ACTA OVERVIEW

Placental abruption: epidemiology, risk factors and consequences

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Abstract

Placental abruption, classically defined as a premature separation of the placenta before delivery, is one of the leading causes of vaginal bleeding in the second half of pregnancy. Approximately 0.4–1% of pregnancies are complicated by placental abruption. The prevalence is lower in the Nordic countries (0.38–0.51%) compared with the USA (0.6–1.0%). Placental abruption is also one of the most important causes of maternal morbidity and perinatal mortality. Maternal risks include obstetric hemorrhage, need for blood transfusions, emergency hysterectomy, disseminated intravascular coagulopathy and renal failure. Maternal death is rare but seven times higher than the overall maternal mortality rate. Perinatal consequences include low birthweight, preterm delivery, asphyxia, stillbirth and perinatal death. In developed countries, approximately 10% of all preterm births and 10–20% of all perinatal deaths are caused by placental abruption. In many countries, the rate of placental abruption has been increasing. Although several risk factors are known, the etiopathogenesis of placental abruption is multifactorial and not well understood.

Abbreviations DIC, disseminated intravascular coagulopathy; IUGR, intrauterine growth restriction/retardation; PPROM, preterm premature rupture of the membranes

Introduction

Placental abruption, classically defined as the complete or partial separation of a normally implanted placenta before delivery, occurs in 0.4–1% of pregnancies (1–6). The incidence varies slightly in different populations (5–9), and has been increasing in some studies (9–11) but not all (5). At least 50 different risk factors or risk markers for placental abruption have been reported with smoking, preeclampsia and history of previous placental abruption being the strongest. Although many risk factors or risk markers are known, the cause of placental abruption often remains unexplained.

Placental abruption is one of the most significant causes of maternal morbidity and perinatal mortality (2,3,7,12,13). Both maternal and perinatal risks associated with placental abruption depend on the severity of abruption. Maternal peripartum risks include obstetric hemorrhage, need for blood transfusion, emergency hysterectomy, disseminated intravascular coagulopathy (DIC), renal failure and even maternal death (12–15). Fetal risks are associated with intrauterine growth restriction (IUGR), low birthweight, preterm delivery, asphyxia, stillbirth and perinatal death (8). Fetal survival depends not only on the severity of the abruption but also on the gestational age (3).

The purpose of this clinically oriented overview is to summarize the current knowledge of epidemiology, risk factors and maternal and perinatal consequences of placental abruption.

Material and methods

Medline database was searched using the keywords 'placental abruption' or 'abruptio placentae' in combination with 'pregnancy', 'risk factors', 'epidemiology', 'adverse perinatal outcome' and 'adverse maternal outcome'. Approximately 2100 English language articles up to October 2009 were identified. Case reports were removed. Only studies focusing mainly on clinical aspects of placental abruption were included. Studies selected were mainly published during the last 10 years, although frequently referenced and highly regarded older reports were not excluded. Reference lists of articles identified by this strategy were also searched, and

articles judged clinically relevant were selected. Finally, 250 articles were judged relevant and 87 were included in this overview. There were no randomized controlled trials that had specifically studied placental abruption. The overwhelming majority of studies were cohort studies, case–control studies or case series examining risk factors associated with placental abruption. Thus, the level of evidence of most of the studies based on the classification of Oxford Centre of Evidence-Based Medicine is mainly II, III or IV.

Results

The overall incidence of placental abruption varies from 0.4 to 1.0% (1–4,6,8,16–18). The rate is generally lower in case–control (0.35%) than in cohort studies (0.69%) (16). In USA-based studies the incidence has been higher both in cohort (0.81%) and case–control studies (0.37%) compared with studies conducted outside the USA (0.60 and 0.26%, respectively) (16). Also, the incidence of placental abruption seems to be lower in the Nordic countries than in the USA (5–9). The incidence is highest at 24–26 weeks of gestation and decreases with advancing gestation (10,19). Over 50% of the cases occur before 37 completed weeks of gestation (3,7,8). Some (9–11) but not all studies (5) report an increase in the overall rates.

Age and parity have been linked to placental abruption (1,4,10,20,21) (Table 1). Abruption occurs more frequently in older women (≥35 years), but usually this increase has been attributed to multiparity (three or more deliveries) independent of age (1). In one study, neither parity nor maternal age increased the risk (22). In another study, maternal age >35 years predicted placental abruption among primiparous but not among multiparous women (1). In many other studies, advanced maternal age has been an independent risk factor (10,20,21). Also, mothers less than 20 years of age have been a risk group in some studies (11,23). However, in most studies the odds ratios of age and parity are low, and the clinical importance might be limited. Being black, unmarried or of lower sosioeconomic status are other risk factors for abruption (8,11,20,22).

The sociodemographic and behavioral risk factors discussed below are summarized in Table 1, maternal and historical risk factors in Table 2, and pregnancy-associated risk factors in Table 3.

Smoking

Several studies have shown that the relative risk for placental abruption associated with maternal smoking during pregnancy varies from 1.5 to 2.5 (5,16,24–26). The dose dependency has been strong in most of the studies (2,5), but not in all (27). There also seems to be a threshold effect at approximately 10 cigarettes per day, after which the risk remains relatively constant (16). Among women with prior abruption, the risk of repeat abruption is increased irrespective of smoking habits (5). Quitting smoking before pregnancy or early in pregnancy seems to reduce the risk of abruption to the level of nonsmokers (5,28,29).

Paternal smoking is also a risk factor for placental abruption, and doubles the risk (7). However, if both partners smoke the risk is additive and nearly fivefold (7). Women with subsequent abruption seem to stop smoking more rarely compared with control subjects. In one study, only 7% of women in the abruption group and 14% of control women stopped smoking during pregnancy (4).

Hypertensive disorders

Chronic hypertension complicates 0.3–0.8% of pregnancies, and increasing maternal age, parity, smoking and black race increase the risk (30). In some (17,20,30), but not all studies (7,18,27,31), chronic hypertension has been a risk factor for placental abruption. In one study, the rate of abruption

Table 1. Sociodemographic and behavioral risk factors for placental abruption.

Risk factor	Odds ratio*	Level of evidence	Reference number
Sociodemographic			
Maternal age ≥35 years	1.3–2.6	II/III	1,2,5,11,18,20,21,63
Maternal age <20 years	1.1–1.5	II	11,20
Parity ≥3	1.1–1.4	II	2,63
Black race	1.3	II	63
Unmarried or single mother	1.2–1.5	II	2,11,20,63
Behavioral			
Cigarette smoking	1.5–2.5	II/III	1,2,4,5,7,18,20,21,26,63
Alcohol use	1.6–2.8	II/III	7,63,70
Cocaine use	3.9–8.6	II/III	60,71
Unexplained infertility	1.2–2.4	II/III	2,97,98

*These estimates are the ranges of odd ratios given in independent studies.

Table 2. Maternal and historical risk factors for placental abruption.

Risk factor	Odds ratio*	Level of evidence	Reference number
Maternal risk factor			
Chronic hypertension	1.8–2.4	II	2,11,20,30,63
Hyperhomocysteinemia	1.8–5.3	II/III	32,33,35
Thrombophilia	1.4–7.7	II/III	32,36,37
Pregestational diabetes mellitus	2.7	II	2
Hypothyreosis	3.0	III	99
Anemia	2.2	II	63
Uterine anomaly	8.1	III	4
Historical risk factor			
Cesarean section	1.3–2.4	II/III	1,4,54,55,57–59
Miscarriage	1.4–3.4	III	1,18,21
Preeclampsia	1.9	II	56
Stillbirth	1.6–13.1	II/III	1,5,18,21
Placental abruption	3.2–25.8	II/III	4,21,56

*These estimates are the ranges of odds ratios given in independent studies.

Table 3. Pregnancy-associated risk factors for placental abruption.

Risk factor	Odds ratio*	Level of evidence	Reference number
Pregnancy-induced hypertension	1.5–2.5	II	2,11,20,30,63
Preeclampsia	1.9–4.4	II/III	2,7,11,18,20,21,31
Superimposed preeclampsia	2.8	II	31
Chorioamnionitis	2.5–3.3	II/III	7,11,20
Premature rupture of membranes	1.8–5.9	II	2,8,11,20,47,63
Oligohydramnion	2.1	II	47
Polyhydramnion	2.5	II	63
Placenta previa	3.2–5.7	III	1,7
Vaginal bleeding $\leq$28 gestational weeks	2.0–3.1	II/III	1,65
Vaginal bleeding $\geq$28 gestational weeks	12.3–18.7	III	1
Multiple gestation	2.0–2.9	II/III	11,21,68
Male fetal sex	1.2–1.3	II/III	1,20,21,23
Small-for-gestational-age fetus	1.3–4.1	II/III	2,8,20,21,30
Velamentous umbilical cord insertion	2.5	III	18

*These estimates are the ranges of odds ratios given in independent studies.

among women with or without chronic hypertension in singleton pregnancies was 1.56 and 0.6%, respectively (30). After adjustment for potential confounders, women with chronic hypertension had 2.4-fold increased risk for abruption (30). In another study, women with chronic hypertension had no increased risk for abruption [risk ratio 1.4; 95% confidence interval 0.5–3.6] (31). Chronic hypertension with superimposed preeclampsia increased the risk for placental abruption 2.8- to 7.7-fold (27,30,31).

Although severe preeclampsia is a strong risk factor for placental abruption (16,27,31), transient hypertension in pregnancy and mild preeclampsia have also been linked to placental abruption in some (20,27), but not all studies (16,31). However, variable criteria have been used to define preeclampsia (16,17,20,31). The risk for abruption is further increased among women with hypertensive disorder who smoke (16,27).

Hyperhomocysteinemia and thrombophilia

There is an association between hyperhomocysteinemia and placental abruption (32–35). Hyperhomocysteinemia is a strong indicator of folate deficiency and vitamin B_{12} deficiency (32). According to a meta-analysis, folate deficiency may also be a risk factor for placental abruption (odds ratio 25.9; 95% confidence interval 0.9–736.3) (32). In another study, high red cell folate decreased the risk for placental abruption (33). In some studies, vitamin B_{12} deficiency has been a risk factor for placental abruption (32,33).

Inherited and acquired thrombophilias increase the risk of venous thromboembolism and adverse pregnancy outcome (36). One of the early studies found that 65% of women with preeclampsia, IUGR, unexplained stillbirth or placental abruption had heritable or acquired thrombophilia (37). The risk found in individual studies has been variable because of

different study designs (36). Homozygous methylenetetrahydrofolate reductase point mutation 677 has been associated with placental abruption in several (32,38,39), but not all studies (37,40). Some studies have shown an association between placental abruption and heterozygous factor V Leiden mutation (37,41,42). However, in a Finnish study M385T polymorphism in the factor V gene, but not Leiden mutation, was associated with placental abruption (43). A Swedish study of 102 women with abruption also failed to show any difference in factor V Leiden carrier rate between cases and control subjects (44). The rate of heterozygous prothrombin gene mutation is increased eight- to ninefold among women with placental abruption (37,45). There are insufficient data of other thrombophilias and placental abruption (36). The combination of hyperhomocysteinemia and thrombophilia increases the risk of placental abruption three- to sevenfold (38).

Premature rupture of membranes and chorioamnionitis

Approximately 4–12% of patients with preterm premature rupture of the membranes (PPROM) before 37 weeks gestation develop placental abruption (17,46). The risk increases with decreasing gestational age at membrane rupture (46). In some women with PPROM, sudden reduction of uterine volume may lead to placental abruption (17). In contrast, women exposed to prolonged PPROM are at increased risk of developing abruption if the time from membrane rupture to delivery exceeds 24 hours (47).

Preterm premature rupture of the membranes is often associated with ascending intrauterine infection. In one study, the rates of abruption among women with or without intrauterine infection were 4.8 and 0.8%, respectively (47). The attributable proportion of intrauterine infections in all abruptions was 6.7% (47). In another study, the rate of histologically confirmed chorioamnionitis was 30% among women with placental abruption (48). Severe chorioamnionitis was strongly associated with placental abruption in both term and preterm pregnancies (48).

Trauma

Approximately 6% of all trauma cases (49) and 20–25% of major trauma cases (50) are associated with placental abruption, but placental abruption is difficult to predict based on the severity of trauma (49). Placental abruption usually becomes manifest within 6–48 hours after trauma but can occur up to 5 days later (49,51,52).

External cephalic version may be regarded as a form of trauma in some cases, particularly if extensive force is used. This procedure is also associated with placental abruption, although the risk is low. In a recent review the rate was only 0.12% (53).

Other risk factors

Other prepregnancy risk factors for placental abruption include previous cesarean section and uterine anomaly (4,54). Furthermore, the risk for placental abruption is increased in the next pregnancy following adverse pregnancy outcomes, including delivery of a small-for-gestational-age newborn, preterm birth, transient hypertension in pregnancy, preeclampsia or stillbirth (13,55,56). This may indicate a common etiologic factor for these conditions (55). Both short and long interpregnancy intervals have also been associated with increased risk of placental abruption (55). According to some studies, cesarean first delivery increases the risk for placental abruption by 30–40% in the next pregnancy compared with women having a vaginal first delivery (4,55,57–59). According to a Finnish study, this risk was 2.4-fold among primiparous and 3.9-fold among multiparous women with cesarean first delivery (54). If the interpregnancy interval is less than 1 year, the risk of abruption is increased by 52% in women with vaginal first delivery and by 111% in women with cesarean first delivery (58). There may also be an association between placental abruption and congenital uterine malformations (4).

Other pregnancy-related risk factors for placental abruption include placenta previa, bleeding during pregnancy, multiple pregnancy, and alcohol and cocaine use (1,7,60–64). Bleeding in early pregnancy increases the risk for abruption in later pregnancy (65). The detection of a subchorionic or retroplacental hematoma in the first trimester by ultrasound examination increases the risk for subsequent placental abruption six- to 11-fold (66,67). Such a hematoma may impair normal placentation. In contrast, a hematoma can result from impaired placentation (67). Approximately 5% of women with placenta previa develop abruption (7). In one study, uterine bleeding > 28 weeks of gestation and placenta previa were the strongest predictors of placental abruption (1). Among women with placenta previa, the risk was three- to fourfold, and among women with uterine bleeding >28 weeks the risk was 12- to 19-fold (1). If uterine bleeding occurred at <28 weeks, the risk for placental abruption was twofold (1).

The risk of placental abruption is two- to threefold in twin pregnancies compared with singleton pregnancies (1,63–68), although not all studies have shown this (7,20). With increasing multiplicity the risk of placental abruption increases, but associated perinatal mortality decreases (64). Discordant growth of twins is a risk factor for placental abruption (69).

The risk of placental abruption is increased due to alcohol consumption during pregnancy in some (7,61,70), but not all studies (20). In one study, the risk of stillbirth was higher among alcohol users, particularly due to placental abruption (61). In the USA, the rate of cocaine use in pregnancy is as high as 10% in selected populations (1,60). The risk for

placental abruption among cocaine users is 3.9- to 8.6-fold (60,71).

Maternal consequences

Placental abruption-associated peripartum risks for the mother are caused by bleeding and include need for blood transfusion, emergency hysterectomy, DIC, renal failure and even maternal death (12–15). Maternal mortality associated with placental abruption decreased from 8% in 1919 to less than 1% in 1995 (14). Still, in the UK in 2000–2002, four maternal deaths and in 2003–2005, two maternal deaths were caused by placental abruption (15,72). Although rare, placental abruption-associated maternal mortality is seven times higher than overall maternal mortality rate (13).

Bleeding caused by placental abruption can lead to maternal hypovolemic shock. Blood loss may be underestimated in placental abruption because concealed bleeding into the myometrium is difficult to quantify. The coagulation cascade then becomes activated. When the placental detachment is large enough to cause fetal death, the risk of DIC is increased. In DIC, coagulation and fibrinolysis result in widespread clotting and bleeding. Placental abruption may also be associated with acute renal failure resulting from hypovolemia or DIC. In one study, 71 (8%) out of 867 emergency hysterectomies were associated with placental abruption (odds ratio 3.2; 95% confidence interval 1.8–5.8) (12). Also, women with prior placental abruption are less likely than other women to become pregnant again (73). After placental abruption with survived newborn, 59% of women had a subsequent delivery, compared with 71% of those without abruption. After perinatal loss, the corresponding rates were 83 and 85% (73). This may reflect maternal anxiety and distress caused by prior placental abruption.

Perinatal consequences

Fetal and neonatal morbidity and mortality associated with placental abruption are linked with preterm birth, low birthweight and fetal distress. Abruption involving more than 50% of placental surface can lead to fetal death (8) due to lack of oxygen and nourishment provided by the placenta. Among cases with placental abruption, perinatal mortality rate varies largely depending on neonatal facilities and has been decreasing in recent decades. Perinatal mortality can be as high as 60% (3), but in developed countries is in the range of 9–12% (2,3,7). High perinatal mortality rate can be explained by a strong link to preterm delivery. However, even term babies with normal birthweight have 25-fold higher mortality with abruption (3) compared with term babies without abruption. More than 50% of all perinatal deaths among cases with placental abruption are stillborns (14).

Of neonates born after placental abruption, 40–60% are preterm (born before 37 gestational weeks) (7,8), and approximately 14% of the abruptions occur before 32 weeks (8). In one study, the rate of giving birth to a low-birthweight infant by women with placental abruption was 46%, compared with 6.4% among those without (3). Other neonatal consequences include fetal growth restriction, anemia and hyperbilirubinemia (25). The association with fetal growth restriction is so strong that growth restriction alone could be used as a marker for the risk of abruption (3). The rate of fetal malformations may be as high as 4.4%, which is two times higher than that in the general population. Most involve congenital heart defects and malformation of the central nervous system (74).

In severe placental abruption cases, Apgar scores and cord blood pH values are low due to antenatal hypoxia and blood loss (18,75–77). In one study, the risk for intrapartum asphyxia was 3.7-fold. Three percent of asphyctic newborns and 0.7% of control newborns had placental abruption (78). Intrapartum asphyxia may lead to long-term consequences among survivors. Neonates born after placental abruption are more likely to develop cystic periventricular leukomalacia or intraventricular hemorrhage (75,79). The risk increases with prematurity and low birthweight (75,79). Severe abruption increases the risk for cerebral palsy (75,80). In one study, 11% of low-birthweight neonates (<2500 g) born after placental abruption and 0% of low-birthweight control neonates without abruption (matched by gestational age) were diagnosed with cerebral palsy 2 years after delivery (75). Placental abruption has also been linked to sudden infant death syndrome (81,82). This may be due to long-term intrauterine hypoxia caused by failure of placentation in early pregnancy, ultimately leading to placental abruption.

Discussion

The prevalence of placental abruption seems to be lower in the Nordic countries than in the USA (5–9). This could reflect differences in diagnostic criteria or different study populations. The population in Nordic countries is often more homogenous compared with the population in the USA. Hypertensive complications, smoking, alcohol and cocaine use are more prevalent in the African-American population in the USA (11). Also, in many studies the lowest gestational age considered has not been defined (2,8,10,11,23) or differs from one study to another. For instance, in Finland perinatal mortality is counted from 22 gestational weeks, in Sweden from 20 weeks and in Norway from 16 weeks.

Smoking is a preventable risk factor, and smoking cessation reduces many adverse pregnancy outcomes, including placental abruption. Although the prevalence of smoking has declined during the past decades, smoking by young women in the Nordic countries is still strikingly high, and only a small proportion of smoking women stop smoking during pregnancy. The association between placental abruption and

smoking was first reported in 1976 (83). Overall, 15–25% of placental abruption episodes may be attributable to smoking (16). Although the mechanisms explaining the association between smoking and placental abruption remain largely speculative, it is known that smoking increases homocysteine levels in the plasma, which may play a role (32). Hyperhomocysteinemia can induce endothelial cell injury and dysfunction, leading to local thromboembolism and defects within the placental vascular bed (84). Also, nicotine has vasoconstrictive effects on uterine and umbilical arteries, and carboxyhemoglobin interferes with oxygenation. The hypoxic changes caused by nicotine and carbon monoxide can lead to placental infarcts, suggesting that increased capillary fragility might result in arterial rupture, leading to placental abruption (28,85). In smoking women, placental function is impaired, although placental weight is increased, which may be due to adaptive angiogenesis in the peripheral villous tree (86). This is reflected by increased levels of proangiogenic placental growth factor and reduced levels of antiangiogenic soluble endoglin and fms-like tyrosine kinase 1 (87). Smokers also have lower concentrations of cellular fibronectin (88), which connects the trophoblast to the uterine decidua (89). Paternal smoking is also a risk factor for abruption (7). Women whose partners smoke might smoke more heavily, or the association may simply be due to passive exposure to smoking (7). The importance of second-hand smoking is probably even more significant in low- and middle-income countries in which women's rights and social status are poor.

Hypertensive disorders in pregnancy have also been linked to placental abruption (7,16,17,20,27,30,31). Comparing these studies is problematic, since definitions vary. Overall, the strength of evidence linking severe preeclampsia and superimposed preeclampsia with placental abruption is strong (16,27,30,31). However, the evidence linking milder hypertensive disorders with placental abruption is weak. The possible mechanisms behind the increased risk of abruption in preeclamptic pregnancies are speculative. However, placental abruption and preeclampsia seem to have a common etiology with failed placentation in early pregnancy (55). This may lead to placental dysfunction and further increase the risk of placental abruption in women with preeclampsia.

Women with unexplained abruption may benefit from screening for thrombophilias and hyperhomocysteinemia (37,38,84). For instance, young women with hyperhomocysteinemia and folate deficiency may be prone to endothelial dysfunction, including the placental vasculature (32), leading to atherosclerosis and thromboembolism (84). Women who screen positive for thrombophilia can be treated with heparin and aspirin in subsequent pregnancies or with folate, vitamin B_6 and vitamin B_{12} in the case of methylenetetrahydrofolate reductase deficiency or hyperhomocysteinemia (19,38). However, the efficacy of these therapies is not supported by strong evidence. According to one study, women using folic acid or multivitamin supplements during pregnancy had 26% lower risk of developing placental abruption than women who had not used such supplements (6). Thus, it might be worth recommending these supplements to women with prior abruption.

Many other risk factors are less common, with relatively small attributable proportion. The true role of these risk factors can only be addressed in larger epidemiological studies. Although PPROM frequently precedes abruption, sometimes placental abruption may lead to PPROM (90). Abruption is associated with marked infiltration of neutrophils in the decidua (90). This is a rich source of proteases that can degrade extracellular matrix, leading to PPROM. It is difficult to determine whether neutrophil infiltration into the decidua is secondary to vascular disruption or whether it is the primary cause of abruption (48).

Chorioamnionitis may precede abruption or abruption may precede chorioamnionitis, or the two conditions may be unrelated and present simultaneously (91). Direct bacterial colonization of decidua with tissue inflammation may initiate a process that ultimately results in placental abruption (91). Sometimes subclinical decidual thrombosis may initiate an inflammatory process (91) that activates cytokines. These cytokines upregulate the production and activity of matrix metalloproteinases in the trophoblast (48). This may result in destruction of the extracellular matrix and cell-to-cell interactions which then may lead to disruption of the placental attachment (48).

Placental abruption is a frightening complication of trauma in pregnancy. The mechanism is directly related to the injury. The relatively elastic uterus is able to alter its shape in reaction to forces applied to the abdomen, whereas the less elastic placenta is not. A shearing effect disrupts the attachment of placenta to the decidua (92). American College of Obstetricians and Gynecologists recommends a minimum of 4 hours post-trauma monitoring. This monitoring should be continued in cases with uterine contractions or tenderness, a nonreassuring fetal heart rate pattern, vaginal bleeding, rupture of amniotic membranes or serious maternal injury (93).

Alcohol easily crosses the placenta and accumulates in the fetus and amniotic fluid (94). Alcohol exposure disturbs the feto-maternal hormonal balance and also causes vasoconstriction in the placenta and umbilical cord (94,95), which might lead to placental abruption. No safe amount of alcohol consumption during pregnancy has been determined. Also, the risk for placental abruption among cocaine users may result from vasoconstrictive effects of cocaine. Although the relationship between placental abruption and cocaine use is confounded by other risk factors, including use of other drugs, tobacco and lack of prenatal care, cocaine use is an independent risk factor (60,71). Amphetamine use is also associated with placental abruption (96).

Significant progress in perinatal medicine has taken place during the last few decades, and as a result, maternal mortality as well as perinatal mortality and morbidity have dramatically decreased in developed countries. It is unfortunate that this favorable development does not apply to placental abruption. Although maternal and perinatal mortality rates have decreased (3,7,13–15,72), this pregnancy complication still causes enormous short-term and long-term morbidity (14,16,18,19,73,75–77,79,80). The impact of this emergency on perinatal outcomes is much larger than approximated simply from its rate. It has been estimated that approximately 10% of all preterm births and 10% or more of all perinatal deaths are caused by placental abruption (3,23). Thus, the disease burden is enormous.

In conclusion, placental abruption is a complex disease. Although several risk factors are known, the etiopathogenesis is not fully understood. Even though placental abruption is relatively rare, the consequences may be severe to the mother and fetus alike. Despite research, placental abruption is still the 'big unknown'.

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