—Review—

Placental Abruption Associated with Cerebral Palsy

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Placental abruption is separation of the placenta from its normal implantation site of the uterine body before delivery of the fetus. The Japan Obstetric Compensation System for Cerebral Palsy (JOCSC) includes it as a major cause of cerebral palsy. Placental abruption is classified as revealed or concealed hemorrhage on the basis of the presence or absence of external bleeding; concealed hemorrhage is associated with a worse prognosis for mothers and infants. If survival of the fetus is confirmed in cases of placental abruption, it should usually be delivered promptly. There is no evidence-based method to prevent placental abruption. Therefore, awareness of early symptoms of placental abruption in pregnant Japanese women is important. (J Nippon Med Sch 2022; 89: 263–268)

Key words: placenta abruption, cerebral palsy, Japan Obstetric Compensation System for Cerebral Palsy

Introduction

Placental abruption (premature separation of normally implanted placenta, *abruptio placentae*) is partial or total separation of the placenta from its normal implantation site in the uterine body before delivery of the fetus[1,2]. Placental abruption starts with necrosis (ischemic changes in the decidua basalis) and rupture (bleeding) of maternal blood vessels in the decidua, followed by hematoma formation along the decidua basalis (retroplacental hematoma or hemorrhage)[1–4]. The retroplacental hematoma further causes separation compression of the adjacent placenta, which ultimately results in complete separation of the placenta from the uterine wall. Although the cause of placental abruption is unclear, impaired trophoblast invasion with subsequent atherosis may be related to inflammation and infection[5–9].

Placental abruption is a severe, life-threatening obstetric complication for the mother and fetus. Extensive separation of the placenta causes fetal asphyxia, leading to cerebral palsy (CP) or death of the fetus. The Japan Obstetric Compensation System for Cerebral Palsy (JOCSC) was launched in January 2009 to compensate parents for the economic burden of children with severe CP associated with delivery and to analyze factors contributing to CP, with the goal of improving the quality of health care[10]. The JOCSC systematically accumulates and organizes individual case data and compiles annual reports that propose evidence-based measures to prevent recurrence. To prevent recurrence of similar cases, the JOCSC publishes an annual report for the public, delivery institutions, related academic societies/organizations, and government agencies, among other organizations. In the second annual report of the JOCSC, in May 2012[11], history of placental abruption was identified in 20 (25.3%) of 79 cases of severe CP. In addition, maternal disseminated intravascular coagulation (DIC) is caused by entry of tissue thromboplastin into maternal blood circulation, which promotes blood coagulation (microthrombosis) in blood vessels, thereby resulting in marked dysregulated coagulation and fibrinolysis related to widespread clotting with bleeding.

This article reviews the literature on placental abruption associated with adverse fetal/neonatal outcomes.

Clinical Course of Placental Abruption

The typical clinical course of placental abruption depends on whether the ruptured blood vessels are arteries or veins, and their size. Because placental findings start with hemorrhage in the decidua basalis, the decidua is accompanied by hemorrhagic degeneration and necrosis, and hematoma adheres to the decidua and becomes difficult to peel off manually over time after formation of a

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retroplacental hematoma. An indentation of villous tissue will appear on the surface of the maternal site in the placenta where the hematoma is present. Histopathological analysis shows blood cells infiltrating the decidua from the hematoma. Possible causes of the rupture/collapse of blood vessels in the decidua are[3–5,12] (1) absence of physiological transformation of the utero-placental arteries because of abnormal vascular structures deep in the myometrium (the result of trophoblastic invasion), (2) attenuated adhesion of the trophoblast to the uterine wall, which is caused by granulocyte elastase, a proteolytic enzyme activated by inflammation in endometrial infection (chorioamnionitis), and (3) weakening of the chorion/decidua with inflammation. Arterial bleeding, associated with (1), tends to develop suddenly and progresses rapidly, whereas venous bleeding, associated with (2) and (3), is triggered by preterm labor or premature rupture of the membranes (PROM) and tends to progress slowly[13–15]. In cases of chorioamnionitis associated with preterm labor and PROM, prematurely delivered placentae are frequently accompanied by marginal venous hemorrhage that undermines the edge of the placenta and originates from deciduitis[4]. Acute reduction in uterine volume and intrauterine surface caused by PROM can disrupt the site of placental attachment in the decidual spongiosa layer, thereby predisposing to abruption[16]. Therefore, placental abruption after the presence of inflammation may often occur after PROM[6]. In contrast, when arterial bleeding develops inside the placenta, the abruption will progress rapidly.

Placental Abruption and Neonatal Sequelae

Neurological sequelae (abnormalities) are present in 15-20% of surviving neonates after placental abruption[1,17,18]. In addition, about 20% of the principal causes of CP registered in JOCSC involve placental abruption, which was the most common causative perinatal complication for CP identified in Japan[10].

According to the Annual Reports on Placental Abruption in the Perinatal Registration System of the Japanese Society of Obstetrics and Gynecology for 2001-2010[17,19], the average onset of placental abruption was at 34.2 weeks of gestation, and about 36% of cases developed before 34 weeks of gestation. The incidences of fetal/neonatal death and an umbilical artery pH lower than 7.0 were both approximately 17%. In a long-term follow-up study of neonates delivered prematurely[20], placental abruption did not increase the risk of hospitalization for neurological symptoms but was associated with increased incidences of CP and developmental disorders (hazard ratio: 6.71 and 3.36, respectively). Although approximately 10% of cases of CP registered in JOCSC had normal umbilical artery pH values, umbilical artery pH was low in cases of CP mainly caused by placental abruption, indicating fetal hypoxic acidemia[21]. Since the fetus is supplied with oxygen and nutrients from the mother through the placenta, if the placenta separates, the oxygen supply to the fetus will be insufficient and fetal hypoxia/acidemia occurs. Outcomes for the fetus/neonate correlate with the placental abruption area: if the area is large, fetal/neonatal death or CP may occur, even if the developed fetus is delivered rapidly after onset of placental abruption.

In Japan, many textbooks, including one formally published by the Japan Society of Obstetrics and Gynecology, used Page's classification, which uses percentage separation of the placenta to define the severity of placental abruption. Unfortunately, this classification is based on an article published almost six decades previously[22,23]. According to the classification, fetal asphyxia/death can occur even in mild cases. Page clarified[22,23] that estimates of percentage separation were made retrospectively and should not serve as guides to therapy. Although percentage separation is undoubtedly an indicator of disease severity, Page's classification is not useful in clinical practice that aims to improve perinatal outcomes. It should only be used for retrospective examination of cases[24].

Magnetic Resonance Imaging Findings in Cerebral Palsy Associated with Placental Abruption

Since there is no single pathological manifestation of CP, magnetic resonance imaging (MRI) findings vary markedly; however, the main conditions that manifest as causes of CP include hypoxic-ischemic encephalopathy in term infants and periventricular leukomalacia (PVL) in preterm infants[25–31]. The former is classified according to severity and duration of hypoxia/ischemia. If severe acidemia/ischemia occurs in a short period of time, because of placental abruption with massive arterial bleeding, widespread hyperintensity of the supratentorial brain, known as the "white cerebrum sign", is a characteristic finding in diffusion-weighted images (total asphyxia)[31]. In cases of acidemia/ischemia caused by severe fetal hypoxia for 20-30 minutes or longer at term, damage to the basal ganglia and thalamus is a characteristic MRI finding (profound asphyxia)[27–29]. Although the prognosis for term fetuses/neonates correlates with the severity and duration of fetal asphyxia, outcomes for preterm

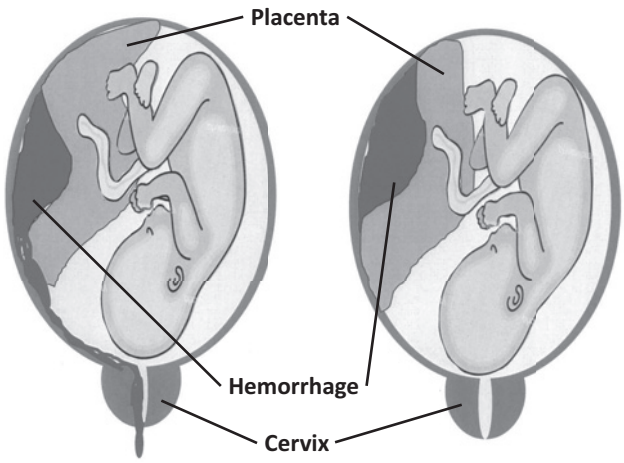

Fig. Revealed hemorrhage/abruption with external bleeding (left) and concealed hemorrhage/abruption without external bleeding (right)

fetuses/neonates may also be associated with gestational age and presence of intrauterine infection (inflammation)[31–33]. Outcomes will also be associated with the presence of chorioamnionitis in the cases managed as preterm labor and will sometimes be revealed by retroplacental hemorrhage, as separation of the placenta from the uterine wall progresses because of preterm RPOM[10,19]. During the preterm period, multiorgan disorders such as chronic lung disease and necrotizing enterocolitis may occur in association with fetal inflammatory responses, even if there are no severe complications such as placental abruption.

Clinical Symptoms of Placental Abruption Associated with Cerebral Palsy

The first subjective symptoms of placental abruption in pregnant women include genital bleeding, abdominal pain, labor pains (uterine contractions) that often do not resolve, and abdominal swelling with or without a feeling of decrease/disappearance of fetal movement, suggesting severe fetal asphyxia due to advanced placental abruption. Placental abruption is classified in relation to the presence of external bleeding as hemorrhage/abruption and concealed hemorrhage/abruption[2,34], as shown in the **Figure**. In the former, separation progresses from the marginal side of the placenta, and external bleeding is observed; in the latter, separation progresses primarily from the central side, and internal bleeding accumulates between the placenta and uterine wall[34]. Placental abruption without external bleeding was reported to be less common and occurs when blood accumulates behind the placenta, with no obvious external bleeding. This type accounts for 20-40% of all placental abruptions[34]; however, it has been reported that the prognosis of affected newborns is worse and that the number of cases of fetal death in utero is increasing[34–37]. In revealed hemorrhage/abruption, in which bleeding occurs outside of the uterus, external bleeding prevents detachment toward the center of the placenta. In concealed hemorrhage/abruption, separation progresses from the center of the placenta, and a massive retroplacental hematoma forms. This increases intrauterine pressure, and thrombin entering the uterine muscle layer further induces uterine muscle contraction, which may increase stress on the fetus. Absence of external bleeding may delay diagnosis of placental abruption[36,37]. The perinatal prognosis may also deteriorate. Furthermore, poor maternal outcomes are attributable to increased release of thromboplastin into maternal circulation as intrauterine pressure increases, thereby inducing maternal DIC[36]. Presentation for hospital treatment may be delayed because both the obstetrician and mother might underestimate the risk of placental abruption[37,38]. Unfortunately, a few cases have been misdiagnosed as preterm labor by medical staff[10].

Diagnosis of Placental Abruption

1. Clinical Findings

Diagnosis of placental abruption is based on the following clinical symptoms: genital bleeding (often noncoagulable), abdominal/labor pain (plate-like stiffness, often persistent contractions with unclear intermittent labor), and abnormal fetal heart rate (suspected preterm labor, frequent or irregular ripple-like, and abnormal fetal heart rate patterns).

2. Ultrasonographic Findings

Ultrasound is almost always the first (and usually the only) imaging modality used to evaluate placental abruption; however, ultrasound is not sensitive for diagnosis of placental abruption[39]. In typical and advanced cases, ultrasonography shows placental thickening and/or the presence of a hematoma between the placenta and uterine wall. However, retroplacental hematoma was identified by ultrasound in only 2-25% of all abruptions[1,40]. The sensitivity of the typical findings was low (24%) despite the high specificity of 96%[40]. In addition, the negative predictive value of ultrasonographic findings for placental abruption was low (53%), despite the high predictive value (88%). Therefore, retroplacental hematoma identified by ultrasonography leads to a definitive diagnosis of placental abruption. However, placental abruption cannot be excluded when ultrasonography findings are normal.

In addition, if the placenta is on the anterior wall of the uterus, abdominal pain is noticed immediately after the onset of abruption, and diagnosis is straightforward with palpation (plate-like hardness) and/or ultrasonography[24]. However, if the placenta is on the posterior wall, onset of subjective symptoms and/or objective findings will be delayed, and ultrasonographic findings may not be diagnostic.

3. Fetal Heart Rate Monitoring

The fetal heart rate pattern reflects the severity of placental abruption[1,41]. Undetectable variability and bradycardia are significantly more frequent in cases of severe placental abruption and so may reflect the severity of placental abruption. According to a previous retrospective study of low-risk pregnant women in Miyazaki Prefecture, Japan[42], if fetal heart rate abnormalities (repeated late deceleration or fetal bradycardia) were observed at the time of hospital visit for labor, placental abruption was more common (46%). In addition, 73% of cases resulted in CP or stillbirth. Therefore, if fetal heart rate abnormalities are observed at the time of hospital visit for (preterm) labor, strict management that considers the possibility of placental abruption is necessary. Again, it is important not to exclude the possibility of placental abruption when the fetal heart rate is abnormal.

Treatment of Placental Abruption

If placental abruption is considered minimal and the results of fetal heart rate monitoring are normal, close monitoring in preparation for vaginal delivery might be possible; however, in other cases the fetus should be delivered promptly if fetal survival is confirmed. Emergency cesarean section is commonly selected, especially when the fetus is expected to survive and prompt vaginal delivery is not possible[1,10,19]. The optimal time for intact survival of the fetus is within 1 hour from onset of placental abruption[10,19]. The risks of perinatal mortality and maternal critical obstetrical bleeding with DIC increase at 2-3 hours after onset. Because placental abruption is often a sudden medical emergency, preoperative examination should be omitted, when possible, to shorten the preoperative time. In addition, in cases of severe fetal bradycardia or fetal death, cardiotocography sometimes erroneously records the maternal heart rate as the fetal heart rate[43], and confirmation of the presence and number of fetal heart beats by ultrasonography is required[1,41].

The prognosis for placental abruption is better for prompt delivery at the facility where placental abruption develops than for delivery after emergent transportation to a facility. In Japan, about 70% of placental abruption cases leading to CP involved delivery after transportation[10]. In Japan, about half of all deliveries are managed in private obstetrics clinics that cannot adequately treat severe neonatal asphyxia or DIC. Because immediate emergent cesarean section and blood transfusion for DIC are often impossible in clinics, early diagnosis and appropriate transfer are a critical issue in Japan. In fact, maternal transport is often carried out if placental abruption is diagnosed at clinics[44]. Therefore, in Japan, an emergency transport system should be developed for situations when prompt delivery is not possible at such clinics[10,44]. Specifically, the following are required for rapid transport: (1) clarification of transportation assessment criteria that accord with the function of obstetrics facilities, (2) smooth communication of information between the transporting and receiving facilities, and (3) preparation for prompt delivery at the receiving facility. It is also desirable to perform advance simulations at facilities, to perfect the necessary procedures, including assembling staff, emergency cesarean section, and preparation for maternal transportation. In addition, since it is often impossible to write a medical record quickly when responding, it is advisable to keep a simple record over time and a detailed record after treatment.

It is also important to educate and encourage pregnant women to visit obstetrics facility at an early stage after developing placental abruption[17,38]. Specifically, all pregnant women should be told of the need to contact an obstetrics facility as soon they have unusual symptoms such as atypical abdominal pain or bleeding at 30 weeks of gestation or later.

Risk Factors and Prevention of Placental Abruption

Studies of postpartum placental findings in singleton pregnancies[45,46] found that abnormally shaped placentae, such as circumvallate placentae, were associated with higher incidences of severe perinatal complications such as placental abruption; however, there is no evidence-based preventive measure against placental abruption. Reported risk factors for placental abruption are a history of placental abruption and pregnancy in young or older females[47]. In a recent study in Japan[48], recurrent placental abruption occurred at an earlier gestational age and followed a more severe course than the first occurrence of placental abruption. A recent review of data from Western countries[47] found a marked effect of maternal age on abruption; however, abruption frequencies and other risk

factors such as smoking suggested marked variation across countries. Ichizuka et al.[49] reported that alcohol consumption, smoking during pregnancy, number of deliveries, polyhydramnios, oral administration of ritodrine hydrochloride, and hypertensive disorders during pregnancy were risk factors for CP after placental abruption in Japan. Among pregnant women who developed placental abruption registered in JOCSC, the rates of smoking during pregnancy and preeclampsia were 9.7% and 18.8%, respectively[10,49]. Educational activities, including health guidance for pregnant women with any risk factor, may be important in increasing their awareness of placental abruption[10,47–49].

In a study by Ruiter et al.[50], the risk of placental abruption in a subsequent pregnancy was significantly higher in women with a previous placental abruption than in women without such a history (5.8% vs. 0.06%, respectively; adjusted odds ratio, 93). Women with a history of placental abruption that occurred at term in their first pregnancy had a higher risk of recurrence than did women with preterm or early preterm (< 32 weeks of gestation) placental abruption in their first pregnancy (adjusted odds ratio, 188 vs. 52 or 39, respectively). Therefore, the authors recommended elective induction from 37 weeks of gestation, especially for women with a history of placental abruption at term in a previous pregnancy.

Conclusion

Unfortunately, no reliable method has been established to prevent placental abruption. To prevent poor perinatal outcomes, healthcare providers should diagnose and treat placental abruption by assessing the severity of the separation and gestational age. Furthermore, it is important to raise awareness of the condition among pregnant women.

Conflict of Interest: None declared.

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