Massive Systemic Air Embolism during Extracorporeal Membrane Oxygenation Support of a Neonate with Acute Respiratory Distress Syndrome after Cardiac Surgery

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Abstract: Extracorporeal membrane oxygenation (ECMO) is universally accepted as a potential lifesaving therapy for neonates suffering severe cardiorespiratory failure, with survival reported as 81% weaning off ECMO and 69% to hospital discharge in this population (1). Although ECMO may reduce mortality in certain neonatal patients, it is associated with significant complications. Air in the circuit complicates 4.9% of neonatal ECMO runs, and it is crucial that all ECMO caregivers are trained in the prevention of air embolism and possess the knowledge necessary to efficiently identify and remove air from the ECMO circuit to prevent life threatening consequences. We present a fatal case of neonatal systemic air embolism leading to massive entrainment of air into the ECMO venous return cannula of a neonatal patient with acute respiratory distress syndrome following repair of obstructed total anomalous pulmonary venous connection. We describe the pathophysiology and presentation of this rare condition and the importance of early recognition, due to its high mortality rate. Keywords: extracorporeal membrane oxygenation, air embolism, neonate, cardiac surgery, acute respiratory distress syndrome, barotrauma. JECT 2011;43:86–88

OVERVIEW

Extracorporeal membrane oxygenation (ECMO) is universally accepted as a potential lifesaving therapy for neonates suffering severe cardiorespiratory failure, with survival reported as 81% weaning off ECMO and 69% to hospital discharge in this population (1). Although ECMO may reduce mortality in certain neonatal patients, it is associated with significant complications. We present a fatal case of neonatal systemic air embolism (NSAE) leading to massive entrainment of air into the ECMO venous return cannula of a neonatal patient with acute respiratory distress syndrome (ARDS) following repair of obstructed total anomalous pulmonary venous connection (TAPVC). Knowledge of this rare condition is important for all ECMO teams that care for neonatal patients with severe lung disease. Institutional Review Board approval was obtained for this case report (X110323001).

DESCRIPTION

A 3.4-kg African American female was diagnosed with supracardiac obstructed TAPVC. During diagnostic workup in the neonatal intensive care unit, she experienced progressive hypoxemia and metabolic acidosis; oxygen saturations steadily declined to 50–60% with mean arterial pressure of 40 mmHg on FiO₂ 1.0, peak inspiratory pressure >35 cm H₂O, mean airway pressure (Paw) 25 cm H₂O, dopamine 20 mcg/kg/min, epinephrine .05 mcg/kg/min, vasopressin .04 units/kg/h, and milrinone .5 mcg/kg/min. Chest x-ray revealed bilateral dense infiltrates. After diagnosis, the patient was taken urgently to the operating room, and upon induction of anesthesia she had cardiac arrest and was emergently placed on cardiopulmonary bypass (CPB). The patient subsequently underwent repair of supracardiac TAPVC via direct anastomosis of venous confluence to posterior left atrium with no residual pulmonary venous obstruction. There was residual ascending vein communication between the superior vena cava and pulmonary confluence. CPB time was 112 minutes, aortic cross...
clamp time was 40 minutes, and the chest was left open. Postoperatively, the child had low cardiac output, pulmonary hypertension and acidosis requiring high frequency oscillatory ventilation (HFOV), nitric oxide, epinephrine .2 mcg/kg/min, vasopressin .04 units/kg/h, phenylephrine 2 mcg/kg/min, and dopamine 20 mcg/kg/min to maintain mean arterial pressure >40 mmHg and oxygen saturations >90%. Echocardiogram 3 hours after admission to cardiac intensive care unit revealed severe right ventricular dysfunction, so the decision was made to place the child on ECMO via central cannulation. During surgical placement of cannulae, the child suffered cardiac arrest and received 5 minutes of cardiopulmonary resuscitation (CPR) until ECMO support was initiated. The child’s hemodynamics quickly stabilized on ECMO with excellent global oxygenation. All cardiovascular medications except milrinone were quickly weaned off, and she remained on HFOV with Paw of 15 cm H₂O, amplitude 10 cm H₂O, Hertz 15, FiO₂ .3. There was minimal bleeding.

Our ECMO circuit has transformed over the years to a rapid response type of circuit. It consists of 1/4" A-V loop, with a venous saturation/hematocrit (SAT/HCT) probe (Terumo Cardiovascular, Ann Arbor, MI), a Capiox SP 45 centrifugal head (Terumo Cardiovascular, Ann Arbor, MI) that is compatible with the Medtronic 560 console (Medtronic, Minneapolis, MN), and a Pediatric Quadrox, Polymethylpentene Oxygenator (Maquet Cardiovascular, Wayne, NJ). We also have a 1/4" bridge with a leur and a stopcock that enables us to flash the bridge without the consequences of delivering old blood to the patient. Total prime is approximately 200 mL. Perfusionists initiate and manage all ECMO runs at the bedside 24/7.

On the second day of ECMO, during routine endotracheal tube bag and suctioning, the perfusionist noticed large amounts of air returning in the venous line with each manual breath. In retrospect, the clinician noted the child’s lungs to be very non-compliant, subjectively requiring significantly more pressure to move the chest than expected. Air completely filled the venous line from the patient to just below the bridge (approximately 2 feet of tubing or 20 mL); it did not reach the centrifugal pump. Bagging was stopped and the perfusionist was able to expel the air by clamping above the bridge on the arterial side of the circuit and below the bridge on the venous side of the circuit; air was worked into the bridge and expelled via a stopcock without any observed air returning to the patient. Air ceased to fill the venous cannula once bagging was stopped, and the circuit was inspected for air prior to restarting ECMO support. The child was returned back to the previous ventilator settings. There was no pneumothorax on chest x-ray or air leak in the chest tubes; echocardiogram confirmed the venous cannula had not been dislodged from its mid-atrium position. Despite no further bagging, there were two more episodes over the next few hours, and the air was again cleared from the venous line. We presumed this complication to be due to barotrauma, so after the third episode all ventilation to the patient was stopped. She was placed on continuous positive airway pressure (CPAP) of 10 cm H₂O, with no further suctioning. There were no more episodes of ECMO circuit air on CPAP for the next 4 days. On ECMO day 7, a head ultrasound demonstrated probable embolic stroke.

Due to two failed ECMO weans, the child underwent cardiac catheterization on ECMO day 6, which revealed severe pulmonary hypertension but no anatomic abnormality. The child’s lungs were then successfully re-recruited without incident over 2 days with gradual increases in the ventilator settings and frequent bagging and suctioning; on ECMO day 8, the child was on full ventilation with HFOV. A transition to the conventional ventilator was planned to facilitate weaning from ECMO, but while bagging and awaiting switch out of ventilators, the child acutely developed pulmonary hemorrhage and the venous circuit quickly filled with massive quantities of air again. The pump was stopped and two perfusionists worked diligently to de-air the circuit, but large columns of air continued to flow down the venous line upon attempting to re-initiate ECMO support. After 10 minutes off ECMO the child had cardiac arrest, and full CPR was initiated. CPR was continued for 15 minutes at which time we noticed a significant amount of air on the arterial side of the ECMO circuit. At this point, resuscitative measures were stopped.

**COMMENT**

Air in the ECMO circuit complicates about 4.9% of neonatal runs (1). This is a potentially fatal complication, especially if air occurs or travels to the arterial side of the circuit. Etiologies may include entrainment of air into the venous side via an open stopcock, central line, or equivalent; cannula dislodgement; inadvertent injection of air into circuit during medication or volume administration; and cavitation of right atrium (RA) in low volume states. Safety systems to address this complication include air detectors with a capability to identify air bubbles .3 mL to .5 mL in the ECMO circuit. Improved venous compliance reservoirs (Better Bladder; Circulatory Technology Inc, Oyster Bay, NY) are an additional component that adds to safety by reducing cavitation of intravascular structures, reducing air bubbles coming out of solution and entering the ECMO circuit.

NSAE is a rare complication of barotrauma due to positive pressure ventilation in patients with severe lung disease, and may also be a rare cause of air in an ECMO circuit. NSAE is thought to occur as result of interstitial emphysema and/or alveolar rupture into small pulmonary veins or capillaries (bronchovenous fistula) with air transit.
to the left atrium and then systemically (2–5). Arterial embolization of air leads to organ ischemia or infarction, most devastating in the coronary arteries or brain. An alternative explanation for NSAE (5) is dissection of interstitial air into the pulmonary lymphatics with transit to central veins via the thoracic duct. Arterial air occurs in this model by right to left shunting through a patent ductus arteriosus or patent foramen ovale often present in neonates. There are a few case reports of NSAE occurring after cardiac surgery (6,7). Diagnosis of NSAE is often made by detection of air within intravascular structures on x-ray or bubbles within an umbilical catheter in a patient with evidence of barotrauma. Echocardiography can be used to identify the origination of air entering the left atrium (7). NSAE is associated with a very high mortality and often has a dramatic clinical presentation such as acute cardiopulmonary collapse or seizures (2,4,5). The initial treatment for NSAE includes head-down position, 100% oxygen, and resuscitative measures as indicated (8). Ventilation pressures should be lowered as much as possible, as the bronchopulmonary venous fistula must be given time to heal. Selective lung ventilation and ECMO have both been used as extraordinary therapies for resolution of the air leak (7).

The patient had severe ARDS as a result of lung damage caused by the cumulative effects of her obstructed pulmonary venous return, barotrauma from high peak pressures, and the deleterious effects of CPB. There were emphysematous changes to her lungs upon arrival to the cardiac intensive care unit, and high Paw on HFOV (30 cm H2O) was still required to oxygenate her postoperatively. It is likely this severe lung damage primed her for the development of an NSAE that started upon manual ventilation on the second ECMO day.

The persistent ascending vein connection from the pulmonary vein confluence to a right superior vena cava may have permitted rapid transit of air to the RA from a bronchopulmonary venous fistula as evidenced by the instantaneous return of air to the venous cannula with each squeeze of the bag. The high airway pressure compared to the negative pressure in the RA (suction from the centrifugal pump) created a very large pressure gradient for rapid transit of the air to the venous ECMO cannula. This pressure gradient also likely prevented massive arterial air embolization to the patient, along with the quick and efficient actions of the perfusionist to de-air the circuit before it could overwhelm the air filtration properties of our oxygenator. The arterial cannula filled with air during CPR on the last ECMO day, which was likely a result of massive NSAE during CPR, as the pressure gradient to the RA was now gone. The patient also suffered an embolic stroke while on ECMO, which may have been the result of arterial embolization of some air during the ECMO run.

The patient’s outcome was consistent with the high fatality reported for neonates with NSAE (2,4,5). Her best chance of a better outcome would have been to avoid barotrauma with earlier initiation of respiratory ECMO support preoperatively or transition straight to ECMO off CPB to rest her lungs. The high pressures necessary to ventilate and oxygenate her postoperatively ultimately led to her fatal NSAE. In addition, there was a few hours delay in providing complete lung rest as we worked toward the diagnosis of NSAE, which likely worsened the bronchopulmonary venous fistula with continued ventilation of her low compliance lungs.

We speculate NSAE may be the cause of air in the venous circuit of occasional neonatal ECMO patients with severe lung disease who have potential intracardiac shunts (i.e., patent foramen ovale or congenital heart disease). Our dramatic case highlights a need for vigilance to identify any air in the ECMO venous return cannula, as cases of NSAE in neonatal ECMO patients with ARDS may present more subtly than our patient. Awareness of this rare condition will allow early diagnosis and treatment including immediate cessation of all ventilation to the patient for at least 48 hours and very judicious manual bagging afterward, which will decrease the risk of systemic embolization of air and the associated high mortality rate in neonates. In addition, it is essential that all ECMO caregivers are trained in the prevention of air embolism and possess the knowledge necessary to efficiently identify and remove air from the ECMO circuit to prevent life threatening consequences.

REFERENCES