Abstract for Case Report

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Title: Challenges in Managing Cardiogenic Shock: A Case of Dobutamine-Induced Lactic Acidosis in a Morbidly Obese Patient

Introduction:

Managing cardiogenic shock secondary to de novo acute heart failure (AHF) and unstable angina presents a significant challenge in emergency medicine, particularly in patients with morbid obesity and no previous comorbidities. This case report sheds light on the complexities associated with the use of dobutamine and its potential contribution to exacerbating lactic acidosis.

Case Presentation:

A 38-year-old Bidayuh man presented to the emergency department with a one-week history of decreased effort tolerance, worsening shortness of breath, and central chest pain at rest. Diagnosed with cardiogenic shock secondary to AHF and unstable angina, initial treatment included intravenous noradrenaline for blood pressure support, intravenous dobutamine (20 mcg/kg/min) to enhance cardiac contractility, bilevel positive airway pressure (BIPAP), and intravenous furosemide 80 mg. However, after 4 hours of therapy, the patient developed worsening lactic acidosis as indicated by arterial blood gas (ABG) analysis. Upon consultation with the cardiologist, the dobutamine dose was reduced to 5 mcg/kg/min, resulting in the resolution of metabolic acidosis. Subsequent ABG analysis 30 minutes later allowed for the safe transfer of the patient to Sarawak Heart Centre via ambulance.

Discussion:

This case is noteworthy as the resolution of metabolic acidosis was achieved following a reduction in the intravenous dobutamine dose. Initially, the high dose of dobutamine was administered due to the patient's tachypnea on BIPAP and mild metabolic acidosis. However, despite therapy with IV furosemide, BIPAP, and dobutamine with noradrenaline, metabolic acidosis worsened. In retrospect, the use of the patient's actual body weight, which was obese at 100 kg, instead of the recommended ideal body weight of 60 kg, may have contributed to this exacerbation. Additionally, there is a lack of studies evaluating the influence of obesity on dobutamine dosing or pharmacokinetics. Dobutamine, with its short onset of action and small volume of distribution, can rapidly titrate to clinical effect after initial

dosing, but caution should be exercised in patients with active myocardial ischemia or recent myocardial infarction, as it can increase myocardial oxygen demand and lead to metabolic acidosis through anaerobic metabolism. Despite this, the patient showed signs of improvement, likely due to early intervention in the emergency department and increased urine output. It's worth noting that long-term use of dobutamine has been associated with increased mortality, necessitating consideration of alternative agents like milrinone and levosimendan, although these are not readily available in Malaysia. Ultimately, a balanced and judicious use of inotropic agents, with appropriate dosage adjustments based on patient characteristics, is paramount in managing critically ill patients.

Conclusion: This case underscores the potential for dobutamine to exacerbate lactic acidosis in patients with severe heart failure. Emergency medicine practitioners must exercise caution with inotropic agents, ensuring close monitoring and appropriate dosage adjustments based on ideal body weight. Timely recognition and management of metabolic derangements are crucial for improving outcomes in patients with cardiogenic shock.

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