Antepartum haemorrhage

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Abstract

The incidence of antepartum haemorrhage (APH) is reported as 3.5% of all pregnancies. It is an important cause of maternal and perinatal mortality. There has been a decline in maternal mortality due to APH due to placenta praevia, but the decline has been marginal in cases of placental abruption. No definite cause can be identified in at least half of cases of APH. The diagnosis of placental abruption is based on clinical presentation and on the examination of the delivered placenta. Major complications resulting from placental abruption require critical care in 5–7% of cases. Mid-pregnancy ultrasound scan is an effective screening tool for the detection of placenta praevia. Ultrasound-based criteria have been developed for managing cases of placenta praevia. The incidence of placenta accreta responsible for APH has increased 10-fold, mainly due to a rise in caesarean section rate. Extraplacental causes of APH can be diagnosed on careful speculum examination.

Keywords antepartum haemorrhage; placental abruption; placenta praevia

Antepartum haemorrhage (APH) is defined as bleeding into and/or from the genital tract in the second half of pregnancy but before delivery of the baby. The incidence of APH is reported as 3.5% and varies with age, parity and social status. The exact cause remains indeterminate in about half of cases, even after investigations. Of the known causes, placental causes contribute to over 90% of cases of APH; the rest are due to local and extraplacental causes. It continues to be an important contributor to perinatal mortality and is responsible for significant maternal and infant morbidity. Vaginal bleeding due to any cause in late pregnancy is associated with preterm labour and low birthweight babies.

Placental abruption

Placental abruption is defined as complete or partial separation of a normally situated placenta, before the delivery of the fetus. The term accidental haemorrhage was first introduced by Rigby in 1776. It has an incidence of 0.5–1.3% of all pregnancies. In large studies, 30–35% cases of antepartum bleeding are contributed to by placental abruption.

Acute inflammation-related conditions and chronic inflammation or vascular dysfunction are two potential mechanisms of placental abruption. There is some evidence to show that the acute inflammatory pathway is dominant in preterm abruptions, whereas chronic inflammation/vasculopathy plays a role in abruption throughout gestation. Inflammatory processes are mediated by cytokines such as interleukin (IL)-1 and tumour necrosis factor (TNF-α), which produce matrix metalloproteinases in trophoblast. The metalloproteinases cause destruction of extracellular matrices and disruption of cell–cell interactions, leading to premature detachment of the placenta.

Fetal complications in placental abruption occur due to prematurity and hypoxia, and maternal complications due to blood loss. Remodelling of uterine arteries is necessary for adequate placentation. Vascular dysfunction leads to reduced invasion of cytotrophoblasts and is associated with the risk of development of placental abruption. This can be detected in the first half of pregnancy from an abnormal pattern of the uterine artery waveforms in Doppler studies. It is postulated that arterial spasm followed by relaxation and subsequent venous engorgement and arteriolar rupture into the decidua basalis with the accumulating blood clot causes the separation of the placenta. The blood can escape through the cervix into the amniotic cavity, producing blood-stained liquor, and into the myometrium, causing sustained tonic uterine contraction.

Risk factors for placental abruption are summarized in Table 1. Other risk factors such as assisted conception, polyhydramnios, oligohydramnios, maternal diabetes, uterine trauma, external cephalic version, short umbilical cord, velamentous insertion, coagulation defects, amniocentesis and placental biopsy have all been historically reported to cause placental abruption. However, these have not been conclusively proven to be risk factors in large series of placental abruption.

The only screening test with any efficacy for the prediction of abruption remains a positive uterine artery Doppler screen in mid-pregnancy. Figures 1 and 2 demonstrate normal and abnormal uterine artery Doppler flow velocity waveforms, respectively. However, this test is of limited clinical utility due to the

Risk factors for placental abruption

Pre-pregnancy risk factors	Risk factors in the index pregnancy
Previous history	Parental smoking
Previous Caesarean section	Alcohol use
Uterine malformations	Placenta praevia
	Pre-eclampsia
	Chorioamnionitis
	Bleeding in the second or third trimester
	Prolonged prelabour rupture of membranes

Table 1

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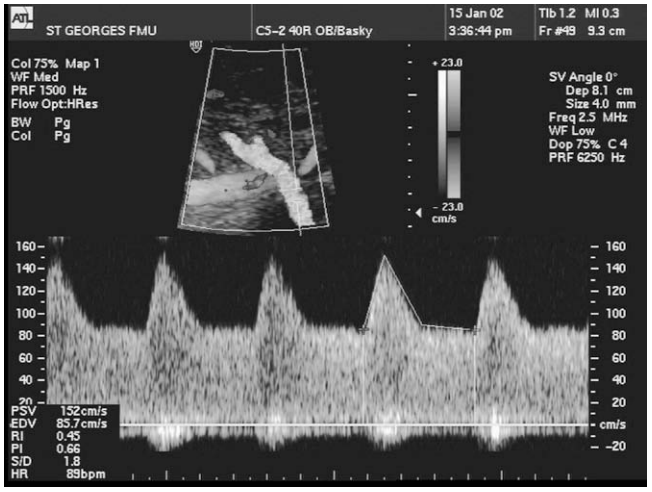

Figure 1 Normal uterine artery Doppler. Note the adequate diastolic flow. There is no diastolic notch.

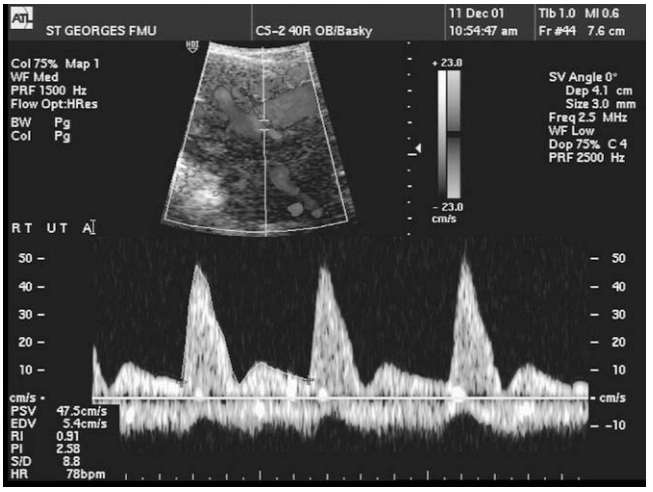

Figure 2 Abnormal uterine artery Doppler. Note the diastolic notching.

low prevalence of abruption and the high rate of false-positive results.

There is no known prevention. Placental abruption seems to represent a final common clinical event that arises from a variety of different causative pathways. A better understanding of these may lead to prevention strategies.

Clinical presentation

In mild cases, the diagnosis is made retrospectively after delivery, on placental examination. Otherwise, painful vaginal bleeding is a cardinal feature. However, vaginal bleeding is a symptom in 70–80% of patients. Half of cases occur after 36 weeks' gestation or in established labour. Abdominal pain and absent or reduced fetal movements are the predominant symptoms. Signs of significant blood loss, like pallor and tachycardia, may be present, and sometimes the patient may present in hypovolaemic shock. The presence of hypertension may mask true hypovolaemia, but increasing abdominal girth or rising fundal height should arouse suspicion of significant concealed haemorrhage. In early cases, uterine irritability with or without uterine tenderness are present. In severe placental separation the uterus is hard, tender to touch and it is difficult to palpate fetal parts. Signs of fetal distress are very common if the fetus is still alive. Blood-stained liquor is present with ruptured membranes.

A useful classification of placental abruption has been described:

- **Grade I** – diagnosis of abruption was made retrospectively. Average retroplacental clot volume was 150–500 ml.
- **Grade II** – APH with classical clinical features, live fetus. The average retroplacental clot volume was 150–500 ml; 25% of patients can have a volume >500 ml. A vast majority (92%) presenting in this grade had fetal heart rate (FHR) abnormalities.
- **Grade III** – cases presenting with fetal demise. Virtually all maternal mortalities occurred in this group. This is further subdivided into:
  - **Grade III (A)** – fetal demise with coagulopathy;
  - **Grade III (B)** – presenting with fetal demise only.

Diagnosis and management

Diagnosis is based on clinical presentation and examination of the delivered placenta. Ultrasound examination is not a sensitive method of diagnosis but is useful in excluding placenta praevia. A negative ultrasound does not rule out abruption.

Management should be individualized and based on the severity of the abruption and the gestational age at which it occurs. When it occurs at or near term and maternal and fetal condition is reassuring, conservative management is reasonable. This involves induction of labour by amniotomy and syntocinon infusion with the goal of achieving a vaginal delivery. Adequate blood products and multidisciplinary team involvement are mandatory. If a patient presents with Grade III abruption, immediate transfusion of at least 2 units of blood is recommended, irrespective of the visible blood loss. Delivery should be achieved by the least traumatic route. The role of caesarean section in placental abruption is difficult to define. Evidence of fetal distress with clinical features of abruption placentae should be treated as an obstetric emergency. It is an indication for urgent caesarean section for a viable fetus. Birth of an hypoxic/acidaemic fetus should be anticipated. Expert neonatal back-up is necessary in view of the possibilities of hypoxia and prematurity.

Caesarean section can be life-saving when conservative treatment fails to control the bleeding, or when the maternal condition is rapidly deteriorating. When there is fetal demise, coagulation screen should be carried out, with blood transfusion or replacement of coagulation factors as needed. Confirmation of fetal presentation, care in an obstetric high-dependency unit, involvement of other specialities and quick delivery by the least traumatic route are recommended. It is important to anticipate postpartum haemorrhage (PPH).

Management of complications

Delay in treatment of placental abruption increases the risk of hypovolaemic shock, coagulation failure and PPH. In case of concealed placental abruption, hypovolaemia and shock may be out of proportion to the blood loss evident before delivery. Underestimation of blood loss can result in hypotension, which causes underperfusion of vital organs such as the kidneys, brain and gut. Central venous pressure monitoring, fluid and electrolyte replacement and blood transfusion of at least 2 units is required in cases of fetal demise. Adequate renal perfusion should result in urine output of 30 ml/h.

Disseminated intravascular coagulation

Progressive separation of the placenta releases thromboplastin-like substances which activate the coagulation cascade. This activation consumes coagulation factors leading to secondary thrombocytopenia. The presence of a large retroplacental clot is followed by formation of fibrin degradation products (FDPs) and D-dimers. Fibrin is deposited in the small vessels of the organ systems, causing ischaemic damage throughout the microvasculature. Tissue hypoxia coupled with microangiopathic haemolysis produces most of the known complications of disseminated intravascular coagulation (DIC), such as renal failure, pulmonary hypoperfusion, PPH, postpartum pituitary necrosis and overt haemorrhagic diathesis. The clinical signs of DIC include bleeding from multiple sites, like the gums, nose, venipuncture, surgical incision, episiotomy and the retroperitoneal space.

Laboratory investigations reveal prolonged prothrombin time (PT) and partial thromboplastin time (PTT), low fibrinogen, presence of FDPs and thrombocytopenia. Overt hypofibrinogenaemia with fibrinogen levels below 100 ng/dl and FDPs above 10 μg/ml confirm the diagnosis of coagulopathy. Monoclonal antibodies to D-dimers can detect fibrinogen derivatives. Treatment is aimed at eliminating the cause (removal of the source of the tissue thromboplastin) by delivering the fetus and placenta. Replacement of red cells, plasma and coagulation products, care in the obstetric high dependency unit, monitoring by laboratory investigations, e.g. fibrinogen levels, FDPs and platelet counts. In cases of uterine inertia, caesarean section may be needed and, if haemorrhage is severe, internal iliac artery ligation or even hysterectomy may be required. General anaesthesia is preferred in such cases and platelet concentrates and FFPs should be available when surgery is required in cases of coagulopathy.

Renal failure

Acute renal failure can occur when the treatment for hypovolaemia is delayed or incomplete (pre-renal), or due to renal damage (renal). Renal damage is most often due to acute tubular necrosis which is reversible. The severe form of renal damage is due to cortical necrosis. This is less common and requires early dialysis. An initial period of oliguria may be followed by polyuria, and there is a possibility of acid–base and electrolyte imbalance. Electrolytes, urea and creatinine are measured every 4 hours. Blood gases may require periodic assessment.

Postpartum haemorrhage

In 2% of cases with placental abruption, the uterus becomes hypotonic and resistant to oxytocics. PPH secondary to abruption placentae is managed by fluids, blood transfusion, oxytocics and bimanual compression. Intrauterine Rusch balloon insertion, brace sutures (such as B-Lynch sutures) or uterine artery embolization may be required. Laparotomy is rarely needed for refractory cases for uterine artery and/or internal iliac artery ligation or hysterectomy as a life-saving procedure.

Placenta praevia

Placenta praevia is defined as the implantation of the placenta in the lower uterine segment. It occurs in approximately 4–5 in 1000 pregnancies at term. Traditionally, placenta praevia has been classified as follows:

- **Grade I** – placenta is in the lower segment but the edge does not reach the internal os.
- **Grade II** – lower edge of the placenta reaches the internal os but does not cover it.
- **Grade III** – placenta covers the internal os asymmetrically.
- **Grade IV** – placenta covers the internal os symmetrically.

This classification is based on digital palpation of the placenta as part of a double set-up examination. In the era prior to the availability of ultrasound, this was the only means of conclusive diagnosis of placenta praevia. Use of ultrasound has become routine in mid-pregnancy in all developed countries. This has made the above classification redundant. Modern management of placenta praevia is based on ultrasound findings of the placenta.

Risk factors for placenta praevia include previous uterine scars, smoking, maternal age over 35 years, grand multiparity, recurrent miscarriage, low socioeconomic status and infertility treatment. In addition to haemorrhagic complications, placenta praevia is associated with placental abruption, congenital malformations, abnormal presentations and preterm delivery.

Screening

It is common practice to perform an ultrasound examination in mid-pregnancy, the so-called anomaly scan. The placental location is routinely reported at this time. This examination is used as a screening test to predict the likelihood of placenta praevia at delivery. Placental edge to internal os distance of 2.0 cm or less is found in approximaately 2% of pregnant women undergoing a routine anomaly scan at 18–23 weeks. A placental edge overlapping the internal os by over 10 mm at 15–24 weeks has 100% sensitivity for a placenta praevia at birth. Therefore, unless the placental edge at least reaches the internal os at mid-pregnancy, placenta praevia at term will not be encountered. When the placental edge is more than 2.0 cm from the internal os, migration always occurs to the extent that a caesarean section for bleeding is not required. If the edge is less than 2.0 cm from the internal cervical os, placental migration can occur in 88.5% of cases. Only 5% of these women undergoing routine scan at 18 weeks are found to have a low lying placenta at 32 weeks and one-third of these have placenta praevia at term. The mean rate of migration is 5.4 mm/week. Placental migration is more likely when the placental edge is thin. When the placental edge is thick, there is a significantly higher rate of APH, abdominal delivery and adherent placenta. Active migration of the placenta is believed to be due to the formation of the lower segment and upward enlargement of the upper segment as pregnancy progresses. A second trimester low lying placenta which is centrally located over the cervical os is likely to persist as placenta praevia at term.

The term placenta praevia should only be used when the placental edge overlaps or is within 2.0 cm of the internal cervical os in late pregnancy. If the placental edge is located more than 2.0 cm but within 3.5 cm from the internal cervical os, the placenta should be termed as low lying. Attempt at vaginal delivery is appropriate, but the risk of PPH remains higher.

Clinical presentation

It presents as painless, unprovoked vaginal bleeding, which may start in the second trimester. More than 50% of episodes occur before 36 weeks' gestation. Placental abruption or labour causing abdominal pain may be associated in 10% of cases, causing

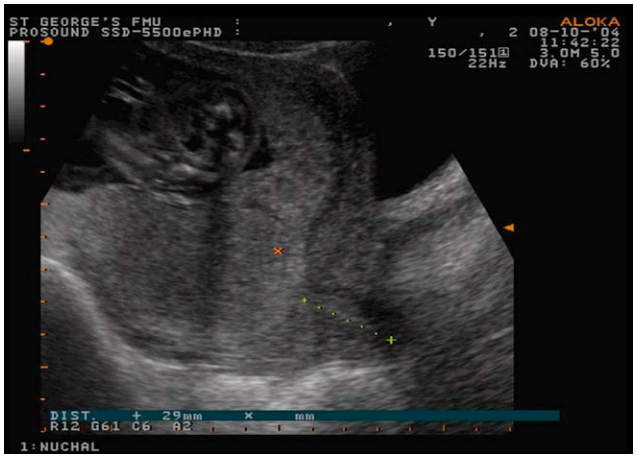

Figure 3 Abdominal ultrasound scan showing placenta covering the internal os.

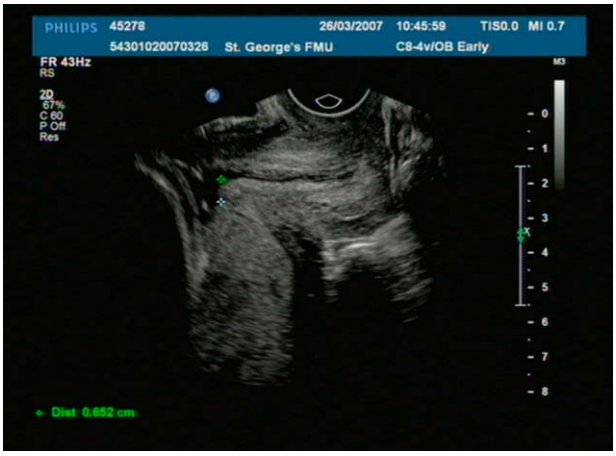

Figure 4 Transvaginal ultrasound scan showing placental edge very close to the internal os.

difficulty in diagnosis. Antepartum bleeding may be absent in 35–40% of cases with placenta praevia. Malpresentations like breech or transverse lie are present in 35% of cases.

Diagnosis and management

Placental localization with ultrasound has become the method of choice because of its safety, accuracy and convenience. Identification of a low lying placenta is recommended at the time of the routine 21–22 weeks' anomaly scan (Figure 3), an episode of APH or an abnormal presentation in the third trimester of pregnancy. All cases where the placenta is found to be low lying at anomaly scan should have a repeat scan at 36 weeks.

A diagnosis of placenta praevia on abdominal ultrasound should be confirmed by a transvaginal scan after any active vaginal bleeding has ceased, particularly in cases of posterior placenta praevia (Figure 4). The whole length of the cervix with the lower part of the uterus and the presence of the lower placental edge is visualized and assessed. The RCOG recommends that trial of vaginal delivery is appropriate if the placental edge to internal os distance is 2.0 cm or more, and a caesarean section is recommended if this distance is less than 2.0 cm. The likelihood of a vaginal delivery increases with increasing distance of the placental edge from the internal os. The new classification of placental location based on ultrasound should be as follows:

- **Normally situated placenta** – placental edge to internal os distance of over 3.5 cm at term.
- **Low placenta** – placental edge to internal os distance of 2.0–3.5 cm at term
- **Placenta praevia** – placental edge to internal os distance of less than 2.0 cm at term. The placental edge may overlap the internal os.

Conservative management

Conservative management of a woman with bleeding from placenta praevia depends upon the degree of haemorrhage and fetal maturity. Inhibition of uterine activity with tocolytics to prolong pregnancy can be considered, but is controversial. The benefits of corticosteroids in promoting fetal lung maturity are well established. All Rhesus-negative women should be given anti-D. Women stabilized following an episode of bleeding at any time in the third trimester are at high risk for further sudden unprovoked bleeding and delivery may be necessary in the interest of the mother and fetus. Surgical intervention is necessary when the mother's life is endangered. A caesarean section may be technically difficult with a low lying placenta. Intraoperative bleeding from the dilated veins and extension of the incision is more likely. PPH is reported in 8–10% of cases. Haemostatic sutures, balloon tamponade, internal iliac artery ligation or even an obstetric hysterectomy may be required in cases of intractable bleeding or placenta accreta.

Placenta accreta

Placenta accreta is an abnormally deep attachment of placental villi to the uterine wall with absence of the normal intervening decidua basalis and fibrinoid layer of Nitabuch. The incidence varies from 1 in 540 to 1 in 70 000 deliveries, with an average incidence of 1 in 7000. Three variants of the condition are generally recognized. In the most common form, placenta accreta is attached directly to the myometrium. Less commonly it may extend into the myometrium (increta) or through the entire myometrial layer (percreta).

The risk factors for placenta accreta include placenta praevia, maternal age over 35 years, grand multiparity, previous curettage, myomectomy, submucous myoma, Asherman syndrome, a short caesarean section to conception interval and a female fetus. The incidence of placenta accreta has increased 10-fold, probably because of the increase in caesarean section rate. The risk of placenta accreta in patients with one caesarean section is eight fold higher compared with an unscarred uterus, and is further increased four-fold with two or more caesarean sections.

Placenta accreta is associated with a 7% mortality rate as well as intrapartum and postoperative morbidity caused by massive blood transfusion, infection and adjacent organ damage. It is important to be aware of this complication as it is now possible to screen for this abnormality. The presence of multiple linear irregular vascular spaces within placenta lacunae is a diagnostic sign which has a high positive predictive value for placenta accreta as compared to diagnosis made in the absence of the retroplacental clear space, which has a false positive rate of 54.3%. Figure 5 shows an ultrasound scan of an anterior placenta praevia accreta.

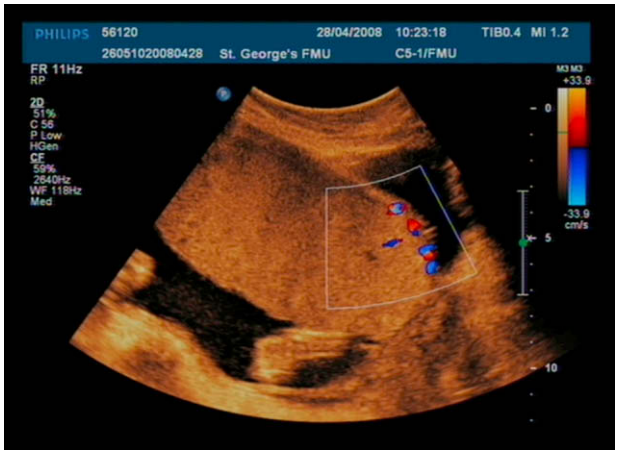

Figure 5 Ultrasound scan showing anterior low placenta. Colour flow mapping shows placenta accreta.

Identification of risk factors and using them as screening criteria for placenta accreta is important because diagnosis by ultrasound, Doppler and magnetic resonance imaging is now possible. In an emergency or where these diagnostic modalities are unavailable, awareness of the clinical risk factors can aid in diagnosis and management of placenta accreta. Proactive management includes availability of adequate blood products, a planned procedure with an experienced surgeon and patient counselling regarding a possible hysterectomy.

In most cases of placenta accreta, the principal concern is the control of haemorrhage. Conservative management includes ligation/embolization of uterine or internal iliac arteries. Women who have undergone hysterectomy have a reported mortality of 5.8–6.6%.

Extraplacental causes of antepartum haemorrhage

These cases are rare. Careful speculum examination is needed to find local causes such as cervical polyps or cancer, varicose veins of vulva and vagina, degenerating fibroid polyp and cervicitis as the source of bleeding. History of abnormal smears may be present.

Vasa praevia

The incidence of vasa praevia is 1 in 6000 deliveries. Antenatal diagnosis is possible and is associated with 97% fetal survival. However, the fetal mortality is 44% if antenatal diagnosis is not made. Pregnancies resulting from assisted conception are at an increased risk for vasa praevia. Vasa praevia should be looked for in particular if the placental edge covers the internal cervical os in mid-pregnancy but recedes later on. The fetal blood vessels unsupported by either the umbilical cord or placental tissue, traverse the fetal membranes of the lower uterine segment, resulting in vasa praevia. Echogenic parallel or circular lines near the cervix, representing the umbilical cord seen ultrasonically, need to be confirmed by colour flow mapping. Caesarian section at term is the choice of treatment.

Antepartum haemorrhage of indeterminate origin

APH is termed indeterminate when other causes of APH have been excluded. Placenta praevia is excluded by ultrasound and placental abruption by absence of the typical clinical features. A normal speculum examination excludes bleeding from local causes. So, it essentially is a diagnosis of exclusion. There is an approximately 1 in 5 risk of delivery in the next 2 weeks. Prophylactic steroid administration should be considered where appropriate. There is a higher risk of abruption and a small baby, and an increase in the perinatal mortality. A recent study reported a small increase in the risk of fetal abnormalities, as well as an increase in the risk of preterm delivery and stillbirth. The study also highlighted the limited role of tests for fetal wellbeing.

Conclusion

Despite advances in medical technology, half of cases of APH remain indeterminate. Accurate prediction or prevention of placental abruption is not possible at the present time. The diagnosis is based on clinical signs and symptoms. Management of complications of placental abruption is team based, requiring involvement of a consultant anaesthetist, haematologist, obstetrician and, sometimes, vascular surgeon and interventional radiologist. The diagnosis and management of placenta praevia is based on ultrasound findings. Vaginal delivery is appropriate if the placental edge to internal os distance is 2.0 cm or more and a caesarean section is recommended if this distance is less than 2.0 cm. In cases of vasa praevia, antenatal diagnosis is possible and is associated with excellent fetal survival. ◆

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