The strategies for management of massive pulmonary embolism (PE) are diverse, and limited by a paucity of data from randomized controlled trials. Institutional preferences shape management of massive PE, with most institutions selecting between surgical embolectomy and systemic thrombolysis. In addition, systemic thrombolytics may be used in conjunction with, or substituted by, catheter-directed approaches including thrombectomy, fragmentation, directed thrombolysis, and ultrasound-assisted thrombolysis. In patients who fail to respond to first line interventions and develop refractory cardiogenic shock secondary to right ventricular (RV) failure, options for escalation of care are limited. Sparse case reports suggest managing refractory RV failure from PE with peripheral extracorporeal membrane oxygenation,1–3 whereas another case report describes the use of a surgically placed right ventricular assist devices (RVAD) after surgical embolectomy.4 Here we describe a patient with persistent cardiogenic shock and RV failure after a massive PE despite treatment with systemic thrombolysis, who was rescued with the percutaneous placement of an RVAD followed by additional catheter-directed thrombolysis.

A 48-year-old man with recent abdominal surgeries, including an ileocolostomy 1 month prior to presentation, had a witnessed syncopal event at home. The patient was found in the field to be unresponsive, with a systolic blood pressure of 70 mm Hg. On arrival to the hospital, the patient had regained consciousness, although his systolic blood pressure remained <75 mm Hg. An electrocardiogram on arrival showed sinus tachycardia at a rate of 110 bpm and a new right bundle branch block. A computed tomography angiogram showed extensive thrombus within the distal main pulmonary arteries (PA) bilaterally, extension of the clot into the segmental upper and lower bilateral pulmonary arteries, and an RV to left ventricle ratio of 1.7 (Figure 1). A bedside echocardiogram revealed an under-filled left ventricle, a normal left ventricular ejection fraction, and a severely enlarged RV with significant impairment in global RV systolic function (Figure 2). His initial treatment included intravenous heparin, multiple vasoactive medications, and fluid resuscitation.

He was deemed to be a poor candidate for surgical embolectomy. Catheter-based thrombectomy procedures were considered but systemic thrombolysis was chosen as the first therapeutic intervention. The patient received 100 mg of intravenous alteplase over 2 hours. Despite thrombolysis, the patient developed progressive acidemia with a pH of 6.98 and hypotension despite support with 4 vasoactive agents. Although arterial oxygenation remained stable, there was clinical evidence and laboratory evidence of hypoperfusion, including cool extremities, rising creatinine, and an elevated lactic acid, consistent with refractory shock.

The patient underwent a right heart catheterization that showed severely elevated filling pressures with an RA pressure of 19 mm Hg, and PA pressures of 53/28 mm Hg. Pulmonary angiography revealed that after systemic thrombolysis, much of the proximal thrombus seen on computed tomography had moved distally. A bedside venous ultrasound and an intra-procedural iliac venogram showed patent common femoral and iliac veins. RV support was accomplished by placing a...
21F intake cannula from the left common femoral vein into the right atrial-vena cava segment and a 21F Tandem Heart outflow cannula from the right common femoral vein into the proximal left PA. During the same procedure, the decision was made to pursue additional low dose catheter-directed thrombolysis via an infusion catheter placed into the left and right lower PAs via the right internal jugular vein. At the end of the procedure, urine output increased, systolic blood pressure augmented to 90 to 100 mm Hg, and clinical evidence of shock improved. The patient was maintained on heparin with a partial thromboplastin time goal of 60 to 80 seconds.

The vaspressors were weaned, and the RVAD was successfully removed after 6 days. Lower extremity ultrasound showed bilateral deep vein thrombosis, and an IVC filter was placed. At this point, the patient was bridged from unfractionated heparin to warfarin. After a 26-day hospitalization, the patient was discharged on room air with no cognitive, renal, or cardiac impairments. Two months later, echocardiography revealed normalization of the RV function and estimated PA systolic pressures (Figure 3).

The optimal treatment strategies for refractory cardiogenic shock after initial management of massive PE are unclear. Use of extracorporeal membrane oxygenation has been reported in few cases where available. Extracorporeal membrane oxygenation bypasses the entire pulmonary circulation and allows time for thrombi to recannulate or dissolve. RVAD also temporarily bypasses the failed RV and allows time for the thrombotic burden to improve, but, unlike extracorporeal membrane oxygenation, does not bypass the abnormal pulmonary vasculature. However, in this case with massive PE without refractory hypoxemia, we found that cardiac output and hemodynamic parameters improved with mechanical support of the RV alone. This case illustrates the successful use of a percutaneous RVAD for a patient with persistent cardiogenic shock after systemic thrombolysis for massive PE.

Disclosures
None.

References

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Percutaneous Right Ventricular Assist Device for Massive Pulmonary Embolism
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