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CASE REPORT

When Bleeding Stops but Organs Fail: Fatal Multiple Organ Dysfunction Syndrome Following Massive Atonic Postpartum Haemorrhage in a Young Primipara — A Case Report

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ABSTRACT

Background: Postpartum haemorrhage from uterine atony can precipitate profound haemorrhagic shock within minutes, and in resource-constrained referral systems the interval between the onset of bleeding and definitive source control is frequently the decisive determinant of survival.

Objective: To document, in granular day-by-day detail, the evolution of fatal multiple organ dysfunction syndrome following massive atonic postpartum haemorrhage in a young woman with no antecedent organ disease, and to translate that trajectory into transferable lessons for haemorrhage systems in resource-limited settings.

Case presentation: We describe a 28-year-old primipara referred four hours after a vacuum-assisted vaginal delivery of twins complicated by severe pre-eclampsia. On arrival she was stuporous, hypotensive (70/68 mmHg), tachycardic (142 beats/min) and oliguric, with active vaginal bleeding and a haemoglobin of 2.4 g/dL. Resuscitation and haemorrhage control proceeded in parallel: high-flow oxygen, dual large-bore access, crystalloid and colloid loading, tranexamic acid, balanced blood-component transfusion, bimanual compression, intubation and emergency laparotomy. A flaccid, bluish, atonic uterus and a fourth-degree perineal tear were identified; subtotal hysterectomy with sphincteroplasty and perineorrhaphy achieved haemostasis. Despite massive transfusion and intensive care, prolonged hypoperfusion evolved into ischaemic hepatitis, severe coagulopathy, stage III acute kidney injury requiring haemodialysis, hospital-acquired pneumonia with drug-resistant sepsis, acute pulmonary oedema and upper gastrointestinal bleeding. Progressive multiple organ dysfunction syndrome culminated in asystolic cardiac arrest on postoperative day 15.

Conclusion: This case illustrates a principle easily overlooked: definitive surgical haemostasis does not reverse organ injury already committed during the pre-operative shock interval. Objective cumulative blood-loss measurement, immediate haemorrhage-bundle and massive-transfusion protocol activation, explicitly documented uterotonic therapy, rapid balanced resuscitation and structured post-haemostasis organ surveillance are the interventions most likely to interrupt the lethal trajectory.

1. Introduction

Postpartum haemorrhage (PPH) remains the single largest direct cause of maternal death worldwide, and its burden falls disproportionately on low- and middle-income countries, where delays in recognition, transport, blood availability and definitive intervention convert a survivable emergency into a fatal one.[1] Contemporary population data show that PPH complicates a substantial and rising share of deliveries; in the United States its rate increased from 2.7% to 4.3% over two decades, mirroring an international trend of increasing severe PPH and transfusion.[2,3] Uterine atony—failure of the myometrium to contract and mechanically compress the placental-bed vessels after delivery—is responsible for the majority of primary PPH and is the phenotype most likely to produce abrupt, high-volume loss, with a well-characterised cluster of antecedent risk factors including uterine overdistension, hypertensive disease and operative delivery.[4,5] When contraction fails, exsanguination can outpace even a well-organised resuscitation team.

The contemporary approach to severe PPH is built on speed and parallelism rather than sequential escalation. A large multicentre trial of a first-response bundle combining calibrated, objective blood-loss measurement with immediate uterotonics, tranexamic acid, uterine massage, fluids and escalation demonstrated that early detection and treatment substantially reduce severe bleeding, establishing objective measurement and bundled response as the cornerstones of modern care.[6] Tranexamic acid, in

particular, reduces death from bleeding when given early, and randomised and pooled evidence confirms its value across operative and vaginal delivery, including in anaemic women who are at especially high risk, while pharmacokinetic work continues to refine optimal dosing.[7-11] When pharmacological and compressive measures fail, a graded menu of interventions exists before hysterectomy: intrauterine balloon tamponade and haemostatic devices, uterine compression sutures, uterine artery embolization and stepwise devascularisation, although the comparative evidence for these devices remains limited.[12] Emergency peripartum hysterectomy remains the definitive procedure of last resort, associated with substantial morbidity but frequently life-saving when conservative options are exhausted or inappropriate.[13]

It is worth being precise about terminology. Primary PPH is conventionally defined as blood loss of 500 mL or more after vaginal birth, or 1,000 mL or more after caesarean section, within the first 24 hours, although the clinically meaningful threshold is better anchored to any bleeding that produces or threatens haemodynamic compromise.[2] Multiple organ dysfunction syndrome (MODS), in turn, denotes altered organ function in an acutely ill patient such that homeostasis cannot be maintained without intervention, and is best conceived as a continuum of severity across two or more organ systems. The unifying physiological currency linking these entities is oxygen debt: the cumulative deficit between oxygen demand and delivery that accrues during hypoperfusion. The magnitude and duration of that debt, more than any single vital sign, predict which patients recover and which descend into progressive organ failure, which is precisely why the pre-hospital and referral interval is so decisive where blood and definitive care are hours rather than minutes away.[1]

What is less frequently emphasised in clinical teaching is what happens after the bleeding stops. Prolonged hypovolaemia and the resulting oxygen debt initiate a biological cascade that can continue to unfold for days after surgical haemostasis. In obstetric intensive-care cohorts, women who survive the initial haemorrhage frequently progress to severe maternal outcomes—organ dysfunction, near-miss events and death—driven by the shock state rather than by continued bleeding.[1] In this sense the haemorrhage is only the inciting event; the shock it produces is a systemic disease with its own momentum. Recognising that momentum, and interrupting it early, may matter as much as the ligature or the hysterectomy itself.

The novelty of this report lies in its illustration of the gap between successful haemostasis and successful resuscitation. Most published PPH case reports end at the point of bleeding control; comparatively few follow the patient through the full arc of post-haemostasis organ failure and use that trajectory to interrogate where the decisive delays occurred. The aim of this study is therefore twofold: first, to document in granular, day-by-day detail the evolution of fatal MODS after massive atonic PPH in a young woman with no antecedent organ disease; and second, to translate that trajectory into concrete, transferable lessons for haemorrhage systems in resource-limited settings—objective blood-loss quantification, bundle and massive-transfusion protocol activation, explicit uterotonic documentation, and continuous organ surveillance after the bleeding has stopped.[6]

2. Case Presentation

Patient information and obstetric history

A 28-year-old primiparous woman was referred from a peripheral maternity hospital to the obstetric emergency unit of Dr. M. Djamil Central General Hospital, Padang, Indonesia, on 3 April 2026. Approximately four hours earlier she had delivered twins by vacuum-assisted vaginal delivery, performed on the background of severe pre-eclampsia. Heavy, fresh vaginal bleeding had continued for roughly two hours before transfer and had soaked four underpads. Prior to referral she had received oxygen through a non-rebreathing mask at 15 L/min, two peripheral intravenous lines, 500 mL of Ringer's lactate, 500 mL of a gelatin-based colloid, and urinary catheterisation. Her antenatal attendance had been limited, and she had no documented history of chronic hypertension, diabetes mellitus, or cardiac, pulmonary, renal or hepatic disease. The combination of a twin gestation, operative vaginal delivery, severe pre-eclampsia and probable intrapartum magnesium sulphate exposure together constituted a formidable cluster of risk factors for atonic haemorrhage.

Clinical findings on admission

On arrival the patient was critically ill and stuporous. Her blood pressure was 70/68 mmHg, heart rate 142 beats/min, respiratory rate 38 breaths/min, temperature 36.7°C, and oxygen saturation 98% while receiving 15 L/min of oxygen through a non-rebreathing mask. The conjunctivae were pale, the extremities were cold, and capillary refill exceeded two seconds—a constellation indicating decompensated hypovolaemic shock with critically impaired peripheral perfusion. Active vaginal bleeding was ongoing. Urine output was approximately 10 mL/hour and the urine was yellow-brown, consistent with established renal hypoperfusion. Abdominal distension, tenderness, rebound tenderness and muscular guarding were absent, and both uterine height and contractility were difficult to assess in the presence of ongoing bleeding and haemodynamic instability.

Diagnostic assessment

Bedside ultrasonography demonstrated an anteflexed uterus with suspected retained placental tissue measuring 7.32 × 3.44 cm and no free intraperitoneal fluid, arguing against occult intra-abdominal haemorrhage and localising the source to the genital tract. Initial laboratory testing revealed profound

anaemia, mild thrombocytopenia, hyponatraemia, an elevated serum creatinine and rising aminotransferases—a pattern already suggestive of early multi-system involvement, as detailed in Table 1. The working diagnosis was haemorrhagic shock due to primary PPH driven predominantly by uterine atony, with suspected retained placental tissue, a fourth-degree perineal tear and severe acute blood-loss anaemia. The admission haemoglobin of 2.4 g/dL is among the lowest compatible with life and, taken together with the stupor, the extreme tachycardia and the oliguria, indicated that oxygen delivery had already fallen far below the threshold required to sustain aerobic metabolism in the vital organs.

Table 1. Selected laboratory findings at admission and six hours postoperatively, with reference ranges.

Parameter	Admission	6 h postoperative	Reference range
Haemoglobin (g/dL)	2.4	7.7	12–16
Haematocrit (%)	8	24	28–40
Platelets (×10^3/µL)	130	115	150–400
Creatinine (mg/dL)	1.7	1.6	0.6–1.2
AST (U/L)	156	1,461	<32
ALT (U/L)	64	934	<31
Albumin (g/dL)	Not available	1.9	3.8–5.0
Prothrombin time (s)	9.4	26.9	9.83–13.23
APTT (s)	25	>180	23.42–30.82
Random glucose (mg/dL)	57	19	70–200

Notes: AST: aspartate aminotransferase; ALT: alanine aminotransferase; APTT: activated partial thromboplastin time.

The paired admission and early-postoperative values detailed in Table 1 capture the paradox at the centre of this case. Transfusion successfully corrected the haemoglobin from 2.4 to 7.7 g/dL within hours, yet over the same interval the aspartate aminotransferase rose almost ten-fold (from 156 to 1,461 U/L), the prothrombin time nearly trebled (from 9.4 to 26.9 s) and the activated partial thromboplastin time became unrecordable (>180 s)—evidence that the organs had already begun to fail even as the circulating red-cell mass was being restored. As shown in Figure 1, these paired trajectories, plotted against their reference ranges, make visually explicit that haematological repletion and biochemical deterioration were occurring simultaneously.

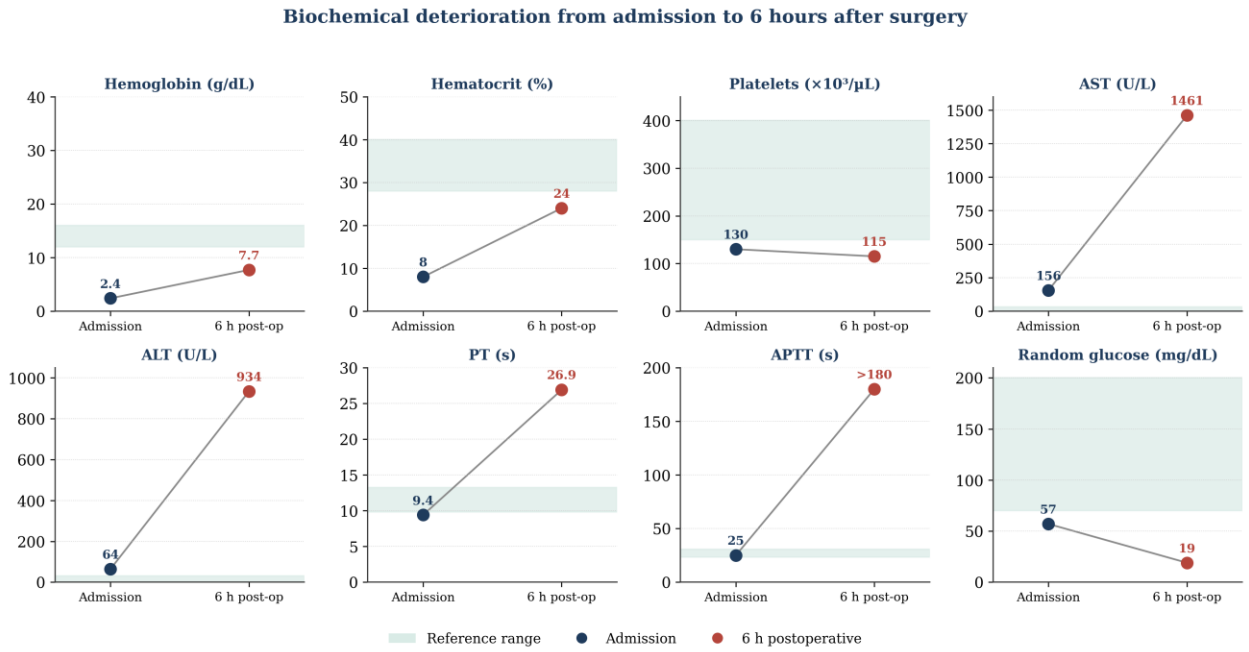

Figure 1. Paired biochemical values at admission (navy) and six hours after surgery (red) for eight key parameters, each plotted against its reference range (shaded band). Haematological repletion (haemoglobin 2.4→7.7 g/dL; haematocrit 8→24%) coincides with marked hepatic (AST 156→1,461 U/L; ALT 64→934 U/L), coagulation (PT 9.4→26.9 s; APTT 25→>180 s) and metabolic (glucose 57→19 mg/dL) deterioration, illustrating that correction of anaemia did not equate to reversal of shock-related organ injury.

Therapeutic intervention

Resuscitation and haemorrhage control were initiated simultaneously rather than sequentially. The patient received 1,500 mL of Ringer's lactate through the first intravenous line and 1,000 mL of gelatin solution together with packed red blood cells through the second. Tranexamic acid 1 g and vitamin K 10 mg were administered intravenously, and external bimanual uterine compression was applied while the team mobilised for theatre. An anaesthesia consultation was obtained, the patient was intubated to secure the airway and support oxygenation in the setting of stupor and extreme tachypnoea, and emergency laparotomy was undertaken without delay.

At laparotomy the uterus was flaccid and mildly bluish, findings consistent with severe uterine atony and early Couvelaire-type infiltration. Because of ongoing haemorrhagic shock and the imperative for rapid definitive source control, a subtotal hysterectomy was performed rather than attempting time-consuming uterus-conserving manoeuvres. Subsequent examination of the genital tract revealed a fourth-degree perineal tear; the urogynaecology team repaired the rectal mucosa, the internal and external anal sphincters, the perineal musculature and the skin. Intraoperative blood loss recorded after arrival in the operating room was 1,000 mL, although the cumulative pre-referral loss was never objectively quantified—a critical gap discussed below. During surgery the patient received seven units of packed red blood cells, four units of fresh frozen plasma and six units of platelet concentrate. The full sequence of resuscitation, source-control and supportive interventions, together with their explicit rationale, is detailed in Table 2.

Table 2. Summary of resuscitation, source-control and supportive interventions with clinical rationale.

Domain	Intervention delivered	Rationale / guideline principle
Airway / breathing	Non-rebreathing O_2 15 L/min; intubation and mechanical ventilation	Maximise oxygen delivery; protect the airway in a stuporous, tachypnoeic patient
Circulation / access	Two large-bore lines; 1,500 mL Ringer's lactate; 1,000 mL gelatin colloid	Restore intravascular volume while awaiting and delivering blood components
Haemostatic pharmacology	Tranexamic acid 1 g IV; vitamin K 10 mg IV	Antifibrinolysis within the effective early window; correct dilutional/consumptive coagulopathy
Blood-component therapy	7 units PRBC, 4 units FFP, 6 units platelets (intra-operative)	Balanced component resuscitation for massive transfusion
Mechanical control	External bimanual uterine compression	Temporising tamponade of the atonic uterus during transfer to theatre
Definitive source control	Emergency laparotomy → subtotal hysterectomy	Rapid haemostasis in an unstable patient when conservative steps are inappropriate
Genital-tract repair	Sphincteroplasty and perineorrhaphy (fourth-degree tear)	Address the "trauma" component of the bleeding and restore continence
Organ support	ICU care, vasopressors, antibiotics, haemodialysis, serial monitoring	Support failing organ systems during and after the shock interval

Notes: PRBC: packed red blood cells; FFP: fresh frozen plasma; IV: intravenous; ICU: intensive care unit.

Postoperative course and outcome

Postoperatively the patient required intensive care, mechanical ventilation, vasopressor support, broad-spectrum antibiotics, repeated blood-component transfusion and serial laboratory monitoring. Six hours after surgery the markedly elevated aminotransferases (AST 1,461 U/L, ALT 934 U/L) were consistent with ischaemic hepatitis, while the prolonged prothrombin time (26.9 s) and unrecordable activated partial thromboplastin time (>180 s) indicated severe coagulopathy. Profound hypoglycaemia (19 mg/dL) and hypoalbuminaemia (1.9 g/dL) reflected failing hepatic synthetic and gluconeogenic function.

Renal function then deteriorated progressively. The serum creatinine climbed from 1.7 mg/dL at admission to 5.8 mg/dL on postoperative day (POD) 4, 6.7 mg/dL on POD 5 and 7.6 mg/dL on POD 8. The acute kidney injury advanced to stage III with oliguria progressing to anuria, mandating haemodialysis. Thrombocytopenia deepened to a nadir of 50 $\times 10^3/\mu L$ before slowly recovering. As illustrated in Figure 2, the serum creatinine rose relentlessly across the first postoperative week despite organ support, while the platelet count fell in parallel.

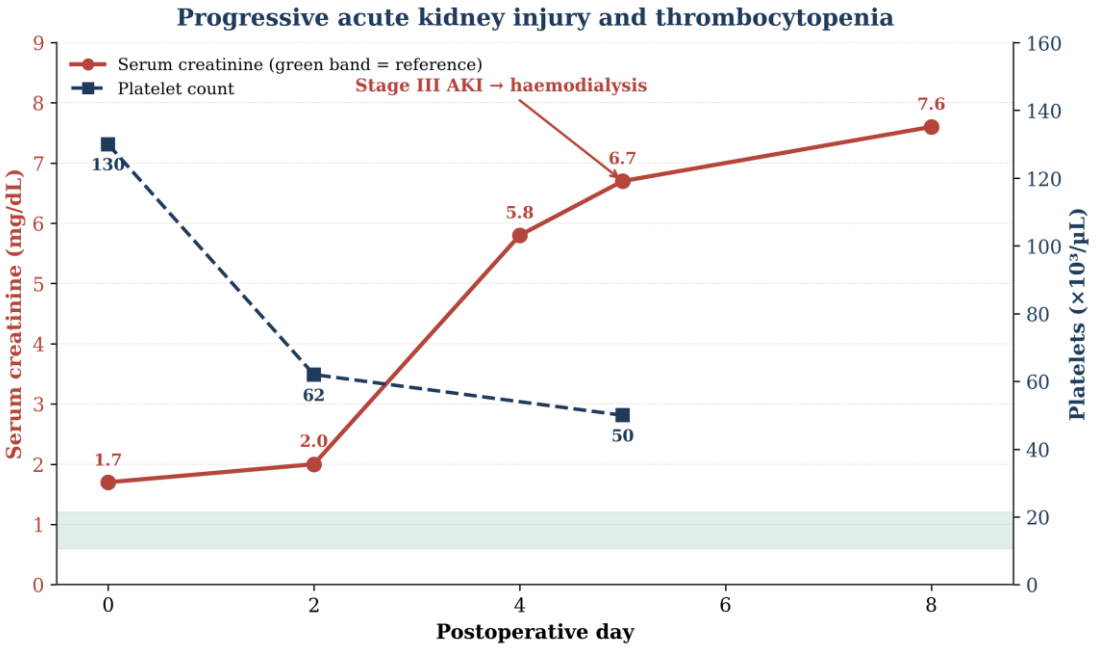

Figure 2. Serial serum creatinine (red, left axis) and platelet count (navy, right axis) across the first eight postoperative days. Creatinine rises progressively from 1.7 to 7.6 mg/dL, corresponding to evolution from prerenal injury to dialysis-dependent stage III acute kidney injury, while platelets fall to a nadir of 50 $\times 10^3$/µL. The shaded band indicates the creatinine reference range.

On POD 9–10 the patient developed dyspnoea, brownish sputum, progressive leukocytosis (28–35 $\times 10^3$/µL), a procalcitonin of 12.5 ng/mL and bilateral pulmonary infiltrates. Hospital-acquired pneumonia, sepsis and acute pulmonary oedema were diagnosed and antimicrobial therapy was escalated. Blood culture subsequently yielded methicillin-resistant*Staphylococcus aureus*, and urine culture grew a carbapenem-resistant *Pseudomonas* species, confirming that the secondary insult was now driven by drug-resistant nosocomial infection superimposed on shock-injured organs. On POD 11–12 melena and suspected haematemesis developed, with a fall in haemoglobin from 8.9 to 4.4 g/dL, consistent with severe upper gastrointestinal bleeding in the setting of critical illness and persistent coagulopathy. As shown in Figure 3, these events form a single temporal map of the organ systems that failed in sequence, and the haemoglobin trajectory across the whole admission, including the secondary gastrointestinal-bleeding nadir, is shown in Figure 4.

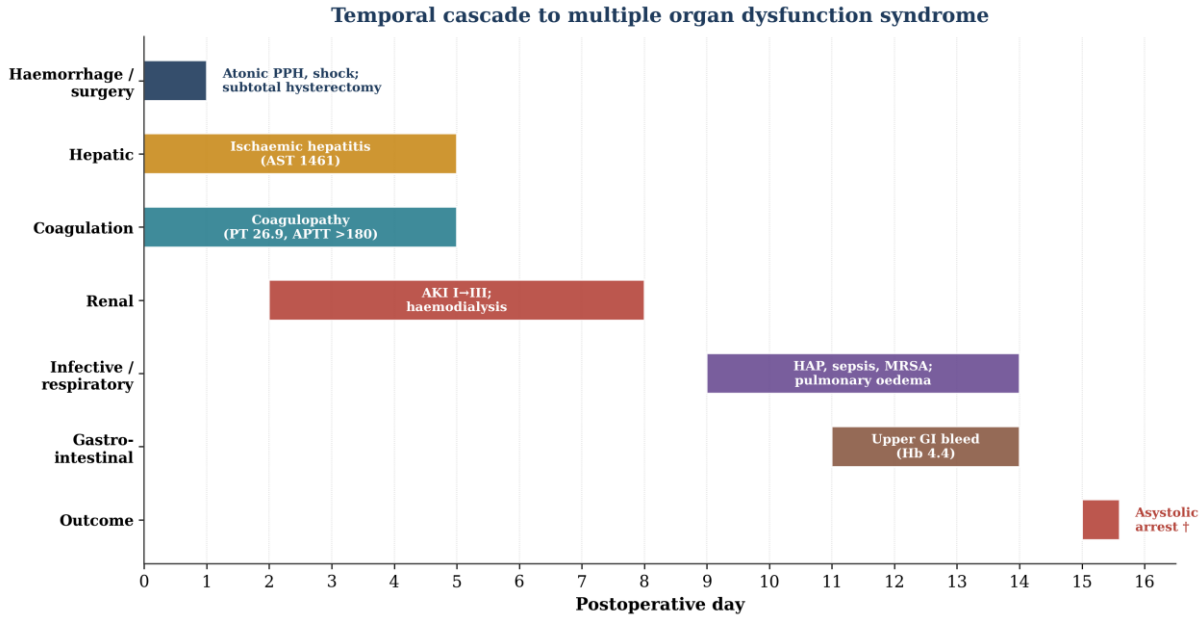

Figure 3. Temporal cascade of organ-system involvement across the 15-day admission. Hepatic and coagulation dysfunction appeared within the first postoperative hours, renal failure evolved over the first week, and infective, respiratory and gastrointestinal complications emerged in the second week, culminating in asystolic cardiac arrest. The figure visualises the sequential, overlapping nature of multiple organ dysfunction syndrome. † denotes death.

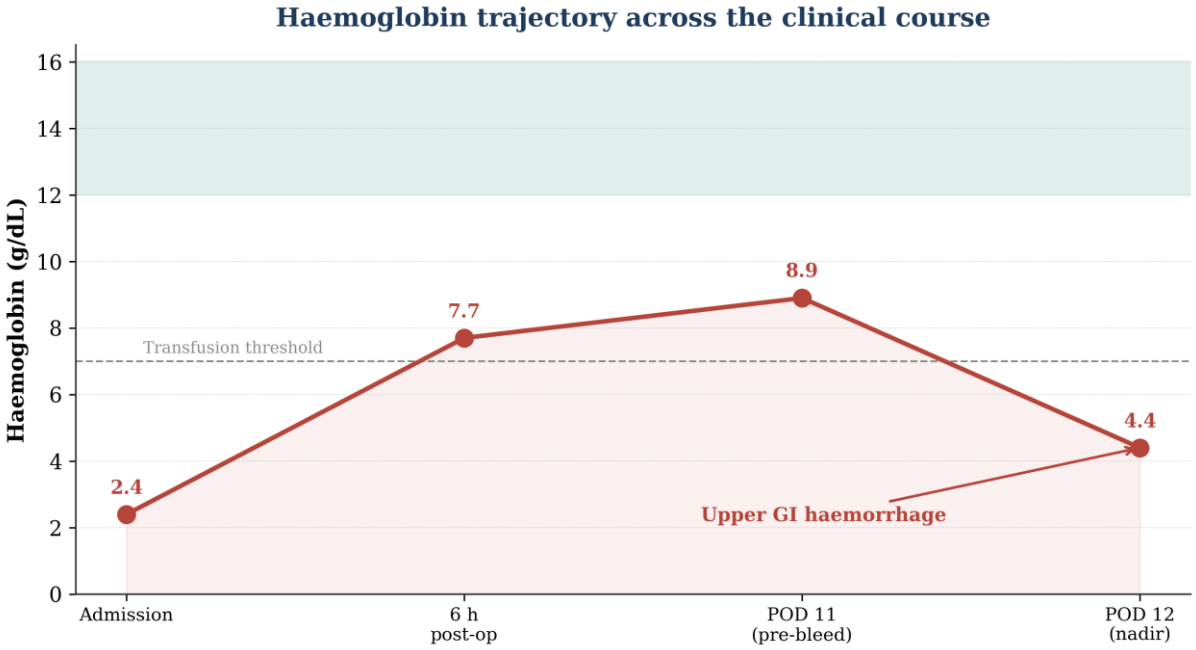

Figure 4. Haemoglobin trajectory across the clinical course, using measured values only. An initial catastrophic nadir of 2.4 g/dL was corrected by transfusion to 7.7 g/dL and 8.9 g/dL, but a second decline to 4.4 g/dL on postoperative day 12 marked the onset of critical-illness upper gastrointestinal bleeding. The dashed line indicates a representative transfusion threshold and the shaded band the normal reference range.

Despite haemodialysis, broad-spectrum antimicrobial therapy, repeated transfusion, continued ventilatory support and vasopressors, the multiple organ dysfunction syndrome progressed inexorably. On POD 15 the patient developed extreme tachycardia, severe desaturation (oxygen saturation 48–54%) and refractory, unrecordable hypotension. In accordance with a previously documented do-not-resuscitate decision reached with the family, cardiopulmonary resuscitation was not performed, and the patient was declared dead at 15:30 following asystolic cardiac arrest. The day-by-day evolution of the postoperative course, aligning the dominant findings, management and interpretation for each interval, is summarised in Table 3.

Table 3. Day-by-day summary of the postoperative clinical course.

Period	Major findings	Management	Interpretation
POD 0–1	Hb 7.7 g/dL; AST/ALT 1,461/934 U/L; PT 26.9 s; APTT >180 s	ICU, ventilation, vasopressors, PRBC/FFP, antibiotics	Ischaemic hepatitis and severe coagulopathy
POD 2–5	Platelets 62→50 $\times 10^3$/µL; creatinine 2.0→6.7 mg/dL	Serial transfusion, renal consultation, dialysis access, haemodialysis	Progressive AKI and thrombocytopenia
POD 6–10	Persistent AKI; leukocytes 28–35 $\times 10^3$/µL; pulmonary infiltrates; procalcitonin 12.5 ng/mL	Haemodialysis, reintubation, broad-spectrum antibiotics	HAP, sepsis, pulmonary oedema
POD 11–14	Melena/haematemesis; Hb nadir 4.4 g/dL; fever; vasopressor dependence	PRBC/FFP, haemostatic therapy, antimicrobial escalation	Upper GI bleeding and worsening MODS
POD 15	Extreme tachycardia; SpO_2 48–54%; unrecordable blood pressure; asystole	Supportive care; DNR applied	Cardiac arrest and death

Notes: POD: postoperative day; Hb: haemoglobin; AKI: acute kidney injury; HAP: hospital-acquired pneumonia; GI: gastrointestinal; MODS: multiple organ dysfunction syndrome; DNR: do not resuscitate.

3. Discussion

Aetiology and severity of the haemorrhage

This patient presented with decompensated haemorrhagic shock arising from primary PPH, and the aetiology was revealingly multifactorial when mapped onto the classic four-T framework. Uterine atony—the dominant cause—supplied the failure of "tone"; the suspected retained placental fragment represented the "tissue" component; the fourth-degree perineal tear contributed "trauma"; and the coagulopathy that emerged during resuscitation added the "thrombin" abnormality. The clinical value of the four-T schema is that it forces parallel rather than serial evaluation: in a patient bleeding this briskly, tone, tissue, trauma and

thrombin must be assessed and addressed almost simultaneously, because fixating on the most obvious cause while another proceeds unchecked wastes the minutes that determine survival. Contemporary risk-factor analyses confirm that severe blood loss requiring surgical intervention and hysterectomy clusters in exactly this kind of patient.[14] In retrospect, the coexistence of at least three of the four mechanisms helps explain why the bleeding was so rapidly decompensating.

The risk-factor profile for atony was, moreover, unusually dense. Twin gestation produces marked uterine overdistension and is among the most consistently reported antecedents of atonic PPH; operative vaginal delivery, severe pre-eclampsia and probable magnesium sulphate exposure each independently impair myometrial contractility or predispose to bleeding.[4,5] Large cohort and cross-sectional analyses of severe PPH repeatedly identify this clustering—multifetal pregnancy, hypertensive disease and instrumental delivery—as the substrate for the most dangerous episodes.[5,14] The admission haemoglobin of 2.4 g/dL, together with stupor, a heart rate of 142 beats/min, hypotension, cold peripheries and oliguria, indicated that oxygen delivery had already collapsed and that a substantial oxygen debt had accumulated before the patient reached definitive care. This pre-operative debt, rather than any intra-operative event, is the single most important determinant of everything that followed, a pattern borne out by obstetric intensive-care cohorts in which severe maternal outcomes track the depth and duration of pre-treatment shock.[1]

The link between severe pre-eclampsia and atonic haemorrhage deserves particular scrutiny, because it is frequently underappreciated. Pre-eclampsia is characterised by a reduced circulating plasma volume and an endothelium primed toward vasoconstriction and microvascular dysfunction; a woman with severe disease therefore enters the peripartum period with a diminished capacity to tolerate acute blood loss and a narrower margin before decompensation. Magnesium sulphate, the standard agent for seizure prophylaxis in severe pre-eclampsia, is itself a smooth-muscle relaxant and can impair myometrial contractility, plausibly contributing to atony in a uterus already overdistended by a twin gestation. The confluence of a contracted intravascular volume, an activated endothelium and pharmacologically blunted uterine tone helps explain why this patient bled so briskly, and it argues for heightened vigilance and pre-emptive uterotonic readiness whenever severe pre-eclampsia, multifetal pregnancy and operative delivery coincide.[4,14]

Initial management and the decision for hysterectomy

Judged against contemporary standards, most of the essential elements of guideline-concordant care were in fact delivered: high-flow oxygen, two intravenous lines, fluid resuscitation, early transfusion, tranexamic acid, urinary catheterisation, bimanual compression, anaesthetic involvement, intubation and emergency surgery. The bundled first-response approach validated in a large multicountry trial—objective blood-loss measurement coupled with immediate uterotonics, tranexamic acid, massage and escalation—captures precisely this philosophy of early, parallel action.[6] The administration of tranexamic acid deserves particular emphasis, because its efficacy is exquisitely time-dependent: pooled individual-patient data show that treatment within three hours of onset reduces death due to bleeding, whereas later administration confers little benefit.[7,8] Its value in anaemic women—directly relevant to a patient presenting with a haemoglobin of 2.4 g/dL—has been examined in a dedicated randomised trial, and pharmacokinetic modelling continues to refine dosing in pregnancy.[9-11]

Two deficiencies nonetheless stand out. First, the documentation of first-line uterotonic therapy was inconsistent. Oxytocin should be recorded explicitly as the initial uterotonic, with additional agents added according to contraindications and response; in a validated first-response bundle, uterotonic administration is an auditable, protocolised step rather than an assumed background activity, and its omission from the record is itself a system failure.[6] Second, and more consequentially, cumulative blood loss was never objectively measured. Visual estimation and the counting of soaked underpads systematically and severely underestimate true loss, whereas calibrated, objective measurement triggers earlier recognition and escalation and is the entry point of the evidence-based bundle.[6] Had the true cumulative loss been quantified at the referring facility, the massive-transfusion pathway might have been activated hours earlier.

The decision to proceed directly to subtotal hysterectomy, bypassing balloon tamponade, compression sutures and arterial embolization, warrants explicit defence. In refractory atony these uterus-conserving measures are ordinarily attempted first, although the comparative evidence for tamponade devices is limited and of low certainty.[12] This patient, however, arrived stuporous, profoundly anaemic, oliguric and in severe shock with active bleeding; in such circumstances the time consumed by sequential conservative attempts is time during which ischaemic organ injury deepens, and proceeding directly to hysterectomy is clinically defensible and often correct.[13] Subtotal (rather than total) hysterectomy is the appropriate operation for rapid haemorrhage control in an unstable patient, since it shortens operative time and reduces the risk of ureteric injury. Systematic worldwide data and national cohort series confirm that emergency peripartum hysterectomy remains an indispensable, life-saving procedure despite its morbidity, with indications dominated by atony and abnormal placentation.[13,15,16]

Massive transfusion and coagulopathy

The patient met every practical definition of massive transfusion, a term that is itself inconsistently defined across the literature but most commonly denotes the transfusion of large red-cell volumes within a short window.[17] Modern practice favours early, balanced blood-component therapy—approximating physiological whole blood through fixed ratios of red cells, plasma and platelets—delivered alongside frequent reassessment, active warming and monitoring of fibrinogen, ionised calcium, acid–base status, lactate and coagulation. There is growing interest in whole-blood-based resuscitation strategies, which reduce the logistical complexity of component ratios and may improve haemostatic efficiency in exsanguinating patients.[18] Fibrinogen replacement is a particular focus, although a large population-based cohort found that its benefit depends critically on early administration.[19] In this case, although substantial quantities of red cells, plasma and platelets were transfused, the exact timing and ratio were not documented as a structured protocol, and fibrinogen, lactate, ionised calcium, temperature and arterial blood-gas values were unavailable—omissions that matter because hypofibrinogenaemia, hypocalcaemia, acidosis and hypothermia constitute the self-reinforcing lethal diamond that perpetuates bleeding.

The coagulopathy in this patient was almost certainly multifactorial—dilutional from resuscitation, consumptive from the bleeding, and exacerbated by hypothermia and the acidosis of shock. Detailed observational work shows that acute obstetric coagulopathy during PPH is driven specifically by hyperfibrinolysis and dysfibrinogenaemia, a mechanism distinct from simple dilution and one that argues for early antifibrinolysis and fibrinogen-directed correction.[20] Fibrinogen is the first coagulation factor to fall to critical levels during major haemorrhage, and its central role in haemorrhage-induced coagulopathy is well characterised.[21] The prothrombin time of 26.9 seconds and unrecordable activated partial thromboplastin time documented six hours postoperatively (Table 1) are the laboratory signature of exactly this process, and the absence of fibrinogen measurement represents a missed opportunity for targeted correction.

A further, easily neglected element of massive transfusion is the metabolic milieu, which the incomplete documentation in this case left largely invisible. Stored blood components are anticoagulated with citrate, which chelates ionised calcium; rapid, high-volume transfusion therefore predictably produces hypocalcaemia, impairing both myocardial contractility and the calcium-dependent steps of the coagulation cascade and thereby compounding the very coagulopathy the transfusion is intended to correct. Potassium, acid–base balance and temperature are perturbed in parallel. Because definitions and triggers for massive transfusion vary so widely between institutions, structured protocols that specify not only component ratios but also the serial measurement and correction of ionised calcium, lactate, temperature and fibrinogen are essential to reliable practice.[17] The absence of these measurements in the present case is therefore not a trivial gap but a genuine blind spot, since each represents a modifiable contributor to ongoing bleeding and organ dysfunction, and each is a parameter that a whole-blood or component-based massive-transfusion pathway is designed to track in real time.[18]

From shock to multiple organ dysfunction syndrome

The most instructive feature of this case is the mechanistic link between the pre-operative shock interval and the cascade of organ failure that followed. Haemorrhagic shock is not merely a deficit of circulating volume but a systemic ischaemia–reperfusion injury, and obstetric intensive-care cohorts confirm that severe maternal outcomes—organ dysfunction, near-miss and death—are concentrated in women who sustain the deepest and most prolonged hypoperfusion.[1] In this framework, the transfusion that raised the haemoglobin to 7.7 g/dL corrected the oxygen-carrying deficit but could not undo the injury already inflicted during the hours of profound hypoperfusion, as the divergence between haematological repletion and biochemical deterioration in Figure 1 makes plain.

The hepatic injury exemplifies this principle. The postoperative surge in aminotransferases to well over a thousand units per litre (Table 1) is the biochemical hallmark of ischaemic (hypoxic) hepatitis, a condition produced by reduced hepatic oxygen delivery during shock and classically associated with high in-hospital mortality when it complicates other low-output states such as cardiogenic shock.[22] The accompanying hypoalbuminaemia and profound hypoglycaemia reflected failure of the liver's synthetic and gluconeogenic functions, and the subsequent drug-resistant sepsis would have compounded the hepatic insult.

The renal trajectory followed the same logic. What began as potentially reversible prerenal azotaemia—the expected consequence of severe hypovolaemia—progressed to established stage III acute kidney injury requiring haemodialysis, as charted in Figure 2. In obstetric critical-care populations, prerenal injury from haemorrhage remains the leading pathway to dialysis, and pregnancy-related acute kidney injury continues to carry substantial morbidity and mortality, particularly where renal replacement therapy is accessed late.[23,24] The relentless rise in creatinine across the first postoperative week signalled that the renal insult sustained during the shock interval had crossed the threshold from reversible to established injury before support could reverse it.

The second-week complications—hospital-acquired pneumonia, drug-resistant sepsis, acute pulmonary

oedema and upper gastrointestinal bleeding—represent the predictable sequelae of prolonged mechanical ventilation, invasive devices, repeated transfusion, hypoalbuminaemia and impaired host defences in a patient whose physiological reserve had already been exhausted, and they mirror the severe-maternal-outcome trajectories described in obstetric intensive-care cohorts.[1] The recovery of methicillin-resistant *Staphylococcus aureus* from blood and carbapenem-resistant*Pseudomonas* from urine underscores two imperatives: culture-directed antimicrobial therapy with careful renal dose adjustment, and disciplined stewardship in a patient in whom every additional nephrotoxic or marrow-suppressive exposure carried disproportionate risk. Critical-illness gastrointestinal bleeding then further reduced oxygen-carrying capacity and aggravated the coagulopathy, producing the secondary haemoglobin nadir of 4.4 g/dL shown in Figure 4. By POD 15 the coexistence of renal failure, respiratory failure, sepsis, pulmonary oedema, recurrent anaemia and vasopressor dependence constituted established MODS, as mapped in Figure 3, and the fatal outcome demonstrated that arresting the haemorrhage had not arrested the disease it had set in motion.

The respiratory failure similarly reflected more than a single mechanism. Acute pulmonary oedema in the peripartum period is frequently multifactorial, combining the hydrostatic burden of large-volume crystalloid, colloid and blood-component resuscitation with the increased pulmonary capillary permeability of sepsis and systemic inflammation and, in a woman with severe pre-eclampsia, a baseline predisposition to fluid extravasation. Transfusion-associated circulatory overload and transfusion-related acute lung injury are recognised hazards of the very massive transfusion that this patient required to survive the initial haemorrhage, and they illustrate the unavoidable therapeutic tension at the heart of resuscitating exsanguinating patients: the products that restore the circulation can themselves injure the lung. The bilateral infiltrates, hypoxaemia and ventilatory dependence documented from the second postoperative week are most coherently understood as the pulmonary manifestation of this convergence, layered upon hospital-acquired pneumonia, and they form one axis of the multi-system failure mapped in Figure 3.

The role of vasopressors and the limits of rescue

The prolonged vasopressor dependence in this patient invites a note of caution. In haemorrhagic shock, vasopressors are a temporising bridge and not a substitute for volume and haemostasis; their early or excessive use in the bleeding patient can worsen tissue perfusion by raising afterload against an underfilled circulation. Once established shock gives way to a distributive, septic physiology, as occurred here in the second week, vasopressor support becomes appropriate and necessary—but by that stage it is supporting organs already injured beyond recovery. The transition of this patient from a haemorrhagic to a septic-distributive vasopressor requirement is itself a marker of the shift from a potentially salvageable to an unsalvageable state, and such transitions are characteristic of the severe maternal outcomes documented in obstetric intensive care.[1]

System-level lessons and the wider context

Beyond the individual physiology, this case is a portrait of a health-system problem. Maternal mortality from PPH is overwhelmingly a phenomenon of low- and middle-income settings, and the modifiable failures cluster at the interfaces of care—recognition at the birth facility, the speed and quality of referral, the availability of blood, and the time to definitive source control.[1,24] The bundled, objective-measurement approach that reduced severe bleeding in a multicountry trial is directly applicable to such settings and offers a concrete template for improvement.[6] In this patient, several system-level opportunities are identifiable in retrospect: cumulative blood loss was never quantified; first-line uterotonic therapy was not clearly documented; the massive-transfusion pathway was not activated as a structured protocol; and the interval from the onset of bleeding to definitive control spanned several hours across two facilities. None of these gaps in isolation was necessarily fatal, but together they lengthened the shock interval that ultimately proved lethal.

It is instructive to translate these observations into the language of quality improvement, because the failures here were not failures of knowledge but of system reliability. Each recommended action—quantifying blood loss, activating a bundle, documenting uterotonics, transfusing in balanced ratios, surveilling organs after haemostasis—is individually well established; what determines outcomes is whether these actions occur consistently, in the right order, and fast enough across the whole chain of care. Bundled, checklist-driven approaches exist precisely because they convert individually known interventions into reliably delivered ones, and their impact is greatest where baseline variability is highest, as in referral networks spanning facilities of differing capability.[6] Viewed through this lens, the present case is best read not as a search for a single missed intervention but as an illustration of how an accumulation of small, individually survivable delays can summate into an unsurvivable oxygen debt.

The coagulopathy also merits framing within the specific obstetric context rather than by simple analogy to trauma. Pregnancy is a physiologically hypercoagulable and hyperfibrinolytic state, and obstetric haemorrhage can evolve rapidly toward a distinct acute coagulopathy characterised by hyperfibrinolysis and dysfibrinogenaemia, particularly when tissue factor is released—as with retained placental tissue and prolonged shock.[20] Fibrinogen, physiologically elevated in late pregnancy, paradoxically becomes an early and sensitive marker of

deterioration, and its role and replacement in haemorrhage-induced coagulopathy are increasingly well defined.[21] The unrecordable activated partial thromboplastin time and markedly prolonged prothrombin time seen six hours postoperatively (Table 1) are consistent with a consumptive process superimposed on dilution, and they reinforce the argument that fibrinogen and viscoelastic monitoring should be embedded in obstetric massive-transfusion pathways rather than borrowed piecemeal from trauma practice.

The logistics of blood supply and inter-facility referral deserve emphasis as a distinct and often rate-limiting link in this chain. A patient who exsanguinates faster than compatible blood can be cross-matched, released and delivered will accrue oxygen debt regardless of how expertly the receiving team performs, and in many resource-limited systems the availability of red cells, plasma, platelets and fibrinogen is the true bottleneck. This patient was referred four hours after the onset of bleeding, having received only limited crystalloid and colloid before transfer, so that the definitive correction of her profound anaemia and coagulopathy could not begin until she reached the tertiary centre. Obstetric intensive-care cohorts from low- and middle-income settings repeatedly show that delayed access to blood products and to renal replacement therapy is associated with worse maternal outcomes, and that strengthening the referral pathway—pre-notification, blood transport, and a receiving team activated before arrival—can compress the interval during which irreversible injury accumulates.[1,24] An objective, cumulative blood-loss figure travelling with the patient would also allow the receiving centre to prepare the appropriate volume of components in advance rather than reactively, shortening still further the time to balanced resuscitation.[6]

Recognising the limits of rescue and the place of comfort-focused care

The eventual do-not-resuscitate decision, reached with the family, is an integral and ethically important part of this narrative rather than a footnote to it. By postoperative day 15 the patient had established, advancing failure of the renal, hepatic, respiratory, haematological and cardiovascular systems despite maximal organ support, a configuration that carries a very high mortality and from which meaningful recovery had become improbable. In such circumstances, honest prognostication, transparent communication with the family and a timely shift toward comfort-focused care are as much a part of good medicine as the earlier heroics of resuscitation and surgery. Recognising the point at which continued escalation no longer serves the patient's interests requires the same rigour and courage as the decision to proceed directly to hysterectomy hours earlier. This case therefore underscores the value of early, structured family communication in the intensive care of catastrophic maternal haemorrhage, so that if the trajectory becomes irreversible, decisions can be made in a considered rather than a crisis-driven manner, and it highlights the importance of building bereavement support and structured team debriefing into the aftermath of a maternal death.

Limitations

Several limitations temper the conclusions that can be drawn from a single case. As a retrospective report, key data were missing—most importantly objective cumulative blood loss, fibrinogen, lactate, ionised calcium, arterial blood-gas values and the precise timing and ratio of blood components—which constrains any definitive attribution of the outcome to specific management steps. The diagnoses of ischaemic hepatitis, sepsis-related organ dysfunction and the several components of MODS are clinical and biochemical rather than histological. A single fatal case cannot establish causation or generalisability, and the counterfactual—whether earlier bundle activation and quantified blood loss would have altered the outcome—cannot be tested. Nonetheless, the day-by-day trajectory documented here is internally coherent and aligns closely with established pathophysiology and with the severe-maternal-outcome literature, and its value lies in illustrating, rather than proving, the central lesson.

Finally, this case has value as a teaching narrative precisely because none of its individual components is exotic. Every clinician who manages obstetric emergencies will recognise the atonic uterus, the falling blood pressure, the race to theatre and the relief of achieving haemostasis; what is less often internalised is the way the clock keeps running afterwards. The sequential, overlapping pattern of organ failure captured in Figure 3—hepatic and coagulation dysfunction within hours, renal failure over the first week, and infective, respiratory and gastrointestinal complications in the second—is the visible expression of an oxygen debt incurred before the first suture was placed. Framing the case in these terms reframes the clinical question from “how do we stop the bleeding?” to the more demanding “how do we shorten the shock interval and then defend every organ that the shock has threatened?”, a question whose answer lies in systems, protocols and surveillance rather than in any single heroic act.[1]

4. Conclusion

Massive atonic postpartum haemorrhage in this young primipara produced profound haemorrhagic shock before definitive treatment could be delivered. Emergency subtotal hysterectomy and repair of the fourth-degree perineal tear controlled the bleeding, but by then prolonged hypoperfusion had already committed the patient to a cascade of ischaemic hepatitis, coagulopathy, dialysis-dependent acute kidney injury, drug-resistant nosocomial sepsis, pulmonary oedema and gastrointestinal haemorrhage, and the resulting multiple organ dysfunction syndrome

ended in fatal cardiac arrest. The overarching lesson is that survival from severe PPH depends not only on eventual surgical haemostasis but on everything that precedes and follows it: early recognition, objective blood-loss measurement, immediate bundle and massive-transfusion protocol activation, explicitly documented uterotonic therapy, rapid balanced resuscitation, decisive source control, and continuous organ surveillance after the bleeding has stopped. Stopping the haemorrhage is necessary but not sufficient; interrupting the shock state it produces, as early as possible, is what saves lives.

For practising clinicians and for the institutions that support them, the actionable message is that the outcome of severe postpartum haemorrhage is decided as much in the minutes and hours around recognition, referral and blood delivery as in the operating theatre. Investing in objective blood-loss measurement, in reliable first-response bundles, in rapid access to balanced blood components including fibrinogen, and in structured post-haemostasis organ surveillance offers a realistic path to preventing deaths that surgery alone cannot avert. This single fatal case cannot prove that any one of these measures would have changed the result, but it illustrates, with unusual clarity, why each of them matters and why they must function together as a system rather than as isolated interventions.

Declarations

Ethics approval and consent to participate

This case report was reviewed and approved by the CMHC Research Center Indonesia (approval reference number 2026/018).

Consent for publication

Written informed consent for publication of this case and any accompanying data was obtained from the patient's next of kin and is documented and available for review by the editor on request.

Competing interests

The authors declare that they have no competing interests.

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Author contributions

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Data availability

The clinical data supporting this report are available from the corresponding author on reasonable request, subject to institutional and ethical restrictions protecting patient confidentiality.

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