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Aspiration Thrombectomy for High-Risk Pulmonary Embolism With Life-Threatening Anemia and Active Uterine Bleeding: A Case Report

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

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Patient: Female, 48-year-old
Final Diagnosis: High-risk pulmonary embolism with severe anaemia and active uterine bleeding from adenomyosis
Symptoms: Transient loss of consciousness • haemodynamic instability (hypotension) • dyspnoea • heavy vaginal bleeding • lower-limb swelling
Clinical Procedure: —
Specialty: Cardiology • Critical Care Medicine • Pulmonology
Objective: Rare coexistence of disease or pathology
Background: High-risk pulmonary embolism (PE) carries significant mortality, and management becomes complex when it coexists with severe anemia and active bleeding, as thrombolysis is then contraindicated. We report the case of a patient with high-risk PE complicated by severe anemia (hemoglobin 41 g/L) and active uterine bleeding from adenomyosis.
Case Report: A 48-year-old woman presented with haemodynamic instability (blood pressure 74/47 mm Hg), critical anemia (hemoglobin 41 g/L), and a 3-year history of irregular vaginal bleeding from adenomyosis. Computed tomography (CT) pulmonary angiography confirmed bilateral PE with right pulmonary artery main-trunk occlusion; echocardiography showed severe pulmonary hypertension, and duplex ultrasound showed extensive left lower-limb deep vein thrombosis. Thrombolysis was contraindicated. Emergency percutaneous aspiration thrombectomy was performed using large-bore catheters (22F, 20F, 16F) with concurrent inferior vena cava filter placement, reducing pulmonary artery pressure from 51/23 to 34/12 mm Hg; the estimated blood loss, quantified from filtered aspirate, was approximately 100 mL. Adjuncts included transfusion of 13 units of packed red cells, dilatation and curettage, gonadotropin-releasing hormone agonist therapy, and titrated anticoagulation. At discharge after a 24-day hospitalization she was hemodynamically stable, and free of dyspnea, with resolved edema and hemoglobin 72 g/L, on oral rivaroxaban.
Conclusions: In this thrombolysis-contraindicated patient, aspiration thrombectomy with inferior vena cava filter placement was a feasible rescue strategy that achieved rapid hemodynamic and angiographic improvement without thrombolysis, with palliative rather than curative intent. As a single case, it cannot establish comparative safety or efficacy, and broader conclusions regarding efficacy cannot be drawn. It illustrates a potentially useful approach in similarly complex scenarios where coordinated multidisciplinary care is essential.
Keywords: Adenomyosis • Anemia • Pulmonary Embolism • Thrombectomy • Uterine Hemorrhage • Vena Cava Filters
Full-text PDF: <https://www.amjcaserep.com/abstract/index/idArt/954225>

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Introduction

High-risk pulmonary embolism (PE), defined by hemodynamic instability or cardiac arrest, carries a short-term mortality exceeding 15% and remains a leading cause of cardiovascular death [1]. The 2019 European Society of Cardiology (ESC) guidelines recommend systemic thrombolysis as first-line reperfusion therapy for high-risk PE [1]. However, thrombolysis is contraindicated when active bleeding is present, creating a critical therapeutic dilemma [1,2].

Adenomyosis is a benign gynecological condition in which endometrial glands and stroma invade the myometrium. It predominantly affects women of reproductive age and is a frequent cause of abnormal uterine bleeding and chronic iron-deficiency anemia [3]. When severe anemia from ongoing uterine bleeding accompanies high-risk PE, a pathophysiological paradox arises: anemia worsens the hemodynamic compromise of right ventricular failure, while anticoagulation or thrombolysis exacerbates the underlying bleeding.

Percutaneous aspiration thrombectomy has emerged as a reperfusion alternative when thrombolysis is contraindicated [4,5]. The EXTRACT-PE trial [6], the FLARE study [7], and the FLASH registry [8] have established the safety and efficacy of catheter-directed mechanical intervention across the spectrum of PE severity. These data, however, are derived predominantly from intermediate-risk populations and do not address the hemodynamically unstable, high-risk patient in whom severe anemia and active bleeding coexist.

The specific knowledge gap addressed here is the rarity of the combined presentation: high-risk PE with a hemoglobin below 50 g/L and active uterine bleeding, in which the anemia and ongoing hemorrhage were themselves the primary determinants of the decision to forgo thrombolysis in favor of mechanical reperfusion. We report such a case, managed with emergency aspiration thrombectomy and inferior vena cava (IVC) filter placement within a multidisciplinary strategy.

Case Report

Written informed consent for publication of this case report and any accompanying images was obtained from the patient and her family. A 48-year-old woman (body mass index 19.92 kg/m²) presented with a 3-year history of irregular heavy vaginal bleeding and a 5-hour history of transient loss of consciousness. She reported dysmenorrhoea requiring oral analgesics. Her obstetric history was G1POA1LO (1 spontaneous abortion at 6 months of gestation). Gynecological ultrasound performed 1 year earlier had demonstrated adenomyosis; the patient had declined treatment and did not attend

follow-up. Her past medical and drug history was otherwise unremarkable.

On arrival at the emergency department, vital signs revealed severe hemodynamic instability: blood pressure 74/47 mm Hg, heart rate 105-110 beats per minute, respiratory rate 28 breaths per minute, oxygen saturation 93% on room air, and temperature 35.0 °C. Examination revealed pale conjunctivae and lips, bilateral diminished breath sounds with moist rales, and left lower-extremity swelling. Gynecological examination demonstrated dark-red blood on the external genitalia. A chronological timeline of key clinical events is provided in **Table 1**.

Emergency complete blood count revealed a critically low hemoglobin of 41 g/L (reference range 110-150 g/L), leukocytosis (white blood cell count $18.75 \times 10^9/L$, neutrophils 92.6%), and a normal platelet count ($138 \times 10^9/L$). D-dimer was 19.46 mg/L fibrinogen equivalent units. B-type natriuretic peptide was 622.04 pg/mL, fibrinogen was 1.48 g/L (hypofibrinogenemia), and procalcitonin was 1.50 ng/mL. Arterial blood gas analysis demonstrated compensated metabolic acidosis (pH 7.39, base excess -9.5 mmol/L) with hyponatremia and hypocalcemia. Serial laboratory parameters are presented in **Table 2**.

Transthoracic echocardiography demonstrated right heart enlargement, moderate-to-severe tricuspid regurgitation (**Figure 1**), severe pulmonary hypertension, and preserved left ventricular ejection fraction (65%). Venous duplex ultrasound confirmed extensive left lower-extremity thrombosis involving the deep veins, the calf intramuscular veins, and the left external iliac vein. CT pulmonary angiography revealed a large filling defect occluding the right pulmonary artery main trunk with absent distal opacification, together with filling defects in the distal left main trunk and multiple segmental branches (**Figure 2**). Gynecological ultrasound confirmed adenomyosis with marked uterine enlargement. The patient was classified as high-risk PE per the 2019 ESC guidelines [1] based on hemodynamic instability, right ventricular dysfunction, and elevated cardiac biomarkers.

Emergency multidisciplinary consultations were obtained from cardiology, intensive care, and respiratory medicine; all teams agreed on the high risk of sudden death and on the need for intensive care unit (ICU) transfer, inferior vena cava (IVC) filter placement, and thrombectomy [9]. Systemic thrombolysis was contraindicated by the combination of active uterine bleeding and severe anemia. Surgical embolectomy was excluded: extracorporeal membrane oxygenation-supported surgery was not available, and the hemodynamic instability made general anaesthesia and cardiopulmonary bypass risky [4]. The patient was transferred to the emergency ICU; resuscitation with intravenous fluids and packed red blood cell transfusion was initiated, and after informed consent was obtained, emergency aspiration thrombectomy and IVC filter insertion were arranged.

Table 1. Chronological timeline of key clinical events.

Day/time	Event
Day 1 (17: 00)	Transient loss of consciousness at home lasting approximately 30 minutes; spontaneous recovery
Day 1 (22: 40)	ED admission: BP 74/47 mm Hg, HR 105-110 bpm, SpO ₂ 93%, Hb 41 g/L. Initial diagnoses: severe anemia, adenomyosis, abnormal uterine bleeding
Day 1 (evening)	Transthoracic echocardiography: right pulmonary artery embolism, right heart enlargement, severe pulmonary hypertension. Venous duplex ultrasound: extensive left lower-extremity thrombosis
Day 2 (01: 00-01: 30)	Multidisciplinary consultations: high risk of sudden death; recommendation for ICU transfer, IVC filter, and thrombectomy
Day 2 (02: 35)	Transfer to emergency ICU
Day 2 (13: 26)	Pre-operative MDT discussion: decision for emergency aspiration thrombectomy and IVC filter
Day 2 (17: 50)	CTPA confirms occlusion of the right pulmonary artery main trunk. Procedure arranged
Day 2 (evening)	Emergency aspiration thrombectomy and IVC filter insertion under local anaesthesia. PA pressure: 51/23 → 34/12 mm Hg
Day 2 (21: 30)	Post-operative anticoagulation initiated: enoxaparin 60 mg SC every 12 hours
Day 3	Hb 65 g/L post-transfusion. Piperacillin-tazobactam started
Day 6	D&C performed; GnRH agonist (leuprolide 3.75 mg SC) initiated
Day 8	Anticoagulation transitioned to oral rivaroxaban 15 mg twice daily
Day 10	Transfer from ICU to emergency department ward. Stable condition
Day 14	Hb 82 g/L (peak)
Day 25	Discharge after 24-day hospitalization. Hb 72 g/L. Stable hemodynamics
Day 29 (4 days post-discharge)	Outpatient follow-up: Hb 63 g/L, WBC $2.77 \times 10^9/L$ (leukopenia)

Abbreviations: BP, blood pressure; bpm, beats per minute; CTPA, CT pulmonary angiography; D&C, dilatation and curettage; ED, emergency department; GnRH, gonadotropin-releasing hormone; Hb, hemoglobin; HR, heart rate; ICU, intensive care unit; IVC, inferior vena cava; MDT, multidisciplinary team; PA, pulmonary artery; SC, subcutaneous; SpO₂, oxygen saturation; WBC, white blood cell count.

Table 2. Serial laboratory parameters during hospitalization.

Day	Hb (g/L)	WBC ($\times 10^9/L$)	PCT (ng/mL)	BNP (pg/mL)	D-dimer (mg/L)	Fibrinogen (g/L)
Ref. range	110-150	3.5-10.0	< 0.05	< 100	< 0.50	2.0-4.0
1	41	18.75	—	—	19.46	—
3	65	15.80	1.50	622.04	6.88	1.48
5	53	20.40	0.36	801.84	—	4.75
7	66	10.90	0.25	674.01	—	5.59
9	74	4.74	0.13	732.11	—	—
14	82	4.06	—	432.64	7.74	—
17	72	3.37	—	—	7.74	—
29 (post-discharge)	63	2.77	—	—	—	—

Abbreviations: BNP, B-type natriuretic peptide; Hb, hemoglobin; PCT, procalcitonin; WBC, white blood cell count. A dash (—) indicates a parameter not measured on that day.

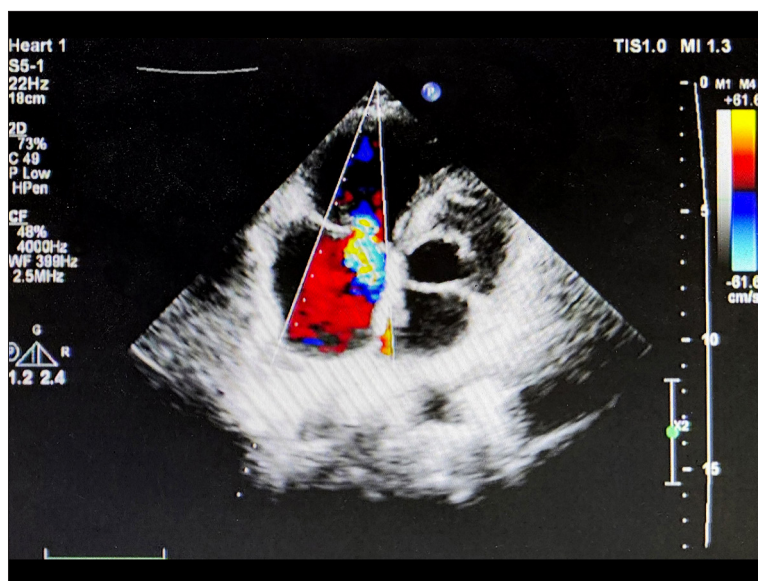


Figure 1. Transthoracic echocardiography (apical four-chamber view with color Doppler) demonstrating right heart enlargement and moderate-to-severe tricuspid regurgitation. The mosaic color jet indicates the regurgitant flow across the tricuspid valve.

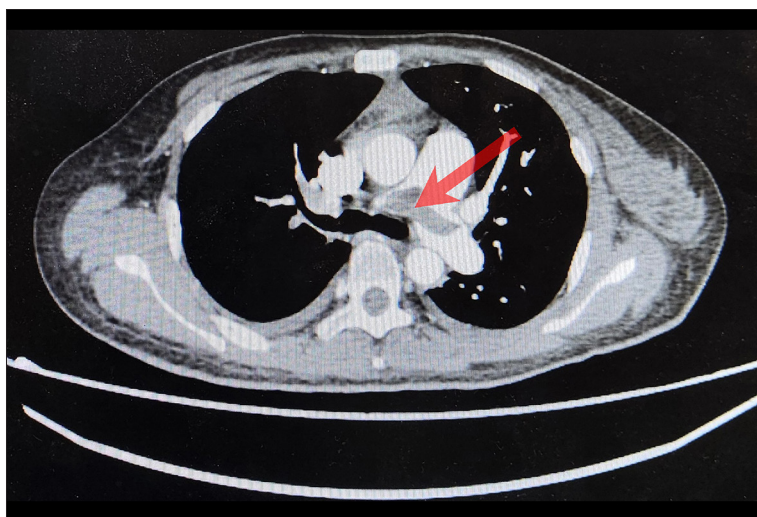


Figure 2. Axial CT pulmonary angiography showing a large filling defect occluding the right pulmonary artery main trunk with absent distal opacification (red arrow). Abbreviations: CT, computed tomography.

The procedure was performed under local anaesthesia (2% lidocaine) via right femoral venous access using the Seldinger technique. The pre-thrombectomy pulmonary artery pressure was 51/23 mm Hg. Multiple aspiration passes were performed using large-bore aspiration catheters (22F, 20F, and 16F) in both pulmonary arteries, retrieving multiple dark-red thrombi from the right and smaller clots from the left (**Figure 3**). Post-thrombectomy angiography demonstrated markedly improved bilateral flow (**Figure 4**); the pulmonary artery pressure decreased to 34/12 mm Hg—a 33% reduction in systolic pressure. The procedural intent was palliative revascularization rather than complete thrombus clearance, and passes were limited accordingly. Aspiration was performed manually with the Tendvia thrombectomy system using its integral 50-mL syringe-based suction; no blood-return or filtration-return circuit was used. Blood loss was quantified by retaining the thrombus and filtering the pooled aspirate from all passes through

gauze into a graduated container, the measured volume totalling approximately 100 mL across the procedure. Access-site hemostasis was achieved with a closure device and manual compression, without hematoma. Both pressures were recorded within this single procedure, with no transfusion running between them. A retrievable IVC filter was deployed under fluoroscopic guidance, with retrieval planned at 3 months [10].

Postoperatively, anticoagulation was initiated with enoxaparin 60 mg (anti-Xa 6000 IU) subcutaneously every 12 hours—a treatment dose selected by multidisciplinary consensus, accepting calculated bleeding risk in favor of thromboembolic prevention, with daily hemoglobin monitoring and ongoing transfusion support. Full-dose rather than prophylactic anticoagulation was chosen because the thrombus was extensive and recent, the patient had already sustained hemodynamic collapse, and the filter was only a temporary safeguard;

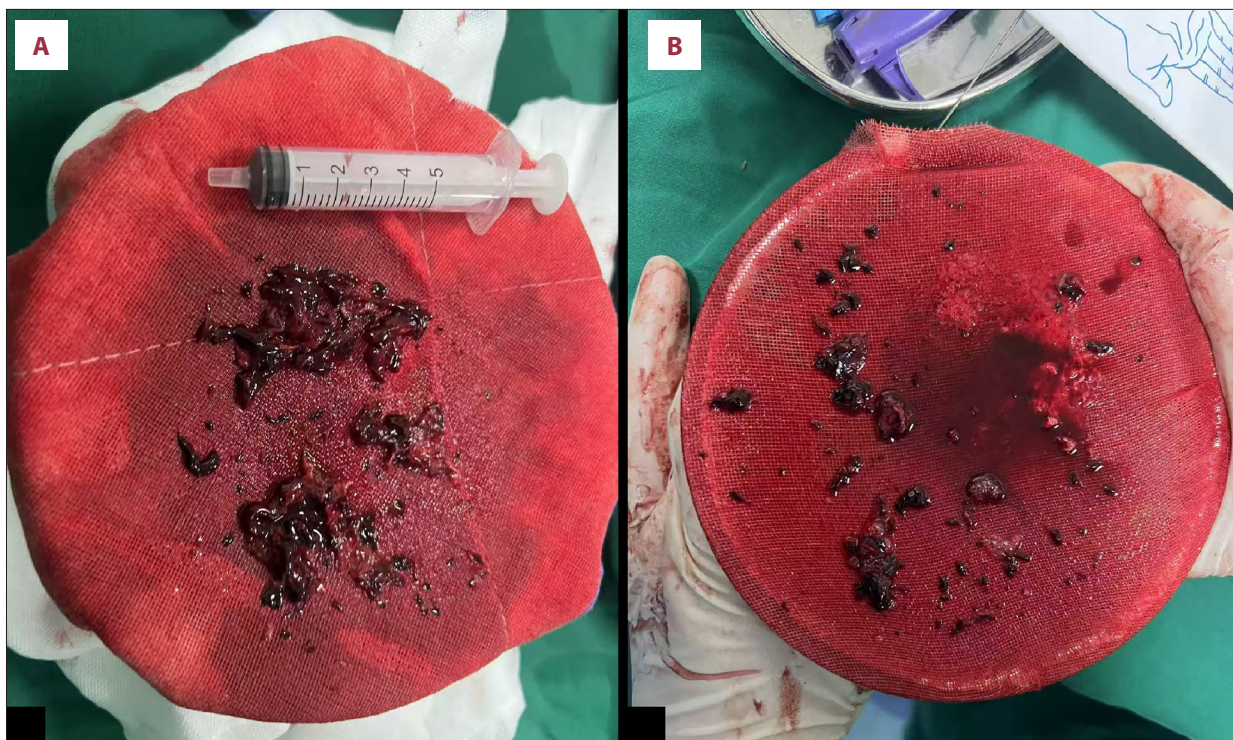


Figure 3. Gross specimens of thrombi (A, B) retrieved during percutaneous aspiration thrombectomy.



Figure 4. Pulmonary angiography before (A) and after (B) aspiration thrombectomy, showing restoration of bilateral pulmonary artery flow and improved distal perfusion.

subtherapeutic anticoagulation was judged to risk fatal re-embolization more than the incremental bleeding from a uterine source accessible to direct control. Bleeding was monitored by quantification of vaginal loss (nursing pad counts), daily hemoglobin and coagulation testing, serial hemodynamic observation, and repeated gynecological review. Anticoagulation was subsequently transitioned to oral rivaroxaban 15 mg twice daily [2]. Empirical antibiotics (piperacillin-tazobactam, with later addition of sitafloxacin) and human fibrinogen concentrate were administered concurrently for pulmonary infection and hypofibrinogenemia, respectively. The total transfusion requirement was 13 units of packed red blood cells and 150 mL of fresh frozen plasma, administered without adverse reactions.

For management of adenomyosis and uterine bleeding, the gynecology team recommended dilatation and curettage (D&C) and subcutaneous leuprolide acetate (gonadotropin-releasing hormone [GnRH] agonist) 3.75 mg every 4 weeks [3]. D&C was performed on day 6 under uninterrupted therapeutic anticoagulation: enoxaparin 6000 anti-Xa IU every 12 hours was continued throughout, with no hold, no bridging interruption, and no reversal agent. Intramuscular oxytocin was given for hemostasis, with quantified blood loss and serial hemoglobin monitoring. Histopathology showed benign endometrial changes with inflammatory hemorrhagic exudate and focal features suggestive of an endometrial polyp. Vaginal bleeding gradually diminished following GnRH agonist therapy.

The patient was transferred from the ICU to the general ward on day 10 and was discharged on day 25 after a 24-day hospitalization. At discharge, the hemoglobin was 72 g/L, there was no dyspnea, the lower-extremity edema had resolved, and only intermittent minor vaginal bleeding persisted. Discharge medications included rivaroxaban 15 mg twice daily and leuprolide acetate 3.75 mg subcutaneously every 4 weeks. At outpatient follow-up 4 days after discharge, the hemoglobin had fallen to 63 g/L, with new mild leukopenia ($2.77 \times 10^9/L$). Uterine bleeding from adenomyosis had not been definitively controlled at discharge, and ongoing loss from this source is the most likely explanation; GnRH agonist therapy requires several weeks for maximal effect, and hysterectomy was planned once hematological stability allowed. A blood film and infectious screen were arranged, but the film result was not available to the authors, and delayed haemolytic transfusion reaction could not be formally excluded. No follow-up beyond this 4-day review was available; IVC filter retrieval and hysterectomy were planned but could not be confirmed.

Discussion

This case illustrates the critical challenge of managing high-risk PE when systemic thrombolysis is contraindicated by active

bleeding. Thrombolysis was associated with major bleeding in 9.24% and intracranial hemorrhage in 1.46% of patients in a meta-analysis of randomized PE thrombolysis trials [11]—risks considered unacceptable in the context of a hemoglobin of 41 g/L and ongoing uterine bleeding. Percutaneous aspiration thrombectomy achieved a 33% reduction in systolic pulmonary artery pressure with immediate angiographic improvement, without recourse to pharmacological thrombolytics. Because both pressures were recorded within a single procedure with no transfusion running between them, this acute change is attributable to mechanical debulking rather than to volume expansion or improved oxygen-carrying capacity. The patient's subsequent recovery, however, cannot be attributed to thrombectomy alone: it occurred alongside 13 units of packed red blood cells, fluid resuscitation, source control of uterine bleeding, antimicrobial therapy, and anticoagulation, and the relative contribution of each cannot be separated within a single case.

The risks of large-bore aspiration in a profoundly anemic patient warrant explicit consideration, since aspiration catheters remove circulating blood alongside thrombus, and at a hemoglobin of 41 g/L significant loss could have precipitated peri-procedural collapse. Aspiration was performed with the Tendvia system using standard manual syringe suction, without any blood-return or filtration-return circuit. Blood loss was estimated by filtering the pooled aspirate from all passes through gauze to retain thrombi and measuring the remaining filtrate, which totalled approximately 100 mL; this bedside method was not independently validated and may not fully distinguish blood from irrigation fluid, so we present it as an intra-procedural estimate rather than a precise measurement. This loss was judged acceptable against the immediate risk of death from obstructive shock, and the palliative intent allowed the number of passes to be limited. Lower-profile or microcatheter systems were not selected because the thrombus occluding the right main pulmonary artery was unlikely to be cleared by small-calibre aspiration; a less effective device would probably have required longer procedures and repeated passes, plausibly increasing rather than reducing cumulative blood loss.

Aspiration thrombectomy is increasingly described as an option for high-risk PE when thrombolysis is contraindicated [4,5]. The EXTRACT-PE trial demonstrated significant reductions in right ventricular to left ventricular (RV/LV) ratio with the Indigo aspiration system in intermediate-risk PE [6]. The FLARE study confirmed reductions in pulmonary artery pressure and right ventricular dilation with the FlowTriever device [7], and the FLASH registry validated these findings in real-world clinical practice [8]. These trials, however, did not enroll hemodynamically unstable patients with severe active bleeding; the present case extends the descriptive experience to this thrombolysis-contraindicated setting in the context of severe

coexisting anemia, without implying equivalent levels of evidence. Published reports of PE complicated by major gynecological bleeding or profound anemia are sparse and largely describe either thrombolysis-eligible patients or bleeding managed after reperfusion; reports in which a hemoglobin below 50 g/L with an actively bleeding uterine source was itself the determinant of device selection are, to our knowledge, not well represented in the literature, which is the specific respect in which this case report is important.

Concurrent IVC filter placement remains the most contestable element of this management. A retrievable filter was placed given the extensive iliofemoral and calf thrombosis and the uncertainty, at the time, as to whether anticoagulation could be sustained against active uterine bleeding [10]. We acknowledge that the evidence for filters in high-risk PE is contested even with coexisting DVT, that filters carry their own risks, and that this patient did subsequently tolerate full-dose anticoagulation, which weakens the retrospective case for the filter and means no clear survival benefit over anticoagulation alone can be claimed. The decision was taken prospectively under genuine uncertainty rather than with knowledge of that outcome, and a retrievable device was chosen for that reason. We do not present it as a generalizable recommendation.

The admission fibrinogen of 1.48 g/L is most consistent with acute consumption and dilution from massive hemorrhage, shock, and resuscitation rather than a chronic hepatic or nutritional defect: after fibrinogen concentrate and stabilization it rose to 4.75 g/L by day 5 and 5.59 g/L by day 7 and remained normal, while the mildly deranged transaminases and albumin followed a similar recovery, consistent with shock-related hepatic hypoperfusion. No pre-morbid fibrinogen was available, so a contributory baseline deficiency cannot be formally excluded.

Curettage without interrupting anticoagulation also merits comment. Standard practice would ordinarily favor a periprocedural hold, but here the thrombus was extensive and recent, the filter was only a temporary safeguard, and interruption risked recurrent embolization in a woman who had already collapsed. Because the operative field was itself the bleeding source, curettage offered direct control of that source rather than creating a new one. Post-procedural bleeding remained within the range anticipated by the gynecology team. We present this as a considered trade-off under competing risks, not a recommended default.

Managing the ongoing uterine bleeding was a constant challenge. Anticoagulation, essential to prevent further venous thromboembolism [1,2], inevitably exacerbated bleeding from the underlying adenomyosis. GnRH agonist therapy suppressed ovarian function and ultimately reduced bleeding, though its maximal therapeutic effect requires several weeks [3]. D&C

provided histopathological diagnosis and short-term haemostatic benefit. Total hysterectomy was recommended as the definitive intervention pending sufficient haemodynamic and hematological stability. The most likely explanation for the continued fall in hemoglobin is ongoing blood loss from adenomyosis, which had improved but not fully resolved at discharge and for which definitive treatment was already planned. A delayed hemolytic transfusion reaction was considered unlikely in the absence of a compatible clinical picture, and no gastrointestinal symptoms were reported; however, as the peripheral blood film was not available to the authors, these could not be formally excluded.

The Pulmonary Embolism Response Team approach was indispensable [9]: coordinated involvement of cardiology, gynecology, critical care, respiratory medicine, and interventional radiology enabled rapid decision-making in a complex scenario. The intersection of active uterine bleeding, chronic anemia, and venous thromboembolism is under-addressed in current guidelines and warrants prospective study.

Applicability and Boundary Conditions

This approach is applicable only to selected high-risk PE cases in which thrombolysis is contraindicated, catheter-directed expertise is immediately available, and individualized bleeding control and anticoagulation planning can be coordinated. Case-specific factors—profound anemia, active uterine bleeding, local unavailability of ECMO-supported embolectomy, and established multidisciplinary capability—shaped both the decision and the management sequence. The role of concomitant IVC filter placement, the anticoagulation strategy, and the sequencing of reperfusion and source control may not generalize beyond these constraints.

What This Case Report Adds

The principal value of this report lies in the integrated management of simultaneously competing risks—obstructive shock, life-threatening hemorrhage and transfusion dependence, therapeutic anticoagulation, and gynecological source control—within a single patient. It illustrates the scenario in which aspiration thrombectomy was selected over thrombolysis and embolectomy, the role of multidisciplinary coordination, and the limits of inference from a single uncontrolled observation. Its limitations are the absence of follow-up beyond 4 days after discharge and the unconfirmed peripheral blood film.

Conclusions

In this single thrombolysis-contraindicated patient with high-risk PE, severe anemia, and active uterine bleeding,

percutaneous aspiration thrombectomy with IVC filter placement was a feasible rescue intervention that produced rapid hemodynamic and angiographic improvement without pharmacological thrombolysis, with palliative rather than curative intent. Because the report describes a single patient without a comparator, it cannot determine the comparative safety or effectiveness of this strategy; it illustrates a potentially useful option in similarly complex scenarios rather than establishing it as proven first-line therapy. Coordinated multidisciplinary care was essential for navigating the competing priorities of reperfusion, bleeding control, and anemia correction. Prospective registries focused on PE complicated by major bleeding are needed to define the comparative role of catheter-directed thrombectomy in such complex scenarios.

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Patient Consent

Written informed consent was obtained from the patient and her family for the publication of this case report and any accompanying images.

Declaration of Figures' Authenticity

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