Placenta Accreta and the Maternal-Fetal Interaction

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Placenta accreta is an increasingly prevalent and potentially life threatening obstetrical condition associated with many maternal and neonatal mortalities and morbidities. Placenta accreta is a generalized term used to describe an abnormally deep implantation of all or part of the placenta to the uterine wall. It is further classified into placenta increta, where the chorionic villi of the placenta invades the myometrium layer of the uterus; and placenta percreta, where the placenta invades through the myometrial wall, possibly attaching to surrounding abdominal organs. By penetrating through the uterine muscle, placenta accreta becomes problematic during the 3rd stage of labor. Because of the deep implementation it is difficult to separate the placenta from the uterus resulting in massive maternal hemorrhage and probable hysterectomy (The American College of Obstetricians and Gynecologists [ACOG], 2012). An increase in cesarean section deliveries (C-sections) has been associated with a 10 fold increase in placenta accreta in the last 20 years. It is now effecting approximately one in every 533 pregnancies (Robinson & Grobman, 2010).

Management of placenta accreta requires a multi-disciplinary approach. Antenatal steroid administration and a planned preterm C-section with a hysterectomy is the current recommendation for known placenta accreta (ACOG, 2012). However not all cases are identified prior to delivery, and there continues to be emerging and conflicting theories regarding the appropriate management of placenta accreta and preterm delivery. Therefore, steroid administration, timing of delivery and surgical management should be individualized to each patient.
The Pathophysiology of Placenta Accreta

The exact cause of placenta accreta is unknown; however, many theories identify a uterine lining defect or primary defect in decidualisation as the cause (Khong, 2008). Decidualisation is the change of maternal uterine cells that occurs during pregnancy in response to the hormone progesterone, and it is believed to regulate the depth of implantation of the placenta. However, prior surgeries such as C-sections or uterine abnormalities can alter the decidua or uterine wall leading to uncontrolled placental growth into or through the uterus (Redline, 2011). Compared to a normal placenta, there are larger dilated arteries associated with placenta accrete which is why attempted removal of the adherent placentas can lead to significant postpartum hemorrhage (Khong, 2008).

There is an increased risk of placental accretion in women with placenta previa (a low-lying placentation) and history of cesarean section, as well as advanced maternal age, high parity, or after myomectomy or curettage (Judge, Morrison, & Lazebnik, 2011).

Placenta accreta is rarely accompanied by maternal or neonatal symptoms and is often only diagnosed during routine ultrasounds when viewing the implementation of the placenta. MRIs are also diagnostic tools used when ultrasounds are inconclusive. If suspected, a blood test to evaluate alpha fetal protein may be done since a rise in this has been linked to placenta accrete.

The Impact of Placenta Accreta on the Fetus

In a study that compared neonatal outcomes between known placental accretion and planned deliveries versus unplanned emergency deliveries, it was identified that there was no difference in length of NICU stays, respiratory distress syndrome or surfactant administration. It was concluded that unless additional complications arise, placenta accreta may not have a direct
effect on neonatal outcomes and therefore, an increased level of fetal surveillance is not necessary unless clinically indicated (Balayla & Bondarenko, 2013). In light of additional maternal complications, the only identified link between placenta accreta and the fetus is intrauterine growth restriction.

The main complications during pregnancy are heavy bleeding during the 3rd trimester and, although very rare, uterine rupture. In the event of uterine rupture and maternal hemorrhage, symptoms depend largely on the extent of injury on the fetus, the compensatory mechanisms of the fetus and the clinical response time (King, 2013). Manifestations of uterine rupture and hemorrhage include fetal distress evidenced by prolonged decelerations or bradycardia, sudden or atypical abdominal pain, a boggy uterus, frank bleeding, tachycardia, hypotension and shock (Chertok, 2013). If uterine rupture is identified, it is managed by emergency C-section, multiple blood transfusions and a potential hysterectomy.

Because the current recommendation by the American College of Gynecology is to schedule a preterm delivery, placenta accreta is associated with preterm birth; however, this neonatal effect is related to the therapy and treatment of placenta accreta and not directly with the obstetrical condition.

**Small for Gestational Age (SGA)**

The most common direct impact of placenta accreta on the fetus is intrauterine growth restriction (IUGR). This is likely related to uteroplacental insufficiency due to abnormal attachment of the placenta and decreased placental area available for gas and nutrient exchange (King, 2013). The impaired blood flow affects the circulation of nutrients and gases thereby causing intrauterine growth restriction (IUGR) and subsequently small for gestational age (SGA) infants (Khong, 2008).
A standardized growth chart, in combination with Ballard scoring, should be utilized to assess the gestational age and significance of below average fetal growth. SGA is usually defined as a birth weight below the 10th percentile for gestational age. Clinical manifestations of SGA infants include infants who are thin with loose peeling skin, little subcutaneous tissue, a scaphoid abdomen and a disproportionately large head. SGA is also associated with hypoglycemia, RDS, persistent pulmonary hypertension (PPHN), PDA, hypothermia, cholestasis, polycythemia, thrombocytopenia, chronic lung disease, hypocalcemia and an increased risk of necrotizing enterocolitis (NEC).

Diagnostic evaluations and management of the suspected SGA neonate includes performing a Ballard assessment and plotting the infant’s weight, head circumference and length on a standardized growth chart. It is likely that the SGA is due to placental insufficiency; however, if any other clinical indicators or concerning data arises, a workup should be done to identify other potential causes of SGA such as maternal infection. Each patient’s care should be individualized and will be based largely on weight, gestational age and clinical status; but, management of the SGA infant will likely include admission to the NICU on a cardio-respiratory and pulse oximetry monitoring to observe for apnea and bradycardia events as well as signs of respiratory distress. If the infant requires respiratory support, blood gasses, chest x-rays or surfactant administration should be considered. The infant should be admitted into a giraffe isolette to prevent heat loss. A baseline CBC should be done to evaluate for infection, neutropenia and thrombocytopenia. Polycythemia is often seen with placental insufficiency and chronic intrauterine hypoxia. Because of suspected impaired blood flow inutero, feedings should be cautiously advanced since these infants are at an increased risk for NEC. Fluid balance should be closely followed with routine electrolytes and blood glucose tests since there is an increased risk of hypocalcemia and
hypoglycemia. The practitioner should also be on the lookout for sings of PPHN since many IUGR infants are subject to chronic intrauterine hypoxia, which resulting in thickening of smooth muscles of the pulmonary arterioles. If clinically indicated, an ECHO should be done to evaluate for PPHN and PDA.

**Perinatal hypoxia and asphyxia**

Spontaneous uterine rupture and severe maternal hemorrhage prior to delivery results in an acute decrease in placental blood flow. This causes fetal distress and potential fetal hypoxia and perinatal asphyxia (King, 2013). Sustained perinatal asphyxia can lead to hypoxic-ischemic encephalopathy (HIE) because of cerebral ischemia. HIE is defined as a disturbance in neurologic function. Maternal hypotension, trauma and uterine hemorrhage account for 20% of HIE cases (Gomella, 2013).

HIE can be further complicated by metabolic acidosis, acute kidney injury with renal failure, fluid and electrolyte imbalances, respiratory failure, hepatic dysfunction, seizures, thrombocytopenia, cerebral hemorrhage, organ damage and death. A physical examination of the infant with laboratory data will be needed to diagnoses perinatal asphyxia. Clinical presentation that suggests perinatal asphyxia will likely include sudden or sustained fetal bradycardia, initial metabolic acidosis, Apgar scores of 0-3 beyond 5 minutes of life, onset of multisystem involvement with first 72 hours and early imaging showing evidence of acute nonfocal cerebral abnormality. Level of consciousness will be altered and may include initial deep stupor, depressed reflexes, respiratory failure or periodic breathing, hypotonia, potential seizures and jitteriness (Gomella, 2013).

If HIE is diagnosed and the infant meets the recommended institutional guidelines, therapeutic hypothermia should be considered. Admission to the NICU on a cardio-respiratory
and pulses oximetry monitoring is likely. Additional evaluation and treatment includes an electroencephalography (EEG) and magnetic resonance imaging (MRI) to evaluate cerebral function, serial blood gasses with intervention to monitor respiratory acidosis and adequate ventilation, and CBC and blood culture to identify infection, thrombocytopenia and anemia. Initial fluid restriction, strict intake and output with daily weights, electrolytes, BUN and creatinine is recommended to monitor fluid and electrolyte balance and kidney function since HIE patients are at risk for fluid overload and cerebral edema. Blood glucose levels should be closely monitored to prevent progression of moderate to severe encephalopathy. Feedings should be advanced cautiously since blood flow is redirected away from the gut, kidney and spleen in the event of fetal hypoxemia. Additionally, consultation with neurology and developmental team is recommended (Gomella, 2013).

The prognosis of HIE varies based on the severity of the insult. Future effects of HIE on the child range from normal outcomes to neurologic disability, cerebral palsy or death. Various models can be utilized in conjunction with the clinical picture in order to predict severity and outcomes (Gomella, 2013).

**Neonatal hypovolemia, anemia, and birth injury**

Additional neonatal complications of maternal hemorrhage and uterine rupture include hypovolemic shock, anemia and potential birth injuries and nonphysiologic hyperbilirubinemia from an emergency C-section. An infant with hypovolemic shock may display tachycardia, prolonged capillary refill, respiratory distress, poor tone, pale color, cold extremities, lethargy, narrow pulses pressures, apnea and bradycardia, metabolic acidosis and weak pulses. Symptoms of anemia due to an acute hemorrhagic event include poor pallor, tachypnea or gasping respirations, decreased peripheral perfusion and poor capillary refill (Gomella, 2013). For
suspected hypovolemia and severe anemia. CBC, reticulocyte count, electrolytes and blood gases may be necessary. If indicated, volume expansion with intravenous crystalloid solution or blood replacement at 10ml/kg should be done. A liver panel may be required for a suspected liver injury or hyperbilirubinemia due to increased bilirubin load from red blood cell breakdown. Clinical manifestations of hyperbilirubinemia may include jaundiced color skin, clay colored stools or dark urine, poor feeding, lethargy, hypotonia and possible seizures (Gomella, 2013).

**Therapy and Treatment Options for Placenta Accreta and how it Effects the Neonate**

Treatment plans must be individualized for each patient. However evidence shows that best practice involves early planning, administration of antenatal steroids, and a scheduled C-section at 34 weeks gestation (Robinson & Grobman, 2010).

**Early Diagnosis and Planning**

Diagnostic ultrasounds have the capability of identifying placenta accreta in the antepartum period. Magnetic resonance imaging (MRI) are also being utilized for further evaluation or when ultrasound findings are inconclusive (Judge et al., 2011). Antenatal diagnosis is critical in order to develop a preoperative plan for delivery. Maternal-fetal medicine specialists should be consulted at this time. Arrangements should be made for the deliveries to take place in a tertiary care center where there are necessary resources to manage potential maternal and neonatal complications including anesthesia, extra delivery personnel, gynecological oncologists, a newborn resuscitation team and in-house neonatal intensive care unit (NICU) (Balaya & Bondarenko, 2013).

**Scheduled Preterm Cesarean Sections**

Studies show that over 40% of women with placenta accreta will experience symptomatic bleeding before 37 weeks. Therefore, a scheduled C-section is recommended 2 days to 2 weeks
following the administration of antenatal steroids (Robinson & Grobman, 2010). Consequently, the neonate will be at a preterm gestation and is thus exposed to complications associated with prematurity. Prematurity is associated with an increased risk of respiratory distress, infection, fluid and electrolyte imbalance, hypoglycemia, anemia of prematurity, temperature instability, reflux, feeding difficulties, NEC, spontaneous intestinal perforation, PDA, apnea and bradycardia, hyperbilirubinemia and intraventricular hemorrhage (IVH) (Bastek, et al., 2008). All of these have a direct effect on the neonate and will likely result in admission to the NICU with prolonged hospitalization and potential respiratory and nutritional support. Diagnostic evaluation and treatment of these complications should be individualized to the neonate and will vary greatly with gestational age and clinical status. Continuous cardio-respiratory and pulse oximetry monitoring, chest and abdominal x-rays, blood gasses, intravenous nutrition, CBC, head ultrasound, blood cultures, metabolic and liver panel labs and ECHOs are some of the evaluations that should be considered in the first 36 hours.

Because a scheduled C-section without labor is the recommended therapy for placenta accreta, regardless of gestational age, the newborn is also at risk for transient tachypnea of the newborn (TTN). This is due to a delayed reabsorption of fetal lung fluid and subsequent impaired gas exchange. Symptoms of TTN will show shortly after birth and usually resolve in 3-5 days. Clinical presentation of TTN usually includes tachypnea, signs of labored breathing, need for respiratory support and increased FiO2, hyperinflation with peripheral streaking of lungs and mild cardiomegaly on x-ray, auscultation of crackles on auscultation, edema and a normal initial CBC (Gomella, 2013).

TTN is often a diagnosis of exclusion, is self-limited and requires only supportive management. Treatment usually includes providing necessary oxygenation, maintaining a neutral
thermal environment, starting IV nutrition to prevent aspiration with tachypnea and close monitoring of fluid balance, electrolytes and hydration (Gomella, 2013).

**Administration of Antenatal Steroids**

The use of antenatal steroids are controversial. Previous randomized studies have demonstrated a link between antenatal steroids and decreased fetal growth and long term neurological conditions such as epilepsy and cerebral palsy. However, many believe these findings are inconclusive or statistically insignificant. There are many documented benefits to antenatal steroid use such as decreased risks of neonatal death, respiratory distress syndrome, chronic lung disease, IVH and ROP (Eriksson, Haglund, Ewald, Odlin, & Kieler, 2009). The discussion on when and if to administer steroids and the use of multiple doses of antenatal steroids remains controversial. It is apparent that additional research and studies are needed in order to draw accurate conclusions.

**Pertinent Theories and Evidenced Based Practice**

The optimal gestational age for scheduled C-section delivery is still controversial; however, literature today recommends the administration of antenatal steroids with a scheduled cesarean delivery and hysterectomy at 34 weeks. Evidence shows this plan is best practice and results in less maternal blood loss and more favorable outcomes for both the mother and neonate. This includes decreased ICU admissions for the mother, decreased perinatal and infant mortality, decreased respiratory distress and decreased infant mental retardation and cerebral palsy (Robinson & Grobman, 2010). In comparison, some studies theorize that if the risk of antepartum hemorrhage is only between 1% and 7%, then the preferred delivery strategy is to wait until 37 weeks gestation (Robinson & Groban, 2010). Scheduled C-sections are
recommended over vaginal delivery because labor can be prolonged and complicated by severe maternal uterine hemorrhage (Balaya & Bondarenko, 2013).

Evidence suggests that antenatal steroids should be given prior to 34 weeks gestations; however, there is little data regarding the effectiveness of corticosteroids after 34 weeks gestation (ACOG, 2011).

Although amniocentesis to evaluate lung maturity was a frequent practice, studies now show that it is an unnecessary procedure. Waiting for fetal lung maturity before delivery does not result in more favorable outcomes and the disadvantages to an amniocentesis outweigh the advantages (Robinson & Grobman, 2010).

The American College of Obstetricians and Gynecologists recommend the use of surgical management with a scheduled cesarean hysterectomy and delivery at 34 weeks as the gold standard for the treatment of placenta accreta (ACOG, 2012). They advise against attempted manual removal of the placenta. Because of placental retention, there is an increased risk of bleeding from the placental tissue which remains in contact with the maternal circulation. A scheduled hysterectomy without placental removal is associated with decreased maternal morbidity (36%) when compared to attempted manual placental removal at (67%) (Balaya & Bondarenko, 2013). There is emerging evidence that a scheduled C-section opposed to emergency C-section is associated with shorter operative times and lower frequency of transfusions, complications and maternal ICU admissions (Robinson & Groban, 2010).

**Economic, emotional and social impact on the family**

Although some families receive a diagnosis of accreta and have time to discuss options and the implications of a hysterectomy, many parents and families are emotionally, mentally and financially unprepared for the birth process and a possible hysterectomy. Since deliveries are
recommended to take place in a tertiary care center, some people are forced to travel for their delivery, possibly separating them from their home and support system. A traumatic birth experience can be associated with immediate and long term psychological distress, physical pain and potential post-traumatic stress disorder (Elmir, Schmied, Jackson, & Wilkes, 2010).

If the delivery results in a hysterectomy, whether planned or unplanned, this can be extremely distressing to family, especially those who desire to have more children. Additional strain is likely to also occur with maternal ICU admissions and death.

It is known that having an infant in the NICU can be an emotional period. Families are overcome with feelings of anxiety, shock, fear, depression, loss of control and overwhelming stress (Obeidt, Bond, & Callister, 2009). There can also be a financial strain on families following a NICU admission. Bloomberg Business week estimates that the average NICU stay costs approximately $41,000 per child, with that number rising for complications and extremely premature infants. This may also be in combination with long term maternal hospitalization and care.

It is important to provide families with as much support and counseling as possible during pregnancy and in the postpartum period. Planning should be done for both emergent situations and planned deliveries, and the families should be included in communication and the plan of care. It is important for practitioners to realize that although birth is usually a joyous occasion, the mother and family unit may be distressed and grieving from a potentially traumatic birth experience or undesired hysterectomy.

**Conclusion**

The management and treatment of placenta accreta necessitates a multidisciplinary approach in order to reduce adverse maternal and neonatal outcomes. Pre-delivery planning and
counseling between the obstetrician, fetal-medicine specialists, neonatologist, mother and family is essential during pregnancy. It is difficult to find a balance between the complications of prematurity and those related to maternal hemorrhage and emergent delivery. There are differing theories on the treatment and management of accreta and the optimal timing of delivery of these patients; therefore, much of the care is individualized. Usually preterm delivery can be avoided, but many times in this situation the continuation of the pregnancy increases the risk of hemorrhage, uterine rupture and fetal death. In these cases preterm delivery will likely be necessary.

Previous cesarean sections are directly linked to placenta accreta and they are largely responsible for the recent rise. Although cesarean sections are more common today and sometimes necessary, they are still associated with increased maternal morbidities. It is important for practitioners and mothers to realize this and alter their practice so that primary cesarean sections are carefully considered and elective repeat cesarean sections are discouraged. Although the only linked fetal effect of placental accreta is IUGR and SGA, additional maternal complications can occur including sudden uterine rupture, maternal hemorrhage, and preterm delivery. Consequently, these maternal complications can result in extensive and supplementary fetal problems. As a practitioner it is important to understand the repercussions of this condition and the physical and emotional effects on both the mother and neonate.
References


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