Hong Kong J Gynaecol Obstet Midwifery 2023;23(1):24-33 | https://doi.org/10.12809/hkjgom.23.1.04

Incidence, risk factors, and clinical outcomes of placental abruption in a tertiary hospital in Hong Kong: a retrospective case-control study

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Introduction: This study aims to identify risk factors for placental abruption and evaluate maternal and fetal outcomes of patients with placental abruption in a tertiary hospital in Hong Kong.
Methods: Medical records of patients with placental abruption treated at the Tuen Mun Hospital between January 2017 and December 2021 were retrospectively reviewed. Data retrieved included patient demographics, alcohol/substance abuse and smoking status, obstetric history, antenatal characteristics, body mass index at first antenatal visit, clinical presentation, intrapartum events, complications, and maternal and perinatal outcomes. Each patient was matched with a control who delivered just before the patient.
Results: Of 22 990 deliveries and 23 230 live births, there were 86 placental abruption cases; the incidence was 0.37%. After adjusting for confounders, the risk factor for placental abruption was a history of antepartum haemorrhage. Compared with controls, patients with placental abruption had higher rates of caesarean sections (91.9% vs 23.3%, p<0.001), postpartum haemorrhage (62.8% vs 15.1%, p<0.001), uterine atony (31.4% vs 3.5%, p<0.001), blood transfusion (25.6% vs 3.5%, p<0.001), and disseminated intravascular coagulopathy (7.0% vs 0%, p=0.029). Compared with controls, neonates complicated with placental abruption had lower Apgar score at 1 minute (7 vs 8, p<0.001), higher preterm birth rate (64.0% vs 9.3%, p<0.001), lower birth weight (2296.4 g vs 3088.8 g, p<0.001), and more perinatal morbidities. Patients with a Couvelaire uterus had higher rates of uterine atony (56.3% vs 27.0%, p=0.026), postpartum haemorrhage (93.8% vs 61.9%, p=0.014), disseminated intravascular coagulopathy (25.0% vs 3.2%, p=0.014), blood transfusion (68.8% vs 17.5%, p<0.001), and secondary intervention (25.0% vs 1.6%, p=0.005). Neonates born from patients with a Couvelaire uterus had higher rates of acidosis (umbilical cord blood pH <7.1) [53.3% vs 5.8%, p<0.001], lower Apgar score at 1 minute (25.0% vs 4.8%, p=0.028), and hypoxic-ischaemic encephalopathy (12.5% vs 0%, p=0.039).
Conclusion: Clinicians should be vigilant for placental abruption in patients with antepartum haemorrhage, especially in high-risk patients with a history of placental abruption, hypertension, or pre-eclampsia. Early and consistent antenatal care is imperative to identify those with risk factors. Proper education and timely preventive management should be provided to improve maternal and fetal outcomes.

Keywords: Abruptio placentae; Risk factors

Introduction

Placental abruption is defined as premature separation of the placenta from the decidua at or after 20 weeks of gestation and can result in maternal and fetal morbidity and mortality. Its incidence is higher in Canada and the United States and lower in Norway, Spain, Finland, Sweden, the Netherlands, and Denmark[1]. The overall incidence is approximately 3 to 10 per 1000 births[1,2]. Clinical presentation of placental abruption includes painful vaginal bleeding with uterine contraction and hypertonicity and a non-reassuring fetal heart rate pattern[3]. Progression of placental abruption can be rapid, especially in cases of severe abruption. Maternal complications include haemorrhagic shock, coagulopathy and disseminated intravascular coagulation, uterine rupture, renal failure, and ischaemic necrosis of distal organs[4,5]. Neonatal complications include death and neurodevelopmental issues[4,6].

There are no reliable diagnostic tests or markers to predict or prevent the occurrence of placental abruption. Its risk factors include advanced maternal age, multiparity, smoking, cocaine and drug use, pre-eclampsia, chronic hypertension, premature rupture of membranes, trauma, polyhydramnios, structural uterine anomalies, and a history of placental abruption[7-10]. This study aims to identify risk factors for placental abruption and evaluate maternal and

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fetal outcomes of patients with placental abruption in a tertiary hospital in Hong Kong.

Materials and methods

This study was approved by the New Territories West Cluster Research Ethics Committee (reference: NTWC/REC/22071). Medical records of patients with placental abruption treated at the Tuen Mun Hospital between January 2017 and December 2021 were identified using the International Classification of Diseases codes. Tuen Mun Hospital is a public hospital in Hong Kong handling around 5000 live births per year. Data retrieved included patient demographics, alcohol/substance abuse and smoking status, obstetric history, antenatal characteristics, body mass index at first antenatal visit, clinical presentation, intrapartum events, complications, and maternal and perinatal outcomes. Each patient was matched with a control who delivered just before the patient.

The diagnostic criteria for placental abruption were: (1) presentation of signs of painful vaginal bleeding and at least one of the following: non-reassuring fetal status, severe abdominal pain, tetanic uterine contractions, and uterine hypertonicity; (2) a freshly delivered placenta showing clinically significant retroplacental bleeding or clots; and (3) a confirmation on prenatal ultrasound. Patients often had a combination of these diagnostic criteria.

Data analysis was performed using SPSS (Windows version 26; IBM Corp, Armonk [NY], United States). A p value of <0.05 was considered statistically significant. The case and control groups were compared using the Student's *t* test and Mann-Whitney *U* test for continuous variables and the Chi-squared test or Fisher's exact test for categorical variables. Multivariate logistic regression analysis was used to determine risk factors for placental abruption. Subgroup analysis was performed to determine association of Couvelaire uterus with maternal and fetal outcomes.

Results

Of 22 990 deliveries and 23 230 live births in Tuen Mun Hospital between 2017 and 2021, there were 86 placental abruption cases; the incidence was 0.37%. The most common symptom of placental abruption was a combination of vaginal bleeding and abdominal pain (38.4%), followed by a combination of vaginal bleeding, abdominal pain, and uterine hypertonicity (24.4%) and vaginal bleeding alone (19.8%) [Table 1]. The most common clinical presentation of placental abruption was retroplacental clot during delivery (96.5%), followed by blood-stained amniotic fluid during delivery (39.5%), a non-reassuring fetal heart rate pattern (38.4%), retroplacental clot on ultrasound (20.9%), and a Couvelaire uterus during caesarean section (18.6%).

As shown in Table 2, the case and control groups were comparable in terms of maternal age (32.4 vs 30.9 years, p=0.052) and the percentage of advanced maternal age (≥35 years) [34.9% vs 22.1%, p=0.063]. Compared with controls, patients with placental abruption had higher rates of pre-eclampsia (11.6% vs 3.5%, p=0.043), preterm prelabour rupture of membranes (16.3% vs 3.5%, p=0.005), a history of antepartum haemorrhage (20.9% vs 7.0%, p=0.008), and use of aspirin during the antenatal period (10.5% vs 0%, p=0.003). However, after adjusting for confounders, only a history of antepartum haemorrhage remained significant (p=0.013, Table 3).

Compared with controls, patients with placental abruption had higher rates of caesarean section (91.9% vs 23.3%, p<0.001), caesarean section for non-reassuring fetal heart rate (33.7% vs 7%, p<0.001), postpartum haemorrhage (blood loss ≥500 ml: 62.8% vs 15.1%, p<0.001; blood loss ≥1000 ml: 31.4% vs 3.5%, p<0.001), uterine atony (31.4% vs 3.5%, p<0.001), blood transfusion (25.6% vs 3.5%, p<0.001), disseminated intravascular coagulation (7.0% vs 0%, p=0.029), and prolonged hospital stay (>4 days) [30.2% vs 15.1%, p=0.018], and longer length of hospital stay (median, 4 vs 3 days, p<0.001) [Table 4]. Treatment performed for severe postpartum haemorrhage included compression sutures (n=4), bilateral uterine artery ligation (n=1), and hysterectomy (n=1).

As shown in Table 5, of 89 neonates (three sets of twins) in the placental abruption group, one was stillbirth. Compared with controls, neonates born from patients with placental abruption had higher rates of premature birth (before 37 weeks: 64.0% vs 9.3%, p<0.001; before 34 weeks: 37.1% vs 2.3%, p<0.001; and before 28 weeks: 11.2% vs 0%, p=0.002), lower birth weight (2296.4 g vs 3088.8 g, p<0.001), lower median Apgar score at 1 minute (7 vs 8, p<0.001), and higher rates of resuscitation care (18.2% vs 0%, p<0.001), mechanical ventilation (25.0% vs 1.2%, p<0.001), admission to neonatal intensive care unit (47.7% vs 1.2%, p<0.001), inotropes for management of haemodynamic instability (12.5% vs 0%, p=0.001), and blood transfusion (17.0% vs 0%, p<0.001). Neonates born from patients with placental abruption had higher complication rates in terms of respiratory distress syndrome (39.8% vs 0%, p<0.001), apnoea of prematurity (25.0% vs 0%, p<0.001), intraventricular haemorrhage (8.0% vs 0%,

Table 1. Symptoms, clinical presentations, and diagnostic criteria of placental abruption

	Patients with placental abruption (n=86)*
Symptom	
Vaginal bleeding alone	17 (19.8)
Abdominal pain alone	7 (8.1)
Uterine hypertonicity alone	1 (1.2)
Vaginal bleeding and abdominal pain	33 (38.4)
Abdominal pain and uterine hypertonicity	1 (1.2)
Vaginal bleeding and uterine hypertonicity	6 (7.0)
Vaginal bleeding, abdominal pain and uterine hypertonicity	21 (24.4)
Clinical presentation	
Non-reassuring fetal heart rate patterns	33 (38.4)
Retroplacental blood clot by ultrasound	18 (20.9)
Blood-stained amniotic fluid during delivery	34 (39.5)
Retroplacental clot/haemorrhage during delivery	83 (96.5)
Couvelaire uterus during caesarean section	16 (18.6)
Diagnostic criteria	
Retroplacental clots/haemorrhage	43 (50.0)
Ultrasound diagnosis	1 (1.2)
Ultrasound diagnosis and retroplacental clots/haemorrhage	12 (14.0)
Painful vaginal bleeding, uterine hypertonicity and non-reassuring fetal heart rate patterns	1 (1.2)
Painful vaginal bleeding, uterine hypertonicity and retroplacental clots/haemorrhage	7 (8.1)
Painful vaginal bleeding, uterine hypertonicity and ultrasound diagnosis	1 (1.2)
Painful vaginal bleeding, uterine hypertonicity, non-reassuring fetal heart rate patterns and retroplacental clots/haemorrhage	8 (9.3)
Painful vaginal bleeding, uterine hypertonicity, ultrasound diagnosis, non-reassuring fetal heart rate patterns and retroplacental clots/haemorrhage	2 (2.3)
Painful vaginal bleeding, uterine hypertonicity, ultrasound diagnosis and retroplacental clots/haemorrhage	2 (2.3)
Painful vaginal bleeding, non-reassuring fetal heart rate patterns and retroplacental clots/haemorrhage	9 (10.5)

* Data are presented as No. (%) of patients

p=0.014), chronic lung disease (8.0% vs 0%, p=0.014), retinopathy of prematurity (8.0% vs 0%, p=0.014), patent ductus arteriosus (12.5% vs 0%, p=0.001), neonatal hypoglycaemia (13.6% vs 3.5%, p=0.017), neonatal sepsis (11.4% vs 2.3%, p=0.019), and increased neonatal hospital stay (median, 7 vs 2 days, p<0.001). There were three (3.4%) early neonatal deaths because of prematurity in the placental abruption group but none in the control group.

15 of the patients had 17 subsequent pregnancies delivered beyond 24 weeks. Three of them had placental abruption; the incidence increased to 17.6% among women with a history of abruption. These three cases were not delivered in our hospital and therefore not included in our cohort.

16 of 79 patients were found to have a Couvelaire uterus intraoperatively by the operating surgeon through visual inspection. All four patients who needed secondary intervention for postpartum haemorrhage underwent compression sutures. Compared with patients without a Couvelaire uterus, patients with a Couvelaire uterus had higher rates of uterine atony (56.3% vs 27.0%, p=0.026), postpartum haemorrhage (93.8% vs 61.9%, p=0.014), disseminated intravascular coagulation (25.0% vs 3.2%, p=0.014), blood transfusion (68.8% vs 17.5%, p<0.001),

Table 2. Characteristics of patients with placental abruption and controls

Characteristic	Patients with placental abruption (n=86)*	Controls (n=86)*	p Value
Maternal age, y	32.4±5.0	30.9±5.4	0.052
Maternal age ≥35 y	30 (34.9)	19 (22.1)	0.063
Maternal age <20 y	1 (1.2)	1 (1.2)	>0.99
Body mass index, kg/m^2	22.7 (5)	22.6 (5)	0.930
Maternal ethnicity			0.496
Chinese	80 (93.0)	83 (96.5)	
South-Asian	6 (7.0)	3 (3.5)	
Tobacco use	6 (7.0)	3 (3.5)	0.496
Alcohol use	2 (2.3)	3 (3.5)	>0.99
Drug abuser	1 (1.2)	1 (1.2)	>0.99
Educational level			0.426
Tertiary or above	33 (38.4)	28 (32.6)	
Below tertiary	53 (61.6)	58 (67.4)	
Unmarried	12 (14.0)	17 (19.8)	0.309
Gravidity	2 (2)	2 (2)	0.266
Parity	1 (1)	1 (1)	0.594
Parity ≥3	6 (7.0)	4 (4.7)	0.515
Nulliparity	35 (40.7)	40 (46.5)	0.442
History of miscarriage/stillbirth	18 (20.9)	9 (10.5)	0.059
History of termination of pregnancy	18 (20.9)	21 (24.4)	0.585
History of caesarean section	21 (24.4)	11 (12.8)	0.050
History of placental abruption	4 (4.7)	0	0.121
Assisted conception	6 (7.0)	2 (2.3)	0.277
Twin pregnancy	3 (3.5)	0	0.246
No antenatal care	0	3 (3.5)	0.246
Hypertensive disorders	12 (14.0)	5 (5.8)	0.074
Chronic hypertension	5 (5.8)	1 (1.2)	0.210
Pregnancy induced hypertension	4 (4.7)	3 (3.5)	>0.99
Pre-eclampsia	10 (11.6)	3 (3.5)	0.043
Diabetes mellitus in pregnancy	13 (15.1)	9 (10.5)	0.361
Pre-existing diabetes mellitus	3 (3.5)	0	0.246
Gestational diabetes mellitus	10 (11.6)	9 (10.5)	0.808
Preterm premature rupture of membranes	14 (16.3)	3 (3.5)	0.005
Polyhydramnios	1 (1.2)	0	>0.99
Oligohydramnios	1 (1.2)	2 (2.3)	>0.99
Uterine anomaly	1 (1.2)	0	>0.99
Placental praevia	5 (5.8)	0	0.059
History of antepartum haemorrhage	18 (20.9)	6 (7.0)	0.008
History of abdominal trauma	1 (1.2)	0	>0.99
Use of aspirin	9 (10.5)	0	0.003
Anaemia	3 (3.5)	4 (4.7)	>0.99

* Data are presented as mean ± standard deviation, median (interquartile range), or No. (%) of participants

Table 3. Risk factors for placental abruption

Variable	Adjusted odds ratio (95% confidence interval)	p Value
History of antepartum haemorrhage	3.59 (1.31-9.81)	0.013
Preterm premature rupture of membranes	3.51 (0.89-13.83)	0.072
Pre-eclampsia	2.44 (0.58-10.24)	0.222
Use of aspirin	-	>0.99

Table 4. Maternal outcomes of patients with placental abruption and controls

Maternal outcome	Patients with placental abruption (n=86)*	Controls (n=86)*	p Value
Mode of delivery			
Caesarean section	79 (91.9)	20 (23.3)	<0.001
Normal vaginal delivery	3 (3.5)	63 (73.3)	<0.001
Assisted vaginal delivery	4 (4.7)	3 (3.5)	>0.99
Caesarean section for non-reassuring fetal heart rate	29 (33.7)	6 (7.0)	<0.001
Blood loss, ml	700 (700)	200 (213)	<0.001
Postpartum haemorrhage of ≥500 ml	54 (62.8)	13 (15.1)	<0.001
Postpartum haemorrhage of ≥1000 ml	27 (31.4)	3 (3.5)	<0.001
Uterine atony	27 (31.4)	3 (3.5)	<0.001
Need for secondary intervention for postpartum haemorrhage	5 (5.8)†	0	0.059
Need for blood transfusion	22 (25.6)	3 (3.5)	<0.001
Hysterectomy	1 (1.2)	0	>0.99
Need for relaparotomy	0	0	-
Transfer to intensive care unit	4 (4.7)	0	0.121
Disseminated intravascular coagulation	6 (7.0)	0	0.029
Death	0	0	-
Length of maternal hospital stay, d	4 (2)	3 (2)	<0.001
Prolonged maternal hospitalisation of >4 d	26 (30.2)	13 (15.1)	0.018

* Data are presented as No. (%) of participants or median (interquartile range)

† Compression sutures (n=4) and bilateral uterine artery ligation (n=1)

and secondary intervention (25.0% vs 1.6%, p=0.005) [Table 6]. Compared with neonates born from patients without a Couvelaire uterus, neonates born from patients with a Couvelaire uterus had higher rates of acidosis (umbilical cord blood pH <7.1) [53.3% vs 5.8%, p<0.001], lower Apgar score at 1 minute (25.0% vs 4.8%, p=0.028), and hypoxic-ischaemic encephalopathy (12.5% vs 0%, p=0.039) [Table 6].

Discussion

The worldwide incidence of placental abruption is 0.3% to 1%[11]; the incidence in our cohort was 0.37%. Having a previous placental abruption is the biggest risk factor, with 10- to 15-fold higher recurrence risk[12]. Recurrent placental abruption is associated with earlier gestational age at delivery, compared with first episodes, although perinatal mortality rates are similar[13]. In our cohort, the incidence of recurrence was 17.6%, but the number of patients with subsequent pregnancies was only 15.

Other risk factors for placental abruption include pre-eclampsia (5-fold increased risk[14]), fetal growth restriction, non-vertex presentations, polyhydramnios, advanced maternal age, multiparity, low body mass index, pregnancy following assisted reproductive techniques, intrauterine infection, preterm prelabour rupture of membranes, abdominal trauma, smoking and drug misuse

Table 5. Perinatal and neonatal outcomes of patients with placental abruption and controls

	Patients with placental abruption (n=89)*	Controls (n=86)*	p Value
Perinatal outcome			
Gestational age at birth, wk	35 (7)	39 (2)	<0.001
Birth before 37 weeks	57 (64.0)	8 (9.3)	<0.001
Birth before 34 weeks	33 (37.1)	2 (2.3)	<0.001
Birth before 28 weeks	10 (11.2)	0	0.002
Livebirth	85 (95.5)	86 (100)	0.121
Stillbirth	1 (1.1)	0	>0.99
Early neonatal death	3 (3.4)	0	0.246
Male sex	52 (58.4)	39 (45.3)	0.083
Birth weight, g	2296.4±835.7	3088.8±469.6	<0.001
Birth weight <10 centile	9 (10.1)	13 (15.1)	0.318
Neonatal outcome			
Apgar score at 1 min	7 (3)	8 (0)	<0.001
Apgar score at 1 min <4	7 (7.9)	0	0.014
Apgar score at 5 min	9 (2)	9 (0)	0.055
Apgar score at 5 min <7	7 (7.9)	1 (1.2)	0.064
Umbilical artery pH <7.1	11/73 (15.1)	-	-
Resuscitation at birth	16 (18.2)	0	<0.001
Mechanical ventilation	22 (25.0)	1 (1.2)	<0.001
Admission to neonatal intensive care unit	42 (47.7)	1 (1.2)	<0.001
Haemodynamic instability required inotropes	11 (12.5)	0	0.001
Need for blood transfusion	15 (17.0)	0	<0.001
Respiratory distress syndrome	35 (39.8)	0	<0.001
Apnoea of prematurity	22 (25.0)	0	<0.001
Transient tachypnoea of the newborn	7 (8.0)	3 (3.5)	0.330
Intraventricular haemorrhage	7 (8.0)	0	0.014
Hypoxic-ischaemic encephalopathy	2 (2.3)	0	0.497
Necrotising enterocolitis	3 (3.4)	0	0.246
Chronic lung disease	7 (8.0)	0	0.014
Retinopathy of prematurity	7 (8.0)	0	0.014
Patent ductus arteriosus	11 (12.5)	0	0.001
Hypoglycaemia	12 (13.6)	3 (3.5)	0.017
Jaundice-required phototherapy	21 (23.9)	18 (20.9)	0.643
Sepsis	10 (11.4)	2 (2.3)	0.019
Epilepsy	2 (2.3)	1 (1.2)	>0.99
Cerebral palsy	2 (2.3)	0	0.497
Length of hospital stay, d	7 (32)	2 (2)	<0.001
Early death	3 (3.4)	0	0.246

* Data are presented as mean ± standard deviation, median (interquartile range), or No. (%) of participants

Table 6. Adverse maternal and neonatal outcomes in patients with placental abruption with or without a Couvelaire uterus

	% of patients		p Value
	Patients with Couvelaire uterus (n=16)	Patients without Couvelaire uterus (n=63)	
Adverse maternal outcomes			
Uterine atony	56.3	27.0	0.026
Disseminated intravascular coagulation	25.0	3.2	0.014
Postpartum haemorrhage	93.8	61.9	0.014
Blood transfusion	68.8	17.5	<0.001
Secondary intervention	25.0	1.6	0.005
Admission to intensive care unit	6.3	4.8	>0.99
Adverse neonatal outcomes			
Birth before 28 weeks	18.8	7.9	0.348
Early neonatal death	6.3	1.6	0.366
Umbilical cord blood pH <7.1	53.3	5.8	<0.001
1-minute Apgar score <4	25.0	4.8	0.028
5-minute Apgar score <7	18.8	6.3	0.143
Resuscitation at birth	31.3	15.9	0.171
Admission to neonatal intensive care unit	62.5	44.4	0.197
Hypoxic-ischaemic encephalopathy	12.5	0	0.039
Cerebral palsy	6.3	1.6	0.366
Epilepsy	6.3	1.6	0.366

(cocaine and amphetamines) during pregnancy[15]. In our cohort, other risk factors for placental abruption were pre-eclampsia, preterm prelabour rupture of membranes, antepartum haemorrhage, and the use of antenatal aspirin, but these became not significant after adjusting for confounders, except for a history of antepartum haemorrhage.

In a systematic review, patients with chronic hypertension with superimposed pre-eclampsia have 2.8-7.7-fold increased risk of placental abruption[16]. Severe pre-eclampsia is a strong risk factor for placental abruption, whereas transient hypertension in pregnancy and mild pre-eclampsia are associated with placental abruption. The common aetiology of placental abruption and pre-eclampsia is related to failed placentation in early pregnancy, which may lead to placental dysfunction and further increased risk of abruption. Among patients with preterm prelabour rupture of membranes, the incidence of placental abruption is 4% to 12%[16]. The association is due to either a sudden reduction of uterine volume or an ascending intrauterine infection.

Among patients who used antenatal low-dose aspirin, nine (10.5%) had placental abruption. In a meta-analysis, prophylactic low-dose aspirin (<100 mg per day) has no effect on the risk of placental abruption or antepartum haemorrhage, irrespective of the gestational age at onset of therapy[17]. However, when the dose is ≥100 mg per day, the risk of placental abruption is lower in women who started treatment before 16 weeks than after 16 weeks. Placental abruption and preeclampsia are due to impaired placentation; aspirin administration for women with an increased risk of impaired placentation may reduce the risk of placental abruption, as it does for preeclampsia. The ASPRE trial recommends that a daily dose of ≥100 mg before 16 weeks of gestation is effective in reducing the risk of preeclampsia[18]. However, the risk of placental abruption or antepartum haemorrhage may increase without reducing the risk of preeclampsia if treatment is started after 16 weeks of gestation, because placentation is mostly complete by 18 weeks of gestation. In patients with persistent abnormal placentation, the use of aspirin (through its antiplatelet properties) can increase the risk of haemorrhage and placental abruption. In our patients, all were on 80 mg aspirin daily started before 16 weeks, and antenatal aspirin use was not found to be a risk factor.

Smoking and drug abuse is a risk factor for placental abruption but was not significant in our study, probably owing to the small sample size and confounding factors. Of 172 patients in our study, only nine were smokers and only two were drug abusers, whereas only one had polyhydramnios and only one had abdominal trauma.

There are no universal diagnostic criteria for placental abruption. The most common clinical presentation leading to the diagnosis of placental abruption is the presence of retroplacental clot/haemorrhage during delivery[19]. 96.5% of our patients with placental abruption had this clinical presentation.

Ultrasound is useful to rule out other causes of antepartum haemorrhage and abdominal pain such as placenta previa and adnexal masses. It is useful in diagnosing retroplacental haematomas, with a positive predictive value of 88% and a sensitivity of 25% to 60%, as it is absent in many patients with placental abruption[20-23]. It is difficult to differentiate a concealed haemorrhage of the surrounding placental tissue during the acute phase of placental abruption, as the blood may be isoechoic. The locations of placental abruption can be subchorionic, retroplacental, and preplacental. Retroplacental haematomas with variable appearance is a common ultrasound finding (Figure 1). They may appear to be solid, complex, hypo-, hyper- or iso-echoic, compared with the placenta, depending on the severity and timing of the abruption. The absence of a positive ultrasound finding does not exclude the diagnosis of abruption.

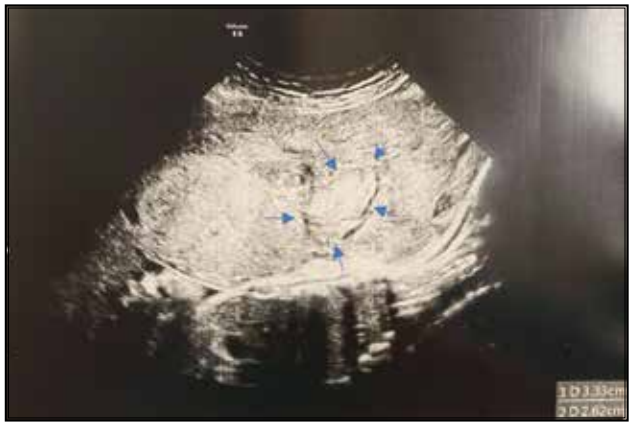

Figure 1. An ultrasound scan showing a retroplacental hematoma (outlined by arrows).

Placental abruption may lead to emergency caesarean birth for fetal or maternal indications, excessive blood loss, and disseminated intravascular coagulation, which can lead to hypovolaemic shock, acute kidney injury, multiorgan failure, respiratory distress syndrome, peripartum hysterectomy, and death[16,24]. In our study, placental abruption was associated with higher rates of caesarean sections, postpartum haemorrhage, uterine atony, blood transfusion, and disseminated intravascular coagulation.

16 of our patients with placental abruption were found to have a Couvelaire uterus intraoperatively (Figure 2), which occurs when a ruptured decidual spiral artery causing bleedings into the decidua basalis and the myometrium during a severe placental abruption. As blood permeates into the uterine serous layer, a blue-violet ecchymosis occurs[25]. The Couvelaire uterus is atonic and very prone to postpartum haemorrhage and thus aggressive and timely management of atony may prevent further worsening of conditions such as disseminated intravascular coagulation and exsanguination. Uterine atony in a

Figure 2. Intra-operative finding of Couvelaire uterus in two patients.

Couvelaire uterus responds less well to standard treatments and is at high risk of hysterectomy. In our patients with a Couvelaire uterus, the rates of uterine atony, postpartum haemorrhage, disseminated intravascular coagulation, and blood transfusion were all higher. Of the five patients who underwent secondary intervention, three of the four patients with compression sutures were those with a Couvelaire uterus. Therefore, it is crucial for surgeons to anticipate the risk of worsened outcomes when a Couvelaire uterus is noted intraoperatively and be proactive in the prevention of deterioration. The more severe the placental abruption, the worse the neonatal outcome.

In our study, there was no maternal death. This may be due to the timely diagnosis and prompt treatment of postpartum haemorrhage by a multidisciplinary team. High vigilance towards uterine atony and postpartum haemorrhage and timely involvement of other specialties (neonatologists, intensivists, anaesthesiologists, and haematologists) and massive transfusion protocol for expeditious transfusion and support are important.

Adverse perinatal outcomes of pregnancies complicated with placental abruption include low Apgar score, preterm birth, lower birth weight, and perinatal mortality[2,11,26-34]. 64% of our patients with placental abruption were delivered prematurely. The risk of preterm birth may be related to preterm labour or preterm prelabour rupture of membranes or a non-reassuring fetal or maternal condition. Neonatal morbidities are associated with both complications of placental abruption and prematurity[2]. In our cohort, neonates born from patients with placental abruption had an increased (but not significantly) risk of acidosis (15%) and cerebral palsy and hypoxic ischaemic encephalopathy. These problems are more likely to be attributable to the acute event of placental abruption leading to perinatal asphyxia than prematurity.

Limitations of the present study include the retrospective nature and the small sample size. The incidence of placental abruption may be underestimated, as antepartum haemorrhage of unknown origin can be marginal bleeding of the placenta, which may be a type of very minor abruption that was not included. 64% of neonates born from patients with placental abruption were preterm, and the neonatal outcome was not controlled for gestation at delivery. Thus, the placental abruption group had higher neonatal morbidities that could be associated with prematurity (rather than placental abruption alone).

Conclusion

Clinicians should be vigilant for placental abruption in patients with antepartum haemorrhage, especially in high-risk patients with a history of placental abruption, hypertension, or pre-eclampsia. Early and consistent antenatal care is imperative to identify those with risk factors. Proper education and timely preventive management should be provided to improve maternal and fetal outcomes.

Contributors

All authors designed the study, acquired the data, analysed the data, drafted the manuscript, and critically revised the manuscript for important intellectual content. All authors had full access to the data, contributed to the study, approved the final version for publication, and take responsibility for its accuracy and integrity.

Conflicts of interest

All authors have disclosed no conflicts of interest.

Funding/support

This study received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Data availability

All data generated or analysed during the present study are available from the corresponding author upon reasonable request.

Ethics approval

The study was approved by the New Territories West Research Ethics Committee (reference: NTWC/REC/22071)). The patients were treated in accordance with the tenets of the Declaration of Helsinki. The patients provided written informed consent for all treatments and procedures and for publication.

References

1. Maeland KS, Morken NH, Schytt E, Aasheim V, Nilsen RM. Placental abruption in immigrant women in Norway: a population-based study. Acta Obstet Gynecol Scand 2021;100:658-65. Crossref

2. Lueth A, Blue N, Silver RM, et al. Prospective evaluation of placental abruption in nulliparous women. J Matern Fetal Neonatal Med 2022;35:8603-10. Crossref
3. Sheiner E. Textbook of Perinatal Epidemiology. Nova Science; 2010.
4. Gaufberg SA. Abruptio placentae from emergency medicine/obstetrics and gynaecology. e-Med J 2001;2(3).
5. Bonnar J. Massive obstetric haemorrhage. Baillieres Best Pract Res Clin Obstet Gynaecol 2000;14:1-18. Crossref
6. Spinillo A, Fazzi E, Stronati M, Ometto A, Lasci A, Guaschino S. Severity of abruptio placentae and neurodevelopmental outcome in low birth weight infants. Early Hum Dev 1993;35:45-54. Crossref
7. Ananth CV, Oyelese Y, Yeo L, Pradhan A, Vintzileos AM. Placental abruption in the United States, 1979 through 2001: temporal trends and potential determinants. Am J Obstet Gynecol 2005;192:191-8. Crossref
8. Ananth CV, Smulian JC, Demissie K, Vintzileos AM, Knuppel RA. Placental abruption among singleton and twin births in the United States: risk factor profiles. Am J Epidemiol 2001;153:771-8. Crossref
9. Jenabi E, Ebrahimzadeh Zagami S. The association between uterine leiomyoma and placenta abruption: a meta-analysis. J Matern Fetal Neonatal Med 2017;30:2742-6. Crossref
10. Panagiotopoulos M, Tseke P, Michala L. Obstetric complications in women with congenital uterine anomalies according to the 2013 European Society of Human Reproduction and Embryology and the European Society for Gynaecological Endoscopy Classification: a systematic review and meta-analysis. Obstet Gynecol 2022;139:138-48. Crossref
11. Sheiner E, Shoham-Vardi I, Hadar A, Hallak M, Hackmon R, Mazor M. Incidence, obstetric risk factors and pregnancy outcome of preterm placental abruption: a retrospective analysis. J Matern Fetal Neonatal Med 2002;11:34-9. Crossref
12. Ananth CV, Savitz DA, Williams MA. Placental abruption and its association with hypertension and prolonged rupture of membranes: a methodologic review and meta-analysis. Obstet Gynecol 1996;88:309-18. Crossref
13. Kojima T, Takami M, Shindo R, Saigusa Y, Miyagi E, Aoki S. Perinatal outcomes of recurrent placental abruption. J Matern Fetal Neonatal Med 2021;34:2192-6. Crossref
14. Bellos I, Pergialiotis V, Papapanagiotou A, Loutradis D, Daskalakis G. Comparative efficacy and safety of oral antihypertensive agents in pregnant women with chronic hypertension: a network metaanalysis. Am J Obstet Gynecol 2020;223:525-37. Crossref
15. Ananth CV, Patrick HS, Ananth S, Zhang Y, Kostis WJ, Schuster M. Maternal cardiovascular and cerebrovascular health after placental abruption: a systematic review and meta-analysis (CHAP-SR). Am J Epidemiol 2021;190:2718-29. Crossref
16. Tikkanen M. Placental abruption: epidemiology, risk factors and consequences. Acta Obstet Gynecol Scand 2011;90:140-9. Crossref
17. Roberge S, Bujold E, Nicolaides KH. Meta-analysis on the effect of aspirin use for prevention of preeclampsia on placental abruption and antepartum hemorrhage. Am J Obstet Gynecol 2018;218:483-9. Crossref
18. Roberge S, Bujold E, Nicolaides K. Aspirin for the prevention of preterm and term preeclampsia : systematic review and metaanalysis. Am J Obstet Gynecol 2018;218:287-93. e1. Crossref
19. Elsasser DA, Ananth CV, Prasad V, Vintzileos AM; New Jersey-Placental Abruption Study Investigators. Diagnosis of placental abruption: relationship between clinical and histopathological findings. Eur J Obstet Gynecol Reprod Biol 2010;148:125-30. Crossref
20. Shinde GR, Vaswani BP, Patange RP, Laddad MM, Bhosale RB. Diagnostic performance of ultrasonography for detection of abruption and its clinical correlation and maternal and foetal outcome. J Clin Diagn Res 2016;10:QC04-7. Crossref
21. Glantz C, Purnell L. Clinical utility of sonography in the diagnosis and treatment of placental abruption. J Ultrasound Med 2002;21:837-40. Crossref
22. Sholl JS. Abruptio placentae: clinical management in nonacute cases. Am J Obstet Gynecol 1987;156:40-51. Crossref
23. Jaffe MH, Schoen WC, Silver TM, Bowerman RA, Stuck KJ. Sonography of abruptio placentae. AJR Am J Roentgenol 1981;137:1049-54. Crossref
24. Oyelese Y, Ananth CV. Placental abruption. Obstet Gynecol 2006;108:1005-16. Crossref
25. Lee YJ, Calvert K, Jape K. Couvelaire uterus resulting in haemoperitoneum. J Surg Case Rep 2022;2022:rjab618. Crossref
26. Ananth CV, Berkowitz GS, Savitz DA, Lapinski RH. Placental abruption and adverse perinatal outcomes. JAMA 1999;282:1646-51. Crossref
27. Ananth CV, Wilcox AJ. Placental abruption and perinatal mortality in the United States. Am J Epidemiol 2001;153:332-7. Crossref
28. Ananth CV, Smulian JC, Srinivas N, Getahun D, Salihu HM. Risk of infant mortality among twins in relation to placental abruption: contributions of preterm birth and restricted fetal growth. Twin Res Hum Genet 2005;8:524-31. Crossref
29. Raymond EG, Mills JL. Placental abruption. Maternal risk factors and associated fetal conditions. Acta Obstet Gynecol Scand 1993;72:633-9. Crossref
30. Rasmussen S, Irgens LM, Bergsjo P, Dalaker K. Perinatal mortality and case fatality after placental abruption in Norway 1967-1991. Acta Obstet Gynecol Scand 1996;75:229-34. Crossref
31. Tikkanen M, Luukkaala T, Gissler M, et al. Decreasing perinatal mortality in placental abruption. Acta Obstet Gynecol Scand 2013;92:298-305. Crossref
32. Faiz AS, Demissie K, Rich DQ, Kruse L, Rhoads GG. Trends and risk factors of stillbirth in New Jersey 1997-2005. J Matern Fetal Neonatal Med 2012;25:699-705. Crossref
33. Brailovschi Y, Sheiner E, Wiznitzer A, Shahaf P, Levy A. Risk factors for intrapartum fetal death and trends over the years. Arch Gynecol Obstet 2012;285:323-9. Crossref
34. Downes KL, Shenassa ED, Grantz KL. Neonatal outcomes associated with placental abruption. Am J Epidemiol 2017;186:1319-28. Crossref