Research Square

Preprints are preliminary reports that have not undergone peer review.
They should not be considered conclusive, used to inform clinical practice,
or referenced by the media as validated information.

Spontaneous Rupture of Uterine Vessels in the Puerperium: Clinical Characteristics, Management, and Outcomes in a 10-Patient Retrospective Series

Qi Zhang[1]
Xijing Hospital
Xin Wang[2]
Xijing Hospital
Wei Zhang[1]
Xijing Hospital
Shu-juan Liu[1]
hanliu@fmmu.edu.cn

Xijing Hospital

Research Article

Keywords: spontaneous uterine vessel rupture, puerperium, postpartum hemorrhage, uterine artery rupture, uterine artery embolization, case series

Posted Date: November 5th, 2026

DOI: https://doi.org/10.21203/rs.3.rs-11166074/v1

License: This work is licensed under a Creative Commons Attribution 4.0 International License. Read Full License

Additional Declarations: No competing interests reported.

Abstract

Background

Spontaneous rupture of uterine vessels during the puerperium is a rare but life-threatening obstetric emergency characterized by acute intraperitoneal hemorrhage and hypovolemic shock. Because of its rarity and often nonspecific clinical presentation, preoperative diagnosis remains challenging and may result in delayed recognition and treatment. This study aimed to characterize the clinical features, diagnostic approaches, management strategies, and maternal and neonatal outcomes associated with spontaneous uterine vessel rupture during the puerperium.

Methods

We conducted a retrospective case series at a tertiary care center. Ten patients diagnosed with spontaneous uterine vessel rupture during the puerperium between 2010 and 2025 were included. Demographic characteristics, obstetric history, clinical presentation, diagnostic findings, surgical findings, treatment modalities, and maternal and neonatal outcomes were retrospectively collected and analyzed.

Results

The mean maternal age was 26.9 ± 3.6 years (range, 20–31 years). Seven patients were primiparous and three were multiparous. Six patients had undergone cesarean delivery, whereas four had delivered vaginally. The onset of bleeding ranged from the immediate postpartum period to 22 days after delivery. The most common presenting features were hypotension or hypovolemic shock and vaginal bleeding. The mean hemoglobin level at presentation was 66.5 ± 14.9 g/L (range, 41–93 g/L), and severe anemia (hemoglobin < 70 g/L) was present in seven patients. Ultrasonography was the primary diagnostic modality. The identified sources of bleeding included uterine artery branches (n = 8, including one pseudoaneurysm), the parametrail venous plexus (n = 1), and placenta accreta with associated vascular injury (n = 1). Management included exploratory laparotomy with vessel ligation or repair, uterine artery embolization (UAE), hysterectomy, and bilateral uterine artery ligation; some patients underwent more than one intervention. Estimated blood loss was documednt in six patients (range, 400–6,000mL,mean, 3,117 mL). All patients received blood product transfusions. The mean hospital stay was 10.0 ± 5.0 days (range, 3–19 days). No maternal or perinatal deaths occurred.

Conclusions

Spontaneous rupture of uterine vessels during the puerperium is a rare but potentially catastrophic condition with heterogeneous clinical and vascular etiologies. Early recognition requires a high index of suspicion. Prompt multidisciplinary management is essential to achieve favorable outcomes. UAE and exploratory laparotomy with direct vessel control constitute the mainstay of treatment. Hysterectomy may be required when bleeding cannot be controlled by conservative approaches.

Background

Spontaneous rupture of uterine vessels during the puerperium is a rare but potentially life-threatening obstetric emergency characterized by acute intraperitoneal hemorrhage and hypovolemic shock[1–2]. Although uterine rupture and vascular injuries have been reported during pregnancy, including rare cases presenting with retroperitoneal hematoma in the first trimester[3], spontaneous rupture of uterine vessels during the puerperium is an exceptionally uncommon condition. The available literature consists predominantly of isolated case reports[2, 4].

The pathogenesis of spontaneous uterine vessel rupture remains incompletely understood. Several factors have been proposed as potential contributors, including increased uterine blood flow during pregnancy, pregnancy-related hormonal changes that may affect vascular wall integrity, vascular abnormalities such as arteriovenous malformations and aneurysms, endometriosis, previous pelvic surgery, connective tissue disorders, and coagulation abnormalities[1, 5–6]. The uterine artery is the vascular site most frequently reported to rupture, followed by the utero-ovarian venous plexus[1].

The clinical presentation is often nonspecific and can resemble more common obstetric emergencies, including placental abruption, uterine rupture, and severe postpartum hemorrhage. Patients may present with features of hypovolemic shock, with or without vaginal bleeding[7–8]. However, these features are not always present, and the condition is infrequently diagnosed before surgery.

Available diagnostic modalities include ultrasonography, computed tomography (CT) angiography, and magnetic resonance imaging (MRI)[9]. Ultrasonography is generally the initial imaging modality and can rapidly identify findings suggestive of hemoperitoneum or a pelvic hematoma[10]. CT angiography can provide more detailed information about the pelvic vasculature and may localize the bleeding vessel or identify an associated pseudoaneurysm[11–12]. However, advanced imaging may not be feasible in hemodynamically unstable patients, in whom immediate exploratory laparotomy may be necessary to achieve hemorrhage control.

Management strategies have evolved over time. Historically, exploratory laparotomy with direct vessel ligation or hysterectomy was the principal approach to definitive hemorrhage control[13]. In recent years, uterine artery embolization (UAE) has emerged as a minimally invasive treatment option, particularly in hemodynamically stable patients and those with pseudoaneurysms[14]. The choice of treatment is influenced by several factors, including hemodynamic status, the suspected source and etiology of bleeding, the availability of interventional radiology services, and the patient's desire for future fertility.

Despite increasing recognition of this condition, the available evidence remains limited because most published reports describe individual cases or small case series. Large cohort studies are lacking due to the rarity of spontaneous uterine vessel rupture.

In this study, we present a retrospective case series of 10 patients with spontaneous uterine vessel rupture during the puerperium who were managed at our institution over a 15-year period. We describe their clinical characteristics, diagnostic evaluation, treatment approaches, and maternal and neonatal outcomes and place these findings in the context of the existing literature.

Materials and Methods

Study Design and Setting

This retrospective study was conducted in the Department of Obstetrics and Gynecology at Xijing Hospital, Fourth Military Medical University, a tertiary referral center in Xi'an, China. The study was approved by the Ethics Committee of Xijing Hospital, Air Force Medical University (approval number: KY20226208-C-1). Given the retrospective nature of the study, the requirement for informed consent was waived.

Study Population

We retrospectively identified all patients diagnosed with spontaneous uterine vessel rupture during the puerperium between May 2010 and August 2025. The puerperium was defined as the first 6 weeks after delivery. Potential cases were identified through a systematic review of the hospital's electronic medical records, operating room logs, and interventional radiology databases. The inclusion criteria were a clinical or surgical diagnosis of spontaneous uterine vessel rupture, occurrence of the event during the puerperium, and availability of complete medical records for review. Patients were excluded if they had traumatic uterine vessel injury, an iatrogenic vessel injury that was clearly recognized and repaired during the primary surgical procedure, or incomplete medical records.

Data Collection

Data were extracted from the hospital electronic medical record system. The collected variables included demographic characteristics, including maternal age; obstetric history, including gravidity, parity, previous cesarean delivery, and previous pelvic surgery; clinical presentation, including chief complaint, presenting symptoms, vital signs, and time of symptom onset relative to delivery; laboratory findings, including hemoglobin level, red blood cell count, and coagulation profile; diagnostic findings, including ultrasonographic findings and the initial clinical diagnosis; intraoperative findings, including the site of vascular rupture, the volume of hemoperitoneum, and uterine status; treatment details, including surgical and interventional radiology procedures and blood transfusion requirements; and maternal outcomes, including duration of hospitalization, complications, and survival. Neonatal survival was also recorded.

Statistical Analysis

Given the descriptive nature and small sample size of this study, clinical and genetic data were summarized individually, and no inferential statistical analyses were performed.

Results

Demographic and Obstetric Characteristics

A total of 10 patients with spontaneous uterine vessel rupture during the puerperium were identified during the study period. Their demographic and obstetric characteristics are summarized in Table 1.

Table 1
Clinical Summary of 10 Cases of Spontaneous Uterine Vessel Rupture in the Puerperium

Case	Age (year)	Gravidity/Parity	Delivery	Onset of Bleeding	Presenting Symptoms	Hemoglobin (g/L)	Site of Rupture	Treatment	Blood Loss (mL)	Hospita Stay (days)
1	30	G3P1	Vaginal delivery	Immediate postpartum (4 h)	Postpartum hemorrhage, shock	58	Uterine artery	Laparoscopy + bilateral uterine artery ligation	6,000	13
2	29	G2P1	Vaginal delivery	Immediate postpartum	Hypotension, dizziness	65	Left parametrial venous plexus	Laparoscopy + laparotomy + venous plexus suture	2,300	4
3	26	G1P1	Cesarean section	Postoperative day 5	Dizziness, lower abdominal discomfort, DIC	41	Uterine artery	Laparotomy + bilateral uterine artery ligation	3,000	19
4	23	G1P1	Cesarean section	Postoperative (16 h after second surgery)	Hypotension	82	Uterine artery	Laparotomy + hysterectomy	2,000	12
5	20	G1P1	Cesarean section	Postpartum day 22	Vaginal bleeding, shock	81	Right uterine artery pseudoaneurysm	Right internal iliac artery embolization	–	10
6	27	G2P1	Cesarean section	Postpartum day 13	Vaginal bleeding	64	Uterine artery	Bilateral UAE + factor VIII replacement	–	8
7	31	G3P2	Vaginal delivery	Postpartum day 11	Vaginal bleeding	58	Uterine artery	Bilateral UAE	–	12
8	27	G2P2	Cesarean section	Postpartum day 15	Vaginal bleeding	61	Uterine artery	UAE (Apr 2024); total hysterectomy (Jun 2025)	400	5
9	31	G3P2	Vaginal delivery	Postpartum day 5	Postpartum hemorrhage, fever	93	Placenta accreta with vessel injury	Laparotomy + subtotal hysterectomy + ureteral reimplantation	5,000	14
10	25	G1P1	Cesarean section	Postpartum day 9	Intermittent vaginal bleeding, shock	62	Uterine artery	Bilateral UAE	–	3

UAE, uterine artery embolization; DIC, disseminated intravascular coagulation; –, not recorded.

The mean maternal age was 26.9 ± 3.6 years (range, 20–31 years). Seven patients were primiparous, and three were multiparous. The mean gravidity was 1.9 ± 0.9 (range, 1–3). Six patients had undergone cesarean delivery, whereas four had delivered vaginally. One patient (case 8) had a history of previous cesarean delivery. One patient (case 9) had undergone previous pelvic surgery for an ovarian cyst. One patient (case 6) had a known coagulation disorder, specifically hemophilia A.

Clinical Presentation

The clinical presentations varied considerably among the 10 patients. The onset of symptoms ranged from the immediate postpartum period, defined as within hours after delivery, to 22 days postpartum. Two patients (cases 1 and 2) presented within the first 24 hours after delivery, seven patients (cases 3, 5, 6, 7, 8, 9, and 10) presented with delayed postpartum hemorrhage between 5 and 22 days after delivery, and one patient (case 4) presented with hypotension 16 hours after a second operation following cesarean delivery.

The most common presenting features were signs of hypovolemic shock, including hypotension, shock, and dizziness, which were observed in five patients (5/10, 50%), and vaginal bleeding, which was also reported in five patients (5/10, 50%). Notably, vaginal bleeding was absent in half of the patients, underscoring the potential for substantial intra-abdominal hemorrhage to occur without prominent external bleeding.

The mean hemoglobin level at presentation was 66.5 ± 14.9 g/L (range, 41–93 g/L). Severe anemia, defined as a hemoglobin level < 70 g/L, was present in seven patients (70%). Disseminated intravascular coagulation (DIC) was documented in one patient (10%).

Diagnostic Evaluation

Ultrasonography was the primary diagnostic imaging modality and was performed in nine patients (90%). Ultrasonographic findings included free intraperitoneal fluid or hemoperitoneum (n = 5), pelvic hematoma (n = 2), and abnormal uterine or cervical findings (n = 3). In one patient (10%), the diagnosis was established on the basis of clinical suspicion alone because severe hemodynamic instability necessitated immediate surgical intervention without further imaging.

A preoperative diagnosis of uterine vessel rupture was suspected in only a minority of patients. Most patients were initially considered to have postpartum hemorrhage of undetermined etiology, placental abruption, uterine rupture, or pelvic hematoma. In most cases, the definitive source of bleeding was established during surgical exploration or angiographic evaluation.

Sites of Vascular Rupture and Treatment Modalities

The sites and patterns of vascular injury are summarized in Table 1. The uterine artery was the most common site of bleeding (n = 8), including one right uterine artery pseudoaneurysm (case 5). Other sites included the left parametrial venous plexus (case 2) and uterine vascular injury associated with placenta accreta (case 9). Treatment was individualized according to the patient's hemodynamic status, suspected bleeding source and etiology, and availability of surgical and interventional radiology resources.

Five patients underwent surgical management. Two patients (cases 1 and 2) were initially managed by laparoscopy; in case 1, laparoscopic bilateral uterine artery ligation achieved hemostasis, whereas in case 2 conversion to laparotomy with suturing of the parametrial venous plexus was required. Three patients (cases 3, 4, and 9) underwent laparotomy: case 3 underwent bilateral uterine artery ligation, case 4 required hysterectomy for uterine artery bleeding presenting 16 hours after a second operation, and case 9 underwent subtotal hysterectomy for placenta accreta-associated vascular injury.

Five patients (cases 5, 6, 7, 8, and 10) underwent transcatheter arterial embolization. Case 5, who had a right uterine artery pseudoaneurysm, underwent right internal iliac artery embolization; case 6, who had hemophilia A, underwent bilateral uterine artery embolization (UAE) combined with factor VIII replacement; and cases 7, 8, and 10 underwent bilateral UAE for suspected uterine artery bleeding. Embolization successfully controlled the bleeding in all five patients. One patient (case 8), with a history of previous cesarean delivery and recurrent postpartum hemorrhage, underwent bilateral UAE in April 2024; because of recurrent bleeding, she subsequently underwent total hysterectomy in June 2025. In total, three patients required hysterectomy (cases 4, 8, and 9).

The estimated blood loss was documented in six patients and ranged from 400 to 6,000 mL (mean, 3,117 mL). Massive hemorrhage (≥ 2,000 mL) was documented in five of these six patients. All patients received blood product transfusions. The mean number of packed red blood cell units transfused was approximately 10.2 units (range, 1.5–29.5 units). Additional blood products, including fresh frozen plasma, platelets, cryoprecipitate, and fibrinogen concentrate, were administered as clinically indicated for coagulopathy.

Maternal and Neonatal Outcomes

All 10 patients survived and were discharged home in stable condition. The mean hospital stay was 10.0 ± 5.0 days (range, 3–19 days). Maternal complications included DIC (n = 1), acute respiratory distress syndrome (ARDS) (n = 1), puerperal infection (n = 1), and ureteral injury requiring reimplantation (n = 1). All neonates survived.

Discussion

Etiology and Pathogenesis

The etiology of spontaneous uterine vessel rupture during the puerperium remains incompletely understood, and the mechanisms are likely multifactorial.

The marked increase in uterine blood flow during pregnancy may play a central role in rendering the uterine vasculature susceptible to rupture. Uterine blood flow increases from approximately 50 mL/min in the nonpregnant state to 700–900 mL/min at term, representing a 10- to 20-fold increase[1]. In addition, the hypercoagulable state of pregnancy, together with sluggish venous flow, predisposes to venous engorgement and varicosity. Estrogen- and progesterone-mediated changes, including intimal hyperplasia, smooth muscle hypertrophy, and loss of mucopolysaccharides and reticulin fibers, may render the vessel wall more susceptible to injury and rupture[15]. Mechanical forces, including progressive uterine enlargement, uterine contractions, and expulsive efforts during labor, may further contribute to susceptibility[16–19]. Consistent with this, two patients in our series developed vascular bleeding after vaginal delivery.

Pre-existing vascular abnormalities, including aneurysms, pseudoaneurysms, arteriovenous malformations, and pelvic varicosities, may increase the risk[20–21]. In our series, one patient had a right uterine pseudoaneurysm, and one had rupture of the parametrial venous plexus. Pseudoaneurysms may develop following surgical trauma, such as cesarean section or curettage, and typically manifest as delayed postpartum hemorrhage. In addition, abnormal placentation (placenta accreta spectrum) and systemic disorders, such as coagulation disorders, may predispose to or exacerbate vascular bleeding[22–23], as illustrated by findings in one patient with hemophilia A in our series.

We acknowledge that the cases in this series represent a heterogeneous spectrum of puerperal uterine vascular bleeding rather than a uniform etiologic group. Several cases were associated with predisposing conditions, including placenta accreta, previous cesarean section, and coagulopathy. We retained these cases to provide a comprehensive clinical overview; strictly spontaneous rupture constitutes only a subset of the presentations included herein.

Clinical Presentation and Diagnostic Challenges

The clinical presentation of spontaneous uterine vessel rupture during the puerperium is variable and often nonspecific, which makes preoperative diagnosis particularly challenging. In our series, the most common presenting features were signs of hypovolemic shock and vaginal bleeding. Importantly, vaginal

bleeding was absent in three patients, highlighting the potential for intra-abdominal hemorrhage to remain clinically concealed despite substantial blood loss[2, 24]. The timing of presentation also varied: two patients presented immediately after delivery, seven patients presented between 5 and 22 days postpartum, and one patient presented after a second operation for postpartum hemorrhage. Ultrasonography is the initial imaging modality and can detect hemoperitoneum or pelvic hematoma, although its sensitivity for identifying the precise bleeding source is limited[20]. In hemodynamically stable patients, CT angiography may localize active extravasation or characterize vascular abnormalities[12]. In unstable patients, definitive intervention should not be delayed for imaging. The differential diagnosis includes placental abruption, uterine rupture, ruptured ovarian cyst, splenic artery aneurysm rupture, and hepatic rupture associated with HELLP syndrome. The diagnosis is rarely established preoperatively.

Management Strategies

Management should be individualized according to hemodynamic status, suspected etiology, availability of interventional radiology, and reproductive preferences. Exploratory laparotomy with direct vessel control remains essential for hemodynamically unstable patients or when the diagnosis is uncertain[1]. Techniques include bilateral uterine artery ligation, utero-ovarian artery ligation, and hypogastric artery ligation[25]. In our series, the uterus was preserved in three of six patients undergoing laparotomy. UAE is an important minimally invasive option, particularly in stable patients with pseudoaneurysms or arteriovenous malformations[14]. Hysterectomy remains the definitive treatment for refractory hemorrhage.

Limitations

This study has several limitations. First, the retrospective design may have introduced selection and information biases. Second, the sample size was small, reflecting the rarity of this condition. Third, the heterogeneity of the underlying etiologies and treatment approaches limits the ability to draw definitive conclusions regarding the optimal management strategy.

Conclusion

Spontaneous rupture of uterine vessels during the puerperium is a rare but life-threatening obstetric emergency with diverse etiologies and nonspecific presentations, often resulting in delayed diagnosis. A high index of suspicion, prompt recognition, and multidisciplinary management are essential to optimize maternal outcomes.

Abbreviations

ARDS Acute respiratory distress syndrome

CT Computed tomography;

DIC Disseminated intravascular coagulation

MRI Magnetic resonance imaging;

PPH Postpartum hemorrhage;

UAE Uterine artery embolization.

Declarations

Ethics approval and consent to participate

This study was conducted in accordance with the Declaration of Helsinki and was approved by the Ethics Committee of Xijing Hospital, Air Force Medical University (approval number: KY20226208-C-1). Given the retrospective nature of the study, the requirement for informed consent was waived.

Consent for publication

Not applicable.

Availability of data and materials

The datasets used and/or analysed during the current study are not publicly available due to patient privacy and institutional regulations but are available from the corresponding author on reasonable request.

Competing interests

The authors declare that they have no competing interests.

Funding

This research received no specific grant from any funding agency in the public, commercial.

Authors' contributions

QZ and XW contributed equally to this work. QZ and XW participated in data collection and analysis and drafted the manuscript. WZ contributed to data interpretation and manuscript revision. SJL conceived and designed the study, supervised the project, and critically revised the manuscript. All authors read and approved the final manuscript.

Acknowledgements

Not applicable.

References

1. Mercier AM, Caldwell MG, Rabie NZ, et al. Spontaneous Uterine Vessel Rupture During Pregnancy or in the Puerperium: A Review of the Literature[J]. Obstet Gynecol Surv. 2022;77(4):227–33. 10.1097/OGX.0000000000001012.
2. Wang HF, Rong SR, Zhang AH, et al. Shock caused by spontaneous rupture uterine vessels during puerperal period: a case report and literature review[J]. Eur Rev Med Pharmacol Sci. 2021;25(13):4451–5. 10.26355/eurrev_202107_26235.
3. Rodrigo V, Chuang TY. A Rare Presentation of Retroperitoneal Hemorrhage Following a Spontaneous Vaginal Delivery[J]. Cureus. 2025;17(6):e85442. 10.7759/cureus.85442.
4. Shibahara M, Kondo E, Shibata E, et al. Spontaneous rupture of an ovarian artery aneurysm complicated by postpartum hypertensive disorders of pregnancy after caesarean section: a case report and literature review[J]. J Med Case Rep. 2024;18(1):553. 10.1186/s13256-024-04871-y.
5. Lung N, McKeown G. Spontaneous rupture of uterine artery in a non-pregnant woman with adenomyosis: a case report and review of current literature[J]. Ulster Med J. 2025;94(1):13–5.
6. Echarfaoui O, Lazhar H, Slaoui A, et al. Uterine arteriovenous malformations as a rare differential diagnosis of abnormal uterine bleeding: A case report[J]. Int J Surg Case Rep. 2025;127:110900. 10.1016/j.ijscr.2025.110900.
7. Melamud K, Wahab SA, Smereka PN, et al. Imaging of Antepartum and Postpartum Hemorrhage[J]. Radiographics. 2024;44(4):e230164. 10.1148/rg.230164.
8. Biju N, Backer C. Hidden Preeclampsia Leading to Placental Abruption and Disseminated Intravascular Coagulation[J]. Cureus. 2025;17(5):e83469. 10.7759/cureus.83469.
9. Alonso-Burgos A, Díaz-Lorenzo I, Muñoz-Saá L, et al. Primary and secondary postpartum haemorrhage: a review for a rationale endovascular approach[J]. CVIR Endovasc. 2024;7(1):17. 10.1186/s42155-024-00429-7.
10. Benacerraf BR, Abuhamad AZ, Bromley B, et al. Consider ultrasound first for imaging the female pelvis[J]. Am J Obstet Gynecol. 2015;212(4):450–5. 10.1016/j.ajog.2015.02.015.
11. Takeda A, Koike W, Imoto S, et al. Three-dimensional computerized tomographic angiography for diagnosis and management of intractable postpartum hemorrhage[J]. Eur J Obstet Gynecol Reprod Biol. 2014;176:104–11. 10.1016/j.ejogrb.2014.02.026.
12. Damodharan K. CT diagnosis and endovascular management of spontaneous rupture of uterine artery in pregnancy[J]. BJR Case Rep. 2016;2(1):20150351. 10.1259/bjrcr.20150351.
13. Taye ST, Abebe TB, Borena AG, et al. Spontaneous uterine rupture in a non-gravid, unscarred uterus secondary to fulminant pelvic infection: a case report[J]. BMC Womens Health. 2025;25(1):630. 10.1186/s12905-025-04191-3.
14. Aryal S, Adhikari S, Sah KK, et al. Postcaesarean Pseudoaneurysm of Uterine Artery Causing Secondary Postpartum Hemorrhage Managed With Embolization: A Case Report[J]. Case Rep Obstet Gynecol. 2026;2026:4977602. 10.1155/crog/4977602.
15. Mandalà M. Influence of Estrogens on Uterine Vascular Adaptation in Normal and Preeclamptic Pregnancies[J]. Int J Mol Sci. 2020;21(7). 10.3390/ijms21072592.
16. Hesselman S, Lampa E, Wikman A, et al. Time matters-a Swedish cohort study of labor duration and risk of uterine rupture[J]. Acta Obstet Gynecol Scand. 2021;100(10):1902–9. 10.1111/aogs.14211.
17. Mengesha MB, Weldegeorges DA, Hailesilassie Y, et al. Determinants of Uterine Rupture and Its Management Outcomes among Mothers Who Gave Birth at Public Hospitals of Tigrai, North Ethiopia: An Unmatched Case Control Study[J]. J Pregnancy. 2020;2020:8878037. 10.1155/2020/8878037.
18. Vachon-Marceau C, Demers S, Goyet M, et al. Labor Dystocia and the Risk of Uterine Rupture in Women with Prior Cesarean[J]. Am J Perinatol. 2016;33(6):577–83. 10.1055/s-0035-1570382.
19. Myers KM, Elad D. Biomechanics of the human uterus[J]. Wiley Interdiscip Rev Syst Biol Med. 2017;9(5). 10.1002/wsbm.1388.
20. Youssef AT. Uterine vascular abnormalities linked to pregnancy complications: color and power Doppler-assisted transvaginal ultrasound evaluation[J]. J Ultrason. 2024;24(96):20240011. 10.15557/jou.2024.0011.
21. Wang Y, Qu S, Yang Y. Late postpartum hemorrhage caused by rupture of a uterine artery aneurysm in a patient with Takayasu arteritis: a case report[J]. J Med Case Rep. 2026;20(1). 10.1186/s13256-026-06226-1.
22. Shteynman L, Monanian G, Torres G, et al. Pathophysiology and Management of Placenta Accreta Spectrum[J]. J Dev Biol. 2025;13(4). 10.3390/jdb13040045.
23. Jha S, Jha S, Praveena P, et al. Recurrent Postpartum Hemorrhage: A Case of Uterine Artery Pseudoaneurysm Probably Induced by Anticoagulants[J]. Gynecol Minim Invasive Ther. 2024;13(1):53–5. 10.4103/gmit.gmit_78_23.
24. Hu L, Ning J, Li L, et al. Two cases of spontaneous rupture of the uterine artery in the perinatal period: A case report[J]. Med (Baltim). 2023;102(20):e33692. 10.1097/MD.0000000000033692.

25. Nabhan AE, AbdelQadir YH, Abdelghafar YA, et al. Therapeutic effect of Internal iliac artery ligation and uterine artery ligation techniques for bleeding control in placenta accreta spectrum patients: A meta-analysis of 795 patients[J]. Front Surg. 2022;9:983297. 10.3389/fsurg.2022.983297.