

Extracorporeal Membrane Oxygenation for Failed TPA Therapy of Pulmonary Embolism

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Abstract

Pulmonary embolism may cause cardiac arrest secondary to obstruction of blood flow. Traditional treatment strategies include anticoagulation, thrombolysis, and mechanical extraction. Some advocate for support with extra corporeal membrane oxygenation (ECMO); however, surgical therapies are contraindicated following thrombolytics. We describe the emergent use of peripheral ECMO following thrombolytic therapy for a saddle pulmonary embolism associated with multiple episodes of cardiac arrest. The patient was stabilized with peripheral ECMO, anticoagulated and subsequently weaned from ECMO without any major bleeding complications. The administration of thrombolytics should not be a contraindication for ECMO in patients with massive pulmonary embolism associated with hemodynamic instability.

Introduction

A saddle pulmonary embolism (SPE) is defined as a thromboembolism spanning the region of the bifurcation of the pulmonary trunk. Adult SPEs are rare, accounting for approximately 2-3% of pulmonary embolism presentations [1]. The recommended intervention for pulmonary emboli in hemodynamically unstable patients is systemic fibrinolytic agents, specifically tissue plasminogen activator (tPA) [2]. ECMO can be used for cardiopulmonary support during embolism fibrinolysis in patients with repeated cardiac arrest or hemodynamic instability [3]. Traditionally, ECMO or pulmonary embolectomy has been considered relatively contraindicated following tPA administration due to the increased risk of bleeding associated with tPA [4].

Case Report

A 61-year-old man presented to the emergency department complaining of chest pain for two days. The EKG indicated sinus tachycardia with a right bundle branch block and a prior inferior infarct. The patient underwent cardiac catheterization, which was negative. Upon examination, the patient was dyspneic, normotensive, nauseous and diaphoretic with cyanotic extremities. Past medical history was unremarkable. The patient developed respiratory distress, was intubated and underwent a transthoracic echocardiogram (TTE), which revealed severely diminished right

ventricular systolic function and right ventricular dilation. Follow-up chest computed tomography (CT) with contrast revealed a large saddle embolus almost completely filling the left and right main pulmonary arteries and segmental branches. During admission to the CTICU in preparation for operative pulmonary embolectomy, the patient suffered cardiac arrest with pulseless electrical activity (PEA). tPA was administered for salvage a 50 mg IV bolus followed by 50mg infused over 90 minutes, but the patient to remained hemodynamically unstable. Despite high doses of inotropes, the patient suffered multiple episodes of cardiac arrest requiring cardiopulmonary resuscitation (CPR). A CentriMag (Thoratec Corporation, Pleasanton, CA) centrifugal pump and a Quadrox Softline coating oxygenator was utilized at the bedside via femoral-femoral arterio-venous cannulation. An 18-French Optisite (Edwards Lifescience) arterial catheter was placed via a percutaneous stick in the left groin. Following placement of the arterial catheter, 15,000 units of heparin were administered. A 25-French Biomedicus Medtronic venous catheter was placed over a guidewire into the right atrium. Distal extremity oxygenation was monitored, negating the need for an antegrade perfusion catheter; no limb ischemia was observed. ECMO flow of 4.2L – a cardiac index of 1.72L/min/m² – was achieved and the patient stabilized hemodynamically. Post-operative echocardiography confirmed

placement of the venous catheter in the right atrium. The patient was anticoagulated to a PTT of 60-80 seconds.

On post-operative day 4, the patient regained normal neurologic status and echocardiography demonstrated improved right ventricular function. ECMO was weaned and removed unremarkably. Right ventricular function was improved, and the patient maintained hemodynamic stability without any therapeutic agents. The patient was discharged 15 days after admission on coumadin, loratadine, and protonix. A hypercoagulability panel was positive for heterozygosity for Factor V Leiden thrombophilia. At 3-month follow up, the patient had no visible clot on CTA, and transesophageal echocardiography (TEE) demonstrated normal right ventricular size and function.

Comments and Conclusion

Pulmonary embolism (PE) is a relatively common condition resulting in obstruction of the pulmonary vasculature by an object (commonly a thrombus) from a distant location. SPEs account for 2.6% of PE presentations.¹ Our patient had a Factor V Leiden mutation, which increases the risk of a thrombotic event significantly when compared to the general population. PE presents in high- and low-risk variants, the difference of which is based on clinical presentation. Therapeutic management is dependent on the risk assessment of the PE. Patients presenting with hemodynamically stable PE are low-risk and have an approximate mortality rate of 8.1%.⁵ Patients presenting with hemodynamic instability are high-risk and carry a mortality rate of 47%.⁶ When PE is accompanied by hemodynamic instability and cardiac arrest requiring CPR, the mortality risk increases to approximately 65% [5]. The American College of Chest Physicians (CHEST) states that in patients with acute PE with hypotension and no high bleeding risk, systemic thrombolytic therapy is recommended.² When systemic thrombolytics are used, the mortality of hemodynamically unstable PE decreases to 15% [6]. Currently CHEST guidelines do not address worsening hemodynamics following tPA, and surgical interventions have traditionally been considered relatively contraindicated for three weeks following administration.⁴ ECMO utility has been suggested to reduce right heart overload, resulting in hemodynamic stabilization and allowance of sufficient time for complete thrombolysis to occur.³ In a case report, Pavlovic et al. demonstrated the efficacy of ECMO salvage in a patient with a massive PE where thrombolytic therapy was contraindicated [7]. In a previous series we had two patients who failed tPA therapy and

had worsening hemodynamics. Surgical pulmonary embolectomy was performed without any major bleeding sequelae [8].

Our patient presented with a massive SPE with hemodynamic instability and cardiac arrest requiring CPR, inotropic and pressor support. In accordance with currently recommended protocol, tPA was administered. The thromboembolism lysed and occluded the pulmonary microvasculature, leading to worsening hemodynamics and episodic cardiac arrest. Venous-arterial peripheral ECMO was utilized for salvage at the bedside. Although ECMO is historically contraindicated following tPA administration, initiation occurred without any bleeding complications. The patient recovered, and three days after ECMO initiation hemodynamic stability was restored. Based on ours and other's experiences, it has been demonstrated that ECMO salvage can be used after tPA therapy following PEA arrest secondary to SPE. Utilization of tPA and invasive procedures remains controversial, but we have demonstrated that hemodynamically unstable PE not responding to tPA therapy can successfully be followed by peripheral bedside ECMO to restore hemodynamic stability.

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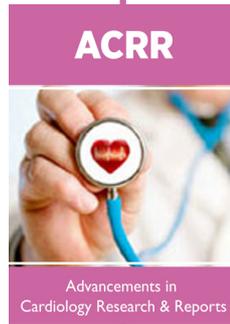
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