Massive pulmonary embolism with intra-hospital cardiac arrest and full recovery of right ventricular function after veno-arterial extracorporeal membrane oxygenation therapy: a case report

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Background
Massive pulmonary embolism (PE) with shock constitutes a life-threatening disease, challenging physicians with the need for fast decision-making in an emergency situation. While thrombolytic treatment or thrombectomy are considered the treatment of choice in high-risk PE, these strategies might not be able to unload the right ventricle (RV) fast enough in some patients with severe cardiogenic shock.

Case summary
We present a case of a patient with massive bilateral central PE who presented in cardiogenic shock, rapidly deteriorating to cardiac arrest. After successful re-establishing spontaneous circulation, the patient remained highly unstable, necessitating a treatment strategy ensuring a quick stabilization of the circulation. Therefore, we decided to use veno-arterial extracorporeal membrane oxygenation (vaECMO) as a supportive strategy allowing for autolysis of the lung to dissolve the thrombi (bridge to recovery). We were able to wean the patient from vaECMO support within 4 days and documented a complete recovery of right ventricular function on echocardiography before hospital discharge.

Discussion
The concept of vaECMO treatment alone might be a valuable alternative in selected patients with massive PE and cardiogenic shock, in whom thrombolytic therapy might not unload the RV fast enough.

Keywords
Veno-arterial extracorporeal membrane oxygenation  •  Pulmonary embolism  •  Right ventricular function  •  Case report
Learning points

- A thorough anamnesis as well as transthoracic echocardiography can provide important information suggesting pulmonary embolism (PE) and therefore help to guide further therapy.
- Heparin-induced clot dissolution and the high spontaneous fibrinolysis capacity of the lung lead to a fast resolution of thrombi and subsequent decrease in right ventricle (RV) afterload.
- Supportive therapy with veno-arterial extracorporeal membrane oxygenation might be a reasonable alternative in selected patients with PE presenting in severe shock or cardiac arrest, in whom thrombolytic treatment might not unload the RV fast enough.

Introduction

The symptoms and prognosis of patients with acute pulmonary embolism (PE) vary significantly depending on the extent of embolism, ranging from incidental findings on computed tomography (CT) to cardiac arrest due to acute pressure overload and consequent output failure of the right ventricle (RV). Stable patients are treated with anticoagulation and depending on further risk assessment might even be released to the ambulatory setting. Treatment options in patients with severe, haemodynamic relevant PE usually include thrombolytic treatment, catheter-based therapy, or surgical embolectomy. However, in case of a cardiac arrest or severe haemodynamic instability, supportive therapy using modern veno-arterial extracorporeal membrane oxygenation (vaECMO) therapy might help stabilize patients with otherwise fatal outcome.

Timeline

| Time         | Event                                                                 |
|--------------|-----------------------------------------------------------------------|
| Day 1        | Admission to emergency department in cardiogenic shock due to massive pulmonary embolism, quickly deteriorating to cardiac arrest. Return of spontaneous circulation after 40 min of resuscitation and subsequent implantation of veno-arterial extracorporeal membrane oxygenation (vaECMO) for circulatory support in ongoing cardiogenic shock. vaECMO removal and thrombectomy and reconstruction of the right femoral artery using a bovine patch cath lab to initiate supportive therapy with a vaECMO. After vaECMO implantation via the right femoral artery and vein, the circulation was stabilized immediately and the catecholamines could be tapered quickly. Coronary artery disease was excluded by coronary angiography. The subsequent cranial CT of the head (cCT), thorax, and abdomen revealed a bilateral massive central PE. However, intracranial bleeding was excluded. However, a comminuted fracture of the alveolar bone was diagnosed as a result of the initial head impact trauma. On arrival at our intensive care unit (ICU), the patient was already without catecholamine support with an established vaECMO flow of 3.5 L/min. The first blood sample taken at the ICU revealed elevated liver enzymes (ASAT/GOT 956 U/L, ALAT/GPT 784 U/L) as well as an increase in serum creatinine (1.5 mg/dL) as laboratory signs of beginning multiorgan failure as a result of cardiogenic shock. Furthermore, elevated levels of high-sensitivity assayed troponin T (972 pg/mL; reference value < 14 pg/mL) and N-terminal pro-B-type natriuretic peptide (NT-proBNP) (600 ng/L; reference value < 155 ng/L) were documented as indicators of high-risk PE. The initial arterial blood gas analysis after admission to the ICU documented a regular gas exchange and decreasing lactate values (4.6 mmol/L).
| Day 4        | Extubation of the patient                                             |
| Day 5        | Transfer from intensive care unit to intermediate care ward           |
| Day 9        | Discharge from hospital with complete recovery of right ventricular function on transthoracic echocardiography |
| 6 months     | Normal biventricular function on transthoracic echocardiography and normal N-terminal pro-B-type natriuretic peptide levels in blood serum; symptom-free patient |
| 6 months follow-up | Normal biventricular function on transthoracic echocardiography and normal N-terminal pro-B-type natriuretic peptide levels in blood serum; symptom-free patient |

Case presentation

A 42-year-old female patient was admitted via our emergency department after a sudden loss of consciousness at home, followed by an impact trauma of the head. Upon arrival of the emergency physician on site, the patient was somnolent and was immediately transferred to our tertiary care hospital. A quick cardiovascular examination revealed arterial hypotension, sinus tachycardia, tachypnoea, and cold and dry hands and feet indicating severe shock. Shortly after arriving in the emergency department, the circulatory situation deteriorated rapidly and the patient suffered cardiac arrest due to pulseless electrical activity. After 40 min of resuscitation with rapid, uncomplicated intubation, spontaneous circulation was restored. However, the patient remained highly unstable with high doses of catecholamines and significantly impaired oxygenation. A venous blood gas analysis obtained shortly after return of spontaneous circulation showed the following values: pCO2 77.4 mmHg, pH 6.86, HCO3− 13.9 mmol/L, BE -20.6 mmol/L, Na+ 135 mmol/L, K+ 4.6 mmol/L, and lactate 14.7 mmol/L. The electrocardiogram (ECG) showed a sinus tachycardia with a right bundle branch block (Figure 1). Echocardiography revealed signs of an acute RV overload and a comminuted fracture of the alveolar bone was diagnosed as a result of the initial head impact trauma. On arrival at our intensive care unit (ICU), the patient was already without catecholamine support with an established vaECMO flow of 3.5 L/min. The first blood sample taken at the ICU revealed elevated liver enzymes (ASAT/GOT 956 U/L, ALAT/GPT 784 U/L) as well as an increase in serum creatinine (1.5 mg/dL) as laboratory signs of beginning multiorgan failure as a result of cardiogenic shock. Furthermore, elevated levels of high-sensitivity assayed troponin T (972 pg/mL; reference value < 14 pg/mL) and N-terminal pro-B-type natriuretic peptide (NT-proBNP) (600 ng/L; reference value < 155 ng/L) were documented as indicators of high-risk PE. The initial arterial blood gas analysis after admission to the ICU documented a regular gas exchange and decreasing lactate values (4.6 mmol/L).

After application of 5000 international units of unfractionated heparin as bolus therapy during vaECMO implantation, further anticoagulation was administered with continuous parenteral infusion of unfractionated heparin with an activated partial thromboplastin time target of 60–80 s. Body temperature was...
Figure 1 Twelve-lead electrocardiogram recorded after successful resuscitation recorded at a paper speed of 25 mm/s. (A) Limb leads and augmented limb leads revealing a right axis deviation. (B) Precordial leads revealing the typical rsR’-pattern of the right bundle branch block.
The patient was admitted to our emergency department in cardiogenic shock as a consequence of massive PE, quickly deteriorating to cardiac arrest. While thrombolytic treatment can be administered without delay, embolectomy requires some beforehand preparation, making it less suitable for use in absolute emergency situations such as circulatory arrest. However, even with currently recommended 2-h regimens for thrombolytic therapy, thrombolysis and subsequent decrease of RV afterload and haemodynamic improvement still requires time. Furthermore, it is known that the haemodynamic benefits of thrombolytic treatment are restricted to the first few days after PE with no benefit on long-term outcome in individuals with intermediate-risk PE. Therefore, thrombolytic treatment is only recommended for patients with high-risk PE. Heparin-induced clot dissolution and the high spontaneous fibrinolysis capacity of the lung lead to a fast resolution of thrombi in PE even without thrombolytic treatment. As a result, RV afterload is substantially reduced in the first few days after PE, allowing fast weaning off vaECMO support.

Prior reports on the use of vaECMO in patients with massive PE have been inconclusive. Based on single centre experiences vaECMO treatment alone has been proposed as a sufficient strategy in high-risk PE, independent of any reperfusion strategy. On the contrary, based on a multicentre series of 52 cases Meneveau et al. postulated that vaECMO therapy alone is not justified in PE, but might be beneficially in complement to surgical embolectomy. However, among the patients with cardiac arrest, no difference in mortality according to treatment strategy was observed.

Treatment with vaECMO alone might be associated with an increased risk for chronic thromboembolic pulmonary hypertension (CTEPH) due to incomplete thrombus resolution. However, at 6 months follow-up, the patient presented asymptomatic with echocardiographic normal RV function, no signs of RV dysfunction on ECG and non-elevated NT-proBNP levels, the combination of which has been suggested to accurately exclude CTEPH. Nevertheless, patients with vaECMO treatment alone should be followed-up closely for signs of CTEPH according to current guidelines.

We hypothesize that in selected patients with massive PE presenting in severe shock or cardiac arrest, in whom thrombolytic treatment might not unload the RV fast enough, supportive therapy with vaECMO (as a ‘bridge to recovery’ concept) might be a reasonable alternative as it immediately stabilizes the circulation preventing further secondary organ damage due to impaired tissue oxygenation.

Additional thrombolytic therapy before or after vaECMO target INR 2.0–3.0) because of the known heterozygous factor V Leiden mutation and insufficient data for non-vitamin K oral anticoagulants in these patients. Taking into account the recurrent thrombotic events, we recommended an indefinite continuation of the therapy. Transthoracic echocardiography before discharge documented a normal biventricular function with no signs of RV overload (Figure 3 and Supplementary material online, Videos). All initially elevated laboratory parameters were within normal ranges at discharge, including NT-proBNP.

Discussion

The patient was transferred to our intermediate care ward after 9 days and was finally discharged after a total of 19 days. We switched anticoagulation to oral therapy with Phenprocoumon (Marcumar);

cooled to 33 °C for 24 h for neuroprotection, and the fracture of the alveolar bone was treated by the colleagues of oral and maxillofacial surgery. A total of three erythrocyte concentrates had to be transfused because of diffuse bleeding on vaECMO therapy. Compression ultrasonography revealed a deep vein thrombosis of the lower left leg.

During the ICU stay, RV function improved rapidly enabling vaECMO removal on Day 4 (duration of vaECMO therapy 77 h). Due to prior detection of a thrombus in the femoral artery, a surgical thrombectomy and reconstruction using a bovine patch were performed in the same procedure. After extubation on the next day a left hemiparesis was observed. A CT showed no sign of a stroke, but magnetic resonance imaging of the head revealed a signal alteration in the area of the basal ganglia on both sides, compatible with hypoxic damage, most likely as a consequence of the initial cardiac arrest. Neurological deficits decreased quickly and at hospital discharge only a slight weakness of the left body side remained (Cerebral Performance Category I).

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.implantation does not seem reasonable to us as this would further increase the high bleeding risk during vaECMO therapy. However, further research is warranted regarding advantages and disadvantages of such an approach.

Lead author biography

Stephan Camen completed his studies at the Medical University of Münster (Germany) in 2015 and is currently in specialist training for internal medicine and cardiology at the University Heart and Vascular Center Hamburg (Germany). His main scientific interest lies in the epidemiology of atrial fibrillation and stroke as well as in the heart–brain interaction. His work has been published in journals such as Europace and Herz.

Supplementary material

Supplementary material is available at European Heart Journal - Case Reports online.

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We would like to thank the patient for agreeing to the publication of this article.

Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as Supplementary data.

Consent: The author/s confirm that written consent for submission and publication of this case report including image(s) and associated text has been obtained from the patient in line with COPE guidance.

Conflict of interest: none declared.

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