

Case Report

Right Homonymous Hemianopia and Inadvertent Subclavian Artery Cannulation During Emergency Peripartum Hysterectomy for Unanticipated Intraoperative Placenta Accreta Spectrum: A Case Report

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Abstract

Background: Unanticipated placenta accreta spectrum can precipitate catastrophic hemorrhage, requiring emergency peripartum hysterectomy and complex multidisciplinary resuscitation maneuvers. **Case:** This case report describes a complex obstetric emergency in which an unanticipated intraoperative diagnosis of placenta accreta spectrum (PAS) resulted in profound maternal hemorrhage and subsequent iatrogenic neurovascular complications. We report a clinical co-occurrence of severe hemorrhagic shock during an emergency peripartum hysterectomy, a resuscitation-related complication involving inadvertent cannulation of the right subclavian artery, and a subsequent left posterior cerebral artery (PCA) territory cortical infarction manifesting as dense right homonymous hemianopia. The patient's primary clinical findings included severe blood loss, requiring an emergency hysterectomy and massive transfusion. Following successful fluoroscopy-guided endovascular stent repair of the subclavian artery injury, the patient developed a persistent right visual field defect. Postoperative brain imaging confirmed an evolving left PCA territory infarction with cortical laminar necrosis, while the major intracranial vasculature remained anatomically patent. **Conclusions:** This obstetric crisis highlights the critical importance of meticulous surgical preparedness for unexpected presentations of PAS. Furthermore, it underscores the need for absolute vigilance during emergency central venous access to avoid inadvertent arterial injury, as well as the necessity of evaluating multi-factorial stroke etiologies, including hemodynamic hypoperfusion, embolic events, and peripartum hypercoagulability, following acute maternal collapse.

Keywords: placenta accreta spectrum; subclavian artery injury; posterior cerebral artery infarction; peripartum hysterectomy; case report

1. Introduction

Placenta accreta spectrum (PAS) remains a major cause of life-threatening obstetric hemorrhage and the need for peripartum hysterectomy [1]. Despite advances in antenatal ultrasound, screening is largely biased toward patients with placenta previa [2,3], potentially allowing cases with atypical fundal implantation to go unnoticed. When PAS is not recognized before delivery, unexpected severe intraoperative hemorrhage significantly increases maternal morbidity and requires urgent, intensive resuscitation by critical care teams [4,5].

This case describes an unexpected intraoperative diagnosis of PAS that significantly complicated clinical management. This crisis resulted in severe maternal hemorrhage, hemodynamic instability, and the need for urgent intervention. During rapid resuscitation to support systemic perfusion, an emergency central venous catheter was inadvertently placed into the right subclavian artery instead of the internal jugular vein. The combination of profound hemorrhagic shock and vascular injury led to acute neurovascular complications. This case emphasizes the clinical challenges of managing unanticipated, rapidly progressive pathology and the secondary risks associated with emergency invasive procedures during major obstetric crises.

A 39-year-old multigravida was admitted for elective cesarean delivery, with a notable surgical history of three previous cesarean sections and three dilatation and curettage (D&C) procedures. Aside from these prior surgeries, her medical, family, and psychosocial histories were unremarkable, with no known genetic predispositions (Table 1).

2. Case Presentation

The patient's antenatal period was uneventful, with no clinical signs of bleeding, pain, or preterm labor. Serial obstetric ultrasonography consistently demonstrated a single viable fetus in cephalic presentation, with a reassuring biophysical profile score of 8/8 and normal umbilical artery Doppler waveforms throughout gestation. The 25-week ultrasound scan (GA 25w1d) identified a high, anterior fundal placenta, confirming the absence of placenta previa or low-lying tissue (Fig. 1A,C). This scan documented a clear retroplacental hypoechoic zone between the placenta and myometrium, uniform uterine wall thickness, and regular retroplacental vascularity without abnormal lacunae. This stable position was confirmed on the 35-week scan (GA 35w1d), which demonstrated a normal amniotic fluid index (AFI = 8 cm) and an anterior-fundal location with no sonographic features of invasion; the lower uterine segment



Table 1. Timeline of clinical course, interventions, and outcomes.

Phase and date	Clinical presentation and diagnostic findings	Interventions and management	Clinical course and outcome
Antenatal phase (Weeks 25 and 35)	High-risk status due to 3 prior cesarean sections and 3 D&Cs. Routine transabdominal gray-scale scans at 25 and 35 weeks' gestation confirm a normal fundal placenta.	Serial obstetric ultrasounds monitoring placental location and fetal growth.	Normal placental implantation confirmed; no prenatal signs of placenta previa or tissue invasion.
Intraoperative phase (0–15 min)	Admitted for planned repeat elective cesarean section at 37 weeks gestation.	Laparotomy initiated under spinal anesthesia; converted to general anesthesia.	Delivery of a healthy infant girl (3.2 kg, Apgar scores 8 and 9). Upper-segment hysterotomy performed.
Intraoperative phase (15–60 min)	Placenta fails to separate; unexpected fundal PAS discovered. Lower uterine segment noted to be paper-thin.	Immediate activation of specialized PAS team; gynecologic oncologist joins surgical team. Attempted manual separation.	Catastrophic maternal hemorrhage occurs (4.5 L blood loss), inducing profound hypotension (lowest recordable NIBP 45/20 mmHg; unmeasurable for 20 mins).
Intraoperative phase (60–120 min)	Failure of primary uterine repair and bilateral vascular ligations. Accidental arterial puncture noted during central line attempt.	Total emergency peripartum hysterectomy; massive transfusion protocol. 8.5 Fr line inadvertently placed in subclavian artery and left <i>in situ</i> .	Definitive hemostasis achieved. Catheter secured to skin to act as mechanical tamponade per vascular surgery advice.
Postoperative day 0	Patient transferred intubated and sedated to the intensive care unit (ICU).	Urgent contrast-enhanced chest CT performed to map catheter trajectory.	Chest CT confirms catheter traverses right subclavian artery with tip at aortic arch; contained injury without active leakage.
Postoperative day 1	Patient remains hemodynamically stable but intubated in the ICU.	Fluoroscopy-guided endovascular repair of right subclavian artery via femoral access.	Successful deployment of covered self-expanding stent graft across injury site; complete vascular exclusion and stable distal perfusion achieved.
Postoperative day 2	Sedation weaned; patient successfully extubated. Conscious patient reports new-onset right visual field defect.	Urgent neurology consultation and non-contrast Brain CT/CTA. Secondary prevention initiated with Aspirin.	Physical examination reveals dense right homonymous hemianopia. Non-contrast brain CT confirms left PCA territory ischemic infarction; CTA confirms patent major intracranial and extracranial vessels without large vessel occlusion.
Postoperative days 5 and 7	Post-stroke medical stabilization and cardiac etiology workup.	Transthoracic echocardiogram and 24-hour Holter monitoring.	Echocardiogram shows borderline enlarged LV, mildly reduced EF. Holter confirms stable sinus rhythm without arrhythmias.
Postoperative day 14	Patient is clinically stable, walking, and displaying excellent wound healing.	Final multidisciplinary discharge assessment, medication reconciliation, and rehabilitation planning.	Discharged home in stable condition with persistent right homonymous hemianopia. Discharge regimen: Aspirin (81 mg daily), Enoxaparin for 10 days, calcium carbonate, ferrous sulfate with folic acid, and a 7-day course of oral antibiotics (cefuroxime and metronidazole).
9 Months postoperative	Long-term multi-specialty follow-up evaluation.	Neuro-ophthalmological workup (Humphrey Visual Field 30-2, macular GCC OCT) and follow-up brain MRI/MRA.	Perimetry confirms persistent right hemianopia with macular sparing; OCT shows left GCC thinning. MRI shows chronic left PCA cortical infarction.

D&Cs, dilatation and curettage; PAS, placenta accreta spectrum; ICU, intensive care unit; CT/CTA, computed tomography/computed tomography angiography; GCC, ganglion cell complex; OCT, optical coherence tomography; MRI, magnetic resonance imaging; MRA, magnetic resonance angiography; PCA, posterior cerebral artery; EF, ejection fraction; LV, left ventricle; MAP, mean arterial pressure.

was completely clear of placental tissue, with the lower placental margin well over 2 cm from the internal cervical os, satisfying criteria for a normally positioned placenta (Fig. 1B,D). The pregnancy was complicated by gestational diabetes mellitus, managed with 750 mg of metformin daily throughout gestation. The patient was admitted for an elective repeat cesarean delivery at 37 weeks of gestation due to her history of multiple prior cesarean deliveries.

2.1 Intraoperative Events and Surgical Management

The elective laparotomy was initially performed under spinal anesthesia but was converted to general anesthesia due to procedural complexity and adequate surgical exposure. Entry into the peritoneal cavity was smooth and without adhesions. Upon exposure of the gravid uterus, marked focal thinning and ballooning were observed over the upper uterine segment and fundus. To avoid tissue transecting the deeply adherent fundal placental mass during fetal extraction, an upper-segment hysterotomy was performed, and the infant was delivered successfully. A healthy female infant weighing 3.2 kg was delivered, with Apgar scores of 8 and 9 at 1 and 5 minutes, respectively.

Following delivery, the placenta failed to detach spontaneously, leading to an intraoperative diagnosis of PAS disorder. The specialized PAS team was immediately activated, and a core team member arrived promptly and participated in the procedure. Before the full extent of the pathologically adherent tissue was recognized, partial manual separation of the placenta was attempted. This disrupted the natural anatomical cleavage plane, resulting in placental fragmentation and precipitating immediate macrovascular hemorrhage from the fundal implantation bed.

In an effort to avoid hysterectomy and stabilize the patient, conservative devascularization maneuvers were immediately performed, including bilateral ligation of the uterine arteries and internal iliac arteries to control severe hemorrhage. At this point, a gynecologic oncologist joined the surgical team to assist with advanced pelvic dissection and hemorrhage control. During these procedures, inspection of the lower uterine segment revealed severe secondary focal thinning and structural weakness, representing an additional challenge during surgical manipulation. Attempts to repair and suture the thinned lower uterine tissue resulted in repeated myometrial tearing with each stitch. The primary source of hemorrhage was the disrupted fundal PAS site, compounded by diffuse pelvic bleeding from tearing of the lower uterine segments during repair attempts.

Attempted manual placental separation and conservative uterine preservation in the setting of undiagnosed PAS contributed directly to the escalation and severity of the hemorrhage. Due to total structural failure of the myometrium, uncontrollable diffuse pelvic hemorrhage, and failure of conservative vascular ligation, the surgical team abandoned uterine-preserving measures and rapidly transitioned to the definitive life-saving intervention. Conse-

quently, an emergency total peripartum hysterectomy was performed, with removal of the uterus and fallopian tubes.

During these procedures, the patient's blood pressure deteriorated rapidly from an operative baseline blood pressure of 115/70 mmHg down to a lowest of 45/20 mmHg (corresponding to a calculated mean arterial pressure [MAP] of 28 mmHg), following this nadir, NIBP readings became completely unmeasurable for an intermittent cumulative period of approximately 20 minutes. Marked sinus tachycardia peaked at 148 beats per minute. Due to structural failure of the uterine tissue and ongoing hemorrhage, the massive transfusion protocol was activated immediately. Resuscitation was aggressively maintained through large-bore peripheral intravenous access using an unheated rapid-infusion device to administer blood products in a 1:1:1 balanced ratio, alongside 2 g of intravenous tranexamic acid and continuous vasoactive support with norepinephrine infusion, titrated to a maximum of 0.3 µg/kg/min to maintain a target MAP above 55 mmHg. Importantly, no blood products, intravenous fluids, or vasoactive medications were administered through the misplaced arterial catheter at any point during resuscitation.

Throughout the surgery, the patient received blood transfusions to manage ongoing blood loss, totaling 6 units of fresh frozen plasma (FFP), 6 units of platelets, and 6 units of packed red blood cells (PRBCs). Extensive irrigation was performed, and definitive hemostasis was achieved. A negative-pressure abdominal drain was placed, an additional dose of cefazolin was administered. The total surgical blood loss was estimated at 4.5 liters. This massive hemorrhage was quantified using a combined approach: direct volumetric measurement of the surgical suction canisters (after explicitly subtracting the known volume of intraoperative irrigation fluids) alongside the gravimetric method, which involved weighing all blood-soaked surgical sponges and laparotomy pads. Urine output remained clear and adequate.

During intraoperative resuscitation, central venous access was attempted via the right internal jugular approach using a landmark technique without real-time ultrasound guidance due to the severity of the ongoing hemodynamic crisis. Upon vascular puncture, bright red, high-pressure pulsatile blood flow was observed. Due to the acute emergency setting, real-time pressure transduction, blood gas analysis, and guidewire position confirmation were not performed prior to catheter insertion, and an 8.5-Fr central venous catheter was accidentally introduced into the arterial circulation. Importantly, as soon as the inadvertent arterial placement was recognized, the catheter was immediately clamped and the lumen locked. No blood products, intravenous fluids, or vasoactive medications were administered through the misplaced catheter at any point during resuscitation.

The vascular surgery team was consulted intraoperatively and strongly advised against immediate catheter removal due to the critical risk of unmanageable, noncom-

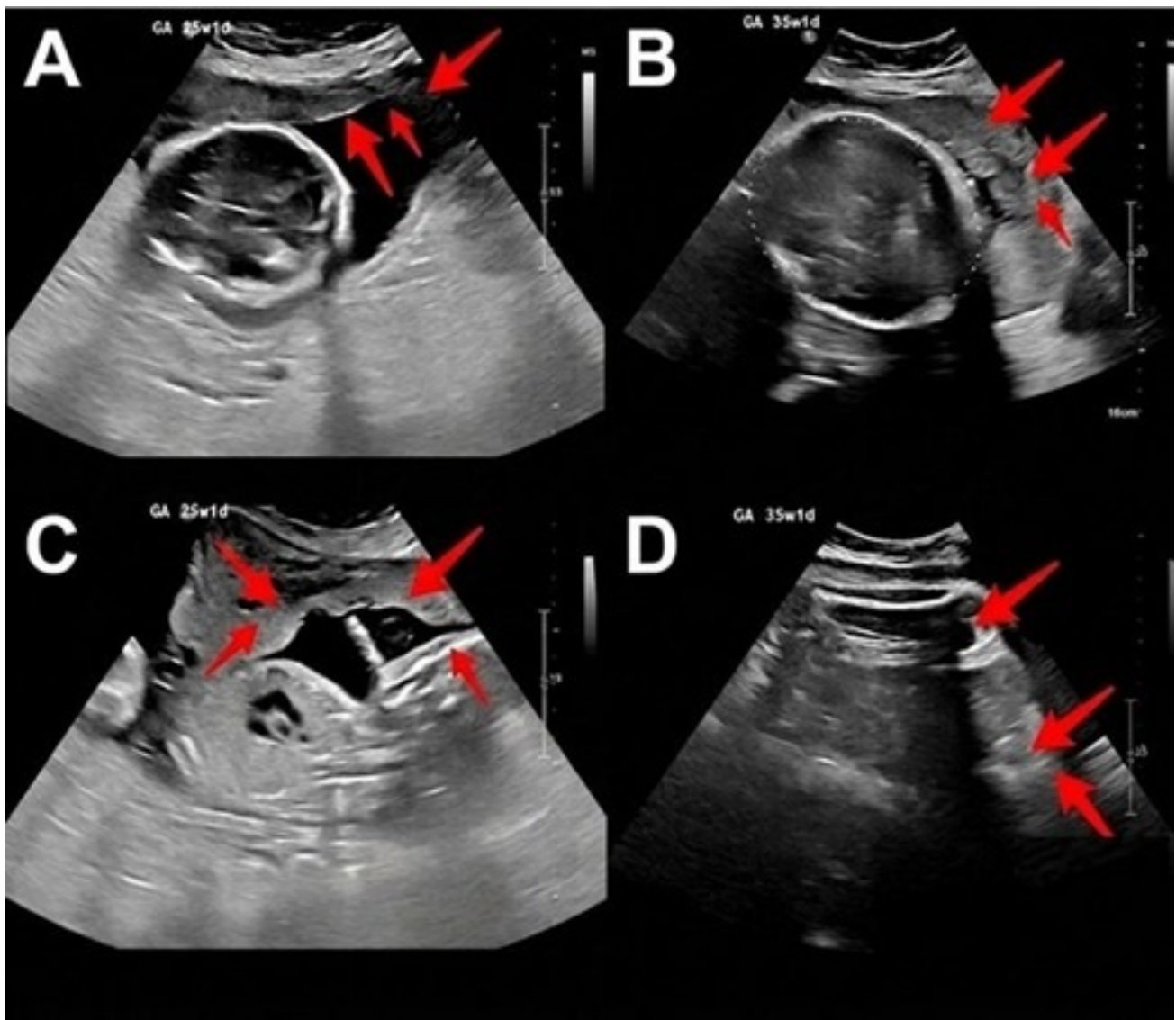


Fig. 1. Longitudinal ultrasound evaluation. Transabdominal gray-scale ultrasound panels tracking stable placental implantation. (A) Initial scan at 25 weeks 1 day (GA 25w1d) showing a high, normally positioned fundal placenta (red arrows). (B) Follow-up scan at 35 weeks 1 day (GA 35w1d) demonstrating the placenta (red arrows) maintaining its normal fundal position as the fetus grows. (C) Detailed view at 25 weeks 1 day confirming an intact retroplacental hypoechoic zone and clear upper-segment implantation (red arrows). (D) Detailed view of the lower uterine segment at 35 weeks 1 day showing the lower placental edge (red arrows) well clear of the internal cervical os, definitively ruling out placenta previa.

pressible thoracic hemorrhage. Instead, they recommended securing the catheter firmly to the skin to maintain mechanical tamponade at the arterial puncture site, followed by a formal computed tomography angiography (CTA) immediately after intensive care unit (ICU) transfer.

On postoperative day 0, diagnostic imaging was performed, including a contrast-enhanced chest computed tomography (CT) (Fig. 2). The imaging revealed malpositioning of the central venous catheter, with its distal tip traversing the right subclavian artery and ending at the aortic arch. Although imaging demonstrated signs of vascular injury, there was no evidence of active contrast extravasation.

On postoperative day 1, the patient underwent successful endovascular repair of the right subclavian artery injury. Under local anesthesia and fluoroscopic guidance, contralateral femoral arterial access was obtained for diagnostic angiography. The 8.5-Fr catheter was found to have entered the right subclavian artery just distal to the origin of the right vertebral artery. Using an over-the-wire exchange technique, the misplaced central venous catheter was carefully withdrawn over a guidewire while a covered self-expanding stent (e.g., 6 mm × 50 mm Viabahn) was deployed across the injury site. Complete vascular exclusion of the perforation was achieved while maintaining uncompromised blood flow into the right vertebral artery along-

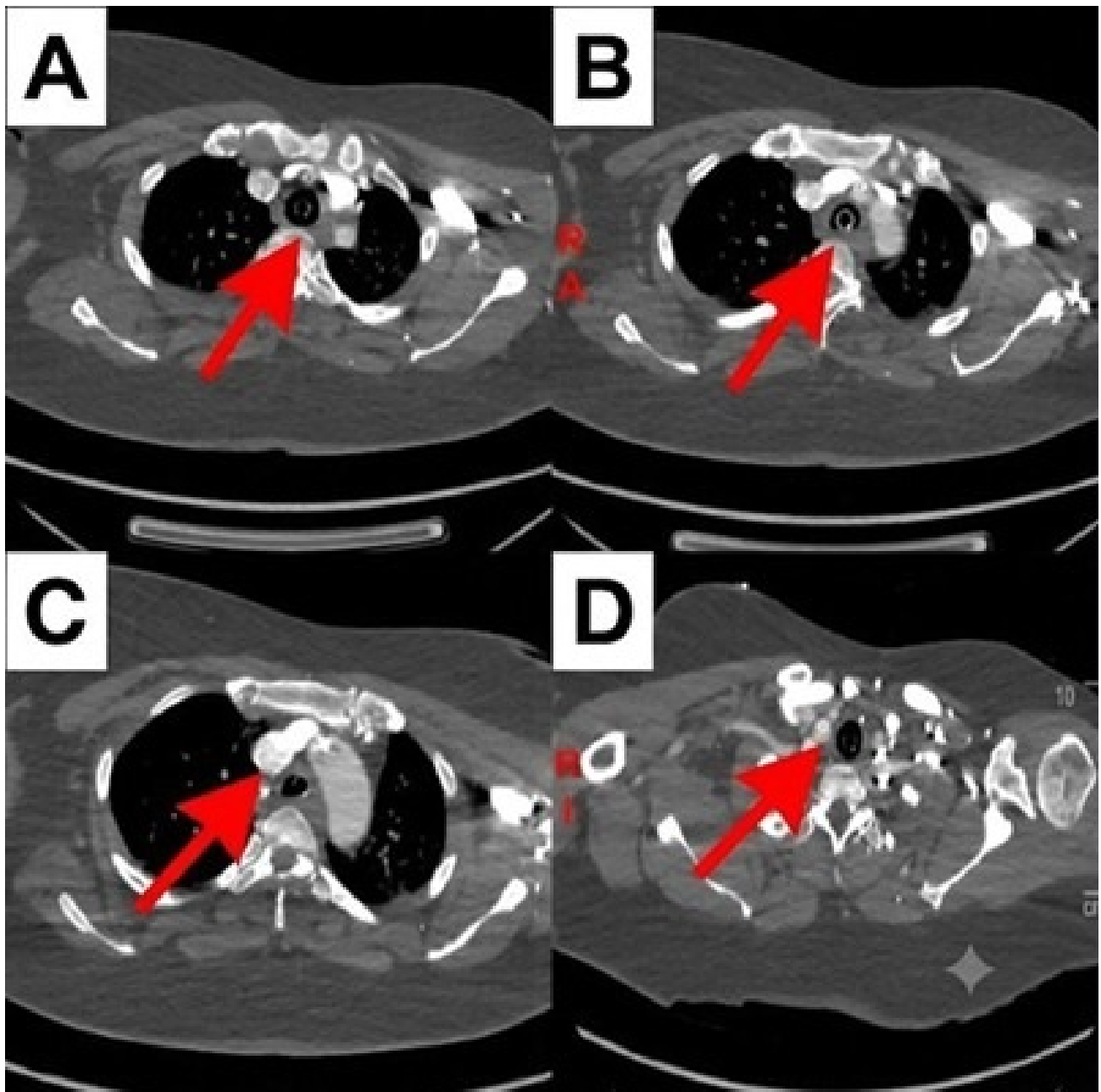


Fig. 2. Iatrogenic vascular injury. Serial axial contrast-enhanced chest computed tomography (CT) images mapping the trajectory of an inadvertently placed 8.5 Fr central venous catheter (red arrows). (A) Superior axial slice capturing the catheter's entry into the right subclavian artery. (B) Descending axial slice showing the catheter traversing the arterial lumen. (C) Lower axial slice tracking the catheter's continuous intravascular course. (D) Distal axial slice confirming the catheter tip terminates at the level of the aortic arch.

side normal distal upper-extremity perfusion on final angiography. Immediate hemostasis was achieved with minimal blood loss.

On postoperative day 2, after weaning from sedation and successful extubation, the patient was transferred to her room. Upon awakening, the patient suddenly reported a right visual field defect. Given that the patient had remained continuously sedated and mechanically ventilated in the ICU since leaving the operating theater, the exact time of stroke onset could not be determined and was masked by pharmacological sedation. The presenta-

tion represented an unwitnessed perioperative stroke, with a diagnostic window extending from the intraoperative period of profound hemorrhagic shock to the first conscious neurological examination post-extubation. An urgent neurology consultation was requested, and physical examination revealed a right homonymous hemianopia without accompanying limb weakness. An emergent non-contrast brain computed tomography (CT) and CT angiography (CTA) of the head and neck were immediately performed. The non-contrast CT confirmed a well-established acute-to-subacute ischemic infarction in the left posterior cerebral

artery (PCA) territory. The concurrent CTA demonstrated patent major intracranial and extracranial vessels, with no evidence of large vessel occlusion, active thrombosis, or high-grade structural stenosis.

Postoperative day 5 and 7 assessments showed a borderline enlarged left ventricle with normal filling pressures, mildly reduced ejection fraction, and mild global hypokinesia. Right Ventricular (RV) function was normal. Holter monitoring revealed no arrhythmias or atrial fibrillation, confirming a reassuring cardiac status. The patient was discharged on postoperative day 14 in stable condition. Her right homonymous hemianopia remained stable. Discharge medications included aspirin (81 mg daily) for secondary stroke prevention, enoxaparin prophylaxis for 10 days, calcium carbonate, ferrous sulfate with folic acid, and a 7-day course of oral antibiotics (cefuroxime and metronidazole).

2.2 Follow-up and Outcomes

During follow-up, neuro-ophthalmological assessments confirmed a stable, dense right homonymous hemianopia without significant improvement. On examination, best-corrected visual acuity was 20/20 in both eyes (OU), color vision was intact, and pupillary light reflexes were normal without a relative afferent pupillary defect (RAPD). Extraocular motility was full, with no nystagmus or strabismus. Automated perimetry (Humphrey Visual Field, 30-2 program) demonstrated a highly congruent, macular-sparing absolute right homonymous hemianopia, with a near-complete loss of sensitivity across the right temporal visual field of the right eye and the right nasal field of the left eye. Optical coherence tomography (OCT) mapping of the peripapillary retinal nerve fiber layer (pRNFL) demonstrated preserved, normal structural thickness in both eyes. However, segmented macular thickness analysis of the ganglion cell complex (GCC) revealed a distinct pattern of homonymous binocular thinning involving the left half of the macula in each eye, consistent with retrograde transsynaptic axonal degeneration secondary to the chronic left visual cortex infarction (Fig. 3). Central visual acuity was preserved, but the patient exhibited a persistent right visual field defect following perioperative resuscitation. The patient adhered well to neurorehabilitation.

Definitive pathologic examination of the total hysterectomy specimen confirmed a diagnosis of placenta accreta spectrum (PAS), classified as grade 1 according to the International Federation of Gynecology and Obstetrics/International Society for Placenta Accreta Spectrum (FIGO/IS-PAS) criteria [6]. The specimen demonstrated third-trimester chorionic villi directly attached to the superficial myometrium without intervening decidua basalis, accompanied by myometrial hypertrophy and focal decidualization, without deeper myometrial infiltration consistent with increta or percreta or serosal involvement. Anatomic tissue sampling confirmed that direct villous adherence was localized to the upper uterine segment and fundus.

Follow-up magnetic resonance imaging (MRI) and magnetic resonance angiography (MRA) 9 months post-surgery (Fig. 4) showed a chronic left medial occipitotemporal infarction with cortical necrosis, accounting for the patient's persistent hemianopia. The MRA demonstrated patent major intracranial arteries without flow-limiting structural stenosis. A focal signal attenuation was observed in the distal branches of the left posterior cerebral artery (PCA) on the 3D time-of-flight sequence. Although this signal attenuation likely represents a functional flow-velocity artifact caused by reduced downstream metabolic perfusion demand within chronic, infarcted occipital tissue, focal distal structural narrowing or vascular remodeling cannot be definitively excluded without invasive catheter-based digital subtraction angiography. Given the stable findings, routine follow-up MRI was scheduled for 6 months.

3. Discussion

PAS is frequently associated with placenta previa and a history of prior cesarean deliveries. However, atypical presentations have also been previously reported, including cases occurring in unscarred uteri [4,7,8] and in primigravidas [9,10,11], which may result in catastrophic postpartum hemorrhage. We describe a rare case of accidental PAS complicated by persistent homonymous hemianopia, and to our knowledge, no similar case has been reported in the literature.

A key limitation of this case is the uncertainty surrounding the precise timing of the stroke's onset, which is a common challenge in perioperative neurovascular medicine. Because the patient required emergent postoperative mechanical ventilation and continuous sedative infusions, focal neurologic assessments were impossible during the immediate postoperative period. Consequently, the ischemic event remained clinically unrecognized until sedation was cleared on postoperative day 2. However, the chronological association with a 4.5-L blood loss, unrecordable intraoperative blood pressure, and a secondary iatrogenic vascular injury strongly supports the intraoperative period of profound systemic hemodynamic collapse as the most plausible time of ischemic initiation.

Attempted manual placental separation before recognition of the underlying PAS, followed by conservative uterine-preservation attempts, directly contributed to escalation and severity of hemorrhage. Manual removal disrupted the underlying myometrial vascular bed and destroyed the anatomical cleavage plane, leaving placental fragments *in situ* and precipitating rapid, uncontrolled macrovascular bleeding.

The diagnostic challenges in our case closely resemble those described in Heim et al. [4] in a case series of four atypical, antenatally unsuspected PAS cases. These cases highlight the importance of maintaining a high index of suspicion during routine grayscale ultrasonographic screening of the lower uterine segment and myometrium in all women

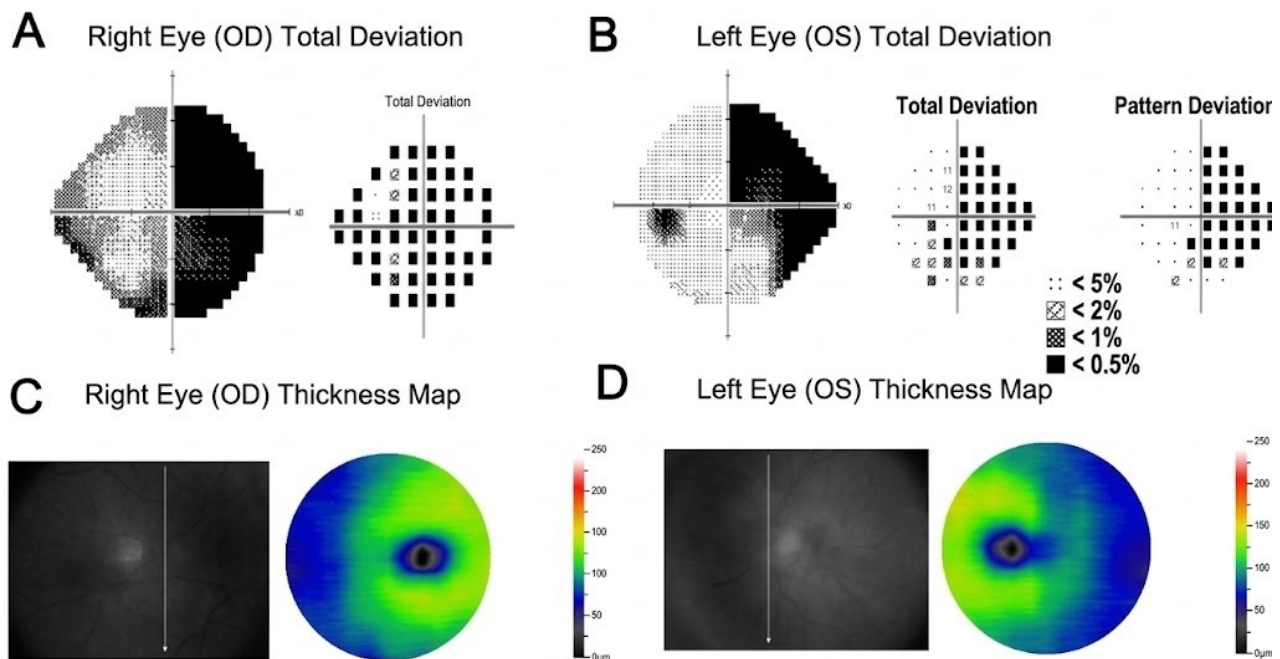


Fig. 3. Neuro-ophthalmological evaluation at 9 months. (A) Automated perimetry (Humphrey Visual Field 30-2) total deviation map for the right eye (OD) demonstrates a highly congruent, absolute right homonymous hemianopia with macular sparing. (B) Automated perimetry total deviation map for the left eye (OS) shows corresponding right-sided field defects. (C,D) Segmented macular Optical Coherence Tomography (OCT) thickness maps of the ganglion cell complex (GCC) for the right (C) and left (D) eyes reveal a homonymous, vertical thinning pattern isolated to the left hemiretina of both eyes, structurally localizing the lesion to the left retrochiasmal visual pathways and visual cortex. OCT, optical coherence tomography; GCC, ganglion cell complex.

with a history of multiple prior cesarean deliveries, even when the placenta appears to be normally implanted in the fundus. Rather than mandating universal Doppler screening, which may not be logistically or economically feasible, clinicians should maintain a low threshold for using color Doppler imaging when subtle grayscale irregularities, such as focal thinning or loss of the retroplacental hypoechoic zone, are identified during routine surveillance. Zhao et al. [12] conducted a multicenter retrospective study of 415,470 deliveries over 10 years to identify key anatomic and clinical risk factors for unsuspected PAS, comparing confirmed cases with antenatally suspected controls using multivariable regression analysis. Their analysis found that 35.4% of PAS cases remained undetected before delivery. Although traditional risk factors, such as prior cesarean delivery, increased the likelihood of prenatal diagnosis, our patient's case illustrates a diagnostic vulnerability. Despite having undergone 3 prior cesarean deliveries, her PAS was not identified antenatally, which substantially affected her intraoperative course. This finding is consistent with Zhao et al. [12], who reported that missed PAS cases were associated with worse outcomes, including higher blood loss and increased transfusion rates.

The diagnostic vulnerability associated with atypical placental implantation is further supported by the retrospec-

tive cohort data reported by Mulla et al. [13]. In their analysis of histologically confirmed PAS cases, prenatal detection rates differed significantly according to the presence of placenta previa, with detection in 72.3% of women with co-existing placenta previa compared with only 6.9% of those without previa ($p < 0.001$). This pattern mirrors our patient's case, in which a high fundal placental location bypassed standard imaging despite her risk profile. Although Mulla et al. [13] reported higher median blood loss and ICU admission rates among patients with placenta previa, they emphasized that missed antenatal diagnoses in the non-previa cohort frequently left surgical teams unprepared for the intraoperative findings. Our case exemplifies the type of intraoperative crisis described in their series, demonstrating that PAS without placenta previa, although statistically less likely to be recognized antenatally, can still lead to catastrophic hemorrhage and systemic hemodynamic collapse. This highlights the importance of immediate multidisciplinary mobilization when unsuspected PAS is discovered at delivery.

A critical lesson from this case concerns the sequence of operative decision-making when unexpected PAS is encountered. Initial manual placental separation disrupted the natural cleavage plane, while conservative maneuvers, including uterine artery ligation and myometrial defect su-

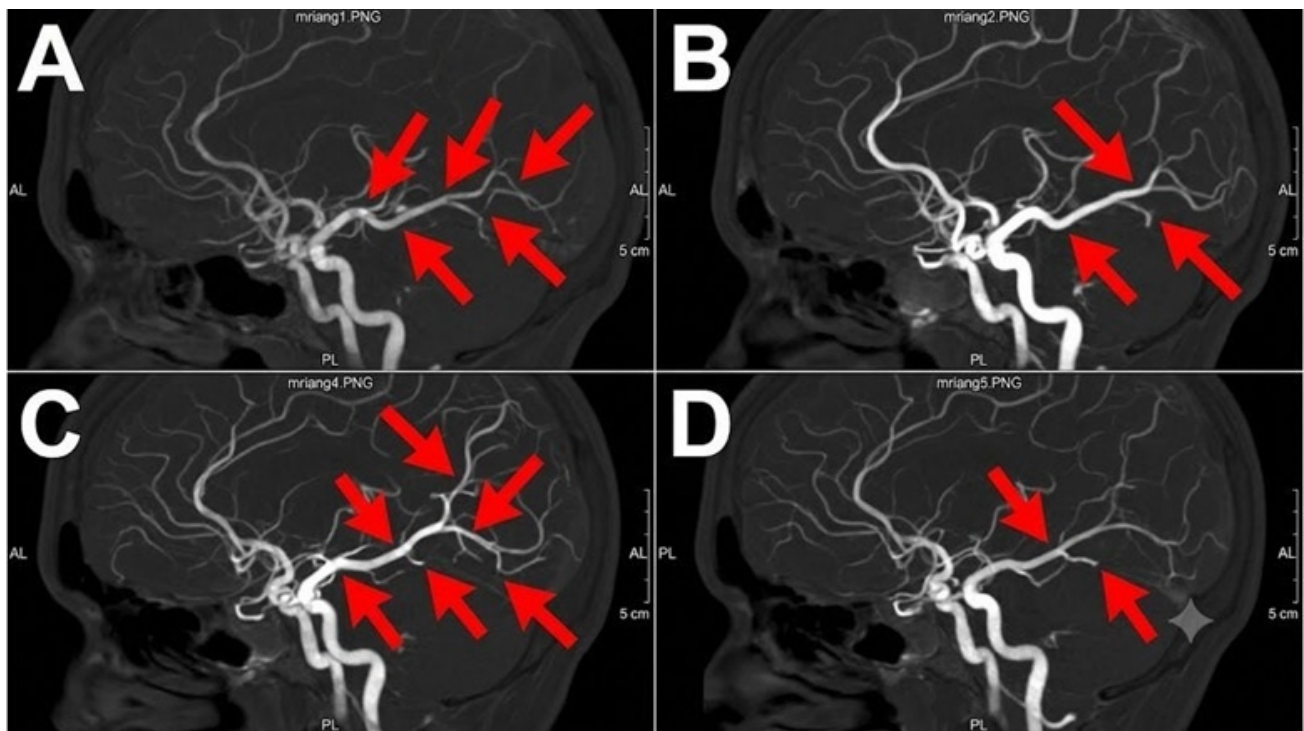


Fig. 4. MRA outcomes. Serial sagittal 3D time-of-flight MRA maximum intensity projection (MIP) views: (A) initial lateral projection demonstrating focal signal attenuation and caliber reduction along the proximal-to-distal left PCA and its distal branching vessels (red arrows). (B) medial adjacent slice tracking the distal cortical PCA segment showing localized signal loss and terminal branch drop-out (red arrows). (C) deeper parasagittal projection delineating the course of the main left PCA trunk and its cortical ramifications with persistent subtle distal signal voids (red arrows). (D) far medial projection revealing distal branch attenuation and localized signal loss in the distal occipitotemporal vasculature (red arrows) against an otherwise patent major intracranial arterial tree. This localized signal loss likely represents a potential flow-velocity artifact secondary to sluggish downstream perfusion in the chronically infarcted occipital territory; however, distal structural narrowing cannot be fully excluded on noncontrast sequences alone. MRA, magnetic resonance angiography; PCA, posterior cerebral artery.

turing, delayed definitive surgical control despite ongoing diffuse hemorrhage. In agreement with international consensus guidelines [14], attempted manual removal of pathologically adherent placental tissue significantly exacerbates hemorrhagic morbidity and blood loss compared with leaving the placenta intact and proceeding directly to primary peripartum hysterectomy. Recognition of these operative pitfalls is essential, as conservative uterine preservation attempts in unsuspected PAS frequently exacerbate intraoperative hemodynamic shock before definitive hysterectomy is undertaken.

To contextualize the unique triad of complications encountered in this case, the clinical findings were compared with established medical literature. The challenges associated with an unexpected intraoperative PAS diagnosis are well documented. Mouna et al. [15] described a similar obstetric emergency in which a 32-year-old woman with 2 prior cesarean deliveries presented with unsuspected PAS identified only after neonatal delivery. Consistent with our case, the absence of a natural cleavage plane and the onset of immediate catastrophic hemorrhage necessitated a rapid intraoperative decision to perform a life-saving hys-

terectomy. This observation reinforces our finding that, despite advances in antenatal surveillance, surgical teams must maintain a high index of suspicion and be prepared for immediate, definitive surgical intervention in any patient with a history of uterine surgery.

Regarding resuscitation-related complications, inadvertent arterial cannulation during emergency central venous access is a recognized high-risk event in critically ill patients. Ananthakrishnan et al. [16] reported 2 cases of iatrogenic subclavian artery puncture, one in a patient with severe hematemesis and the other in a patient with peritonitis secondary to upper gastrointestinal perforation, in which large-bore 7-Fr catheters were inadvertently introduced into the subclavian artery and terminated in the aortic arch, closely matching the anatomical trajectory observed on our patient's postoperative chest CT. Their literature review validates our management strategy: rather than performing an immediate, blind catheter removal, which carries a high risk of uncontrollable mediastinal or thoracic hemorrhage, the catheter should be secured in place to maintain mechanical hemostasis. The literature further demonstrates that subsequent transfer for minimally inva-

sive endovascular repair, such as the use of collagen closure devices or covered stents via axillary access, can provide effective, definitive hemostasis with minimal blood loss [17,18].

A focal unilateral PCA territorial infarction is not, in itself, sufficient evidence of a purely hemodynamic mechanism. Because the patient remained continuously sedated and mechanically ventilated until postoperative day 2, the exact clinical onset of stroke remains unknown. While severe hemorrhagic shock (4.5 L blood loss) and sustained hypotension (with a lowest recordable MAP of 28 mmHg and 20 minutes of unmeasurable non-invasive blood pressures) induce critical systemic hypoperfusion to which the visual cortex is exquisitely vulnerable, multiple alternative and competing mechanisms cannot be excluded. These potential mechanisms include thromboembolic events arising from subclavian artery catheter manipulation or endovascular stent deployment near the vertebral artery origin, *in situ* arterial thrombosis or vasospasm from high-dose vasopressor therapy, and the physiological hypercoagulable state of the peripartum period compounded by massive transfusion. Therefore, this perioperative stroke should be interpreted as a multifactorial event rather than a confirmed single-cause ischemic insult.

4. Conclusions and Take-Home Message

This case highlights three primary educational lessons for multidisciplinary obstetric critical care. First, PAS may occur in the absence of placenta previa, and attempted manual placental separation can disrupt the natural cleavage plane, exacerbate hemorrhage, and require prompt transition to primary hysterectomy. Second, inadvertent subclavian artery cannulation during emergency resuscitation should be managed by securing the catheter locked *in situ* to maintain mechanical tamponade and by avoiding infusion until definitive endovascular repair is performed. Third, visual loss after obstetric hemorrhage warrants a comprehensive neurovascular evaluation incorporating systemic hypoperfusion alongside embolic, catheter-related, and hypercoagulable stroke mechanisms.

Availability of Data and Materials

All data in this manuscript are available from the corresponding author upon reasonable request, subject to patient privacy considerations.

Author Contributions

NA designed the research study, conducted the data collection, analyzed the clinical data, provided expert clinical consultation, and performed the literature review. As the sole author, NA drafted and critically revised the manuscript for important intellectual content, read and approved the final version, and took full public responsibility and accountability for all aspects of the work.

Ethics Approval and Consent to Participate

This case report was conducted in accordance with the ethical principles set forth in the Declaration of Helsinki. Consistent with our institution's policy, formal Institutional Review Board (IRB) approval is not required for a single case report. Informed consent was obtained from the patient for the publication of this case report and any accompanying clinical images.

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Conflicts of Interest

The author declares no conflicts of interest.

Declaration of AI and AI-Assisted Technologies in the Writing Process

During the preparation of this work, the author used Gemini to check spelling and grammar and to combine images into composite figures. The author explicitly confirms that Gemini was used solely for formatting and layout purposes; no generative modification, alteration, enhancement, addition, or removal of any underlying medical image content was performed. After using this tool, the author reviewed and edited the content as needed and takes full responsibility for the content of the publication.

Supplementary Material

Supplementary material associated with this article can be found, in the online version, at <https://doi.org/10.31083/CEOG55251>.

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