40 y/o gravida 5 para 4, presents to your hospital with painless vaginal bleeding at 38 weeks' gestation. She had one episode of painless vaginal bleeding at 28 weeks' gestation which resolved spontaneously. She has a past surgical history of three cesarean sections. Her obstetrician wants to take her back for a cesarean section now for continued vaginal bleeding. Her vital signs are as follows: blood pressure 90/36 mm Hg, heart rate 112 beats per minute, respiratory rate 22 per minute, and Sp02 97 % on room air. Her hematocrit is 25.

Discussion questions:

Discuss anesthesia-related maternal mortality.

Anesthesia-related maternal mortality has decreased from 4.3 per million live births during 1979 to 1981 to 1.7 per million live births during 1988 to 1990. Most deaths occurred during general anesthesia for cesarean sections, with 73% arising from airway problems (aspiration, induction, or intubation problems; inadequate ventilation; or respiratory failure). Since 1984, use of regional anesthesia has been correlated with a decrease in the number of maternal deaths, whereas general anesthesia-related maternal deaths have remained unchanged during the 1979 to 1990 period.


How would you diagnose placenta previa in this patient?

Patients who present with placenta previa usually have painless bleeding in either the second or third trimester. Placenta previa occurs in up to 1% of all full term pregnancies. The first event of bleeding typically occurs preterm, is not associated with increased uterine activity, and generally resolves spontaneously. If placenta previa is suspected, then a vaginal examination is avoided due to the risk of massive hemorrhage. Ultrasonography is most frequently used to diagnose placenta previa. Not only can it help to localize the placenta but it also can be used to rule out a placental abruption which may also be present. Magnetic resonance imaging (MRI) may also be used to help with the diagnosis of placenta previa, but it is not the first modality chosen due to cost and availability.


How is placental abruption diagnosed?

Placental abruption usually presents with painful vaginal bleeding. It is typically associated with uterine tenderness and increased uterine activity. The amount of bleeding is not always easy to quantify. Much of the bleeding can be confined behind the placenta, making an estimate of blood loss difficult. Placental abruption can be diagnosed with ultrasonography or magnetic resonance imaging (MRI).
Who is at risk for uterine rupture and how is uterine rupture diagnosed?

Uterine rupture is a rare occurrence with an incidence of 1 in 15,000 patients who have an unscarred uterus. However, the risk for uterine rupture or dehiscence is increased in patients with a previous uterine scar. Similarly, in patients attempting vaginal birth after cesarean section (VBAC), use of prostaglandins have been shown to increase the relative risk of uterine rupture by a factor of 15. Additional risk factors associated with uterine rupture include uterine trauma, a tumultuous labor, midforceps delivery, breech version and extraction, inappropriate uterotonic use, uterine anomalies, placenta percreta, tumors, fetal macrosomia, and fetal malposition. The most consistent sign of uterine rupture is fetal distress. Other signs that may be present include hypotension, vaginal bleeding, abdominal pain, change in the uterine contour, changes in the uterine contraction pattern, and cessation of labor. True diagnosis is made either during manual inspection of the uterus or during a laparotomy.


What is vasa previa, how is it diagnosed, and what is the management?

Vasa previa develops when the intramembranous fetal vessels overlie the cervical os in front of the fetal presenting part. The patient usually presents with painless vaginal bleeding and decreased fetal movement. Fetal mortality is high because the bleeding is from a fetal source and can lead to fetal hemorrhage. The incidence of vasa previa was found to be 1.6 per 10,000 pregnancies in a study that looked at 93,874 patients.

Diagnosis is made by either palpation or ultrasonography. Color Doppler ultrasonography can be used to help with the diagnosis. If blood is present then it can be tested by either a Wright's stain or an Apt test. A Wright's stain is conducted to look for fetal nucleated red blood cells (RBCs). An Apt test is performed by adding an alkaline solution to the blood. Adult RBCs will rupture in this environment and turn the solution brown as opposed to fetal RBCs, which will remain bright red.

If a vasa previa is suspected then the patient should have an emergent cesarean section. Owing to the small blood volume of the fetus delay in delivery could lead to fetal demise. Because the bleeding is primarily from a fetal source the mother is usually hemodynamically stable. A regional or a general anesthetic could be selected and should be based on maternal factors in addition to the need for a rapid surgical anesthetic.

How is placenta accreta diagnosed?

The frequency of placenta accreta is between 1 in 1,667 and 1 in 70,000. Antepartum diagnosis of placenta accreta would be preferred so that management could be appropriately planned. Unfortunately this is often not the case and diagnosis of placenta accreta is made postpartum when separation of the placenta does not occur, hematuria develops, uterine inversion occurs, or massive hemorrhage follows placental removal. Patients with a prior uterine scar who present with previa should be suspected as possibly having placenta accreta. Patients at a high risk could have gray-scale ultrasonography, color Doppler ultrasonography, or MRI to help identify placenta accreta. The diagnosis can be confirmed during laparotomy.
In a patient with placenta previa who is bleeding profusely, fetal maturity does not impact the decision to proceed with immediate cesarean delivery. Patients with a small, self-limited episode of bleeding and a premature fetus can be treated conservatively with bed rest and corticosteroids to accelerate fetal lung maturity. Once the patient is closer to term, cesarean delivery is performed. With the use of antenatal corticosteroids and exogenous surfactant to prevent respiratory distress syndrome, neonatal survival is now greater than 80% after 26 weeks' gestation and approximates 100% after 32 weeks' gestation.


After delivery of the fetus and placenta you notice generalized oozing from the surgical site. What is disseminated intravascular coagulation (DIC)?

DIC occurs when consumption of the coagulation factors, platelets, and fibrinogen occurs with deposition of thrombi in the microcirculation. The thrombi can diminish the blood flow to organs, which may ultimately lead to multisystem organ failure. At the same time, fibrinolysis occurs with the formation of fibrin split products. Owing to the inappropriate consumption of the factors and platelets bleeding can be difficult to control. Coagulation can be impaired to the level of spontaneous bleeding from any disturbed area including the uterus and intravenous sites. Often DIC occurs in the setting of postpartum hemorrhage, which worsens an already tenuous situation. DIC can develop rapidly in the peripartum period. DIC is seen in up to 10% of placental abruptions and it occurs more frequently when there is fetal demise. Patients who also have developed pregnancy-induced hypertension or amniotic fluid embolism are at risk for developing DIC. DIC is also seen in patients with sepsis, severe trauma, solid tumors, and hematologic cancers. Laboratory data can help to guide management and should include prothrombin time/partial thromboplastin time/international normalized ratio (PT/PTT/INR), platelets, fibrin degradation products, and fibrinogen. Differentiation between DIC and dilutional coagulopathy can sometimes be done by
observing the fibrinogen levels. Treatment should center on taking care of the underlying disorder and supporting the patient. Low doses of unfractionated heparin at a rate of 300 to 500 units per hour may be helpful in treating DIC. Antifibrinolytic drugs are not recommended for patients in DIC due to the fact that the patients already have inadequate fibrinolysis.


You are called to see a 27-year-old woman who just delivered and is continuing to hemorrhage. What is the definition and differential diagnosis of postpartum hemorrhage?

Postpartum hemorrhage can develop in as many as 5% to 10% of deliveries. It has been classically defined as greater than 500 mL of blood loss in the first 24 hours postdelivery. It is accepted that accurate measurement of blood loss is difficult and may be misleading. The American College of Obstetrics and Gynecology has defined postpartum hemorrhage as greater than a 10% decrease in the hematocrit level or the need for a blood transfusion in the postpartum period. Patients with unexplained hypotension, tachycardia, or low urine output should be suspected as having a postpartum hemorrhage and resuscitation should commence.
The most common cause of postpartum hemorrhage is uterine atony, which accounts for up to 80% of the cases. Other uterine causes include retained products of conception, placental accreta, uterine inversion, and uterine rupture. Nonuterine causes include genital tract lesions, genital tract hematomas, intraabdominal lacerations, pelvic lacerations, and coagulopathies.


What are the treatment options for uterine atony?

Initially, physical compression of the uterus by bimanual compression or uterine massage should be initiated for atony. During this time, oxytocin is started as an intravenous infusion. Synthetic oxytocin (Pitocin, Syntocinon) is the drug of choice that is given once the fetus is born and the placenta is delivered. The uterus increases the number of oxytocin receptors throughout pregnancy, peaking at near term. Oxytocin differs from antidiuretic hormone (ADH) by only two amino acids and is produced in the posterior pituitary. It may exhibit a small antidiuretic effect but clinically this does not seem to be concerning unless very large doses are given. In this situation, systemic and/or pulmonary hypertension, and water intoxication can occur. If a bolus of oxytocin is given, then hypotension can follow and should be treated with appropriate vasopressors (ephedrine or phenylephrine). The hypotension is caused by a decrease in peripheral vascular resistance. Patients may also exhibit tachycardia and arrhythmias. Once the oxytocin binds onto the receptors, the frequency and duration of the uterine contractions increase. The contractions are most likely mediated through an increase in intracellular calcium levels. Ergot alkaloids (ergonovine and methylergonovine) can be used to treat uterine atony. The agent is usually given intramuscularly and has an effect in 2 to 5 minutes. The ergot alkaloids produce tetanic contractions of the uterus, which is probably mediated through a-
adrenergic receptors. The agents have many potential side effects. Hypertension can be prominent due to vasoconstriction such that these agents should be avoided in chronic hypertensive patients or in patients with pregnancy-induced hypertension. The ergot alkaloids may have other cardiovascular affects including coronary artery spasm leading to myocardial infarction, arhythmias, or cerebrovascular accidents. Pulmonary complications have been reported with the ergot alkaloids including pulmonary artery vasoconstriction and pulmonary hypertension. The ergot alkaloids are also associated with side effects such as headache, dizziness, and nausea and vomiting. Prostaglandins increase through labor reaching a peak level after the placenta is delivered. The prostaglandins cause the intracellular levels of calcium to increase, leading to an increase in myosin light-chain kinase activity and then uterine contraction. Side effects with the prostaglandins include nausea and vomiting, fever, and diarrhea. IS-Methyl prostaglandin F2-a (carboprost, Hemabate) can be given either intramuscularly or intramyometrially. The prostaglandins should be avoided in patients with reactive airway disease due to the bronchospasm that can develop. Prostaglandin E1 (Misoprostol) can be given rectally to help when uterine atony is persistent. A review of randomized controlled trials comparing misoprostol (PO/rectal) verses injectable (IV/IM) uterotonics(oxytocin / ergotamine / combination) found that misoprostol was less effective than injectable uterotonics.


Define amniotic fluid embolism and discuss treatment.
Amniotic fluid embolism can occur when there is direct communication between the amniotic fluid and the maternal circulation. This most likely occurs in multiparous patients who are experiencing a complicated labor. The communication can occur at the level of the endocervical vessels, at the level of the placenta, or at a uterine trauma site. The patients may experience sudden onset of hypotension, dyspnea, hypoxemia, cyanosis, loss of consciousness, and possible seizures. The symptoms are due in part to mechanical blockage of the pulmonary vasculature and then subsequent pulmonary vasoconstriction, which may be mediated through the release of substances such as prostaglandins, histamine, serotonin, or leukotrienes. The patients may develop acute cor pulmonale and right heart failure. More than 80% of these patients experience cardiopulmonary arrest. The patients are at a high risk for developing disseminated intravascular coagulation (DIC). The patients need to have their airway secured and their ventilation supported. Application of positive end-expiratory pressure may help to oxygenate the patients. An arterial line should be placed to monitor hemodynamics. Central monitoring may help to evaluate fluid requirements. The blood pressure should be supported with vasopressors and DIC should be treated as discussed previously. The mortality of patients who develop an amniotic fluid embolism is higher than 80%. Amniotic fluid embolism is often a diagnosis of exclusion. Diagnosis is usually based on clinical signs and symptoms. Blood aspirated from pulmonary artery catheters would often show squamous cells, fat, and mucin.


What is the role for uterine artery balloon placement and uterine artery embolization?

Uterine artery balloon placement and uterine artery embolization have both been used in the peripartum setting due to either anticipated or ongoing obstetric hemorrhage. Owing to the improved imaging modalities and known associations with abnormal placentation, prophylactic internal iliac artery occlusion balloon catheters or embolization catheters can be placed preoperatively. The balloon catheters may help reduce the bleeding associated with abnormal
placentation or uterine anomalies (e.g., arteriovenous malformation, atony). Abnormal placentation is often associated with collateral circulation and balloon dilation may not completely stop bleeding. It has been suggested that the use of balloon dilation may reduce the overall blood loss in these cases and may provide a more stable surgical situation. Although balloon placement with or without embolization may help to control bleeding, a hysterectomy may still have to be performed for definitive control of blood loss. Prophylactic catheterization may allow the patient to avoid a general anesthetic and all the complications associated with general anesthesia. It must be understood that conversion to a general anesthetic may need to be performed during the case, if continuing resuscitation is needed and airway control is required.

In an emergent situation, arterial embolization may help control hemorrhage. Some patients may experience complications due to emergent embolization including thrombosis of the left popliteal artery, vaginal necrosis, and paresthesia of the right leg.

