Obstetrics - Dr. Sundus – Lecture 4 – Abruptio Placentae

DEFINITIONS

Abruptio placentae, or premature separation of the normally implanted placenta, complicates 0.5% to 1.5% of all pregnancies (1 in 120 births). Abruption severe enough to result in fetal death occurs in 1 per 500 deliveries.

PREDISPOSING FACTORS

1. Maternal hypertension.
2. Placental abruption in a prior pregnancy.
3. Trauma (ECV, cordocentesis, road traffic accidents).
5. Premature rupture of membranes.
6. Short umbilical cord.
7. Tobacco use and smoking.
8. Folate deficiency.
10. Increased maternal age.

The most common of these risk factors is maternal hypertension, either chronic or as a result of preeclampsia. The risk of recurrent abruption is high: 10% after one abruption and 25% after two.

Approximately 50% of placenta abruption cases severe enough to cause fetal death are associated with hypertension. Placental abruption may also be associated with:

1. Fetal abnormality: ↑ maternal serum α-fetoprotein, ↑ recurrence of abruption. ?? poor placentaion (↓ adhesiveness).
3. Chorioamnionitis.

Classification

Complete separation: no vaginal bleeding
Partial separation: vaginal bleeding will be apparent
Marginal separation: vaginal bleeding will be apparent

Grades:

*Asymptomatic: retroplacental clot seen after placental delivery.
*Mild: vaginal bleeding (revealed) + uterine tenderness; visible retroplacental clot after placental delivery.
*± revealed bleeding; enough placental separation producing fetal compromise and visible retroplacental clot after placental delivery.
*± revealed bleeding with maternal signs (uterine tetany, hypovolaemia, abdominal pain) and late stage fetal compromise or fetal death. 30% of these women will develop DIC.

If the placental location is higher in the uterus, or if the bleeding is more central and the margins of the placenta remain attached to the underlying uterus, blood may not escape into the vagina. Thus the amount of vaginal bleeding is extremely variable, from none to heavy.
PATHOPHYSIOLOGY

Placental separation is initiated by hemorrhage into the decidua basalis with formation of a decidual hematoma. The resulting separation of the decidua from the basal plate predisposes to further separation and bleeding, as well as compression and destruction of placental tissue.

The inciting cause of placental separation is unknown. It has been postulated that it may be due to an inherent weakness or anomaly in the spiral arterioles. Blood may either dissect upward toward the fundus, resulting in a concealed hemorrhage, or extend downward toward the cervix, resulting in an external or revealed hemorrhage.

CONCEALED HEMORRHAGE. Retained or concealed hemorrhage is likely when:

1. There is an effusion of blood behind the placenta but its margins still remain adherent.
2. The placenta is completely separated yet the membranes retain their attachment to the uterine wall.
3. Blood gains access to the amnionic cavity after breaking through the membranes.
4. The fetal head is so closely applied to the lower uterine segment that the blood cannot make its way past it.

Most often, however, the membranes are gradually dissected off the uterine wall, and blood sooner or later escapes.

DIAGNOSIS AND MANAGEMENT

Clinically, the diagnosis of a placental abruption is entertained if a patient presents with painful vaginal bleeding in association with uterine tenderness, hyperactivity, and increased tone.

The signs and symptoms of placental abruption are, however, variable. The most common finding is vaginal bleeding, seen in 80% of cases. Abdominal pain and uterine tenderness are seen in 66% of cases, fetal distress in 60%, uterine hyperactivity and increased uterine tone in 34%, and fetal demise in 15%.

The diagnosis of placental abruption is primarily a clinical one. Ultrasonography may detect only 2% of abruptions. Because placental abruption may coexist with a placenta previa, the reason for doing an initial ultrasonic examination is to exclude the latter diagnosis.

Diagnosis of Placenta Abruption

Clinical presentation:

* Bleeding: revealed/concealed, so clinical picture is important.
* Pain on the uterus and this increases in severity.
* Signs of shock (hypovolaemia): fainting and collapse.
* Hard tender uterus (uterine tetany).
* Difficult to palpate the fetal parts and to hear the fetal heart.
* The diagnosis is clinical.

*U/S: is to Confirm fetal viability, assess fetal growth & normality, measure liquor, do umbilical artery Doppler velocities. Exclude placenta praevia.

Management of the patient with an abruption includes careful maternal hemodynamic monitoring, fetal monitoring, serial evaluation of the hematocrit and coagulation profile, and delivery.
Intensive monitoring of both the mother and the fetus is essential because rapid deterioration of either one’s condition can occur. Blood products for replacement should always be available, and a large-bore (16- to 18-gauge) intravenous line must be secured. Red blood cells should be given liberally if indicated.

**Management of Placenta Abruption**

**Principle of management:**
- Early delivery (50% of abruption present in labour).
- Adequate blood transfusion.
- Adequate analgesia.
- Detailed maternal and fetal monitoring.
- Coagulation profile (30% develop DIC).
- **C/S:** distressed baby, severe bleeding, alive baby & not in advanced labour. Perinatal mortality rate is 15-20%.
- **Vaginal delivery:** very low gestation, dead baby, cervix is fully dilated (Ventouse delivery).
- **Conservative:** small abruption, well mother and fetus, if the gestational age < 34, give steroids.

**Conservative:** Time taken to achieve delivery depends on:
1/rate of the bleeding.
2/The rate of change in the clotting studies.
3/The clinical condition of the mother and fetus.
- **CTG:** twice/day.
- **Serial U/S and umbilical artery Doppler waveform.**
- **No conservative after 38 week’s gestation.**
- **Anti-D if the mother is rhesus positive.**
- **Anticipate PPH.**
- **In cases of previous CS, discuss hysterectomy.**

**MATERNAL-FETAL RISKS**

Abruption places the fetus at significant risk of hypoxia and, ultimately, death. The **perinatal mortality rate due to placental abruption is presently 35%,** and the condition accounts for 15% of third-trimester stillbirths. Fifteen percent of live-born infants have significant neurologic impairment.

**Placental abruption is the most common cause of DIC in pregnancy.** This results from release of thromboplastin from the disrupted placenta and the subplacental decidua into the maternal circulation, causing a consumptive coagulopathy. Clinically significant DIC complicates 20% of cases and is most commonly seen when the abruption is massive or fetal death has occurred.

**Hypovolemic shock and acute renal failure due to massive hemorrhage may be seen with a severe abruption if hypovolemia is left uncorrected. Sheehan’s syndrome (amenorrhea as a result of postpartum pituitary necrosis) may be a delayed complication resulting from coagulation within the portal system of the pituitary stalk.**
**Coagulation abnormalities**

*Hypofibrinogenemia
*Increasing levels of fibrin degradation products
*Decreasing platelet count
*Increasing prothrombin time and partial thromboplastin time
*Decreasing other serum clotting factor

Placental abruption is the most common cause of consumptive coagulopathy in pregnancy.

**Prevent Coagulopathy by:**

1. Restore blood volume by IV fluid (normal saline or Ringers lactate)
2. Treat the possible cause of coagulation failure
3. Give fresh blood or blood products for e.g. FFP (15ml / Kg body weight) (normal saline or Ringers lactate) to replace clotting factor or packed or sedimented RBC for RBC replacement or Cryoprecipitate to replace fibrinogen or platelet concentrates if platelet count is less than 20,000/ml.

Whole blood helps to replace not only volume but also oxygen carrying capacity. Fresh whole blood may replace RBCs and all procoagulants, but often difficult to obtain. Many experts recommend component therapy.

**Complications of abruptio placentae (summary)**

1. Premature delivery.
2. Fetal distress and death.
3. Haemorrhagic shock.
4. Acute renal failure: acute tubular or cortical necrosis.
5. DIC (release of tissue thromboplastin).
6. Uterine atony (Couvelaire uterus).
7. PPH.

**Follow Up After Delivery**

1. Check P.R., B.P. every 30 min for two hours then hourly for six hours then every 4 hours
2. Perform gentle uterine massage every 30 min. to prevent PPH
3. Check for vaginal bleeding
4. Check U.O. every 2 hours

**BLEEDING OF UNKNOWN ETIOLOGY**

In many cases of antepartum hemorrhage, no definite cause is ever found. The bleeding is usually minimal in amount. This diagnosis can be made only after exclusion of all other causes.

**Antepartum Haemorrhage**

- Exclude abruption, uterine rupture, placenta praevia with labour
  - Is she stable? - ?BP, pulse
- Check Abdomen - previous C/S scar, fundal height and uterine tenderness
- Check FH - Vaginal examination and ARM