International Journal of Reproduction, Contraception, Obstetrics and Gynecology
Aatif S et al. Int J Reprod Contracept Obstet Gynecol. 2026 Dec;15(12):5037-5040
www.ijrcog.org
pISSN 2320-1770 | eISSN 2320-1789

DOI: https://dx.doi.org/10.18203/2320-1770.ijrcog20264426

Case Report

Uterus preservation in severe obstetric disseminated intravascular coagulation secondary to placental abruption using bilateral uterine artery embolization: a case report

Saira Aatif[1]*, Uzma Sharif[2], Muhammad Aayan Mumtaz[3], Shahad Al Zabi[2]

[1]Danat Al Emarat Hospital Abu Dhabi, United Arab Emirates
[2]Sheikh Khalifa Hospital, Fujairah, United Arab Emirates
[3]Newcastle University, Malaysia

Received: 08 August 2026
Revised: 19 November 2026
Accepted: 20 November 2026

***Correspondence:**
Dr. Saira Aatif,
E-mail: Saira.munir80@gmail.com

Copyright: © the author(s), publisher and licensee Medip Academy. This is an open-access article distributed under the terms of the Creative Commons Attribution Non-Commercial License, which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

ABSTRACT

Placental abruption is a leading obstetric cause of disseminated intravascular coagulation (DIC) and uncontrolled bleeding in this setting often ends in peripartum hysterectomy. A 27-year-old woman in her third pregnancy with two previous caesarean deliveries, presented at 34 weeks 6 days of gestation with sudden severe abdominal pain, a tense and tender uterus and intrauterine fetal death. Emergency caesarean section confirmed complete placental separation and was followed by persistent bleeding of non-clotting blood. Prothrombin time was 27.3 seconds, activated partial thromboplastin time 72.8 seconds and fibrinogen below 0.30 g/L with a markedly raised D-dimer, giving an International Society on Thrombosis and Haemostasis overt DIC score of 7. Bleeding continued despite uterotonics, tranexamic acid and massive transfusion. After abdominal packing, bilateral uterine artery embolization achieved angiographic stasis and re-laparotomy showed a well-contracted uterus without active bleeding. The patient recovered fully and was discharged with her uterus preserved.

Keywords: Placental abruption, Disseminated intravascular coagulation, Uterine artery embolization, Postpartum haemorrhage, Uterine preservation

INTRODUCTION

Placental abruption is the separation of a normally sited placenta from the uterine wall before the birth of the fetus, occurs in approximately 0.4-1% of pregnancies and remains an important cause of maternal morbidity and perinatal death.[1,2] Hypertensive disorders of pregnancy, including pre-eclampsia are among its strongest recognized risk factors with other reasons being a previous caesarean delivery, abruption in an earlier pregnancy, smoking, abdominal trauma and advanced maternal age.[1,3]

Of all obstetric conditions, abruption is the one most closely linked with DIC. Bleeding into the decidua basalis forms a retroplacental hematoma and tissue factor released from the damaged decidua and placenta enters the maternal circulation and drives widespread thrombin generation. Platelets and clotting factors, fibrinogen in particular are then consumed faster than they can be replaced and secondary fibrinolysis further increases the tendency to bleed.[2,4] The risk of coagulopathy is greatest when the separation is extensive and the fetus has died.[2]

When obstetric haemorrhage does not respond to medical treatment, the remaining options are surgical such as compression sutures, vessel ligation or hysterectomy. Uterine artery embolization is an established uterus-sparing technique but coagulopathy is repeatedly identified as a predictor of embolization failure because the occlusive effect of several embolic agents depends

partly on the patient's own ability to form thrombus.[5,6] Successful embolization in the presence of overt DIC has therefore been described only occasionally.[7] We report a woman with severe DIC following placental abruption with fetal death in whom the uterus was preserved by bilateral uterine artery embolization combined with massive transfusion and coordinated multidisciplinary care.

CASE REPORT

A 27-year-old woman in her third pregnancy, who had two previous lower segment caesarean deliveries, presented to the emergency department at 34 weeks 6 days of gestation with sudden onset of severe abdominal pain. Her antenatal course had been uneventful apart from gestational diabetes mellitus controlled by diet alone and no hypertensive disorder had been recorded. A growth scan at 32 weeks 6 days had shown appropriate fetal growth.

On arrival she was in severe pain. Blood pressure was 100/60 mmHg, pulse rate 100 beats/min and respiratory rate 19 breaths/min. There was no vaginal bleeding. The abdomen was tense and the uterus was tender on palpation. Bedside ultrasonography showed absent fetal cardiac activity and a clinical diagnosis of concealed placental abruption with intrauterine fetal death was made. In view of two previous uterine scars and the severity of the concealed abruption, a category 1 emergency caesarean section was performed. Intravenous fluid resuscitation was started, four units each of packed red blood cells and fresh frozen plasma were arranged and samples were sent for complete blood count, coagulation profile, renal and liver function tests and serum electrolytes.

At laparotomy the placenta was completely separated, with a large retroplacental clot. A fresh stillborn baby weighing 2180 g was delivered and the uterine incision was closed in two layers. Generalized oozing nevertheless continued from the incision, the uterine surface and the abdominal wall with the blood being thin and not clotting. The estimated blood loss, including retroplacental clot and vaginal loss was 2000 mL. Coagulation tests confirmed severe consumptive coagulopathy, with an International Society on Thrombosis and Haemostasis (ISTH) overt DIC score of 7 (Table 1).[8]

The massive transfusion protocol was activated (Table 2). Uterotonic treatment consisted of carbetocin, an oxytocin infusion (40 units) and methylergometrine. Tranexamic acid and prothrombin complex concentrate were given, together with cefazolin and metronidazole for antibiotic prophylaxis. Despite these measures, diffuse bleeding continued.

As bleeding persisted and the coagulopathy worsened, care was escalated. A second consultant obstetrician, the consultant anaesthesiologist and the interventional radiology, vascular surgery and intensive care teams were called to the theatre. The patient's husband was informed of the gravity of her condition and of the possible need for uterine artery embolization, ligation of pelvic vessels or hysterectomy. Because the patient was young and her hemodynamic status had improved with transfusion, the team chose a uterus-preserving approach. The abdomen was packed and temporarily closed and she was moved to the angiography suite.

Through a right common femoral artery approach, selective angiography of the left uterine artery showed a hypertrophied, tortuous vessel with marked vascular blush over the uterine body (Figure 1 A). The right uterine artery, catheterized after formation of a Waltman loop, showed a similar appearance (Figure 2 A). Both arteries were embolized with polyvinyl alcohol particles and trisacryl gelatin microspheres, supplemented with gelatin sponge, until stasis was achieved. Completion angiography showed no residual blush on either side (Figures 1 B and 2 B). The laparotomy packs remained in place throughout the procedure.



Figure 1 (A and B): Left uterine artery angiography; (A) Pre-embolization image showing a hypertrophied, tortuous uterine artery with diffuse vascular blush: radio-opaque laparotomy packs are seen in the pelvis; (B) Post-embolization image showing stasis with loss of the vascular blush.

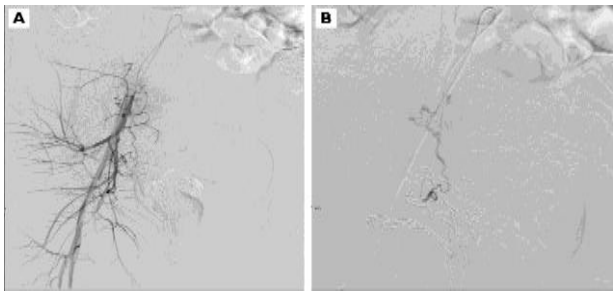


Figure 2 (A and B): Right uterine artery angiography, catheterized using a Waltman loop; (A) Pre-embolization image showing a hypertrophied uterine artery with vascular blush; (B) Post-embolization image showing stasis in the uterine artery with no residual blush.

She was returned to the operating theatre for re-laparotomy. The uterus was well contracted and there was no active bleeding from it; a mild residual ooze from the myometrium was controlled with a thrombin-gelatin hemostatic matrix. Bladder integrity was confirmed by

instillation of methylene blue. The packs were removed, a 19 Fr drain was placed and the abdomen was closed.

The patient was managed in the intensive care unit under strict monitoring. On the first postoperative day she remained intubated and sedated for monitoring. She was hemodynamically stable, her coagulation values were improving and there was no evidence of further intra-abdominal bleeding. By the second postoperative day she had been extubated and was mobilizing, renal function was normal, there was no metabolic acidosis and the only abnormal finding was mild abdominal distension without vaginal bleeding. Thromboprophylaxis with enoxaparin was started once coagulation had normalized. She continued to improve and was discharged in a stable condition on postoperative day.

Patient was kept in ICU under strict monitoring and extubated on 2nd postoperative day.

Table 1: Hematological and coagulation parameters during intraoperative bleeding.

Parameters	Results	ISTH score (points)[a]
Platelet count	91-111 × 10^9/l (serial values)	1
Prothrombin time	27.3 s (INR 2.11)	2[b]
Activated partial thromboplastin time	72.8 s	-
Fibrinogen	<0.30 g/l	1
D-dimer	>20 mg/l FEU	3
Hemoglobin	7.3 g/dl	-
Total ISTH score		7 (overt DIC ≥5)

*[a]International Society on Thrombosis and Haemostasis overt disseminated intravascular coagulation score. [b]Prolongation of ≥6 s over the laboratory reference value. INR: international normalized ratio; FEU: fibrinogen equivalent units.

Table 2: Blood components and hemostatic agents administered.

Products	Amount (units/dose)
Packed red blood cells	6 units
Fresh frozen plasma	4 units
Cryoprecipitate	6 units
Platelet concentrate	6 units
Prothrombin complex concentrate	1 dose
Tranexamic acid	1 g Intravenous

DISCUSSION

This case shows how quickly a concealed abruption can progress to overt consumptive coagulopathy and that the uterus can still be preserved when correction of coagulation and arterial devascularization are carried out in parallel. Abruption accompanied by fetal death carries the highest risk of DIC, reflecting the large quantity of tissue factor released from an extensively separated placenta.[2,4] In our patient the diagnosis rested on both clinical and laboratory grounds i.e. non-clotting blood oozed from every cut surface and the ISTH score of 7 was well above the threshold of 5 for overt DIC.[8] Because fibrinogen rises and clotting times shorten in normal pregnancy, non-pregnant cut-offs can understate obstetric coagulopathy and a pregnancy-adapted score has been proposed[9] by either system, this patient's values were unequivocally abnormal.

A falling fibrinogen level is the earliest and most informative laboratory marker of severe obstetric haemorrhage and values of 2 g/L or less strongly predict progression to severe bleeding.[10] With a fibrinogen concentration below 0.30 g/L, the patient could not form a stable clot which explains why uterotonic drugs alone were ineffective. Current guidance therefore stresses early replacement of fibrinogen with cryoprecipitate or concentrates alongside red cells and plasma and administration of tranexamic acid within 3 hours of birth, which reduces death from bleeding.[11-13] The patient received these components through a massive transfusion protocol, together with platelets and prothrombin complex concentrate.

When the fetus is dead after abruption, vaginal birth is generally preferred if the mother is stable because abdominal delivery in a woman with developing coagulopathy adds the bleeding of a surgical wound.[14] Caesarean section may however be needed for maternal reasons. In this patient the decision was influenced by 2 previous uterine scars, the extent of concealed abruption and the need to achieve delivery and haemostasis without delay.

Pelvic arterial embolization controls postpartum haemorrhage in most women but coagulopathy and the volume of blood transfused before the procedure are among the factors most consistently associated with failure.[5,6] The likely explanation is that gelatin sponge and to a lesser extent particulate agents achieve occlusion partly by inducing local thrombosis, which is impaired when fibrinogen and platelets are depleted. In our patient, stasis was obtained by combining permanent particulate agents (polyvinyl alcohol particles and microspheres) which occlude mechanically with gelatin sponge, while component replacement continued during the procedure. Sugai et al described a woman with postpartum haemorrhage and DIC in whom bleeding was finally controlled by repeated uterine artery embolization, an experience that illustrates both the value of the technique and the risk of recurrent bleeding while coagulopathy persists.[7] Temporary abdominal packing before transfer provided tamponade of the raw surfaces during embolization and the subsequent re-laparotomy allowed haemostasis to be confirmed and the bladder to be checked before definitive closure.

Preserving the uterus is particularly important for a young woman who has just lost her baby. Most women resume menstruation after pelvic embolization for postpartum haemorrhage and successful later pregnancies have been reported, although careful antenatal surveillance is advisable.[15] Rare complications such as uterine ischemia, necrosis and infection should be watched for during follow-up.

The favorable outcome depended on early escalation to a team that included senior obstetricians, anaesthesiology, interventional radiology, vascular surgery, transfusion services and intensive care and on clear communication with the family about each possible step, including hysterectomy. This report has limitations. It describes a single patient; the approach depends on immediate access to interventional radiology and blood components which is not available in every unit and longer-term menstrual and reproductive outcomes have not yet been documented.

CONCLUSION

Severe DIC following placental abruption is a life-threatening complication in which hysterectomy is often considered inevitable. In this patient, prompt recognition of the coagulopathy, early fibrinogen-rich component replacement and bilateral uterine artery embolization, coordinated by a multidisciplinary team controlled the bleeding while preserving the uterus. Where interventional radiology is readily available embolization may be considered even in overt obstetric DIC, provided that correction of coagulopathy proceeds at the same time.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES

1. Tikkanen M. Placental abruption: epidemiology, risk factors and consequences. Acta Obstet Gynecol Scand. 2011;90(2):140-9.
2. Brandt JS, Ananth CV. Placental abruption at near-term and term gestations: pathophysiology, epidemiology, diagnosis, and management. Am J Obstet Gynecol. 2023;228(5S):S1313-29.
3. 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(2):309-18.
4. Erez O, Othman M, Rabinovich A, Leron E, Gotsch F, Thachil J. DIC in pregnancy-pathophysiology, clinical characteristics, diagnostic scores, and treatments. J Blood Med. 2022;13:21-44.
5. Sentilhes L, Gromez A, Clavier E, Resch B, Verspyck E, Marpeau L. Predictors of failed pelvic arterial embolization for severe postpartum hemorrhage. Obstet Gynecol. 2009;113(5):992-9.
6. Poujade O, Zappa M, Letendre I, Ceccaldi PF, Vilgrain V, Luton D. Predictive factors for failure of pelvic arterial embolization for postpartum hemorrhage. Int J Gynaecol Obstet. 2012;117(2):119-23.
7. Sugai S, Nonaka T, Tamegai K, Sato T, Haino K, Enomoto T, et al. Successful repeated uterine artery embolization in postpartum hemorrhage with disseminated intravascular coagulation: a case report and literature review. BMC Pregnancy Childbirth. 2021;21(1):710.
8. Taylor FB Jr, Toh CH, Hoots WK, Wada H, Levi M. Towards definition, clinical and laboratory criteria, and a scoring system for disseminated intravascular coagulation. Thromb Haemost. 2001;86(5):1327-30.
9. Erez O, Novack L, Beer-Weisel R, Dukler D, Press F, Zlotnik A, et al. DIC score in pregnant women-a population based modification of the International Society on Thrombosis and Hemostasis score. PLoS One. 2014;9(4):e93240.
10. Charbit B, Mandelbrot L, Samain E, Baron G, Haddaoui B, Keita H, et al. The decrease of fibrinogen is an early predictor of the severity of postpartum hemorrhage. J Thromb Haemost. 2007;5(2):266-73.
11. World Health Organization. Consolidated guidelines for the prevention, diagnosis and treatment of postpartum haemorrhage. Geneva: World Health Organization. 2025.
12. WOMAN Trial Collaborators. Effect of early tranexamic acid administration on mortality, hysterectomy, and other morbidities in women with post-partum haemorrhage (WOMAN): an international, randomised, double-blind, placebo-controlled trial. Lancet. 2017;389:2105-16.
13. Mavrides E, Allard S, Chandraharan E, Collins P, Green L, Hunt BJ, et al. Prevention and management of postpartum haemorrhage: Green-top Guideline No. 52. BJOG. 2017;124(5):e106-49.
14. Royal College of Obstetricians and Gynaecologists. Antepartum haemorrhage. Green-top Guideline No. 63. London: RCOG. 2011.
15. Fiori O, Deux JF, Kambale JC, Uzan S, Bougdhene F, Berkane N. Impact of pelvic arterial embolization for intractable postpartum hemorrhage on fertility. Am J Obstet Gynecol. 2009;200(4):384.e1-4.

Cite this article as: Aatif S, Sharif U, Mumtaz MA, Al Zabi S. Uterus preservation in severe obstetric disseminated intravascular coagulation secondary to placental abruption using bilateral uterine artery embolization: a case report. Int J Reprod Contracept Obstet Gynecol 2026;15:5037-40.