

Ultrasound-Accelerated Thrombolysis and Extracorporeal Membrane Oxygenation in a Patient With Massive Pulmonary Embolism and Cardiac Arrest

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A 72-year-old man presented to the emergency department in cardiogenic shock; 4-days before he was diagnosed with right femoro-popliteal deep vein thrombosis after trauma. During routine monitoring and diagnostic workup, cardiac arrest with pulseless electric activity rapidly ensued and cardiopulmonary resuscitation was initiated. A transthoracic echocardiogram showed severe right ventricular dilatation and acute pulmonary embolism was suspected. No Return of Spontaneous Circulation was obtained after 10 minutes of cardiopulmonary resuscitation. The patient was transferred to the cardiac catheterization laboratory under resuscitation with autopulse (ZOLL, Chelmsford, MA) and femoro-femoral veno-arterial extracorporeal membrane oxygenation (ECMO; PLS Maquet GmbH, Rastatt, Germany) was percutaneously initiated with a 23 French (Fr) venous cannula and 17 Fr arterial cannula with restoration of systemic blood flow and oxygen delivery (5 L/min, 4000 rpm). Baseline pulmonary angiography demonstrated a large amount of thrombus in both the right (Movie I in the online-only Data Supplement) and the left pulmonary arteries (Figure 1; Movie II in the online-only Data Supplement). The contralateral femoral vein was cannulated with two 6 Fr sheaths and 2 Ekosonic ultrasound-accelerated thrombolysis (EKOS) catheters were placed directly into the thrombus of both pulmonary arteries (Figure 2). Local thrombolysis facilitated by ultrasound was initiated with infusion of recombinant tissue-type plasminogen activator at a rate of 0.5 mg/h for 24 hours in each EKOS catheter. After 6 hours of treatment, at full ECMO flow, transesophageal echocardiography evaluation showed persistence of right ventricular dilatation and dysfunction (tricuspid annular plane systolic excursion, 9 mm; Tissue Doppler imaging, 7 cm/s). Because of the lack of hemodynamic improvement, a 1-mg bolus recombinant tissue-type plasminogen activator was administered bilaterally. According to our institutional protocol with special regard to the risk of access-related bleeding,¹ systemic anticoagulation was started after 10 hours with bivalirudin, at a rate of 0.025 mg/kg per hour and purified antithrombin supplementation was given to maintain

antithrombin activity >100%. Despite the large bore vascular accesses, no bleeding was noted and the patient required only 1 U of packed red blood cells. After 1 day, TEE showed complete recovery of right ventricular function and the patient was successfully decannulated the day after, with manual compression of vascular accesses. Unfortunately, anoxic brain damage as a result of the cardiac arrest was evident and the patient was discharged from hospital with severe neurological sequelae.

Massive pulmonary embolism includes not only hypotension or shock, but also pulselessness requiring cardiopulmonary resuscitation.² Resuscitation guidelines suggest therapy with thrombolytics in patients with pulmonary embolism and cardiac arrest³; however, in case of refractory cardiac arrest, because of the inherent risk of bleeding of systemic

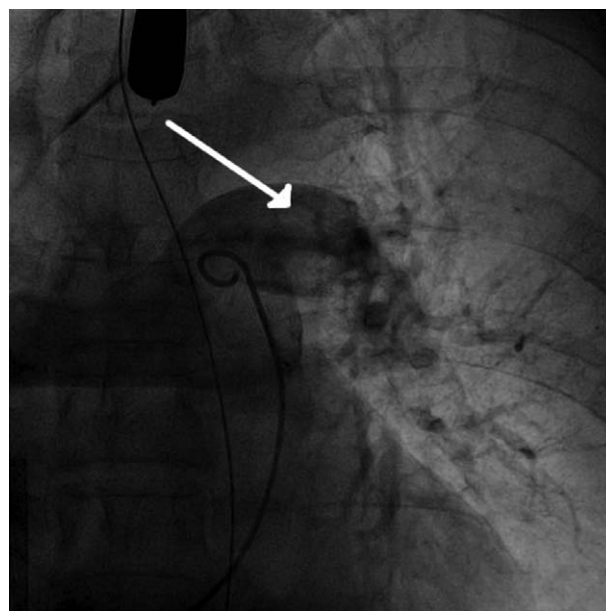


Figure 1. Pulmonary angiography, a thrombus burden in the left pulmonary artery (white arrow).

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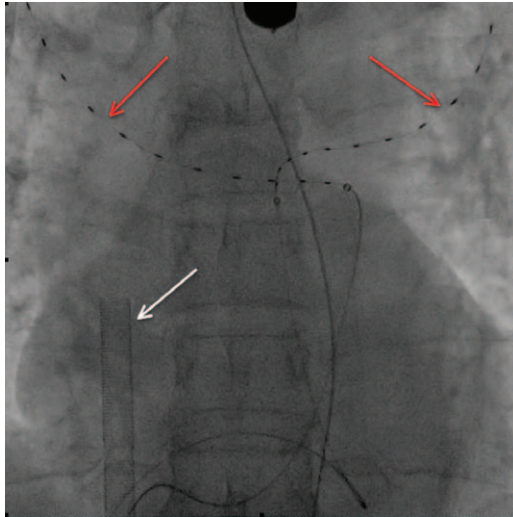


Figure 2. Demonstrating the ekosonic ultrasound-accelerated thrombolysis catheters (red arrows) placed in both pulmonary arteries and the veno-arterial extracorporeal membrane oxygenation cannulae (white arrow).

thrombolysis, no further options can be offered to patients with treatment failure.²

ECMO is recommended to provide pulmonary and circulatory support for the emergency treatment of patients with

massive pulmonary embolism and cardiac arrest. It decreases right heart volume and allows recovery of ventricular function and optimizes oxygen transport by improving cardiac output and oxygen content. However, systemic thrombolysis is generally considered an absolute contraindication for ECMO, in fact disastrous bleeding complications can occur particularly during and after ECMO placement. Patients can be supported with ECMO while endogenous thrombolysis proceeds with concomitant heparin administration, but the time lag for recovery cannot be foreseen and eventually, ECMO-related complications can ensue; thrombolytic agents may hasten hemodynamic recovery and, therefore, weaning from ECMO, lowering the risk for complications.

EKOS enhances catheter-directed thrombolysis by accelerating the fibrinolytic process via the application of ultrasound. Improving the efficiency of the thrombolytic process reduces the treatment time and total lytic dose delivered. The risk of an associated bleeding complication, which is extremely increased by the concomitant ECMO procedure, is, therefore, reduced.^{4,5}

We report a case of a patient with cardiac arrest because of acute massive pulmonary embolism, managed with percutaneous ECMO and EKOS. In conclusion, ECMO may give cardiopulmonary support, whereas EKOS can lyse the emboli with a lower risk of bleeding. We think that this combined therapeutic strategy could be used in this clinical scenario

Cardio Pulmonary Resuscitation in Pulmonary Embolism

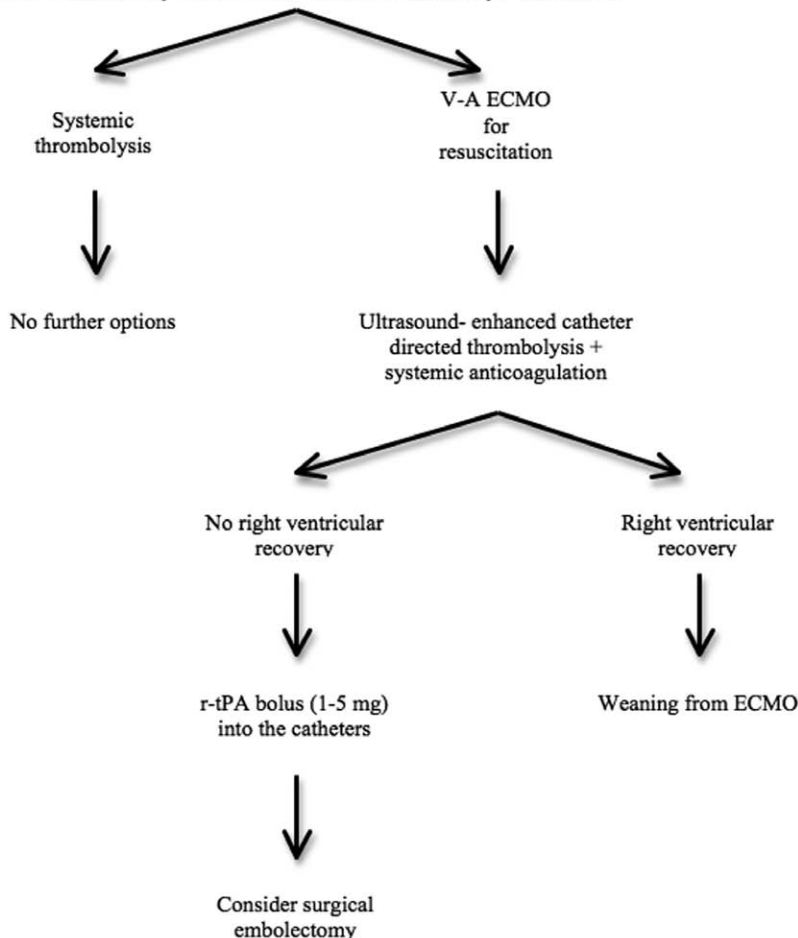


Figure 3. Algorithm for management of massive pulmonary embolism with cardiac arrest. V-A ECMO indicates veno-arterial extracorporeal membrane oxygenation; and rtPA, recombinant tissue-type plasminogen activator.

(Figure 3) in centers that have these facilities. In the light of this experience, the use of these devices together warrants further studies.

Disclosures

None.

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KEY WORDS: ECMO ■ pulmonary embolism ■ cardiac arrest

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Legends for the Video files

Video 1: Pulmonary angiography, a thrombus burden in the right pulmonary artery

Video 2: Pulmonary angiography, a thrombus burden in the left pulmonary artery