# Abruptio Placenta and Placenta Previa

# 50

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**CASE** Called stat by obstetrician for a 29-year-old female patient who is presenting pregnant at 30 weeks gestation with fetal bradycardia, vaginal bleeding, and complaining of abdominal pain.

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| Medications: | Prenatal vitamins |
| | Iron Supplementation |
| Allergies: | NKA |
| Past Medical History: | Gestational GERD |
| | Iron Deficiency anemia |
| Physical Exam: | Vital Signs: BP 110/60, HR 105 bpm, RR 30/min, oxygen saturation 99%. |
| | Patient appears anxious, slow trickle of bright red blood from vagina. |
| Cardiac: | Regular rhythm, grade 2/6 systolic ejection murmur heard only at left sternal boarder |
| Otherwise: | insignificant |

1. **What are the four major causes of antepartum hemorrhage? What are the incidences of each?**

The major causes of antepartum hemorrhage include placenta previa, placental abruption, uterine rupture, and vasa previa. Placenta previa occurs when the placenta implants over the cervical os. This implantation may be marginal, partial, or total in its covering of the os. The incidence of placenta previa is 4.0 per 1000 pregnancies [1]. Placental abruption refers to the complete or partial separation of the placenta from the uterine wall before delivery of the fetus. The incidence of placental abruption varies with the population studied 3–10 per 1000 pregnancies [2]. Uterine rupture following previous vaginal delivery has an incidence of 0.18 per 1000 pregnancies. This increases significantly to 9 per 1000 pregnancies in women who have had a previous cesarean section [3]. When fetal vessels have a velamentous insertion over the cervical os, the fetal vessels are not protected by the umbilical cord or the placenta. This is diagnosed as vasa previa and has a traditionally reported incidence of 1 in 2500 to 1 in 5000 [4, 5].

2. **What are the risk factors for placenta previa, placental abruption, and uterine rupture?**

The risk factors for placenta previa and placental abruption were studied in over 16 million pregnancies, in the United States, from 1995–2000. Risk factors for placenta previa include advanced maternal age, multiparity, and history of Cesarean delivery. Notably, these risk factors exist *prior* to pregnancy. Risk factors for placental abruption include conditions occurring *during* pregnancy: cigarette smoking, alcohol intake, preterm premature rupture of membranes, and chorioamionitis [6–8].

3. **What are the risk factors for uterine rupture?**

The most common risk factor for uterine rupture is previous Cesarean section or uterine surgery [9]. Other reported risk factors include induction of labor, macrosomia ≥4 kg, gestational age greater than 42 weeks, maternal age over 35, and short maternal stature [3]. Uterine rupture is rare but catastrophic and requires prompt identification and management.

4. **What are normal vital signs for a pregnant patient in her third trimester?**

Normal baseline heart rate (HR) increases up to 25% during pregnancy (by 10–20 bpm on average), however maternal tachycardia is still defined as HR ≥100 bpm [10–12]. Blood pressure (BP) slowly decreases from the first to

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second trimester but returns to back to baseline by term gestation [13]. Maternal hypertension is defined as BP ≥ 140/90 [14].

Respiratory rate does not increase significantly with pregnancy, despite a 40% increase in tidal volume and a 30–50% increase in minute ventilation. Tachypnea (RR ≥ 20 bpm) should be considered abnormal in the pregnant patient [15]. Oxygen saturation should remain normal in a pregnant patient at sea level and mild altitude [15].

5. **What are the cardiovascular physiologic changes seen in pregnancy and what is the most likely etiology of the murmur?**

The parturient undergoes a number of physiologic changes with impact on the cardiovascular system. There is a notable decrease in afterload as well as increase in cardiac output, heart rate, and stroke volume. These adaptive hemodynamic changes are likely triggered by an early fall in systemic vascular tone in pregnancy [16]. There is no significant change in pulmonary capillary wedge pressure, central venous pressure, left ventricular stroke work index, or mean arterial pressure [11]. In order to accommodate for the increase in plasma and stroke volume, the left ventricle dilates and hypertrophies. There is an increase in myocardial contractility by the second trimester [17].

The 50% increases in cardiac output and plasma volume may be difficult to tolerate if a pregnant patient has valvular heart disease. The increase in cardiac output worsens myocardial oxygen demand, exacerbates CHF, and the low SVR may decrease coronary perfusion, causing myocardial ischemia. The drop in SVR may also be significant in women with shunt pathology or congenital heart defects [18]. Labor contractions can rapidly increase the already increased cardiac output. During the second stage of labor cardiac output can increase by an additional 50% [11].

Up to 90% of women will develop a systolic ejection murmur during pregnancy. They are commonly heard best at the second left intercostal space or along the left sternal border. These murmurs are related to the alterations of blood flow through normal valves and can be attributed to the increase in blood volume, cardiac output, and blood velocity [19].

6. **What are the four classes of hemorrhagic shock and how much blood will this pregnant patient lose before she becomes hypotensive?**

Based on the ATLS hemorrhagic shock classification (Table 50.1) [20], a healthy pregnant patient will not show signs of hypotension until Class III shock, or 30% of blood loss. In a 70 kg term gestation pregnant patient who has a 50% increase in plasma volume this means that the patient may have up to 2 L of blood loss before she displays signs of tachycardia and hypotension.

7. **What are some clinical signs to indicate this patient is in hemorrhagic shock?**

In practice, staff physicians, residents, and nurses are grossly inaccurate at estimating blood loss after vaginal or cesarean delivery. There tends to be an underestimation which only gets worse with increasing blood loss [21]. Care providers should rely on the clinical picture of the patient to guide fluid and resuscitation management. Clinical signs of severe hemorrhagic shock include decreased urine output, altered mental status (confusion, anxiety, and lethargy), tachypnea, decreased pulse pressure and the late signs of tachycardia and hypotension [20].

**Table 50.1** The ATLS classification of hypovolemic shock [20]

| | Class I | Class II | Class III | Class IV |
|---|---|---|---|---|
| Blood loss in % | <15 | 15–30 | 30–40 | > 40 |
| Pulse rate | <100 | 100–120 | 120–140 | >140 |
| Blood pressure | Normal | Normal | Decreased | Decreased |
| Pulse pressure | Normal or increased | Decreased | Decreased | Decreased |
| Respiratory rate | 14–20 | 20–30 | 30–40 | >35 |
| Mental status | Slightly anxious | Mildly anxious | Anxious, confused | Confused, lethargic |
| Urine output (ml/h) | >30 | 20–30 | 5–15 | Negligible |
| Fluid replacement | Crystalloid | Crystalloid | Crystalloid and blood | Crystalloid and blood |

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8. **What are the immediate steps in management of this patient?**

This patient has multiple emergent issues that must be concurrently diagnosed and treated in the immediate management of this patient. Foremost, this patient is having ongoing vaginal bleeding and is showing early signs of hemorrhagic shock including tachycardia (HR > 100), tachypnea (RR > 20), and altered mental status (anxiety). There is also acute abdominal pain which may shed light into the etiology of the bleed. Finally, there are two patients to consider in this clinical scenario—the parturient and the fetus. Fetal bradycardia is an obstetric emergency and may indicate fetal hypoxia and/or impending death.

Obstetric, anesthesia and neonatal teams should be emergently called to the patient's bedside. The patient should be in a monitored setting with BP, HR, $SpO_2$, and ECG monitoring. 100% $O_2$ should be applied to this patient and two large bore IV's should be started above the diaphragm with initiation of a crystalloid bolus of 1–2 L. The patient should get urgent bloodwork including a complete blood count, coagulation profile, and type and crossmatch for potential blood transfusion. It may be appropriate at this time to call for blood products. Vital signs should be measured at minimum every 5 min including level of consciousness. Attempts should be made to keep the patient euthermic. The operating suite should be notified and prepared for an emergency cesarean delivery[22].

9. **If this patient's blood type is unknown, which type of blood should be given in an emergency resuscitation?**

This patient should receive type-O negative or uncrossmatched packed red blood cells if crossmatched are not immediately available [23]. Type-AB fresh plasma and apheresis platelets should be given in an emergency-release transfusion if the patient's blood group is unavailable [24, 25]. If an Rh negative patient receives Rh positive platelets in an emergency transfusion, she should receive Rho(D) immune globulin afterwards.

The decision on when to transfuse plasma, platelets and cryoprecipitate should be guided by laboratory and clinical factors. Guidelines have recommended transfusion thresholds of platelet count $\leq 50 \times 10^9$/L, INR > 1.5, and fibrinogen < 1.0 g/L [22].

10. **Should this patient receive a general anesthetic or neuraxial anesthetic?**

In many obstetrical emergencies, the type of anesthetic must be individualized to the patient and clinical scenario. If the fetal heart rate is persistently bradycardic despite adequate resuscitation of the mother, or if the vaginal bleeding remains uncontrolled, an emergency "stat" cesarean delivery should be anticipated. In the setting of uncontrolled and uncorrected hypovolemia a general anesthetic may be favored. A neuraxial technique is contraindicated if the maternal hemorrhage persists to the point of suspected coagulopathy or DIC. The anesthesiologist should keep in mind the time required for induction of neuraxial anesthesia; with persistent fetal bradycardia a general anesthetic may be indicated [26].

11. **Which optimization techniques could be considered prior to induction of this patient?**

If time and the clinical situation permits, optimization of both the parturient and fetus should be explored. These optimization techniques should not delay delivery if there are maternal and/or fetal indications for emergency delivery [27, 28]. A 30 week fetus with threatened preterm labor can be optimized with corticosteroid therapy using either betamethasone 12 mg IM two doses 24 h apart or dexamethasone 6 mg IM four doses 12 h apart. This has been shown to reduce perinatal mortality, respiratory distress syndrome, and other morbidities in the infant [29].

Magnesium sulphate ($MgSO_4$) should be considered for women at ≤31 + 6 weeks with imminent preterm birth. It is dosed as 4 g IV load over 30 min then 1 g/hr infusion until birth for a maximum of 24 h. Risks and side effects of magnesium include muscle atony. An ominous sign is the loss of deep tendon reflexes. Uterine atony may lead to further bleeding postpartum. Fetal hypotonia and apnea may have implications for the neonatal care provider [27, 28].

This pregnant patient is at risk for pulmonary aspiration of gastric contents and should be given prophylaxis for aspiration pneumonitis. A combination of antacids (e.g., 30 mL of oral sodium citrate) plus $H_2$ antagonist (Ranitidine 50 mg IV) have been shown to prevent low gastric pH [30]. All women undergoing elective or emergency Casarean section should receive antibiotic prophylaxis [31].

12. **What are the 4 major classifications of postpartum hemorrhage? Which is the most common?**

A common way to think of the differential for postpartum hemorrhage (PPH) is the '*4 T's.*' **T**one—uterine atony or distended bladder. Uterine atony is the most common cause of PPH. **T**issue—retained tissue from the placenta or blood clots can cause persistent postpartum bleed and can also contribute to poor uterine tone. **T**rauma—lacerations to the vaginal wall, cervix, or uterine injury may contribute a significant amount of blood loss until appropriately repaired. **T**hrombin—a woman may have an undiagnosed

coagulopathy presenting for the first time with childbirth, or it may be an acquired or consumptive coagulopathy [32]. DIC has been associated with placental abruption, placenta previa, amniotic fluid embolism, pre-eclampsia, HELLP syndrome, intrauterine fetal demise, intrauterine infection, and acute fatty liver of pregnancy [33].

13. **What are the risk factors for uterine atony?**

Uterine atony can be broken down into sub-categories in order to organize a differential and remember clinical risk factors. Over-distension of the uterus can be caused by polyhydramnios, multiple gestation, or macrosomia. Uterine muscle exhaustion may be secondary to rapid or prolonged labor, high parity and oxytocin use. Intra-amniotic infection should be suspected if there is maternal fever or prolonged rupture of membranes. Uterine abnormality risks include fibroids, placenta previa, bladder distension, or other anomaly. Finally, uterine-relaxing medications can lead to uterine atony including halogenated anesthetics and nitroglycerin [32].

14. **Name four pharmacologic interventions and four mechanical interventions that can be used to treat uterine atony**

Ongoing blood loss with decreased uterine tone requires administration of uterotonics. The following are recommended medications for the management of PPH due to atony:

*Oxytocin* 10 units IM or 10–40 units in 1 L of crystalloid run in continuously. Rapid undiluted IV boluses should be avoided because they can cause hypotension.

*Methylergonovine* (Methergine) 0.2 mg IM every 2–4 h can be used but avoided if the patient is hypertensive.

*15-methyl PGF-2α* (Carboprost or Hemabate) 0.25 mg IM every 15–90 min, 8 doses maximum. This should be avoided in asthmatic patients and is relatively contraindicated in hepatic, renal, and cardiac disease. Common side effects include diarrhea, fever, and tachycardia.

*Dinoprostone* (Prostaglandin E2) is a vaginal or rectal suppository of 20 mg every 2 h. It should be avoided if the patient is hypotensive. Fever is a common side effect.

*Misoprostol* (Cyotec, PGE1) 800–1000 mcg rectally [34].

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