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Case Report

Survival following traumatic thoracic compartment syndrome managed with VV-ECMO

Timothy Amos[b,c,*], Meei Yeung[a,b], Julian Gooi[e], Mark Fitzgerald[a,b,d]

[a] Trauma Services, The Alfred Hospital, Melbourne, Victoria, Australia
[b] National Trauma Research Institute, Australia
[c] Emergency and Trauma Centre, The Alfred Hospital, Melbourne, Victoria, Australia
[d] Monash University School of Medicine, Melbourne, Victoria, Australia
[e] Cardiothoracic Surgery Unit, The Alfred Hospital, Victoria, Australia

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ABSTRACT

Whilst post-traumatic respiratory failure is the most common indication for use of VV-ECMO in trauma patients, its use in traumatic thoracic compartment syndrome is not yet well described. Thoracic compartment syndrome, a rare complication of thoracic trauma, occurs in the setting of chest wall injuries, impaired chest wall compliance, pulmonary contusions and subsequent high ventilatory pressures. This in turn impairs venous return and increases risk of circulatory arrest due to obstructive shock.

This case study describes the successful use of VV-ECMO in a young male with thoracic compartment syndrome following severe blunt chest trauma sustained in a high speed motor vehicle crash. Following brief circulatory arrest, thoracic compartment syndrome was relieved during thoracotomy but reoccurred on chest closure. The use of VV-ECMO for oxygenation permitted lower ventilatory pressures, allowing venous return and primary closure of the thoracotomy. The patient subsequently had an excellent functional outcome.

This case describes the successful use of VV-ECMO for a novel indication. The indications for ECMO in thoracic trauma patients continue to evolve.

1. Introduction

Thoracic compartment syndrome is a rare complication of severe thoracic trauma, defined as resolution of shock/traumatic circulatory arrest upon opening of the chest by thoracotomy. While veno-venous extracorporeal membrane oxygenation (VV-ECMO) has been shown to be an effective treatment of respiratory failure in a specific cohort of trauma patients [1], its use in traumatic thoracic compartment syndrome and subsequent traumatic circulatory arrest is not well described. This case report describes the effective use of VV-ECMO as part of early trauma management and raises the possibility of including traumatic thoracic compartment syndrome as an indication for ECMO. (see Tables 1 and 2)

2. Case report

A 21 year old male was involved in a high speed motor vehicle crash during which the vehicle rolled off a bridge, coming to rest

* Corresponding author. National Trauma Research Institute, Level 4, 89 Commercial Rd, Melbourne, Victoria, 3004, Australia.
E-mail address: T.Amos@alfred.org.au (T. Amos).

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Table 1
Traumatic thoracic compartment syndrome.

1. Risk factors
Chest wall trauma
Large volume fluid resuscitation
Impaired chest wall compliance
Pulmonary contusion
2. Clinical Signs
High airway pressures
Impaired oxygenation
Haemodynamic instability and symptoms of obstructive shock
Resolution of symptoms upon opening of the chest

Table 2
ECMO use in Trauma.

Indications for ECMO in trauma
ARDS (Acute respiratory distress syndrome) [1]
Thoracic compartment syndrome (proposed)
Cardio-respiratory support in patients with both thoracic and brain injuries to provide safe conditions for clinical assessment [14]
Contraindications for ECMO in trauma
Uncontrolled haemorrhage (relative contraindication in VV-ECMO)
Traumatic brain injury (relative contraindication [15,16])
Established multi-organ failure [17]
Poor short term prognosis [17]
Advanced comorbidities [17]

overturned, but not submerged, on the bank of a river. The patient was initially GCS 15 but during a prolonged extrication became hypoxic and combative, with GCS dropping to 3 whereupon he was intubated and ventilated by paramedics. The patient was transported via helicopter retrieval service to a major trauma centre approximately 160km away.

On arrival at the trauma centre, the patient had a heart rate of 137/minute, blood pressure 88/50 mmHg and was profoundly hypoxic with oxygen saturations of 55–69% - despite being ventilated on 100% FiO2, with high airways pressures > 40cmH2O and tidal volumes of approximately 250ml. Bilateral finger thoracostomies were performed finding bilateral, non tensioned haemo/pneumothoraces. Bilateral 32Fr intercostal catheters were inserted, central access obtained and a massive transfusion protocol commenced. Initial chest x-ray demonstrated bilateral white-out of both lung fields suggestive of early post-traumatic ARDS. Progressive hypotension predicated removal of intercostal catheters with repeat finger thoracostomies again finding no tension pneumothorax, with no improvement of oxygenation or blood pressure. Intercostal catheters were reinserted with a subsequent chest x-ray raising the suggestion of tracheobronchial disruption.

At 1 h post arrival the primary cause of worsening hypoxia and hypotension remained unclear, with ongoing moderate blood loss through bilateral intercostal catheters. Given the diagnostic uncertainty and ongoing instability the resuscitation was moved to the operating theatre for clamshell thoracotomy. During transport to theatre the patient became progressively bradycardic, hypotensive and subsequently went into circulatory arrest. Adrenaline and metaraminol were given while a clamshell thoracotomy was performed. On opening the chest return of spontaneous circulation (ROSC) was obtained, oxygenation rapidly improved and peak inspiratory pressures decreased to 30–33 cmH2O. The patient had bilateral haemothoraces (previously placed intercostal catheters were found to be functioning and in appropriate positions with no evidence of tension) and chest wall bleeding, but no definitive surgical cause for the patient's presentation was found. During the procedure persistently large volumes of frothy pink sputum were suctioned from the airway. Arterial blood gas demonstrated pH 6.91, pCO2 77, PO2 74 on FiO2 of 1.0 for a P/F ratio of 74.

On closure of the chest, oxygenation and ventilation again became impaired with FiO2 increasing from 0.4 for SpO2 100%, to 1.0 for SpO2 88%. PEEP was increased incrementally to 20cmH2O with peak inspiratory pressures > 40cmH2O. The patient became increasingly hemodynamically unstable with rapidly increasing doses of noradrenaline and vasopressin administered to support blood pressure. The working diagnosis was obstructive shock with severe bilateral pulmonary contusions/oedema. This suggested early ARDS with thoracic compartment syndrome demonstrated by impairment of ventilation temporally related to opening and closure of the chest. At this point the patient had received 2700ml crystalloid, 14 units of packed red cells, 16 units of fresh frozen plasma, one unit of platelets and 8 units of cryoprecipitate. The post-operative haemoglobin level was 78g/L.

The decision was made to commence the patient on rescue veno-venous extra-corporeal membrane oxygenation (VV-ECMO) with no systemic anticoagulation. The rationale was that ECMO would allow the use of much lower ventilatory pressures, thus decreasing intra-thoracic pressure to allow venous return, relieving obstructive shock. As oxygenation markedly improved (SpO2 100%) on opening of the chest, it became clear that ARDS was not the sole pathology. Additionally ECMO would allow for primary chest

closure. ECMO cannulation was performed in the operating theatre as closure of the chest was being finalised, with marked improvement in haemodynamics and oxygenation as ECMO was commenced and ventilatory pressures were decreased.

Subsequent CT trauma series demonstrated extensive bilateral pulmonary contusions and dependent consolidation, bilateral multiple rib fractures, a sternal fracture and multi-level thoracic spine fractures consistent with high velocity blunt thoracic trauma. The patient was successfully weaned from VV-ECMO on day 11 and extubated on day 16 of admission. He was discharged from hospital on day 35 well, with minimal functional impairment.

3. Discussion

3.1. Thoracic compartment syndrome

Compartment syndrome occurs when the pressure in a compartment exceeds the tissue perfusion pressure and is more often associated with abdominal or orthopaedic pathology. Nevertheless thoracic compartment syndrome can present as a rare and life threatening complication of trauma, manifesting as impaired ventilation, elevated airway pressures and haemodynamic instability [2] with the possibility of traumatic circulatory arrest due to obstructive shock. Thoracic compartment syndrome is more commonly seen in cardiothoracic surgery as a complication of delayed sternal closure after a prolonged cardiac procedure [3]. A key characteristic is the resolution of symptoms upon opening of the chest and recurrence of symptoms on chest closure as demonstrated in this case [4].

Previous case reports describe management of thoracic compartment syndrome with an open chest approach [2] and delayed thoracotomy closure when able. The early implementation of VV-ECMO in this case allowed primary closure of the thoracotomy and avoided certain associated risks of having an open chest such as mediastinitis [3], osteomyelitis and difficulties of nursing an open chest in the intensive care unit. None the less, ECMO is not without significant risks such as haemorrhage [4] or accidental decannulation as well as challenges in patient transportation and nursing. In this case ECMO did lead to delayed operative fixation of an unstable spinal injury due to both issues of anticoagulation and the difficulties of lying the patient prone. ECMO is an expensive intervention with improved survival when implemented at high volume centres [5]. Availability and relative experience should be taken into account when considering how to approach future cases such as this.

3.2. ECMO in trauma

The primary risk of using ECMO in trauma is uncontrolled haemorrhage, though this is weighed against the ability to correct hypoxia, acidosis and hypothermia [6]. A recent retrospective review of ECMO use in trauma patients by Swol et al. [1] showed increasing use of VV-ECMO with thoracic trauma, with ARDS the most common indication. An overall survival rate of 70% was achieved. The development of ARDS following trauma is associated with mortality of between 21 and 40% [7,8] and generally involve lung protective ventilation, recruitment manoeuvres and strategies such as proning - which present extra challenges in the multi-trauma patient. Rates of posttraumatic ARDS have decreased however with the advent of restrictive transfusion practices and lung protective ventilation [9,10]. Criteria for use of ECMO in traumatic ARDS has been defined as severe hypoxaemia, decreased total thoracic compliance and bilateral pulmonary infiltrates [11] which were met in this case. Using P/F ratio as a trigger for ECMO rescue in ARDS, for example < 50 for > 3 hours [12] was not considered in this case, as the cause of hypoxia and haemodynamic instability was multifactorial and not solely ARDS. Of note there are case reports of the use of ECMO in cases of tracheal-bronchial disruption as a bridge to definitive management [13] which was a diagnosis initially considered in this case.

4. Conclusion

In this case the patient progressed through the standard ATLS traumatic circulatory arrest algorithm [14], with ROSC achieved on clamshell thoracotomy despite no cardiac injury or severe hypovolaemia identified. Hypoxia and impaired ventilation with subsequent obstructive shock due to thoracic compartment syndrome was thought to be the key issue. with veno-venous ECMO relieving the need for high ventilatory pressures and allowing venous return. While a rare cause of traumatic circulatory arrest, managing future cases of traumatic thoracic compartment syndrome with VV-ECMO is worth considering. The indications for ECMO in thoracic trauma patients continue to evolve

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