Case Report

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Resuscitative Endovascular Balloon Occlusion of the Aorta to Facilitate Continuous Venovenous Hemodiafiltration in a Patient with Methanol Intoxication

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BACKGROUND

Resuscitative endovascular balloon occlusion of the aorta (REBOA) was first described by Hughes in the Korean War [1] and regained popularity in the late 2000s. It is successfully used in patients with non-compressible torso hemorrhage due to trauma [2] as well as situations associated with non-traumatic bleeding such as placenta percreta [3] and uncontrollable gastrointestinal bleeding [4]. As REBOA use evolves with time, the life-saving potential of the procedure, especially in bleeding patients, has made itself a useful tool in Emergency Departments (EDs) and also in prehospital environments [5].

CASE REPORT

A 61-year-old male presented to the ED with progressive loss of vision and impaired consciousness for 3 hours. According to his relatives, the patient was a chronic cologne abuser and had ingested an unknown amount of cologne one day prior. He had no medical history and was not on any medication. His initial vital signs were a blood pressure of 80/60 mmHg, a pulse of 98 beats/min, oxygen saturation of 94% and a temperature of 36.2 °C. Due to the patients Glasgow Coma Scale (GCS) of 6 (E4M1V1), he was intubated with rapid sequence intubation. Laboratory studies showed profound lactic acidosis with a calculated level of methanol of 319 mg/dl (pH: 6.76, HCO_3: 6.3 mEq/L, PO_2: 115 mmHg, PCO_2: 37 mmHg, lactate: 6 mmol/L, anion gap: 44). The patient was catheterized for urgent hemodialysis (HD) through his right internal jugular vein and transferred to the intensive care unit (ICU). While awaiting preparations for HD, fluid resuscitation, bicarbonate and norepinephrine infusion was initiated along with 10 cc/kg of intravenous ethanol bolus over 30 minutes followed by a continuous ethanol infusion of 2 cc/kg/h iv infusion. A right radial artery catheter was also placed for monitoring blood pressure. A continuous veno-venous hemodiafiltration (CVVHDF) was initiated due to persistent hypotension and profound acidosis. The blood ethanol level was measured at 6-hour intervals and the infusion was regulated to keep serum ethanol level over 150 mg/dl. As the patient became more hypotensive during CVVHDF, dopamine and epinephrine infusions were added and doses were escalated to reach a norepinephrine infusion of 40 mcg/min, dopamine infusion of 50 mcg/min, and epinephrine infusion of 40 mcg/min. After 24 hours, CVVHDF began to fail because the patient had persistent profound hypotension despite triple inotropic support and this yielded a loss of efficacy in the performance of the HD device.

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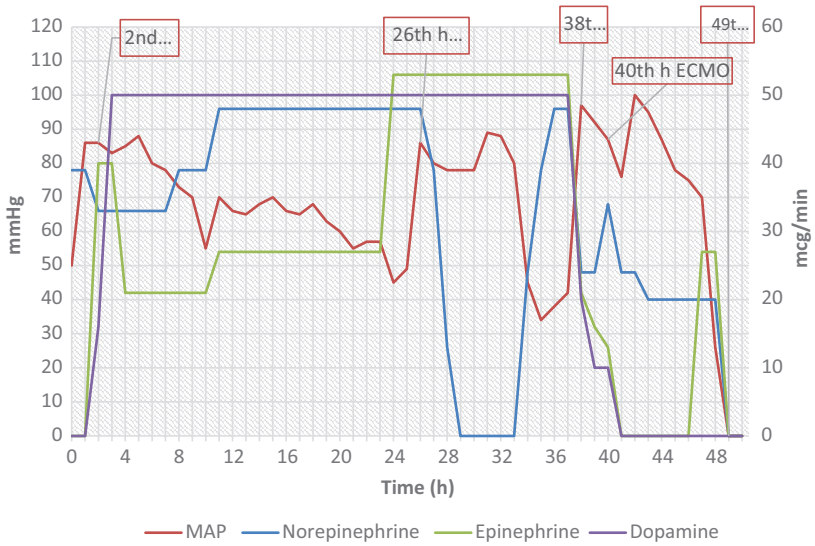

Figure 1 The clinical and interventional course of the patient.

To facilitate CVVHDF, we decided to perform a zone 3 partial REBOA (pREBOA) to augment systolic blood pressure. A 6 Fr introducer sheath was placed into the right common femoral artery (CFA) with ultrasound (US) guidance followed by a 6 Fr Fogarty catheter. After confirmation that the tip of the catheter was at zone 3 by US, the balloon was fully inflated. Another 6 Fr introducer sheath was placed with the same method into the contralateral CFA to monitor blood pressure below the balloon so that pREBOA could be titrated to maintain a distal systolic blood pressure of 80 mmHg. With pREBOA, a blood pressure of 130/80 mmHg was achieved proximal to the balloon and the systolic blood pressure distal to the balloon was 45 mmHg. In the meantime, an extracorporeal membrane oxygenation (ECMO) protocol was initiated and 118 minutes after balloon inflation veno-arterial ECMO was commenced following the balloon deflation and withdrawal of the occlusion catheter. Systolic blood pressure distal to the balloon just before the deflation was 80 mmHg. Concomitantly to ECMO, inotrope support was weaned as tolerated until only a 26 mcg/min of norepinephrine infusion was required (Figure 1). Once a proximal mean arterial pressure greater than 90 mmHg was achieved, CVVHDF resumed with ethanol and bicarbonate infusion. Despite all efforts, lactate levels did not decrease less than 12.8 mmol/L and on the 9th hour of ECMO, the patient developed bradycardia and then cardiac arrest. After 50 minutes of CPR, the patient was declared dead.

DISCUSSION

Although REBOA is a promising new therapy for the management of non-compressible torso hemorrhage even in austere combat environments [6], we presume that it may be an evolving bridging therapy for management of various critically ill patients.

To the best of our knowledge, our case is the first example of REBOA in the literature utilized to facilitate CVVHDF in an intoxicated patient. In a recent case report, REBOA was unconventionally used to improve hemodynamic support in a patient with proximal aortic aneurysm rupture [7]. Similarly, we only aimed to slightly augment mean arterial pressure (MAP) without occluding the renal arteries, thus we preferred a zone 3 REBOA. We also did not want to bear the consequences of limb ischemia and reperfusion injury in an already critically ill patient with profound lactic acidosis. This was the main reason for us to perform a pREBOA rather than a complete one (cREBOA). Unfortunately, we were obliged to use a 6 Fr Fogarty balloon catheter with a 13 mm balloon diameter because we could not find a larger catheter at our institution. However, this lack of resources served our purposes well. We inflated the balloon with volume increments of 0.5 ml until we reached a volume of 2 ml which is the maximum balloon volume allowed and left it fully inflated via a stopcock. Thus, we aimed to protect the balloon from blowing.

Reva et al. demonstrated that with a 40–49% partial occlusion the aorta yields a significant increase in MAP in a bleeding sheep model [8]. As we experienced, placing a contralateral CFA sheath might be useful while monitoring the blood pressure distal to the balloon of the catheter. While providing a distal systolic blood pressure over 80 mmHg to avoid limb ischemia, it may also provide valuable information about the efficacy of the aortic occlusion. Along with the concept of Endovascular Perfusion Augmentation for Critical

Care (EPACC) by Williams et al. [9], the main reason for us to perform an aortic occlusion was to augment the blood flow proximal to the occlusion balloon, in particular, renal arteries at the cost of possible lower limb ischemia. However, this possibility constitutes the main reason for monitoring the blood pressure distal to the balloon to predict any potential lower limb hypoperfusion. In a brief report [10], DuBose recommended using an 8 Fr ER-REBOA catheter to measure the distal arterial pressure below the occlusion balloon and deflating the balloon gradually until the distal arterial waveform below the balloon was observed. However, with the lack of an 8 Fr catheter in our institution, we were not capable of performing a cREBOA. Therefore, there was not an interrupted but only slightly diminished blood flow distal to the balloon. In addition, we presume that using a smaller catheter to achieve pREBOA might be practical unlike DuBose's method. In this case, we tried to focus on the balance of the augmentation of the proximal blood pressure versus introducing enough blood flow below the balloon while avoiding limb ischemia.

REBOA may serve as a bridge to numerous life-saving interventions such as ECMO, selective aortic branch occlusion, coiling, and embolization. In our case, the catheter balloon was inflated in order to 'step up' the ECMO. pREBOA acted as the 'fourth inopressor' along with dopamine, norepinephrine, and epinephrine and provided time for ECMO setup and priming.

CONCLUSION

REBOA may be a useful tool in the management of critically ill patients with a variety of etiologies, even for intoxicated patients. In addition to serving as a bridge to advanced life-saving interventions, REBOA may act as a substitute inotrope when combinations of other inotropic agents fail to preserve the mean arterial pressure.

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