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Case Report

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A Case of Septic Cardiomyopathy

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Introduction

Sepsis includes a life-threatening organ dysfunction as a result of a dysregulated host response to an infection [1]. Sepsis can sometimes result in septic cardiomyopathy, a sequela that is associated with increased mortality in sepsis [2,3]. Septic cardiomyopathy implies a temporary and non-ischemic cardiac dysfunction that can happen in septic states. Septic cardiomyopathy is defined as a newly discovered reduction in left ventricular ejection fraction (LVEF) of </= 50% or a 10% reduction in LVEF with known heart failure with reduced ejection fraction (HFrEF) [2]. Septic cardiomyopathy is characterized by rapid onset and it is usually reversible (usually in 7 to 10 days) with no identifiable coronary lesions [4].

Case Report

A 65-year-old male with a history of hypertension, coronary artery disease (CAD), hyperlipidemia, chronic obstructive pulmonary disease (COPD), and prostate adenocarcinoma with a recent robotic prostatectomy and ileal neobladder construction did present to the emergency department (ED) with fevers, chills, anorexia, and flank pain over the previous 3 days. In the ED, the patient was noticed to be septic with underlying septic shock. The patient was given a dose of intravenous (IV) ceftriaxone, and vancomycin. The patient was given 3 liters of IV crystalloids with minimal improvement in blood pressure (BP) and persistent low BP that necessitated starting the patient on norepinephrine and vasopressin to maintain adequate organ perfusion pressure. Despite requiring norepinephrine for maintaining adequate organ perfusion pressures, the patient was very alert and oriented to place, person, time, and purpose. He was still breathing spontaneously and requiring only minimal supplemental oxygen (at 2 liters/min via nasal cannula). The patient was admitted to the Intensive Care Unit (ICU) for further care.

While in the ICU, the patient had an episode of pulseless electrical activity (PEA) without any identifiable triggers and therefore, a code blue was activated. Advanced cardiac life support (ACLS) algorithm was followed with eventual return of spontaneous circulation (ROSC). The patient had an advanced airway placed during the code and the patient received multiple doses of epinephrine pushes with achievement of ROSC and adequate perfusion pressures only upon initiation of dobutamine drip. After attaining ROSC, a point of care ultrasound (POCUS) was done, which showed a dilated left ventricle with poor contractility and no evidence of right ventricular failure (please see figure 1). The patient subsequently underwent a transthoracic echocardiogram (TTE) that showed severely reduced ejection fraction of 32% with akinesis of the septal, anteroseptal, and inferior walls along with inferolateral wall hypokinesis. The TTE also revealed normal right ventricular size and systolic function. Post cardiac arrest, the patient was maintained on norepinephrine, vasopressin, and dobutamine drips to keep adequate organ perfusion pressure (titrated to keep a mean arterial pressure of 65 mm of mercury). The patient could not tolerate even minimal decreases in dobutamine drip. As such, norepinephrine and vasopressin were titrated down initially before dobutamine was titrated down. The patient was maintained on dobutamine drip at a rate of 10 micrograms/kilograms/min for 30 hours before this can be titrated down to keep his MAP more than or equal to 65 mm Hg to ensure adequate vital organ perfusion. Dobutamine drip was eventually turned off in 72 hours following the cardiac arrest once the patient had a MAP >/= 65 mm Hg without any vasopressor requirements.

The patient's lactic acid post cardiac arrest was as high as 22 millimols/liter and had a serum bicarbonate of 10 millimols/liter. Serum cortisol was 113 micrograms/deciliter with no prior documented steroid therapy. Troponin following cardiac arrest that necessitated chest compressions was 0.123 nanograms/milliliters (normal high >=0.034; critically high>0.120). The patient's blood cultures from admission was positive for Klebsiella oxytoca (pan sensitive except for ampicillin) and Enterococcus faecalis (vancomycin sensitive). Urine culture from admission (sample obtained from ileal conduit) was positive for Klebsiella oxytoca (colony count >=100,000 cfu/ml) and Enterococcus faecalis (colony count 40,000-49,000 cfu/ml). Klebsiella oxytoca was pan sensitive to penicillin except to ampicillin. Enterococcus faecalis was sensitive to vancomycin. The patient's antibiotics were tailored according to the sensitivities and the patient was treated with the antibiotics for the required duration. The patient had a prolonged hospital course because of difficulty in weaning from mechanical ventilator and eventually had a tracheostomy placed before he was fully weaned off from the ventilator. The patient was discharged to a long-term acute care facility before he was discharged home. Fortunately, the patient did not have any evidence of anoxic brain injury from the cardiac arrest.

A repeat TTE was done in 20 days following the cardiac arrest, which showed normal systolic function with a LVEF of 75% with normal regional wall motion. The right ventricular size was normal with normal systolic function. A review of the patient's prior medical records showed that he had a coronary angiogram

done in 2016 that showed mid left anterior descending artery 30% stenosis that was treated with balloon angioplasty. He had a TTE done in October 2021 that showed a LVEF of 63%. As such, this patient was admitted with septic shock with no documented ischemic cardiomyopathy but had a new onset cardiomyopathy (LVEF = 32%) that was reversed eventually (LVEF = 75%), suggesting that the patient had septic cardiomyopathy. There was no evidence of apical ballooning in the POCUS or TTE done following the cardiac arrest suggesting that the cardiomyopathy was not stress cardiomyopathy (Tako-Tsubo cardiomyopathy). In addition, this case of cardiomyopathy was in a 65-year-old male, while Tako-Tsubo cardiomyopathy is almost exclusively seen in postmenopausal women [5].

Discussion

The identification of shock secondary to septic cardiomyopathy is challenging as the presentation mimics other causes of shock specifically if a patient presents with all the pathognomic signs of septic shock. In this case, a perfusing rhythm and adequate organ perfusion pressures could not be attained without the addition of an inotrope (dobutamine). Routine evaluation of the hemodynamics in shock states utilizing POCUS has become the standard of care. However, if the POCUS suggests pump failure (low ejection fraction), it is difficult to identify if a patient has a known history of heart failure with reduced ejection fraction (HFrEF) suggesting cardiogenic shock or if it is a new onset heart failure (while acute ischemia is ruled out using negative troponins and EKG), specifically if the patient is a poor historian or if the patient is not in a condition to provide adequate history. As such, the selection of the ideal vasopressors or ionotropes that could be used in the clinical scenario becomes more challenging. Despite being a reversible phenomenon, septic cardiomyopathy has high mortality. Therefore, appropriate recognition and timely management of septic cardiomyopathy are essential in sepsis. Expert guidelines suggest addition of dobutamine to norepinephrine if there is evidence of cardiac dysfunction in septic shock despite adequate arterial blood pressure and organ perfusion [6].

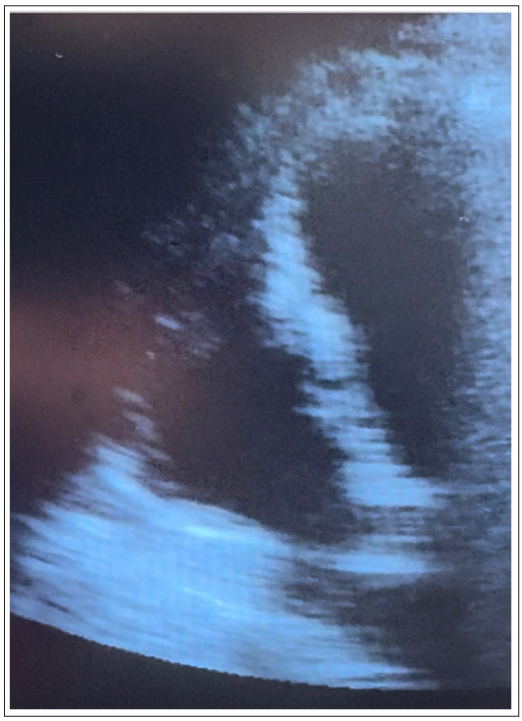

Figure 1: POCUS showing Dilated Left Ventricle with Hypokinesis of the Interventricular Septum

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