

Successful Rescue of an Adult With Refractory Anaphylactic Shock and Abdominal Compartment Syndrome With Venoarterial Extracorporeal Membrane Oxygenation and Bedside Laparotomy

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Abstract

The incidence of life-threatening anaphylactic reactions related to anesthesia is approximately 1 in 6000 anesthetics administered, and is associated with mortality as high as 5%. In such cases the use of extracorporeal membrane oxygenation (ECMO) in the setting of refractory shock following anaphylaxis may be life saving. Abdominal compartment syndrome (ACS) itself and in this case complicating ECMO support, is a potentially devastating complication of high-volume resuscitation. Decompressive laparotomy is the treatment of choice for ACS. We present a patient treated with venoarterial ECMO for refractory shock following anaphylaxis who developed ACS that was successfully treated with urgent decompressive laparotomy performed in the intensive care unit. This case report highlights the role of abdominal compartment syndrome as a rare but potentially fatal cause of low circuit flow in ECMO-supported patients and proposes a stepwise approach to decision making in this setting. Urgent decompressive laparotomy is potentially lifesaving in this circumstance, and should be urgently considered once other causes of low ECMO flow have been excluded.

Keywords

cardiopulmonary bypass, critical care, intensive care unit, therapeutic hypothermia, transesophageal echocardiography

Introduction

The incidence of life-threatening anaphylactic reactions related to anesthesia is approximately 1 in 6000 anesthetics administered and is associated with mortality as high as 5%.¹ The use of extracorporeal membrane oxygenation (ECMO) in the setting of refractory shock following anaphylaxis has been documented.^{2–5} However, only Zhang et al³ and Lafforgue et al⁴ have documented the successful use of ECMO in anaphylaxis in adults leading to neurologically intact discharge from the hospital and neither cases were complicated by abdominal compartment syndrome (ACS). ACS complicating ECMO support has been previously described.^{6,7} ACS is a condition of raised intra-abdominal pressure, typically greater than 20 cm H₂O.^{6,8} ACS, also known as intra-abdominal hypertension (IAH) carries an intensive care unit (ICU) mortality rate of 30.8% with the only independent predictors being high SOFA score and positive fluid balance on admission.⁹ ACS has many causes,^{6,10} including massive capillary leak such as can occur with anaphylaxis.¹⁰ A recent review examining physician

awareness, understanding and management of ACS reveals significant deficiency and variation in recognizing ACS and a general lack of consensus and knowledge regarding guidelines.⁸ During ECMO support, either venoarterial (VA) or venovenous (VV), venous drainage is typically via a large cannula placed in the femoral vein and advanced into the inferior vena cava (IVC) with the tip ultimately being sited in the right atria.¹¹ A dual lumen venovenous cannula is now available allowing for singular access via the right internal jugular vein. Raised intra-abdominal pressure associated with ACS can impair drainage from the IVC cannula and therefore severely restrict ECMO flow. Since the access cannula on VV ECMO relies on adequate return of blood to the right atrium, compression of the IVC due to ACS may limit

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drainage especially at high flow rates on VA ECMO. In a previous report, ACS occurred 48 hours after initiation of VA ECMO following cardiac surgery due to a large retroperitoneal hematoma secondary to arterial injury that occurred during placement of an intra-aortic balloon pump.⁶ However, despite a laparotomy and attempts at surgical hemostasis, the patient died. We present a patient treated with VA ECMO for refractory shock following anaphylaxis who developed ACS that was successfully treated with urgent decompressive laparotomy performed in the ICU.

Case Report

An otherwise healthy 61-year-old man who was 172 cm tall and weighed 84 kg, presented for general anesthesia for catheter-based pulmonary vein isolation for paroxysmal atrial fibrillation. Endotracheal intubation was planned to facilitate transesophageal echocardiography (TEE), as per routine. Prior to induction of anesthesia, a 20-G catheter was placed in the patient's right radial artery, and 14-G intravenous catheter was placed in his right forearm. General anesthesia was induced with propofol 100 mg, fentanyl 200 µg, and atracurium 30 mg, and the trachea was intubated uneventfully. Cefazolin 2 g was also administered concurrently with the induction agents. Immediately following induction of anesthesia, the patient became profoundly hypotensive (blood pressure 81/45 mm Hg; mean arterial pressure [MAP] 57 mm Hg) while securing the endotracheal tube decreasing to 50/30 mm Hg (MAP 37 mm Hg) as noted on anesthesia record. Owing to the initial absence of obvious signs of anaphylaxis—bronchospasm and high airway pressures—tachycardia (heart rate 80-90), typical rash (rash restricted to arms and only seen after removal of blanket, end tidal CO₂ low (18 mmHg) and hypotension were the only indicators initially. The patient's hypotension was refractory to escalating doses of metaraminol (1 mg then 5 mg then 10 mg), which was initially administered assuming hypotension was related to propofol induction. Epinephrine 1 mg was given quickly followed by a second dose of 1 mg when metaraminol had failed and a rash was observed on removal of the blanket covering the patient's arms. Initial cardiac rhythm on the monitor was pulseless electrical activity (sinus rhythm) closed chest massage was initiated and help was called for. A good wave form and MAP of 60 to 80 mm Hg was generated with cardiopulmonary resuscitation (CPR). During the resuscitation a 9-French (Fr) introducer was placed in the right internal jugular vein and subsequently a quad-lumen central line was placed just distal to the introducer in lieu of ongoing high volume fluid administration. The first arterial blood gas (ABG) obtained shortly after initiation of CPR revealed a pH of 7.12, pCO₂ of 82 mm Hg, pO₂ of 55 mm Hg, and a bicarbonate of 19 mEq/L. A TEE probe was not placed because of ongoing CPR; however, a

brief transthoracic examination revealed a severely dilated and hypokinetic left ventricle and a severely impaired and hypokinetic right ventricle. While high quality CPR continued end tidal CO₂ remained more than 20 mm Hg, MAP varied in the 70 to 80 mm Hg range. Over the next 20 minutes, in spite of aggressive advanced life support, adequate spontaneous perfusion was not achieved, and a decision was made to initiate VA ECMO.

The ECMO circuit consisted of a polymethylpentene oxygenator (Quadrox PLS, MAQUET Cardiovascular, Hirrlingen, Germany) and a centrifugal pump (Rotaflow, MAQUET Cardiovascular). A 29-Fr multiport drainage cannula (HLS, MAQUET Cardiovascular) was placed percutaneously in the patient's right femoral vein and a 19-Fr return cannula (HLS MAQUET Cardiovascular) was placed percutaneously in what was thought to be the patient's left femoral artery. Both cannulae were placed using landmark methods initially due to vascular ultrasound not being readily available. However, initiation of ECMO did not result in an improvement in the patient's hemodynamics or oxygenation. ECMO was discontinued and guide-wires were placed in both cannulae. Fluoroscopic imaging demonstrated the return cannula was in the femoral vein, not artery, it further confirmed the position of the access cannula appropriately in the right atrium. The return cannula was then inserted in the left femoral artery using vascular ultrasound since an ultrasound machine had since arrived. After 60 minutes of advanced life support, involving continuous closed chest compressions, 19 mg epinephrine, 150 mL 8.4% sodium bicarbonate, and 20 L crystalloid fluid, VA ECMO was successfully initiated at a pump speed of 4000 rpm, which achieved a circuit flow of 5.1 L/min. At that time, the post CPR ABG revealed a pH of 7.41, pCO₂ of 72 mm Hg, pO₂ of 66 mm Hg, and a bicarbonate of 26 mEq/L. VA ECMO at this flow rate resulted in a dramatic improvement in the patient's cardiorespiratory state. Repeat ABG after 10 minutes of VA ECMO revealed an increase in pO₂ to 88 mmHg, and a reduction in pCO₂ to 38 mm Hg with an ECMO sweep of 5 L/min. Arterial pH was maintained at 7.48 and bicarbonate at 24 mEq/L. A vascular surgeon was called to surgically place a distal perfusion cannula in the left femoral artery, which occurred during ongoing resuscitation in the catheter laboratory. An initial hemoglobin level was drawn immediately following successful cannulation and 2 units of packed red blood cells were administered for a hemoglobin of 6.4 g/dL. No significant blood loss was noted and the drop in hemoglobin was thought to be dilutional. A TEE probe was placed concurrently with initiation of VA ECMO revealing a severely dilated and globally hypokinetic left ventricle, the access cannula positioned in the right atria, and a hypokinetic right ventricle. Approximately 30 minutes after initiation of VA ECMO, TEE demonstrated a dramatic improvement in left ventricular function. The patient was transferred to the cardiovascular ICU.

Prior to and following arrival in the ICU difficulties were experienced achieving adequate circuit flow. Despite pump speeds of 3500 to 4000 rpm, maximal achievable flows were 3 to 3.5 L/min. Because capillary leaking and redistribution of intravascular volume into the bowel wall and other extra-vascular spaces is progressive, as is ACS in general, initial flows were better than subsequent flows. Attempts to increase pump speed resulted in repeated suction events, necessitating reducing pump speed and administering further fluid boluses. Suction events or access insufficiency results when insufficient blood return from the body to the venous cannula exists to support the ECMO flow goal for a given speed. These events are characterized by shaking or chatter of the venous (access) limb of the circuit and reduction in flow. Volume status was assessed utilizing TEE which not only revealed dramatically improved biventricular function but a well filled heart and progressive aortic valve ejection. A small left-sided pleural effusion was also noted on TEE with no evidence of significant fluid collection in either pleural space on chest radiograph. The patient's abdomen had become grossly distended, and abdominal palpation resulted in immediate suction events. The absence of hemoglobinuria and echo confirmation of laminar flow at the access cannula, at least when suction events were not present, led us to rule out cannula obstruction or thrombus as a cause. Additionally, as cardiac function improved oxygenation of the patient's upper body became severely impaired, due to the ejection of deoxygenated blood by the left ventricle into the central circulation also known as differential hypoxia. The ventilator in the ICU was set in pressure control mode with a peak pressure of 20 cm H₂O, peak end expiratory pressure of 10 cm H₂O, FiO₂ of 100% at a rate of 12 breaths/min, resulting in tidal volumes of approximately 2 mL/kg. This poor compliance was correlated with an admission to ICU chest radiograph showing bilateral ground glass opacifications encompassing the entirety of both lungs. Ventilator settings were aimed at lung protective strategy keeping peak pressure below 30 cm H₂O and providing increased peak end expiratory pressure. The high FiO₂ was necessary due to increasing difficulty with oxygenation as heart function improved. Arterial blood gas analysis taken from the right radial arterial catheter revealed a SaO₂ that varied between 40% and 75%. Bilateral upper extremity pulse oximetry revealed only intermittent but less than 50% SaO₂ considered unreliable due to initially limited pulsatility and lack of reliability of such low peripheral SaO₂ measurements. Pre- and post-oxygenator SaO₂ were as expected with pre-oxygenator values ranging from 50% to 64% and post-oxygenator values being 100%. The patient was given 50 mg of rocuronium to facilitate relaxation in an effort to improve ventilation and access insufficiency with no improvement. To help reduce left ventricular ejection, the epinephrine infusion was discontinued and an infusion of norepinephrine commenced to support

blood pressure (target MAP >60 mm Hg). During the acute phase and because of consideration for laparotomy, anticoagulation was withheld.

A discussion was had regarding possible therapeutic options, and a decision was made to request a decompressive laparotomy with vacuum dressing. This was performed at the bedside by a general surgeon. Moderate ascites and severely edematous bowel were identified at laparotomy with no evidence of mesenteric ischemia. Following abdominal decompression, pump speed could be increased to 4000 rpm without causing suction events, and circuit flow increased from 3.5 to 6.0 L/min. Additionally, SaO₂ immediately increased from 71% to 91% and tidal volumes increased modestly to 4 mL/kg at 20 cm H₂O indicating some improvement in lung compliance. Two hours post-laparotomy, pump speed was 4150 rpm, circuit flow was 6.0 L/min, SaO₂ was 99%, pH 7.44, and lactate was 2.0 mmol/L. Thereafter support with VA ECMO was uneventful, there were no further suction events and no further need for intravenous resuscitation fluid.

The patient was cooled to a target core temperature of 33°C to 34°C for 24 hours as monitored by indwelling urinary catheter temperature probe, following which he was slowly passively rewarmed so as to not create hyperthermia, a target temperature of 36°C was targeted. Sedation was maintained with midazolam and fentanyl infusions and 2 subsequent doses of 50mg of rocuronium were administered between laparotomy and normothermia to prevent shivering and to facilitate continued ECMO. During this time the patient had a massive spontaneous diuresis of 4020 mL in 24 hours. Repeat TEE demonstrated normal cardiac function. Anticoagulation was still withheld due to the patient's open abdomen and to spite mild thrombosis seen on the access side of the oxygenator, flows were not adversely affected and no evidence of excessive hemolysis was present reflected by normal plasma free hemoglobin levels. After 48 hours, ECMO was discontinued and sedation was stopped. The patient awoke and was neurologically intact. The laparotomy wound was closed uneventfully at 72 hours. After 10 days, the patient was discharged from hospital in good health. Tryptase levels obtained at 2 hours and 4 days postarrest were 44.7 and 4.9 µg/L, respectively. Krishna et al¹² showed that a tryptase level >33.6 µg/L carried a positive predictive value of 86.4% for an IgE-mediated anaphylaxis. Laroche et al¹³ further describe a tryptase of 7.35mcg/L indicating an allergic mechanism with a sensitivity and specificity of 92%. Skin testing at 3 months demonstrated cefazolin was the causative agent for the anaphylaxis, which according to current evidence is consistent with the second most common cause of anesthesia related allergic reactions being antibiotics.¹² Laroche et al¹³ also showed that aggressive resuscitation in itself does not affect serum tryptase levels. Repeat pulmonary vein isolation with a nontriggering general anesthetic was planned for a later date.

Discussion

The use of VA ECMO for treating cardiogenic shock is well established, as are the many pitfalls and complications associated with its use.¹⁰ VA ECMO may also be used for treating anaphylactic shock,²⁻⁵ although its role is less well established than for treating cardiogenic shock of other etiology.

Anaphylactic shock is characterized by widespread arteriolar vasodilation and severe capillary leak that typically results in intravascular volume depletion and tissue edema. Additionally, in the context of severe hypotension and the need for advanced cardiac life support, myocardial function can also become impaired. Thus, in its most severe form anaphylactic shock may have features of both vasodilatory and cardiogenic shock. Kounis, in several papers, describes an allergic angina or Kounis syndrome typified by inflammatory mediators and arachidonic acid products leading to allergic myocardial infarction and subsequent cardiac dysfunction.^{14,15} While these products of mast cell degranulation and the inflammatory cascade are likely implicated empirical evidence is lacking and future investigation is needed. Whether or not myocardial dysfunction is due to related ischemia and allergic acute coronary syndrome, or by direct depression of the myocardium is yet to be determined.¹⁵

In this case, ACS was thought to be primarily due to the tissue edema associated with anaphylaxis. Progressive extravasation of fluid into the abdominal tissues led to raised intraabdominal pressure, impairing venous drainage to the ECMO circuit, which in turn necessitated further fluid administration to maintain ECMO circuit flow. In addition, the increase in intraabdominal pressure decreases functional residual capacity, decreasing compliance and promoting volume loss and in turn hypoxia. In this case a self-reinforcing cycle of progressive ACS, severely impaired lung function and deteriorating ECMO circuit flow was established. Decompressive laparotomy allowed ECMO circuit flow to be maintained without the ongoing need for fluid administration. In addition, a reduction in the severity of the anaphylaxis-induced capillary leak presumably also occurred around the same time as the laparotomy, further improving the clinical situation.

Abdominal compartment syndrome has been previously described in 3 patients receiving VA ECMO, all of whom received VA ECMO for postoperative cardiogenic shock.⁶ In 2 of 3 patients, decompressive laparotomy resulted in a significant improvement in ECMO flows and the patients' hemodynamics. However, both patients died, one from anoxic brain injury and one from multiple organ failure. In the third case, the patient died before decompressive laparotomy could be performed. To our knowledge, our patient is the only reported survivor following urgent decompressive laparotomy for ACS complicating ECMO support.

In addition to ACS, other potential causes of low ECMO flow despite adequate pump speed include hypovolemia, patient wakefulness or ventilator dysynchrony, malposition of the venous drainage cannula, cardiac tamponade, and obstruction within the ECMO circuit (eg, thrombus in the oxygenator or pump head, kinking of circuit tubing). Each of these factors must be excluded as potential causes of low ECMO flow before considering decompressive laparotomy. An approach to investigating and treating the cause low circuit flow in a patient supported by VA ECMO is outlined in Table 1.

This case highlights 2 further issues that can arise in patients placed on emergency peripheral VA ECMO. First, the problem of upper-body or differential hypoxemia when pulmonary function is impaired and arterial return is via a femoral cannula.⁹ In this case, cardiac function recovered very rapidly with initiation of ECMO and restoration of systemic perfusion resulting in significant ejection of blood from the left heart. Since pulmonary function was impaired due to pulmonary edema, the blood ejected from the left heart was poorly oxygenated resulting in upper-body hypoxemia. Upper body hypoxemia was exacerbated by low ECMO circuit flows (allowing more systemic venous return to pass through the pulmonary circulation) and the need for large volume fluid resuscitation (exacerbating pulmonary edema). Attempts to minimize ventricular ejection by reducing the epinephrine infusion were largely ineffective. Restoration of adequate ECMO flow with decompressive laparotomy solved the problem.

Second, this case highlights the difficulty in clinically identifying the femoral artery in a hypoxemic patient receiving chest compressions. In this circumstance, both venous and arterial blood will appear "blue" and tend to "spurt" from the seeker needle. Accurate identification of the correct femoral vessel with ultrasound is also challenging in this situation. Advancing a long guidewire in to the femoral vessel and confirming its presence in the thoracic aorta (as opposed to the inferior vena cava) with fluoroscopy correctly identified the femoral artery. At this institution and in this instance anesthesia-trained intensivists place the percutaneous ECMO cannulae the alternative being surgical cut-down provided a surgeon trained to institute ECMO is present, which was not the case in this instance.

In summary, this case report highlights the role of ACS as a rare but potentially fatal cause of low circuit flow in ECMO-supported patients. Urgent decompressive laparotomy is potentially lifesaving in this circumstance, and should be urgently considered once other causes of low ECMO flow have been excluded.

Declaration of Conflicting Interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Table 1. Causes, Diagnosis, and Treatment of Low Circuit Flow Despite Adequate Pump Speed in a Patient Supported by Venoarterial ECMO.

Cause	Diagnosis	Treatment
Inadequate sedation	Patient moving or coughing	Sedate and/or paralyze the patient
Hypovolemia	Low central venous pressure	Administer fluid bolus
Cardiac tamponade	Evidence of cardiac compression on TEE	Surgical reexploration for relief of tamponade
Malposition of drainage cannula	Check position and flow pattern of drainage cannulae with TEE	Reposition the drainage cannulae under TEE guidance
Obstruction within circuit		
Kinking of ECMO tubing	Visible kink in ECMO tubing	Unkink ECMO tubing
Thrombus in the oxygenator	Visible thrombus in oxygenator Falling post-oxygenator PaO ₂ ; increased pressure drop across oxygenator	Replace oxygenator or entire circuit
Thrombus in the pump head	Raised plasma hemoglobin Visible thrombus in pump head Abnormal sound of from pump head	Replace pump head or entire circuit
Abdominal compartment syndrome	Distended abdomen Suction events with abdominal palpation Measure bladder pressure (symptoms occur with bladder pressure >15-20 mm Hg)	Early intervention with decompressive laparotomy ^a

Abbreviations: ECMO, extracorporeal membrane oxygenation; TEE, transesophageal echocardiography.

^aConsideration must be made to overall viability and comorbidity.

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