Extracorporeal Cardiopulmonary Resuscitation for Intraoperative Massive Pulmonary Embolism Complicating Hip Surgery: A Rescue Bridge to Recovery

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Received: 10-20-2015
Accepted: 11-17-2015
Published: 12-01-2015
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Abstract
Fat embolism syndrome is a clinical complication commonly associated with traumatic bone fractures. The pathophysiology is not yet completely understood, but seems to be of multifactorial determination. The only intervention possible after detection of FES is supportive care of the involved organ systems. Here we report the case of successful intervention with a veno-arterial extracorporeal membrane oxygenation in a patient with peri-operative FES and circulatory arrest during surgery for an acetabulum fracture.

Keywords: In-Hospital Resuscitation; Refractory Cardiac Arrest; Extracorporeal Membrane Oxygenation (ECMO); Extracorporeal Life Support; Extracorporeal Cardiopulmonary Resuscitation (E-CPR)

Introduction
Patients sustaining pelvic traumas are known to be at increased risk for developing thromboembolic complications [1]. Following orthopaedic surgery for traumatic acetabular fractures the incidence of fatal and non-fatal pulmonary embolism (PE) may be as high as 2% and 10%, respectively [2-4]. Catheter-directed intrapulmonary thrombolytic therapy, catheter fragmentation of the thrombus, rheolytic thrombectomy, and surgical thrombectomy are available treatment options for thrombotic PE in the post-operative setting [5]. However, there are no specific treatment options for PE due to circulation of fat globules leading to the fat embolism syndrome (FES) [6,7]. Only supportive care is feasible once FES is diagnosed, using the criteria described by Gurd and Wilson (table 1) [7,8].

Extracorporeal membrane oxygenation (ECMO) has been shown to be an effective supportive treatment option for circulatory and respiratory support in patients developing cardiogenic and respiratory failure [9]. Here we report the successful treatment of refractory circulatory failure secondary to echocardiographic diagnosed PE caused by FES by means of extracorporeal resuscitation (E-CPR) in a patient with an acetabular fracture. This article is submitted with written consent.
A 67-year old male was admitted with a left-sided acetabular fracture. The fracture involved the acetabular roof, anterior wall and anterior column with protrusion of the femoral head through the quadrilateral plane. The patient underwent surgical reduction and fixation of the acetabular fracture through the ilio-inguinal approach several days following admission due to the sub-acute nature of the injury and the further CT assessment of the fracture and the hemodynamically stable condition of the patient. Preoperative assessment revealed no relevant prior medical history and no use of any medication prior to this hospital admittance.

After establishing standard anesthetic monitoring including ECG, oxygen saturation and invasive arterial blood pressure measurement, general anesthesia was induced with 0.5 mcg/kg sufentanil, 1.5 mg/kg propofol and 0.6 mg/kg rocuronium. After uncomplicated endotracheal intubation the patient was ventilated with controlled ventilation with tidal volumes of 7-8 ml/kg, positive end expiratory pressure of 5 cm H2O, respiratory rate of 12/min and a fraction of inspired oxygen of 0.4 which resulted in normocapnia and normal oxygen saturation.

Anesthesia was maintained with sevoflurane at a minimum alveolar concentration of approximately 0.8. Perioperative analgesic regime contained continuous intravenous administration of s-ketamin, lidocaine and boluses of sufentanil.

Initial blood pressure measurements were stable prior to and after induction with a mean arterial pressures between 60 and 75 mmHg.

During the reduction of the quadrilateral plane of the acetabular fracture, the procedure was complicated by sudden arterial hypotension and a decrease in end-tidal CO2 from 4.0 kPa to 1.9 kPa, refractory to volume resuscitation and administration of dobutamine, norepinephrine and epinephrine, necessitating mechanical and pharmacological resuscitation.

Transesophageal echocardiography confirmed the suspicion of massive PE (Figure 1) exhibiting floating emboli in the dilated right ventricle with elevated right ventricular pressure.

PE caused by FES was suspected because of the clinical course such as petechial rash and sustained blood pH<7.3 in combination with a traumatic acetabular fracture and surgical manipulation.

After 75 minutes of resuscitative efforts with short-term return of spontaneous circulation (ROSC) and high dose vasopressor medication, circulatory arrest was considered to be refractory and E-CPR was initiated applying full support femoral veno-arterial ECMO by surgical cutdown after which the patient was transported to the intensive care unit (ICU).

The clinical course in the ICU was further complicated by renal failure requiring temporary renal replacement therapy, a bacteraemia with enterobacteriaeae, a hospital-acquired pneumonia confirmed by radiographic imaging, critical illness neuropathy and heparin induced thrombocytopenia.

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Discussion

FES is an ill-defined clinical entity that arises from the systemic manifestations of fat emboli within the microcirculation [8]. In case of traumatic fractures movement of bone fractures leads to bone marrow release in the peripheral microcirculation [10]. Once a patient develops FES the only treatment is supportive care of the involved organ systems [6]. In order to diagnose FES the Gurd and Wilson criteria can be used. In the 1970s they observed and described three major and five minor features (table 1). Gurd et al stated that the diagnosis of FES could be determined if one major and at least four minor criteria are present [8]. Thus, FES may manifest itself with the major features of respiratory insufficiency, cerebral dysfunction and a petechial rash, but it has been described with a variety of clinical signs.

Why should an Emergency Physician be Aware of this?

FES is difficult to diagnose for any physician because of the great variety of clinical symptoms with serious consequenc -
treatment options for PE due to FES, the use of ECMO may be a novel lifesaving option when other options fail [6, 13, 14].

Table 1. Diagnosis of fat embolism syndrome using the Gurd and Wilson major and minor criteria: one major and four minor criteria/laboratory findings required for diagnosis.

<table>
<thead>
<tr>
<th>Major features</th>
<th>Respiratory insufficiency</th>
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<td>Cerebral involvement</td>
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<td>Petechial rash</td>
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<td>Minor features</td>
<td>Pyrexia</td>
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<td>Tachycardia</td>
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<td>Retinal changes</td>
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<td>Renal changes</td>
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<td>Thrombocytopenia</td>
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<td>Fat macroglobulaemia</td>
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Declaration of Interest section

There was no conflict of interest declared by any author. The authors alone are responsible for the content and writing of the paper.

References


