Placental abruption is defined as separation of the placenta from the decidua basalis before delivery of the fetus. Bleeding occurs from the exposed decidual vessels, and may be extensive. However, because haemorrhage is often occult - with blood collecting around the placenta and fetus or in the myometrium and broad ligaments, the amount of blood lost is easily underestimated.

Fetal distress occurs because of loss of area for maternal-fetal gas exchange. Abruption is an important cause of intrauterine growth retardation, premature labour and fetal death.

**Incidence of placental abruption**

In a large retrospective study in Sweden on 894,619 births, the incidence was estimated at 0.5% of all pregnancies, with a perinatal mortality of 20%.

The cause of abruption is not known, but several factors are known to be associated which include:

- Trauma
- Chronic hypertension
- Premature rupture of the membranes
- Pre-eclampsia and eclampsia
- History of previous abruption (the risk is increased ten times).
- Advanced maternal age and parity
- Cocaine use
- Smoking
- Black ethnic origin
- Some uterine and fetal malformations
- Thrombophilia

**Clinical Presentation**

Placental abruption usually arises unexpectedly. Classically it presents with vaginal bleeding, severe, intense abdominal pain, uterine contractions, and intrauterine death of the fetus. Proteinuria and hypertension may occur but may be a secondary manifestation. There may be haemorrhagic shock if there is a large retroplacental haematoma - when the uterus may contain more than 1500mls of blood. The amount of vaginal bleeding may be very much less than the true blood loss. This classical presentation is present in less than one third of cases, and placental abruption can therefore present as a threat of premature delivery or acute fetal distress or an unexplained fetal death. In Lille our experience of 102 placental abruptions out of 18,082 deliveries in 4 years (0.56%), only 19 presented with classical clinical symptoms. 45 were diagnosed by examination of the placenta and 36 were suspected before or during delivery, either during Caesarean section for fetal distress or premature labor associated with vaginal bleeding.

Placental abruption can be difficult to diagnose especially if associated with a placenta praevia and can complicate this diagnosis in 4% of cases. Ultrasound examination often shows the presence of a retroplacental haematoma, but a normal ultrasound does not exclude the diagnosis.

Abruption may be missed because the clinical signs are hidden by the onset of labour associated with a hyperkinetic and hypertonic uterus. Epidural analgesia for labour may also obscure the symptoms. The diagnosis is sometimes made by examination of the placenta after a normal vaginal delivery or at caesarean section when a haematoma may be found. Alternatively, severe cases with fetal death and disseminated intravascular coagulation (DIC) have been described in which the presentation was a failure to progress in labour.

**Classification of abruption**

Taking into account the differences in clinical presentation, two classifications are suggested. The classification by Page has four stages;

- **Stage 0**: a diagnosis purely on pathology without symptoms.
- **Stage 1**: quiescent form with a live baby.
- **Stage 2**: mild form with onset of clotting problems.
- **Stage 3**: severe form with coagulation defects and fetal death in utero.

**Sher’s classification has 3 stages:**

- **Stage I** is mild with unexplained vaginal bleeding and a retrospective diagnosis of a small haematoma post-partum.
- **Stage II** is the intermediate form with a hypertonic uterus and a live baby.
- **Stage III** is the severe form with an intrauterine death, subdivided into IIIA without coagulopathy and stage IIIB with coagulopathy.

**Are there any warning signs of a placental abruption?**

No symptoms predictive of placental abruption have been found. However elevation of fibrin and fibrinogen degradation products (FDPs) in the last few days prior to abruption have been detected. D-dimers (the products of the degradation of stabilised fibrin), have been shown to be significantly elevated in patients who subsequently suffer an abruption between 32 and 40 weeks. A persistent increase in the fetal heart rate has also been associated with subsequent abruption. In addition two other parameters have been shown to be present in 60% of cases in the weeks before the event: lowering of the maternal plasma volume (which appears as an increase in haematocrit) and the presence of a notch on the uterine artery Doppler curve. However in 30% of cases neither of these abnormalities are found and so their value as predictors of abruption are limited.

**Complications of Abruption**

The major complications are:

- Haemorrhagic shock
- Disseminated intravascular coagulation (DIC )
Most cases however will require prompt delivery, and the preferred scalp electrode should be placed. Fetal heart rate should be monitored and where available a fetal monitoring of blood pressure, heart rate and urine output. The left lateral tilt should be ensured and there should be regular and coagulation screen. The mother should be given oxygen, a crossmatching and for measurement of haemoglobin, haematocrit bore intravenous cannulae should be sited and blood taken for or maternal compromise, the pregnancy may be allowed to

Delivery of the fetus and placenta is the definitive treatment. Anaesthetic management of placental abruption

is sometimes possible to inhibit uterine contractions to allow distress syndrome. Where there are no signs of fetal distress, it and mortality. As many as 50% of these babies have respiratory distress syndrome. Where there are no signs of fetal distress, it is sometimes possible to inhibit uterine contractions to allow further fetal maturation. However the advantages of this must be balanced against the risks of giving tocolytic drugs and of intrauterine fetal death.

Anaesthetic management of placental abruption

Delivery of the fetus and placenta is the definitive treatment. However if the degree of abruption is minor, and there is no fetal or maternal compromise, the pregnancy may be allowed to continue, to allow fetal lung maturation.

If the diagnosis of a significant abruption is suspected, 2 large bore intravenous cannulae should be sited and blood taken for crossmatching and for measurement of haemoglobin, haematocrit and coagulation screen. The mother should be given oxygen, a left lateral tilt should be ensured and there should be regular monitoring of blood pressure, heart rate and urine output. The fetal heart rate should be monitored and where available a fetal scalp electrode should be placed.

Most cases however will require prompt delivery, and the preferred route of delivery depends on several factors.

Vaginal delivery is recommended when the fetus is dead. This avoids the risks of caesarean section in a patient who may be hypovolaemic and coagulopathic. It may also be chosen where there is no fetal distress and the cervix is favourable. Epidural analgesia may be provided for these patients when clotting studies are normal, and there is no evidence of hypovolaemia.

Some studies have shown an increase in survival rate when the baby is delivered by caesarean section, and this is the most common mode of delivery where there is acute fetal distress. General anaesthesia is preferred for most of these cases and management of haemorrhage and associated hypovolaemia together with coagulopathy is critical.

Aggressive volume resuscitation is required with crystalloid or colloid, being guided by heart rate, blood pressure and urine output. Central venous pressure monitoring is also often helpful in managing severe haemorrhage. A line can be placed via an antecubital or an internal jugular vein (sites which can be compressed if there is haemorrhage from accidental arterial puncture) if the clotting is abnormal. Transfusion of cross-matched blood is necessary to keep the haemoglobin above 7gdl.

Coagulopathy should be corrected, ideally guided by clotting studies where these are available. The coagulopathy is most frequently due to disseminated intravascular coagulation with fibrinolysis predominating. However where large volumes of fluids and/or blood have been transfused, there may also be a dilutional element to it.

The object during caesarean or vaginal delivery is to try to make the blood coagulate at the moment of delivery. This can be done by various methods and no one has proved to be superior than the others: infusion of 20ml/kg of fresh frozen plasma, the infusion of 0.1g/kg of fibrinogen or the use of antifibrinolytic agents, particularly aprotinin, which is a plasmin inhibitor but also retains anticoagulant properties. Aprotinin may be given as up to 500,000 kallikrein units immediately by slow intravenous injection, followed by 200,000 units every hour by continuous intravenous infusion until haemorrhage is controlled. In the presence of DIC up to 1,000,000 units or more may be necessary. Persistent haemorrhage after delivery may be due to the coagulopathy. However it may also be due to failure of the uterus to contract down once it is empty, which occurs more commonly in these patients. An oxytocin infusion should be commenced after delivery (20-40units in 500mls of saline over 4 hours). Persistent uterine atony requires administration of other drugs such as ergometrine and 15methyl PGF2 alpha (Hemabate).

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Conclusion

Placental abruption is a complication of 0.25 - 1% of all pregnancies and occurs in 4% of those with severe pre-eclampsia. It occurs suddenly and is often unexpected. Its clinical presentation may be mistaken for isolated vaginal bleeding, fetal distress or labour with a hypertonic, hyperkinetic uterus.

The apparent blood loss may greatly underestimate the true amount of haemorrhage which is usually due to a retroplacental haematoma. Disseminated intravascular coagulation may also be present. Caesarian section may reduce the risk of perinatal mortality in the case of placental abruption with a live baby but vaginal delivery is recommended if the baby is dead. General anaesthesia is usually required because of the frequent presence of maternal hypovolaemia due to haemorrhage, the presence of a coagulopathy, and the potential for further intra-operative bleeding.

Further Reading

Dear Reader

If you would like to receive Update in Anaesthesia please write to: Dr C Collins, Secretary, World Anaesthesia, Royal Devon and Exeter Hospital (Wonford), Exeter EX2 5DW, UK. The fax number is +44 1392 402472. Alternatively contact the editor by email - iain.wilson5@virgin.net When writing please include your name, address, email (if available), your title and role, and a few details about your hospital and work. If you would like extra copies of Update to distribute to other anaesthetists, please let us know how many you require, and the names of the readers.

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http://www.anesthesiology.org/      Animation of gas flows in an anaesthesia machine

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