Management of Decreased Consciousness Caused by Hypovolemic Shock: Postpartum Hemorrhage in Critical Care Setting

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1. Introduction

Postpartum hemorrhage life-threatening condition, with a prevalence of approximately 1%-10% of all deliveries. Postpartum hemorrhage is one of the leading causes of maternal death.1 Postpartum hemorrhage is a blood loss of more than 500ml or after vaginal birth and 1000 ml in cesarean delivery.2,3 According to the American College of Obstetricians and Gynaecolgists, PPH is defined as a cumulative blood loss greater than or equal to 1000 mL or blood loss accompanied by signs or symptoms of hypovolaemia within 24 hours after the birth process (includes intrapartum loss) regardless of route of delivery.4 Hypovolemic shock due to bleeding requires a well-integrated health management system. It requires a wide range of resources, such as advanced surgical and radiology capabilities, laboratory services, high-dependency nursing care, and blood transfusion.5

According to the World Health Organization in 2014, maternal deaths in the world are caused by bleeding 30.3%, labor complications 15.3%, infection 16.5%, unsafe abortion 10.8%, high blood pressure during pregnancy 27.1%. Maternal mortality in Indonesia still very high according to the Indonesian Demographic and Health Survey maternal. Within the literature, postpartum clinical conditions are mentioned that are classified as “the 4Ts”: tone, trauma, tissue and thrombin that can trigger complications in this stage. Tone alterations develop more frequently (70%), and uterine atony is characteristic, presenting in 60%-85% of women, followed by uterine hypotony (82%).6

2. Case Presentation

A 33-year-old female patient came to the emergency department with decreased consciousness 1 hour before hospital admission, with postpartum hemorrhage, placental abruption following cesarean section, and uterine atonia following B-Linch. The patient underwent a B-Linch operation after undergoing a cesarean section at a previous Hospital due to antepartum hemorrhage and placental abruption. The patient also experienced ongoing bleeding during the treatment due to uterine atonia. Following surgery, a post-operative laboratory evaluation revealed significant thrombocytopenia (platelets: 45,000/mm3) and anemia (Hb: 4.6 g/dl).

Conclusion: The management of postpartum hemorrhage, especially with shock, requires comprehensive critical care management. Appropriate management with good multidisciplinary team involvement will improve patient outcomes and prevent multi-organ failure.
undergoing a cesarean section at a previous Hospital due to antepartum hemorrhage and placental abruption. The patient also experienced ongoing bleeding during the treatment due to uterine atonia. Following surgery, a post-operative laboratory evaluation revealed significant thrombocytopenia (platelets: 45,000/mm$^3$) and anemia (Hb: 4.6 g/dl). Due to ICU limitations in the previous Hospital, the patient was referred to our hospital. Before transferring the patient, the patient received two units of Whole blood transfusion, stabilization, and transfer.

When admitted to our resuscitation room, he was found to have soporous consciousness with a GCS value of E2M4V1, with a blood pressure of 72/42 with a mean arterial pressure of 52, a heart rate of 148, and oxygen saturation in the emergency department was 89%. The patient was given fluid resuscitation and a blood transfuse immediately. While in the emergency room, the patient was also given oxytocin and a methergine drip. Immediately, the patient was intubated, and then the patient was transferred to the ICU for intensive monitoring.

From the results of the labor examination in the ICU, the Hb value was 6.0, platelets 83,000, Albumin 1.5. The patient was given a blood transfusion of whole blood 2 units, PRC 3 units, platelet apheresis 1 unit, fresh frozen plasma 4 units, and 25% albumin transfuse. In addition to fluid and blood resuscitation, the patient was also given inotropic and vasopressor to achieve the target mean arterial pressure.

This patient was also accompanied by impaired renal function, ureum 47, and creatinine 2.1, accompanied by severe metabolic acidosis, with pH 7.03, pCo2 34, pO2 185.6, BE -19.7, HCO$_3$-9 and SaO$_2$ 98.9%, the patient was given meylon correction. The coagulopathy examination also showed INR 2.16 and D dimer >10,000. Blood pressure in the ICU after blood resuscitation was obtained. Hemodynamic blood pressure was 90/57 mmHg, with a heart rate of 155x/min.

Abdominal examination revealed fundus uteri 2 fingers below umbilical, contraction was good, and there was no vaginal bleeding. From the ultrasound examination, no abnormalities were found in the uterus, and there was no free fluid in the abdomen. The patient had a body mass index of 27.05 and was categorized as obese.

On the second day in the ICU, the Hb was found to be 8.6, a transfusion of 2 units of PRC was planned, the platelet value was 30,000, 1 unit of apheresis platelet transfusion was carried out, the patient was carried out periodic hematological examinations every 6 hours, ureum value 66, creatinine 3.0. The patient was then referred to the renal hypertension department to be planned for hemodialysis. In this patient, hemodialysis was performed 3 times a week.

The patient also had seizures; seizures occurred throughout the body with a tonic-clonic pattern, with seizure duration <1 minute. The patient was also referred to the neurology, pulmonology, and cardiology departments. From the neurology department, the patient received levetiracetam 2 x 2000 mg and clobazam 2 x 10 mg.

At the beginning of admission to the ICU, the patient’s procalcitonin value was 3.05, and leukocytes 23.920. However, on day three, the procalcitonin value rose to 161.39; the patient received antibiotic therapy cefepime 3 x 1 gram, moxifloxacinc 3x 400 mg, tranexamic acid 3 x 1 gram, vit k 3 x 10 mg, paracetamol 3 x 1 gram, omeprazole 2 x 40 mg.

On the seventh day of treatment in the ICU, the patient was planned for tracheostomy due to prolonged intubation. The patient’s level of consciousness still had no improvement on day seventh, and blood pressure was also still unstable with the support of norepinephrine, dobutamine, and vasopressin.

### 3. Discussion

A 33-year-old female patient came to the emergency department with decreased consciousness 1 hour before hospital admission, with postpartum hemorrhage, placental abruption following cesarean section, and uterine atonia following B-Linch. In postpartum hemorrhage, the goal is to find the cause of the bleeding. The amount of blood lost during labor
and delivery should be monitored closely. The most common causes of postpartum hemorrhage are the 4 T’s: Trauma (birth canal), Tone (decreased uterine muscle tone/atony), Tissue (placental remnants or pieces of placenta), and Thrombin (coagulopathy).

The patient was also given oxytocin and a methergine drip. Oxytocin is the first-line therapy for atony uteri. This drug stimulates rhythmic uterine contractions, especially in the upper segment. ergometrin can also be given. ergometrin works by increasing sustained myometrial contractions. ergometrin also works on vascular smooth muscle.

The management of postpartum hemorrhage, especially with shock, requires comprehensive critical care management. Appropriate management with good multidisciplinary team involvement will improve patient outcomes and prevent multi-organ failure. The majority of obstetric patients requiring treatment in the ICU are postpartum hemorrhage with complications such as shock and sepsis, the management of which requires a multidisciplinary team including obstetricians, intensive care physicians, anesthesiologists, hematologists, blood bank technicians, nurses, and other related health workers.

Systemic resuscitation is performed during the management of postpartum hemorrhage causing hypovolemic shock, first, by securing airway patency through endotracheal intubation and preventing the risk of aspiration, especially with patients with decreased consciousness. Second, large-caliber venous accesses are required to administer resuscitation fluids, with a volume of 1-2 liters administered during the first hour, closely monitoring vital signs; the solutions used are crystalloid, colloid, and blood. Finally, vasopressor drugs such as Desmopressin 0.3µg/kg uterotonic drugs such as Oxytocin 30 IU/30 minutes are given, and if these drugs are ineffective, additional sodium bicarbonate to overcome lactic acidosis.

From the results of the labor examination in the ICU, the Hb value was 6.0, platelets 83,000, Albumin 1.5. The patient was given a blood transfusion of whole blood 2 units, PRC 3 units, platelet apheresis 1 unit, fresh frozen plasma 4 units, and 25% albumin transfuse.

Acute circulatory failure or shock is a condition in which tissue perfusion is inadequate. The main goal of management is to restore perfusion to the compromised area to provide adequate oxygenation to the tissues. Insufficient blood flow can be clinically identified as the development of one or more of the following clinical and laboratory assessments: altered mental status, lactic acidosis, oliguria, and tachycardia. Clinical assessment of the patient’s hemodynamic status is essential in critical management. Clinical parameters that can be seen include the level of consciousness (Glasgow coma scale), pulse rate, blood pressure, respiratory rate, capillary refill, and urine output.

Acute respiratory distress syndrome is one of the complications of massive bleeding, especially in the presence of shock, which may require ventilatory support. Respiratory management aims to maintain adequate gas exchange by ensuring a patent airway, appropriate and sufficient oxygen therapy, and continuous ventilation.

The fundamental purpose of intubation is to ensure airway patency and adequate ventilation and oxygenation. In patients with decreased consciousness [Glasgow coma scale (GCS) ≤ 8], bleeding shock, hypoventilation, or hypoxemia, intubation is one of the important management. However, other aspects should also be considered. For example, administering positive pressure may cause potentially life-threatening hypotension in hypovolemic patients.

This patient was also accompanied by impaired renal function, ureum 47, and creatinine 2.1, accompanied by severe metabolic acidosis, with pH 7.03, pCO2 34, pO2 185.6, BE -19.7, HCO3 -9 and SaO2 98.9%, the patient was given meylon correction. The coagulopathy examination also showed INR 2.16 and D dimer >10,000 DIC.

Acute renal failure is one of the major contributing factors to maternal mortality after massive postpartum hemorrhage. The primary management goal is to minimize damage to viable nephrons and reduce
further nephron damage. Improvement of circulatory function by fluid or blood, along with judicious use of vasopressors, is needed in the critical management of shock patients with renal impairment. Adequate blood pressure increases perfusion pressure and maintains adequate urine output (> 0.5-1 mL/kg/hour). Electrolyte disturbances, especially abnormalities in potassium values, must be corrected. Severe acidosis may require sodium bicarbonate therapy.

Replenishment of blood loss aims to prevent or attenuate the phenomenon of mandatory ischemia-reperfusion, which is proportional to the degree and length of the hypoxemia period. In addition to carrying oxygen, red blood cells also carry nutrients, inflammatory and immune cells, hormones, and clotting cells and factors. Replacing whole blood components is the ideal replacement fluid for massive blood loss. Especially whole blood that has been fresh for 24 hours or whole blood that is still in the bank. Alternatively, RBCs and plasma as a second choice and RBCs and lactated Ringer's as a third choice are commonly used.

In hemorrhagic shock, patients with low initial Hb levels are a predictive factor for massive transfusion. In addition to fluid and blood resuscitation, the patient was also given inotropic and vasopressor to achieve the target mean arterial pressure. If there is life-threatening hypotension, vasopressor administration, in addition to fluids, can be given to maintain the target mean arterial pressure. If there is myocardial dysfunction, inotropic agents can be given. Vasopressors may also be required temporarily, even when fluid expansion is ongoing and hypovolemia has not been corrected, to maintain tissue perfusion in the presence of life-threatening hypotension.

In bleeding situations, colloid infusion provides hemodynamic stabilization with less tissue edema when compared to crystalloids. Dilutional coagulopathy may be exacerbated by aggressive volume administration. Hence, limited volume replacement (1-2 mL of crystalloid for every 1 mL of blood loss) to avoid secondary hypervolemia and dilution of coagulation factors. However, maintaining normovolemia is essential to compensate for blood loss and stabilize hemodynamics in this patient population.

In non-major bleeding, the target for starting blood transfusion is a Hb level of <7 mg/dl, but in case of massive bleeding, an Hb level of 7-9 g dL may be targeted, depending on the patient’s condition and the clinical condition. Transfusion of fresh frozen plasma (FFP) (15-20 mL/kg) and platelet concentration (5-10 mL/kg). Physiologic acid-base status and calcium homeostasis should be monitored. In patients with massive bleeding, the patient’s temperature should also be monitored, as even mild hypothermia can increase blood loss. In case of massive bleeding, a ratio-based protocol is recommended as soon as possible, according to the European Society of Anesthesiology Guidelines, to treat acquired coagulation factor deficiency.

4. Conclusion
The management of postpartum hemorrhage, especially with shock, requires comprehensive critical care management. Appropriate management with good multidisciplinary team involvement will improve patient outcomes and prevent multi-organ failure.

5. References


