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Case Reports

Use of Extracorporeal Membrane Oxygenation–Facilitated Large-Bore Catheter Embolectomy in the Treatment of Acute Pulmonary Embolism Complicated by Shock

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Abstract

This article reports the case of a 42-year-old man who presented with a saddle pulmonary embolus complicated by normotensive cardiogenic shock. The patient was first stabilized with venoarterial extracorporeal membrane oxygenation. Then, while the patient was still on extracorporeal membrane oxygenation, thrombectomy with a large-bore catheter device was performed that resulted in a large decrease in pulmonary artery pressures and a clinically significant increase in cardiac index, with rapid clinical improvement. Complete recovery of the patient's cardiopulmonary status has been maintained at intermediate-term follow-up. This treatment strategy should be considered favorably in the treatment of patients presenting with pulmonary embolism complicated by cardiogenic shock.

Keywords: Pulmonary embolism; extracorporeal membrane oxygenation; shock, cardiogenic.

Case Report

Presentation and Physical Examination

A 42-year-old man presented to the emergency department with progressive dyspnea over the preceding 2 days. He denied chest pain. He was in moderate respiratory distress.

Blood pressure was 93/81 mm Hg. Heart rate was 130/min, and oxygen saturation on room air was 83%. Swelling of his right leg was noted, as was a scar from recent right Achilles tendon surgery. Arterial blood gas readings on presentation were pH 7.4, partial pressure of carbon dioxide was 4.4 kPa (33 mm Hg), and partial pressure of oxygen was 6.9 kPa (52 mm Hg). Cardiac troponin T level was elevated at 0.07 ng/mL, as were the B-type natriuretic peptide level (978 pg/mL) and lactate level (4 mmol/L). He was immediately put on a bilevel positive airway pressure machine, with prompt increase in oxygen saturation to 100%.

A 12-lead electrocardiogram showed sinus tachycardia. Chest x-ray showed mild prominence of the central pulmonary vasculature without overt pulmonary edema. Bedside echocardiography showed marked dilatation of the right ventricle (RV), with signs of RV pressure overload and moderate impairment of RV systolic function. Duplex ultrasound of the right lower extremity showed appearances suggestive of thrombosis of the popliteal vein.

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Medical History

The patient had no history of cardiac or respiratory disease. He had no personal or family history of venous thromboembolism. The patient had undergone surgical treatment for a ruptured Achilles tendon 2 months previously and was still immobilized in a surgical boot.

Differential Diagnosis

The differential diagnosis generated at admission after clinical evaluation, chest x-ray, and electrocardiography included pulmonary embolism (PE), pulmonary edema, pneumonia, acute asthma, and acute respiratory distress syndrome.

Technique

Computed tomography of the pulmonary artery demonstrated a saddle embolus involving the left and right main pulmonary arteries, with near occlusion of the left pulmonary artery (Fig. 1A). The calculated RV-to-left ventricle diameter ratio was markedly elevated, at 2.5 (normal, <1.0) (Fig. 1B). Heparin infusion then commenced, and the PE response team (PERT) was immediately activated. The PERT, concerned that the patient had several indicators for normotensive cardiogenic shock, recommended a right-heart catheterization study to determine the need for mechanical circulatory support before catheter-based pulmonary embolectomy. This study revealed a profoundly depressed cardiac index (1.4 L/min/m^2). Given this documentation of cardiogenic shock, the decision was made to proceed with venoarterial extracorporeal membrane oxygenation (VA-ECMO) placement to stabilize the patient before performance of large-bore aspiration thrombectomy with full mechanical cardiovascular support. Before VA-ECMO cannulation, a preemptive 5F catheter sheath was placed antegrade in the right superficial femoral artery to maintain antegrade flow. Extracorporeal membrane oxygenation cannulation was then performed percutaneously, under fluoroscopic and ultrasound guidance, with placement of a 19F cannula in the right common femoral artery and a 29F cannula in the right common femoral vein. The VA-ECMO circuit was then initiated at 4 L/min. Computed tomography of the pulmonary artery 24 hours later showed no clinically significant interval change in the size of the pulmonary embolus and demonstrated persistent

Key Points

- ECMO may facilitate treatment of the hemodynamically unstable patient with PE.
- Large-bore catheter embolectomy is a valuable treatment option in acute PE.
- The PERT plays a key role in deciding treatment strategy in complex PE cases.

Abbreviations and Acronym

ESC, European Society of Cardiology
FLASH, FlowTriever All-Comer Registry for Patient Safety and Hemodynamics
PE, pulmonary embolism
PERT, pulmonary embolism response team
RV, right ventricle
SCAI, Society for Cardiovascular Angiography and Interventions
VA-ECMO, venoarterial extracorporeal membrane oxygenation

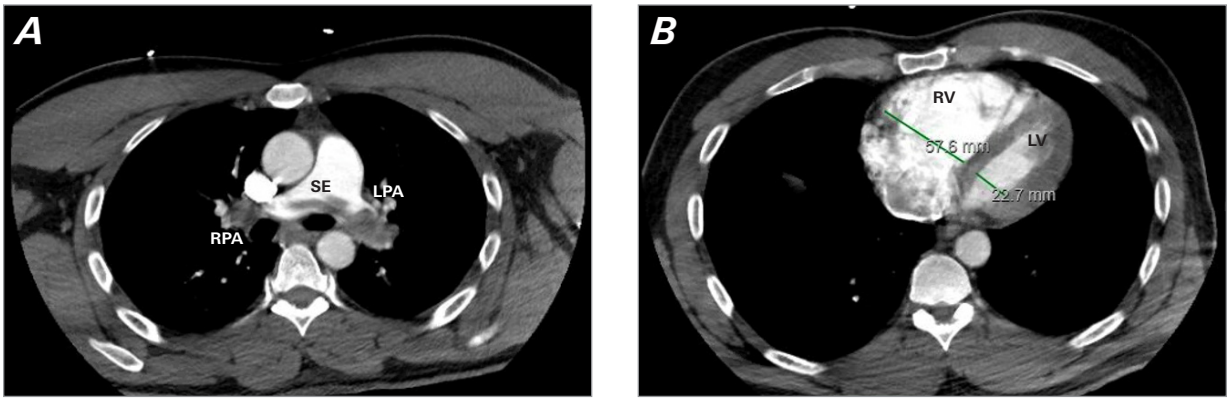

Fig. 1 Computed tomography pulmonary angiogram shows (**A**) a saddle embolus (SE) involving the left pulmonary artery (LPA) and right pulmonary artery (RPA), with near occlusion of the left pulmonary artery noted. **B**) Computed tomogram (axial imaging) of the heart demonstrates marked dilatation of the right ventricle (RV), with an increased RV-to-left ventricle (LV) diameter ratio of 2.5.

right-heart strain. Large-bore catheter thrombectomy was then performed via a 24F catheter sheath in the left femoral vein with the Inari FlowTriever (Inari Medical) (Fig. 2). Extensive clot was removed from the right and left pulmonary arteries (Fig. 3). Right-heart catheterization was then repeated; the cardiac index had increased to 2 L/min/m^2 (Table I). After thrombectomy, the patient was decannulated. The arterial and venous sheaths were removed via a surgical cutdown on the femoral vessels, with primary repair of the artery and vein.

Outcome

After sheath removal, the patient had no dyspnea; oxygen saturation was 100% on 2 L oxygen/min. Systemic blood pressure was recorded at 119/60 mm Hg; heart rate was 93/min. The next day, the patient was asymptomatic on room air and hemodynamically stable. Echocardiography showed that the RV was mildly dilated, with a mild reduction in RV systolic function. The patient was discharged home on day 4 on apixaban 5 mg twice daily.

Latest Follow-Up

Twelve months after discharge, the patient remains well, with no cardiopulmonary symptoms. Echocardiography shows complete resolution of the RV dilatation and normal RV systolic function.

Discussion

According to the American Heart Association classification scheme,[1] massive PE is defined as a systolic blood pressure less than 90 mm Hg, a drop of more than 40 mm Hg for at least 15 minutes, or a need for vasopressor support. The equivalent term adopted by the European Society of Cardiology (ESC)[2] is *high-risk PE*. *Submassive PE* is defined by the American Heart Association as RV strain without hypotension; the equivalent term used by the ESC is *intermediate-risk PE*.

The ESC then categorizes patients with intermediate-risk PE into 2 subgroups according to whether patients have both RV dysfunction and RV injury (intermediate risk–high) or only 1 or neither of these findings (intermediate risk–low).[2] This patient's systolic blood pressure was higher than 90 mm Hg, but he had both RV dilatation on echocardiography and elevated cardiac troponin levels; accordingly, he would be classified as having submassive (American Heart Association) or intermediate risk–high (ESC) PE. In such patients, anticoagulation

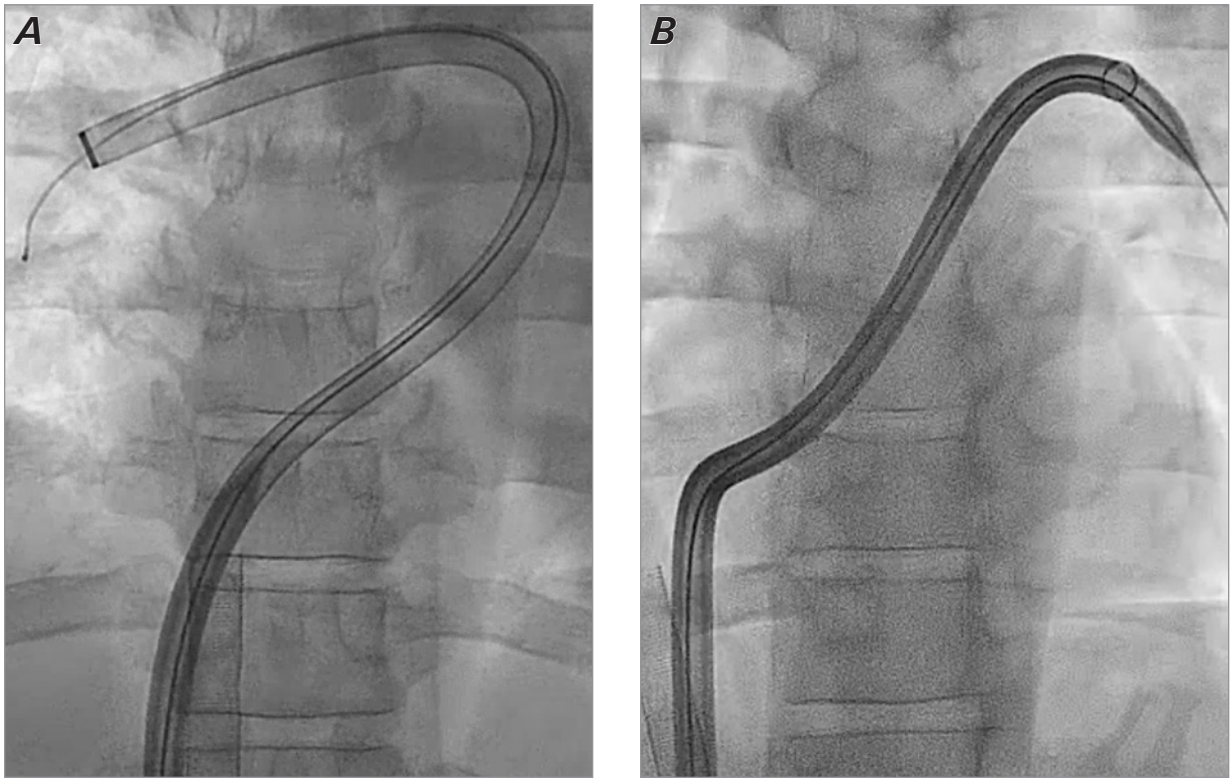

Fig. 2 Fluoroscopy image shows the Inari FlowTriever catheter in the (**A**) right pulmonary artery and (**B**) left pulmonary artery.

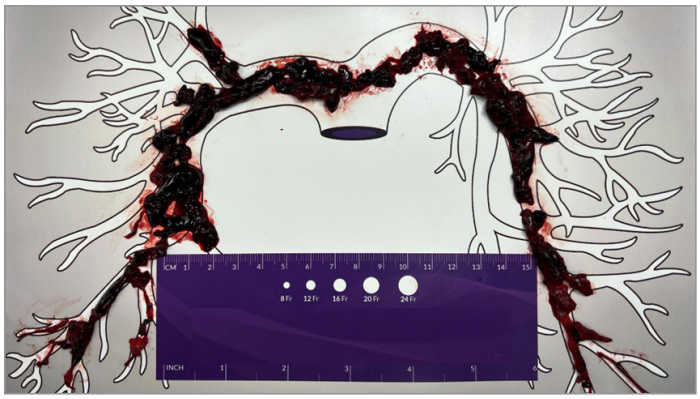

Fig. 3 Extensive thrombus retrieved by the thrombectomy device laid out over a schematic representation of the pulmonary artery branches. Hemodynamic measurements before and after large-bore catheter thrombectomy are detailed in Table I.

TABLE I. Hemodynamic Measurements Obtained at Right-Heart Catheterization Before ECMO Implantation and Immediately After Large-Bore Catheter Thrombectomy

Variable	Before ECMO implantation	After thrombectomy
Mean right atrium pressure, mm Hg	8	1
RV systolic/end-diastolic pressure, mm Hg	44/14	19/1
Pulmonary artery systolic/end-diastolic pressure, mm Hg	45/18	24/19
Mean pulmonary artery pressure, mm Hg	30	15
Cardiac index, L/min/m^2	1.4	2.0

Abbreviations: ECMO, extracorporeal membrane oxygenation; RV, right ventricle.

(preferably with direct agents, such as apixaban or rivaroxaban) is the standard treatment.[3] In recent years, large-bore catheter embolectomy has been found to be a useful alternative to anticoagulation therapy in such patients.[4-9] Catheter embolectomy devices include suction thrombectomy devices such as the Indigo system (Penumbra), the Inari FlowTriever device, and the AlphaVac device (Angiodynamics, Inc).[10] The initial plan was to proceed directly to the cardiac catheterization laboratory and immediately perform large-bore thrombectomy with the Inari FlowTriever device, but based on recent insights from the FlowTriever All-Comer Registry for Patient Safety and Hemodynamics (FLASH),[8] the PERT raised concerns that this patient, although normotensive, may have already developed cardiogenic shock. Specifically, analysis of this registry identified 6 factors associated with the development of this condition: elevated troponin and B-type natriuretic peptide level levels, moderately or severely reduced RV function, saddle PE, concomitant deep vein thrombosis, and tachycardia.[8] Strikingly, all 6 predictive factors were present in this patient. An alternative way of classifying the patient's clinical status would be according to the Society for Cardiovascular Angiography and Interventions (SCAI) shock classification schema; this patient would be best described as having stage C (confirmed) cardiogenic shock.[11,12]

Given these considerations, this patient, on arrival in the catheterization laboratory, first underwent a formal right-heart catheterization study. The finding of a profoundly depressed cardiac index confirmed the suspicion of established cardiogenic shock. Because of the possibility of the patient developing further hemodynamic deterioration, VA-ECMO rather than immediate thrombectomy was initiated.

Venoarterial ECMO is an effective treatment modality in the stabilization and prevention of hemodynamic collapse associated with massive PE. It is a reliable and quick method to decrease RV overload, improve RV function, and decrease pulmonary edema and resultant tissue hypoxia.[13-15] Successful use of VA-ECMO in the treatment of a patient with massive PE was first

reported nearly 30 years ago.[16] Subsequent single-center studies reported encouraging survival rates (>60%) in patients with massive PE treated with VA-ECMO,[17,18] but there has been an absence of randomized controlled trials examining the value of ECMO in the treatment of high-risk PE.[15] As a result, there are no particularly supportive guidelines for the use of ECMO in such patients. The ESC guidelines state, "V-A ECMO may be considered, in combination with surgical embolectomy or catheter-directed treatment, in patients with PE and refractory circulatory collapse or cardiac arrest."[2] The latter indication was given a weak (IIb, C) recommendation.[2] In the past decade, however, investigators have shown renewed interest in the potential benefits of VA-ECMO in the treatment of patients with high-risk PE and, in particular, patients with circulatory collapse or cardiac arrest.[15,19]

Use of ECMO for the reported patient was based on a strategy previously reported by Ghoreishi et al[20] at the University of Maryland. The authors adopted a standard treatment protocol in 41 patients with confirmed massive PE. According to this protocol, patients were supported for 3 to 5 days before echocardiographic re-evaluation of RV function. If RV function was found to have recovered, VA-ECMO was discontinued without further intervention. If persistent RV dysfunction was noted, surgical pulmonary embolectomy was performed. Interestingly, more than 70% of these study patients ultimately recovered with anticoagulation alone.[20]

After the patient was stabilized, thrombectomy was performed via the FlowTriever system. This system allows for a combination of manual aspiration thrombectomy through a Triever Aspiration Catheter (Inari Medical) using a 60-mL large-bore smart locking syringe to generate negative pressure and mechanical thrombectomy using self-expanding nitinol mesh disks to engage, disrupt, and deliver clot to the Triever Aspiration Catheter for extraction.[4] Excellent results with regard to the clinical utility of the Inari system in the treatment of patients with high-risk PE were observed in the recently reported FlowTriever for Acute Massive Pulmonary Embolism study.[6]

Patel et al[21] have reported the use of VA-ECMO followed by large-bore catheter embolectomy in the successful treatment of a patient with SCAI stage E (in extremis) cardiogenic shock secondary to massive PE. Specifically, their reported patient suffered a postoperative pulseless electrical activity arrest secondary to massive PE and needed 18 minutes of cardiopulmonary resuscitation before return of spontaneous circulation. In contrast, the reported patient was at an earlier stage of the shock process (SCAI stage C), and early use of mechanical circulatory support with ECMO was an anticipatory strategy. The goal was to prevent deterioration of the patient to SCAI stage D/E shock.

Treatment with systemic thrombolytics has been recommended as primary therapy in patients with PE who do not have contraindications to this therapy and are at high risk of death, as was the case for this patient.[2,3] Because of the high bleeding risk associated with systemic thrombolysis, however, thrombolysis was not considered for this patient. Specifically, thrombolysis for massive PE is associated with a 10% risk of severe bleeding and a 1.7% risk of intracranial hemorrhage.[2] Moreover, unsuccessful thrombolysis has been reported in 8% of patients with high-risk PE.[2] Large-bore mechanical thrombectomy devices such as the device used to treat this patient may be a superior alternative treatment modality because they can enable rapid cardiopulmonary recovery without thrombolytic bleeding risk, as evidenced by the recently reported FlowTriever for Acute Massive Pulmonary Embolism trial.[6]

In the FLASH study, hemodynamics were measured immediately before thrombectomy and again at least 5 minutes after thrombectomy.[7] In the normotensive shock group of patients, there was a modest but clinicaly significant increase in cardiac index (median, 1.87-1.93 L/min/m^2) and a clinically significant decrease in mean PA pressure (median, 33-24 mm Hg). In this patient, hemodynamics were measured at only 2 points: immediately before insertion of ECMO and 10 minutes after thrombectomy. The increase in cardiac index (from 1.4 to 2.0 L/min/m^2) and decrease in mean pulmonary artery pressure (from 30 to 15 mm Hg) observed in this patient were markedly greater than the median results seen in the FLASH study.[8] This finding likely reflects the additional effects of VA-ECMO and thus supports the idea that ECMO may indeed enhance the salutary hemodynamic effects of mechanical thrombectomy. Encouragingly, at intermediate-term follow-up, complete recovery of the patient's cardiopulmonary status has been maintained.

Conclusion

This case report describes a treatment strategy that should be considered favorably in the treatment of

patients presenting with PE complicated by cardiogenic shock. This treatment strategy includes early recognition of the development of shock and rapid interventional treatment. The latter combines use of a long-standing mechanical circulatory support system (VA-ECMO) and the evolving technology of large-bore mechanical thrombectomy devices. It is important to note, however, that VA-ECMO is limited to a few medical centers. Moreover, after implantation, it is critical to have a multidisciplinary team of perfusionists, ECMO-trained physicians, and nurses to perform constant patient monitoring and to troubleshoot the device. Few centers have such extensive resources.[14]

Article Information

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