

BLUE OCEAN STRATEGY : METHYLENE BLUE IN REFRACTORY SHOCK

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INTRODUCTION

Majority patients in shock will respond to vasopressors for hemodynamic stabilization besides the treatment of etiology. Common vasopressors used are noradrenaline, adrenaline and vasopressin. Methylene blue is an evolving vasoactive agent and not routinely used. We share our experience in using methylene blue for refractory shock at our centre.

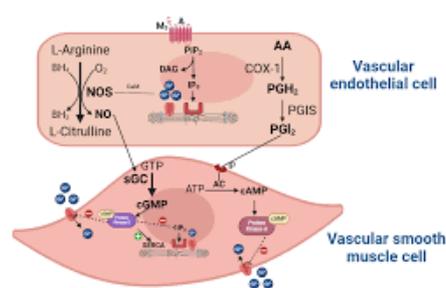
CASE DESCRIPTION

A 49 years old male with no known medical illness and allergy presented at our centre with persistent vomiting, loose stool and rapid breathing. He was profoundly hypotensive and tachypnoeic. However, he was afebrile and not tachycardic. The patient was in severe metabolic and lactic acidosis with pH of 7.09, bicarbonate of 8.2 and lactate of 8.0. His full blood count revealed leukocytosis, blood urea 19.7 and creatinine 454. He was also oliguric. The patient was intubated and started on intravenous broad-spectrum antibiotic. Due to refractory shock despite fluid resuscitation and required three vasopressors at maximum dosage, he was started on methylene blue 2mg/kg slow bolus followed by infusion 0.25mg/kg/hr for 6 hours. Following that, the patient's hemodynamics improved and the acidosis resolved. Patient was admitted to intensive care unit (ICU) and underwent continuous veno-venous hemofiltration (CVVH) for 8 hours. Adrenaline was off on day 4 whereas noradrenaline and vasopressin on day 5 post methylene blue. His repeated blood urea and creatinine post CVVH were normal. His blood for leptospirosis, malaria and atypical screening were negative. His blood and urine culture shows no growth. He was treated for severe infective acute gastroenteritis enterotoxin producer and successfully extubated on day 9 of intubation. He was transferred out to medical ward and discharged well.

DISCUSSION

Sepsis often causes distributive shock which can cause multiorgan failure. It is caused by the release of nitric oxide via inducible nitrite synthetase (iNOS) in vascular endothelium. Nitric oxide (NO) will activate the guanylate cyclase that convert guanosine triphosphate (GTP) to cyclic guanosine monophosphate (cGMP) which lead to smooth muscle relaxation and severe vasoplegia. Methylene blue inhibit the nitric oxide-cyclic guanosine monophosphate (NO-cGMP) pathways, decreases the vasodilation effect and increases responsiveness to vasopressors.

Regulation of vascular tone by cGMP and cAMP



CONCLUSION

Our case shows that methylene blue can be used as an adjunctive agent for refractory septic or other vasodilatory shock. It could be considered when other vasopressors had been maximized.

Keywords

Methylene blue, refractory shock, sepsis

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