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Optimizing Patient Blood Management: Successful Intraoperative Cell Salvage During Cesarean Hysterectomy for Placenta Accreta Spectrum - A Case Report

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A B S T R A C T

Introduction: Placenta accreta spectrum disorders represent a critical maternal health concern with a high risk of massive obstetric hemorrhage, which conventionally necessitates substantial allogeneic blood transfusion. Intraoperative cell salvage serves as a highly efficient autotransfusion alternative within modern patient blood management frameworks. **Case presentation:** A 37-year-old female (Gravida 4, Para 1) at 37-38 weeks of gestation presented with total placenta previa and a Placenta Accreta Index score of 6, correlating to a 69% probability of placenta accreta. A transperitoneal profunda cesarean section with subsequent hysterectomy was planned. A combined spinal-epidural anesthesia technique was utilized, justified by favorable airway metrics and supported by a proactive massive transfusion protocol. Surgical estimated blood loss was 3,500 mL. An intraoperative cell salvage device processed 2,438 mL of shed fluid, which included 1,000 mL of surgical irrigation. This yielded 451 mL of washed packed red blood cells that were successfully reinfused. The patient's hemodynamics were stabilized using a continuous norepinephrine infusion. The patient received zero allogeneic blood products throughout her admission. Hemoglobin levels were maintained from 10.1 g/dL preoperatively to 9.2 g/dL at discharge. Postoperative coagulation profiles remained stable. The patient was discharged on postoperative day 5 without complications. **Conclusion:** The application of intraoperative cell salvage in major obstetric surgery is demonstrably safe and clinically beneficial. This technology provides a resource-optimized alternative to allogeneic transfusion.

1. Introduction

Placenta accreta spectrum encompasses a range of severe abnormalities associated with placental implantation, characterized primarily by the direct adherence of chorionic villi to the myometrium due to a partial or complete absence of the decidua basalis. Under normal physiological conditions, the decidua basalis acts as a critical interface, providing a highly regulated plane of cleavage that facilitates the safe separation and expulsion of the placenta following the delivery of the fetus. However, in cases of placenta accreta spectrum (PAS), this regulatory layer is structurally compromised or entirely deficient. This deficiency is most frequently localized over areas of prior uterine trauma, particularly the focal scarring generated by previous cesarean sections or significant uterine curettage.[1] Driven by the invasive properties of the extravillous trophoblasts, the placental villi penetrate beyond the functional layer of the endometrium, anchoring directly into the underlying

myometrial muscle fibers, or in more severe iterations of the spectrum, invading deep into the myometrium (increta) or completely penetrating the uterine serosa and adjacent pelvic organs (percreta).[2]

The epidemiological trajectory of this spectrum globally has demonstrated a continuous upward trend, which is inextricably linked to the rising rates of surgical deliveries and advanced maternal age.[3] Over the past several decades, the global incidence of primary and repeat cesarean deliveries has surged, fundamentally altering the baseline risk profile of the modern obstetric population. Each subsequent hysterotomy incrementally degrades the integrity of the lower uterine segment, creating an increasingly hospitable environment for abnormal trophoblastic invasion during subsequent pregnancies. When this rising surgical rate is compounded by the demographic shift toward delayed childbearing—which carries independent risks for abnormal placentation and diminished uterine vascular compliance—the result is a synergistic exacerbation of PAS incidence.[3] Consequently, the presence of abnormal placentation dramatically elevates maternal morbidity and mortality metrics, transforming standard obstetric deliveries into highly complex surgical interventions that carry profound physiological risks. Anticipating a PAS delivery requires moving beyond standard obstetric protocols and transitioning toward a multidisciplinary surgical approach, often requiring the concerted expertise of maternal-fetal medicine specialists, advanced obstetric anesthesiologists, urologists, and vascular surgeons to navigate the anticipated catastrophic complications.[4]

Among the most critical perioperative complications is severe, rapid-onset hemorrhage, which frequently necessitates immediate and high-volume blood transfusions to prevent hypovolemic shock, dilutional coagulopathy, and end-organ ischemia. The fundamental etiology of this hemorrhage stems from the surgical attempt to separate a placenta that lacks a physiological cleavage plane. Because the maternal spiral arteries have undergone extensive physiological remodeling to become high-capacitance, low-resistance vessels, any traumatic avulsion of the placenta results in an unrestricted, high-pressure arterial flow directly into the uterine cavity and surgical field.[5] Unlike normal postpartum hemorrhage, where the myometrium can contract to tamponade bleeding vessels, the myometrium in PAS is often deeply infiltrated and dysfunctional. Consequently, patients can exsanguinate their entire circulating blood volume in a matter of minutes. This acute loss of red cell mass and plasma precipitates a severe hypoxic state, while aggressive crystalloid resuscitation inadvertently dilutes the remaining clotting factors and platelets, pushing the patient rapidly toward the lethal triad of acidosis, hypothermia, and irreversible coagulopathy.

While allogeneic blood transfusion remains a cornerstone in the management of major intraoperative bleeding, it is accompanied by inherent and sometimes severe risks. The empirical reliance on massive transfusion protocols involving uncrossmatched or emergency-release blood products exposes the vulnerable maternal physiological state to a myriad of potential hazards. Potential complications include ABO blood incompatibility, transfusion-related acute lung injury, transfusion-associated circulatory overload, and the transmission of blood-borne pathogens. Transfusion-related acute lung injury (TRALI), characterized by sudden-onset non-cardiogenic pulmonary edema, is a particularly devastating immunologic response triggered by donor leukocyte antibodies reacting with the recipient's pulmonary endothelium.[6] Similarly, transfusion-associated circulatory overload (TACO) poses a severe risk in obstetric patients who already possess an expanded plasma volume and may struggle to accommodate rapid, high-volume fluid and blood resuscitation, leading to acute hydrostatic heart failure.

Furthermore, donor blood continues to be a finite and often severely constrained resource. The global blood supply chain is frequently subjected to critical shortages, seasonal fluctuations in donation rates, and complex logistical hurdles involving strict temperature controls and limited product shelf-life.[7] In massive obstetric hemorrhage, a single patient may deplete a significant portion of a regional hospital's blood bank reserves, jeopardizing the care of other trauma or surgical patients. Beyond supply chain limitations, the ethical and sociological dimensions of transfusion

medicine must be navigated. Certain patient populations may also actively decline allogeneic transfusions based on personal beliefs, requiring clinicians to employ viable alternatives to maintain hemodynamic stability.[8]

To address these profound logistical, physiological, and ethical challenges, intraoperative cell salvage has emerged as a crucial technological component within modern patient blood management frameworks. Patient blood management (PBM) represents an evidence-based, multidisciplinary paradigm shift away from the reflexive use of donor blood, focusing instead on optimizing a patient's own red cell mass, minimizing iatrogenic blood loss, and optimizing physiological tolerance to anemia. Intraoperative cell salvage (ICS) operates precisely at the intersection of these principles. This technology facilitates the continuous aspiration, anticoagulation, centrifugal washing, and rapid reinfusion of the patient's own erythrocytes lost during the surgical procedure. Through a highly automated mechanical process, shed blood is immediately drawn from the surgical field into a sterile reservoir, where it is instantly mixed with a precise ratio of heparinized saline to prevent clotting.[9] Once a sufficient volume is collected, the fluid is routed into a centrifuge bowl. Through variations in specific gravity, the dense erythrocytes are driven to the periphery of the bowl, while the lighter plasma, free hemoglobin, activated clotting factors, inflammatory cytokines, and surgical debris are isolated and discarded as waste.

The resulting product is a highly concentrated suspension of washed autologous packed red blood cells, which can be safely reinfused into the patient's central or peripheral venous circulation.[10] The primary physiological benefits include the immediate availability of biochemically compatible blood, the preservation of normal erythrocyte morphology, and the total elimination of immunologic and infectious complications associated with donor blood products. Unlike banked allogeneic red blood cells, which undergo progressive biochemical degradation during storage—known as the storage lesion—salvaged autologous erythrocytes maintain optimal levels of 2,3-diphosphoglycerate (2,3-DPG) and adenosine triphosphate (ATP). This ensures that the salvaged cells possess superior deformability for microvascular perfusion and an optimal oxygen dissociation curve, facilitating immediate and efficient oxygen offloading to ischemic maternal tissues. Furthermore, by returning the patient's own antigens, the systemic inflammatory response frequently associated with the human leukocyte antigens found in donor blood is completely circumvented.

The aim of this study is to thoroughly evaluate the clinical efficacy, specific hemodynamic management strategies, and pathophysiological outcomes associated with the use of intraoperative cell salvage during a cesarean hysterectomy in a patient presenting with a 69% probability of placenta accreta. While the broader literature increasingly supports the safety of ICS in obstetrics, granular analyses regarding the true physiological yield of these devices during massive, uncompensated hemorrhagic shock remain sparse. Specifically, the primary aim of this manuscript is to critically analyze the exact red blood cell mass recovered via ICS and its direct impact on postoperative hematological stability without the reliance on allogeneic transfusion. The novelty of this study lies in its rigorous, mathematics-based methodological re-evaluation of ICS yield—utilizing a precise mass-balance calculation of shed erythrocyte volume rather than superficial fluid recovery metrics. Furthermore, this study contributes highly granular, resource-optimized patient blood management data from a developing healthcare infrastructure setting, adding valuable clinical evidence to a globally debated topic. By documenting the successful management of a predictable catastrophic hemorrhage in a clinical environment entirely devoid of advanced viscoelastic point-of-care coagulation testing, this report provides an indispensable, highly translatable blueprint for optimizing maternal survival and blood conservation in resource-constrained medical environments worldwide.

2. Case Presentation

Ethical considerations

Ethical protocol adherence and patient autonomy are paramount in the dissemination of clinical case reports. In accordance with the ethical standards established by the Declaration of Helsinki and the

prevailing institutional guidelines for human clinical reporting, comprehensive informed consent was formally obtained from the patient prior to the surgical intervention, encompassing both the high-risk procedure and the subsequent publication of this case report. The patient was thoroughly counseled regarding the academic utilization of her intraoperative hemodynamics, hematological mass-balance data, and clinical outcomes. To ensure strict patient confidentiality and protect personal privacy, all potentially identifiable demographic information has been systematically de-identified and anonymized throughout the manuscript. As this study constitutes a retrospective narrative of a single clinical encounter rather than a prospective experimental trial, formal evaluation by the local Institutional Review Board (IRB) was deemed exempt by institutional policy, contingent upon the rigorous maintenance of medical ethics and patient anonymity.

Preoperative assessment and patient blood management optimization

A 37-year-old female patient (Gravida 4, Para 1) at 37-38 weeks of gestation with an obstetric history notable for two prior early first-trimester miscarriages and one prior viable cesarean delivery in 2013 presented for a scheduled transperitoneal profunda cesarean section and planned definitive management via total abdominal hysterectomy. The patient's clinical presentation was asymptomatic, with a negative review of systems for vaginal spotting, cardiopulmonary distress, or infectious etiologies. A critical component of Patient Blood Management (PBM) is the preoperative optimization of red cell mass. Laboratory investigations upon admission revealed a mild preoperative anemia, with a hemoglobin concentration of 10.1 g/dL and a hematocrit of 31.2%. Within an idealized PBM framework, the identification of maternal anemia necessitates proactive treatment with intravenous iron or recombinant erythropoietin to maximize physiological reserves prior to a surgery associated with massive hemorrhage. However, due to the patient's late presentation to our tertiary center and the imminent necessity of the surgical intervention at 38 weeks gestation, the temporal window for effective pharmacological erythropoiesis was closed. Consequently, we proceeded with a high-risk surgical intervention in a patient with a suboptimal baseline oxygen-carrying capacity. Baseline coagulation studies were within normal physiological limits, with a robust platelet count of 294,000 cells/mm^3 and a fibrinogen level of 410 mg/dL. Advanced obstetric ultrasonography confirmed total placenta previa covering the internal cervical os, accompanied by pathognomonic signs of abnormal invasion: grade III placental lacunae, bridging veins, and a myometrial thickness of less than 1 mm. These imaging parameters generated a Placenta Accreta Index score of 6, correlating directly to a 69% statistical probability of placenta accreta (Table 1).

Intraoperative management

Following comprehensive informed consent, the patient was transferred to the operating theater, where bilateral large-bore intravenous access (16-gauge and 18-gauge) was established, and a massive transfusion protocol was proactively activated. The blood bank crossmatched and reserved 2 units of whole blood, 4 units of packed red blood cells, 600 cc of thrombocyte concentrate, and 600 cc of fresh frozen plasma. Continuous invasive arterial blood pressure monitoring was instituted via a radial arterial line prior to anesthetic induction. The anesthetic management utilized a combined spinal-epidural technique, utilizing 0.25% ropivacaine and 1 mg of preservative-free morphine. We acknowledge that the selection of a neuraxial technique—specifically one incorporating a rapid-onset spinal component—for a planned cesarean hysterectomy in a patient with a high probability of Placenta Accreta Spectrum (PAS) represents a profound deviation from conventional obstetric anesthesiology guidelines. Standard protocols heavily favor either a slowly titrated continuous epidural to mitigate sudden sympathectomy or general anesthesia (GA) to ensure immediate, secure airway control and superior hemodynamic stability during anticipated catastrophic hemorrhage. The decision to employ regional anesthesia over general anesthesia was firmly justified by the patient's strong preference to remain awake for the delivery, combined with a highly favorable airway

assessment (Mallampati classification of 2, thyromental distance of 7 cm, unrestricted cervical mobility). To mitigate the extreme risks associated with this approach, a comprehensive airway backup plan was established; a videolaryngoscope, supraglottic airway devices, and induction agents (propofol and rocuronium) were drawn and immediately available (Table 2).

Table 1. Preoperative Assessment and Patient Blood Management

PATIENT DEMOGRAPHICS & CLINICAL HISTORY	
Patient Profile	37-year-old female, 37-38 weeks of gestation.
Obstetric History	Gravida 4, Para 1; history notable for two prior early first-trimester miscarriages and one prior viable cesarean delivery in 2013.
Clinical Presentation	Asymptomatic; negative review of systems for vaginal spotting, cardiopulmonary distress, or infectious etiologies.
Planned Intervention	Scheduled transperitoneal profunda cesarean section with planned definitive management via total abdominal hysterectomy.
HEMATOLOGY & PBM OPTIMIZATION	
Preoperative Hemoglobin (Hb)	**10.1 g/dL** (indicative of mild preoperative anemia).
Preoperative Hematocrit (Hct)	**31.2%**.
Baseline Coagulation Studies	Within normal physiological limits; Platelet count of 294,000 cells/mm³ and fibrinogen level of 410 mg/dL.
PBM Optimization Status	Temporal window for effective pharmacological erythropoiesis was closed due to late presentation and imminent surgical necessity. Patient proceeded to surgery with suboptimal baseline oxygen-carrying capacity.
IMAGING DIAGNOSTICS & RISK STRATIFICATION	
Imaging Modalities	Advanced obstetric ultrasonography.
Placental Position	Total placenta previa covering the internal cervical os.
Signs of Abnormal Invasion	Pathognomonic signs present: grade III placental lacunae, bridging veins, and a myometrial thickness of less than 1 mm.
Risk Scoring	**Placenta Accreta Index score of 6**.
Statistical Probability	Correlates directly to a **69% probability** of placenta accreta.

The physiological consequences of this anesthetic choice became rapidly apparent. Upon delivery of the fetus, attempts to manage the highly vascularized placental bed resulted in immediate, profuse hemorrhage. The surgical estimated blood loss was 3,500 mL. The rapid exsanguination, compounded by the dense sympathetic efferent blockade induced by the intrathecal ropivacaine, obliterated the patient's compensatory mechanisms. The inability to mount a sympathetically driven vasoconstrictive or tachycardic

response to the acute hypovolemia precipitated a predictable and severe hemodynamic collapse, marked by a nadir mean arterial pressure of 55 mmHg. This acute hypotensive episode, exacerbated by the sympathectomy from the neuraxial block, was aggressively managed with rapid crystalloid infusion and a continuous intravenous norepinephrine infusion titrated between 0.03 and 0.08 mcg/kg/min to restore vascular tone.

Table 2. Intraoperative Management

PRE-INDUCTION PREPARATION & MONITORING	
Vascular Access	Bilateral large-bore intravenous access (16-gauge and 18-gauge) was established.
Hemodynamic Monitoring	Continuous invasive arterial blood pressure monitoring was instituted via a radial arterial line prior to anesthetic induction.
Massive Transfusion Protocol	Proactively activated; blood bank crossmatched and reserved 2 units of whole blood, 4 units of packed red blood cells, 600 cc of thrombocyte concentrate, and 600 cc of fresh frozen plasma.
ANESTHETIC MANAGEMENT & AIRWAY	
Anesthetic Technique	Combined spinal-epidural technique utilizing 0.25% ropivacaine and 1 mg of preservative-free morphine.
Airway Assessment & Justification	Regional anesthesia chosen due to patient preference and a highly favorable airway assessment (Mallampati classification of 2, thyromental distance of 7 cm, unrestricted cervical mobility).
Airway Backup Plan	A videolaryngoscope, supraglottic airway devices, and induction agents (propofol and rocuronium) were drawn and immediately available.
HEMODYNAMIC SEQUELAE & RESUSCITATION	
Surgical Event	Attempts to manage the highly vascularized placental bed resulted in immediate, profuse hemorrhage.
Hemodynamic Collapse	Severe hemodynamic collapse marked by a **nadir mean arterial pressure of 55 mmHg** .
Vasoactive Resuscitation	Aggressively managed with rapid crystalloid infusion and a continuous intravenous norepinephrine infusion titrated between 0.03 and 0.08 mcg/kg/min to restore vascular tone. .
FLUID MASS-BALANCE & CELL SALVAGE (ICS)	
Estimated Blood Loss (EBL)	**3,500 mL** calculated using multimodal assessment (gravimetric weighing, wall-suction, ICS volume). .
Dual-Suction Technique	Primary, non-salvage wall suction catheter utilized to evacuate approximately 800 mL of amniotic fluid **[cite: 60]**. Laparotomy sponges absorbed an estimated 1,262 mL of whole blood.
ICS Processing & Yield	The cell saver aspirated 2,438 mL of fluid (including 1,000 mL of surgical irrigation). The system successfully produced **451 mL** of washed autologous packed red blood cells.
PHARMACOLOGICAL ADJUNCTS	
Antifibrinolytic Therapy	A prophylactic 1-gram intravenous dose of tranexamic acid (TXA) was administered over 10 minutes immediately following umbilical cord clamping.
Electrolyte Management	Intravenous calcium gluconate was administered to counteract potential citrate toxicity from the cell saver anticoagulant and support the coagulation cascade.

Hematological management

To systematically account for the 3,500 mL estimated blood loss and correct omissions in the intraoperative narrative regarding fluid dynamics, a rigorous mass-balance approach was utilized. The mitigation of massive hemorrhage relied heavily on an

intraoperative cell salvage device. The total estimated blood loss (EBL) of 3,500 mL was calculated using a multimodal assessment: gravimetric weighing of laparotomy sponges, measurement of fluid in a primary wall-suction canister, and the volume processed by the ICS system. To prevent contamination of the ICS reservoir with amniotic fluid, a strict dual-suction technique was employed. Prior to and immediately following hysterotomy, a primary, non-salvage wall suction catheter was utilized to evacuate amniotic fluid, capturing approximately 800 mL of fluid. Concurrently, laparotomy sponges absorbed an estimated 1,262 mL of whole blood.

The cell saver device aspirated a total collected volume of 2,438 mL of fluid from the surgical field, which included approximately 1,000 mL of 0.9% sodium chloride used for surgical irrigation. Following automated centrifugation and saline-washing phases, the system successfully produced 451 mL of high-hematocrit, washed autologous packed red blood cells. Regarding pharmacological adjuncts, early administration of tranexamic acid (TXA) is the gold standard for antifibrinolytic therapy in obstetric hemorrhage. While initially reported solely as a postoperative intervention, the administration of TXA was initiated intraoperatively. Immediately following umbilical cord clamping and the onset of profound hemorrhage, a prophylactic 1-gram intravenous dose of TXA was administered over 10 minutes to inhibit the conversion of plasminogen to plasmin. This therapy was subsequently continued in the postoperative period with tranexamic acid 1 gram administered three times daily to stabilize formed clots. Because of this intervention, the patient received zero allogeneic blood products throughout the entire intraoperative and postoperative period.

3. Discussion

The pathophysiology of placenta accreta spectrum (PAS) disorders represents a profound disruption of normal placentation, primarily driven by the defective decidualization of the maternal endometrium. Under normal circumstances, the decidua basalis provides a highly specialized, immunologically privileged, and structurally friable interface that regulates trophoblastic invasion and facilitates the clean separation of the placenta post-delivery. However, in patients with a history of uterine trauma, the decidual layer is compromised over the surgical scar, and consequently, the chorionic villi anchor directly into the myometrium. This unchecked infiltration fundamentally alters the architecture of the lower uterine segment.[11] This structural abnormality fundamentally prevents the normal physiological mechanism of placental separation following fetal delivery. The interface transitions from a physiological cleavage plane to a rigid, fibrotic fusion between fetal and maternal tissues. Attempts to extract the placenta inevitably result in the avulsion of the heavily remodeled, high-flow maternal spiral arteries, leading to catastrophic and intractable hemorrhage. The spiral arteries in a late-term pregnancy lack the muscular tunica media necessary for robust vasoconstriction; thus, when torn, they present an unrestricted, high-capacitance conduit for rapid maternal exsanguination (Figure 1).

Managing this hemorrhage under regional anesthesia presents distinct pathophysiological challenges. As clearly demonstrated in this case, the utilization of a Combined Spinal-Epidural (CSE) technique must be critically analyzed not merely as a clinical success story, but as a critical learning point highlighting the severe limitations of neuraxial blockade in PAS.[12] The physiological response to acute hemorrhage typically involves a massive surge in endogenous catecholamines, which act upon alpha-adrenergic receptors to induce systemic vasoconstriction, and beta-adrenergic receptors to increase inotropy and chronotropy. However, the administration of neuraxial ropivacaine blocks sympathetic efferent pathways, resulting in profound vasodilation and an inability to mount a compensatory tachycardic response to acute hypovolemia.[13]

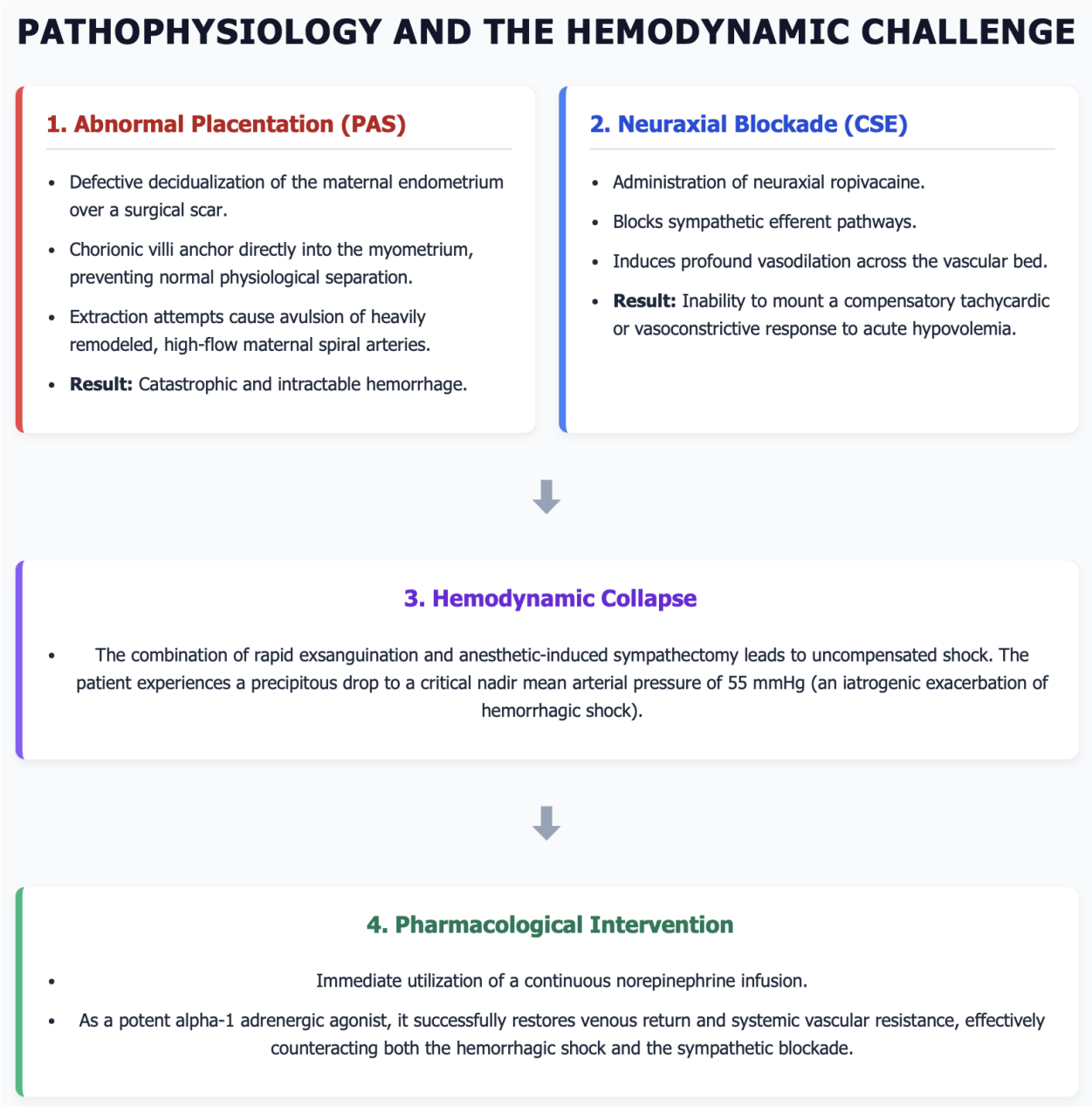

Figure 1. Pathophysiology of PAS and hemodynamic challenge.

When the sympathetic chain, specifically the cardioaccelerator fibers (T1-T4) and the splanchnic vascular beds (T5-L2), are subjected to dense local anesthetic blockade, the patient's autonomic nervous system is effectively rendered blind to the catastrophic volume loss occurring in the surgical field.[14] Consequently, the precipitous drop to a critical nadir mean arterial pressure of 55 mmHg in our patient was a predictable, iatrogenic exacerbation of hemorrhagic shock. The regional anesthetic obliterated the very compensatory mechanisms required to survive rapid blood loss, shifting the hemodynamic state from compensated to uncompensated hemorrhagic shock in a matter of minutes.

This necessitated immediate and targeted pharmacological intervention. Standard fluid resuscitation alone is vastly insufficient when the vascular container has been pathologically expanded by sympathectomy. The utilization of a continuous norepinephrine infusion was vital; as a potent alpha-1

adrenergic agonist, it restored venous return and systemic vascular resistance, counteracting both the hemorrhagic shock and the anesthetic-induced sympathectomy. By stimulating peripheral alpha-1 receptors, norepinephrine forces the constriction of venous capacitance vessels, thereby increasing the mean systemic filling pressure and actively driving blood back to the right atrium to sustain cardiac output. Concurrently, it increases arterial tone to maintain a perfusion pressure gradient across vital end-organs, specifically the cerebral and coronary vasculature. For future cases with a high Placenta Accreta Index, slowly titrated epidural anesthesia (which allows for a gradual onset of sympathectomy and fluid loading) or general anesthesia must remain the unequivocally preferred modalities to ensure maternal safety during massive fluid shifts. General anesthesia, in particular, provides a secure airway, allows for the use of amnestic and analgesic agents with minimal vasodilatory profiles, and preserves the intrinsic sympathetic response to surgical stress and hemorrhage.[15]

The integration of intraoperative cell salvage directly addresses the hematological pathophysiology resulting from the surgical hemorrhage. By actively collecting shed blood, washing it of impurities, and reinfusing the concentrated erythrocytes, the clinical team can aggressively defend the patient's oxygen-carrying capacity. However, evaluating the efficacy of the ICS system requires rigorous mathematical contextualization. Historically, the efficiency of cell salvage has often been reported using a rudimentary volume-to-volume ratio.[16] Previous analyses calculated a superficial recovery yield of 18.5%. This figure is derived by dividing the 451 mL of packed red blood cells produced by the 2,438 mL of total fluid aspirated. This metric is clinically and scientifically flawed, as the 2,438 mL of aspirated fluid was not pure whole blood; it was heavily diluted with approximately 1,000 mL of surgical irrigation fluid and residual amniotic fluid. Calculating efficiency based on total fluid aspirated penalizes the technology for performing in a surgical environment that necessitates copious irrigation to maintain visualization. A more accurate metric relies on calculating the estimated red blood cell (RBC) mass lost versus the RBC mass recovered. This mass-balance approach isolates the specific physiological variable of interest—the erythrocyte—and eliminates the confounding variables of saline, plasma, and amniotic fluid. Given an estimated blood loss of 3,500 mL and a preoperative maternal hematocrit of 31.2%, the total shed RBC volume can be calculated. To find the precise volume of lost erythrocytes, we apply the following calculation: Shed RBC mass= 3500 mL x0.312 = 1092 mL. This calculation reveals that of the 3,500 mL of fluid considered blood loss, exactly 1,092 mL consisted of functional red blood cells. The ICS device returned 451 mL of washed, highly concentrated erythrocytes. The centrifugation process within the cell saver device aggressively packs the red cells, stripping away plasma and irrigation fluid. Assuming an average hematocrit of 60% for cell-saver processed blood (which is heavily concentrated during centrifugation), the recovered RBC mass is: Recovered RBC Mass = 451 mL x 0.60 = 270.6 mL. With the actual red cell mass of both the shed blood and the reinfused product quantified, the true efficiency of the autotransfusion process can be determined. Therefore, the true physiological recovery efficiency is: Recovery Efficiency = 270.6 mL / 1092 mL = 24.78%.

Recovering approximately 25% of the total lost red cell mass is highly significant. This represents a quarter of the patient's critical oxygen delivery infrastructure that was seamlessly returned to her intravascular space. When factoring in the patient's baseline anemia, this targeted replacement of oxygen-carrying capacity successfully averted critical tissue hypoxia without exposing the maternal immune system to human leukocyte antigens or the inherent risks of allogeneic transfusion, such as transfusion-associated circulatory overload or acute lung injury. By utilizing her own erythrocytes, the patient bypassed the storage lesion associated with banked blood, ensuring that the reinfused cells possessed optimal levels of 2,3-diphosphoglycerate for immediate oxygen offloading at the tissue level, while simultaneously preserving limited community blood bank resources.

The mitigation of red cell loss is only one facet of managing massive obstetric hemorrhage; defending the coagulation cascade is equally critical. A 3,500 mL obstetric hemorrhage represents an acute loss of

approximately 50-60% of total maternal blood volume.[16] Resuscitating a deficit of this magnitude primarily with crystalloids and washed, factor-depleted autologous erythrocytes fundamentally risks the precipitation of severe dilutional coagulopathy. This phenomenon occurs when the volume of the intravascular space is restored, but the concentration of essential procoagulant proteins, fibrinogen, and circulating platelets falls below the critical thresholds required to maintain hemostasis. Cell salvage provides packed red blood cells devoid of platelets and coagulation factors. Because the washing process of the ICS device intentionally strips away plasma to remove activated clotting factors and debris, the reinfused product contributes absolutely nothing to clot formation.

Given these physiological parameters, it is a remarkable clinical outcome that postoperative laboratory trends confirmed safe physiological stability without the administration of fresh frozen plasma (FFP), cryoprecipitate, or platelet concentrates.[17] Postoperative Day 1 laboratories demonstrated a platelet count of 195,000 cells/mm^3 and an aPTT of 25.10 seconds. Most notably, the fibrinogen level decreased from a preoperative baseline of 410 mg/dL to 285 mg/dL immediately postoperatively. Fibrinogen is typically the first coagulation factor to reach critically low levels during massive hemorrhage, functioning as the primary substrate for definitive clot formation. While this reduction in fibrinogen clearly reflects consumption and dilution, remaining above the critical obstetric threshold of 200 mg/dL indicates that the patient's intrinsic hepatic reserves, combined with careful surgical hemostasis, were sufficient.[18]

Furthermore, the pharmacological management played a synergistic role in maintaining this delicate hemostatic balance. The early intraoperative administration of tranexamic acid played a critical role in inhibiting uncontrolled fibrinolysis, and intravenous calcium gluconate was administered to counteract potential citrate toxicity from the cell saver anticoagulant and support the coagulation cascade. Tranexamic acid, a synthetic lysine analogue, competitively binds to plasminogen, preventing its activation into plasmin and thus halting the premature degradation of forming fibrin clots. Calcium, functioning as Factor IV in the coagulation cascade, is an absolute requirement for the assembly of coagulation factor complexes on phospholipid surfaces.[19]

However, a critical limitation must be explicitly acknowledged regarding our management of massive hemorrhage within a developing healthcare infrastructure. The gold standard for guiding complex coagulation resuscitation is the use of Point-of-Care (POC) viscoelastic testing, such as Rotational Thromboelastometry (ROTEM) or Thromboelastography (TEG). These advanced modalities analyze the viscoelastic properties of whole blood as it clots under low shear stress. These modalities provide rapid, real-time visual assessment of clot formation, strength, and lysis, allowing for targeted, factor-specific therapy. Instead of blindly administering ratios of FFP and platelets, clinicians can pinpoint exact deficiencies—whether a lack of fibrinogen, weak platelet contribution, or hyperfibrinolysis—and administer specific concentrates accordingly.[20]

Due to resource constraints in our setting, ROTEM/TEG was unavailable. This technological void forces clinicians back into traditional, empiric paradigms of care. Our clinical team was forced to rely entirely on standard laboratory coagulation panels (PT, aPTT, Fibrinogen), which possess turnaround times of 45-60 minutes. These standard tests are performed on plasma, completely ignoring the complex cellular interactions of in vivo clotting, and their delayed results merely provide a historical snapshot of the patient's coagulation status from an hour prior. During active, massive obstetric exsanguination, such delays render standard tests practically obsolete for acute decision-making. The patient's physiological state will have drastically evolved by the time the results are populated in the electronic medical record. Future protocols in resource-limited settings must focus heavily on empirical, ratio-driven massive transfusion protocols and early antifibrinolytic therapy to bridge the technological gap caused by the absence of viscoelastic monitoring.

4. Conclusion

Obstetric hemorrhage secondary to abnormal placentation remains a leading etiology of severe maternal morbidity. As cesarean section rates globally continue to climb, the incidence of Placenta Accreta Spectrum will inextricably follow, demanding that obstetric, surgical, and anesthetic teams continuously refine their protocols for catastrophic hemorrhage. Cesarean hysterectomy for placenta accreta spectrum necessitates careful anesthetic planning, aggressive hemodynamic control with vasoactive infusions, and proactive volume management. The physiological perturbations associated with regional anesthesia in the face of rapid exsanguination highlight the absolute necessity for immediate sympathomimetic support, such as continuous norepinephrine infusions, to override iatrogenic vasodilation and sustain critical perfusion.

The implementation of intraoperative cell salvage in parturients facing severe bleeding risks provides a biologically superior mechanism for oxygen-carrying capacity replacement. By rigorously evaluating the recovered red cell mass rather than mere fluid volumes, it is evident that autotransfusion is highly efficient, returning a significant percentage of lost erythrocytes directly to the patient. This technology aligns perfectly with modern Patient Blood Management frameworks, respecting both physiological needs and resource constraints. As demonstrated in this case, autotransfusion successfully averted the need for allogeneic blood products, maintained postoperative hematological stability, and facilitated the patient's successful discharge without long-term sequelae. It represents a cornerstone intervention in bridging the gap between life-threatening obstetric pathology and the optimization of safe, high-quality, and resource-conscious maternal healthcare.

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