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## Hemodynamic and respiratory support using venoarterial extracorporeal membrane oxygenation (ECMO) in a polytrauma patient

*Uso de suporte hemodinâmico e respiratório por meio de oxigenação extracorpórea por membrana (ECMO) venoarterial em um paciente politraumatizado*

### ABSTRACT

There are few reports in the literature regarding the use of venoarterial extracorporeal membrane oxygenation (ECMO) for double-dysfunction from both heart and lung contusions in polytrauma patients. This article reports a 48-year-old patient admitted after a traffic accident. He rapidly progressed to shock with low cardiac output due to myocardial contusion and refractory hypoxemia due to

pulmonary contusion, an unstable chest wall and bilateral pneumothorax. ECMO was an effective rescue procedure in this dramatic situation and was successfully discontinued on the fourth day after the trauma. The patient also developed an extensive brain infarction and eventually died on the seventh day after admission.

**Keywords:** Oxygenation; Shock, cardiogenic; Acute lung injury; Craniocerebral trauma; Case reports

### INTRODUCTION

The use of extracorporeal membrane oxygenation (ECMO) as respiratory support has been widely acknowledged as a rescue technique for refractory hypoxemia in H1N1-infected patients.<sup>(1)</sup> Recently, our institution adopted the use of ECMO in the intensive care unit (ICU) for selected refractory cardiopulmonary dysfunction cases.

In the venoarterial modality, venous blood is oxygenated and pumped back into the arterial system, providing total/nearly total cardiorespiratory support. This method is mostly used in patients who are difficult to wean from cardiopulmonary bypass following acute myocardial infarction or in cases of refractory cardiac arrest.<sup>(2)</sup>

Few investigators have reported the use of ECMO for simultaneous post-traumatic cardiac and pulmonary dysfunctions.<sup>(3,4)</sup> In this article, we report the case of a 48-year-old male patient with cardiogenic shock and hypoxemia due to cardiac and pulmonary contusions, who was successfully supported by venoarterial ECMO until cardiorespiratory recovery.

### CASE REPORT

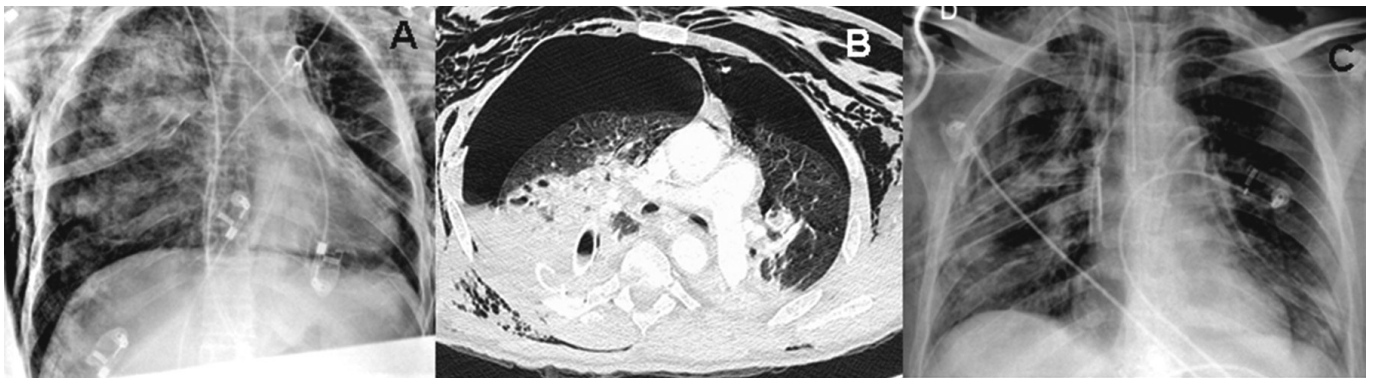
A 48-year-old male patient, with no previous comorbidities, was brought to the emergency service of Hospital das Clínicas after a serious traffic accident (automobile versus motorcycle). The patient was a motorcycle rider who was not wearing a helmet. At the accident site, he had an oxygen saturation of

90% (while breathing room air), an unstable chest wall, a heart rate of 130 beats per minute, an arterial blood pressure of 80/40 mmHg, and a Glasgow coma scale (GCS) rating of 12. During transport to the hospital, orotracheal intubation, right hemithorax relief puncture and volume expansion with 1,000 mL of saline solution were performed.

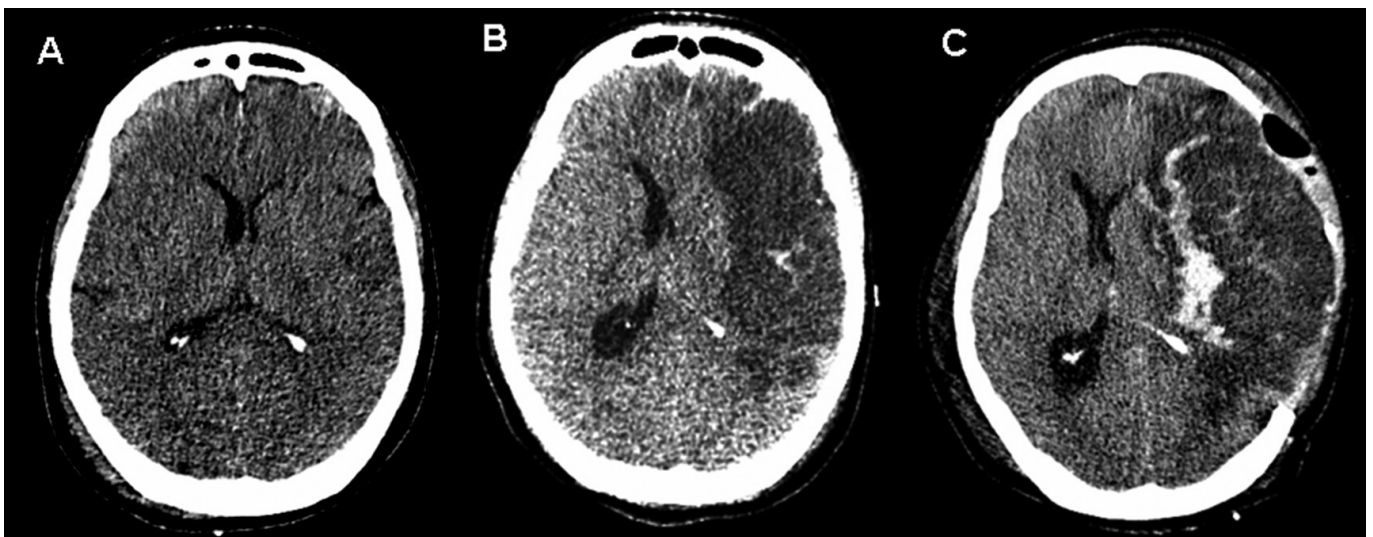
Upon admission to the emergency room, the patient was lying on a rigid bed with cervical collar in place. He had a level 3 GCS and miotic pupils and was mechanically ventilated. He had reduced breath sounds and severe chest subcutaneous emphysema and was hypotensive. Focused assessment with sonography for trauma (FAST)

was negative. Chest tubes were placed bilaterally, and 350 mL of blood had drained from the right side. The patient was persistently hypotensive, despite volume expansion with crystalloid solutions and administration of vasopressor drugs.

Computed tomography (CT) imaging of the head, chest, abdomen and pelvis showed a small left frontal contusion (without an indication for surgical treatment) with mild lateral ventricular asymmetry, suggesting probable right brain edema; multiple costal fractures; extensive pneumothorax and pneumopericardium; bilateral pulmonary contusions; vertebral spinous process fractures; fractures of the lumbar transverse



**Figure 1 - A)** Admission chest radiograph showing bilateral pneumothorax and extensive right lung consolidation, in addition to several costal fractures. **B)** Initial chest computed tomography showing pneumothorax and extensive bilateral consolidation, compatible with polytrauma and pulmonary contusion. **C)** Chest radiograph upon withdrawal of extracorporeal support showing improvement in the pulmonary contusion and pneumothorax.



**Figure 2 - A)** Head computed tomography showing the initially mild asymmetric lateral ventricles, suggesting brain edema likely from ischemia. **B)** Head computed tomography on the 5<sup>th</sup> day from admission, already without extracorporeal support, showing extensive left hemisphere infarction. **C)** Hemorrhagic transformation of the infarction after left frontoparietal craniectomy.

processes; and a fracture of the left ilium extending to the pubis and acetabulum (chest and head CT shown in figures 1 and 2).

After admission to the ICU, difficulty with adequate ventilation persisted due to the patient's extensive pulmonary contusions, even after effective bilateral lung drainage. Additionally, the patient required increasing doses of noradrenalin and dobutamine due to persistent signs of low cardiac output (diaphoresis, coldness and slow capillary filling). Bedside echocardiography was performed, and the subcostal window showed an extremely dilated (diastolic diameter 6 cm) and hypokinetic left ventricle, with an estimated ejection fraction of 0.08 (Teicholz). The esophageal Doppler measured a cardiac index of 0.8 L/m<sup>2</sup>.

About 18 hours after the trauma, despite the administration of 4 mcg/kg/min noradrenalin and 20 mcg/kg/min dobutamine, the patient's hemodynamics progressively worsened, with a mean blood pressure of 50 mmHg, profuse sweating and delayed peripheral perfusion. The patient was then placed under assisted pressure controlled mechanical ventilation with an inspired oxygen fraction (FiO<sub>2</sub>) of 1.0, a positive end-expiratory pressure (PEEP) of 10 cmH<sub>2</sub>O, an inspiratory

pressure of 25 cmH<sub>2</sub>O (15 cmH<sub>2</sub>O driving pressure), an inspiratory time of 0.75 seconds and a respiratory rate of 30. Using these parameters, arterial blood gas showed a PaO<sub>2</sub> of 56 mmHg, an oxygen saturation of 84% and 3.1 mEq/L (28 mg/dL) lactate. Subsequent tests showed progressive worsening of the physiological parameters, with a central venous saturation of 57% (see Baseline column in Table 1).

Given the imminent risk of death from cardiogenic shock and refractory hypoxemia, our institution's ECMO team chose to start venoarterial ECMO support as a rescue procedure. Using the Seldinger technique, 22 Fr draining cannulas were inserted into the right common femoral vein. A return cannula was placed in the right femoral artery with an 8F catheter for distal perfusion of the right lower limb. A centrifuge magnetic pump with a polymethylpentene oxygenation membrane (Rotaflow/Jostra Quadrox, Maquet Cardiopulmonary AG, Hirrlinger, Germany) was used. The blood flow was initially at 4,500 mL/min with a 6,000 mL/min gas flow (pure oxygen Sweeper).

The ECMO team of Hospital das Clínicas de São Paulo and Hospital Sirio-Libanês consists of nurses, physicians and physiotherapists. The entire shift team

**Table 1 – Clinical progression including hemodynamic, respiratory, neurological (Sedation-Agitation Scale) and organ dysfunction (Sequential Organ Failure Assessment score – SOFA) parameters**

	Baseline	ECMO start	1 <sup>st</sup> ECMO day	Last ECMO day	After decannulation
PEEP (cmH <sub>2</sub> O)	10	10	10	10	5
Ventilator FIO <sub>2</sub>	1	0.4	0.3	0.21	0.6
Respiratory rate (ipm)	30	10	10	10	14-30
Noradrenalin (mcg/kg/min)	4	0.5	0.19-1.38	0.06-0.03	0
Dobutamine (mcg/kg/min)	20	0	0	0	8
ECMO blood flow (L/min)	na	4-4.5	4	4-4.5	na
ECMO gas flow (L/min)	na	4-6	4	4	na
FIO <sub>2</sub> ECMO	na	1	0.4	0.4	na
Heart rate (bpm)	160	120	64-156	104-120	100-127
Mean blood pressure (mmHg)	50	70	64-71	65-80	53-91
pH	7.28	7.19	7.11	7.4	7.42
pO <sub>2</sub>	56	316	126	58	62
Arterial O <sub>2</sub> saturation	84	99.8	97	90	92
pCO <sub>2</sub>	31	32	25	43	44
Bicarbonate	14	12	14	27	28
Base excess	-11	-15	-19	2	3.1
Lactate	28	59	96	32	19
SAS	1-2	1-2	1-2	2	2
SOFA	17	17	16	14	14

ECMO – extracorporeal membrane oxygenation; PEEP – positive end-expiratory pressure; FiO<sub>2</sub> – inspired oxygen fraction; SAS - Sedation-Agitation Scale; SOFA - Sequential Organ Failure Assessment score.

Baseline column data represent the patient's condition 18 hours after the trauma, immediately before the start of ECMO.

rather than one specific person was responsible for managing the device.

Progressive hemodynamic and respiratory improvement occurred about 8 hours after ECMO was started. This allowed us to wean the patient from dobutamine and taper the noradrenaline dose to 0.5 mcg/kg/min. a mean blood pressure of 70 mmHg was maintained. The absence of a pressure curve and a pulse pressure led us to infer that the entire blood flow was mediated by the ECMO. Minimal mechanical ventilation parameters were maintained, with a PEEP of 10 cmH<sub>2</sub>O, an inspiratory pressure of 20 cmH<sub>2</sub>O and a FiO<sub>2</sub> of 0.3 (controlled pressure mode).<sup>(5)</sup> Blood gas analysis showed that the patient's hypoxia had been corrected; however, he continued to have metabolic acidosis and significant hyperlactatemia. The ECMO parameters were adjusted according to the perfusion and oxygenation indices (Table 1).

During the ICU stay, this patient was given analgesia with continuous fentanyl (0.25 – 0.5 mcg/kg/minute). He continued to be obtunded (GCS 5T; Sedation Agitation Scale (SAS) 1-2). On the second day following admission, bedside cranial ultrasonography showed optic sheath widening (6 mm) and midline shift; however, due to the patient's critical clinical status, no new CT scans were possible during ECMO. Because the nature of the intracranial event could not be precisely established, we chose to maintain analgesia with fentanyl while monitoring the consciousness level until imaging could be performed. Pain was assessed based on behavioral and physiological reactions.

Because of heavy bleeding from the chest tube (1.5 L during the first day), which required multiple transfusions, and the above described neurological conditions, an anticoagulant was not given during the ECMO.

Despite the presence of acute renal failure (requiring hemodialysis), low platelet counts and signs of extremity ischemia (worse in the right leg where the arterial return cannula was located), cardiac and pulmonary functions progressively improved. On the 4<sup>th</sup> day of support, a pulse pressure curve was detected by invasive blood pressure monitoring, and the echocardiogram-estimated left ventricle ejection fraction was 0.3. The pulmonary condition also improved, as assessed by chest x-ray. Dobutamine inotropic support was restarted, and the patient was successfully decannulated (Table 1).

The day after decannulation, the patient had anisocoria and a decreased consciousness level (GCS 3T, SAS1). Repeat head CTs showed a left hemisphere infarction (Figure 2). Left frontotemporal decompressive craniectomy with duraplasty was performed. Postoperative

imaging showed significant post-decompression bleeding (Figure 2) with neurological deterioration. The next day, clinical examinations were compatible with brain death, which could not be confirmed due to intraoperative use of thionembutal. About 24 hours later, somatic death was diagnosed.

## DISCUSSION

The use of extracorporeal support for severe hypoxemia in children is supported by relatively strong clinical evidence.<sup>(2)</sup> Little evidence has supported the use of this technique in adult patients.<sup>(5)</sup> Recently, however, interest in the use of ECMO in adults has intensified, partly due to technological advances (such as biocompatible and durable membranes) and especially due to the large number of refractory hypoxemia cases that occurred during the H1N1 influenza epidemics.<sup>(1)</sup>

Extracorporeal support was a key component of the successful Australian treatment regimen for H1N1-related refractory hypoxemia.<sup>(1)</sup> A recent randomized trial showed a possible benefit from extracorporeal support in patients with severe acute respiratory failure secondary to acute lung injury/adult acute respiratory distress syndrome. In this trial, ECMO was used to prevent lung injury caused by mechanical ventilation using low ventilation volumes and pressures. However, this study has been criticized because ECMO was only used in 68 of the 90 randomized patients, and there was a relatively high rate of death among patients during transfer to ECMO-specialized sites.<sup>(5)</sup>

The lack of well-designed clinical trials of ECMO in adult patients prevents us from drawing clear conclusions about the utility of the procedure in adult critical care patients, as highlighted in a recent systematic review.<sup>(6)</sup> Therefore, the current status of ECMO is that of a rescue measure for failed traditional therapeutics. Because of this, a multidisciplinary team was established for using ECMO in selected refractory hypoxemia cases. With similar indications to those proposed by CESAR,<sup>(5)</sup> this team aims to offer an alternative for patients in whom usual hypoxemia management measures (e.g., alveolar recruitment maneuvers, nitric oxide and high-frequency ventilation) are ineffective and/or harmful (e.g., barotrauma or high airway pressure needed for maintaining acceptable ventilation).

Significant hypoxemia is a common complication of pulmonary contusion, and the use of extracorporeal oxygenation in some of these patients has been reported.<sup>(7)</sup>

However, the case reported here differs from prior

reports in several important ways. This patient had refractory hypoxemia from several injury mechanisms (pulmonary contusion, bilateral extensive pneumothorax and an unstable chest wall) and had a PaO<sub>2</sub>/FiO<sub>2</sub> ratio of 56. Measures frequently used in this context would be inappropriate or even harmful. Alveolar recruitment maneuvers could worsen the bilateral air fistulae, and prone positioning is contraindicated due to the extreme hemodynamic instability.

Additionally, the patient had shock that was refractory to volume expansion, vasopressors and inotropic drugs. Echocardiography revealed clear cardiogenic shock, likely due to myocardial contusion. Considering the imminent risk of death and that there were no other therapeutic options, we chose to use venoarterial ECMO with total cardiorespiratory support to simultaneously support the patient's respiratory and hemodynamic functions. Significant improvement was quickly achieved (Table 1), allowing the progressive recovery of cardiac and pulmonary functions, and we were able to discontinue extracorporeal support after 4 days.

During this time, clinical (consciousness level) and imagery signs (transcranial ultrasound and widened optical sheath) were indicative of intracranial hypertension. However, the patient's complete dependency on hemodynamic and respiratory support prevented him from undergoing a head CT. Unfortunately, the patient died from his intracranial injury, which could not be assessed and treated in a timely fashion.

Few reports discuss full cardiopulmonary support with venoarterial ECMO in trauma patients. Perchinsky et al. reported 50% survival in a series of 6 patients using this procedure as a rescue measure for severe polytrauma patients deteriorating in spite of the conventional therapy.<sup>(3)</sup> More recently, Masiakos et al. reported the successful management of a patient with pulmonary and myocardial contusions in addition to right ventricular papillary muscle rupture with significant tricuspid regurgitation, who presented with significant hypoxemia, hemodynamic instability and difficult-to-manage ventricular arrhythmias.<sup>(4)</sup>

In our report, we successfully used extracorporeal support as a rescue measure for cardiorespiratory dysfunction that would otherwise have been rapidly fatal. ECMO was effective as a bridging strategy, allowing

decannulation on the 4<sup>th</sup> day after the trauma. The patient died from head trauma, which was not related to nor treated with extracorporeal support. Unlike the report by Masiakos et al.,<sup>(4)</sup> no additional ICU professionals (e.g., a perfusionist) were necessary during the extracorporeal support; only our institutional ECMO team was involved.

In summary, this article reports a polytrauma patient with refractory hypoxemia due to pulmonary contusion and refractory cardiogenic shock due to cardiac contusion. Venoarterial ECMO was successfully used as a bridging strategy for cardiac and pulmonary recovery, and the extracorporeal support was discontinued on the 4<sup>th</sup> day after trauma. This extracorporeal support method can be lifesaving in selected patients. However, additional studies are necessary to evaluate how this promising technology may best be used clinically.

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#### **RESUMO**

Existem poucos relatos na literatura sobre o uso de oxigenação extracorpórea por membrana venoarterial por dupla disfunção decorrente de contusão cardíaca e pulmonar no paciente politraumatizado. Relatamos o caso de um paciente de 48 anos, vítima de acidente de motocicleta e automóvel, que evoluiu rapidamente com choque refratário com baixo débito cardíaco por contusão miocárdica e hipoxemia refratária decorrente de contusão pulmonar, tórax instável e pneumotórax bilateral. O suporte extracorpóreo foi uma medida efetiva de resgate para esse caso dramático, e o seu uso pôde ser interrompido com sucesso no 4º dia após o trauma. O paciente evoluiu com extenso infarto cerebral, morrendo no 7º dia de internação.

**Descritores:** Oxigenação; Choque cardiogênico; Lesão pulmonar aguda; Traumatismos craniocerebrais; Relatos de casos

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