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Case Report

Pulse contour analysis guided management of a case of puerperal uterine inversion and hemorrhagic shock – ‘Giving what it takes’

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insight into the hemodynamics of such delicately poised patients.

Case report

A 22-year-old gravida two, para one with anemia and previous caesarean section, came in advanced labor and delivered vaginally. After an hour, excessive vaginal bleeding was noticed. Genital tract trauma was ruled out and resuscitation started with crystalloids and colloids. Uterine atony persisted inspite of carboprost, oxytocin infusion and uterine massage. She appeared pale, had supra-pubic tenderness and was in shock. The estimated blood loss was 1000 ml. In view of a history of a previous caesarian section, abdominal pain and features of hypovolemic shock, a uterine rupture was suspected and an emergency exploratory laparotomy was planned. Enroute to the operation theater she became drowsy, unresponsive and pulseless with an unrecordable blood pressure (NIBP). Resuscitation was continued with a rapid infusion of tetra starch and normal saline.

She was rushed to the operating room and standard monitoring was instituted. The heart rate (HR) was 150/min with a thready pulse. SpO₂ and NIBP were not recordable and the patient was very drowsy though responsive now. The trachea was intubated following a rapid sequence induction

Introduction

Acute uterine inversion is difficult to diagnose and is associated with significant blood loss and shock out of proportion to the blood loss. Pulse contour analysis guided (Flotrac™/Vigileo™-system Edwards Lifesciences, Irvine, CA, USA) monitoring is a new tool which provides a precise and invaluable

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Table 1 – Hemodynamic parameters, blood gas analysis, hemoglobin levels and requirement of vasoactive agents, fluids and blood products.

	Pre op	Intra op	ICU adm	After 6 h	After 12 h
BP mmHg	Unrecordable	55/32–98/40	80/40	130/70	112/68
HR beats/min	150	170–109	132	98	76
CVP cm of H ₂ O		10–12	18	10	10
CO l/min			5.0	3.0	4.0
SVV%			28	<15	<15
SVR dynes/sec/cm ⁻⁵			560	2133	1052
Arterial blood gases					
pH		6.878	7.12	7.35	7.49
PaO ₂ mmHg		433.3	206	118	139.2
PaCO ₂ mmHg		47.3	30	30.9	27.7
HCO ₃ mmol/l		7.8	11	18.3	23.5
Vasoactive agents					
Dopamine mcg/kg/min		10.5–15	15	Tapered off	
Noradrenaline mcg/kg/min		0.2–0.6	1.0	0.1	Tapering
Vasopressin units/hr		0.02	0.04	0.02	Tapering
Adrenaline mcg/kg/min		0.1	1	tapered off	
Dobutamine mcg/kg/min				3	3
Fluids	2.5 L crystalloids 1.5 L colloids	0.5 L crystalloids			
Blood components		2 PRBC	4 PRBC 6 RDP 6 FFP 6 Cryo		
Hb g%	8	6.1		9	10
Hct	19	18.3		27	30

with ketamine and succinylcholine. Anesthesia was maintained with an infusion of ketamine. The radial artery was cannulated for invasive blood pressure monitoring. The central line introduced in the left internal jugular vein revealed a watery aspirate. The hemodynamic parameters and relevant patient details are tabulated as shown in Table 1.

She remained in shock and therefore, dopamine and noradrenaline were started. The arterial blood gas (ABG) analysis revealed metabolic and respiratory acidosis. 50 ml of sodium bicarbonate was injected over 10 min and the minute ventilation increased. Intraoperative echocardiography showed global hypokinesia. A uterine inversion was detected at laparotomy with buckling of uterine fundus (Fig. 1), and was corrected by gentle traction. The arterial blood pressure trace became flat, HR dipped to 46/min and pulseless electrical activity (PEA) was noted. CPR was therefore started. Atropine 0.6 mg and adrenaline 1 mg were administered. Dopamine and noradrenaline infusions were stepped up. Vasopressin 0.02 U/min and adrenaline 0.1 mcg/kg/min were started and sodium bicarbonate 100 ml repeated, followed by an infusion at 20 ml/h 500 ml of crystalloids and 2 units of packed red blood cells (RBCs) were transfused intra-operatively. With the above measures, the hemodynamics improved (Table 1). However, the uterus remained atonic and an obstetric hysterectomy was done.

In view of transient PEA, deliberate hypothermia was instituted in the ICU for cerebral protection and the core temperature was maintained at 32–34 °C. Propofol was started at 120 mg/h for sedation and cerebral protection. A repeat ABG showed improvement in the acidosis. Hct had increased to 20.5% and International normalized ratio (INR) was 1.9. Since the patients hemodynamics were unstable, requiring

high doses of vasoactive agents, cardiac output monitoring in the form of Flotrac™ was instituted to optimize the hemodynamics. Cardiac output (CO) was 2.4 L/min, stroke volume variation (SVV) was 28% though the central venous pressure (CVP) at this stage was 12 cm H₂O, systemic vascular resistance (SVR) was 560 dynes/s/cm⁻⁵ and her blood pressure (BP) was 80/40 mmHg. Therefore, the rate of vasopressors was stepped up further. Additional blood components to a total 2880 ml were transfused over the next 6 h. The hourly urine output was adequate. Sodium bicarbonate was stopped once the pH was 7.2. The SVR had now increased to 2133 dynes/s/cm⁻⁵ and the BP improved to 130/70 mmHg. Noradrenaline and vasopressin were tapered, dopamine &

Fig. 1 – Uterine inversion at laparotomy.

adrenaline were stopped, and dobutamine was now started. The patients' hemodynamics stabilized over the next 6 h.

By the next day, the Hb had increased to 9 g %. Liver enzymes were found to be elevated with serum bilirubin of 3.1 mg/dl bedside echocardiography showed a normal left ventricular function. She was extubated on the 2nd post-operative day after a weaning trial and discharged from hospital on the 6th post-operative day.

Discussion

The incidence of uterine inversion averages around 1 in 3000 amongst 116,500 deliveries.¹ The most common clinical presentation is pain, vaginal bleeding and shock.² In the past, the understanding was that shock was disproportionate to blood loss, possibly mediated by parasympathetic stimulation caused by stretching of tissues. The need in many cases for transfusion of large volumes of blood, however, illustrates that blood loss is usually massive and greatly underestimated.^{1–6} Our patient too needed to be transfused a total of 2880 ml of blood components for stabilization.

In a robust review including 24 studies involving 830 patients, the authors have concluded that there is no association between CVP and the circulating blood volume, and that CVP does not predict fluid responsiveness across a wide spectrum of clinical conditions.⁷

Numerous clinical and experimental studies have shown the usefulness of dynamic indices based on heart–lung interactions for guiding volume resuscitation.^{8,9} Amongst the less invasive advanced hemodynamic monitors, Flotrac™ is an arterial waveform analysis that doesn't require external calibration and is relatively independent of the vascular tone.¹⁰ It detects each waveform via an arterial catheter which is then analyzed at a frequency of 100 Hz. From the given stroke volume and heart rate, the cardiac output is determined, and updated every 20 s. A filter in the computer adjusts for excesses in systolic blood pressures and heart rates. Software updates have improved the accuracy of the monitor.¹¹ Many studies have demonstrated that SVV measured using pulse contour analysis is highly accurate in predicting responders to fluid expansion.⁸ Moreover, SVV is the physiologic variable that needs to be measured when assessing cardiopulmonary interaction.¹² The limitations of pulse contour analysis are its inaccuracy during arrhythmias, aortic valve regurgitation, intra-aortic balloon pumping, peripheral vascular disease and inability to provide information in a patient with an open chest.

In our patient the initial CVP was high inspite of the requirement of a further transfusion of blood products as evidenced by a high SVV though the SVR was very low (Table 1). She remained in shock intra-operatively in spite of a normal CVP. However, two factors which were causing shock at that stage were metabolic acidosis as a result of poor tissue perfusion and probably peripheral vasodilatation following uterine inversion and excessive fluid and colloid resuscitation. The patient was therefore, managed with vasopressors to improve tissue perfusion and sodium bicarbonate to normalize the pH, thus allowing for better cardiac contractility.

In the ICU, the SVV was found to be high, in spite of a normal CVP and the SVR was very low. Thus blood component therapy was continued under SVV guidance also vasopressors were added and had to be used at doses much higher than routinely used "giving what it took" to bring up the perfusion pressures at all costs. This resulted in an improvement in the peripheral perfusion as also the cardiac output leading to a rapid correction of the metabolic acidosis. As the patient improved, the peripheral vasculature became more responsive to the vasopressors which could be rapidly tapered off once the SVR increased.

Thus the hemodynamic variables derived from the Flotrac provided rapidly changing real time information about the actual interplay of preload contractility and afterload in our patient allowing timely appropriate intervention and a favorable outcome.

Conflicts of interest

All authors have none to declare.

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