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Case Report

Management of Neurogenic Pulmonary Edema and Differential Hypoxemia in an Adult Supported on Venoarterial Extracorporeal Membrane Oxygenation

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PATIENTS SUPPORTED on peripheral venoarterial extracorporeal membrane oxygenation (VA-ECMO) can have differential hypoxemia if cardiac function is regained in the clinical setting of poor gas exchange within the native pulmonary bed. Differential hypoxemia is demonstrated by hypoxemia of the upper extremities, brain, and myocardium while adequate oxygen perfusion is maintained in the lower extremities through extracorporeal membrane oxygenation (ECMO). Management of differential hypoxemia in this patient was complicated by acute onset of neurogenic pulmonary edema (NPE), an uncommon complication of anoxic brain injury that compromised oxygenation through the native pulmonary bed. To the authors' knowledge, this is the first case report of rescue VA-ECMO for cardiac arrest resulting in differential hypoxemia in the setting of NPE.

Case Report

A 19-year-old man was found unresponsive by his college roommate after taking an unknown illicit substance. Because of his ongoing asystole, paramedics immediately initiated 15 minutes of cardiopulmonary resuscitation before achieving a return of spontaneous circulation, after which time he was transferred to a local hospital for further care.

Initial physical examination found the patient nonresponsive to painful stimuli, with fixed 5-mm pupils and coarse breath sounds but with retained ventilatory drive with tachypnea to more than 40 breaths/min on pressure-support mode of mechanical ventilation. Multiorgan failure was evident, including cardiogenic shock (necessitating concurrent norepinephrine, epinephrine, and vasopressin infusions), acute hypoxic respiratory failure (PaO_2 of 62 with fraction of inspired oxygen [F_IO_2] of acute kidney injury) (creatinine, 150 μmol/L), metabolic disturbances (lactate concentration of 9.9 mmol/L and pH of 6.9), and coagulopathy (international normalized ratio of 1.4 and partial thromboplastin time of 34 s).

Transfer to the cardiac operating room was accomplished emergently, with initial transesophageal echocardiography showing profound biventricular hypokinesis, with estimated ejection fraction less than 10% with severe right ventricular failure. Given the patient's rapid decompensation, the decision was made to initiate mechanical support. An oblique 4-cm right groin incision was made to expose the femoral artery and vein, followed by performance of a purse string on the femoral

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vein using 4-0 polypropylene. Intravenous heparin, 200 U, was administered. Placement of the 29-Fr venous cannula was performed using a modified Seldinger technique (direct surgical approach and percutaneous technique for insertion of the guidewire and cannula) and was advanced to the right atrium with position confirmed by transesophageal echocardiography. An 8-mm end-to-side graft was sutured to the right common femoral artery with continuous 4-0 polypropylene. The femoral arterial chimney graft was cannulated using an 18-Fr arterial cannula. Cardiopulmonary bypass was initiated and induced hypothermia to 32°C was performed. Due to the absence of an immediately reversible etiologic factor and ongoing profound cardiogenic shock, the patient's care was transitioned subsequently to a dedicated portable ECMO pump and oxygenator. The cannulae were sutured to the skin, temporary dressing was applied, and he then was transported to a tertiary referral ECMO center for further treatment.

On admission, the patient had persistent therapeutic hypothermia (34.0°C), mean arterial pressures between 60 and 70 mmHg, a circuit flow of 4 L/min corresponding to a cardiac index of 2.1, sweep gas flow of 3 L/min, and sweep gas F_IO_2 of 1.0. Initial mechanical ventilator settings were the following: mode, continuous mandatory ventilation; rate, 12; tidal volume, 400 mL, F_IO_2, 0.5; and positive end-expiratory pressure (PEEP), 6 mmHg. Neurologic examination revealed 8-mm pupils that were fixed and dilated with no oculocephalic response or corneal reflexes. Computed tomography of the head showed diffuse loss of the gray-white differentiation accompanied by complete obliteration of the sulci, effacement of the ventricles, and fullness of the foramen magnum consistent with diffuse anoxic brain injury and associated herniation (Fig 1). A discussion was held with the neurologic intensivists and the family about the patient's condition. A formal brain death examination could not be performed at that time because multiple confounders were present, including hypothermia and a pending comprehensive toxicology evaluation. Importantly, the family expressed the patient's wishes for organ donation in such a situation. Hence, the authors elected to continue full support until further information became available and an official examination was possible.

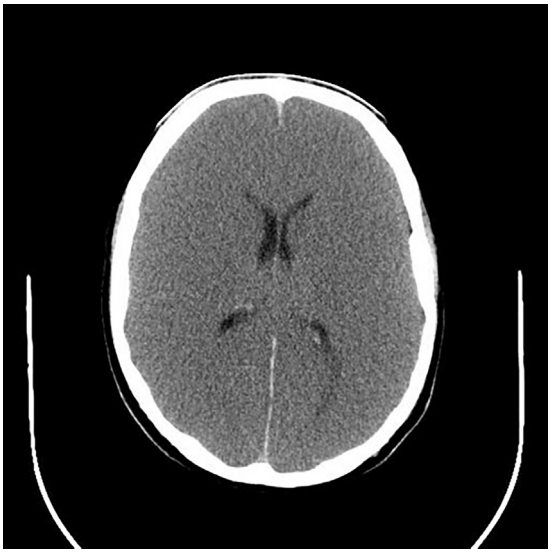

Fig 1. Computed tomography of the head without intravenous contrast medium. Diffuse loss of the gray-white differentiation accompanied by complete obliteration of the sulci and effacement of the lateral ventricles. These findings are consistent with diffuse anoxic brain injury with associated descending herniation.

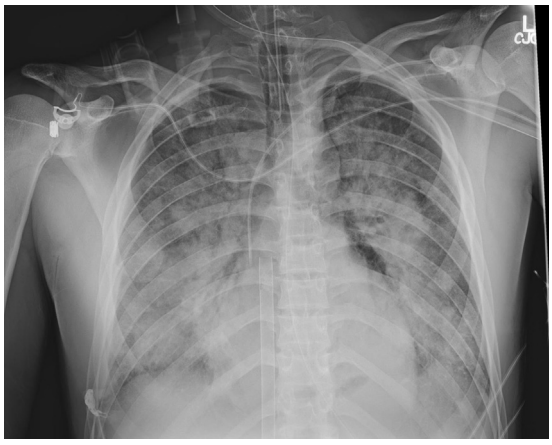

Fig 2. Chest radiograph during differential hypoxemic episode. Diffuse bilateral perihilar and lower-lung-predominant patchy opacities are shown.

Six hours after the patient's arrival, sinus tachycardia developed, with a heart rate of 100-to-130 beats/min. Concurrently, hypotension rapidly resolved over approximately 30 minutes, prompting down-titration and subsequent discontinuation of vasopressin and norepinephrine infusions. The patient's urine output was 400-to-1,800 mL/h. In addition, serum osmolality was 304 mOsm/kg, urine osmolality was 301 mOsm/kg, and serum sodium concentration was 152 mmol/L, leading to a diagnosis of central diabetes insipidus. Crystalloid fluids and desmopressin were administered followed by a marked decrease in urine output to 200 mL/h within 3 hours. At this time, pulse oximetry showed marked asymmetry between the upper and lower extremities. Arterial blood gases (ABG) drawn from the ECMO circuit and the right radial arterial line showed marked discrepancy, with PaO_2 of 64 mmHg (arterial line) and 283 mmHg (postoxygenator), respectively.

The widely disparate oxygenation between the postoxygenator sample and proximal (relative to the aortic valve) sample was diagnostic for differential hypoxemia. Despite this, the etiologic factors of proximal hypoxemia remained unclear, with a differential that included vascular occlusion of retrograde perfusion impairing ECMO flow from reaching the proximal monitoring site and increased native cardiac ejection of hypoxemic blood, suggestive of improved contractility and concurrent suboptimal oxygenation within the native pulmonary bed. Emergent transthoracic echocardiography noted an improvement in left ventricular ejection fraction to 40% and appropriate venous cannula position. A chest radiograph showed acute development of diffuse bilateral perihilar and lower lung patchy opacities consistent with edema or pneumonitis (Fig 2). Subsequent bronchoscopy revealed gross pulmonary edema with noted absence of mucus plugging,

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