Pheochromocytoma-Associated Cardiogenic Shock due to Metoclopramide Managed with ECMO

Sheinberg R, Mitter
Johns Hopkins Medical Institute, Baltimore, MD, USA

Introduction: Pheochromocytoma is a catecholamine-secreting neuroendocrine tumor with typical symptoms of paroxysmal headache, palpitations, and severe hypertension. We present a case of cardiogenic shock due to use of Metoclopramide secondary to a pheochromocytoma that was managed with ECMO.

Case Presentation: A 45-year-old female presented with a severe headache associated with nausea, vomiting and palpitations. After a dose of Metoclopramide for nausea, the patient exhibited acute hypertension, tachycardia and diaphoresis. She deteriorated further with EKG elevations, hypotension and respiratory failure requiring intubation. Coronary catheterization revealed normal vessels but echocardiography revealed severe global hypokinesis with an ejection fraction of 5-10%. Laboratory values demonstrated elevated metanephrines. After placement of an intra-aortic balloon pump (IABP), the patient was transferred to our institution. She was placed on veno-arterial extracorporeal membrane oxygenation (VA-ECMO) via her left femoral vessels. Abdominal ultrasound revealed a right adrenal mass. ECMO was discontinued after two days because of improved systolic function. Six hours after decannulation, systolic blood pressure was measured above 200mm Hg and an asystolic cardiac arrest ensued. ACLS was instituted and ECMO reinstated. Despite these measures the patient continued to decompensate and expired.

Discussion: Only a small percentage of patients diagnosed with pheochromocytoma present with heart failure. This case highlights the cardiac surgical management of a condition normally seen in the general operating rooms as well as drug management in a patient suspected to have a pheochromocytoma. Dopamine type-2 receptors are presynaptic on sympathetic nerve endings and stimulation inhibits release of catecholamines. Metoclopramide blocks this inhibition and may act as a potent trigger of severe catecholamine release in the presence of a pheochromocytoma which can lead to acute cardiogenic shock, as it did in our patient. 2

There are only about 5 case reports of pheochromocytoma-induced acute heart failure being treated with percutaneous cardiopulmonary bypass systems.1 The mechanism underlying pheochromocytoma-induced heart failure may include excess sympathetic stimulation, myocardial stunning, ischemia due to coronary artery spasm and direct toxic effects of catecholamines on myocytes.3 Standard preoperative treatment using alpha blockers to reverse coronary vasoconstriction or beta blockers for tachycardia is not feasible when blood pressure is low, nor is the administration of inotropic agents in patients who already have a surplus of endogenous catecholamines.4 ECMO and VADs provide more complete circulatory support than IABPs and have been used in patients with catecholamine-induced cardiogenic shock due to pheochromocytoma. Given the high mortality rate associated with medical management and that the clinical course of myocardial dysfunction in pheochromocytoma-induced cardiogenic shock is reversible, the early use of percutaneous cardiopulmonary bypass systems should be considered.