Critical Care for the Obstetrician and Gynecologist

Obstetric Hemorrhage and Disseminated Intravascular Coagulopathy

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KEYWORDS

- Hemorrhagic shock • Massive transfusion protocol
- Disseminated intravascular coagulopathy • Transexamic acid
- Special populations of hemorrhage

KEY POINTS

- Hemorrhage in pregnancy is common and a subset of patients succumb to hemorrhagic shock with disseminated intravascular coagulopathy.
- Massive transfusion protocol aids in complex resuscitations while avoiding acidosis and dilutional coagulopathy.
- Aside from uterotonics, there are other medications that can be used in correction of coagulopathy.

BACKGROUND

Obstetric hemorrhage accounts for 5% of all deliveries and is usually defined as greater than 500 mL and greater than 1000 mL of estimated blood loss following a vaginal delivery and cesarean section, respectively.[1] Risk factors include, but are not limited to, grand multiparity, prolonged induction, chorioamnionitis, multiple gestation, and abnormal placentation.[2]

Mostly hemorrhage is secondary to uterine atony, and this is usually resolved with medical therapies, such as oxytocin, carboprost, methergine, and misoprostol.[1,2] However, in complex hemorrhages, which can be secondary to a multitude of factors, the obstetrician must be astute in the management of hemorrhagic shock and its resultant coagulopathy and electrolyte disturbances. This article focuses on the definition, management, and therapies for such a complex hemorrhage.

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DEFINITION OF HEMORRHAGIC SHOCK AND DISSEMINATED INTRAVASCULAR COAGULOPATHY

Shock is state of hypoperfusion and anaerobic metabolism. In hemorrhagic, hypovolemic shock, it is further characterized into four classes (**Table 1**). Although intravascular volume increases up to 50% in the mid second trimester pregnancy, hemorrhagic classification does not change for pregnancy.[3]

As shown in **Table 1**, hemorrhagic shock is not only based on loss of blood volume, but also on clinical factors, such as mental status, heart rate, blood pressure, and urine output. In the setting of postpartum hemorrhage (PPH), which is often rapid, large-bore intravenous access should be confirmed and transition from crystalloid resuscitation to blood products should be made once the patient shows signs of class III shock.

Although defined in the nineteenth century and clinically relevant, the precise definitions of disseminated intravascular coagulation (DIC) have not been completely adopted and there have been no criteria that have shown improvement in clinical outcomes.[4–6] DIC affects 12.5 per 10,000 pregnancies and is reported to be the second most common severe maternal morbidity indicator for obstetric admissions.[7,8] The pathophysiology of clinical DIC is defined as a general inflammatory response with a release of cytokines, proteases, and hormones that leads to extensive microvascular endothelial damage.[5] This further causes vasodilation, capillary leak, and shock, which dysregulates the coagulation pathway leading to excessive thrombin generation and microthrombus formation.[5] This is clinically seen with exhaustion of both platelets and coagulation factors (ie, fibrinogen).[5] Clinically this is manifested as thrombosis of small arterial and venous vessels resulting in organ dysfunction and severe bleeding.

Outside of pregnancy, the three most common clinical conditions associated with DIC are sepsis, acute leukemia, and solid cancers.[4] However, pregnancy-related true DIC is secondary to placental abruption, preeclampsia, amniotic fluid embolism, and acute fatty liver of pregnancy (AFLP).[9] Acute hemorrhage is not as much intrinsic DIC as it is a dilutional coagulopathy. However, many times in acute hemorrhage and trauma there is a release of cytokines and proinflammatory agents that can trigger an intrinsic coagulopathy. Secondary to the lack of widely accepted guidelines, it is important for providers to treat DIC in the context of the underlying clinical disease

Table 1
Classes of hemorrhagic shock

	Class I	Class II	Class III	Class IV
Blood loss (mL)	Up to 750	750–1500	1500–2000	>2000
Blood loss (% of blood volume)	Up to 15%	15%–30%	30%–40%	>40%
Pulse rate	<100	100–120	120–140	>140
Blood pressure (mm Hg)	Normal	Normal	Decreased	Decreased
Pulse pressure (mm Hg)	Normal	Decreased	Decreased	Decreased
Respiratory rate	14–20	20–30	30–40	>35
Urine output (mL/h)	>30	20–30	5–15	Negligible
Mental status	Slightly anxious	Mildly anxious	Anxious, confused	Confused, Lethargic
Fluid replacement	Crystalloid	Crystalloid	Crystalloid + blood	Crystalloid + blood

From Advanced Trauma Life Support (ATLS) student course manual. 9th edition. Chicago (IL): American College of Surgeons; 2012.