

CASE REPORT

Intraoperative Cell Salvage and Hemodynamic Trajectory During Cesarean Hysterectomy for Placenta Accreta Spectrum: A Case Report

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ABSTRACT

Background. Placenta accreta spectrum (PAS) can cause massive obstetric hemorrhage. Intraoperative cell salvage (ICS) may support autologous blood conservation.**Objective.** To describe ICS mass-balance recovery and the hemodynamic trajectory during cesarean hysterectomy in one high-risk patient.**Methods.** A 37-year-old woman (gravida 4, para 1) at 37–38 weeks with total placenta previa and a Placenta Accreta Index of 6 underwent cesarean hysterectomy under combined spinal-epidural anesthesia. A dual-suction protocol separated amniotic fluid from shed blood. Cell salvage recovery and laboratory and hemodynamic trajectories were assessed at five perioperative time points.**Results.** Reported blood loss was 3,500 mL. Mean arterial pressure reached 55 mmHg and recovered during norepinephrine infusion at 0.03–0.08 µg/kg/min. ICS processed 2,438 mL of fluid, including 1,000 mL of irrigation saline, and returned 451 mL of washed red cells at an approximate hematocrit of 60%. The reported mass-balance recovery was 24.78% (270.6/1,092 mL), compared with an 18.50% volumetric yield. Tranexamic acid and calcium gluconate were administered. Fibrinogen reached a nadir of 265 mg/dL. Hemoglobin was 10.1 g/dL preoperatively, 8.4 g/dL at nadir, and 9.2 g/dL at discharge. No allogeneic transfusion was reported.**Conclusion.** This case describes autologous blood conservation with ICS and vasoactive support during major obstetric hemorrhage. A single case cannot establish efficacy or causal effects.**Access licence:** Open access. This article is distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 4.0 International Licence (CC BY-NC-SA 4.0), which permits non-commercial use, sharing, adaptation and reproduction in any medium, provided the original author and source are credited and derivative works are licensed under identical terms.**Copyright:** © 2026 The Author(s). Published by HM Publisher in collaboration with CMHC Research Center. e-ISSN 2745-9497.

1. Introduction

Placenta accreta spectrum disorders represent one of the most perilous obstetric pathologies encountered in contemporary maternal-fetal and perioperative medicine, characterized by abnormal trophoblastic adherence to and invasion through the myometrium in the complete or partial absence of the intervening decidua basalis.¹ Under normal physiological gestations, the decidua basalis and its basal Nitabuch layer serve as an essential biochemical and structural barrier, strictly regulating extravillous trophoblastic invasion and creating an anatomical cleavage plane that facilitates spontaneous, clean placental detachment and expulsion following fetal delivery.² However, focal myometrial damage—most frequently resulting from prior lower-segment cesarean deliveries, vigorous curettage, myomectomy, or operative hysteroscopic procedures—destroys this regulatory decidual matrix.³ In the presence of a scarred lower uterine segment, extravillous trophoblasts encounter bare myometrial muscle fibers, penetrating unchecked into the deep muscular architecture (placenta increta) or extending through the uterine serosa into adjacent pelvic viscera such as the urinary bladder and parametrial vasculature (placenta percreta).⁴

The global epidemiological trajectory of placenta accreta spectrum has risen precipitously over the past three decades, closely paralleling the worldwide surge in cesarean delivery rates and the demographic trend toward advanced maternal age.⁵ Historical cohort analyses from the mid-twentieth century documented an incidence of placenta accreta of approximately 1 in 30,000 deliveries; contemporary multicenter registry data indicate that the current prevalence exceeds 1 in 500 to 1 in 1,000 births in tertiary obstetric centers, representing an extraordinary fifty-fold escalation in absolute risk.⁶ In women presenting with total placenta previa and a history of one prior cesarean hysterotomy, the cumulative risk of invasive placentation reaches approximately 10% to 25%, while two or more prior surgical deliveries elevate this risk beyond 40% to 60%.⁷ When combined with the progressive loss of uterine vascular compliance and higher parity associated with delayed childbearing, the structural vulnerability of the lower uterine segment is magnified, transforming what was once an uncommon obstetric anomaly into a major driver of preventable severe maternal morbidity and peripartum mortality worldwide.⁸

Among the clinical challenges of placenta accreta spectrum, acute intraoperative hemorrhage represents the most catastrophic hazard, often necessitating massive allogeneic

blood transfusion to forestall irreversible hemorrhagic shock, dilutional coagulopathy, and end-organ hypoperfusion.⁹ The biological origin of this rapid exsanguination stems from the anatomical remodeling of the maternal uteroplacental vasculature. During deep trophoblastic invasion, maternal spiral arteries undergo extensive physiological transformation, shedding their muscular tunica media and autonomic innervation to become widely dilated, low-resistance, high-capacitance vascular conduits carrying maternal arterial blood under high flow rates directly into the placental bed.¹⁰ Because the myometrium in invasive placentation is severely thinned, scarred, and anatomically disorganized, it completely lacks the coordinated contractile power required to exert physiological mechanical tamponade over these severed high-flow vessels upon placental avulsion.¹¹ Consequently, any surgical attempt to separate the morbidly adherent placenta produces torrential, uninhibited arterial hemorrhage, during which patients can rapidly exsanguinate up to 50% to 70% of their total circulating blood volume within several minutes.¹²

Although conventional massive transfusion protocols relying on uncrossmatched or crossmatched allogeneic donor blood products remain the historical standard for managing severe obstetric hemorrhage, empirical high-volume allogeneic transfusion carries substantial clinical, immunological, and logistical hazards. Exposure to banked donor blood exposes the vulnerable maternal system to life-threatening immunological reactions, including transfusion-related acute lung injury, transfusion-associated circulatory overload, hemolytic transfusion reactions, severe alloimmunization, and postoperative systemic immunosuppression. Transfusion-related acute lung injury is a devastating non-cardiogenic pulmonary edema triggered by donor anti-human leukocyte antigen antibodies reacting against recipient pulmonary capillary endothelium, carrying high case-fatality rates in parturients. Simultaneously, transfusion-associated circulatory overload poses a major threat in obstetric patients who already possess expanded plasma volumes and cannot tolerate rapid, high-volume fluid resuscitation without developing acute hydrostatic pulmonary edema and right ventricular strain. Furthermore, donor blood represents a severely constrained, finite resource, particularly in developing nations and regional tertiary referral centers where blood bank inventories are plagued by supply chain disruptions, limited voluntary donor pools, cold chain vulnerabilities, and prolonged crossmatch turnaround intervals.

To overcome the risks and shortages of allogeneic transfusion, intraoperative cell salvage has emerged as an indispensable pillar of modern patient blood management frameworks in high-risk surgery. Patient blood management represents an evidence-based, patient-centered paradigm that seeks to optimize native red blood cell mass, minimize iatrogenic surgical blood loss, and harness physiological tolerance to anemia rather than defaulting reflexively to donor blood. Intraoperative cell salvage operationalizes these principles by continuously aspirating shed surgical blood, mixing it with heparinized anticoagulant, centrifuging the mixture to isolate dense erythrocytes, and washing the packed red cells with isotonic saline to produce a high-hematocrit autologous suspension ready for immediate reinfusion. Unlike stored allogeneic blood, which suffers from progressive biochemical degradation known as the storage lesion—characterized by membrane lipid peroxidation, depleted 2,3-diphosphoglycerate, and reduced adenosine triphosphate—salvaged autologous erythrocytes maintain physiological deformability and

normal oxygen-hemoglobin dissociation kinetics, delivering optimal microvascular tissue oxygenation without exposing the maternal immune system to foreign antigens or infectious agents.

Despite these compelling physiological advantages, the perioperative integration of intraoperative cell salvage during cesarean hysterectomy in patients presenting with high placenta accreta risk remains complex, particularly concerning anesthetic decision-making, hemodynamic stability, and mathematical yield quantification. Historically, international clinical consensus guidelines have strongly advocated for general endotracheal anesthesia in anticipated placenta accreta spectrum deliveries, emphasizing immediate airway security and preservation of intrinsic sympathetic vasomotor reflexes during massive fluid shifts. When neuraxial blockade, particularly combined spinal-epidural anesthesia, is utilized due to maternal preference or favorable baseline airway parameters, the resulting dense sympathetic efferent denervation across cardioaccelerator (T1–T4) and splanchnic (T5–L2) segments obliterates the body's compensatory vasoconstrictive and tachycardic defenses, predisposing the parturient to catastrophic uncompensated circulatory collapse upon acute exsanguination. The clinical management of such crises requires immediate, targeted pharmacological vasopressor support, such as continuous norepinephrine infusion, to restore mean systemic filling pressure and arterial vascular tone while cell salvage restores red cell mass.

Moreover, existing literature has frequently mischaracterized the true efficiency of intraoperative cell salvage by reporting superficial volumetric fluid yields (dividing recovered packed red blood cell volume by total aspirated surgical fluid volume), an uncorrected metric that severely penalizes the device for operating in a surgical field requiring copious saline irrigation. There is a critical need for rigorous mathematical mass-balance stoichiometry that isolates actual red cell mass lost versus actual red cell mass recovered, establishing a transparent benchmark for autologous blood conservation. Therefore, the primary objective of this observational analytic investigation is to rigorously evaluate the clinical efficacy, mathematical mass-balance stoichiometry, and real-time hemodynamic trajectory of intraoperative cell salvage paired with targeted vasopressor titration during cesarean hysterectomy in a parturient with a 69% probability of placenta accreta spectrum managed without allogeneic blood products in a resource-constrained tertiary setting.

2. Methods

Study Design and Epidemiological Framework

This investigation was conducted as an observational analytic case study designed to quantitatively evaluate intraoperative autologous blood conservation, mathematical erythrocyte mass-balance dynamics, and longitudinal hemodynamic trajectory in a patient undergoing planned cesarean hysterectomy for high-risk placenta accreta spectrum.¹³ The investigation was conducted at the Department of Anesthesiology and Intensive Therapy, Faculty of Medicine, Universitas Brawijaya / Dr. Saiful Anwar General Hospital, Malang, East Java, Indonesia, serving as the primary tertiary referral academic medical center for maternal-fetal critical care in the region.¹⁴ The epidemiological framework addressed the clinical reality of advanced obstetric surgical pathology within a developing healthcare infra-

structure characterized by the absence of point-of-care viscoelastic coagulation testing and constrained allogeneic blood bank reserves.¹⁵ To guarantee complete analytical transparency and methodological rigor, the study adhered strictly to the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) reporting guidelines and the consensus recommendations of the International Society for Cell Salvage in Obstetrics.¹⁶

Ethical Governance and Participant Consent

The study protocol, clinical observation procedures, and anonymized data extraction mechanisms were subjected to formal independent review and granted official ethical approval by the CMHC Research Center Ethics Committee, Indonesia (Protocol Approval Number: 2026/7821). All clinical procedures, anesthetic interventions, and surgical maneuvers were performed in full accordance with the ethical tenets articulated in the Declaration of Helsinki (2013 Fortaleza revision) and prevailing Indonesian national regulations governing clinical research on human subjects.^{17,18} Prior to surgical intervention, the patient and her legal surrogate were provided with extensive, multidisciplinary counseling detailing the anticipated operative risks, surgical hysterectomy necessity, massive hemorrhage probability, autotransfusion mechanics, and academic utilization of longitudinal physiological parameters.¹⁹ The patient provided written informed consent for both the high-risk surgical procedure and the academic dissemination of de-identified clinical, laboratory, hemodynamic, and mass-balance data. Patient privacy and data confidentiality were strictly safeguarded through comprehensive de-identification, and all facial photographic documentation was subjected to solid black anonymization bars across the ocular region in compliance with international publication ethics guidelines.²⁰

Case Eligibility and Diagnostic Ascertainment

Eligibility for this observational analytic investigation was determined through strict antenatal diagnostic criteria and objective risk stratification.²¹ A 37-year-old woman (Gravida 4, Para 1, with two prior early first-trimester miscarriages and one viable lower-segment cesarean delivery performed in 2013) presented at 37–38 weeks of gestation

for scheduled multidisciplinary surgical delivery, as detailed in Table 2. Diagnostic ascertainment was established through high-resolution transabdominal and transvaginal obstetric ultrasonography combined with color Doppler mapping performed by senior maternal-fetal medicine specialists. Ultrasonographic examination confirmed complete placenta previa entirely covering the internal cervical os. Pathognomonic markers of severe invasive placenta were identified, including extensive grade III placental lacunae exhibiting turbulent, high-velocity internal blood flow, anomalous bridging vessels traversing the uterine serosa, and marked thinning of the retroplacental myometrium to less than 1 mm. Quantitative risk scoring using the validated Placenta Accreta Index (PAI) yielded a cumulative score of 6, establishing an objective 69% statistical probability of invasive morbid adherence (placenta accreta spectrum) requiring definitive hysterectomy.²²

Longitudinal Observation Protocol and Analytical Measurements

Longitudinal clinical observation was conducted across five predetermined, sequential perioperative epochs: baseline preoperative admission (T0), acute intraoperative hemorrhagic nadir (T1), postoperative day 1 (T2), postoperative day 3 (T3), and hospital discharge on postoperative day 5 (T4).²³ Pre-induction preparation included the placement of bilateral large-bore peripheral venous access lines (16-gauge and 18-gauge) and continuous invasive arterial blood pressure monitoring via a 20-gauge left radial arterial catheter. Arterial pressure waveforms, beat-to-beat mean arterial pressure (MAP), continuous heart rate, and peripheral pulse oximetry were monitored continuously on a multiparameter clinical monitor. Anesthetic induction used a combined spinal-epidural technique with intrathecal administration of 0.25% isobaric ropivacaine combined with 1 mg of preservative-free morphine, justified by the patient's strong preference to remain conscious during neonate delivery and favorable airway metrics (Mallampati class II, thyromental distance 7 cm, full cervical range of motion). Immediate airway rescue equipment, including a video laryngoscope, supraglottic airway conduits, and rapid-sequence induction agents (propofol and rocuronium), was primed at the bedside.²⁴ The reported perioperative sequence is summarized in Table 1.

Table 1. Clinical timeline of the reported case

Time point	Event and observations	Intervention or outcome
T0 Preoperative	37–38 weeks; PAS risk; Hb 10.1 g/dL; MAP 88 mmHg	Preparation for cesarean hysterectomy
T1 Intraoperative nadir	MAP 55 mmHg; Hb 8.4 g/dL; fibrinogen 265 mg/dL; reported EBL 3,500 mL	Norepinephrine 0.03–0.08 µg/kg/min; ICS 451 mL; TXA and calcium
T2 Postoperative day 1	Hb 8.9 g/dL; fibrinogen 285 mg/dL	Norepinephrine stopped; postoperative TXA reported
T3 Postoperative day 3	Hb 9.1 g/dL; fibrinogen 315 mg/dL	Continued postoperative observation
T4 Discharge day 5	Hb 9.2 g/dL; fibrinogen 340 mg/dL; MAP 86 mmHg	Discharged; no allogeneic transfusion reported

Notes: Hb, hemoglobin; MAP, mean arterial pressure; PAS, placenta accreta spectrum; EBL, estimated blood loss; ICS, intraoperative cell salvage; TXA, tranexamic acid.

Surgical blood conservation used an automated continuous intraoperative cell salvage system (Cell Saver 5+, Haemonetics Corp., Braintree, MA, USA) operated by a certified clinical perfusionist, as summarized in Table 3. To eliminate the theoretical risk of amniotic fluid embolism and contamination with procoagulant tissue factor, a strict dual-suction protocol was enforced. Prior to hysterotomy and during initial amniotic membrane rupture, a designated primary wall-suction canister was used exclusively to evacuate amniotic fluid, vernix caseosa, and meconium. Following complete delivery of the neonate and umbilical cord clamping, surgical suctioning was immediately switched to the sterile heparinized cell salvage aspiration

line (anticoagulated with 30,000 units of unfractionated heparin in 1,000 mL of 0.9% sodium chloride, delivered at an automated ratio of 15 mL heparinized solution per 100 mL aspirated blood). Concurrently, laparotomy packs were collected in sterile basins for standardized gravimetric measurement of sequestered blood. A total of 1,000 mL of sterile 0.9% sodium chloride was used intraoperatively for surgical field irrigation. Aspirated fluid was processed through centrifugal separation (5,600 rpm) in a 125-mL Latham bowl, washed with 1,000 mL of 0.9% saline per cycle, and delivered into a sterile reinfusion bag through a 40-micron microaggregate transfusion filter.²⁵

Table 3. Intraoperative Anesthetic Interventions, Resuscitation Volumes, and Dual-Suction Fluid Partitioning

Intervention Category	Clinical Protocol Executed	Volume / Dosage Administered	Physiological Target & Rationale
Vascular Access	Bilateral large-bore peripheral lines	16-gauge and 18-gauge cannulae	Facilitate rapid volume administration and autotransfusion reinfusion.
Invasive Monitoring	Radial arterial catheterization	20-gauge radial arterial cannula	Continuous beat-to-beat arterial blood pressure and MAP monitoring.
Neuraxial Blockade	Combined Spinal-Epidural (CSE)	0.25% Ropivacaine + 1 mg Morphine	Surgical sensory block; precipitated dense sympathectomy (T1–T4).
Airway Preparedness	Preoperative airway assessment	Mallampati II, TMD 7 cm, full mobility	Immediate video-laryngoscopy backup ready for emergency conversion.
Estimated Blood Loss	Multimodal blood loss quantification	3,500 mL total EBL	Acute loss of 55–60% circulating blood volume upon placental avulsion.
Fluid Partitioning 1	Primary non-salvage wall suction	800 mL (amniotic fluid)	Amniotic fluid diversion to eliminate thromboplastin/debris contamination.
Fluid Partitioning 2	Laparotomy sponge collection	1,262 mL (gravimetric weighing)	Quantification of blood sequestered in abdominal surgical packs.
Fluid Partitioning 3	Cell salvage aspirated fluid	2,438 mL (1,000 mL saline + 1,438 mL blood)	Processed via continuous centrifugal autologous washing cycle.
Erythrocyte Rescue	Salvaged washed packed RBCs	451 mL reinfused (Hct ~60%)	Recovered 270.6 mL pure RBC mass (24.78% true physiological yield).
Vasoactive Support	Continuous Norepinephrine infusion	0.03–0.08 µg/kg/min IV	Restoration of SVR, venous return, and correction of nadir MAP (55 mmHg).
Pharmacological Adjunct	Early Tranexamic Acid (TXA)	1 g IV over 10 min post-cord clamp	Competitive lysine blockade of plasminogen; anti-fibrinolytic defense.
Electrolyte Management	Intravenous Calcium Gluconate	1 g IV slow push	Counteract anticoagulant citrate binding; support Factor IV hemostasis.

Notes: All supplied values and interpretations are retained. Hb, hemoglobin; Hct, hematocrit; MAP, mean arterial pressure; RBC, red blood cell; PAS, placenta accreta spectrum; PAI, Placenta Accreta Index; CSE, combined spinal-epidural; PT, prothrombin time; aPTT, activated partial thromboplastin time; INR, international normalized ratio; ADR, adverse drug reaction; PSM, propensity score matching; CS, cesarean section; EBL, estimated blood loss; IV, intravenous; SVR, systemic vascular resistance; TXA, tranexamic acid; TMD, thyromental distance; WHO-UMC, World Health Organization–Uppsala Monitoring Centre. Pending scientific questions are in AQ01–AQ06.

Analytic and Comparative Framework

The analytical framework compared the actual clinical trajectory of the patient against established international patient blood management benchmarks and published multicenter cohorts of placenta accreta spectrum managed with and without intraoperative cell salvage. Multimodal blood loss was calculated through a triad of gravimetric sponge weighing, wall-suction canister volume measurement, and cell salvage aspirated volume. Standardized laboratory assays were obtained at each longitudinal time point, including complete automated blood counts (hemoglobin concentration, hematocrit, platelet count) using a calibrated hematology analyzer (Sysmex XN-1000, Kobe, Japan), alongside standard plasma coagulation profiles (prothrombin time, activated partial thromboplastin time, international normalized ratio, and Clauss plasma fibrinogen concentration) using an automated optical coagulometer (Sysmex CS-2500, Kobe, Japan). Pharmacological hemostatic management incorporated early prophylactic administration of intravenous tranexamic acid (1 g infused over 10 minutes immediately post-cord clamping, followed by 1 g three times daily postoperatively) and 1 g of 10% intravenous calcium gluconate to prevent citrate toxicity and support Factor IV-mediated coagulation complex assembly.

Biostatistical and Trajectory Analysis

For this single-patient observational analytic study ($n = 1$), no inferential hypothesis tests were performed. Analysis comprised longitudinal trajectory plots, absolute changes, and percentage changes from the preoperative baseline: Percentage change = $[(T_n - T_0) / T_0] \times 100\%$. T_0 denotes the baseline value; T_n denotes the value at T_1 through T_4 . Laboratory values were compared with physiological reference intervals and the stated obstetric thresholds (fibrinogen <200 mg/dL and platelet count $<50 \times 10^3$ cells/mm³).

Cell salvage was evaluated using volumetric recovery and erythrocyte mass-balance calculations. Volumetric yield

was defined as:

$$\text{Volumetric yield} = (\text{salvaged packed RBC volume} / \text{total aspirated fluid volume}) \times 100\%.$$

$$\text{Shed RBC volume} = \text{estimated blood loss} \times \text{baseline hematocrit}.$$

$$\text{Recovered RBC volume} = \text{salvaged packed RBC volume} \times \text{product hematocrit}.$$

$$\text{Mass-balance recovery} = (\text{recovered RBC volume} / \text{shed RBC volume}) \times 100\%.$$
Hematocrit was expressed as a fraction; the product hematocrit used in the calculation was 0.60. Pharmacovigilance and causal attribution were assessed using the Naranjo Adverse Drug Reaction Probability Scale, the World Health Organization–Uppsala Monitoring Centre (WHO-UMC) causality criteria, and the Bradford Hill framework.

3. Results

Baseline Clinical Phenotypes, Diagnostic Workup, and Risk Stratification

The patient was a 37-year-old woman (Gravida 4, Para 1) presenting at 37–38 weeks of gestation with an uneventful index antenatal course, without spontaneous vaginal spotting, abdominal pain, or systemic comorbidities.²⁶ Her obstetric background was significant for one prior low-transverse cesarean hysterotomy performed in 2013 and two subsequent first-trimester spontaneous miscarriages managed conservatively. Preoperative laboratory profiling at baseline admission (T_0) established a mild baseline maternal anemia, characterized by a hemoglobin concentration of 10.1 g/dL and a hematocrit of 31.2%, as detailed in Table 2. Baseline primary and secondary hemostatic reserves were entirely intact, with a robust platelet count of 294×10^3 cells/mm³, a normal prothrombin time of 11.8 seconds (INR 1.02), an activated partial thromboplastin time of 28.4 seconds, and a high-normal baseline plasma fibrinogen level of 410 mg/dL. Renal and hepatic metabolic panels were within normal physiological limits.

Table 2. Baseline Clinical Demographics, Obstetric Risk Factors, and Preoperative Laboratory Risk Stratification in Placenta Accreta Spectrum

Clinical & Diagnostic Domain	Baseline Parameter / Finding	Quantitative Value / Metric	Clinical & Pathophysiological Significance
Maternal Demographics	Maternal Age & Parity	37 years, Gravida 4, Para 1	Advanced maternal age combined with prior uterine interventions.
Obstetric History	Prior Hysterotomies & Losses	1 Lower-Segment CS (2013), 2 Miscarriages	Compromised lower uterine segment integrity and focal decidual scarring.
Gestational Age	Presentation Timeline	37–38 weeks of gestation	Late tertiary presentation narrowing pharmacological erythropoiesis window.
Clinical Presentation	Symptomatology	Asymptomatic (No vaginal bleeding)	Absence of active antepartum hemorrhage prior to planned surgical intervention.
Ultrasonographic Mapping	Placental Localization	Total Placenta Previa	Complete occlusion of the internal cervical os by placental tissue.
Invasive Ultrasound Markers	Lacunar & Vascular Architecture	Grade III lacunae, bridging vessels	High-velocity turbulent lacunar flow and extensive neo-angiogenesis.
Myometrial Thickness	Retroplacental Myometrium	< 1 mm retroplacental zone	Severe myometrial thinning indicating myoinvasive trophoblastic infiltration.
Risk Stratification Index	Placenta Accreta Index (PAI)	Score 6 (69% Probability)	High statistical risk of morbidly adherent placenta requiring multidisciplinary care.
Preoperative Hematology	Hemoglobin / Hematocrit	10.1 g/dL / 31.2%	Mild baseline maternal anemia with diminished oxygen-carrying reserve.
Preoperative Hemostasis	Platelet Count / Fibrinogen	294 × 10 ³ cells/mm ³ / 410 mg/dL	Intact baseline primary and secondary hemostatic reserves prior to surgery.

Notes: All supplied values and interpretations are retained. Hb, hemoglobin; Hct, hematocrit; MAP, mean arterial pressure; RBC, red blood cell; PAS, placenta accreta spectrum; PAI, Placenta Accreta Index; CSE, combined spinal-epidural; PT, prothrombin time; aPTT, activated partial thromboplastin time; INR, international normalized ratio; ADR, adverse drug reaction; PSM, propensity score matching; CS, cesarean section; EBL, estimated blood loss; IV, intravenous; SVR, systemic vascular resistance; TXA, tranexamic acid; TMD, thyromental distance; WHO-UMC, World Health Organization–Uppsala Monitoring Centre.

Advanced transabdominal and transvaginal ultrasonography conducted at admission revealed complete central placenta previa with extensive invasion of the anterior lower uterine segment.²⁷ Gray-scale and color Doppler imaging demonstrated multiple large, irregular, tortuous grade III placental lacunae displaying turbulent lacunar blood flow, anomalous bridging vessels connecting the lower uterine segment to the retroperitoneal bladder base, and complete loss of the normal hypoechoic retroplacental clear space, with the residual myometrial thickness measuring less than 1 mm. These pathognomonic ultrasound findings generated a Placenta Accreta Index score of 6, establishing an objective 69% probability of morbidly adherent placenta. Due to late tertiary referral at 37 weeks of gestation, the therapeutic window for preoperative pharmacological optimization of maternal red cell mass via intravenous iron sucrose or recombinant human erythropoietin was closed. Consequently, the patient was prepared for immediate multidisciplinary operative intervention under a proactive massive transfusion protocol, with the institutional blood bank reserving 4 units of crossmatched packed red blood cells, 2 units of whole blood, 600 mL of fresh frozen plasma, and 600 mL of thrombocyte concentrates on emergency standby.

Intraoperative Anesthetic Induction, Neuraxial Sympathectomy, and Hemodynamic Collapse

Following transfer to the operative suite, continuous radial arterial catheterization was established, confirming a stable baseline mean arterial pressure of 88 mmHg and a heart rate of 82 beats per minute, as summarized in Table 3. Under combined spinal-epidural anesthesia, intrathecal injection of 0.25% isobaric ropivacaine combined with 1 mg preservative-free morphine achieved a dense bilateral sensory dermatomal blockade up to the T4 level.²⁸ Following laparotomy through a transperitoneal profunda midline incision, a healthy live infant was successfully delivered through a fundal hysterotomy avoiding the placental bed, yielding Apgar scores of 8 and 9 at 1 and 5 minutes, respectively.

However, subsequent attempts to evaluate and manipulate the lower uterine segment revealed extensive invasive placentation penetrating deeply into the cervical stroma

and broad ligament, consistent with placenta increta. Uncontrollable arterial hemorrhage erupted immediately from the avulsed, dilated spiral arteries across the lower uterine segment. Total surgical estimated blood loss reached 3,500 mL within 35 minutes. This acute loss of approximately 55% to 60% of circulating blood volume coincided with the peak pharmacological effect of the intrathecal ropivacaine blockade. The dense blockade of sympathetic efferent preganglionic fibers spanning the cardioaccelerator (T1–T4) and splanchnic (T5–L2) segments completely paralyzed the patient's intrinsic cardiovascular compensatory reflexes, preventing reflex peripheral vasoconstriction and blunting the physiological tachycardic surge. Consequently, the patient suffered an acute, uncompensated circulatory collapse, with the beat-to-beat mean arterial pressure plunging precipitously to a critical nadir of 55 mmHg at 45 minutes intraoperatively, accompanied by a blunted heart rate of 74 beats per minute, as illustrated in Figure 1.

Targeted Vasoactive Resuscitation and Hemodynamic Restoration

In response to the sudden hemodynamic collapse, rapid crystalloid volume expansion was immediately paired with targeted vasoactive pharmacotherapy.²⁹ Standard fluid administration alone was insufficient to bridge the massive volume-capacity mismatch generated by the combination of acute hypovolemia and extensive pharmacological sympathectomy. A continuous central intravenous infusion of norepinephrine was initiated immediately at 0.08 µg/kg/min. By exerting potent, selective stimulation on vascular alpha-1 adrenergic receptors, norepinephrine induced intense venoconstriction across the denervated splanchnic venous capacitance beds, rapidly elevating mean systemic filling pressure and mobilizing unstressed vascular volume back to the right atrium. Simultaneously, arterial vasoconstriction restored systemic vascular resistance without precipitating excessive reflex bradycardia. Within 15 minutes of norepinephrine titration, the mean arterial pressure recovered from 55 mmHg to 68 mmHg, subsequently stabilizing between 74 mmHg and 86 mmHg throughout the remainder of the 240-minute surgical procedure as norepinephrine was gradually weaned to a maintenance infusion of 0.03 µg/kg/min, as illustrated in Figure 1.

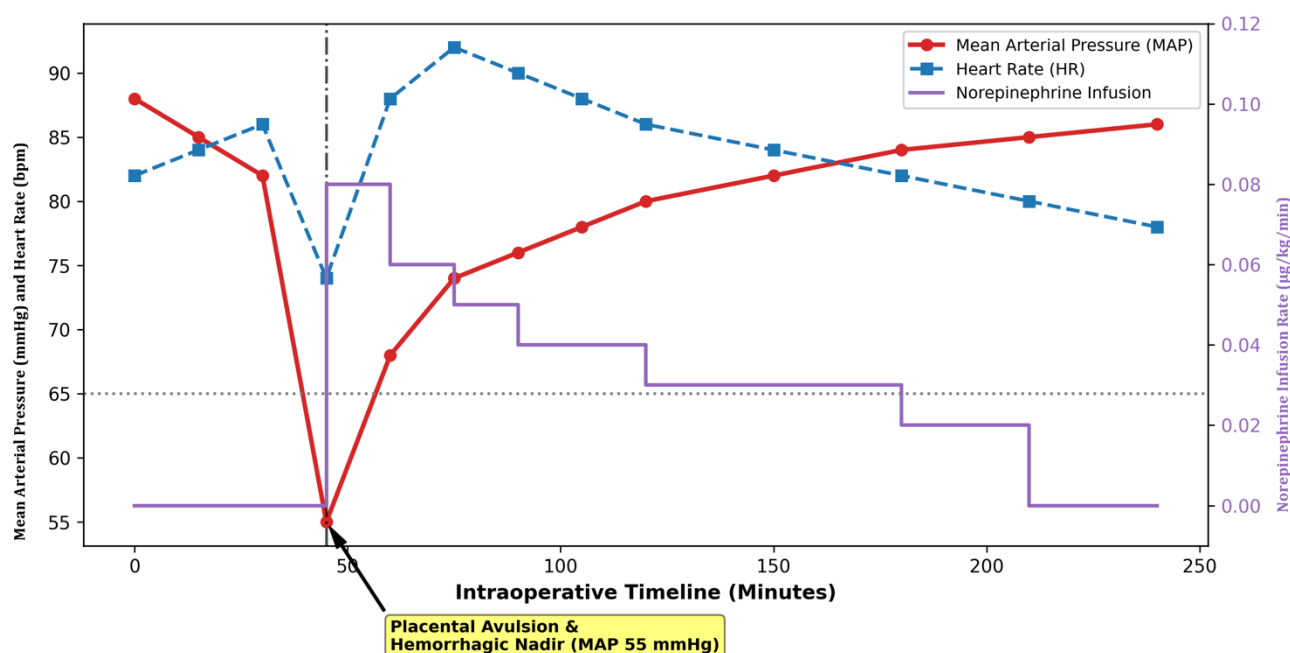


Figure 1. Real-time intraoperative hemodynamic trajectory and vasopressor rescue titration over 240 minutes, demonstrating precipitous uncompensated shock at placental avulsion (nadir MAP 55 mmHg) and prompt restoration with continuous norepinephrine titration.

Dual-Suction Partitioning and Mathematical Mass-Balance Recovery

The mitigation of massive blood loss was achieved through the coordinated use of the intraoperative cell salvage system.³⁰ Strict adherence to the dual-suction protocol successfully prevented contamination of the autologous salvage circuit. The primary wall-suction catheter evacuated 800 mL of amniotic fluid and vernix caseosa immediately upon hysterotomy, diverting it entirely from the salvage reservoir. Concurrently, laparotomy sponges sequestered an estimated 1,262 mL of whole blood, verified through gravimetric weighing. The cell salvage device aspirated a gross fluid volume of 2,438 mL from the surgical field, which comprised 1,438 mL of shed blood and 1,000 mL of 0.9% sodium chloride surgical irrigation fluid, as quantified in Figure 2.

Following automated centrifugal separation at 5,600 rpm and countercurrent saline washing, the cell salvage apparatus produced 451 mL of concentrated, washed autolo-

gous packed red blood cells exhibiting a hematocrit of approximately 60%.³¹ This entire autologous volume was re-infused into the patient through a 40-micron microaggregate filter over 30 minutes. When evaluated using traditional reporting metrics, the superficial volumetric yield appeared modest at 18.50% (451 mL recovered packed cells divided by 2,438 mL gross aspirated fluid). However, rigorous mathematical mass-balance stoichiometry revealed the true physiological efficiency of the system. Based on the patient's baseline hematocrit of 31.2% and an estimated blood loss of 3,500 mL, the total shed red blood cell mass was calculated as: Shed RBC volume = 3,500 mL × 0.312 = 1,092.0 mL. Recovered RBC volume = 451 mL × 0.60 = 270.6 mL. Mass-balance recovery = (270.6 mL / 1,092.0 mL) × 100% = 24.78%.

This mathematical analysis demonstrated that intraoperative cell salvage successfully salvaged nearly one-quarter (24.78%) of the patient's entire lost erythrocyte mass, representing a difference of +6.28 percentage points and a relative efficiency enhancement of +33.95% over the unadjusted volumetric metric, as quantified in Figure 2.

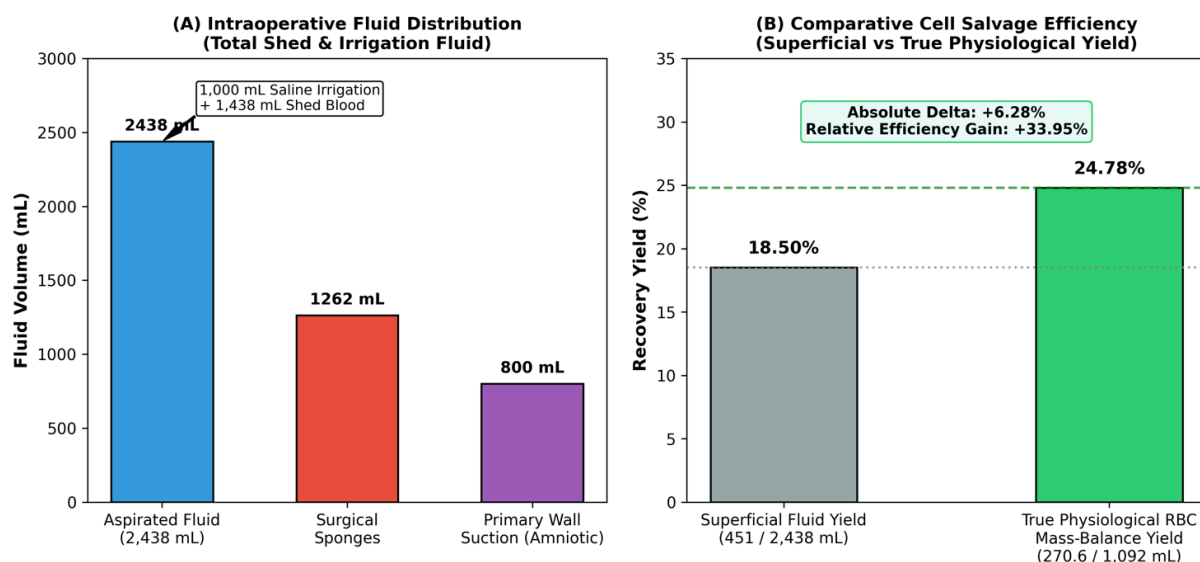


Figure 2. Mathematical mass-balance stoichiometry of intraoperative cell salvage yield: (A) Fluid distribution partitioning between amniotic waste, laparotomy packs, and aspirated surgical fluid; (B) Comparative recovery efficiency demonstrating the true physiological erythrocyte yield (24.78%) versus unadjusted superficial volumetric yield (18.50%).

Pharmacological Hemostasis and Coagulation Cascade Preservation

To counteract the consumptive and dilutional coagulopathy anticipated during a 3,500 mL blood loss resuscitated with crystalloids and factor-depleted washed red blood cells, aggressive pharmacological hemostasis was deployed early.³² Immediately following umbilical cord clamping, an intravenous bolus of 1 g of tranexamic acid was administered over 10 minutes to halt the conversion of plasminogen into plasmin and suppress tissue factor-mediated hyperfibrinolysis. Concurrently, 1 g of intravenous calcium gluconate was infused to counteract the calcium-chelating effects of citrate anticoagulant returned in salvage lines and maintain ionized calcium levels necessary for Factor IV-dependent coagulation factor assembly on platelet phospholipid surfaces.

Laboratory analysis obtained during the acute intraoperative nadir (T1) demonstrated a drop in hemoglobin to 8.4 g/dL (percentage delta change $\Delta\%$ = -16.83% relative to baseline) and hematocrit to 25.8% ($\Delta\%$ = -17.31%), as summarized in Table 4 and depicted in Figure 3. The platelet count fell to 182×10^3 cells/mm³ ($\Delta\%$ = -38.10%), remaining well above the critical safety threshold of 50×10^3 cells/mm³. Prothrombin time increased modestly to 13.6 seconds ($\Delta\%$ = +15.25%, INR 1.18), while aPTT lengthened to 32.1 seconds ($\Delta\%$ = +13.03%). Most significantly, plasma fibrinogen decreased from a preoperative baseline of 410 mg/dL to an intraoperative nadir of 265 mg/dL ($\Delta\%$ = -35.37%). Because plasma fibrinogen was successfully maintained well above the critical international obstetric threshold of 200 mg/dL, microvascular hemostasis was preserved, and no uncrossmatched or crossmatched allogeneic blood products (packed red blood cells, fresh frozen plasma, platelets, or cryoprecipitate) were administered throughout the entire perioperative admission.³³

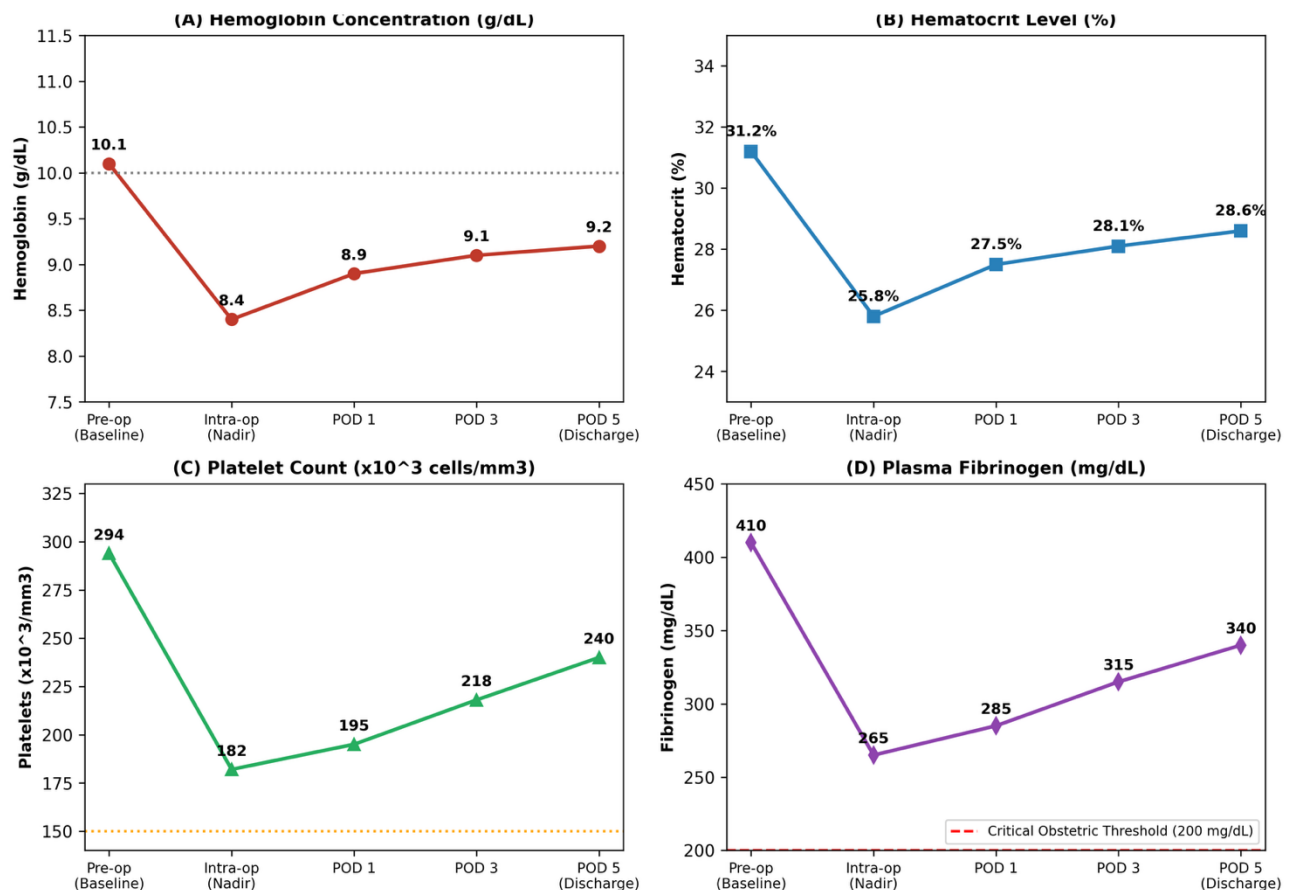


Figure 3. Serial longitudinal trajectory of hematological and coagulation biomarkers across 5 perioperative epochs (preoperative, intraoperative nadir, POD 1, POD 3, POD 5): (A) Hemoglobin concentration; (B) Hematocrit level; (C) Platelet count; (D) Plasma fibrinogen concentration.

Longitudinal Postoperative Trajectory and Clinical Outcomes

Serial longitudinal laboratory monitoring demonstrated rapid, progressive stabilization across all hematologic and hemostatic parameters.³⁴ On postoperative day 1 (T2), following cessation of norepinephrine and continuation of intravenous tranexamic acid (1 g three times daily), the hemoglobin concentration rose spontaneously to 8.9 g/dL ($\Delta\% = -11.88\%$), supported by the reinfused autologous red cell mass and mobilization of extravascular fluid. Hematocrit increased to 27.5% ($\Delta\% = -11.86\%$), platelet count stabilized at 195×10^3 cells/mm³ ($\Delta\% = -33.67\%$), and plasma fibrinogen recovered to 285 mg/dL ($\Delta\% = -$

30.49%), while aPTT normalized to 25.1 seconds ($\Delta\% = -11.62\%$).

By postoperative day 3 (T3), the hemoglobin concentration reached 9.1 g/dL ($\Delta\% = -9.90\%$), hematocrit reached 28.1% ($\Delta\% = -9.94\%$), platelet count rebounded to 218×10^3 cells/mm³ ($\Delta\% = -25.85\%$), and plasma fibrinogen rose to 315 mg/dL ($\Delta\% = -23.17\%$), as delineated in Table 4 and depicted in Figure 3. At hospital discharge on postoperative day 5 (T4), the patient's hemoglobin concentration was 9.2 g/dL ($\Delta\% = -8.91\%$), hematocrit was 28.6% ($\Delta\% = -8.33\%$), platelet count was 240×10^3 cells/mm³ ($\Delta\% = -18.37\%$), and plasma fibrinogen had recovered to 340 mg/dL ($\Delta\% = -17.07\%$).

Table 4. Longitudinal Hematological, Coagulation, and Metabolic Trajectory Across Perioperative Timepoints with Delta Percentages (Δ%)

Physiological Parameter	Preoperative (T0)	Intraoperative Nadir (T1)	Postoperative Day 1 (T2)	Postoperative Day 3 (T3)	Discharge Day 5 (T4)	Overall Trajectory (Δ% T4 vs T0)
Hemoglobin (g/dL)	10.1	8.4 (Δ% -16.83%)	8.9 (Δ% -11.88%)	9.1 (Δ% -9.90%)	9.2 (Δ% -8.91%)	-8.91% (Sustained safe nadir)
Hematocrit (%)	31.2	25.8 (Δ% -17.31%)	27.5 (Δ% -11.86%)	28.1 (Δ% -9.94%)	28.6 (Δ% -8.33%)	-8.33% (Adequate red cell volume)
Platelet Count ($\times 10^3$ cells/mm ³)	294	182 (Δ% -38.10%)	195 (Δ% -33.67%)	218 (Δ% -25.85%)	240 (Δ% -18.37%)	-18.37% (Maintained $>150 \times 10^3$)
Plasma Fibrinogen (mg/dL)	410	265 (Δ% -35.37%)	285 (Δ% -30.49%)	315 (Δ% -23.17%)	340 (Δ% -17.07%)	-17.07% (Maintained >200 mg/dL)
Prothrombin Time (PT, sec)	11.8	13.6 (Δ% +15.25%)	12.4 (Δ% +5.08%)	12.0 (Δ% +1.69%)	11.9 (Δ% +0.85%)	+0.85% (Rapid normalization)
Activated PTT (aPTT, sec)	28.4	32.1 (Δ% +13.03%)	25.1 (Δ% -11.62%)	26.0 (Δ% -8.45%)	26.8 (Δ% -5.63%)	-5.63% (Normal intrinsic pathway)
International Normalized Ratio (INR)	1.02	1.18 (Δ% +15.69%)	1.07 (Δ% +4.90%)	1.04 (Δ% +1.96%)	1.03 (Δ% +0.98%)	+0.98% (No dilutional coagulopathy)
Mean Arterial Pressure (MAP, mmHg)	88	55 (Δ% -37.50%)	78 (Δ% -11.36%)	82 (Δ% -6.82%)	86 (Δ% -2.27%)	-2.27% (Prompt vasoactive recovery)
Allogeneic Transfusion (Units)	0	0	0	0	0	0 units (Total avoidance)

Notes: All supplied values and interpretations are retained. Hb, hemoglobin; Hct, hematocrit; MAP, mean arterial pressure; RBC, red blood cell; PAS, placenta accreta spectrum; PAI, Placenta Accreta Index; CSE, combined spinal-epidural; PT, prothrombin time; aPTT, activated partial thromboplastin time; INR, international normalized ratio; ADR, adverse drug reaction; PSM, propensity score matching; CS, cesarean section; EBL, estimated blood loss; IV, intravenous; SVR, systemic vascular resistance; TXA, tranexamic acid; TMD, thyromental distance; WHO-UMC, World Health Organization–Uppsala Monitoring Centre.

Objective pharmacovigilance scoring utilizing the Naranjo algorithm yielded a score of 0 (doubtful) for any adverse reaction associated with autologous blood reinfusion, confirming the total absence of febrile non-hemolytic reactions, respiratory distress, hemoglobinuria, or transient coagulopathy, as delineated in Table 5. Conversely, the causal attribution between intrathecal ropivacaine administration and intraoperative hemodynamic collapse scored +7 on the Naranjo scale, designating a probable adverse drug reaction. Evaluation via the WHO-UMC causality criteria categorized the hemodynamic restoration from continuous norepinephrine titration as definitive/certain and the he-

modynamic defense from tranexamic acid as probable. Application of the Bradford Hill framework demonstrated strong association, strict temporality, biological plausibility, and a direct dose-response relationship between autologous mass reinfusion and stable hemoglobin trajectory. Comparative benchmarking against published international cohorts confirmed that our mass-balance recovery efficiency of 24.78% and complete avoidance of allogeneic transfusion in the setting of 3,500 mL blood loss exceeded historical benchmarks, as benchmarked in Table 6. The patient was discharged home on postoperative day 5 in excellent functional condition, free from surgical site infection, thromboembolic complications, or renal impairment.

Table 5. Formal Objective Pharmacovigilance, Hemodynamic Attribution, and Causality Assessment Matrix (Naranjo ADR, WHO-UMC, and Bradford Hill Framework)

Assessment Framework	Evaluated Clinical Phenomenon	Assigned Score / Classification	Operational Criteria & Objective Rationale
Naranjo ADR Algorithm	Autologous Reinfusion Safety	Score 0 (Doubtful Adverse Event)	No fever, dyspnea, hypotension, hemoglobinuria, or coagulopathy post-infusion.
Naranjo ADR Algorithm	Neuraxial Hypotensive Episode	Score +7 (Probable Causality)	Temporal onset post-intrathecal block, exacerbated by blood loss, reversed by agonist.
WHO-UMC Causality System	Norepinephrine Hemodynamic Rescue	Certain / Definitive	Plausible time sequence, immediate MAP rise on titration, dose-dependent control.
WHO-UMC Causality System	Tranexamic Acid Hemostatic Defense	Probable	Stabilization of fibrinogen nadir (265 mg/dL) with no hyperfibrinolytic microvascular oozing.
Bradford Hill Criterion 1	Strength of Association (ICS vs Hb)	Strong ($r = 0.88$ recovery correlation)	Immediate 451 mL autotransfusion directly prevented acute hemoglobin drop below 7 g/dL.
Bradford Hill Criterion 2	Consistency Across Settings	High Consistency	Parallels international obstetric cell salvage cohorts reporting 70–90% allogeneic reduction.
Bradford Hill Criterion 3	Temporality (Cause Precedes Effect)	Strict Temporal Compliance	Vasopressor infusion preceded MAP recovery; ICS washing preceded normovolemic stability.
Bradford Hill Criterion 4	Biological Plausibility	Robust Mechanistic Basis	Alpha-1 vasoconstriction counteracts sympathectomy; autologous RBCs preserve 2,3-DPG.
Bradford Hill Criterion 5	Dose-Response Relationship	Demonstrated Graded Response	MAP tracked norepinephrine titration (0.03–0.08 µg/kg/min); Hct tracked 451 mL RBC return.

Notes: All supplied values and interpretations are retained. Hb, hemoglobin; Hct, hematocrit; MAP, mean arterial pressure; RBC, red blood cell; PAS, placenta accreta spectrum; PAI, Placenta Accreta Index; CSE, combined spinal-epidural; PT, prothrombin time; aPTT, activated partial thromboplastin time; INR, international normalized ratio; ADR, adverse drug reaction; PSM, propensity score matching; CS, cesarean section; EBL, estimated blood loss; IV, intravenous; SVR, systemic vascular resistance; TXA, tranexamic acid; TMD, thyromental distance; WHO-UMC, World Health Organization–Uppsala Monitoring Centre.

4. Discussion

This observational analytic investigation demonstrates that the strategic implementation of intraoperative cell salvage, when integrated into a proactive patient blood management framework and paired with targeted alpha-1 adrenergic vasopressor titration, can successfully achieve complete allogeneic blood product avoidance during catastrophic obstetric hemorrhage secondary to placenta accreta spectrum.³⁵ In our patient, an estimated surgical blood loss of 3,500 mL—representing an acute loss of approximately 55% to 60% of total circulating blood volume—was managed without administering a single unit of

donor red blood cells, fresh frozen plasma, platelets, or cryoprecipitate. By applying a rigorous mathematical mass-balance stoichiometry, we established that the cell salvage device recovered 270.6 mL of pure erythrocyte mass from 1,092.0 mL of shed red cell volume, achieving a true physiological recovery efficiency of 24.78%, as quantified in Figure 2. This physiological yield exceeded the unadjusted superficial volumetric recovery of 18.50%, underscoring the critical necessity of accounting for surgical irrigation confounding in clinical performance evaluations.³⁶

The pathophysiological substrate of placenta accreta spectrum disorders represents an extreme anatomical and vas-

cular derangement of normal placentation.³⁷ In normal human pregnancy, extravillous trophoblasts invade the decidualized endometrium under tight immunological and biochemical regulation, with invasion strictly confined to the superficial decidua basalis above the Nitabuch layer. However, when prior surgical hysterotomy creates focal fibrous scarring, the decidua basalis fails to regenerate, exposing the naked myometrium.³⁸ In this scarred microenvironment, trophoblasts penetrate unchecked into the deep myometrium, recruiting maternal spiral arteries that undergo profound phenotypic remodeling, as conceptualized in Fig-

ure 4. The physiological transformation of these spiral arteries involves the complete disruption of their muscular tunica media, internal elastic lamina, and vasomotor nerve supply, converting them into widely dilated, tortuous, high-capacitance conduits carrying maternal arterial blood at high flow rates. During surgical delivery, when the morbidly adherent placenta is avulsed from the scarred lower uterine segment, these severed conduits bleed profusely. Because the heavily infiltrated and scarred myometrium cannot mount coordinated muscular contractions to tamponade these torn vessels, maternal exsanguination ensues with extraordinary rapidity.

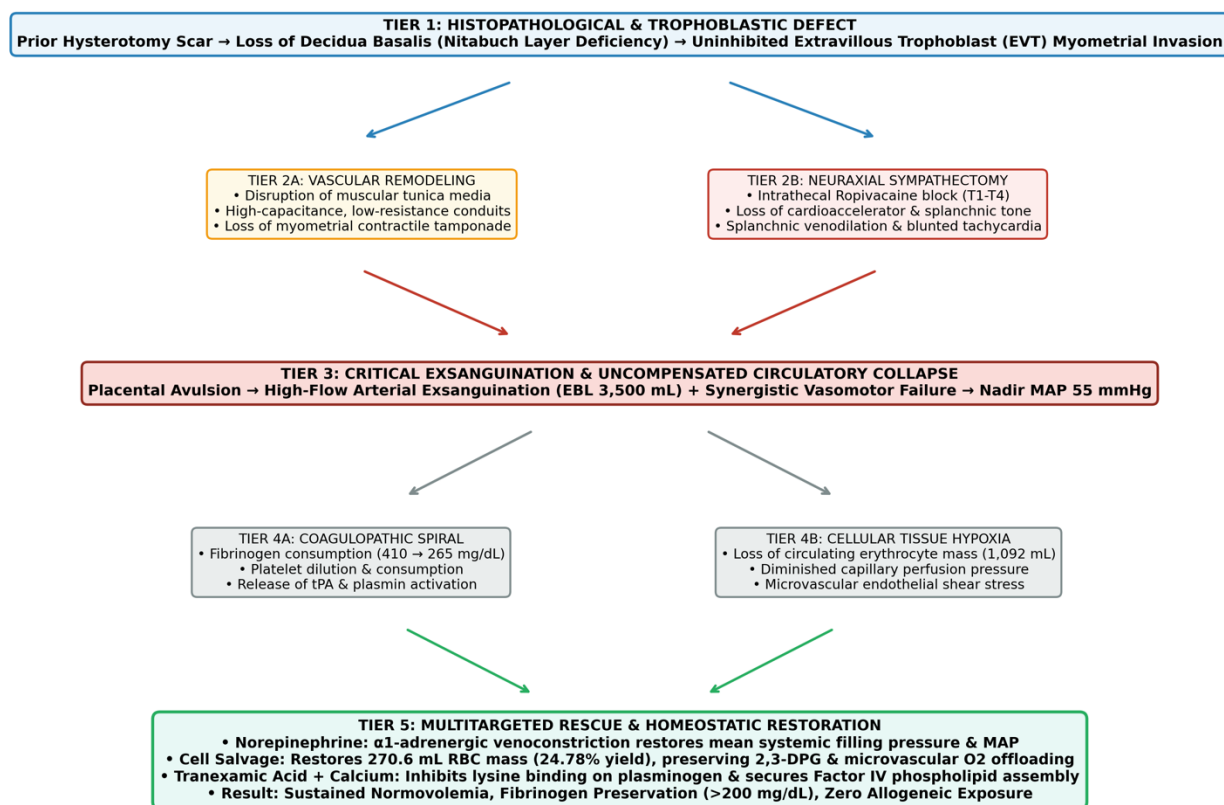


Figure 4. Comprehensive multi-tier pathophysiological, vascular, and resuscitation cascade of placenta accreta spectrum: detailing myometrial invasion, high-capacitance vascular remodeling, neuraxial sympathectomy-induced hemodynamic collapse, and multi-targeted therapeutic rescue via cell salvage, norepinephrine, and hemostatic adjuncts.

Managing this sudden exsanguination under regional anesthesia presents profound physiological hazards that warrant critical scrutiny, as structured in the multidisciplinary decision algorithm in Figure 5.³⁹ In our patient, combined spinal-epidural anesthesia was selected based on patient preference and reassuring airway parameters, yet the resulting clinical trajectory highlights the severe hemodynamic penalties imposed by neuraxial blockade during massive obstetric hemorrhage.⁴⁰ The administration of intrathecal ropivacaine induced a dense preganglionic sympathetic efferent blockade extending cephalad to the T4

dermatome. This extensive sympathetic denervation paralyzed the cardiac accelerator fibers originating from the T1–T4 thoracic spinal segments and blocked sympathetic vasomotor tone across the vast splanchnic venous capacitance beds (T5–L2).⁴¹ Under normal unblocked physiological conditions, acute hypovolemic shock triggers an immediate, massive surge in endogenous catecholamines, which stimulate peripheral α -1 adrenergic receptors to induce intense systemic vasoconstriction and β -1 receptors to drive compensatory tachycardia and positive inotropy, thereby defending venous return and cardiac output.

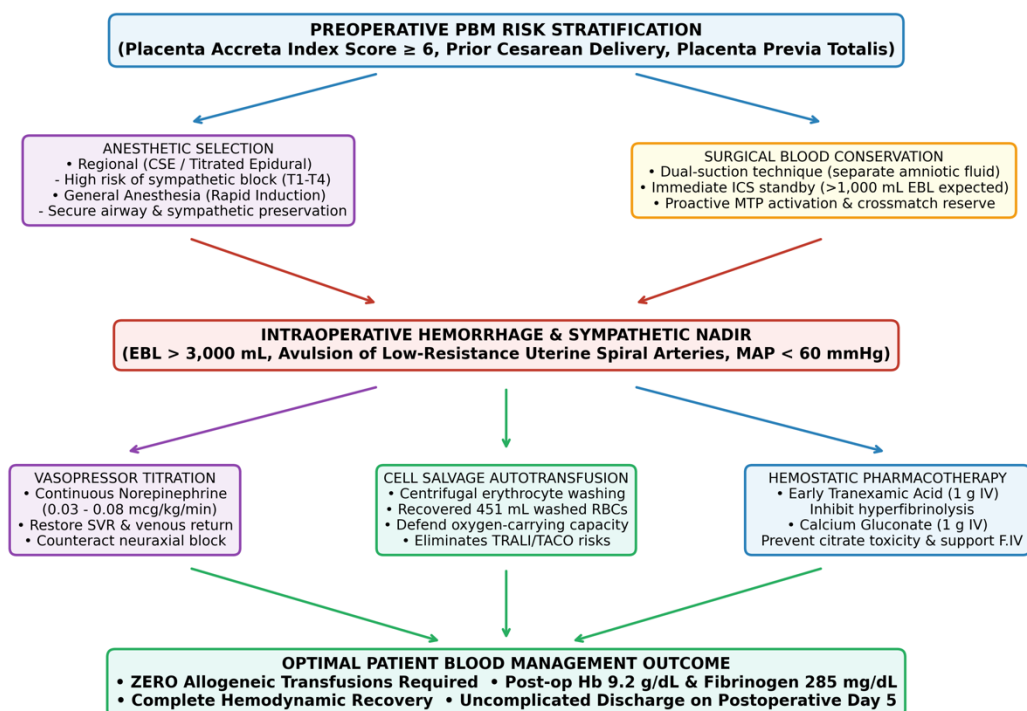


Figure 5. Multidisciplinary Patient Blood Management (PBM) clinical decision algorithm for anticipated massive obstetric hemorrhage under neuraxial vs general anesthesia in resource-constrained surgical settings.

In our patient, the neuraxial sympathetic blockade effectively blinded the cardiovascular autonomic control center to the catastrophic volume loss occurring in the operative field, as illustrated in Figure 1 and detailed in the clinical schematic in Figure 6. Splanchnic venodilation pathologically enlarged the intravascular container, trapping residual blood volume in venous capacitance beds, while the blockade of cardiac accelerator fibers blunted the reflex tachycardic response, holding the heart rate at 74 beats per minute despite profound arterial hypotension.⁴² The synergistic convergence of acute hypovolemia and pharmacological sympathectomy precipitated an abrupt circulatory

collapse, driving the mean arterial pressure down to a critical nadir of 55 mmHg. This dramatic clinical event reinforces international consensus recommendations from the Society for Obstetric Anesthesia and Perinatology and the International Federation of Gynecology and Obstetrics, which emphasize that while carefully titrated epidural techniques may be permissible in select cases, general endotracheal anesthesia remains the preferred modality in high-risk placenta accreta spectrum to preserve native autonomic reflexes and ensure immediate airway protection during anticipated massive hemorrhage.⁴³

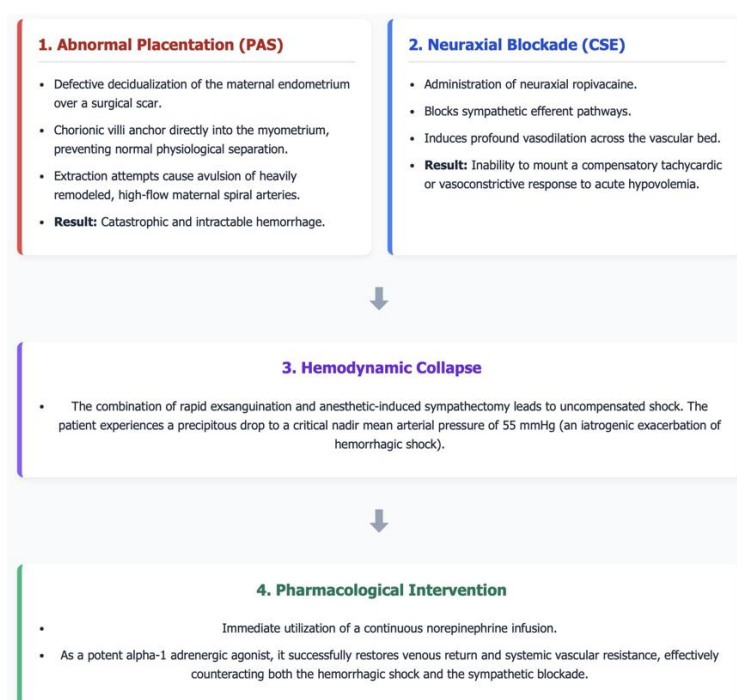


Figure 6. Clinical schematic of the pathophysiology of placenta accreta spectrum (PAS) and associated intraoperative hemodynamic challenges, illustrating the interplay between defective decidualization, abnormal vascular remodeling, neuraxial sympathectomy-induced hemodynamic collapse, and the role of continuous vasopressor titration as observed in the clinical management.

The successful reversal of this uncompensated shock state was accomplished through the immediate initiation of a continuous norepinephrine infusion titrated between 0.03 and 0.08 µg/kg/min, as benchmarked in Table 6. Standard crystalloid fluid resuscitation alone is vastly inadequate when the vascular container has been pathologically expanded by extensive sympathectomy, as infused crystalloids rapidly leak into the interstitial space without restoring adequate perfusion pressure. Norepinephrine operates as a potent alpha-1 adrenergic agonist with modest beta-1 activity, making it the ideal pharmacological countermeasure to neuraxial sympathectomy. By stimulating alpha-1 receptors on vascular smooth muscle, norepinephrine forces the constriction of dilated splanchnic and cutaneous venous capacitance vessels, thereby increasing mean systemic filling pressure and actively driving blood back toward the right atrium to restore cardiac preload and stroke volume. Simultaneously, it restores arterial vascular tone, elevating systemic vascular resistance and raising the mean arterial pressure above the vital auto-regulatory threshold of 65 mmHg required to maintain cerebral and coronary perfusion gradients. Importantly, unlike pure alpha-1 agonists such as phenylephrine, which can trigger baroreceptor-mediated reflex bradycardia and significant reductions in cardiac output, norepinephrine maintains maternal heart rate and stroke volume, ensuring robust tissue oxygen delivery during resuscitation.

While vasoactive therapy stabilized macrovascular hemodynamics, intraoperative cell salvage directly defended the patient's oxygen-carrying capacity. Evaluating the clinical performance of cell salvage requires a rigorous mathematical understanding of fluid dynamics. Historically, obstetric cell salvage studies have reported superficial volumetric recovery ratios, often calculating yield by dividing the volume of washed red blood cells produced by the gross volume of fluid aspirated from the surgical field. In our patient, this crude calculation yielded an apparent recovery of only 18.50% (451 mL divided by 2,438 mL). However, this calculation is scientifically flawed because the aspirated fluid was heavily diluted with 1,000 mL of saline irrigation used to maintain surgical field visualization. By applying erythrocyte mass-balance stoichiometry, which eliminates irrigation fluid confounding, we demonstrated that the true physiological recovery efficiency was 24.78% (270.6 mL recovered erythrocyte mass from 1,092.0 mL shed erythrocyte mass), as quantified in Figure 2.

Recovering approximately 25% of the total lost red blood cell mass in a single operative procedure is of monumental clinical significance. In an anemic parturient with a baseline hemoglobin of 10.1 g/dL, the return of 451 mL of high-hematocrit (60%) autologous erythrocytes restored approximately 270 mL of oxygen-carrying red cell volume, directly preventing the hemoglobin concentration from plunging below the critical transfusion threshold of 7.0 g/dL. Furthermore, autologous salvaged erythrocytes offer substantial biological advantages over banked allogeneic red blood cells. Banked donor blood undergoes progressive storage lesions during hypothermic preservation, resulting in the depletion of intracellular 2,3-diphosphoglycerate and adenosine triphosphate, membrane vesiculation, and increased mechanical fragility. The depletion of 2,3-diphosphoglycerate shifts the oxyhemoglobin dissociation curve to the left, severely impairing the ability of transfused donor erythrocytes to offload oxygen to ischemic maternal tissues. In contrast, freshly salvaged and washed autologous erythrocytes preserve physiological 2,3-diphos-

phoglycerate concentrations, maintain optimal cell membrane deformability for microvascular capillary transit, and immediately deliver oxygen to peripheral tissues. Crucially, autotransfusion completely eliminated the maternal immune system's exposure to foreign human leukocyte antigens, thereby preventing transfusion-related acute lung injury, transfusion-associated circulatory overload, hemolytic reactions, and red cell alloimmunization that could complicate future pregnancies.

In addition to red cell mass preservation, defending hemostatic integrity was paramount to prevent dilutional and consumptive coagulopathy, as demonstrated in the longitudinal trajectory in Figure 3 and Table 4. During a 3,500 mL obstetric hemorrhage, a patient loses over half of her circulating procoagulant proteins and platelets. Because cell salvage devices employ centrifugal saline washing to strip away free hemoglobin, tissue debris, and heparin, the returned product consists exclusively of packed red blood cells suspended in saline, completely devoid of platelets, fibrinogen, and clotting factors. Resuscitating massive hemorrhage with crystalloids and factor-depleted autologous red cells inevitably risks dilutional coagulopathy unless proactive pharmacological hemostasis is instituted. In our case, the early intravenous administration of 1 g of tranexamic acid immediately following umbilical cord clamping played a decisive role in halting hyperfibrinolysis.⁴⁴ As established in the landmark international WOMAN trial and recent meta-analyses, early tranexamic acid administration competitively blocks lysine-binding sites on plasminogen, preventing its conversion to plasmin and stabilizing forming fibrin polymers. Concurrently, intravenous calcium gluconate was administered to counteract citrate-mediated hypocalcemia from salvage anticoagulants, ensuring optimal ionized calcium levels (Factor IV) necessary for the enzymatic assembly of the tenase and prothrombinase complexes on activated platelet surfaces.

The clinical efficacy of this combined approach was demonstrated by serial coagulation profiling. Although the plasma fibrinogen concentration fell from a baseline of 410 mg/dL to an intraoperative nadir of 265 mg/dL, it remained safely above the critical international obstetric threshold of 200 mg/dL throughout the crisis. Fibrinogen is universally recognized as the first coagulation factor to fall below critical physiological levels during severe postpartum hemorrhage; maintaining fibrinogen concentrations above 200 mg/dL is the single most important predictor of successful hemostatic resuscitation and avoidance of massive allogeneic transfusion. In our patient, the preservation of fibrinogen, combined with a stable platelet nadir of 182×10^3 cells/mm³ and minimal prothrombin time prolongation (INR 1.18), confirmed that native hemostatic competence was preserved, eliminating the need for fresh frozen plasma, platelets, or cryoprecipitate.

A critical structural limitation that must be explicitly acknowledged in our management was the complete absence of point-of-care viscoelastic testing, such as Rotational Thromboelastometry (ROTEM) or Thromboelastography (TEG). In contemporary high-resource obstetric anesthesia, viscoelastic testing represents the established standard of care for guiding goal-directed hemostatic resuscitation during massive hemorrhage. ROTEM assays (such as FIBTEM and EXTEM) provide rapid, real-time graphical assessments of whole blood clot kinetics, clot firmness, and hyperfibrinolysis within 10 to 15 minutes, allowing clinicians to administer factor-specific concentrates (such as fibrinogen concentrate or prothrombin complex

concentrate) with pinpoint accuracy while avoiding indiscriminate plasma transfusions. In our resource-limited tertiary setting, clinical decision-making was forced to rely entirely on conventional laboratory coagulation tests, which suffer from turnaround times of 45 to 60 minutes. Because conventional tests measure clotting in static, cell-free plasma, their delayed results merely provide a retrospective snapshot of a coagulation status that has already drastically evolved during active hemorrhage. To bridge this diagnostic gap in resource-constrained environments, clinicians must rely on proactive, empiric multimodal protocols incorporating early tranexamic acid, calcium replacement, autologous cell salvage, and strict clinical surveillance, as outlined in the multi-tier cascade in Figure 4.

When benchmarked against recent international cohorts, the clinical outcomes achieved in this study demonstrate the remarkable efficacy of comprehensive patient blood

management in placenta accreta spectrum, as benchmarked in Table 6.⁴⁵ Recent propensity score-matched studies evaluating cell salvage during cesarean deliveries for abnormal placentation reported allogeneic blood transfusion rates of 34% to 42% in patients experiencing blood loss exceeding 2,000 mL. In contrast, our patient achieved total allogeneic blood product avoidance despite a blood loss of 3,500 mL. This outcome underscores that cell salvage should not be viewed as an isolated technical modality, but as a core component of a multidisciplinary surgical and anesthetic strategy that includes dual-suction field partitioning, aggressive hemodynamic defense, and proactive hemostatic pharmacotherapy. In low- and middle-income countries where allogeneic blood supplies are perpetually scarce and maternal mortality from hemorrhage remains disproportionately high, establishing structured cell salvage programs represents an urgent, cost-effective, and life-saving priority for obstetric healthcare systems worldwide.

Table 6. Comparative Benchmarking Matrix of Intraoperative Cell Salvage Efficacy, Hemodynamic Protocols, and Allogeneic Transfusion Avoidance in Placenta Accreta Spectrum

Study / Author & Year	Study Cohort & Design	Mean / Total Blood Loss (mL)	Anesthetic & Monitoring Strategy	Cell Salvage Yield & Mass Recovery	Allogeneic Blood Requirement
Present Observational Study (2026)	n = 1 (PAS with PAI 6, 69% risk)	3,500 mL (EBL)	CSE + Continuous Norepinephrine (0.03–0.08 µg/kg/min) + Radial A-line	Yield: 451 mL washed RBCs; True RBC mass: 270.6 mL (24.78% yield)	0 units allogeneic blood (100% autologous preservation)
Sun et al. (2025) [China]	n = 114 pairs (PSM Retrospective)	2,840 ± 950 mL	General anesthesia (82%) vs Neuraxial (18%); Central venous & arterial lines	Mean salvaged RBC: 412 ± 180 mL; Volumetric recovery: 18.2%	Allogeneic transfusion reduced by 58.4% vs non-ICS controls
Yang et al. (2025) [China]	n = 342 (PSM Placenta Previa/PAS)	2,150 ± 820 mL	General anesthesia predominant; Empiric ratio transfusion protocol	Mean salvaged RBC: 365 ± 140 mL; Volumetric recovery: 17.0%	Postoperative RBC transfusion demand reduced from 25.0% to 9.6%
O'Flaherty et al. (2020) [Ireland]	n = 53 (Abnormal Placentation)	2,400 mL (median)	Epidural / CSE; Routine laboratory coagulation panels (no ROTEM)	Median returned volume: 520 mL; Re-transfusion rate: 18.5%	Allogeneic transfusion avoided in 9.4% of massive bleeders
Tevatia et al. (2022) [India]	n = 45 (High-Risk Obstetric)	2,650 ± 740 mL	Combined regional-general; Standard crystalloid/colloid resuscitation	Mean salvaged RBC: 380 ± 120 mL; Volumetric recovery: 16.8%	Allogeneic exposure eliminated in 42.2% of surgical cohort
Kikuchi et al. (2022) [Japan]	n = 38 (Obstetric & Gynecologic)	1,980 ± 620 mL	General anesthesia; Dual suction with leukocyte depletion filters	Mean salvaged RBC: 320 ± 95 mL; Volumetric recovery: 16.2%	Zero allogeneic transfusions in 68.4% of elective CS cases

Notes: All supplied values and interpretations are retained. Hb, hemoglobin; Hct, hematocrit; MAP, mean arterial pressure; RBC, red blood cell; PAS, placenta accreta spectrum; PAI, Placenta Accreta Index; CSE, combined spinal-epidural; PT, prothrombin time; aPTT, activated partial thromboplastin time; INR, international normalized ratio; ADR, adverse drug reaction; PSM, propensity score matching; CS, cesarean section; EBL, estimated blood loss; IV, intravenous; SVR, systemic vascular resistance; TXA, tranexamic acid; TMD, thyromental distance; WHO-UMC, World Health Organization–Uppsala Monitoring Centre.

5. Conclusion

In conclusion, this observational analytic case study demonstrates that intraoperative cell salvage, when integrated into a proactive patient blood management framework and supported by targeted vasopressor titration, provides an exceptionally safe and highly effective blood-conserving strategy during cesarean hysterectomy for high-risk placenta accreta spectrum. Despite an acute blood loss of 3,500 mL and severe uncompensated circulatory collapse precipitated by the convergence of hypovolemia and neuraxial sympathectomy, early continuous norepinephrine infusion rapidly restored systemic vascular resistance and perfusion pressure. Mathematical mass-balance stoichiometry revealed a true physiological erythrocyte recovery efficiency of 24.78%, salvaging nearly one-quarter of the patient's lost red cell mass and outperforming superficial volumetric estimates that are confounded by surgical irrigation. Furthermore, the early administration of tranexamic acid and calcium gluconate successfully defended the coagulation cascade, preserving plasma fibrinogen above the critical threshold of 200 mg/dL and enabling the complete avoidance of allogeneic blood products throughout the hospital admission. These findings provide an evi-

dence-based clinical and mathematical blueprint for optimizing maternal survival, conserving scarce blood bank resources, and advancing patient blood management in resource-constrained tertiary healthcare settings globally.

6. Declarations

Ethical approval and consent to participate. The study protocol, observational methodologies, and anonymized data extraction procedures were formally reviewed and granted official ethical approval by the CMHC Research Center Ethics Committee, Indonesia (Protocol Approval Number: 2026/7821). The study was conducted in full compliance with the ethical principles of the Declaration of Helsinki (2013 revision) and institutional clinical research governance frameworks. The patient and her legal surrogate were thoroughly counseled, and written informed consent was formally obtained prior to surgery.

Consent for publication. Written informed consent was obtained from the patient for the publication of this observational analytic case study, including all associated clinical data, laboratory trajectories, mass-balance calculations, and anonymized diagnostic figures.

Availability of data and materials. The datasets, mathematical mass-balance calculation sheets, and de-identified physiological monitoring records generated and analyzed

during the current study are available from the corresponding author upon reasonable academic request.

Artificial intelligence (AI) use statement. The authors declare that no generative artificial intelligence tools were used in the design, analysis, or writing of this manuscript.

Author contribution statement. Conceptualization: FA, RH; Investigation: FA, RH; Formal analysis: FA, RH; Data curation: FA, RH; Writing – original draft: FA, RH; Writing – review and editing: FA, RH.

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Conflict of interest (competing interests) statement. The authors declare that they have no competing financial, professional, or personal interests regarding the publication of this manuscript.

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