Placental abruption leading to disseminated intravascular coagulation: a clinical case and short review

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We present the case of a primigravida with disseminated intravascular coagulation at 21 weeks' gestation. Furthermore, we performed a short review of the evidence-based management of the condition. The patient presented with pain and vaginal bleeding. Clinical examination, laboratory studies, and an abdominal ultrasound produced inconclusive results about the origin of her disseminated intravascular coagulation. She was transferred to a tertiary facility where blood and plasma product transfusions were performed, and further investigations revealed fetal demise caused by placental abruption as the underlying cause of her disseminated intravascular coagulation. Cervical preparation was conducted with a balloon catheter and misoprostol. Surgical evacuation of her uterus was performed and she made a full recovery.

Key words: blood component transfusion, cervical ripening, disseminated intravascular coagulation, misoprostol, placental abruption

Introduction

The expected presentation of placental abruption is with severe abdominal pain, vaginal bleeding, and a compromised fetus, usually in the third trimester. However, the condition can occur earlier in pregnancy and have an atypical presentation. Because of severe maternal morbidity caused by abruption, a hysterotomy has been reported to be appropriate treatment.[1] We present a case managed with uterine evacuation and judicious provision of blood products.

Patient information

A 25-year-old primigravida was transferred to our tertiary referral hospital. The patient had presented a day earlier at 21 weeks' gestation with abdominal pain and vaginal spotting. She was clinically stable and diagnosed with a urinary tract infection and was discharged with antibiotic treatment. A second presentation later that day with pain and bleeding provoked further workup.

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This is a case report, and the division leadership is aware of the submission. The patient has provided written consent for this case report to be written and submitted.

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Clinical findings

A live fetus was noted during an ultrasound examination. The growth was within normal limits overall, but a small abdominal circumference was noted. The placenta was noted to not be low lying, and no evidence of abruption was reported. The patient's laboratory findings demonstrated evidence of disseminated intravascular coagulation (DIC) (Table). The patient received intravenous ceftriaxone and metronidazole because her white blood cell count was increased. The patient was transfused with 2 units of packed red cells, 4 units of fibrinogen-rich cryoprecipitate, 2 units of fresh frozen plasma, and 1 unit of platelets without improvement of coagulopathy. Because of the marked coagulopathy, a bone marrow aspirate was collected to exclude a leukemic process as the cause of DIC. The vaginal bleeding increased, and the patient's abdominal pain worsened, raising concern for preterm labor. The patient was transferred to a tertiary unit on day 2 of admission with a diagnosis of DIC of unknown origin and preterm labor.

Timeline

The obstetrical consultant was asked to provide an opinion at transfer to the tertiary care facility. The diagnostic process and treatment occurred over the following timeline.

Diagnostic assessment

On arrival, the patient was in obvious discomfort. Observations revealed a pulse of 70 beats per minute, blood pressure of 100/55 mm Hg, and O_2 saturation of 97% on room air. The patient's uterus was tight, tender, and was approximated to be that of a 32-week gestation uterus. A bedside ultrasound revealed fetal demise and large collections presumed to be a blood clot. Cervical examination revealed that the cervix was closed, uneffaced, at −2 station, hard, and anterior. A large, firm mass was palpated at the vaginal apex and assumed to be the distended uterus. Further laboratory findings confirmed DIC (Table).

Therapeutic intervention

Treatment goals were set to achieve a hemoglobin level of >10.0 g/L, fibrinogen level of >200 mg/dL, and platelet level of $>70\times10^9$g/L. Initial replacement was with 4 units fibrinogen-rich cryoprecipitate, 2 units of packed red blood cells, and 1 unit of platelets.

TABLE
Hematologic findings in relation to timing of the clinical events

Parameter	Early pregnancy bloods	Day 1 6:40 PM (presentation)		Day 1 7:30 PM	Day 1 7:50 PM	Day 2 4 AM	Day 2 11:03 AM	Day 2 1:30 PM	Day 2 3:45 PM	
Hemoglobin g/dL (10–14.5)	12.9	10.4		9.9	10.1	8.6	7.1		8.4	
Platelets g/L (150–400)	361	61		59	62	47	83		58	
APTT s (22.0–34.0)				46	43	49	34	37	42	
Fibrinogen mg/dL (180–400)				30	30	40	100	100	80	
D dimer μg/L (<500)						>4000				
	Transfer to tertiary unit	Day 2 8:20 PM		Day 2 10:35 PM	Day 3 2:45 AM	Delivery	Day 3 7:30 AM	Day 3 12:10 PM	Day 4 9:45 AM	Day 5 9 AM	Follow-up results
Hemoglobin g/dL (10.0–14.5)		7.2		8.4	9.5		11.0	11.0	11.2	10.5	13.0
Platelets g/L (150–400)		43		42	85		83	85	129	183	585
APTT s (22.0–4.0)		38		39	36		36	35	28	29	
Fibrinogen g/L (1.8–4.0)		160		150	230		230	230	240	230	

APTT, activated partial thromboplastin time.

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Presuming the DIC was caused by a consumptive coagulopathy owing to abruption, expedient evacuation of the uterus was a priority. While being transfused, 400 μg of buccal misoprostol was administered and repeated every 3 hours for a total of 4 doses. After the second dose, the cervix was dilated and soft enough to permit passage of a Foley catheter. During direct visualization, a couvelaire sign was noted on the cervix, indicating infiltration of the tissues with blood.[2] The catheter was inflated to 30 mL and attached to a 1-liter intravenous bag that was hung off the bed to apply constant traction. Regional anesthesia was contraindicated because of the platelet count and coagulopathy. A patient-controlled morphine pump was used for pain relief. The Foley was expelled in 2 hours when a cervical dilatation of 3 cm was noted at that time. Considering a cephalic presenting part, a decision was made to perform an amniotomy and to await delivery instead of performing a dilatation and evacuation procedure because a small uterine or cervical laceration would likely bleed profusely.[3] After 2 further hours, the patient felt the need to push. She was transferred to the operating room where a seemingly normal stillborn fetus was delivered weighing 340 g. Fundal massage led to rapid evacuation of a 1300 mL blood clot. The blood clot was adherent to the entire placental surface. A general anesthetic was administered and suction curettage was performed. Sharp curettage was avoided to minimize the chance of uterine perforation and Asherman syndrome.[4,5] A mechanical tamponade was not needed, nor was a local injection of dilute vasopressin to decrease blood loss, which is an option of care before a dilatation and evacuation procedure.[3] Rectal 1000 μg misoprostol was given. The total blood loss was 2000 mL. The total transfusion support given in our center was 8 units fibrinogen-rich cryoprecipitate, 5 units packed red cells, and 1 unit of platelets.

Follow-up and outcomes

The patient made a full recovery after uterine evacuation. She was discharged

to the care of her general practitioner with instructions to have a follow-up consultation for debriefing and future pregnancy planning. She was also instructed to have a repeat consultation with a high-risk obstetrician about any future pregnancy.

Discussion

Our case highlights several important points, including the challenges of diagnosing placental abruption, uterine evacuation procedures, management of DIC, and obstetrical hemorrhage.

Challenges in diagnosis of placental abruption

Although in retrospect it is apparent that placental abruption caused both the presenting symptoms and coagulopathy, it is important to recognize the strong element of hindsight bias introduced following a poor clinical outcome.[6] Abruption in the second trimester that leads to profound maternal coagulopathy is an uncommon event. Moreover, placental abruption is often accompanied by significant vaginal bleeding. In this case, the abruption initially led only to vaginal spotting, which confused the clinical picture. Although the correct diagnosis initially eluded the clinicians caring for this patient, their significant concern led to multiple investigations and ultimately to transfer to a tertiary care facility.

Placental abruption is a common obstetrical complication.[7,8] The diagnosis is a clinical one, although it can frequently be confirmed on gross inspection of the placenta postpartum and pathologic examination. Risk factors include previous abruption, maternal hypertension, low body mass index, and maternal smoking or drug use, which were not present in this patient.[7] Her symptoms of cramping and bleeding are common in pregnancy, and their perceived severity are subjective. Objective testing that may have guided the clinicians would have been a Kleihauer test, although this is not a guideline-based recommendation.[7] Indeed, a Kleihauer test was performed after her delivery and was negative. However, fetal red cells in the maternal circulation may be present in other patients with similar symptoms and would raise suspicion of abruption. A formal ultrasound was performed, and there was a live fetus. This confused the diagnosis because significant abruption often leads to fetal demise.[8,9] Ultrasonography is a poor tool for identification of placental abruption.[7,10] However, if an abruption is suspected on ultrasound because of the presence of subchorionic hematoma or retroplacental clot, the chance of an abruption is high, making a suggestive finding on ultrasound important.[7,10] The placenta in this case measured up to 8 cm in thickness (Figure) and seemed abnormally thick on an informal bedside ultrasound when the patient presented to our center. A placenta measuring >4 cm in width should be viewed with concern, especially in the second trimester.[11] Placental abruption is not the only cause of placentomegaly but should be considered when the finding is present.

FIGURE
Ultrasound image

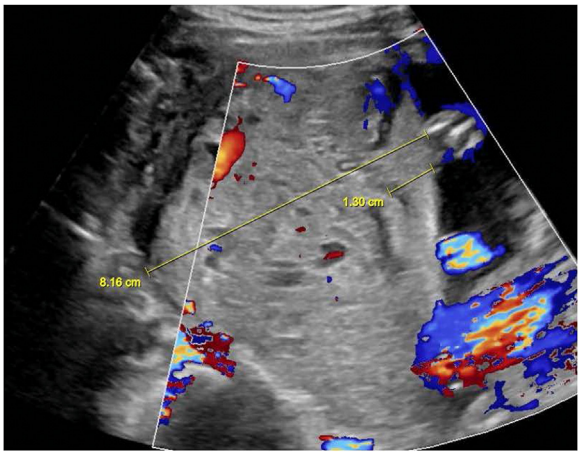

Image shows uterus on the day of transfer. The *large measurement* is the width of the area presumed to be placenta at 8.16 cm. The *small measurement* is the suspected actual placental width at 1.3 cm, lifted off the myometrium by what was later confirmed to be blood.

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Uterine evacuation

In relation to treatment when the abruption was recognized, uterine evacuation was a priority. Hysterotomy was an option and is expedient.[1] However, this invasive approach was reserved as a last resort considering the gestational age and the fact that the fetus was demised. The scarring from a hysterotomy has long-term effects for the reproductive health of the patient and we would caution that a laparotomy in patients with DIC frequently results in bleeding from multiple sites.[12] In a primigravid patient, cervical preparation for dilatation and evacuation is frequently a prolonged process, often with laminaria.[3] Instead, misoprostol was prescribed regularly.[3] Use of a Foley catheter in combination with misoprostol may be the best option when cervical dilatation is required expediently.[3] We were fortunate that the patient delivered within 12 hours of the start of cervical ripening and had an uneventful uterine instrumentation. This is consistent with existing data that suggest a correlation between time to birth and blood loss during placental abruption, with labors being shorter as blood loss increases.[13] Contingency plans were in place for either a Foley or Bakri balloon tamponade to be placed in the uterus in combination with local dilute vasopressin injection if bleeding continued postprocedure.[3]

Management of obstetrical DIC

DIC is a life-threatening condition. Obstetrical causes include placental abruption, as occurred here.[14] Cases of

concealed hemorrhage that can occur with abruption confuse the diagnosis, especially when a live fetus is present. Correction of the cause of the DIC and identification of the underlying cause is paramount,[14] which, in this case, involved placental removal and uterine involution. Without ongoing hemorrhage, judicious replacement to improve laboratory parameters, as was performed here, is indicated.[7] Medical management of coagulopathy was central to the treatment of the patient.[14,15] The fibrinogen deficiency of DIC is best treated with cryoprecipitate because it contains the highest concentration per unit volume of fibrinogen. Blood products can and should be infused while awaiting laboratory results in cases in which hemorrhage and coagulopathy are clinically evident.[7] Packed red cells were indicated because the patient was anemic with ongoing bleeding into her uterus. They provided volume after her concealed hemorrhage and in preparation for the anticipated blood loss at delivery. Platelets were also infused before the evacuation of the uterus. Ideally, the platelet level should be 50 g/L or above before commencement of surgery.[16]

Obstetrical hemorrhage

Lastly, we feel it was important to reiterate that obstetrical hemorrhage is the leading contributor to maternal mortality worldwide.[17] Although hemorrhage heads the list of causes of maternal mortality, death from abortion is also a significant contributor to maternal mortality internationally.[17] The technique for performing this uterine evacuation with correction of the patient's coagulopathy was vital to the health and well-being of this woman. Importantly, it minimized the operative morbidity that a hysterotomy would confer and did not deprive her of having a pregnancy in the future. Death caused by obstetrical hemorrhage is no longer a major contributor to mortality in New Zealand[18] and remains uncommon in other developed countries[16] because of the availability of blood products, uterotonics, and medical care. However, it remains one of the main contributors to morbidity. We anticipated the potential for life-threatening hemorrhage owing to coagulopathy and corrected this before the obstetrical intervention. This case emphasizes the importance of expediting delivery, managing coagulopathy and hemodynamic instability, and anticipating further hemorrhage as a risk to general and future reproductive health. ■

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