Methylene Blue Action to Treat Vasoplegic Syndrome is Time-Dependent

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Abstract

We present a case report of an ultrasound-guided femoral nerve block in a seven-year-old patient with an aberrant femoral nerve. The utilization of ultrasonography enabled the identification and successful blockade of an aberrant femoral nerve within the belly of the iliopsoas muscle. A review of the literature regarding aberrant femoral nerve anatomy is included.

As stated by Blacker and Whaler, there is strong evidence that methylene blue (MB), an inhibitor of guanylate cyclase, is an excellent therapeutic option for Vasoplegic Syndrome (VS) in heart surgery. The authors reported a case of VS during an on pump coronary artery bypass grafting with no response to MB. The patient was a 63 year old male with chest pain was found to have multivessel disease and referred for elective three vessels CABG. He was 72 inches and 89 kg with a history of hypertension, hyperlipidemia, and benign prostate hypertrophy. Medications included metoprolol 50 mg once a day, enalapril, hydrochlorothiazide, Zocor, Avodart and tamsulosin. Hydrochlorothiazide had been the only held morning medication. Anesthesia procedures, including intubation and insertion of a right pulmonary artery catheter, were accomplished uneventfully. Coincidently with tranexamic acid infusion, before the beginning of CPB, a severe hypotensive episode ruled the patient hemodynamic status. Arterial hypotension persisted uncontrollable, during and after CPB, despite the use of high doses of norepinephrine. Out of CPB, CI was 2.6 L/minute·m² with a Systemic Vascular Resistance (SVR) of 663 dyne·sec·cm⁻⁵, and the initial LV function appeared normal on TEE. CPB was re-initiated. Vasopressin and norepinephrine infusions continued. In consideration of adrenal insufficiency, 125 mg hydrocortisone was given intravenously. Despite no other signs or symptoms other than hypotension, anaphylaxis were considered and IV ranitidine and diphenhydramine were given; the latex Foley was replaced. After around 6 hours, an intra-aortic balloon pump (IABP) was placed. CPB was weaned and the blood pressure was in the high 90/50's mmHg. Vasopressin was continued as weaning would result in a MAP of 50 mmHg and tachycardia of 120 bpm. CPB was re-instituted due to persistent hypotension, a worsening lactate of 11.3 despite the IABP use. After discussion, vasopelia was considered, and 200 mg MB was given intravenously, 6 hours after the initial episode of hypotension. There was a brief rise in the MAP to the 70's mmHg but lasted only three minutes [1]. I have been “watching this movie” since the 80 s, a time-dependent. J Anesth Clin Res 4: 349. doi: 10.4172/2155-6148.1000349

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was proposed by Evora et al. However, the clinical practice shows that the MB sometimes works and sometimes not [5-7]. A possible explanation, emphasized by Blacker & Whaler, was given by Fernandes in his academic thesis. Using a mouse sepsis model, three eight-hour windows of guanylate cyclase activity were evidenced [8]. Sepsis like VS is a state of profound refractory vasodilation. In the first eight hours, there is increased nitric oxide synthase activity and upregulation of guanylate cyclase. The second eight hours there is an absence of guanylate cyclase expression and a down regulation of nitric oxide synthase. The third eight hour window occurs and there is an upregulation of guanylate cyclase and nitric oxide synthase.

Retrospectively, discussing the presented case: 1) SV diagnostic by observing amine resistant hypotension after tranexamic acid infusion; 2) MB 2 mg/kg infusion before starting the CPB; 3) MB continuous infusion during CPB based on the need of overflow and high dose NOR; 3) The IABP would be contraindicated because it is a situation of vasoplegia and 4) As additional measures, consider the use anti-inflammatory doses of corticosteroids and low doses of metoprolol to counteract the phenomenon of beta receptors “downregulation”.

The motivation of this long letter to the editor was the authors’ contribution, practical and education, singularly fundamental aspects: 1) The disclosure of the team to use the MB window opportunity syndrome vasoplegic in heart surgery, and; 2) The need for the establishment of this window in humans, perhaps choosing cGMP as biomarker since our attempt to use nitrite/nitrate, measured by chemiluminescence was frustrating [3]. Congratulations to the authors by the attitude of seeking, in retrospect, why the MB had a transient effect not just dismissing its action as often happens. In addition, I guess that it has introduced an easier concept to understand than the “Window of Opportunity” definition....”MB ACTION TO TREAT VASOLEPIC SYNDROME IS TIME-DEPENDENT”.

References