METHYLENE BLUE ADMINISTRATION IN REFRACTORY VASOPLEGIC TOXIC SHOCK SYNDROME

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INTRODUCTION: Vasoplegia, or hypotension refractory to vasopressor therapy, is a key risk factor for death in shock. Catecholamine-sparing agents play an essential role in ensuring adequate perfusion. We report a case of methylene blue (MB) administration in a patient with refractory distributive shock secondary to Streptococcal Toxic Shock Syndrome (TSS), with subsequent sustained decreased vasopressor and inotropic requirements.

CASE PRESENTATION: A 34 year old male, recently diagnosed with Influenza B, presented with dyspnea and hemoptysis. On arrival he was afebrile (37.6 °C), hypotensive (74/62 mmHg), tachycardic (110 bpm), tachypneic (39 bpm) and hypoxemic (SpO2 80%). Chest CT showed extensive bilateral lung infiltrates, consistent with multifocal pneumonia. Despite aggressive fluids, broad-spectrum antibiotics, Oseltamivir, stress-dose steroids, and vitamin C, he decompensated requiring multi-agent vasopressor support, and was intubated for respiratory failure. Blood and sputum cultures grew Streptococcus pyogenes, and Clindamycin was added with concern for TSS. His course progressed to acute respiratory distress syndrome requiring veno-venous extracorporeal membrane oxygenation (VV-ECMO), and was complicated by renal failure requiring continuous renal replacement therapy and septic cardiomyopathy. He remained hypotensive and acidotic despite prolonged high doses of norepinephrine, epinephrine, vasopressin, and dobutamine, which precipitated severe rhabdomyolysis, critical limb ischemia, and dry gangrene of all 4 limbs. In the setting of vasoplegia a methylene blue loading lose and continuous infusion was initiated on hospital day (HD) 4, with subsequent sustained significant decline in vasopressor requirement.

DISCUSSION: Risks associated with catecholamine vasopressors, such as peripheral and mesenteric ischemia, have prompted interest in alternative agents. MB is a non-adrenergic vasopressor, with data to support blood pressure elevation and acceleration of catecholamine discontinuation (1, 2). However, MB still lacks robust evidence, especially in patients with septic shock. One review concluded that use of MB in sepsis increased systemic vascular resistance and mean arterial pressure, though the studies examined predated more recent advances in critical care (3). Our use of MB in a patient with TSS supports its subsequent decrease in catecholamine requirements, allowing for lower vasopressors doses and reduced adverse effects.

CONCLUSIONS: Following MB administration our patient was progressively tapered from inotropic and vasopressor support, and was weaned from ECMO on HD8. He had ongoing multi-organ failure and was ultimately transitioned to comfort measures before dying. His case illustrates how methylene blue can be an effective treatment of refractory hypotension related to septic shock. Clinical trials are needed to define administration guidelines and strengthen the evidence for MB usage.


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