MAYO CLINIC

BRIEF REPORT

Severe Acute Cardiopulmonary Failure Related to Gadobutrol Magnetic Resonance Imaging Contrast Reaction: Successful Resuscitation With Extracorporeal Membrane Oxygenation

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Abstract

Nonanaphylactic noncardiogenic pulmonary edema leading to cardiorespiratory arrest related to the magnetic resonance imaging contrast agent gadobutrol has rarely been reported in the literature. Rarer is the association of hypokalemia with acidosis. We report 2 patients who had severe pulmonary edema associated with the use of gadobutrol contrast in the absence of other inciting agents or events. These cases were unique not only for their rare and severe presentations but also because they exemplified the increasing role of extracorporeal membrane oxygenation in resuscitation. Emergency extracorporeal membrane oxygenation resuscitation can be rapidly initiated and successful in the setting of a well-organized workflow, and it is a viable alternative and helps improve patient outcome in cases refractory to conventional resuscitative measures.

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Except for nephrogenic systemic fibrosis syndrome in patients with renal failure and rare anaphylactic reactions, magnetic resonance imaging (MRI) contrast agents are considered harmless.[1] We describe 2 patients with an extremely rare adverse reaction to an MRI contrast agent and the concerted utilization of extracorporeal membrane oxygenation (ECMO) to successfully manage life-threatening refractory cardiorespiratory failure. Both cases were associated with use of the gadolinium (a paramagnetic metal in the lanthanide series) chelates available as gadobutrol.

CASE 1

In October 2014, a 63-year-old man with history of isolated dextrocardia underwent a routine annual outpatient cardiac MRI for suspected dilated nonischemic cardiomyopathy. This was a follow-up from 2 previous cardiac MRIs completed to monitor for a low ejection fraction of 42%, initially documented by cardiac imaging in December 2010. The previous cardiac MRIs were performed using gadolinium chelate–based contrast agents other than gadobutrol (ie, gadodiamide and gadomenate dimeglumine). Other comorbidities included obstructive sleep apnea and hypertension. During the latest scan, he received 20 mL of intravenous (IV) gadobutrol contrast material (Gadavist, 1 mmol/mL; Bayer HealthCare Pharmaceuticals), and the scan was uneventfully completed without patient sedation. Ten minutes after completing the scan, the patient developed acute dyspnea and was immediately transferred to the nearby emergency department (ED) at Mayo Clinic.

On arrival at the ED, the patient was anxious, diaphoretic, and producing copious frothy white oral secretions. His respiratory rate was 30 breaths/min, and hemoglobin saturation (via pulse oximetry) was 60% while the patient was breathing ambient air. He was normotensive, with a blood pressure of 111/83 mm Hg, and his pulse rate was

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95 beats/min. An initial electrocardiogram demonstrated left bundle branch block. Oxygen supplementation was provided via a nonrebreathing mask, and he received epinephrine 0.3 mg intramuscularly, dexamethasone 10 mg IV, diphenhydramine 50 mg IV, and albuterol-ipratropium 0.3 mg/0.5 mg in an inspired aerosol to treat a suspected anaphylactic reaction. Arterial blood gas analysis showed pH 7.39; P_{O_2}, 53 mm Hg; P_{CO_2}, 39 mm Hg; and bicarbonate, 23 mmol/L. His blood lactate concentration was mildly elevated (3.0 mmol/L), but other laboratory values, such as hematocrit, leukocytes, platelets, coagulation profile, troponin, and creatinine, were normal. His serum tryptase level was 7.6 ng/mL (reference range, <11.5 ng/mL). The initial chest radiograph suggested pulmonary edema (Figure 1).

His condition continued to deteriorate, and he became hypotensive within 30 minutes of presentation to the ED. Emergent tracheal intubation was performed, and a right internal jugular central venous catheter was placed for norepinephrine administration. Vasopressin and epinephrine were subsequently given IV in incremental doses, for a total of 10 U and 75 μg, respectively. He also received IV methylene blue for ongoing hypotension and IV diuretics for worsening hypoxemia and pulmonary edema. He was transferred to the coronary care unit, where a repeat electrocardiogram showed dynamic T-wave changes in the lateral leads suggestive of possible coronary ischemia. He continued to be hypoxemic despite intensive ventilatory support with 100% oxygen and positive end-expiratory pressure of 18 cm H_2O. He underwent emergent coronary angiography, which demonstrated normal coronary arteries. Transesophageal echocardiography was performed in the coronary catheterization laboratory and revealed markedly reduced right and left ventricular systolic function. Repeat blood analysis 2 hours after arrival at the ED was significant for acute hypokalemia (potassium level, 2.8 mmol/L) despite metabolic acidosis with a blood pH of 7.2.

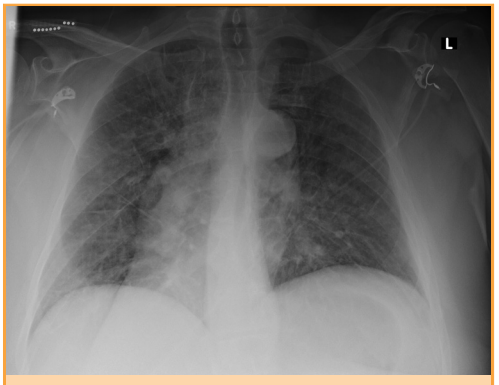

FIGURE 1. Initial chest radiograph of case 1 showing features of pulmonary edema.

In view of the tenuous hemodynamics requiring high vasopressor support and the persistent, severe hypoxemia (arterial P_{O_2} of 49 mm Hg), the decision was made to initiate peripheral venoarterial ECMO (VA-ECMO). Percutaneous cannulation was performed using a 17 French arterial cannula to the right femoral artery and a 25 French venous cannula to the right femoral vein. The cannulas were positioned under fluoroscopic and transesophageal echocardiographic guidance. By then, the patient developed junctional cardiac rhythm with global left ventricular hypokinesia (ejection fraction of 10%-15%). A pulmonary angiogram did not show evidence of pulmonary embolism. A temporary transcutaneous pacemaker was placed for episodes of bradycardia, and atrial septostomy was performed to preempt left ventricular overload and prevent pulmonary edema or hemorrhage. After a period of stabilization, a left femoral artery Dacron chimney graft was fashioned in the operating room for arterial inflow to maintain adequate lower-extremity perfusion. The original right venous cannula was retained.

Twenty-four hours after ECMO initiation, his hemodynamics significantly improved, and vasopressors were discontinued. Repeat bedside transesophageal echocardiography showed improved left ventricular function (ejection fraction of 40%) on day 3 of the intensive care unit stay. ECMO was discontinued, and the associated catheters were removed. The trachea was extubated the following day. The patient had no neurologic deficits, and was discharged from the intensive care unit.