Antepartum Haemorrhage

R. Giordano A. Cacciatore P. Cignini R. Vigna M. Romano

Summary

Antepartum haemorrhage (APH) defined as bleeding from the genital tract in the second half of pregnancy, remains a major cause of perinatal mortality and maternal morbidity in the developed world. In approximately half of all women presenting with APH, a diagnosis of placental abruption or placenta praevia will be made; no firm diagnosis will be made in the other half even after investigations. In cases presenting with APH, the evaluation consists of history, clinical signs and symptoms and once the mother is stabilized, a speculum examination and an ultrasound scan.

- · Causes include:
- Abruptio placentae
- Placenta praevia
- APH of indeterminate origin
- Vasa praevia
- · Bleeding from the lower genital tract

Placental abruption

Usually the placenta is situated in the upper uterine segment. Placental abruption is the premature separation of a normally situated placenta from the uterine wall, resulting in haemorrhage before the delivery of the fetus. It occurs in around one in 80 deliveries and remains a significant source of perinatal mortality and morbidity. Incidence

Recent large epidemiological studies report an incidence ranging from 5.9 to 6.5 per 1000 singleton births and 12.2 per 1000 twin births. Perinatal mortality is reported to be 119 per 1000 births complicated by abruption. The risk of abruption recurring in a subsequent pregnancy is increased as much as 10-fold.

Pathology and aetiology

The precise cause of abruption is unknown. Abruption arises from haemorrhage into the deciduas basalis of the placenta, which results in the formation of haematoma and an increase in hydrostatic pressure leading to separation of the adjacent placenta. The resultant haematoma may be small and self-limited or may continue to dissect through the decidual layers. However, the bleeding may be in whole or in part concealed, if the haematoma does not reach the margin of the placenta and cervix for the blood loss to be revealed. Therefore the amount of revealed haemorrhage poorly reflects the degree of blood loss. The bleeding may infiltrate the myometrium resulting in so-called Couvelaire uterus.

A causal relationship between hypertension and abruption is controversial. Most explanations implicate vascular or placental abnormalities, including increased fragility of vessels, vascular malformations, or abnormalities in placentation. The absence of transformation from muscular arterioles to low-resistance, dilated vessels as in normal

pregnancy and the lack of trophoblastic invasion of uterine vessels is thought to result in decreased placental blood flow and dysfunctional endothelial responses to vasoactive substances. These abnormal placental vessels may predispose to ischaemia and rupture of involved vessels, thus causing placental abruption. Placental abruption is seen more often in gestational hypertensive disease, advanced maternal age, increasing parity, the presence of multiple gestations, polyhydramnios, chorioamnionitis, prolonged rupture of membranes, trauma, and possibly thrombophilias. Potential preventable risk factors include maternal cocaine and tobacco use. Unexplained elevated maternal serum alpha-fetoprotein (MSAFP) levels in the second trimester is associated with pregnancy complications such as placental abruption.

Clinical presentation

The diagnosis of placenta abruption is made clinically and then confirmed by evaluation of the placenta after delivery. It presents classically with vaginal bleeding, abdominal pain, uterine contractions and tenderness. On clinical examination, the uterus is irritable, with increased baseline tone. There may be evidence of fetal distress. In severe cases, the mother may show cardiovascular decompensation with evidence of hypovolaemia. The fetal heart may be absent, and there is a serious risk of development of coagulopathy in the mother due to consumption of clotting factors. The clinical signs of blood loss are out of proportion to the amount of vaginal bleeding. Ultrasound is an insensitive and unreliable tool for detecting or excluding placental abruption, as negative sonographic findings are common with clinically significant abruptions. The diagnosis may be confirmed postpartum on gross examination of the placenta, which reveals a clot and/or depression in the maternal surface, known as a delle. In less severe cases, the diagnosis of placental abruption may not be obvious, particularly if the haemorrhage is largely concealed and it may be misdiagnosed as idiopathic preterm labour. The majority of fetal morbidity is thought to be due to prematurity, with low birth weight, fetal growth restriction, anaemia, and hyperbilirubinaemia significantly more common. Placental abruption cannot be eliminated as a potential diagnosis in the absence of vaginal bleeding, as haemorrhage may be retroplacental and concealed. Placental

abruption is concealed in 20–35% and revealed in 65-80% of cases.

In severe abruption, complications include haemorrhage requiring transfusion, disseminated intravascular coagulopathy (DIC), infection and rarely, maternal death. Couvelaire uterus may occur and occasionally may require hysterectomy. The incidence of stillbirth is related to the size of the abruption. Separation exceeding 50% of the placenta causes a marked elevation in stillbirth rate.

Management

Once placental abruption has been suspected, action should be swift and decisive because the prognosis for mother and fetus is worsened by delay. Treatment consists of initial resuscitation and stabilization of the mother, treatment of the abruption, and recognition and management of complications. It is individualized based on the extent of the abruption, maternal and fetal reaction to this insult, and gestational age of the fetus. Maternal resuscitation and treatment of hypovolaemic shock are a subject of a review in its own right, and will not be discussed further. For the purpose of management or abruption, Sher and Statland divided placental abruption into three degrees of severity.

These are mild (grade 1): not recognized clinically before delivery and usually diagnosed by the presence of a retroplacental clot; moderate (grade 2): intermediate, the classical signs of abruption are present but the fetus is still alive; and severe (grade 3): the fetus is dead and coagulopathy may be present.

There are three practical options for management:

- Expectant: in the hope that the pregnancy will contin-
- ue
- Immediate caesarean section

Rupture the membranes and aim at vaginal delivery In mild placental abruption, the bleeding may stop and the symptoms gradually resolve with satisfactory fetal monitoring and the patient can often be managed as an outpatient. The management of moderate or severe placental abruption is resuscitation, delivery of the fetus and observation for and correction of any coagulation defect that arises. This requires management in the labour ward with intensive monitoring of both mother and fetus. A trial of labour and vaginal delivery is recommended whenever tolerated by the maternal-fetal pair. Labour is usually rapid and progress should be monitored with continuous fetal heart rate assessment. If fetal distress is present then delivery should be expedited in the form of Caesarean section. Major abruption should be regarded as an emergency, requiring multidisciplinary input from the obstetrician, anaesthetist and haematologist. A fulminant maternal DIC can ensue within hours of a complete abruption and delivery should be effected, as it is the only means with which to halt the DIC. Replacement of blood and its components should begin before surgery. Abruption also places the patient at risk of severe postpartum haemorrhage. This is as a result of a combination of uterine atony and coagulation failure. Invasive monitoring with arterial lines and central venous access may be necessary, and patients are best treated in the high-dependency unit. Urine output should be closely monitored, as renal failure is a potential complication. Multiple studies have shown expectant management with or without tocolytics to be safe and effective in a select population of patients with preterm placental abruption. In some observational studies, tocolysis allowed a median delay of delivery of several days without increasing neonatal or maternal morbidity, including the need for transfusion or delivery by Caesarean section. However, in the absence of randomized controlled trials, the benefits of tocolysis remain uncertain.

Placenta praevia

Placenta praevia is defined as a placenta that lies wholly or partly within the lower uterine segment. The prevalence of clinically evident placenta praevia at term is estimated to be approximately 4 or 5 per 1000 pregnancies.

Classification

Classification of placenta praevia is important in making management decisions because the incidence of morbidity and mortality in the fetus and mother increases as the grade increases.

Classically, placenta praevia is divided in four types or grades (Table 1). Types I and II are regarded as minor, and types III and IV as major degrees of placenta praevia. Care must be taken not to confuse these grades with grades of placental maturity.

The classification is difficult to use in practice, because the definition of lower uterine segment is more conceptual than anatomical. In any case, with the availability of ultrasound, this classification has become obsolete. Currently, the condition is most commonly diagnosed on ultrasound examination. Ultrasound remains the method of choice because it is relatively cheap and readily available.

Aetiology and associated factors

Placenta praevia is caused by implantation of the blastocyst low in the uterine cavity. Factors associated with the development of placenta praevia include increasing maternal parity, advancing maternal age, increasing placental size (multiple pregnancy), endometrial damage (previous dilatation and curettage), previous Caesarean section, uterine scars and pathology (previous myomectomy or endometritis), placental pathology (marginal cord insertions and succenturiate lobes), previous placental praevia, and curiously, cigarette smoking.

Clinical presentation and diagnosis

Most women in the UK will have a routine scan at 21–23 weeks (anomaly scan). The placenta will be low-lying in some, necessitating a repeat scan later in pregnancy, typically at 34–36 weeks. Women classically present with minor degrees of painless vaginal bleeding in the absence of labour pains. The bleeds tend to occur due

to the formation of the lower uterine segment. Fetal malpresentation or unstable lie is found in one-third of cases and many cases of placenta praevia do not bleed until the onset of labour. The diagnosis of placental praevia is most commonly made on ultrasound examination. Up to 26% of placentas are found to be low lying on ultrasound examination in the early second trimester.

Several studies have demonstrated that unless the placental edge is at least reaching the internal cervical os at midpregnancy, placenta praevia at term will not be encountered.

Transvaginal ultrasound is safe in the presence of placenta praevia, and is more accurate than transabdominal ultrasound in locating the placental edge. Ultrasound has been used to observe and document the phenomenon of placental migration from the lower uterine segment. It is thought that this process is not a true migration of placental tissue but, rather, a degeneration of the peripheral placental tissue that receives a suboptimal vascular supply and has slow placental growth in better perfused uterine areas at the same time, so-called placental trophotropism.

None of the cases presented with placenta praevia at term, unless the placental edge overlapped the internal os at least by 1 cm at the mid-trimester scan. There was a minimal placental migration rate of 0.1 mm/week in this group. In contrast, cases where the placenta eventually migrated away from the internal os showed a mean rate of migration of 4.1 mm/week. Placental edge overlapping the internal os at the mid-trimester scan, and a thick placental edge (where the angle between the placental edge and the uterine wall is o1351) are known to be associated with reduced likelihood of placental migration. In addition, those cases, where the placentas failed to migrate were associated with increased rates of interventional Caesarean delivery and manual placental removal, and a higher prevalence of placenta accreta.

Table 1 Classification of placenta praevia.

- Type I The placenta encroaches into the lower uterine segment and lies within 5 cm of the internal cervical os
- Type II The placenta reaches the cervical os but does not cover it
- Type III The placenta covers the cervical os but the placental site asymmetric with most of the placenta being on one side of the cervical os
- Type IV The placenta is centrally located over the cervical os

Management

The management of placenta praevia depends upon clinical presentation, severity of bleeding and degree of praevia. Currently, the diagnosis of placenta praevia is made using ultrasound. Most cases presenting with APH would already be known to have a low-lying placenta. Those cases, in which the placenta was low-lying at the time of routine anomaly scan should receive a repeat ultrasound scan at 36 weeks to check placental location. Some of these cases will present with antepartum bleeding. Initial haemorrhages, referred to as 'warning haemorrhages' are often small and tend to stop

spontaneously. Delivery may be needed for severe, intractable or recurrent bleeding. Fetal morbidity is associated with iatrogenic prematurity. In the report of confidential enquiries into maternal mortality over 2000-2002 in the UK ('Why mothers die 2000-2002'), there were 17 maternal deaths due to haemorrhage. Four out of these 17 deaths were due to placenta praevia. Controversy surrounds the antepartum management of those cases found to have a low-placenta at the anomaly scan, particularly the ones who have never had antepartum bleeding. Moreover, many women will be admitted with vaginal bleeding due to known low-lying placenta, but the bleeding would stop spontaneously, and not recur for several days. Current guidelines by the Royal College of Obstetricians and Gynaecologists (RCOG) recommend that such women be kept admitted to the hospital. This advice is based on a small randomized trial that showed no difference between inpatient and outpatient management of cases of placenta praevia. However, the authors of the RCOG guideline felt that uncommon, but potentially serious, maternal complications are unlikely to come to light with a trial with small numbers. The recommendation for inhospital management is not based on the presence of evidence of benefit of hospitalization, but due to absence of large good quality trials. Traditionally, Caesarean section has been the recommended mode of delivery for major placenta praevia (type III and IV), whereas for minor praevia (type I and II) an attempt at vaginal delivery was deemed appropriate. Until recently, no evidence-based protocol was available for management of delivery guided by the findings of the ultrasound scan. We reported that when the placental edge was within 1 cm of the internal cervical os within 2 weeks of delivery, all patients required a Caesarean delivery due to bleeding. We proposed that cases with placental edge to internal os distance of less than 2 cm be referred to as major placenta praevia. An elective Caesarean section should be recommended. In contrast, if the placental edge to internal cervical os distance was 2-3.5 cm at the last ultrasound scan within 2 weeks of delivery, the likelihood of achieving a vaginal delivery was at least 60%. It is recommended that these cases be still referred to as low-lying placenta, because the risk of postpartum haemorrhage remains high in this group. An attempt at vaginal delivery is appropriate. RCOG guidelines recommended that any women going to the operation theatre with known major placenta praevia should be attended by an experienced obstetrician and anaesthetist, with consultant presence available, especially if these women have previous uterine scars, an anterior placenta or are suspected to be associated with placenta accreta. Four units of cross-matched blood should be kept ready, even if the mother has never experienced vaginal bleeding. Delivery of women with placenta praevia should not be planned in units where blood transfusion facilities are unavailable. The choice of anaesthetic technique for Caesarean sections is usually made by the anaesthetist conducting the procedure.

Placenta accreta

Although placenta accreta is very rare (0.004%) in women with a normally situated placenta, it occurred in 9.3% of women with placenta praevia according to data

from Southern California. Ultrasound features of placenta accrete in second and third trimesters include visualization of irregular vascular sinuses with turbulent flow, abnormalities of the bladder wall on ultrasound inspection and possibly myometrial thickness of less than 1 mm. Absence of the sono-luscent space between myometrium and the placenta is not a reliable sign. Colour Doppler and magnetic resonance imaging are not yet completely sensitive and specific tests for the diagnosis of placenta accreta.

When a probability of placenta accreta is raised, multidisciplinary input involving the patient and the family, the anaesthetist, obstetrician and the sonographer should be arranged. Advance planning should be made for management of delivery. The options are subsequent hysterectomy after delivery or leaving the placenta in-situ in order to reduce surgical complications and blood loss. Of the four maternal deaths due to placenta praevia in the triennium 2000–2002, all had at least on previous Caesarean, and three had a history of placenta accreta.

Bleeding of uncertain origin

The exact cause of bleeding in late pregnancy is unknown in about half of cases. The woman typically presents with painless vaginal bleeding without ultrasound evidence of placenta praevia. Placenta praevia can be excluded by an ultrasound scan, but the diagnosis of placental abruption is based on clinical signs and symptoms, and is difficult to confirm in mild cases. Approximately 15% of women with unexplained APH will go into spontaneous labour within 2 weeks of the initial haemorrhage. In the majority of cases, the bleeding is mild and settles spontaneously. Further management will either be expectant or delivery will be expedited. If pregnancy is beyond 37 weeks gestation and the bleeding is recurrent or associated with fetal growth retardation, labour induction is the management of choice. If episodes of bleeding are recurrent and significant, there may be a need for immediate delivery even if the gestation is below 37 weeks. If a policy of expectant management is adopted, fetal well-being should be monitored. Once the bleeding has settled and the woman has been observed as an inpatient for 24-48 h, it may be considered safe to allow her to be managed as an outpatient. If the gestational age is below 34-36 weeks, antenatal steroids should be administered in view of the risk of preterm delivery. In a small proportion of cases where placenta praevia and placental abruption have been excluded, a cause may still be found. They include 'show', cervicitis, trauma, vulval varicosities, genital tumours, haematuria, genital infections and vasa previa. Many of these conditions are evident on the initial speculum examination.

Vasa praevia

Vasa praevia is the presence of unsupported fetal vessels below the fetal presenting part, where the cord insertion is velamentous. It is rare, but consequences are disastrous, if not prenatally diagnosed. Vasa praevia has an incidence of approximately one per 6000 deliveries. Classically, vaginal bleeding follows amniotomy with subsequent fetal bradycardia suggests vasa praevia.

Immediate Caesarean delivery is needed if fetal blood is confirmed to be present in the vaginal bleeding. Oyelese et al. demonstrated the importance of prenatal diagnosis. In the group where prenatal diagnosis had been made, 97% infants survived, as opposed to only 44% where the diagnosis had not been made before birth. Echogenic parallel or circular lines near the cervix representing the umbilical cord, seen by grey-scale ultrasound, should raise the possibility of vasa praevia. The diagnosis of vasa praevia can be confirmed by Doppler and endovaginal ultrasound studies if aberrant vessels over the internal cervical os are suspected. Several reports have linked vasa praevia to invitro fertilization. The diagnosis should be kept in mind in cases of in-vitro fertilization pregnancies with low placenta, and cases where the placenta had been low-lying at the midtrimester scan, but has receded from the internal os on repeat assessment. Delivery by elective Caesarean section after fetal pulmonary maturity is established and prior to the onset of labour should be recommended unless obstetric complications supervene.

Practice points

The cause of APH remains undetermined in about half of the cases.

Diagnosis of placental abruption is clinical, whereas that of placenta praevia, based on an ultrasound scan.

In cases of abruption presenting with intrauterine death, at least a 2 unit blood transfusion should begin because average blood loss is about 1 l.

Unless the placental edge overlaps the internal os by at least 1.0 cm at 21–23 weeks scan, placenta praevia at term will not be encountered. A repeat scan at 34–36 weeks should be organized.

Caesarean section for placenta praevia should involve the most senior available staff in the anaesthetic and obstetric service. At least 4 units of blood should be crossmatched.

The possibility of placenta accreta should be kept in mind in cases of placenta praevia. Absence of an echoluscent line behind the placenta is not a reliable sign. Sonographic visualization of irregular sinuses with turbulent flow in the placenta is the most reliable sign.

Antepartum identification of vasa praevia leads to significant improvement in perinatal mortality.

A multi-disciplinary massive obstetric haemorrhage protocol should be available in all units. It should be regularly updated and rehearsed in conjunction with the blood bank.

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