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Placental Abruption

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Placental abruption complicates about 1% of pregnancies and is a leading cause of vaginal bleeding in the latter half of pregnancy. It is also an important cause of perinatal mortality and morbidity. The maternal effect of abruption depends primarily on its severity, whereas its effect on the fetus is determined both by its severity and the gestational age at which it occurs. Risk factors for abruption include prior abruption, smoking, trauma, cocaine use, multifetal gestation, hypertension, preeclampsia, thrombophilias, advanced maternal age, preterm premature rupture of the membranes, intrauterine infections, and hydramnios. Abruption involving more than 50% of the placenta is frequently associated with fetal death. The diagnosis of abruption is a clinical one, and ultrasonography and the Kleihauer-Betke test are of limited value.

The management of abruption should be individualized on a case-by-case basis depending on the severity of the abruption and the gestational age at which it occurs. In cases where fetal demise has occurred, vaginal delivery is preferable. Disseminated intravascular coagulopathy should be managed aggressively. When abruption occurs at or near term and maternal and fetal status are reassuring, conservative management with the goal of vaginal delivery may be reasonable. However, in the presence of fetal or maternal compromise, prompt delivery by cesarean is often indicated. Similarly, abruption at extremely preterm gestations may be managed conservatively in selected stable cases, with close monitoring and rapid delivery should deterioration occur. Most cases of placental abruption cannot be predicted or prevented. However, in some cases, maternal and infant outcomes can be optimized through attention to the risks and benefits of conservative management, ongoing evaluation of fetal and maternal well-being, and through expeditious delivery where appropriate.

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Placental abruption, defined as the premature separation of the placenta, complicates approximately 1% of births.[1] Abruption is an important cause of vaginal bleeding in the second half of pregnancy and is associated with significant perinatal mortality and morbidity. The purpose of this review is to describe the epidemiology of placental abruption with particular emphasis on its incidence, temporal trends, and risk factors and to present an evidence-based approach to the diagnosis and management of the condition, with consideration of the severity of the abruption and the gestational age at which it occurs.

STUDY SELECTION

We carried out a MEDLINE search using the keywords "abruption," "abruptio," and "bleeding" AND "pregnancy," limiting our search to publications in the English language between 1966 and 2006. Further studies were identified through cross-referencing. There are no randomized controlled studies that have specifically examined abruption, and the overwhelming majority of studies are observational (ie, cohort, case–control, or case series). Most large studies dealing with abruption have examined risk factors for the

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condition. Studies that have examined management strategies for the condition are typically limited by small numbers. The levels of available evidence for the diagnosis and management of abruption, based on the classification of the United States Task Force on "Levels of Evidence," are mainly II-1, II-2, and III.

DEFINITION

Placental abruption is defined as premature separation of a normally implanted placenta. Although some degree of placental separation often occurs when there is a placenta previa, these cases are not conventionally considered abruptions in the true sense. Abruption may be "revealed," in which case blood tracks between the membranes and the decidua, and escapes through the cervix into the vagina (Fig. 1A). The less common "concealed" abruption occurs when blood accumulates behind the placenta, with no obvious external bleeding (Fig. 1B). Finally, abruption may be total, involving the entire placenta, in which case it typically leads to fetal death, or partial, with only a portion of the placenta detached from the uterine wall.

CLINICAL IMPORTANCE

Placental abruption has a wide spectrum of clinical significance, varying from cases with minor bleeding and little or no consequences, to massive abruption leading to fetal death and severe maternal morbidity. Abruption may be implicated in up to 10% of preterm births.[1] The risk to the fetus depends on both the severity of the abruption and the gestational age at which the abruption occurs (Fig. 2 and 3), whereas the danger to the mother is posed primarily by the severity of the abruption. A U.S. population-based cohort study of 7,508,655 pregnancies found a perinatal mortality rate of 119 per 1,000 births among pregnancies complicated by abruption, compared with 8.2 per 1,000 among all other births.[2] More recent U.S. data corroborate these previous findings (Fig. 3). This high perinatal mortality is largely due to preterm delivery, because approximately one half of the excess perinatal deaths are associated with early delivery (Fig. 2 and 3).

Although placental abruption is an important cause of spontaneous preterm birth, it is also often an indication for iatrogenic preterm delivery.[1] Premature separation of the placenta before delivery may deprive the fetus of oxygen and nutrition, leading to long-term handicap among survivors. A case–control study of 29 neonates, delivered after abruption, at a median gestational age of 29 weeks, found that 34% of them developed cystic periventricular leukomalacia, a 10-fold increase over controls.[3] Similarly, the rate of intraventricular hemorrhage among the abruption cases was higher than that of controls.[3]

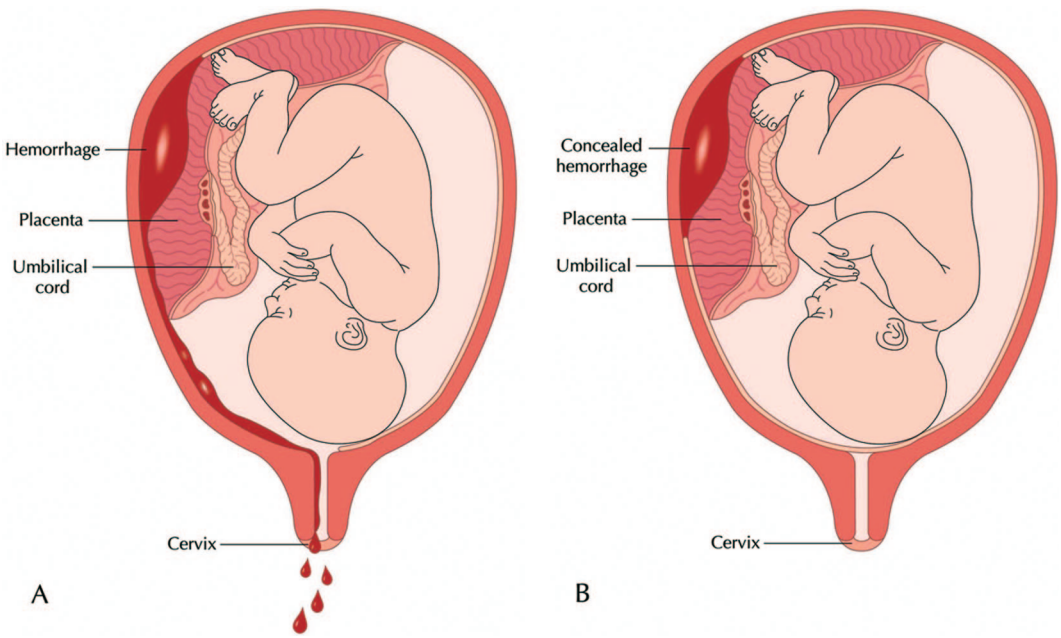

Fig. 1. Types of abruption. **A.** Revealed abruption. Blood tracks between the membranes, and escapes through the vagina and cervix. **B.** Concealed abruption. Blood collects behind the placenta, with no evidence of vaginal bleeding. Illustration: John Yanson. Modified from University Health Care at the University of Utah. High-risk pregnancy: Bleeding in pregnancy/placenta previa/placental abruption. Available at: http://uuhsc.utah.edu/healthinfo/pediatric/hrpregnant/bleed.htm.
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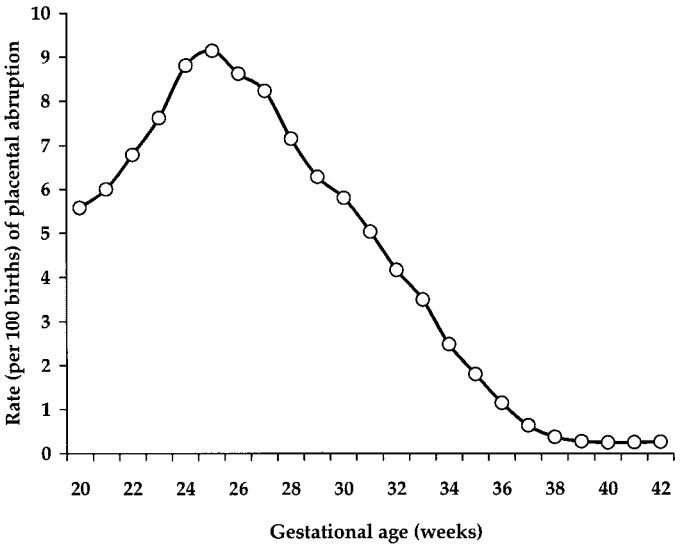

Fig. 2. Rates of abruption across gestation, United States, 2000–2002 (N=11,635,328).
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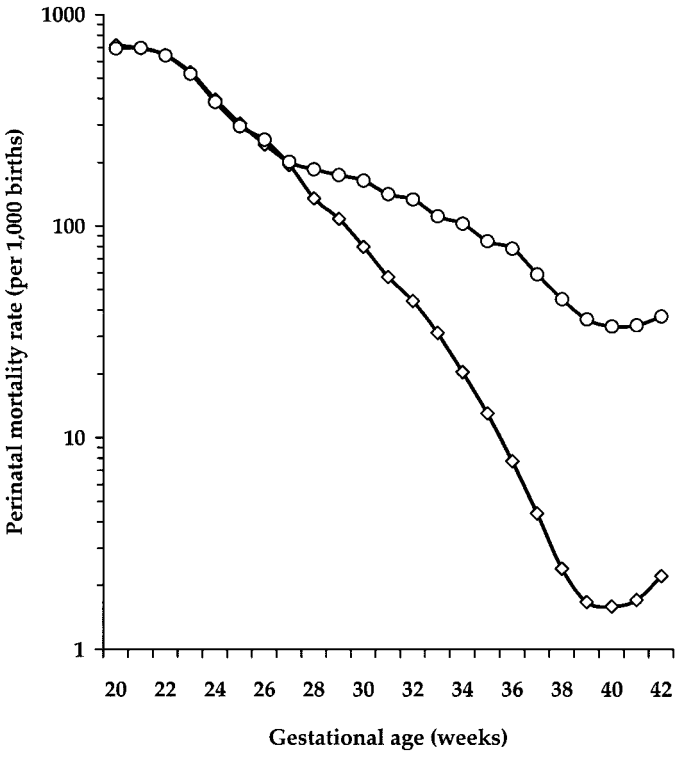

Fig. 3. Perinatal mortality in pregnancies with and without abruption across gestation, United States, 2000–2002 (N=11,635,328). Circles, pregnancies with abruption. Diamonds, pregnancies without abruption.
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Although preterm premature rupture of the membranes frequently precedes abruption, in some cases, placental abruption may cause weakening and premature rupture of the membranes.[4] Placental abruption is associated with intrauterine growth restriction.[5,6] It appears that, in the vast majority of cases, abruption is the end result of a chronic process and that both fetal growth restriction and abruption share a common cause. Maternal risks associated with abruption include, but are not limited to, disseminated intravascular coagulopathy, renal failure, obstetric hemorrhage, need for blood transfusions, hysterectomy, and less commonly, maternal death.

INCIDENCE OF PLACENTAL ABRUPTION

Several epidemiologic cohort studies have found that placental abruption complicates approximately 1% of deliveries.[2,5,7–9] However, when Bernsichke and

Gille[10] performed pathologic examination of 7,038 consecutive placentas, they found evidence of abruption in 3.8%. Similarly, in the U.S. Collaborative Perinatal Project, a prospective cohort study of 55,908 pregnancies, Niswander and Gordon[11] found evidence of abruption in 2.12% of pregnancies. When the diagnosis of abruption is made by examination of the placenta by the pathologist, the majority of cases are noted to have had an unremarkable obstetric history.[12] Thus, there is significant discrepancy between the rates of diagnosis of abruption between clinicians and pathologists.[12] Because cases of abruption diagnosed solely on the basis of pathology examination typically have no obvious clinical consequences, we would recommend that obstetricians reserve the term "abruption" for those cases diagnosed on clinical grounds. An obvious exception to this rule would be cases of pregnancies with an adverse outcome in which examination of the placenta by the pathologist reveals evidence of an otherwise unrecognized abruption. Interestingly, the incidence of abruption is highest at 24–26 weeks gestation, and drops precipitously with advancing gestation (Fig. 2).

TEMPORAL TRENDS IN PLACENTAL ABRUPTION

Ananth and colleagues[8] recently evaluated temporal trends in the rate of placental abruption among singleton births in the United Sates between 1979 and 2001. The overall rate of abruption in the United Sates increased from 0.81% in 1979–1981 to 1.0% in 1999–2001–a relative increase of 23% (95% confidence interval 22–24%). There was a strong race disparity in the temporal trends in abruption risk in that the rate of abruption increased among white women by 15% (from 0.82% to 0.94% between 1979–1981 and 1999–2001), and increased by 92% among black women (from 0.76% to 1.43% between 1979–1981 and 1999–2001).[8] These overall trends in placental abruption were similar in a Norwegian population, where Rasmussen et al[9] noted the frequency of placental abruption increased from 5.3 per 1,000 births in 1971 to 9.1 per 1,000 births in 1990.

RISK FACTORS FOR ABRUPTION

Risk factors for placental abruption are summarized in Table 1.[5,7,13–17] Other risk factors include trauma,[18] thrombophilias,[19] dysfibrinogenemia, hydramnios, advanced maternal age, and intrauterine infections. There is a dose–response relationship between the number of cigarettes smoked and the risk of abruption.[13,16] At least 2 recent population-based retrospective cohort studies have indicated that women who have a cesarean first birth have an increased risk of placental abruption in a second pregnancy when compared with women who had a vaginal first birth.[20,21]

Table 1. Evidence and Strength of Association Linking Major Risk Factors with Placental Abruption Based on Published Studies

	Evidence	
Risk Factors	Strength	RR or OR
Maternal age and parity	+	1.1–3.7
Cigarette smoking	++	1.4–2.5
Cocaine and drug use	+++	5.0–10.0
Multiple gestations	++	1.5–3.0
Chronic hypertension	++	1.8–5.1
Mild and severe preeclampsia	++	0.4–4.5
Chronic hypertension with preeclampsia	+++	7.8
Premature rupture of membranes	++	1.8–5.1
Oligohydramnios	+	2.5–10.0
Chorioamnionitis	++	2.0–2.5
Dietary or nutritional deficiency	+/–	0.9–2.0
Male fetus	+/–	0.9–1.3

RR, relative risk; OR, odds ratio.

These estimates are the ranges of RR or OR found in independent studies.

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Numerous case–control, cohort, and population-based studies have attempted to determine the association between abruption and thrombophilias.[19,22–24] Retrospective case–control studies that have examined the frequency of thrombophilias among women with abruption have mostly found increased rates of thrombophilias.[19,24] Conversely, those that have compared rates of abruption between thrombophilias and controls have generally found no significant differences.[23] Prochaczka and colleagues,[22] in a retrospective case–control study of 102 women with abruption, failed to show any difference in incidence of factor V Leiden carriage status between the cases and controls. Secondary analysis of a large National Institutes of Health–funded prospective cohort study also failed to find an association between maternal and fetal factor V Leiden carrier status and placental abruption in women with no history of thromboembolism.[23] Mean levels of homocysteine are higher among patients with abruptions that among controls.[24]

Bleeding in early pregnancy carries an increased risk of abruption in later pregnancy.[25,26] An elevated second-trimester maternal serum alpha-fetoprotein may be associated with an up to 10-fold increased risk

of placental abruption.[27] Similarly, notching of the uterine artery waveform in the second trimester, a marker of impaired uteroplacental blood flow, carries an increased risk of abruption.[28]

Perhaps the greatest determinant of abruption risk, however, is an abruption in a prior pregnancy.[29] The recurrence risk of abruption in subsequent pregnancies was quantified by Ananth and colleagues[14] in a meta-analysis. The risk increased 15- to 20-fold in subsequent pregnancies when an earlier pregnancy was complicated by abruption.[14] The relative risk of recurrence was less than 9 in only one of the 11 studies examined.[14]

PATHOPHYSIOLOGY

The precise pathophysiology that leads to placental abruption is unknown in many cases. Abruption results from hemorrhage at the decidual–placental interface.[12] It seems that acute vasospasm of small vessels may be the event that immediately precedes the placental separation. There may be thrombosis of the decidual vessels with associated decidual necrosis and venous hemorrhage.[12] Doubtlessly in some cases, abruption is an acute process. Shearing forces resulting from trauma may lead to acute placental separation.[18] This may also be the mechanism by which abruption occurs when there is sudden uterine decompression resulting from membrane rupture with hydramnios, or after delivery of a first twin. With cocaine usage, acute vasoconstriction may lead to placental separation. However, it seems that in the majority of cases, placental abruption may be the consequence of a long-standing process that probably dates back to the first trimester.[12,25] There is abundant support for this concept. A recent large cohort study of 34,271 women indicated that women with first-trimester low levels of pregnancy-associated plasma protein A (in the lowest fifth percentile) had an increased risk of placental abruption.[30] A small case series of placental bed biopsies in 12 women with abruption demonstrated a lack of adequate trophoblastic invasion in seven (58%) of these.[31] These changes are also observed in placentas of women with preeclampsia, suggesting that the two conditions share some common causes.[12] Indeed, abruption occurs frequently in the setting of preeclampsia.[12] There is also an association of growth restriction with abruption, again implicating uteroplacental insufficiency as a possible causative factor.[5] Placentas in cases of abruption more frequently have evidence of chronic pathologic lesions than placentas from pregnancies without abruption.[25] Furthermore, a prospective cohort study has found an association between notching of the Doppler waveform of the uterine artery, a marker of impaired uteroplacental blood flow, at 20–24 weeks and the subsequent development of placental abruption.[28] Thus, uteroplacental insufficiency seems to play a role in the cause of abruption.[12] Finally, bleeding in the first two trimesters of pregnancy is associated with an increased risk of subsequent placental abruption.[25,26] Thrombin is a potent uterotonic agent, and uterine contractions are frequently present. Histologic examination of placentas of women with preterm labor often have evidence of old placental bleeding, supporting the concept that thrombin production from placental abruption is implicated in a significant proportion of cases of spontaneous preterm birth.[32]

Acute separation of the placenta deprives the fetus of oxygen and nourishment, with the consequence that the fetus frequently dies.[12] The coagulation cascade is activated with consumption of coagulation factors and consequent disseminated intravascular coagulopathy (DIC). This risk is highest when there is such a large placental detachment as to cause fetal death. Hemorrhage associated with DIC leads to further consumption of coagulation factors, setting off a vicious circle. Bleeding may occur into the uterine myometrium, leading to a beefy boggy uterus, called a Couvelaire uterus. When there is a recent abruption, pathologic examination frequently reveals fresh clot attached to the maternal surface of the placenta, whereas in older cases there may be fibrin deposits at the site of the abruption, and there may be infarcts of the overlying placenta.[12] In these cases, there may be a depression in the maternal surface of the placenta.[12] Microscopic examination reveals hemosiderin-laden macrophages and evidence of villous hemorrhage.[12]

CLINICAL PRESENTATION

The clinical presentation of abruption varies widely from totally asymptomatic cases to those where there is fetal death with severe maternal morbidity. The classically described symptoms of placental abruption are vaginal bleeding and abdominal pain. It is important to realize, however, that severe abruption may occur with neither or just of one of these signs. The amount of vaginal bleeding correlates poorly with the degree of abruption. The severity of symptoms depends on the location of the abruption, whether it is revealed or concealed, and the degree of abruption. There is a correlation between the extent of placental separation and the risk of stillbirth, with stillbirth occurring in most cases in which there is greater than 50% placental separation.[1,12] Typically, there is uter-

ine hypertonus with associated high-frequency, low-amplitude uterine contractions. The uterus is frequently tender and may feel hard on palpation. Backache may be the only symptom, especially when the placental location is posterior. There may be acute fetal distress, and in cases where more than 50% of the placenta has separated, fetal demise. Rarely fetal death due to abruption may occur with no other symptoms or signs. In some cases, evidence of abruption may be found on ultrasonographic examination of asymptomatic patients. Finally, abruption may present as idiopathic preterm labor.

A variety of fetal heart rate patterns have been described in association with abruption. There may be recurrent late or variable decelerations, reduced variability, bradycardia, or a sinusoidal fetal heart rate pattern. More infrequently, in cases of concealed abruption associated with fetal death, the first clinical sign may be of evidence of abnormal bleeding, the result of disseminated intravascular coagulopathy. In addition, there may be maternal hypovolemic shock. Labor typically proceeds fairly rapidly in cases of abruption. Placental abruption may be associated with acute tubular necrosis and acute cortical necrosis, leading to oliguria and renal failure. Although tubular necrosis may be due to acute hypovolemia, it seems that cortical necrosis is the result of damage to the kidney resulting from products of the coagulation cascade. Renal cortical necrosis may result in chronic renal failure.

DIAGNOSIS

Clinical

The diagnosis of abruption is a clinical one and the condition should be suspected in women who present with vaginal bleeding or abdominal pain or both, a history of trauma, and those who present in otherwise unexplained preterm labor. The differential diagnosis includes all causes of abdominal pain and bleeding. These include placenta previa, appendicitis, urinary tract infections, preterm labor, fibroid degeneration, ovarian pathology, and muscular pain.

Ultrasonography

The ultrasonographic appearance of abruption depends to a large extent on the size and location of the bleed (Fig. 4), as well as the duration between the abruption and the time the ultrasonographic examination was performed.[33] In cases of acute revealed abruption, the examiner may detect no abnormal ultrasonographic findings. Nyberg and colleagues,[33] in a retrospective cohort study of images in 57 cases of abruption, found that the ultrasonographic appearance of abruption in the acute phase was hyperechoic to isoechoic when compared with the placenta. Later on, as the hematomas resolved, they became hypoechoic within 1 week and sonolucent within 2 weeks. In some cases, only a thickened heterogenous placenta could be seen. Thus, it is important to realize that abruption may have a variety of ultrasonographic appearances (Fig. 4; Fig. 5, B–D). The placenta may "jiggle" when sudden pressure is applied with the transducer, the so-called "jello" sign. Glantz and colleagues,[34] in a retrospective cohort study, found that the sensitivity, specificity, and positive and negative predictive values of ultrasonography for placental abruption were 24%, 96%, 88%, and 53%, respectively. Thus, ultrasonography will fail to detect at least one half of cases of abruption. However, when the ultrasonogram seems to show an abruption, the likelihood that there is indeed an abruption is extremely high.[34] Importantly, a negative ultrasonogram does not rule out an abruption.[34] Sholl[35] identified ultrasonographic evidence of a clot in only 25% of abruptions, whereas Jaffe and colleagues[36] found that ultrasonography identified only 50% of abruptions confirmed by pathology. Yeo and colleagues[37] found, in a prospective cohort study of 73 patients presenting

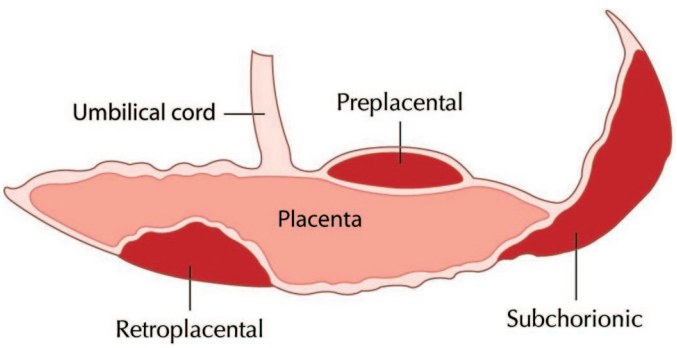

Fig. 4. Diagram showing the different sites at which ultrasonographic evidence of abruption may be observed. Subchorionic hematomas are thought to rise from marginal abruptions. "Preplacental hemorrhage" describes both subamniotic hematoma and massive subchorial thrombosis. Illustration: John Yanson. Adapted from: Nyberg DA. Finberg HJ. Placenta, placental membranes, and umbilical cord. In: Nyberg DA, Mahony, BS, Pretorius DH. Diagnostic ultrasound of fetal anomalies. Chicago (IL): Year Book Medical Publishers; 1990. Copyright 1990, with permission from Elsevier.

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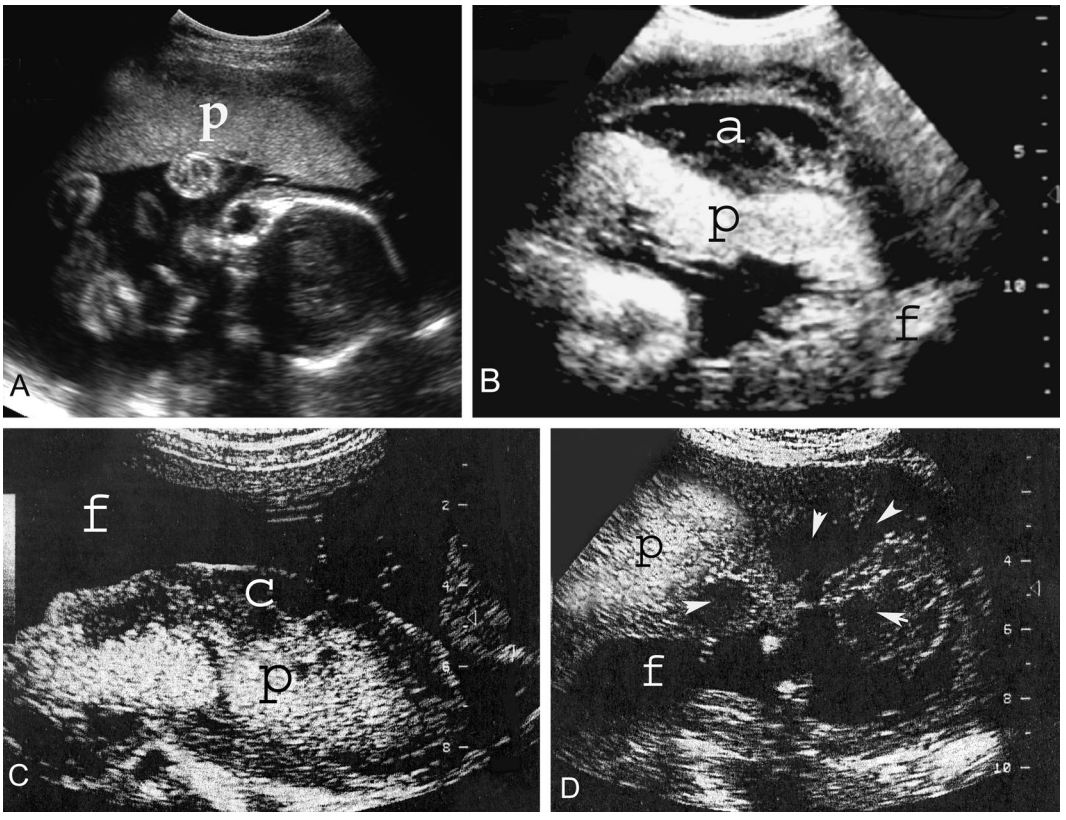

Fig. 5. Varying ultrasonographic appearances of normal placenta and placental abruption. **A.** Normal placenta *(p)*. Note the retroplacental hypoechoic space. It is important not to mistake this for abruption. **B.** Large, retroplacental abruption *(a)* between the placenta *(p)* and the uterus. Fetus *(f)*. This hypoechoic area is the typical appearance of abruption. **C.** Large, extensive ultrasonographic preplacental collection *(c)* beneath the chorionic plate, amniotic fluid *(f)*, and placenta *(p)*. **D.** Thickened placenta *(p)* with heterogenous appearance. The arrowheads point to areas of hemorrhage. Parts B, C, and D reprinted from Yeo L, Ananth CV, Vintzileos AM. Placental abruption. In: Sciarra J, editor. Gynecology and obstetrics. Vol 2. Hagerstown (MD). Lippincott, Williams & Wilkins; 2003. © 2003 Lippincott Williams & Wilkins.

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with vaginal bleeding in the second half of pregnancy, using 7 ultrasonographic parameters (see Box) that the sensitivity of ultrasound for placental abruption was 80%, whereas the specificity was 92%.[37] Positive and negative predictive values were 95% and 69%, respectively.[37] However, no other studies have replicated this accuracy for the ultrasonographic diagnosis of abruption. Ultrasonography may also predict prognosis in abruption; Nyberg and colleagues,[38] in a retrospective review of 69 cases of abruption, found that fetal mortality correlated with the ultrasonographically estimated percentage of abruption and with the location, with the worst prognosis occurring in retroplacental abruptions. An important role of ultrasonography in evaluation of bleeding in the second half of pregnancy is placental location; if there is a placenta previa, it makes it less likely that abruption is the cause of the bleeding. The ultrasonographer must be careful, though, not to mistake a clot over the cervix for placenta previa. The presence of a fundal placenta makes it unlikely that the mass covering the cervix is placenta. A clot may "jiggle" with movement of the fetus or ultrasound transducer.[37]

Kleihauer-Betke Test

The Kleihauer-Betke test is frequently performed in women in whom abruption is suspected. Emery and colleagues[39] carried out a retrospective cohort study of the use of the Kleihauer-Betke test at their institution. There were no positive Kleihauer-Betke tests among the 27 placentas that showed evidence of abruption on pathologic examination. Nine percent of patients with no evidence of abruption had positive Kleihauer-Betke tests. A retrospective case–control study comparing 100 low-risk women in the third trimester with 151 women of similar gestational ages who had undergone evaluation for abdominal trauma found that the incidence of

Ultrasonographic Criteria for Diagnosis of Placental Abruption

1. Preplacental collection under the chorionic plate (between the placenta and amniotic fluid) (see Fig. 5C)
2. Jello-like movement of the chorionic plate with fetal activity.
3. Retroplacental collection. (See Fig. 5B)
4. Marginal hematoma
5. Subchorionic hematoma
6. Increased heterogenous placental thickness (more than 5 cm in a perpendicular plane) (Fig. 5D)
7. Intra-amniotic hematoma

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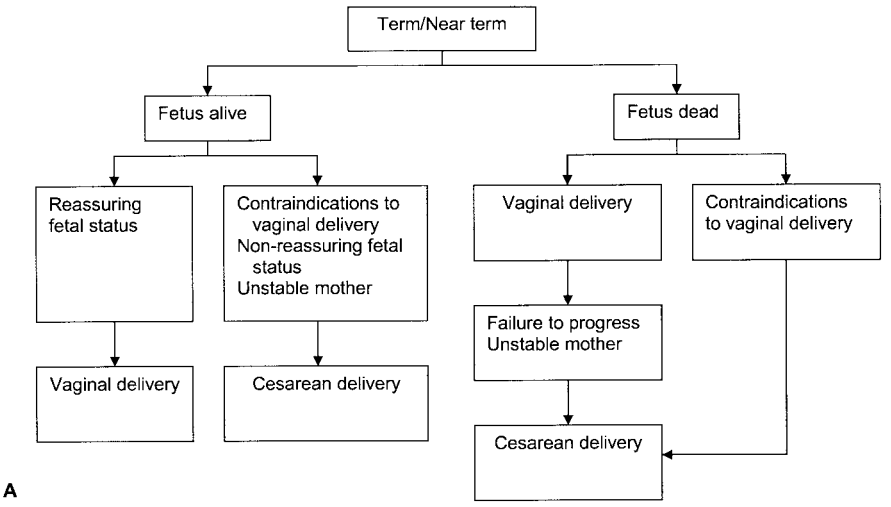
positive Kleihauer-Betke tests were similar in the two groups.[40] There was no association between a positive test and abruption. Thus, the Kleihauer-Betke test has limited usefulness in the diagnosis of abruption. A negative test should not be used to rule out abruption, nor does a positive test necessarily confirm abruption. However, a Kleihauer-Betke test allows quantification of fetomaternal transfusion to guide dosing of Rh-immune globulin in Rh-negative women.

MANAGEMENT

The management of placental abruption depends on the presentation, the gestational age, and the degree of maternal and fetal compromise. (Fig. 6). Because the presentation is widely variable, it is important to individualize management on a case-by-case basis. More aggressive management, desirable in cases of severe abruption, may not be appropriate in milder cases of abruption.

In cases of severe abruption with fetal death, regardless of gestational age, as long as the mother is stable, it is reasonable, in the absence of other contraindications, to allow the patient to have a vaginal delivery. Typically, the uterus is contracting vigorously, and labor rapidly progresses. Amniotomy is frequently sufficient to speed up delivery. There is a significant risk of coagulopathy and hypovolemic shock. Intravenous access should be established and blood and coagulation factors should be replaced aggressively. Meticulous attention should be paid the amount of blood loss; clinicians frequently underestimate this. Blood should be taken for complete blood count, coagulation studies and type and crossmatch, and the blood bank should be informed of the potential for coagulopathy. A Foley catheter should be placed and the hourly urine output should be monitored closely. It is prudent to involve an anesthesiologist in the patient's care early. When labor does not progress rapidly, and in cases in which there is feto-pelvic disproportion, fetal malpresentation, or a prior classical cesarean delivery, cesarean delivery may be necessary to avoid worsening of the coagulopathy. Bleeding from surgical incisions in the presence of DIC may be difficult to control, and it is important to stabilize the patient and to correct any coagulation derangement during surgery. After delivery, the patient should be monitored closely, with particular attention paid to vital signs, amount of blood loss, and urine output. In addition, the uterus should be observed closely to ensure that it remains contracted and is not increasing in size, and blood loss should be monitored closely. The uterus may be hypotonic, and occasionally hysterectomy may be necessary. Blood should be drawn for complete blood count and coagulation studies at regular intervals until the patient is stable. Finally, some cases of abruption may be associated with severe preeclampsia, which may be masked because the patient may be normotensive due to hypovolemia. Thus, there should be a high index of suspicion for severe preeclampsia in patients with abruption not resulting from an obvious cause such as trauma or cocaine use. In such cases, the patients may benefit from close volume status monitoring, early recognition of hypovolemia, and adequate blood replacement.

In cases of abruption at term or near term with a live fetus, prompt delivery is indicated. The main question is whether vaginal delivery can be achieved without fetal or maternal death or severe morbidity. In cases in which there is evidence of fetal compromise and delivery is not imminent, cesarean delivery should be performed promptly, because total placental detachment could occur without warning. When both maternal and fetal status are reassuring, conservative management, with the goal of vaginal delivery, is reasonable. Labor, if established, should be allowed to progress, otherwise induction of labor should be considered. Both mother and fetus should be monitored closely during labor. Should the fetal heart rate tracing become nonreassuring, with bradycardia, loss



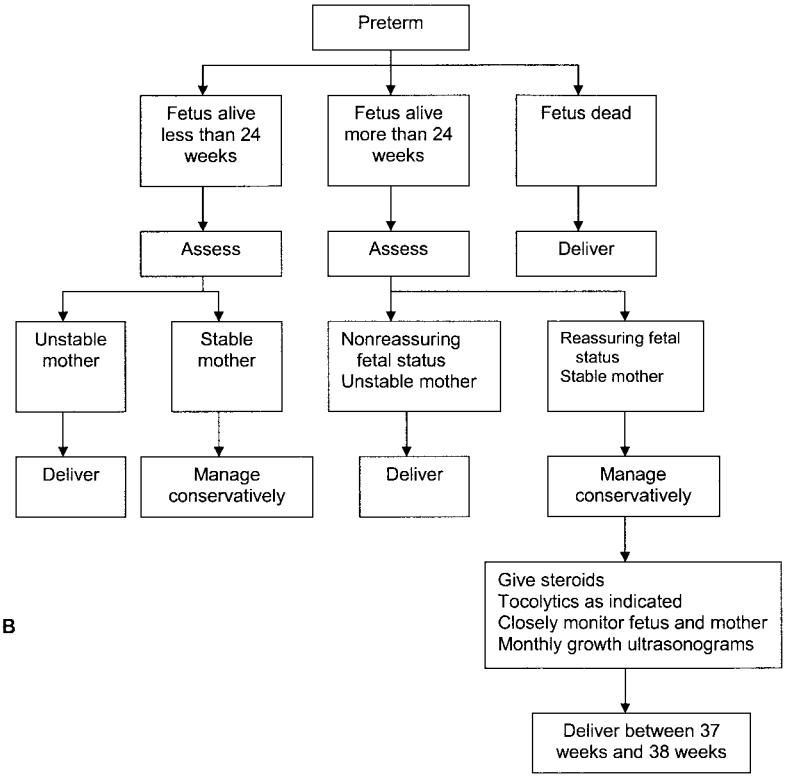


Fig. 6. Algorithm for the management of placental abruption in term or near term **(A)** and preterm births **(B)**. In all cases, complete blood count and coagulation indices should be checked; blood or blood volume should be replaced; coagulopathy should be corrected; and intake, output, and renal function should be monitored.

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of variability, or persistent late decelerations, prompt cesarean delivery is indicated. Similarly, should maternal compromise occur, the fetus should be delivered promptly.

A few older retrospective cohort studies suggested that outcomes in cases of abruption where the fetuses were alive were superior when there was a cesarean delivery to when vaginal delivery occurred.[41–43] In a case–control study examining the relationship between decision–delivery interval and perinatal outcome in 33 patients with severe abruption and fetal bradycardia, Kayani and colleagues[41] found that longer decision–delivery intervals were associated with poorer perinatal outcomes. It must be emphasized that in the setting of significant abruption with fetal bradycardia, minutes may make a difference between death and survival.

At more preterm gestational ages (between 20 and 34 weeks of gestation), when there is partial placental abruption and the maternal and fetal status

are reassuring, the patient may be managed conservatively.[44,45] Preterm birth is the leading cause of perinatal death in women with abruption, and to optimize perinatal outcomes, it is desirable, if possible, to prolong gestation. However, it cannot be overemphasized that these patients require extremely close monitoring, because there is a significant risk of fetal death. In cases where the gestational age is between 24 and 34 weeks, steroids should be administered to promote fetal lung maturation. Patients should be delivered in a center with adequate neonatal facilities and the parents should be counseled by a neonatologist regarding potential treatments and outcomes for the neonate. Prolonged hospitalization and monitoring may be necessary. It may be possible to discharge these patients to outpatient management if the fetal status is reassuring once they have remained stable for several days.

Abruption suspected on the basis of an incidental finding on ultrasound should be managed on a case-by-case basis. Thorough history and physical examination should be conducted for evidence of trauma, cocaine use, hypertension, preeclampsia, or any other predisposing factors. Subsequent management may follow the recommendations above, taking into consideration the gestational age and the state of maternal and fetal well-being. If ultrasonography suggests an abruption in a term fetus, delivery is reasonable. At preterm gestations, if fetal status is reassuring, conservative management should be the goal.[45] In a retrospective cohort study of conservative management of 40 cases of placental abruption in preterm gestations after 20 weeks gestation, Combs and colleagues[45] were able to delay delivery until term in 33%. The perinatal mortality rate was 22%, and all cases of perinatal death except one were attributable to extreme prematurity. Of those who delivered before term, 63% had at least one other risk factor (twins, advanced cervical dilation, rupture of membranes) that predisposed to preterm delivery.

In cases where conservative management is chosen, initial hospitalization for further evaluation and assessment of fetal well-being is reasonable. We recommend serial ultrasonograms to evaluate progression or regression of the abruption.

TRAUMA IN PREGNANCY

Women who sustain trauma in pregnancy, such as those in motor vehicle accidents, are at risk of abruption.[18] This is usually the result of shearing forces, may occur even without direct abdominal trauma, and is independent of placental location.[18] Current American College of Obstetricians and Gynecologists guidelines[18] recommend that women involved in trauma should have a minimum of 4 hours of fetal monitoring. This duration should be extended and further evaluation carried out in the presence of uterine contractions or irritability, nonreassuring fetal heart rate tracing, uterine tenderness, vaginal bleeding, severe maternal trauma, or rupture of the membranes. When the fetal heart rate tracing is nonreassuring, delivery is generally indicated, depending on gestational age and individual circumstances.

SCREENING FOR THROMBOPHILIAS

In women with abruption without a known cause, such as trauma or cocaine usage, screening for congenital or acquired thrombophilias should be considered. Thrombophilias that may be associated with abruption include factor V Leiden, antithrombin III, prothrombin gene mutation, protein S and protein C deficiency, methyltetrahydrofolate reductase deficiency, lupus anticoagulant, and anticardiolipin antibodies. Women who screen positive should be treated with heparin and aspirin in subsequent pregnancies or with vitamin B6 and B12 in the case of methyltetrahydrofolate reductase deficiency.

TOCOLYSIS

It is generally taught that tocolytics, especially β-sympathomimetics such as terbutaline, are contraindicated in the presence of vaginal bleeding, because side effects such as tachycardia could mask the clinical signs of blood loss. However, a few retrospective cohort and case–control studies have evaluated the use of tocolytics (including β-sympathomimetics) in the presence of bleeding in the second half of pregnancy, including patients with suspected stable placental abruptions.[35,45,46] Bond and colleagues[44] expectantly managed 43 women with clinical evidence of placental abruption before 35 weeks gestation, using tocolysis in cases where there were contractions. There were no intrauterine deaths. They achieved a mean latency period to delivery of 12.4 days. Of these, in 23 cases, delivery occurred within 1 week of admission, while in the remaining 20 patients, the mean time to delivery was 26.8 days. However, there was no comparison group. Towers and colleagues[47] reviewed 236 cases of third trimester bleeding, which included 131 cases of placental abruption, with a mean gestational age of 28.9 weeks at the time of first bleeding. In 95 (73%) of these women, tocolysis had been used. The mean time from bleeding until delivery was 18.9 days, the median time from bleeding until delivery was 7 days, and the neonatal mortality

rate was 51 deaths per 1,000 live births. All cases of mortality were related to prematurity and no adverse maternal or fetal effects of tocolysis occurred. Thus, it seems reasonable to use tocolytics with caution in stable women who have partial placental abruption but are remote from term.[46] Because of the aforementioned concerns, probably magnesium sulfate rather than terbutaline should be the first-line tocolytic in cases of stable suspected abruption.

MANAGEMENT IN SUBSEQUENT PREGNANCY

Women with an abruption are at approximately ten-fold increased risk of abruption in a subsequent pregnancy.[48] In addition, they are at increased risk of other adverse pregnancy outcomes, including preterm birth and preeclampsia.[48] Although no interventions have been demonstrated to reduce this risk, some recommendations are possible. Women who smoke tobacco or use cocaine should be counseled on the adverse effects of exposure to these substances, and encouraged to quit before the next pregnancy. Hypertension should be controlled before and during the subsequent pregnancy. Although no clear benefit in reducing recurrent abruption risk has been demonstrated, it is reasonable to treat women with inherited thrombophilias with thromboprophylaxis, as indicated, in subsequent pregnancies. Because patients with abruption have an increased risk of impaired uteroplacental perfusion in subsequent pregnancies,[48] it is reasonable to consider serial growth scans every 4 weeks in the second half of pregnancy. In cases where the mother has had two or more prior abruptions, amniocentesis for lung maturity and delivery at about 37 weeks gestation seems reasonable.

CONCLUSION

Placental abruption remains an important cause of perinatal mortality and morbidity. Perinatal mortality is determined by the severity of the abruption and the gestational age at which it occurs. Unfortunately neither accurate prediction nor prevention of abruption are possible at the present time. Despite advances in medical technology, the diagnosis of abruption is still a clinical one. When abruption does occur, there are some strategies that may help minimize the risks of morbidity and mortality associated with this condition.

These include early recognition and prompt delivery in cases in which the fetus is mature and, in stable cases remote from term, conservative management to enable steroid administration, allow transfer to a center with facilities for care of the preterm infant, and in some cases, permit fetal maturation before delivery. Finally, close attention to maternal condition, with replacement of blood and blood products as indicated, may improve outcomes for the mother.

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