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Clinical paper

Extracorporeal membrane oxygenation in severe trauma patients with bleeding shock☆

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ABSTRACT

Aim of the study: Death to trauma is caused by disastrous injuries on scene, bleeding shock or acute respiratory failure (ARDS) induced by trauma and massive blood transfusion. Extracorporeal membrane oxygenation (ECMO) can be effective in severe cardiopulmonary failure, but preexisting bleeding is still a contraindication for its use. We report our first experiences in application of initially heparin-free ECMO in severe trauma patients with resistant cardiopulmonary failure and coexisting bleeding shock retrospectively and describe blood coagulation management on ECMO.

Methods: From June 2006 to June 2009 we treated adult trauma patients ($n = 10$, mean age: 32 ± 14 years, mean ISS score 73 ± 4) with percutaneous veno-venous (v-v) ECMO for pulmonary failure ($n = 7$) and with veno-arterial (v-a) ECMO in cardiopulmonary failure ($n = 3$). Diagnosis included polytrauma ($n = 9$) and open chest trauma ($n = 1$). We used a new miniaturised ECMO device (PLS-Set, MAQUET Cardiopulmonary AG, Hechingen, Germany) and performed initially heparin-free ECMO.

Results: Prior to ECMO median oxygenation ratio (OR) was 47 (36–90) mmHg, median $paCO_2$ was 67 (36–89) mm Hg and median norepinephrine demand was 3.0 (1.0–13.5) mg/h. Cardiopulmonary failure was treated effectively with ECMO and systemic gas exchange and blood flow improved rapidly within 2 h on ECMO in all patients (median OR 69 (52–263) mm Hg, median $paCO_2$ 41 (22–85) mm Hg. 60% of our patients had recovered completely.

Conclusions: Initially heparin-free ECMO support can improve therapy and outcome even in disastrous trauma patients with coexisting bleeding shock.

1. Introduction

Severe trauma is a leading cause of death in young adults.[1,2] Fatal injuries without any treatment option (e.g., cervical spinal injury and aortic rupture) result in immediate death at the scene. Life-threatening complications in the early course are bleeding shock and/or severe respiratory failure following chest trauma or massive blood transfusion. The early goal in trauma care is to combat shock. Bleeding shock can be treated effectively on scene by controlling the source of bleeding and rapidly initiating fluid resuscitation. In cases of severe trauma, early damage control surgery and extensive blood transfusion are immediately necessary.[3] About 15% of polytrauma patients are in need of massive blood transfusion (MBT) defined as more than 10 units of packed red blood cells (PRBC). The prognosis of trauma patients receiving MBT is considered to be poor.[4–8] Even if massive blood transfusion (MBT) is effective in the treatment of hemorrhagic shock, it is associated with potential complications[9,10] like severe acidosis and hypothermia. Hypothermia (body temperature < 34 °C) has deleterious effects on blood coagulation in trauma patients and when occurring in conjunction with metabolic acidosis can result in a mortality rate up to 90%.[11–14] MBT can overwhelm the patient's cardiocirculatory function resulting in the early decrease of cardiac output and pulmonary gas exchange. Late complications can appear as transfusion-related lung injury (TRALI) which magnifies the impairment of pulmonary gas exchange by blunt chest trauma and pulmonary contusions.[12] Both-, MBT and pulmonary contusion can lead to acute respiratory distress syndrome (ARDS). ARDS is most frequently observed in the early course of intensive care in patients with severe trauma and shock. The mortality rate in these patients reaches up to 40% despite advanced ventilatory treatment.[15,16] The use of extracorporeal membrane oxygenation (ECMO) as lifesaving treatment in respiratory failure was introduced by the pioneer work of Robert Bartlett in 1972[17] and revolutionised the treatment of resistant

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Table 1
Median and minimum–maximum of arterial pressure, blood pH, blood lactate and vasopressor support before, 2 h and 24 h on ECMO ($n = 10$). MAP: mean arterial pressure; OR: oxygenation ratio (paO_2 [mm Hg]/FiO_2).

Measurement	Before ECMO	2 h on ECMO	24 h on ECMO
MAP [mm Hg]	68 (45–90)	74 (56–92)	72 (52–84)
Norepinephrine [mg/h]	3.0 (1.0–13.5)	0.9 (0.0–5.0)	0.75 (0.0–2.5)
Lactate [mg/dl]	60 (25–105)	68 (11–122)	73 (9–126)
OR [mm Hg]	47 (36–90)	69 (52–263)	144 (48–218)
$paCO_2$ [mm Hg]	67 (36–89)	41 (22–85)	34 (29–45)
pH	7.21 (6.89–7.47)	7.41 (7.30–7.61)	7.43 (7.30–7.59)

hypoxemia in patients worldwide.[18,19] Depending on the site of cannulation, ECMO takes over the gas exchange function of the lung (veno-venous ECMO) or provides additional circulatory support (veno-arterial ECMO).[17–19] Despite ongoing technical improvement of ECMO devices like centrifugal pumps and heparin coating of the circuits (which allows a reduction of systemic heparinisation), bleeding is still the most common complication in patients on ECMO. Bleeding can be induced by cannulation of the vessels, underlying coagulation disorders or the full-dose heparinisation commonly used.[20] Since the first ECMO in a trauma victim was performed by Hill et al.,[20] there have been several reports on ECMO in the subsequent course of post-traumatic respiratory failure in patients with blunt chest trauma.[21,22] However, the use of ECMO in severe trauma victims with preexisting bleeding shock is still unusual.[23] A recently published report on ECMO support in post-traumatic ARDS patients highlights the effort to extend this lifesaving technology to trauma patients.[23] We report our first experiences in application of initially heparin-free ECMO in severe trauma patients with resistant cardiopulmonary failure and coexisting bleeding shock. We reviewed our data retrospective and describe cannulation procedures and blood coagulation management during emergency v-v and v-a ECMO.

2. Methods

In addition to being a level I trauma centre, our institution provides an interdisciplinary specially trained ECMO team consisting of a senior perfusionist, consultant anaesthesiologist with special skills in cardiothoracic anaesthesia and emergency medicine and on demand one cardiac surgeon. This specialised team allows round-the-clock extracorporeal life support for indoor and out-of-centre use.[19] The indication for v-v ECMO support in this report was resistant hypoxemia (OR < 100 mm Hg) despite advanced respiratory and positive end-expiratory pressure management, transfusion regime and chest tube placement. Indications for v-a ECMO support were persisting shock despite fluid resuscitation, blood transfusion and vasopressor support. Shock was defined as inadequate blood flow with signs of tissue hypoperfusion despite fluid resuscitation and vasopressor support. If massive blood transfusion MBT (defined > 10 units of packed red blood cells PRBC) was necessary shock was defined as bleeding shock. Contraindications for extracorporeal life support in our trauma patients were fatal cerebral lesions and states of observed prolonged hypoxemia (e.g., resuscitation on scene in cases of vehicle entrapment) or potentially fatal preexisting disease. When possible, we performed trauma CT-scan before the decision was made regarding extracorporeal life support to identify injury patterns (e.g., unstable cervical spine injury), as well as options for clinical improvement and main vessel pathology (e.g., aortic dissection). We thus assessed the patient's options for clinical improvement. In cases of severe trauma without complete trauma CT-scan, it is necessary to protect the patient's cervical spine especially during emergency cannulation procedures using jugular vessel access. Before setting a patient "on-pump", the ECMO team must be convinced that no improvement in cardiopulmonary function by other means is possible. Depending on the patient's priority in trauma care and his actual cardiopulmonary deterioration, we initiated extracorporeal life support in our patient group either in the emergency room (ER) or the operating room (OR) during damage control surgery. The percutaneous cannulation procedure was carried out by Seldinger technique without routinely use of skin incision to prevent cannula site bleeding. In leading hypoxic pulmonary failure with preserved cardiocirculatory function, we used femoro-jugular veno-venous (v-v) cannulation. In cases of circulatory failure or cardiopulmonary resuscitation (CPR) we performed femoral–femoral veno-arterial (v-a) cannulation. Depending on the patient's biometric data, we used a 21 or 23 Fr cannula (Femoral cannula, DLP Medtronic, Minneapolis) for venous outflow in v-v and v-a ECMO and a 15 or 17 Fr cannula (Novaport™, Novalung, Talheim, Germany) for venous and arterial backflow in v-v and v-a ECMO. In femoro-jugular veno-venous ECMO, the tip of the outflow cannula was placed in the inferior vena cava and the inflow cannula was placed in the superior vena cava superior. To avoid recirculation within the ECMO circuit, it is necessary to prevent venous "inflow" proximity to venous "outflow". For veno-arterial ECMO, the tip of the arterial cannula (inflow) was positioned in the common iliac artery or the distal abdominal aorta, whereas the tip of the venous cannula (outflow) was placed in the inferior vena cava. To initiate extracorporeal membrane oxygenation, we used a new miniaturised ECMO device (PLS-Set, MAQUET Cardiopulmonary AG, Hechingen, Germany) and performed initially heparin-free ECMO. The PLS-Set has a priming volume less than 600 ml normal saline and consists of a plasma-resistant polymethylpentene membrane oxygenator (Quadrox PLS™) and a centrifugal pump (Rotaflow™). The system is heparin-coated (Bioline™) "tip-to-tip" and has a 14 days CE approval. The Rotaflow drive and steering unit incorporates flow sensing and bubble detecting. The circuit includes a special shunt line for drug and volume application and, if required, an effective high-speed fluid resuscitation line. The centrifugal pump and the membrane oxygenator are mounted on a specialised lightweight holder system which allows fixation in close proximity to the patient. The Rotaflow drive unit has an integrated battery pack and can act as a standalone device during patient transfer. The oxygenator also acts as a heating or cooling device to control the patient's blood temperature. In ECMO, pump flow rates were up to 4.5 l/min and gas supply to the oxygenator was initially pure oxygen with a flow rate of up to 12 l/min to utilise maximum gas exchange capacity. Shortly after successful implementation of the ECMO pump, gas flow rates were adjusted to the patient's actual requirements and gas exchange function. The blood flow achieved with v-a ECMO support should be in the range of physiologic cardiac output, adapted to the patient's biometric data. Gas supply to the oxygenator is regulated to achieve blood gas values in the normal arterial range to avoid the adverse effects of hyperoxygenation or hypocapnia. For patients on ECMO assistance, lung protective ventilation including reduction of high inspiratory oxygen concentration was performed as soon as possible. Blood temperature was maintained between 33 and 35 °C in patients who had suffered cerebral injury or those who had episodes of cardiopulmonary resuscitation. In all other