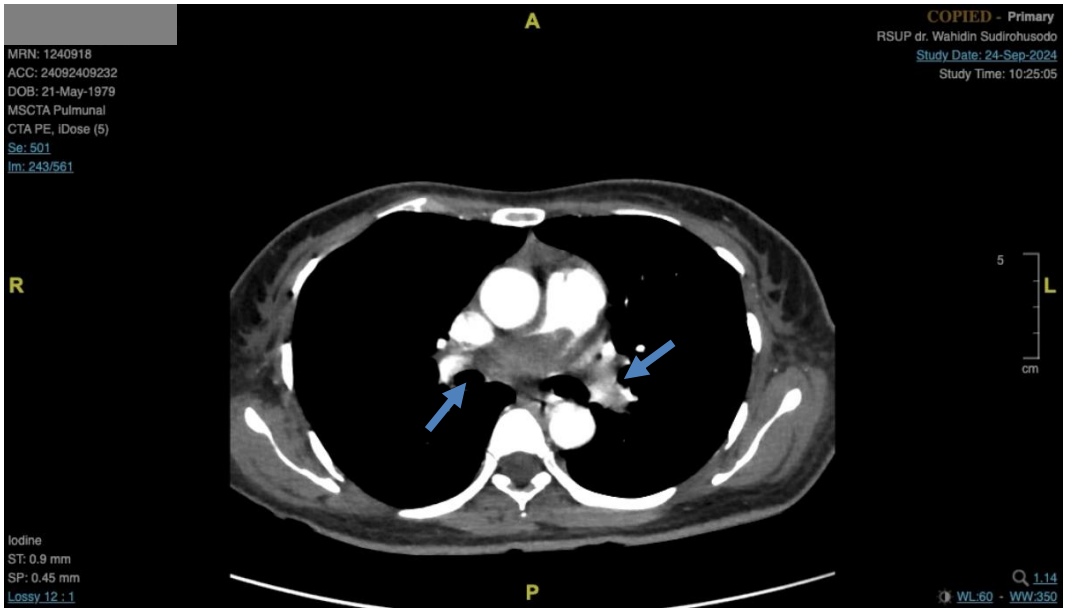

Figure 4. CT pulmonary angiography findings revealed a filling defect nearly occluding the entire lumen of the main pulmonary artery, extending from the bifurcation to both the right and left pulmonary artery.

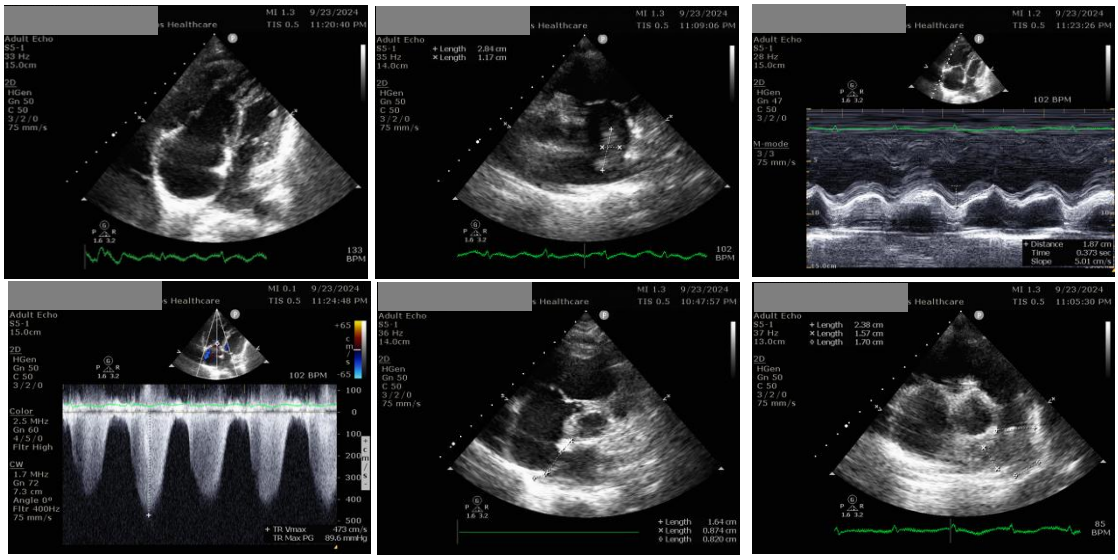

Figure 5. Echocardiography results on initial assessment revealed a secundum atrial septal defect.

Case III illustration

A 36-year-old woman presented with worsening SOB over 10 days prior to admission, associated with orthopnea, exertional dyspnea, and paroxysmal nocturnal dyspnea (PND). She reported a history of SOB during physical exertion, which began after the birth of her third child one month earlier. Upon examination, the patient was hemodynamically stable, with a JVP of +3 cm H_2O. Auscultation revealed bilateral rhonchi, and physical examination noted ascites with shifting dullness and bilateral pretibial edema. Hb of 10.4 g/dL and thrombocyte of 152.000/mcl were found on laboratory examinations. An ECG revealed sinus tachycardia, poor R-wave progression (PRWP), and LVH with strain, findings that correlated with chest X-ray results indicating cardiomegaly and pulmonary edema. Echocardiography revealed multiple LV thrombi were found with the largest size of 1.54x1.99 cm in the apical LV and RA thrombi measuring 2.1 x 2.5 cm. In addition, the LVEF was 23.9% on BIPLANE analysis (Figure 6).

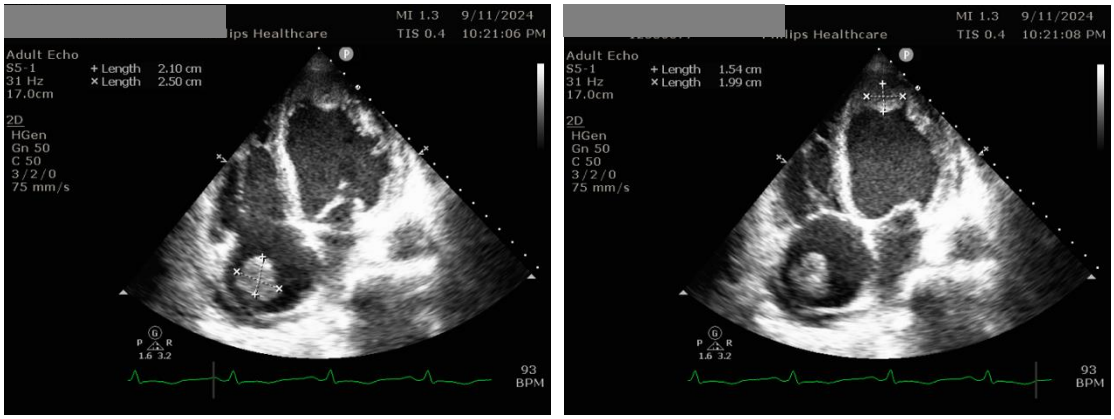

Figure 6. Echocardiogrphy revealed a left ventricular ejection fraction of 23.9% alongside multiple thrombi in the left ventricle and a mobile thrombus on the right ventricle.

The patient was diagnosed with New York Heart Association (NYHA) Class IV congestive heart failure, peripartum cardiomyopathy (PPCM), RA thrombus, and multiple LV thrombi. Consequently, she received heart failure management, which included an initial bolus of heparin at a dose of 60 IU/kg body weight, followed by a continuous maintenance infusion of 12 IU/kg body weight per hour via a syringe pump. Targeted aPTT monitoring was conducted, aiming for a range of 1.5 to 2 times the baseline. Initial measurements increased from 29.9 IU to 42.4 IU. The therapeutic target was achieved by the following day, and uptitration was scheduled for the second day.

On the second day of treatment, the patient experienced an abrupt onset of SOB,

accompanied by cold sweats. Oxygen saturation plummeted from 98% to 73%, blood pressure fell to 77/50 mmHg, and the respiratory rate increased to 32 breaths per minute. An echocardiographic evaluation for intracardiac thrombus revealed that a thrombus in the RA had migrated (Figure 7). The patient was subsequently diagnosed with Acute PE.

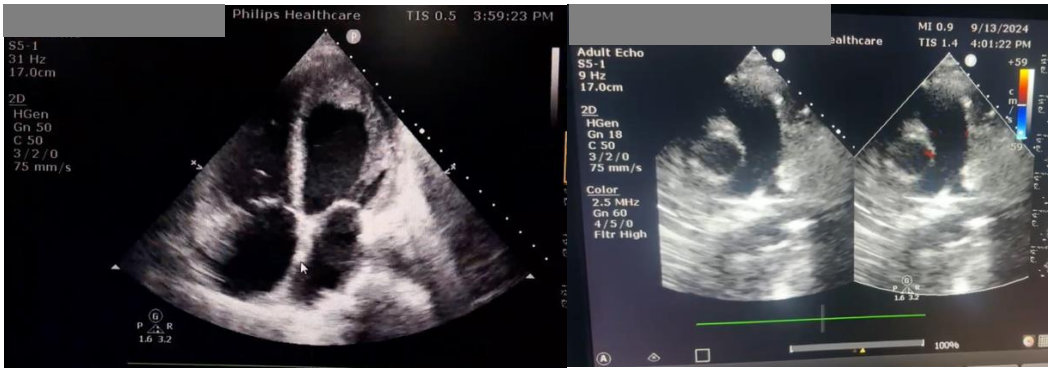

Figure 7. Echocardiography evaluation revealed that a thrombus in the right atrium had migrated.

Pulmonary arteriography demonstrated a filling defect in the RPA, suspected to be a thrombus, resulting in total occlusion of the lower lobe of the RPA (Figure 8). Consequently, thrombolytic therapy was initiated with CDT using Alteplase at a rate of 1 mg/hour for 24 hours. A 6F pigtail catheter was utilized for CDT via femoral vein access. Due to limited resources, EKOS monitoring was not implemented. The CDT was conducted over 24 hours with a total alteplase (tPA) dose of 24 mg. Notably, echocardiographic evaluation of RV systolic function, specifically the TAPSE parameter, was not carried out in this instance. Post-CDT, oxygen saturation increased from 77% to 97% on room air; however, the patient still presented with tachycardia at 130 beats per minute and tachypnea at 25 breaths per minute. Consequently, pulmonary arteriography was repeated to assess flow in the inferior segment of the right pulmonary artery. The RV/LV ratio and pulmonary artery pressure (PAP) were not evaluated. To manage bleeding risk, an evaluation was conducted, resulting in an ARC-HBR score indicative of 1 Major (high bleeding risk), leading to the decision for CDT over systemic thrombolysis. Given that the thrombus remained at a proximal level, CDT was deemed the preferred approach.

Follow-up arteriographic evaluation after this period showed persistent total occlusion of the lower lobe of the RPA. As a result, the procedure was extended to include percutaneous transluminal angioplasty (PTA) targeting the lower lobe lesion. Mechanical thrombectomy with PTA was conducted utilizing a Boston Scientific Mustang balloon catheter measuring 8

mm x 80 mm (135 cm), which gradually inflated to pressures ranging from 10 to 18 atm. Aspiration was not performed due to limitations within the facility, and no stents were deployed during the procedure. The PTA intervention proceeded without any reported intraprocedural complications. The final outcome revealed restored flow to the middle and lower lobes of the RPA, leading to the conclusion of the procedure. Heparinization was recommended for five days. Table 1 summarizes the demographics and clinical timelines for all three presented cases.

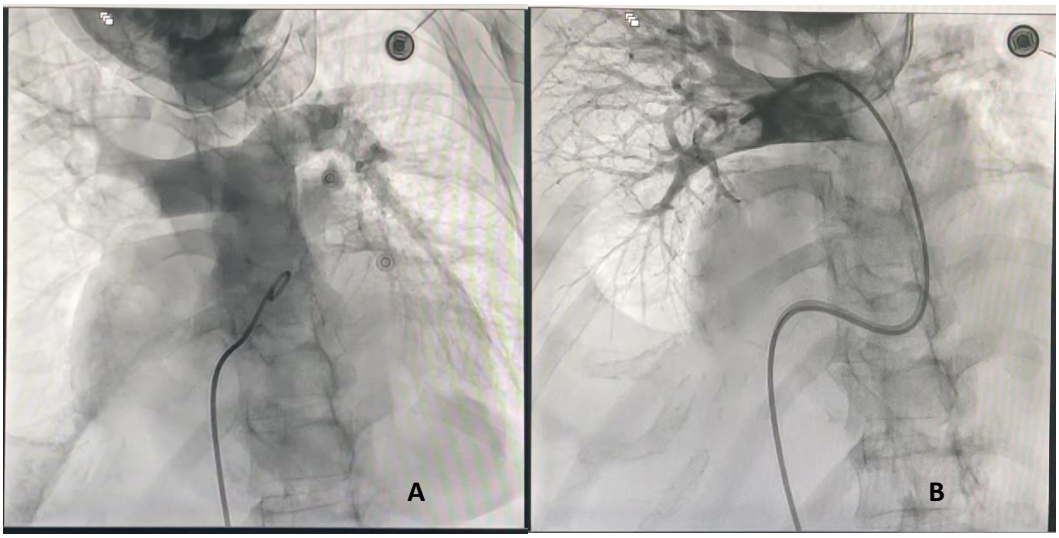

Figure 8. Results of pre-CDT (A) and post-CDT arteriography accompanied by PTA (B).

Discussion

Comparison of diagnosis, severity, and risk stratification of pulmonary embolism between three cases

The diagnosis of PE is grounded in the assessment of clinical presentation and hemodynamic stability. Initially, the identification of PE is guided by the presence of specific symptoms, followed by probability evaluations that incorporate the Pulmonary Embolism Rule-out Criteria (PERC) score, plasma D-dimer levels, and various imaging modalities. These imaging techniques include CTPA, lung scintigraphy (V/Q scintigraphy), pulmonary angiography, magnetic resonance angiography, echocardiography, compression ultrasonography, and computed tomography venography. For patients with stable hemodynamics, this diagnostic algorithm can be undertaken; however, in cases of unstable hemodynamics, direct imaging—such as echocardiography or CTPA—should be prioritized over probability testing.[3]

Table 1. Summarization of demographics and clinical timelines of presented cases.

Descriptor	Case 1	Case 2	Case 3
Sex	Female	Female	Female
Age	66 years old	45 years old	36 years old
Comorbidities	Hypertension, NSTEMI, acute heart failure, chronic venous insufficiency	Hypertension Pneumonia Cardiomegaly associated with ASD and Eisenmenger syndrome	Congestive heart failure, peripartum cardiomyopathy
Onset-admission-intervention intervals	**Onset-admission (cardiac center):** 15 hours **Admission-intervention:** 24 hours (admission at weekend)	**Onset-admission (cardiac center):** 1 month **Admission-intervention:** No intervension (Sudden cardiac death before procedure)	**Onset-admission (cardiac center):** 10 days **Admission-intervention:** 2 hours
Key lab values	Leukocytosis 12,900/mcl Elevated hs-Troponin I	Leukocytosis (10,600/mcl) and an elevated D-dimer level of 585.46 ng/mL	Hb 10.4 g/dL, Thrombocyte 152,000/mcl
Imaging findings	Reduced RV systolic function, McConnell sign (+), thrombus at right, left, and base of main PA	Filling defect at entire lumen of MPA, RPA, and LPA MPA dilation with thrombus, ASD with bidirectional shunt, RA and RV dilation	LVEF 23.9% (BIPLANE), Migrated trhombus from RA, total occlusion of the lower lobe RPA
Interventions performed	CDT + Alteplase	Nil (sudden cardiac death before intervention)	1st: CDT + Alteplase (Nonresponse) 2nd: PTA
Short-term outcomes	Changes in TAPSE (+, 1.4 cm > 1.5 cm Day 1, 1.7 cm Day 2), oxygen saturation (97%)	Oxygen saturation (73%) following with sudden cardiac death	Oxygen saturation (77%>97% room air)
Long-term outcomes	Survive	Not Survive	Survive

ASD: Atrial Septal Defect, CDT: Cathether Directed Thrombolysis, LPA: Left Pulmonary Artery, MPA: Main Pulmonary Artery, NSTEMI: Non-ST Elevation Myocardial Infarct, PA: Pulmonary Artery, PTA: Percutaneous Transluminal Angioplasty, RA: Right Atrium, RPA: Right Pulmonary Artery, RV: Right Ventricle

In cases of acute PE, patients exhibiting unstable hemodynamics are classified into a

high-risk category. Conversely, in patients with stable hemodynamics, severity and risk stratification are assessed based on the presence of RV dysfunction, as detected via echocardiography or CTPA, along with elevated troponin levels. The first and third patients in this report are identified as having acute PE with unstable hemodynamics, meeting criteria for obstructive shock; the first patient presented with hypotension, recording a blood pressure of 60/40 mmHg, while the third had a blood pressure of 77/50 mmHg. The second patient, classified as having intermediate-low severity and risk, exhibited an sPESI score of 1, indicating tachycardia with a heart rate of 125 beats per minute.[2]

Echocardiography is not necessary for diagnosing PE in hemodynamically stable patients; however, it can be utilized to exclude other potential causes of dyspnea. In contrast, in patients with hemodynamic instability suspected of having acute PE, clear indicators of RV pressure overload, particularly with definitive echocardiographic findings such as the 60/60 sign, McConnell sign, or right heart thrombus, warrant immediate reperfusion interventions if a CTPA cannot be performed promptly, especially in cases with high suspicion and no alternative explanation for RV pressure elevation.[3] This was evident in the first case, where the hemodynamically unstable patient presented in shock, prompting echocardiographic evaluation to exclude other potential causes such as cardiac tamponade, acute valve dysfunction, LV dysfunction, aortic dissection, or hypovolemia. Notably, signs of RV dysfunction were identified, including reduced TAPSE and a McConnell sign, along with a thrombus in the RPA. In the third case, a thrombus was discovered in the RA, which subsequently resolved with significant hemodynamic deterioration. Conversely, the second patient, though hemodynamically stable, underwent echocardiography to rule out differential diagnoses contributing to their symptoms. This assessment revealed a bidirectional shunt due to an ASD, which could mimic RV pressure overload, typically associated with pulmonary embolism during RV function evaluation.

In the first case, the patient underwent a CTPA despite being eligible for immediate reperfusion therapy, given the positive McConnell sign observed on echocardiography and the unstable hemodynamics at the time. A CTPA was also conducted for the second patient to further assess the diagnosis of PE in individuals exhibiting a low to moderate probability of PE, along with elevated D-dimer levels. CTPA findings for the first patient revealed filling defects in both the right and left pulmonary arteries, confirming acute PE. Similarly, the second patient's CTPA displayed substantial filling defects throughout the pulmonary trunk and extending into the right and left pulmonary arteries. However, contrast was still evident distally in the RPA and LPA, thereby corroborating the diagnosis of PE. In contrast, the third patient

could not undergo CTPA due to the examination's unavailability during their acute episode of PE. This patient presented with unstable hemodynamics and demonstrated migration of a previously identified mobile thrombus in the RA. CTPA is a minimally invasive diagnostic tool that offers accuracy comparable to traditional pulmonary angiography, with reported sensitivities of 83% and specificities of 96%. In cases deemed to have a moderate to high probability of PE, the positive predictive value of CTPA escalates to 92-96%. This was exemplified in the first patient, classified with high probability, and the second patient with moderate probability, thereby providing conclusive evidence of PE in both cases.[3]

Pulmonary angiography remains the gold standard for diagnosing PE; however, its use has declined due to the high accuracy of CTPA. A definitive diagnosis of acute PE is established when there is direct evidence of a thrombus, characterized by either a filling defect or an interruption of blood flow within a pulmonary artery branch. This was evidenced in the first and third patients who underwent pulmonary angiography, which confirmed the presence of thrombus in the pulmonary artery.

Comparison of acute pulmonary embolism therapy selection between the three cases

The classification of severity and risk stratification for PE is crucial for determining the timing of therapeutic intervention as per the established algorithms.[3] In this case, the first and third patients were assessed using the first algorithm, while the second patient followed the second algorithm—the first patient presented with suspected high-risk acute PE characterized by unstable hemodynamics. An echocardiographic examination was conducted immediately, revealing RV dysfunction. Subsequently, a CTPA was performed after the patient's condition was stabilized with supportive therapies, including norepinephrine and dobutamine. It is recommended that CTPA be conducted promptly once hemodynamic stability is achieved with such support. The CTPA results indicated a filling defect consistent with thrombus in the pulmonary artery, prompting the decision to initiate immediate reperfusion therapy. Notably, RV dysfunction identified via echocardiography alone is considered adequate to commence reperfusion therapy in PE patients with unstable hemodynamics. This was illustrated in the third patient, where echocardiography conducted prior to embolism revealed a thrombus in the RA, further supporting the need for urgent intervention in such clinical scenarios.

In cases where PE is suspected and hemodynamic stability is present, the initial evaluation should include a D-dimer assay, particularly for patients categorized as having a low-to-moderate probability. Should the D-dimer results be elevated, a subsequent CTPA is warranted. Conversely, in patients deemed to have a high probability of PE, CTPA should be prioritized as the first-line diagnostic tool.[3] This approach was exemplified in our second

patient, who, despite stable hemodynamics, was assessed using a probability test that yielded intermediate results. The elevated D-dimer levels prompted a further evaluation with CTPA, which confirmed the diagnosis. Consequently, the patient was initiated on therapy for PE.

In cases of suspected PE accompanied by hemodynamic instability, the preferred approach is primary reperfusion therapy, which may involve systemic thrombolysis or surgical or transcatheter embolectomy. Administration of reperfusion therapy within 48 hours of symptom onset is crucial, as it has demonstrated a significant reduction in both mortality and the risk of recurrent PE.[3] In patients with acute PE, the risk of death and major bleeding complications (intracerebral hemorrhage) is lower with catheter-directed thrombolysis (CDT) compared with systemic thrombolysis. When compared with anticoagulation alone, CDT is associated with a lower risk of death but the same risk of intracerebral haemorrhage.[5–7] This suggests that CDT has a better safety profile than ST, making it recommended as first-line therapy in cases of acute PE.[6,7]

Before initiating primary reperfusion therapy in the initial patient, several supportive measures were implemented. Following the management algorithm for high-risk PE, this patient was identified as a suspected high-risk case. In cases classified as either high or intermediate probability, parenteral anticoagulants should be administered even prior to the availability of diagnostic test results. The preferred anticoagulant options are low-molecular-weight heparin (LMWH) or fondaparinux, as they present a lower risk of major bleeding and heparin-induced thrombocytopenia (HIT) compared to unfractionated heparin (UFH). Current guidelines limit the use of UFH primarily to patients exhibiting significant hemodynamic instability or those on the verge of hemodynamic decompensation who are scheduled to receive primary reperfusion treatment.[3] In the case of the first patient with unstable hemodynamics, the recommended parenteral anticoagulant regimen involved an initial bolus of 80 IU/kg body weight of intravenous heparin. The patient was classified as ARC-HBR 1 Major, indicating a high risk of bleeding. Consequently, CDT was selected over systemic thrombolysis. Despite a PE-CH score of 0 and a BACS score of 0, reflecting a low predicted risk of intracranial bleeding following thrombolysis, CDT was preferred due to the significant mortality associated with PE post-systemic thrombolysis, which ranges from 40-50%. Additionally, the thrombus in this patient remained at a proximal level, further supporting the choice of CDT.

The patient received 200 cc of normal saline intravenously, leading to an increase in blood pressure; however, the patient remained classified as hypotensive. In response, inotropic and vasopressor agents were administered, along with oxygen supplementation, due to desaturation with oxygen saturation levels below 90%. Typically, the initial approach to

manage hemodynamic instability includes enhancing RV preload through intravenous fluid boluses, as demonstrated in the first patient who was given normal saline to address hypotension. Nonetheless, excessive volume resuscitation can exacerbate RV failure by overstretching the RV, heightening wall stress, aggravating RV ischemia, diminishing contractility, and inducing further interventricular septal shift towards the LV. This shift can impede LV filling and systemic cardiac output. An initial trial of intravenous fluids is most effective in patients with no signs of elevated right-sided preload, particularly those with central venous pressure (CVP) under 15 mmHg. In contrast, for patients with a CVP exceeding 15 mmHg, volume administration should be avoided, and the initial intervention should focus on administering vasopressors and inotropics while awaiting definitive therapy.[8] Consequently, as normal saline administration did not improve the patient's hemodynamic status, the subsequent course of action involved administering Norepinephrine and Dobutamine.

In patients with high-risk PE, the ideal agent for hemodynamic support should enhance RV function through positive inotropic effects while ensuring adequate systemic arterial perfusion pressure. Norepinephrine, epinephrine, and dopamine exhibit a dual mechanism, acting as both inotropes and vasopressors.[8] Norepinephrine, in particular, has the capacity to improve systemic hemodynamics by increasing coronary perfusion without significantly altering pulmonary vascular resistance (PVR).[3] Thus, it is recommended as the initial agent for hemodynamic support in patients with high-risk PE. In cases where RV contraction needs to be improved to boost cardiac output, inotropic agents such as dobutamine become essential. Furthermore, the use of vasopressors is critical when administering these inotropic agents to ensure adequate end-organ perfusion.

Thrombolysis with CDT delivers thrombolytic agents directly to the pulmonary artery, utilizing a lower total dose than systemic administration and can be performed with or without ultrasound guidance. The use of ultrasonography in CDT enhances the binding of tPA to fibrin cross-links, accelerating thrombolysis. Small, randomized trials involving patients with intermediate-risk PE have demonstrated that ultrasound-facilitated CDT is more effective in reducing pulmonary artery pressure and right ventricular strain indicators on imaging compared to anticoagulation alone. Dosing regimens reported in various studies range from 8 to 24 mg of tPA administered over 4 to 24 hours.[9] Other analyses indicate that alteplase may be given at doses of 0.5 to 1.0 mg/hour into one or both main pulmonary arteries for durations of up to 24 hours, typically accumulating to a total dose of less than 30 mg and not exceeding 24 hours of infusion.[10] In the first and third patients, neither exhibited absolute contraindications to systemic thrombolysis. However, both presented high-risk bleeding profiles; the first patient

met one major criterion (hemoglobin at 10.2 g/dL) and one minor criterion (eGFR at 54 mL/min/1.73 m^2), while the second met one major criterion (hemoglobin at 10.4 g/dL). To mitigate the risk of intracerebral hemorrhage, which is associated with systemic thrombolysis, we opted for CDT in these cases, given the low dose of thrombolytic agents employed. Additionally, the patients were treated in a facility equipped with the appropriate infrastructure for CDT, solidifying its selection as the optimal therapeutic approach.

Local CDT enables the targeted infusion of low-dose thrombolytic agents into the pulmonary artery or directly onto the thrombus, with the option to fragment the thrombus prior to drug injection. Thrombolytic agents can be administered through a pigtail catheter for intrathrombus delivery, and nearly all thrombolytics are applicable in this manner. Although definitive evidence is lacking, it is recommended that CDT be initiated within 60 minutes of establishing the indication for the procedure (rather than from the time of PE diagnosis) in patients already at a healthcare facility equipped for CDT. For patients requiring transfer to such a facility, this intervention should commence within 90 minutes of the initial assessment. It is crucial to highlight that the allotted time frame pertains strictly to the clinical indication for CDT, not the diagnostic timeline of the PE. In cases of hemodynamic instability, immediate initiation of CDT is indicated. However, before proceeding with invasive interventions, a CTPA is advisable to evaluate the location of the embolus. Patients with acute lobar or proximal pulmonary emboli derive greater benefit from CDT compared to those with segmental disease. In scenarios where patients are hemodynamically unstable and unable to undergo CTPA, direct pulmonary angiography may be performed ahead of CDT.[10] This approach is consistent with the presentations of the initial and third patients referenced, who exhibited pulmonary emboli in the lobar arteries, highlighting the appropriateness of CDT in such cases.

In the context of performing CDT, it is essential to note that the administration of anticoagulants should continue post-procedure, unless there are absolute contraindications, while consistently monitoring activated clotting time (ACT) or activated partial thromboplastin time (aPTT) values. Currently, there is no clear guideline indicating whether heparin dosing should be adjusted during CDT.[10] Anticoagulants are typically prescribed following a standard 5-day regimen. After CDT, the transition to oral anticoagulants can occur once heparin administration has been completed.[3] Potential complications associated with this procedure include hemodynamic decompensation, respiratory failure, alveolar hemorrhage, pulmonary artery perforation, contrast-induced acute kidney injury, hemolysis, and hematoma formation at the vascular access site. These complications may vary based on the technique and system utilized.[10]

In the first patient, after 24 hours of CDT using rt-PA (Alteplase at 0.5 mg/hour), a follow-up pulmonary angiography demonstrated a reduction in the size of the RPA and LPA filling defects. This was accompanied by clinical improvement and enhanced RV systolic function, as indicated by TAPSE parameters obtained via echocardiography. These findings align with the fundamental objective of acute PE management: reducing RV afterload through the dissolution of pulmonary artery thrombus. Notably, in this case, complete thrombus removal is not typically required to achieve hemodynamic stabilization. Indicators such as increased systemic blood pressure and/or decreased heart rate reflect a reduction in pulmonary obstruction and enhanced RV dimensions and function, as substantiated by the elevated TAPSE values, which can result from partial recanalization. Consequently, in this initial case, reperfusion therapy with CDT can be deemed successful.

In the third patient, pulmonary angiography performed post-CDT using the rt-PA regimen (Alteplase at 1 mg/hour) revealed similar findings to those seen in the pre-CDT angiography. Specifically, a significant filling defect was noted, indicating a total occlusion in the inferior lobe of the RPA. Consequently, the decision was made to proceed with mechanical thrombectomy via PTA. Despite improvements in several hemodynamic parameters, including a mean arterial pressure (MAP) of 83 mmHg without support, the patient was still categorized as having failed reperfusion therapy. This was evidenced by persistent tachycardia and tachypnea, albeit with a slight reduction in severity. Therefore, an escalation in reperfusion therapy was warranted for this patient. Furthermore, research has shown that mechanical thrombectomy leads to a reduced incidence of perioperative bleeding complications and improved hemodynamic outcomes, including decreased pulmonary hypertension and myocardial infarction, compared to CDT. Additionally, long-term rates of chronic pulmonary hypertension and right heart failure were also found to be lower, underscoring the advantages of pulmonary thrombectomy over CDT in this scenario.[11] Although specific guidelines for escalating reperfusion therapy in high-risk acute PE are lacking, evidence suggests that early intervention (within 12 hours) correlates with improved outcomes, characterized by decreased hemodynamic decompensation, reduced intensive care requirements, and shorter hospital stays.[12] In this case, the implementation of early reperfusion management in the third patient had a significant impact on survival outcomes.

In patients with acute PE, "treatment failure" pertains to the ineffectiveness of primary reperfusion or anticoagulation therapies. Currently, there are no specific guidelines to evaluate treatment outcomes, whether in high-risk or non-high-risk categories. The assessment of treatment efficacy involves the monitoring of clinical indicators, including consciousness level,

systemic blood pressure, heart rate, respiratory rate, arterial oxygen saturation, and signs of organ hypoperfusion, such as decreased urine output and elevated lactate levels. Echocardiography may be employed to assess changes in right ventricular function. In hemodynamically unstable patients, the escalation of treatment regarding reperfusion therapy is influenced by several factors, including the availability of equipment, the expertise of the healthcare team, and logistical constraints.[10]

In recent years, mechanical thrombectomy has seen significant advances, enabling intervention without the need for thrombolysis. The primary aim of mechanical thrombectomy is to extract the thrombus from the pulmonary artery. Key indicators of clinical success include hemodynamic stabilization, resolution of hypoxia, and survival rates from PE, which stand at approximately 86.5%.[9] Mechanical thrombectomy utilizing the pigtail catheter for thrombus fragmentation is the predominant catheter-based intervention for acute PE, owing to its widespread availability, ease of use, and cost-effectiveness.[10] In this procedure, a modified pigtail catheter is manually rotated around a guidewire that exits through a side port. This technique can often be enhanced by the incorporation of a peripheral balloon, typically smaller than the arterial lumen's diameter. Such methods are particularly beneficial for hypotensive patients with proximal occlusions, as they facilitate the recanalization of central embolic obstructions, thereby rapidly restoring flow to the occluded pulmonary artery.[13]

The second patient presented with an intermediate probability of PE, which was subsequently confirmed by positive results from CTPA. In the risk stratification process, the patient was categorized as intermediate risk, despite the absence of troponin results, with a PESI class III classification (score of 95) and an sPESI of 1. Consequently, the patient was admitted for monitoring of hemodynamic status and was initiated on a maintenance dose of UFH at 18 IU/kg body weight intravenously, aiming for a target aPTT of 1.5 to 2.3 times the control value.[14] This patient was administered sildenafil to enhance pulmonary hemodynamics and cardiac output, aligning with findings documented in multiple studies. Evidence supports the efficacy of sildenafil in managing pulmonary hypertension, demonstrating benefits in both short-term and long-term outcomes at a dosage of 50 mg every 8 hours.[15,16]

When a patient with normotensive PE progresses to high-risk PE during anticoagulation therapy, this is classified as treatment failure, necessitating the immediate initiation of reperfusion therapy. Current guidelines advise monitoring patients exhibiting RV dysfunction on echocardiography or CTPA, along with a positive troponin test, for the first 2 to 3 days due to the potential for early hemodynamic decompensation and circulatory collapse. However, specific parameters for monitoring have yet to be established. Clinical indicators, such as

persistent or worsening tachycardia, hypotension, elevated respiratory rates, and signs of organ hypoperfusion (e.g., reduced urine output and increased lactate levels), commonly precede hemodynamic collapse. However, deterioration can occasionally occur abruptly without these warning signs. As it stands, there are no concrete criteria for assessing worsening in intermediate-risk PE cases; thus, the evaluation of anticoagulation failure relies solely on the absence of improvement after several days of anticoagulation. In scenarios involving patients with intermediate to high-risk PE, CDT should be considered if no progress is observed within 24 to 48 hours of initial anticoagulation.[10]

The treatment options presented in this report should be considered alongside the distinct triggering factors associated with each case. In the first case, the acute PE occurred following EVLA for varicose veins, a scenario that is rarely documented. A related case report from 2008 by Nwaeijke et al. also highlighted additional confounding risk factors, such as previous great saphenous vein ligation one year earlier, the presence of malignancy, and tributary variceal phlebitis. The patient was managed per British Thoracic Society (BTS) guidelines, given a Wells score of 3 (high-risk), and received intravenous heparin followed by a 6-month course of warfarin post-discharge.[17] Notably, confounding risk factors in our case could include heart failure and hypertension.[18] Despite the high-risk nature of both cases, the patient in this report was treated with more advanced therapy, incorporating CDT with Alteplase, which resulted in clinical improvement. These cases underscore the importance of investigating potential confounding risk factors and underlying causes without undermining the immediate treatment of PE.[17]

In the second case, the incidence of acute PE associated with ASD is relatively uncommon. A national database study revealed that ASD was present in only 0.4% of patients primarily diagnosed with acute PE. However, those with acute PE and concurrent ASD exhibited a heightened risk of adverse outcomes, including stroke, mortality, extended hospital stays, and post-discharge disability. This increased risk correlates with a greater likelihood of chronic kidney disease (CKD), congestive heart failure (CHF), valvular heart disease, peripheral vascular disease, a prior cerebrovascular accident (CVA), episodes of PE, atrial fibrillation/flutter, and obesity. Additionally, the ASD cohort experienced a higher incidence of cardiogenic shock, acute renal failure, and respiratory failure. Consequently, these patients were more frequently managed with advanced interventions such as balloon counter pulsation, extracorporeal membrane oxygenation (ECMO), and blood transfusions, which are critical in addressing acute PE.[19] As illustrated in the second case of this report, rapid deterioration followed the Valsalva maneuver, necessitating resuscitation efforts. The limitation of

resources, particularly ECMO, resulted in suboptimal resuscitation and contributed to an instance of sudden cardiac death in this case.[10]

In the third case presented, the patient exhibited PPCM alongside intracardiac thrombi, which was linked to acute PE. The incidence of acute PE in patients with PPCM is estimated to be between 2% and 6%.[20,21] Conversely, the occurrence of intracardiac thrombi in this population is reported to be 17.9%.[22] Identified risk factors for PE in PPCM patients include elevated coagulation factor levels, diminished activity of protein C and S, impaired fibrinolysis, and reduced LVEF.[21] Furthermore, the formation of intracardiac thrombi is associated with lower LVEF, higher hemoglobin levels, and increased platelet counts.[22] In this case, the patient presented with an LVEF of 23.9%, categorizing them as at risk for both intracardiac thrombus formation and acute PE. Additionally, PE in the context of PPCM is associated with elevated mortality rates. Key predictors of mortality in this condition encompass atrial fibrillation, non-ST elevation myocardial infarction (NSTEMI), chronic kidney disease, and cardiogenic shock.[20] Notably, these factors were absent in this case, suggesting that the implementation of reperfusion therapy may enhance survival outcomes for this patient, especially as treatment escalated from CDT to mechanical thrombectomy.

This case report has several limitations. Complete outcome parameters, including changes in TAPSE, oxygen saturation, RV:LV ratio, and pulmonary artery pressures, were not reported. Furthermore, quantitative changes in thrombus burden on angiography were not reported. Furthermore, long-term follow-up data, including anticoagulant regimen, functional status, and echocardiography and other imaging studies, were not reported.

Conclusion

Pulmonary embolism is a cardiovascular emergency with high mortality that requires immediate treatment based on risk stratification. This case series includes three patients with different profiles: (1) a patient with obstructive shock after an EVLA procedure who was successfully treated with CDT, (2) a stable patient with ASD and pulmonary hypertension who was treated with anticoagulation, and (3) a patient with peripartum cardiomyopathy with a right heart thrombus who required escalation with angioplasty after CDT failed. Lessons learned from this case series report emphasize the importance of risk-based therapy, including systemic thrombolysis or CDT for high-risk PE, anticoagulation for intermediate- to low-risk PE, and hemodynamic support with vasopressors/inotropic agents in cases of shock.

Conflict of Interest

Nothing to declare

Funding Sources

Nothing to declare

Ethics and Patient Consent

This case report was conducted by the ethical standards of the ethics committee of our institution. Written informed consent was obtained from each patient for the publication of this case report and any accompanying images.

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