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Case Report

Choosing an Appropriate Treatment Modality in Patients with Acute Massive Pulmonary Embolism and an Algorithm for Management

Mukherji J*, Burcar K and Freilich A
Loyola University Medical Center, Maywood, IL, USA

***Corresponding author:** Jayanta Mukherji, Loyola University Medical Center, Maywood, IL, USA

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Abstract

Patients suspected of Acute Massive Pulmonary Embolism (AMPE) require immediate attention as early diagnosis and management help reduce the high mortality associated with severe cardiogenic shock and or cardiac arrest. Death from AMPE can occur rapidly if the diagnosis is missed or the patient arrives to the hospital late. While unstable patients need to be resuscitated rapidly, choosing appropriate modalities of treatment needs to be an informed decision that could possibly impact patient morbidity and survival. We present a case report and discuss the treatment modalities used in our scenario.

Introduction

Most patients who present with AMPE usually present in extremis with cardiogenic shock. They need to be stabilized to undergo a rapid and reliable diagnosis to confirm these findings and then proceed to a quick management strategy. An understanding of underlying pathophysiology is essential in order to provide appropriate rapid resuscitation for the management of profound cardiogenic shock. Our case study illustrates the rationale in choosing the appropriate therapeutic modality for managing AMPE with hemodynamic instability.

Case Presentation

32-year male presented with acute right sided pleuritic chest pain and dyspnea after a period of prolonged immobilization following a ten hour long bus ride. He had not been diagnosed with any prior hypercoagulable disorder, but had a strong family history of venous thromboembolic disease in his father and sibling. He has had no other comorbidities. He was a 1/2 pack day smoker for 5 years. His symptoms were insidious with mild shortness of breath which progressively worsened over a day.

He presented with sinus tachycardia with HR of 120-130/min, RR of 28-30/min and systolic BP of 80-90 mm Hg. He was also hypoxemic and was on a non-rebreather at 10L/min oxygen with a SpO_2 of 92%.

Workup with CT angiogram confirmed the diagnosis of AMPE. (Figures 2,3). Imaging findings included massive, completely occlusive emboli within each main pulmonary artery extending distally into lobar and segmental arterial branches and right heart strain. Additionally, there were multifocal parenchymal opacities concerning for infarctions in both lungs.

Transthoracic Echocardiogram (TTE) showed severe right ventricular and Right Atrium (RA) dilation with significant global hypokinesis of the Right Ventricle (RV). An echogenic mass was also located in the right atrium and tricuspid valve (Figure 1).

It was decided that given the extent of a large clot burden in the pulmonary arteries, a catheter directed thrombolysis might not resolve the obstruction and may put the patient at risk of a longstanding right heart strain or failure from pulmonary hypertension caused by thromboembolism. The presence of a large RA clot was an additional factor against proceeding with catheter directed thrombolysis.

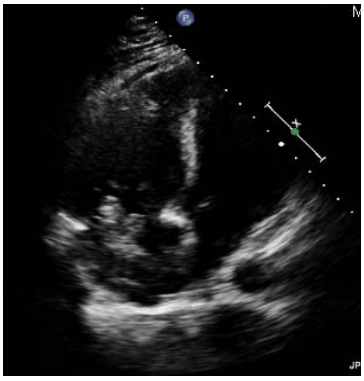

Figure 1: Transthoracic Echocardiogram (TTE); apical four chamber view. The right ventricle (seen on left side of screen) is distended with shifting of the interventricular septum to the left side. An echogenic mass is seen straddling across the tricuspid valve.

The patient's hemodynamic status was unstable and he was emergently taken to the operating room. He was induced with IV etomidate and rocuronium with rapid sequence intubation. He continued to have impaired oxygenation with PaO_2 of 50-57 mm Hg on mechanical ventilation and Fio_2 of 1.0. A right internal jugular and pulmonary artery catheter was placed which showed a Central Venous Pressure (CVP) exceeding 20mm Hg and a Mean Pulmonary Artery Pressure (MPAP) exceeding 55mm Hg. He was maintained on epinephrine (0.08-0.12 mcg/kg/min) and norepinephrine infusions (0.1-0.12 mcg/kg/min) to maintain a target Mean Arterial Pressure (MAP) greater than 60mm Hg. Inhaled nitric oxide was used for RV afterload reduction. Intraoperative Transesophageal Echocardiogram (TEE) showed a normal LV systolic function with ejection fraction of 50%, a dilated RA, and a RV with severely reduced RV function. The RA showed multiple echogenic masses straddling the tricuspid valve

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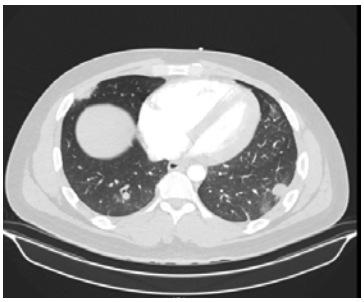

Figure 2: CT angiogram showing distended right ventricle with flattening of the interventricular septum and an underfilled left ventricle.

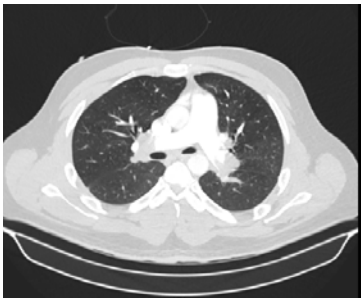

Figure 3: CT angiogram showing filling defects from thrombus occluding the right and left main pulmonary arteries.

with severe tricuspid regurgitation. The interatrial septum was bowed to the left with no interatrial communication.

The patient was emergently placed on Cardiopulmonary Bypass (CPB) and underwent bilateral pulmonary embolectomy and removal of the RA thrombus. Intraoperative findings revealed clots within each main pulmonary artery extending distally into lobar and segmental pulmonary arterial branches.

Difficulty was encountered with the inability to come off CPB, and TEE showed incomplete recovery of the RV function with dilatation and global hypokinseis. Hemodynamic support with multiple vasopressors and inotropes (epinephrine, dobutamine. and norepinephrine infusions) were inadequate to maintain MAP greater than 60mm Hg and sufficient organ perfusion. In order to support the RV, infusions included dobutamine 5mcg/kg/min, epinephrine 0.1-0.12 mcg/kg/min, norepinephrine 0.1-0.12 mcg/kg/min, and inhaled nitric oxide at 80 parts per million for RV afterload reduction. The patient was then placed on Venoarterial Extracorporeal Membrane Oxygenation (VA ECMO) to improve oxygenation and to achieve hemodynamic stability, while allowing time for the RV to recover.

In the ICU the patient was anticoagulated for VA ECMO and maintained with flows of 4L/min, sweep of 2-3 L/min. and Fio2 of 1.0. The patient's oxygenation and hemodynamic status stabilized by postoperative day 3, and we were able to wean vasopressors and continue with diuresis. We did not encounter excessive bleeding from use of heparin infusion started on postoperative day 1 for VA ECMO.

The patient's hemodynamic status improved, and we were able to wean off inhaled nitric oxide and vasopressors.

There was recovery of RV function by 72 hours, which permitted successful decannulation of VA ECMO. There were no complications, and the patient regained full recovery of end organs and functional status. The patient underwent Inferior Vena Cava (IVC) filter placement and was continued on full therapeutic anticoagulation with a heparin infusion.

Discussion

AMPE is referred to as thrombus occlusion of more than 50% of the pulmonary artery cross-sectional area or occlusion of two or more lobar arteries or clinically hemodynamic compromise or severe RV dysfunction detected by echocardiography [1]. The incidence is estimated to be between 4.5% and 10% of all cases of Pulmonary Embolism (PE) [2]. This life-threatening condition carries a high

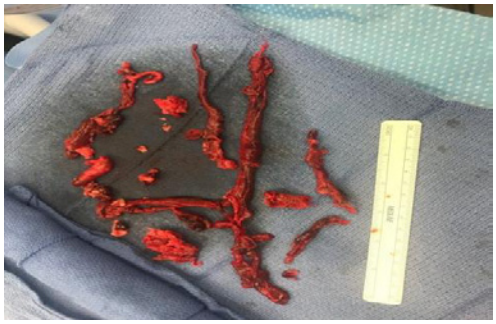

Figure 4: Large clots extracted from patient during Surgical Embolectomy.

mortality (30-50%) even with aggressive treatment [3].

The care of patients with AMPE needs to be prioritized as they could rapidly progress to sudden cardiac arrest with a delay in diagnosis and management. Even with aggressive treatment, a patient could develop cardio-pulmonary compromise. Poor prognostic indicators as described in our case include severe RV dilatation, hypokinesis and shock [4].

Choosing an optimal therapeutic strategy for patients with AMPE is controversial because to date there are no randomized controlled trials in support of an ideal treatment modality. Therapeutic anticoagulation is the mainstay treatment for all patients diagnosed with PE. However, RV dysfunction with impending cardiogenic shock warrants a more aggressive approach. Specific therapeutic modalities described to treat patients with AMPE include Surgical Embolectomy (SE), Thrombolytic Therapy (TL), and catheter-directed thromboembolectomy. Treatment modalities are guided by both a patient's underlying presentation and condition.

Thrombolytics

Thrombolytic therapy is an important consideration for AMPE in symptomatic patients with worsening respiratory failure or severe RV dysfunction. Systemic TL along with anticoagulation may be considered for massive PE according to the 2016 ACCP Antithrombotic Guidelines.

TL in patients with AMPE and cardiogenic shock has suggested a significant reduction of death or PE recurrence compared to heparin treated patients [5]. The use of TL has been validated in unstable patients diagnosed with AMPE and has been associated with a reduction in pulmonary-artery pressure, pulmonary vascular

Table 1: Algorithm for choosing treatment modality in patients diagnosed with Acute Massive Pulmonary Embolism (AMPE).

Acute Massive Pulmonary Embolism (APME)	
A: Hemodynamically stable	1. Consider thrombolytics (TL) if not contraindicated
	2. If thrombolytics are contraindicated, then consider surgical embolectomy (SE)
	3. SE can be considered first line of treatment at an experienced surgical center with or without evidence of RV strain
	4. If high risk for open heart surgery and thrombolytics are contraindicated, then consider catheter based intervention techniques
	5. Continue therapeutic anticoagulation with heparin/ enoxaparin +/- IVC filter
B: If hemodynamically unstable* (with presence of varying degrees of RV dysfunction*and/or myocardial injury*** is present)**	1. Consider TL unless contraindicated
	2. Consider emergent SE with failure of TL
	3. Failure of TL with or without the presence of RV dysfunction and myocardial injury
	4. Echocardiographic evidence of free floating thrombus within the right atrium, right ventricle, or patent foramen ovale with risk of imminent paradoxical embolism
C. Adjunct therapy in Hemodynamically unstable patients	**VA ECMO placement**
	1. VA ECMO if hemodynamic status remains unstable or deteriorates rapidly following TL or at any stage perioperatively during SE
	2. Consider VA ECMO early for end-organ failure and metabolic/respiratory acidosis even before AMPE treatment
	3. Cardiogenic shock from RV failure as evidenced by profound hypotension, dilated RV, elevated RV end diastolic pressure, elevated CVP, hepatic dysfunction, renal insufficiency
	4. Ongoing treatment of RV failure in conjunction with RV support with epinephrine, norepinephrine, dobutamine
	5. Hypoxemia, hypercarbia, acute respiratory failure, mechanical ventilation, intraparenchymal (lung) hemorrhage and failure to ventilate lung; use VA ECMO in conjunction with pulmonary vasodilators inhaled nitric oxide and inhaled epoprostenol 6. Continue therapeutic anticoagulation with heparin/enoxaparin +/- IVC filter in all of these patients.
***Criteria for hemodynamic instability**	a) Need for cardiopulmonary resuscitation
	b) Systolic blood pressure less than 90 mm Hg
	c) Signs of end-organ hypoperfusion (cold extremities, urinary output less than 30 mL/h, or mental confusion)
	d) Need for catecholamine infusion to maintain end organ perfusion.
** **Criteria for RV dysfunction**	**Echocardiographic**
	a) RV end-diastolic diameter greater than 30 mm (parasternal long-axis or short-axis)
	b) Hypokinesis of the RV free wall
	c) Tricuspid regurgitant jet velocity greater than 2.6 m/s
	CT scan
	Right/left short-axis diameter ratio greater than 0.9 (transverse plane)
*** **Myocardial injury**	Confirmed by a positive troponin I or T test

resistance, and hemodynamic recovery [6].

A favorable response to TL has been reported in 92% of these patients based on their clinical and echocardiographic improvement within the first 36 hour [7]. TL therapy is generally initiated within 48 hour of symptom onset, but it can still be beneficial to patients who have had symptoms lasting for 6-14 days [8]. Earlier reports suggest that TL may be considered in patients with PE associated with free-floating thrombi in the right heart if the risk of open-heart surgery is considered to be extremely high [9].

The major disadvantage of TL includes major bleeding risks in about 13% of patients. The most dreaded complication, intracranial hemorrhage, can occur in up to 3% of patients [25]. Long-term sequelae include recurrent emboli, failure to completely retrieve all of the thrombus materials, and inability to completely resolve the intra-luminal thrombus, with the potential risk of developing chronic pulmonary hypertension [1,10].

Contraindications to TL in patients with AMPE include trauma, surgery within the previous 3 weeks, history of hemorrhagic stroke or any cerebral ischemic event within 6 months, oral anticoagulant use, advanced hepatic disease, active peptic ulcer, pregnancy, and CPR [11,12].

Catheter-Directed Thromboembolectomy

Catheter-directed thomboembolectomy has been successfully used for the treatment of massive PE. It is a minimally invasive technique and is efficacious in removal of clots and recovery of RV function. The American Heart Association (AHA) guidelines for management of massive and submassive PE, iliofemoral deep vein thrombosis and chronic thromboembolic pulmonary hypertension, has recognized catheter-based interventions or Percutaneous Catheter-Based Thrombectomy (PCBT), as an alternative to

thrombolysis or surgical embolectomy [4].

Catheter-based interventions are considered as level C, class IIA, recommendations by AHA since there is no randomized trial of medical management [10,13]. The mechanism of the catheter involves using positive pressure for the saline spray and negative pressure to suck the clots using the Bernoulli principle. The procedure entails rheolytic thrombectomy, fragmentation, and aspiration thrombectomy [14].

Hybrid procedures where local thrombolysis can be administered along with mechanical embolectomy, appear to be the best option in managing AMPE [15].

Disadvantages include bleeding complications from anticoagulation, bradycardia, tricuspid regurgitation, mechanical hemolysis, fluid overload, and denudation of the intimate layer from use of catheter size exceeding 10 French [15]. Other complications include injury or perforation of the RV and pulmonary arteries, arrhythmias from catheter passage through the right heart, PE recurrence, pulmonary hypertension and acute pancreatitis [11]. Clot fragmentation has the potential to shower emboli distally into the peripheral pulmonary vasculature, and can lead to chronic thromboembolic pulmonary hypertension and decreased long-term survival [11,13]. The long-term implications of this therapeutic modality have not yet been fully studied.

Surgical Embolectomy (SE)

Mechanical removal of clots by Surgical Embolectomy (SE) may need to be considered to improve oxygenation and achieve hemodynamic stability. An emergency SE performed in a critically ill patient can be lifesaving as it results in swift removal of thrombus obstructing the main pulmonary artery and its branches, thereby reducing RV afterload and causing rapid improvement of RV function [16]. It has been utilized in failed TL.

There has been a paradigm shift with the implementation of SE early on rather than reserving this modality for patients as a last therapeutic resort. Operating early is preferable as SE is associated with an acceptable morbidity and mortality. Potential future complications such as recurrence of PE and chronic thromboembolic pulmonary hypertension can also be prevented [17].

Current indications for SE include patients with massive central PE with contraindications to thrombolytics and those with RV dysfunction and cardiogenic shock after receiving thrombolytic therapy (Class IIa; Level of Evidence C) [1]. An aggressive approach to early surgical intervention in severely compromised patients can be advocated since results and have been shown with low mortality [10,18].

Evidence points to a more favorable result with surgical intervention in hemodynamically stable patients with moderate to severe RV dysfunction on echocardiography [10,19]. Hemodynamically stable patients with massive central clot burden and signs of RV dysfunction on echocardiogram are being considered more frequently for surgery [20].

Another indication of SE includes the echocardiographic evidence of an embolus sited in a patent foramen ovale or present in the Right Atrium (RA). A free-floating thrombus seen on imaging within the RA or RV in patients with a patent foramen ovale poses an imminent risk for paradoxical embolization [21]. A massive thrombus seen proximally in the RV, main pulmonary artery and extra pulmonary branches of the pulmonary arteries are technically more amenable to surgical removal.

SE is not without its drawbacks. It carries a high mortality, particularly in elderly patients with multiple comorbidities (6% to 46%) [22]. SE in unstable patients carries a higher operative mortality compared to stable patients with pulmonary embolism (10% *versus* 4%) [23].

Conditions that can worsen outcomes and result in death following SE include interstitial pulmonary edema, RV failure, cardiac arrest before surgery requiring CPR, peripheral thrombus extension beyond the sub segmental pulmonary arteries, and massive lung parenchymal bleeding [24]. IVC filter placement, in addition to full anticoagulation with heparin or enoxaparin, may need to be considered early as recurrence of PE can occur soon after SE and is one of the most important causes of early postoperative mortality. The recurrence rate of PE can be as high as 5% [25].

VA ECMO

VA ECMO has been reported in multiple case reports and has been used for massive PE. With advances in ECMO technology, it has been increasingly used for patients unresponsive to standard treatment modalities or as adjunct or a bridge to SE. VA ECMO can be initiated if thrombolytic intervention fails or is contraindicated. Placement of VA ECMO as an adjunct to SE in the post-CPB period, as in our patient with acute cardiogenic shock, assists recovery of RV function [26]. It is preferable that ECMO be initiated early for patients with AMPE with hemodynamic instability since reports indicate dismal survival (29%) for patients where ECMO was initiated later in the setting of cardiac arrest [27].

ECMO cannot be utilized in all patients as it requires full anticoagulation to maintain the integrity of the system. There is always a concern for acute bleeding risk in patients having recently received thrombolytics. Recent surgery, hemorrhagic stroke, and prolonged cardiopulmonary resuscitation with failure to achieve end organ perfusion are known contraindications to ECMO. The role of ECMO has not be well defined in randomized control trials and has not received mention by 2016 CHEST Antithrombotic Therapy in the management of APME [28].

Critical Care Considerations

Management of cardiogenic shock from massive pulmonary embolism includes the following aspects: institution of mechanical ventilation and the use of ventilator strategies to lower airway pressures, optimization of oxygenation and gas exchange, and prevention of acidosis help prevent worsening of pulmonary hypertension.

Inotropes, diuretics, and pulmonary vasodilators (inhaled nitric oxide, prostaglandins) are used for RV support to help achieve circulatory stability.

Summary

A multidisciplinary approach with rapid diagnosis by imaging

with CT angiogram and echocardiography is essential when PE is suspected. Choosing the appropriate treatment modality depends on a patient's comorbidities, presentation, and hemodynamic status. See (Table 1). With advances of VA ECMO technology the decision to provide circulatory support to hemodynamically unstable patients should be considered early in order to avoid cardiac arrest [29].

SE may be a first treatment option in patients with AMPE in whom thrombolysis is contraindicated or those with refractory RV dysfunction and cardiogenic shock after receiving thrombolytic therapy. However, surgical experience at the treatment center should be factored in the decision process. Survival can be influenced by poor hemodynamic status or cardiac arrest in patients undergoing SE, and VA ECMO should be considered in these situations. Emergent SE should occur before cardiac arrest ensues in order to increase the likelihood of a successful outcome. Echocardiographic evidence of an embolus trapped within a patent foramen ovale and/or thrombus in the RA and RV supports the choice of surgical intervention.

TL should be considered if there are no contraindications. There is a concern for major bleeding including intracranial hemorrhage and incomplete resolution of thrombus and chronic pulmonary thromboembolism. Catheter based intervention has shown promise but no studies are available regarding long-term outcome in patients with APME.

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