OBG MANAGEMENT

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Managing postpartum hemorrhage: establish a cause

Although postpartum hemorrhage may be both sudden and massive, this condition has only 4 causes: uterine atony, genital-tract lacerations, retained placenta, and coagulopathy. Thus, the first step in management is determining the cause of bleeding.

While even optimal management may not prevent all maternal deaths, a reasoned and scientific approach to postpartum hemorrhage can dramatically improve maternal outcome.

Still, even with modern blood-banking techniques and effective medical and surgical approaches for combating bleeding, this common obstetric dilemma remains a significant cause of maternal morbidity and, occasionally, mortality—even in developed nations.[1]

Postpartum hemorrhage is not a medical condition in and of itself, but rather a clinical sign of other conditions that differ widely in both pathophysiology and treatment. For that reason, when excessive bleeding occurs, the clinician's first step should be to establish a cause. In most cases, the appropriate treatment then becomes apparent.

Defining hemorrhage

Traditionally, postpartum hemorrhage has been defined as blood loss exceeding 500 cc in a vaginal delivery.[1] This definition persists in a number of textbooks despite the fact that it has been well established that *mean* blood loss during vaginal delivery is 500 cc.[2] Thus, there is no uniformly accepted volume of bleeding that defines postpartum hemorrhage. In general, the diagnosis should be considered whenever experienced observers perceive blood loss to exceed the norm, or when otherwise unexplained drops in maternal blood pressure (BP) occur in the postpartum period.

Because many gravidas have BPs significantly lower than the general population (80/50 mm Hg is by no means unusual in a

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KEY POINTS

- The most widely used agent for both prevention and management of postpartum hemorrhage is misoprostol.
- The primary contraindications to 15 methyl prostaglandin $F_{2\alpha}$ are asthma and cyanotic cardiac disease.
- In most cases of failed medical management of uterine atony, hysterectomy is necessary.
- Although routine inspection of the placenta for completeness is essential following every delivery, portions may remain behind even when the delivered placenta appears to be complete.

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TABLE 1

Causes of postpartum hemorrhage

- Uterine atony
- Genital-tract laceration
- Retained placenta
- Coagulopathy

pregnant woman at term), the traditional definition of hypotension—a systolic BP below 90 mm Hg—is not always applicable in pregnancy. Because epidural anesthesia also may cause hypotension, it sometimes is difficult to determine whether the hypotension is a result of hemorrhage. Clinicians therefore may need to compare a gravida's pre- and intrapartum BP measurements and consider maternal pulse to determine the significance of "low" readings obtained during labor and delivery.

While sudden massive hemorrhage can be associated with maternal bradycardia, most young, healthy patients tend to respond to hemorrhage with progressive tachycardia—another meaningful sign of developing hypovolemia.

All told, however, there are only 4 causes of postpartum hemorrhage (TABLE 1).

Uterine atony

The most common cause of excessive postpartum bleeding is uterine atony, which is linked to several predisposing factors (TABLE 2). While a knowledge of these factors may help the clinician identify patients at increased risk, many women—even some with severe atony—have no risk factors at all. In 1 review of women with atony that was unresponsive to medical therapy and required hysterectomy, 20% lacked any identifiable risk factors.[3] Thus, the obstetrician needs a clear understanding of the management of uterine atony and should be prepared to quickly initiate a sequence of well-defined steps aimed at its elimination. While there likely are a number of ways to appropriately address this condition, I tend to take the following approach.

Uterine compression. By manually manipulating the uterus in a serious case of uterine atony, we aim to mimic the firm, steady contraction seen in the normal postpartum uterus. After all, direct pressure is an effective means of stopping most bleeding—at least temporarily. The uterus is no exception. Note that this is a separate entity from fundal massage, which can help a clinician express postpartum clots.

For serious uterine atony, place 1 hand in the vagina toward the posterior fornix and the other on the patient's abdomen, trapping the uterus between the 2. Apply firm pressure. As long as this pressure continues, bleeding will be significantly reduced. In some instances, this pressure is all that is needed to stop the bleeding, as the uterus eventually responds and contracts on its own. In other cases, manual compression may simply buy the clinician some time to obtain additional intravenous (IV) access, administer pharmacologic agents, arrange for blood transfusion, or summon assistance.

Oxytocin infusion. This step generally is carried out at the same time as uterine compression. Resist the temptation to administer oxytocin as an IV bolus; this can lead to paradoxical hypotension in a small percentage of women. Instead, infuse a dilute solution (20 to 40 U per liter).

Medical treatment. The combination of simple compression and oxytocin infusion resolves most cases of uterine atony. When these measures are insufficient, additional pharmacologic therapy is necessary. While each of the following agents is often effective in combating atony, there is no established

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order for their administration. In some instances, maternal medical conditions may contraindicate some medications. In others, the clinician's personal preference and experience will guide management. It usually is possible to go through a series of these additional medications within 15 to 20 minutes while maintaining fundal compression to decrease the bleeding.

15 methyl prostaglandin $F_{2\alpha}$ (carboprost tromethanine). Of all the prostaglandins used in the United States today, this 1 has the longest track record in managing uterine atony. Administer it into a peripheral muscle or directly into the uterus itself. In general, I prefer the first approach. If the patient is hypotensive, however, peripheral muscular perfusion will be diminished, and direct injection into the myometrium may be more effective.

One vial of 15 methyl prostaglandin $F_{2\alpha}$ ($PGF_{2\alpha}$) contains 250 μg. The medical literature suggests the use of 2 to 6 vials before considering the intervention a therapeutic failure.[1] My own approach, generally, is to inject 2 vials initially in cases of serious uterine atony, then move on to other drugs if this proves ineffective. In most cases, however, it is entirely appropriate to repeat this 500-μg dose once or twice. (There is no evidence supporting the routine initial administration of 500 μg as opposed to 250 μg.) Patients generally respond within 5 minutes.

The primary contraindications to 15 methyl $PGF_{2\alpha}$ are asthma and cyanotic cardiac disease. Since this drug is a general bronchoconstrictor, clinically insignificant desaturation occurs in all women. Because asthmatic women are more sensitive to these bronchoconstrictive effects, the use of this agent in an asthmatic patient may have disastrous results. For the same reason, a woman with cyanotic heart disease—who may be only marginally oxygenated—might be unable to tolerate a degree of oxygen desaturation that would be insignificant in most patients.

Methylergonovine maleate. This drug has the longest history of use in the management of uterine atony. It is a potent smooth muscle constrictor, affecting not only smooth muscle of the uterus but vascular smooth muscle as well. In the vast majority of cases, the drug, dosed at 0.2 mg, should be given intramuscularly (IM). As with 15 methyl $PGF_{2\alpha}$, the patient generally responds within 5 minutes. Most physicians give 1 to 2 doses of methylergonovine at 5-minute intervals before abandoning the therapy. However, if the atony is slow, chronic, or intermittent, additional doses at longer intervals may be appropriate, as may oral therapy.

Because it may lead to hypertensive crisis and cerebral vascular accident, the drug is contraindicated in hypertensive patients.

Misoprostol. The most widely utilized agent in the world for both prevention and management of postpartum hemorrhage is misoprostol. It was not commonly used in the

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TABLE 2

Uterine atony: predisposing factors

- Uterine overdistension (twins or macrosomia)
- Prolonged labor
- Oxytocin augmentation
- Chorioamnionitis
- Magnesium sulfate infusion
- Precipitous labor

United States until recently, but is increasingly valued for its effects against postpartum atony. The drug comes in 100-μg pills. Although very small doses (25 μg) are used in labor induction, much larger quantities are needed for postpartum atony—generally in the range of 600 to 800 μg. Misoprostol is readily absorbed through any mucous membrane and may be given rectally, buccally, or orally, as the situation demands. (Avoid the vaginal route in patients with hemorrhage because the pills will wash out.)

While neither the obstetrician nor the patient wants to go from a normal spontaneous vaginal delivery to **peripartum hysterectomy**, such **lifesaving decisions** must occasionally be made.

Misoprostol generally takes effect within 10 to 15 minutes. There are no known contraindications to its use. In such high doses, however, violent shivering is a relatively frequent occurrence. This may be frightening for both patient and physician, but is transient and requires no treatment.

Surgery. When pharmacologic therapy fails, laparotomy is indicated. A number of surgical approaches to uterine atony have been described.[3-5] Although some clinicians advocate uterine and hypogastric artery ligation, most series touting the effectiveness of these techniques do not include patients for whom medical management has failed.[6] In my experience, these women are extremely unlikely to respond to either uterine or hypogastric artery ligation. Indeed, any technique that causes further ischemia of an organ would seem to stand little chance of improving the function of that organ and increasing contraction.

In most instances of failed medical management, hysterectomy is necessary. While neither the obstetrician nor the patient wants to go from a normal spontaneous vaginal delivery to peripartum hysterectomy, such lifesaving decisions must occasionally be made. Death due to uterine atony alone is almost always preventable.

Lacerations

Genital-tract lacerations are the second most common cause of postpartum bleeding. When the palpated uterus is found to be firm in a hemorrhaging patient, a thorough search for lacerations is indicated. Begin this process with careful inspection of the vagina and cervix. Adequate anesthesia often is essential for such exploration. In some instances, it may be necessary to move the patient to the operating room (OR), as surgical assistance may be needed to obtain appropriate exposure.

Lacerations should be surgically repaired. Unless the patient has undergone a vaginal birth after cesarean (VBAC) or the bleeding is observed to originate in the uterus, it is not necessary initially to inspect the uterine cavity manually.

Retained placenta

After delivery, inspection of the placenta helps to determine if all cotyledons have been expelled intact. Be aware, however, that portions may remain behind even when the delivered placenta appears to be complete. When the uterus is firm and careful inspection of the lower genital tract reveals no bleeding sites, uterine exploration is necessary.

Manually explore the uterine cavity and remove any retained placenta that is encountered. Adequate anesthesia—even, at times, general anesthesia—is crucial under these circumstances. In some cases, postpartum curettage performed with a specially designed curet may help. Complete removal of all portions of the placenta generally leads to the prompt cessation of bleeding.

When the preceding methods fail to resolve bleeding due to retained placenta,

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prompt exploratory laparotomy is mandatory. On occasion, broad-ligament hematomas may be detected, the laceration corrected, and hysterectomy avoided. In other instances, hysterectomy is performed as a lifesaving measure without exact knowledge of the source of the bleeding. This is often followed by the pathologic diagnosis of placenta accreta.

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Other surgical approaches. Ligation of the uterine or internal iliac arteries is more likely to be effective in cases of uterine rupture. The classic approach to treating placenta accreta is hysterectomy. While a theoretical benefit exists for hypogastric artery ligation in some cases, the rarity of indications for this procedure means that very few recently trained obstetricians will be proficient enough to perform it under emergent conditions. A lack of such knowledge does not indicate inadequate training or technique. Rather, internal artery ligation has limited clinical value and is never required by the standard of care. With an experienced operator, however, the technique may occasionally make uterine conservation possible.

Coagulopathy

There are only 2 causes of acute consumptive coagulopathy in obstetrics: massive placental abruption and amniotic fluid embolism. Most chronic coagulation defects are uncovered long before pregnancy, usually during a workup for menorrhagia. Thus, in most cases of acute disseminated intravascular coagulopathy (DIC), such clotting abnormalities will be readily apparent.

In the bleeding patient with DIC, rapidly infuse platelets and/or fresh frozen plasma, depending on laboratory values. In general, fresh frozen plasma should be administered when a bleeding patient has a fibrinogen level below 100 mg/dL. Platelets are reserved for the bleeding patient with a platelet count below 30,000 per cubic centimeter.

If large amounts of lost blood are replaced only with crystalloid solution and packed red blood cells, the patient may develop a dilutional coagulopathy that resembles DIC in many ways. Treatment is the same. If shock intervenes, elements of both dilutional and consumptive coagulopathy may be present.

Other considerations

In managing postpartum hemorrhage, a number of other nuances or options may be relevant.

Placenta accreta. The prevalence of placenta accreta is on the rise—some experts have even described it as epidemic.[7] The principal predisposing factors are placenta previa and 1 or more prior cesarean deliveries: In a patient with placenta previa and 1 prior cesarean, the risk of placenta accreta approaches 25%; in the presence of 2 or more cesareans, this risk exceeds 50%.[7] Thus, the increased prevalence of placenta accreta is not surprising, considering the rise in cesarean sections over the past decade.

A number of diagnostic techniques, including color-flow ultrasound, power Doppler, and magnetic resonance imaging (MRI), may be helpful in the antenatal diagnosis of placenta accreta. In fact, in certain centers, these techniques appear to have a high degree of sensitivity and reasonable positive predictive value.[8,9] Still, no diagnostic test is so sensitive that the clinician can perform a repeat cesarean in a patient with placenta previa without being prepared for cesarean hysterectomy. Prior to delivery, these patients should be informed of their high risk of requiring hysterectomy. In addition, blood generally should be prepared as type and cross rather than type and screen, and the OR should have the instrumentation and personnel necessary to perform an emergent hysterectomy.

One of the most important variables is the decision to perform hysterectomy. If the placenta does not readily shell out when manual removal is attempted, the operator should not attempt to pull it out in fragments. Rather, he or she should proceed immediately to hysterectomy.

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In cases of placenta accreta or increta, simple hysterectomy generally will suffice. In rare cases of accreta, however, the uterus may invade other organs, making immediate surgery difficult, if not impossible. Under such circumstances, abnormal vascularity may be evident. These cases often require the expert assistance of a urologist, gynecologic oncologist, or vascular surgeon.

In some instances, the placenta cannot be successfully removed without maternal exsanguination. In these cases, delivery of the fetus through a fundal incision, so that the placenta is left intact, has been described. Some patients benefit from chemotherapeutic agents such as methotrexate to enhance placental shrinkage in the postpartum period. In most cases, however, a return to the OR will eventually be necessary. Even so, significant placental/uterine involution will have occurred by then, making surgery significantly easier.

Angiographic embolization. This is an exciting technique that is rapidly developing into an effective way to control postpartum

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hemorrhage. Its main limitation is that it is time-consuming and, thus, generally unsuitable for the rapid treatment of massive intraoperative or peripartum hemorrhage. However, a slowly evolving retroperitoneal hematoma or other forms of gradual, persistent hemorrhage often are amenable to angiographic identification followed by embolization. Indeed, a number of studies have suggested good outcomes in carefully selected, hemodynamically stable patients.[10,11] In these cases, angiographic embolization may help the physician avoid laparotomy and hysterectomy.

Blood-product infusion. When trying to stop a hemorrhage, do not overlook the need for blood and component therapy. The appropriate use of packed red blood cells, crystalloid solution, fresh frozen plasma, and platelets is beyond the scope of this discussion. While an experienced physician certainly can direct both blood-banking infusion and the surgical approach to hemostasis, it sometimes is helpful to formally delegate blood and component replacement to another specialist, e.g., the anesthesiologist.

Conclusion

Although postpartum hemorrhage carries the potential for serious maternal morbidity—even mortality—it generally can be managed successfully if it is approached in a systematic manner. The first step is determining the cause of bleeding: uterine atony, genital-tract laceration, retained placenta, or coagulopathy. While the cause of bleeding usually is singular, things aren't always that simple. A laceration may accompany uterine atony, or retained placenta may contribute to persistent uterine atony.

Uterine compression, oxytocin infusion, and/or other medical management often are effective approaches to atony. For lacerations and retained placenta, a surgical approach often is necessary. Although it should be the last resort, hysterectomy sometimes is required. The wise clinician anticipates this possibility and plans accordingly. ■

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