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CASE REPORT **ANESTHESIA FOR TRAUMA**

Anesthetic management of a patient with hollow viscus perforation due to blunt abdominal trauma with grade IV hemorrhagic shock

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Abstract

Hemorrhagic shock is a form of hypovolemic shock in which severe blood loss leads to inadequate oxygen delivery at the cellular level. Death from hemorrhage represents a substantial global problem, with more than 60,000 deaths per year in the United States and an estimated 1.9 million deaths per year worldwide, 1.5 million of which result from physical trauma. This case report aims to stress the need of handling cases of hemorrhagic shock in accordance with damage control protocol.

Hemorrhagic shock management using permissive hypotension management, bleeding control, massive transfusion protocol (MTP), minimal crystalloid therapy, and adjuvant therapy is the best approach to get optimal outcome to prevent triad of death. In this case, the application of damage control resuscitation has not been fully implemented because of several constraints.

Key words: Hemorrhage; Hemorrhagic shock; Permissive hypotension; Massive Transfusion Protocol; MTP; Resuscitation; Damage control

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1. Introduction

Shock is a condition of acute circulatory failure which results in impaired organ perfusion due to ineffective oxygen supply to tissues resulting in organ dysfunction. In hypovolemic shock, a decrease in cardiac output, right and left ventricular filling pressures (preload) and an increase in afterload (systemic vascular resistance) can be found through compensated vasoconstriction. Inadequate delivery of oxygen to peripheral tissues results in a change in metabolism from aerobic to anaerobic. Anaerobic metabolism in turn produces lactate, which is a marker of hypoxemia and the severity of shock. However, persistence of cellular hypoxia in the long term can result in irreversible organ dysfunction.[1]

Hemorrhagic shock is a form of hypovolemic shock due to massive blood loss that causes inadequate oxygen delivery at the cellular level. If the bleeding continues, death will occur immediately. Death from hemorrhage is a global problem with more than 60,000 deaths per year in the United States and an estimated 1.9 million deaths per year worldwide. Furthermore,

those who survived hemorrhagic shock had a poor outcome and significantly increased long-term mortality. The damage control protocol is a protocol used in dealing with the incidence of hemorrhagic shock due to trauma with the aim of minimizing iatrogenic resuscitation injuries, preventing progression of the initial traumatic shock, and obtaining definitive hemostatic management.[2]

This case report discusses anesthetic management for preoperative, intraoperative and postoperative phase in a patient with hollow viscus perforation due to blunt abdominal trauma with grade IV hemorrhagic shock, who underwent emergency exploratory laparotomy with general anesthesia. It is interesting to discuss whether the preoperative, intraoperative, and postoperative treatments that were carried out including fluid resuscitation and pharmacological therapy for the management of hemorrhagic shock in this case were in accordance with the damage control protocol.

2. Case report

We report the anesthesia management in a patient with hollow viscus perforation due to blunt abdominal trauma with grade IV hemorrhagic shock. Written informed consent was obtained from the next of kin of the patient. A 22 years-old male with 60 kg body weight and 160 cm of height was scheduled for emergency exploratory laparotomy. The patient was brought to hospital emergency after 3 h of a traffic accident. We found injuries on the patient's left chest and left lower abdomen. No open wounds were found. There was no history of previous surgery and anesthesia, no history of allergy, and no history of comorbidities. Recent eating or drinking history was unknown.

In the primary survey, an examination of airway showed a clear airway, good airway patency, a nasogastric tube in situ with production of 50 ml dark colored fluid. The patient was breathing spontaneously oxygen 8 L/min with a non-rebreather mask, at a respiratory rate 45/min. Rhonchi and wheezing were absent, and oxygen saturation was not measured. On circulatory examination, blood pressure was not measurable, radial pulse was not palpable, extremities were cold, S1-S2 heart sounds were regular, murmur was absent. An intravenous cannula 20 G with a 3-way was attached to left hand and a cannula 18 G was attached to his right foot. In the disability examination, the patient's awareness was in sopor with GCS E1M4V1. The patient was found to be in hypoglycemia (blood sugar 35 mg/dL). In the primary survey, the patient was found in a grade IV hemorrhagic shock with suspected intra-abdominal bleeding with hypoglycemia. The initial management that had been done was a loading of 2 L of ringer's lactate and 1 L colloid, and infusion of 2 bags of D40% for the treatment of hypoglycemia. Packed red cells (PRC) and fresh frozen plasma (FFP) were ordered. Ringer's lactate was given 120 ml/h, then a Foley catheter was inserted. The initial urine output was zero. The patient was then intubated with 2 mg of midazolam.

In the post-resuscitation assessment, it was found that the patient's awareness increased with GCS E3M5Vt, blood pressure 90/60 mmHg, pulse still at 136 beats/min, respiration was 24/min, oxygen saturation 95% on 10 L/min oxygen using Jackson–Reese circuit.

On secondary survey, conjunctiva pale, sclera icteric, a lesion was found on the head, pupils were round and isochore, rhinorrhea (+), a tear in the dental mucosa was found, fracture in the left mandibular parasymphysis was found, and NGT was attached with a bag filled with black discharge. There was no injury on neck. Trachea was not deviated. There was a lesion on the left chest. Chest walls movements were symmetrical, emphysema subcutis was found in the left hemithorax. Breath sounds were symmetrical with no rhonchi and wheezing, with no intercostal retraction. Heart sounds were within normal limits. Laboratory investigations showed the following results (Table 1).

On chest x-ray, there was an impression of left lung contusions, subcutis emphysema, but no fracture of ribs and scapulae and no cardiomegaly. Abdominal CT scan exhibited hepatorenal and splenorenal fluid collections.

From the results of the history, physical examination, laboratory and imaging results, the patient was diagnosed with grade IV hemorrhagic shock with diffuse peritonitis due to hollow viscus perforation due to blunt abdominal trauma with ASA IV–E. After resuscitation, the patient was then transferred to the operating room for surgical management for bleeding control.

Table 1: Preoperative laboratory results

Parameter	Value	Parameter	Value	Parameter	Value
Hb	6.9	PT	16.2	pH	7.205
Ht	20.4	APTT	33.4	pCO_2	30.7
Leukocyte	3.060	INR	1.5	pO_2	297
Thrombocyte	102.000	Ur	59	HCO_3	12.2
GDS	129	Cr	2.91	BE	-13.7
Lactate	3.6	Alb	2.7	Saturation	98.9
Ca	3.93	Mg	1.7		
Post resuscitation Lactate: 3.0					

Table 2: Postoperative laboratory results

Parameter	Value	Parameter	Value
Hb	10,3	pH	7,265
Ht	31,4	pCO_2	24
Leukocyte	1,230	pO_2	43
Thrombocyte	33.000	HCO_3	11
PT	16,7	BE	***
APTT	41,2	Saturation	67,4
INR	1,55		
Lactate	3,0		

At induction time, findings were; GCS E3M5Vt, blood pressure 112/74 mmHg, pulse rate 122 beats/min, respiratory rate 24 /min, SpO_2 98% on 10 L/min of O_2. An ETT No. 7.5 was directly connected to the anesthesia machine. Induction was performed with 3 mg of midazolam, 25 µg of fentanyl, and 50 mg of rocuronium. Anesthesia was maintained with sevoflurane 0.5-1%, O_2: air in 60:40 ratio. Intraoperative systolic blood pressure ranged from 98 to 121 mmHg, diastolic blood pressure ranged from 61 to 72 mmHg, heart rate ranged from 108 to 122 beats/min, and SpO_2 99%. Intraoperative bleeding was 1250 ml with urine output of 150 ml/h. During surgery, 3 L of crystalloid fluid and 2 L of *Gelofusin* were infused. The operation lasted for 3 h.

Postoperatively, the patient was admitted to the Intensive Care Unit to monitor the vital signs. Postoperative lab results were obtained (Table 2).

3. Discussion

Perioperative evaluation showed that the patient had decreased level of consciousness, tachycardia, hypotension, decreased oxygen saturation, and decreased urine production. These symptoms indicated a state of shock as a result of hemorrhage and hollow viscus perforation. Hemorrhagic shock is a systemic perfusion disorder resulting in widespread cellular hypoxia and vital organ dysfunction due to a mismatch between demand and supply of substrates at the cellular level.[1,2]

Signs and symptoms of shock vary depending on the severity of the shock itself. These symptoms can include systolic pressure less than 90 mmHg, increased heart rate due to sympathetic stimulation and increased level of epinephrine and norepinephrine, weak and rapid pulse due to decreased cardiac output, cold and pale skin, and increased skin moisture due to

Table 3: Classification of hemorrhagic shock

Parameter	Class 1	Class 2	Class 3	Class 4
Blood loss (ml)	> 750	> 750-1500	> 1500-2000	> 2000
Blood loss (% blood volume)	>15%	15-30%	30-40%	> 40%
Average heart rate	< 100	100-120	120-140	> 140
Blood pressure	Normal	Normal	Decreased	Decreased
Pulse pressure	Normal/ increased	Decreased	Decreased	Decreased
Respiratory rate	14-20	20-30	30-40	> 35
Urine output (ml/h)	>30	20-30	5-15	Negligible
Central nervous system	Slightly anxious	Mildly anxious	Anxious, confused	Confused, lethargic

vasoconstriction of blood vessels on skin and sweating, decreased consciousness due to decreased oxygen supply to the brain, decreased urine production due to increased aldosterone and anti-diuretic hormone (ADH), patients appear thirsty due to loss of extracellular fluid, and decreased blood pH due to increased production of lactic acid.[3]

In primary survey, our patient was found to be in a state of shock with a drop in blood pressure due to blood loss caused by trauma. As a form of compensation against blood loss and lack of oxygen supply, the body retains fluids so that the patient's urine production decreased and the patient's body released epinephrine and norepinephrine hormones, which resulted in an increased heart rate. Peripheral vasoconstriction caused the patient's body to feel cold. In this patient, despite various compensatory mechanisms, his body was unable to meet his own needs, as proven by the physical signs and symptoms and the laboratory results. In this patient, there was an increase in base excess which indicated that the patient was still in a hypovolemic state and increased lactate which contributed to a decreased cellular hypoxia. According to The American College of Surgeons, hemorrhagic shock is divided into 4 classes as shown in Table 3.[5]

In this patient, there were signs of grade 4 hemorrhagic shock, namely, blood pressure was not measurable at the time the patient arrived, the pulse was feeble, the respiratory rate was 45/min, and there was no urine output. The laboratory examination also revealed anemia with an Hb of 6.9, and the patient was in a state of uncompensated metabolic acidosis with a pH of 7.2 and a base excess of −13.7. Therefore, when the patient came to the resuscitation room, we performed the management of shock by administering 2 L of crystalloid fluid and 1 L of colloid fluid for approximately one hour. In principle, recognizing shock as early as possible and managing it as soon as possible to stop bleeding is the key to save the life of a patient with hemorrhagic shock. Therefore, immediately controlling the source of bleeding and restoring the patient's intravascular volume, may prevent the worsening of shock to irreversible shock.[2] In addition to administering fluids, we also collaborated with surgeons to urgently control bleeding through surgical approach.

After fluid resuscitation, the patient's hemodynamics became stable with blood pressure 90/60 mmHg, pulse rate 136 beats/min and respiratory rate 24 breaths/min with 95% saturation on 10 L/min O_2, and the patient's consciousness began to increase with GCS E3M5Vt. The initial resuscitation had a good impact on the patient. Adequate volume of fluids and good oxygenation may improve hemodynamic disorders and replenish the patient's oxygen needs.

Successful resuscitation requires aggressive measures to prevent further accumulation of oxygen deficiency and to replenish oxygen demand by stopping all sources of bleeding and restoring intravascular volume as quickly as possible. After major bleeding is identified, it is advisable to apply damage control management with permissive hypotension, control the source of bleeding, administration of minimal

crystalloid fluids, administration of plasma and other blood products in an equal ratio (preferably 1: 1: 1), and administration of hemostatic drugs.[4]

Intraoperatively, 1250 ml of bleeding was recorded, then 3 L of crystalloid fluid, 2 L of *gelofusin*, 4 bags of PRC and 4 bags of FFP were infused. Postoperatively, the Hb was 10.3 gm and hematocrit 31.4. However, thrombocytopenia and prolonged coagulation and risk of DIC still persisted. The lab also showed uncompensated metabolic acidosis. The conditions mentioned above were parameters that still needed to be treated, therefore the patient was treated in the Intensive Care Unit (ICU) postoperatively for further mangement.

When referring to the concept of damage control, the application of damage control management had not been fully applied in this case, namely the administration of crystalloid and colloid fluid in this case which should have been limited to 1-2 L before surgery with a target systolic blood pressure of 80-90 mmHg. The availability of blood products were still constrained by the preparation time. Bleeding control through surgery also required a relatively long time before the surgery was performed. Therefore, adequate knowledge regarding the management of hemorrhagic shock still needs to be improved, and fast and good coordination regarding the availability of blood and coordination with surgical team regarding surgical management must be rapid.

4. Conclusions

Successful resuscitation requires aggressive measures to prevent worsening oxygen deficiency, and to replenish oxygen demand by stopping all sources of bleeding and restoring intravascular volume as quickly as possible. Permissive hypotension, administration of crystalloids, and activation of massive transfusion protocols are the allowable approaches to treat hemorrhagic shock and prevent the occurrence of the triad of death.

Adequate liaison and good multidisciplinary collaboration is needed so that the management of hemorrhagic shock is not delayed.

5. Conflict of interest

None declared by the authors.

6. Authors' Contribution

DKP: Concept, organize the study, review of the manuscript

MBK: Literature review, evaluation of findings, manuscript writing

RWS: Logistic support to conduct the study

HNR: Responsible for biological materials and referred patients, follow–up, collection of relevant biological materials, regulation and reporting of data

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