Current Science
CS 5 (4), 3676-3687 (2025)
Received 15 April 2025 | **Revised 11 May** 2025 | **Accepted 12 June**. 2025 | **Online Available 24 July** 2025 **https://doi.org/ 10.52845/CS/2025-5-4-29**

Current Science

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OPEN ACCESS

ISSN (O) 2795-8639

Original Article

Adenomyosis as a Predisposing Factor for Pregnancy-Associated Hemolytic Uremic Syndrome: A Case-Based Mechanistic Study with Literature and Pathophysiological Correlation

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Abstract:

Background: Hemolytic uremic syndrome (HUS) is a rare obstetric complication with significant renal morbidity. Recent evidence suggests that uterine pathologies like adenomyosis may predispose to thrombotic microangiopathy (TMA), but mechanistic links remain unclear.

Methods: We describe a case of post-abortion pregnancy-associated HUS (pHUS) in a patient with adenomyosis and analyze clinical, histological, and immunological data in the context of TMA mechanisms. We also review existing literature and propose a plausible biological model.

Results: The patient presented with thrombocytopenia, hemolytic anemia, and acute kidney injury, consistent with pHUS. Renal biopsy confirmed TMA. Plasma exchange and eculizumab led to full recovery. A hypothesized model suggests endometrial necrosis, cytokine storm, and complement activation in adenomyosis may trigger pHUS.

Conclusion: This is the first reported case of post-abortion HUS in a patient with adenomyosis. The interplay between decidual necrosis, high secretory endometrial collapse, and microvascular thrombosis may mimic tumor lysis syndrome, triggering TMA. Early recognition and prompt treatment are crucial for a favorable outcome.

Keywords: Hemolytic uremic syndrome; Thrombotic microangiopathy; Adenomyosis; Induced abortion; Eculizumab; Obstetric complications

Introduction

Pregnancy-associated hemolytic uremic syndrome (pHUS) is a rare but severe form of thrombotic microangiopathy (TMA), characterized by the clinical trial of non-immune (microangiopathic) hemolytic anemia, thrombocytopenia, and acute kidney injury (AKI) [1,2]. Unlike typical, Shiga toxin-mediated HUS, pHUS is primarily complement-mediated and most commonly occurs in the postpartum period [3], with an estimated incidence of approximately 1 in 136,000 pregnancies [2]. Its occurrence following first-trimester induced abortion is exceedingly rare, with only sporadic case reports in the literature [4].

Adenomyosis is a chronic, estrogen-dependent gynecologic disorder defined by the presence of ectopic endometrial glands and stroma within the myometrium. It is commonly associated with menorrhagia, dysmenorrhea, and chronic pelvic pain [6,7]. Although traditionally regarded as benign, mounting evidence implicates adenomyosis in systemic and local pathophysiological processes. These include chronic inflammation with elevated uterine levels of IL-1β, IL-6, TNF-α, and COX-2 [8,9], aberrant angiogenesis mediated by VEGF, activin, and

follistatin [10-12], and a hypercoagulable state characterized by increased tissue factor expression and elevated coagulation markers during menstruation [13,14].

Furthermore, adenomyosis has been associated with endothelial dysfunction, immune dysregulation—such as altered Th17/Treg balance—and production of antiphospholipid antibodies [15,16]. These disturbances are thought to contribute to a uterine microenvironment prone to inflammation, thrombosis, and vascular injury [16]. Inflammatory cytokines, such as IL-6 and TNF-α, can upregulate tissue factor and activate the coagulation cascade, bridging inflammation and thrombosis [17,18]. The discount on anticoagulant mechanisms commonly observed in chronic inflammatory conditions further exacerbates this risk [19].

Given these changes, it is biologically plausible that in patients with adenomyosis, systemic stressors—such as abrupt HCG decline and trophoblastic necrosis following abortion—may amplify endothelial activation and complement pathway engagement, potentially triggering TMA. This scenario mirrors mechanisms seen in atypical HUS (aHUS), where complement dysregulation plays a central role [20,21]. Indeed, complement gene variants have been identified in several cases of pregnancy-associated aHUS [22,23].

Here, we describe a rare and instructive case of pHUS in a 35-year-old woman with untreated adenomyosis following a first-trimester surgical abortion. The case underscores the need for heightened clinical vigilance for pHUS in similar contexts and supports a hypothesized pathophysiological link between adenomyosis and complement-mediated microvascular injury. Understanding this association is essential for tracking early warning signs, guiding timely intervention, and informing future research on preventive strategies in women with gynecological comorbidities undergoing pregnancy-related procedures.

Methods

Patient Description and Clinical Data Collection

This study is based on a single, clinically well-documented case of a 35-year-old woman diagnosed with pHUS following an induced abortion. The patient had a known 2-year history of adenomyosis and presented with acute lower abdominal pain, progressive oliguria, and hematologic abnormalities one week after the procedure. Detailed clinical data including vital signs, physical examination findings, obstetric history, medication use, and comorbidities were recorded upon admission and throughout hospitalization. The diagnostic workflow and therapeutic interventions followed standard institutional protocols and were approved by the ethics committee of the affiliated hospital. The patient provided informed consent for anonymous data publication and histological image usage.

Laboratory and Hematologic Evaluation

Serial laboratory assessments were performed between January 10 and January 23, 2025. Blood samples were analyzed for:

1) Complete blood count (CBC): including hemoglobin, hematocrit, platelet count, and white blood cell count.
2) Markers of hemolysis: lactate dehydrogenase (LDH), indirect bilirubin, haptoglobin, and peripheral blood smear for schistocytes.
3) Coagulation parameters: prothrombin time (PT), activated partial thromboplastin time (APTT), thrombin time (TT), fibrinogen, D-dimer, and international normalized ratio (INR).
4) Renal function tests: serum creatinine, urea, estimated glomerular filtration rate (eGFR), uric acid, and electrolyte profile.
5) Inflammatory markers: C-reactive protein (CRP), procalcitonin (PCT), and serum ferritin.
6) Complement testing: levels of complement components (C3, C4) and CH50 were measured to evaluate alternative pathway activity.

All tests were conducted in the hospital's central laboratory using standardized automated methods.

Renal Histopathology and Immunologic Workup

Percutaneous renal biopsy was performed under ultrasound guidance after stabilization of the patient's coagulation status. The tissue specimens were evaluated by:

1) Light microscopy with hematoxylin-eosin

(HE), periodic acid-Schiff (PAS), Masson's trichrome, and Jones methenamine silver stains;

2) Immunofluorescence for IgG, IgA, IgM, C3, C1q, fibrinogen, kappa, lambda, and PLA2R;

Histopathologic features consistent with TMA—including endothelial swelling, fibrinoid necrosis, capillary occlusion, and interstitial inflammation—were documented.

Complement-mediated atypical HUS was diagnosed based on renal pathology, laboratory features, and exclusion of other causes such as sepsis or disseminated intravascular coagulation (DIC).

4. Therapeutic Protocol

Upon diagnosis, treatment was initiated with:

1) Intravenous methylprednisolone (80 mg/day) to modulate systemic inflammation;

2) Therapeutic plasma exchange (TPE) once daily for 3 consecutive days, using fresh frozen plasma as a replacement fluid;

3) Eculizumab (900 mg IV weekly) administered as per recommended dosing guidelines for complement-mediated TMA;

4) Supportive care included intravenous fluids, electrolyte correction, blood pressure control with amlodipine and labetalol, and thromboprophylaxis with low molecular weight heparin after stabilization.

Treatment efficacy was monitored using serial laboratory parameters and clinical signs of renal recovery.

5. Literature Review Methodology

A narrative literature review was conducted to contextualize the observed clinical phenomenon and construct a plausible pathophysiologic mechanism linking adenomyosis to pHUS. Electronic searches were performed in PubMed, Web of Science, and Scopus databases using the following keywords: "adenomyosis", "hemolytic uremic syndrome", "pregnancy-associated HUS", "thrombotic microangiopathy", "complement activation", "induced abortion", "eculizumab", "cytokine storm", and "hypercoagulable state".

Inclusion criteria were limited to English-language studies published between 2015 and 2025. Articles involving case reports, case series, reviews, and experimental research were included. References cited within relevant papers were also screened for additional sources.

6. Mechanistic Framework Construction

Based on patient data and literature synthesis, a hypothetical pathophysiological model was developed to illustrate how adenomyosis may predispose to complement-mediated endothelial injury in the setting of post-abortion tissue necrosis. The model integrates clinical, molecular, and immunologic factors such as:

1) Inflammatory cytokine release (e.g., IL-6, TNF-α);

2) Overexpression of tissue factor and VEGF in adenomyotic tissue;

3) Collapse of secretory endometrium post-hormonal withdrawal;

4) Complement activation via the alternative pathway.

Results

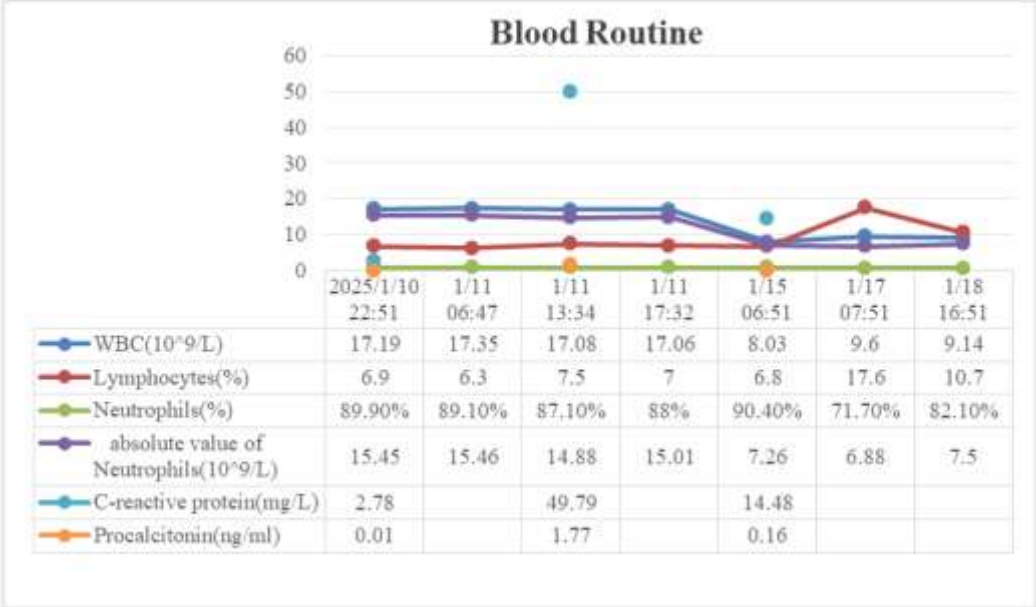
1. Clinical Course and Presentation

A 35-year-old woman with a two-year history of untreated adenomyosis presented with sudden-onset lower abdominal pain and oliguria one week after an uncomplicated first-trimester surgical abortion (8+ weeks gestation). Initial evaluation revealed suprapubic tenderness and uterine enlargement (~10-week size), without fever or signs of peritonitis. Vaginal bleeding was minimal. Within 48 hours of symptom onset, she developed progressive fatigue, hematuria, and generalized edema. Notably, there was no history of hypertension, autoimmune disease, or prior renal dysfunction.

2. Hematological and Inflammatory Laboratory Findings

Serial complete blood counts demonstrated progressive anemia (hemoglobin nadir: 78 g/L) and thrombocytopenia (lowest platelet count: 29×10^9/L). Blood smear showed schistocytes, consistent with microangiopathic hemolytic anemia. Inflammatory markers were markedly elevated at admission, including white blood cell count (WBC: 17.5×10^9/L), C-reactive protein (CRP: 97 mg/L), and procalcitonin (PCT: 5.2 ng/mL). These decreased significantly after

empirical antibiotics (Figure 1), suggesting sterile inflammation rather than overt sepsis.



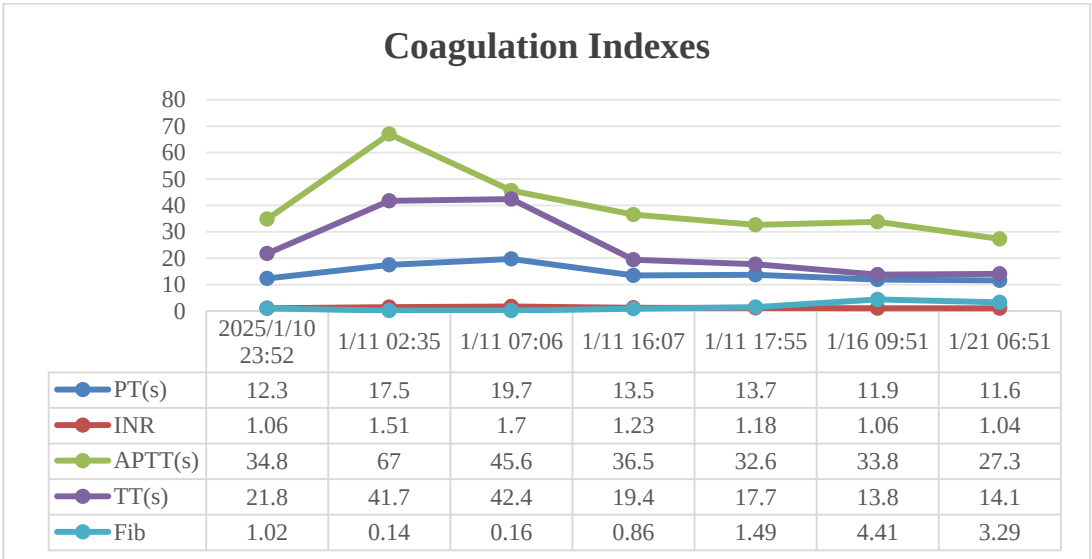
	2025/1/10 22:51	1/11 06:47	1/11 13:34	1/11 17:32	1/15 06:51	1/17 07:51	1/18 16:51
WBC(10^9/L)	17.19	17.35	17.08	17.06	8.03	9.6	9.14
Lymphocytes(%)	6.9	6.3	7.5	7	6.8	17.6	10.7
Neutrophils(%)	89.90%	89.10%	87.10%	88%	90.40%	71.70%	82.10%
absolute value of Neutrophils(10^9/L)	15.45	15.46	14.88	15.01	7.26	6.88	7.5
C-reactive protein(mg/L)	2.78		49.79		14.48		
Procalcitonin(ng/ml)	0.01		1.77		0.16		

Figure 1. Results of Blood Routine

Repeated blood routine examinations from January 10 to 11 showed a significant increase in white blood cells, C-reactive protein and procalcitonin. 48 hours after the application of antibiotics, white blood cells, C-reactive protein and procalcitonin all decreased significantly.

3. Coagulation Profile and Evidence of Consumptive Coagulopathy

Coagulation testing revealed early and dynamic changes suggestive of TMA. Fibrinogen was initially low (1.02 g/L), with elevated D-dimer levels (>5000 ng/mL) and prolonged activated partial thromboplastin time (APTT: 67 s) and thrombin time (TT: 42 s). Prothrombin time (PT) was mildly increased (15.8 s), and international normalized ratio (INR) peaked at 1.5. These findings were consistent with consumptive coagulopathy in the context of evolving TMA. Following plasma exchange and complement inhibition, coagulation parameters gradually normalized (Figure 2).



	2025/1/10 23:52	1/11 02:35	1/11 07:06	1/11 16:07	1/11 17:55	1/16 09:51	1/21 06:51
PT(s)	12.3	17.5	19.7	13.5	13.7	11.9	11.6
INR	1.06	1.51	1.7	1.23	1.18	1.06	1.04
APTT(s)	34.8	67	45.6	36.5	32.6	33.8	27.3
TT(s)	21.8	41.7	42.4	19.4	17.7	13.8	14.1
Fib	1.02	0.14	0.16	0.86	1.49	4.41	3.29

Figure 2. Coagulation profile in a patient with pHUS

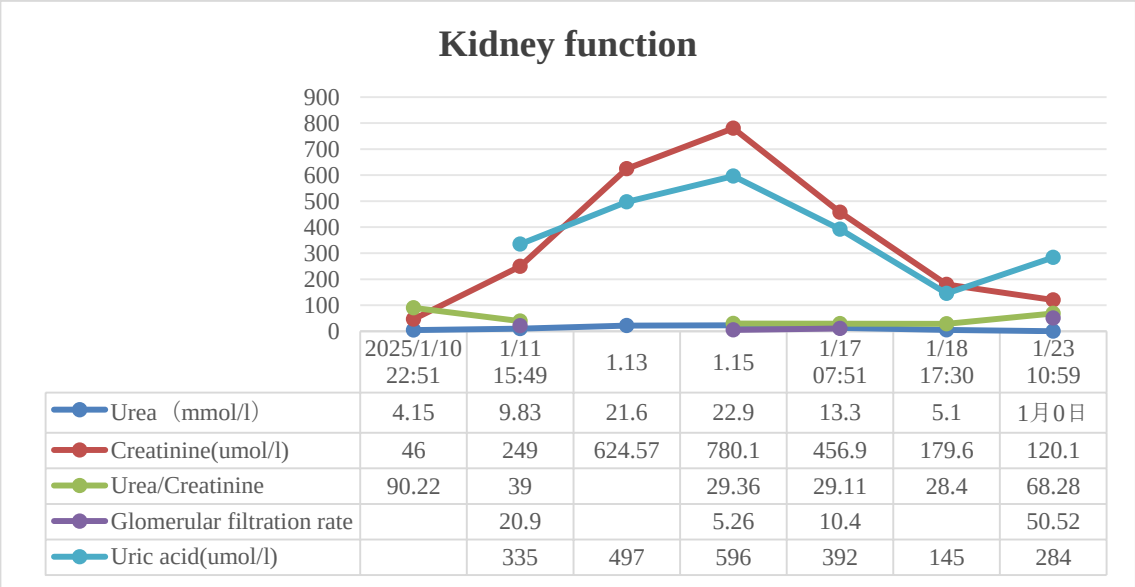
Serial laboratory monitoring of coagulation parameters from January 10 to January 21, 2025, revealed dynamic changes associated with microangiopathic coagulopathy. Notably,

activated partial thromboplastin time (APTT) and thrombin time (TT) peaked early in the clinical course, with APTT reaching 67 seconds and TT exceeding 40 seconds on January 11. Prothrombin time (PT) and international normalized ratio (INR) were mildly elevated in the early phase but gradually normalized. Fibrinogen (Fib) levels were initially low (1.02 g/L), consistent with consumptive coagulopathy, but subsequently rebounded above the normal range, indicating recovery. These trends reflect the evolving coagulopathy characteristic of pHUS and the therapeutic response to plasma exchange and complement blockade.

4. Renal Function Deterioration and Recovery

Renal function deteriorated rapidly after admission. Serum creatinine rose from 92 μmol/L on day 1 to 327 μmol/L by day 5, and estimated glomerular filtration rate (eGFR) dropped to 22 mL/min/1.73m². Urea and uric acid levels also increased substantially. The patient developed oliguria but did not require dialysis.

Following treatment with plasma exchange, methylprednisolone, and eculizumab, renal function improved progressively, with creatinine declining to 103 μmol/L by day 14 and eGFR increasing to 68 mL/min/1.73m² at discharge (Figure 3).



	2025/1/10 22:51	1/11 15:49	1.13	1.15	1/17 07:51	1/18 17:30	1/23 10:59
Urea（mmol/l）	4.15	9.83	21.6	22.9	13.3	5.1	1月0日
Creatinine(umol/l)	46	249	624.57	780.1	456.9	179.6	120.1
Urea/Creatinine	90.22	39		29.36	29.11	28.4	68.28
Glomerular filtration rate		20.9		5.26	10.4		50.52
Uric acid(umol/l)		335	497	596	392	145	284

Figure 3. Kidney function trends in a patient with pHUS

The levels of urea (mmol/L), creatinine (μmol/L), urea/creatinine ratio, glomerular filtration rate (GFR, mL/min/1.73m²), and uric acid (μmol/L) were recorded from January 10 to January 23, 2025. A rapid rise in creatinine and urea was observed between January 11 and January 15, corresponding to acute kidney injury. Following initiation of plasma exchange and eculizumab therapy, renal function progressively improved, as shown by declining urea and creatinine levels and rising GFR.

5. Renal Histopathological Findings

Renal biopsy revealed 18 glomeruli, of which one showed complete global sclerosis. The remaining glomeruli exhibited no mesangial hypercellularity or matrix expansion. The mesangial areas contained 1–3 mesangial cells per area, and the glomerular capillary walls were of normal thickness. Endothelial cells showed no proliferation. Notably, foot process effacement was observed, indicating podocyte injury.

Tubulointerstitial examination showed diffuse swelling and granular degeneration of tubular epithelial cells. Patchy tubular dilation was present, with epithelial cell shedding and the formation of scattered proteinaceous and leukocyte casts. Approximately 10% of the interstitial area exhibited edema and fibrosis, with multifocal infiltration of lymphocytes, monocytes, neutrophils, and eosinophils. Small-caliber interstitial blood vessels demonstrated mild segmental wall thickening, suggestive of microvascular injury.

Masson's trichrome staining revealed no significant immune complex deposition. Congo red staining was negative, ruling out amyloid deposition. Immunohistochemical staining for PLA2R was negative, excluding primary membranous nephropathy.

Taken together, the histological findings supported the diagnosis of TMA involving acute tubular injury, podocyte damage, and interstitial inflammation, consistent with a clinical diagnosis of pHUS. The result of light microscopy showed multifocal acute and chronic interstitial inflammation, patchy acute tubular necrosis, and mild glomerular lesions. There was no fluorescence distribution: IgG-, IgA-, IgM-, C3-, Fib-, C1q-, Kappa-, Lambda-, IgG1-, IgG2-, IgG3-, IgG4-(Figure 4).

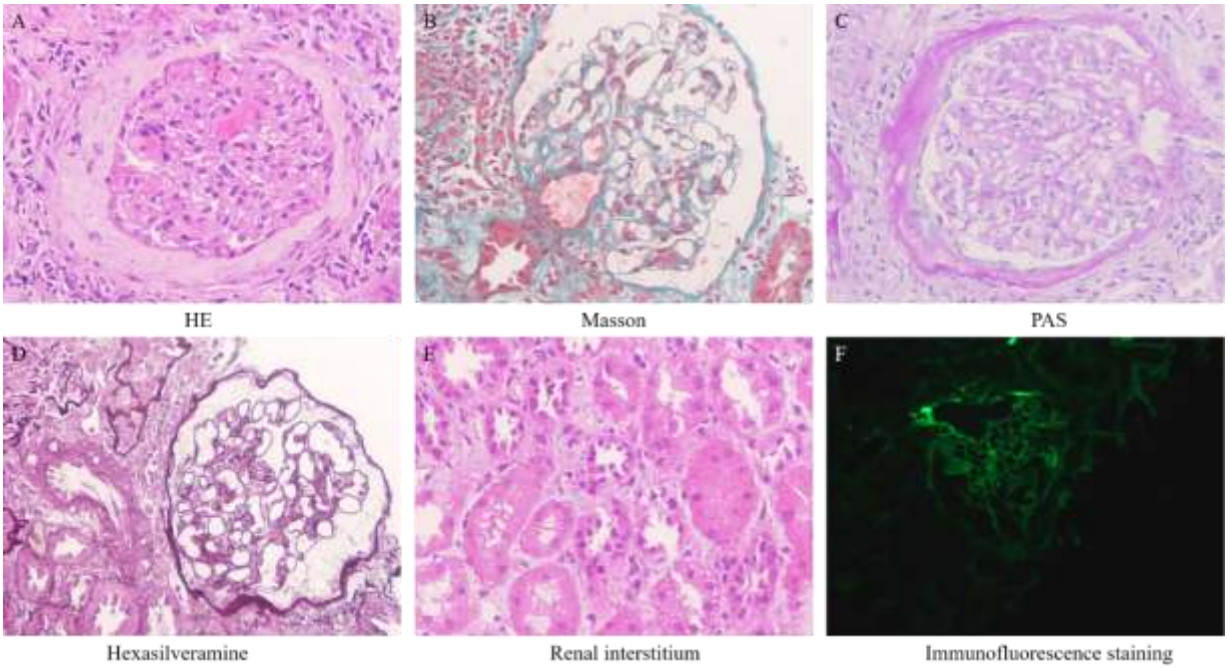


Figure 4. Renal histopathological and immunofluorescence findings in a patient with pregnancy-associated hemolytic uremic syndrome (pHUS)

(A) Glomerular HE staining shows a preserved glomerular structure without mesangial cell proliferation or capillary wall thickening. One glomerulus (not shown here) showed global sclerosis. (×400); (B) Masson's trichrome stain reveals no evidence of immune complex or fibrin deposition, and the mesangial matrix appears normal. (×400); (C) PAS stain demonstrates intact glomerular basement membrane with no obvious thickening or duplication. (×400); (D) Silver stain (Jones methenamine) shows clear glomerular basement membranes and intact capillary loops without spike formation or double contouring. (×400); (E) Tubulointerstitial, HE staining indicates tubular epithelial swelling, granular degeneration, and interstitial lymphocyte and eosinophil infiltration. Tubular lumen dilation and epithelial shedding are also observed. (×400); (F) Immunofluorescence staining shows negative PLA2R expression, indicating no immune complex deposition or membranous nephropathy. (×400)

6. Clinical Outcome

The patient received three consecutive days of plasma exchange, corticosteroid therapy, and a single dose of eculizumab. Her hematologic and renal parameters steadily improved. She was discharged on day 21 in stable condition with full recovery of renal function, normal platelet count (190×10^9/L), and resolved hemolysis (LDH and bilirubin normalized). Follow-up at 4 weeks confirmed complete clinical remission, without evidence of relapse or persistent renal impairment.

7. Comparison with Similar Reported Cases

Table 1 compares the clinical characteristics of previously reported cases of pHUS with the current case. A comprehensive review of the literature reveals that nearly all documented cases of pHUS have occurred in the late third trimester

or postpartum period, most often in association with systemic disorders such as preeclampsia, autoimmune disease (e.g., SLE), or infection. Notably, none of the published reports have described pHUS following first-trimester abortion, nor have any identified uterine adenomyosis as a contributing gynecological comorbidity. This case is therefore the first to report pHUS arising after an early surgical abortion in a patient with symptomatic, untreated adenomyosis—highlighting a potentially novel and previously unrecognized pathophysiological link between chronic uterine inflammation and complement-mediated TMA. In contrast to prior cases, our patient exhibited several distinguishing features: (1) a known history of adenomyosis without prior intervention; (2) absence of infection, hypertensive disorders, or systemic autoimmune disease; (3) a rapid and favorable clinical response to early complement blockade with eculizumab; and (4) renal histopathology consistent with complement-mediated TMA, but without immune complex deposition. Together, these findings underscore the novelty of this presentation and suggest that adenomyosis may represent an underappreciated systemic risk factor for TMA in reproductive-age women undergoing pregnancy termination.

Table 1 The comparison of the clinical characteristics between previously reported cases of pHUS and the current case

Author	Age (Year)	Pregnancy Type	Onset Timing	Underlying Conditions	Initial Symptoms	Treatment	Outcome
Wu et al. (2021)	32	Term vaginal delivery	Postpartum day 2	None	Oliguria, anemia, thrombocytopenia	Plasma exchange + eculizumab	Full recovery
Alex D-Vet al. (2024)	25	emergency cesarean section	4 days after presentation	preeclampsia	anemia, thrombocytopenia, mild transaminitis	eculizumab pneumococcal vaccination and prophylactic penicillin treatment	Full recovery stable renal function
Haider Ghazanfar et al. (2024)	36	emergent cesarean delivery	postoperative day 5	preterm premature rupture of the membranes	anemia, low platelet count, and worsening renal function,	Plasmapheresis + eculizumab	Full recovery
Marc Tshilanda et al. (2020)	31	cesarean section	postoperative day 2	high blood pressure	anemia, low platelet count, acute kidney injury, declining liver function	hemodialysis	Full recovery
Bair Cadet et al. (2021)	18	emergent cesarean section	5 weeks	epilepsy, hypertension, atypical hemolytic uremic syndrome and end-stage	acute kidney injury	plasma exchange, steroids, eculizumab and hemodialysis	recurrent

				kidney disease			
Yoshihiro Miyazaki et al. (2024)	31	cesarean section.	postoperative day 1		anemia, decreased platelet count, and oliguric acute kidney injury	plasma exchange (PE), and hemodialysis (HD) Ravulizumab	No recurrences
Arif Asif et al. (2016)	33	cesarean section		thrombocytopenia	anemia, thrombocytopenia	plasma exchange +eculizumab	recovery
Andreas Kouroukl aris et al. (2014)	23	premature delivery,	Three months		no dialysis-dependent renal failure and without signs of hemolysis.	eculizumab	No recurrences
Mae-Lan Winchesteret al. (2019)	37	vaginal delivery	postpartum day 4		anemia, thrombocytopenia acute kidney injury	eculizumab	Full recovery
Ayako Inatomi et al. (2025)	33	emergency cesarean section	postoperative day 5	SLE	hemolytic anemia, thrombocytopenia, and renal dysfunction	Plasma exchange +eculizumab	
Hong Sang Choi et al. (2021)	33	Caesarean section	12th day postpartum		Microangiopathic anemia, thrombocytopenia and renal dysfunction	Plasma exchange +eculizumab	no recurrence

8. Mechanistic Correlation with Adenomyosis

Given the patient's known history of adenomyosis and lack of other autoimmune or genetic thrombotic risk factors, we postulate that post-abortion trophoblastic necrosis, abrupt hormonal withdrawal, and local uterine cytokine surge in the setting of adenomyosis may have triggered complement activation and endothelial injury. This is supported by the temporal proximity of abortion to symptom onset, the rapid systemic inflammatory response, and the biopsy-confirmed TMA pathology in the absence of sepsis or HELLP syndrome. A hypothetical model linking adenomyosis to complement-mediated thrombotic microangiopathy is illustrated in Figure 5.

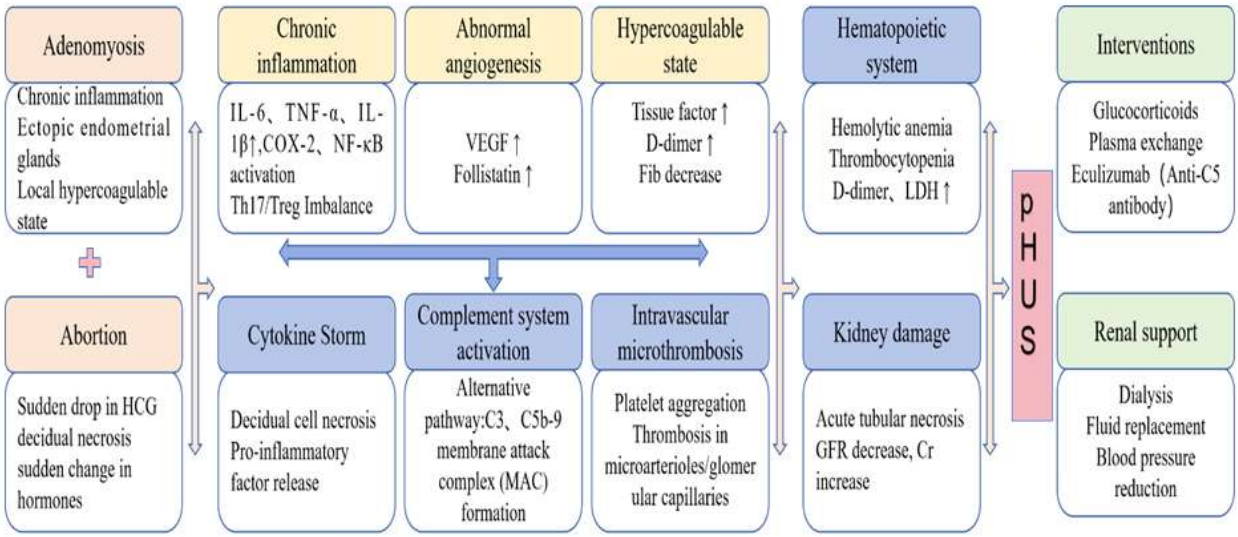


Figure 5. Mechanistic Correlation with Adenomyosis

Discussion

Pregnancy-associated hemolytic uremic syndrome (pHUS) is a rare but potentially fatal form of thrombotic microangiopathy (TMA), typically triggered by complement dysregulation in the peripartum period. Although most reported cases occur postpartum, this study presents an unusual case of pHUS arising after a first-trimester induced abortion in a woman with a history of adenomyosis. To our knowledge, this is the first reported instance linking adenomyosis to the pathogenesis of post-abortion HUS, thereby raising the possibility of a novel uterine-inflammatory trigger for systemic TMA.

Adenomyosis has traditionally been viewed as a benign uterine condition, primarily associated with menorrhagia and dysmenorrhea. However, increasing molecular evidence has demonstrated that adenomyosis is characterized by chronic inflammation, aberrant angiogenesis, and a local procoagulant environment [28]. The ectopic endometrial tissues in the myometrium produce high levels of pro-inflammatory cytokines such as IL-1β, IL-6, and TNF-α, alongside elevated expression of tissue factor and other coagulation mediators [29]. These changes may contribute to a localized hypercoagulable state which, under systemic stress, could spill over into systemic circulation and activate endothelial injury pathways.

In this case, the patient experienced a rapid and severe onset of thrombocytopenia, hemolytic anemia, and acute kidney injury within one week of surgical abortion. Renal biopsy confirmed typical histopathological findings of TMA, including tubular epithelial necrosis, interstitial inflammation, and glomerular endothelial injury, but without immune complex deposition. Complement-mediated TMA (aHUS) was diagnosed, and the patient responded favorably to plasma exchange and eculizumab, an anti-C5 monoclonal antibody that halts terminal complement activation. The positive response to eculizumab supports the hypothesis that complement activation played a pivotal role in this case.

We propose a pathophysiological model whereby abrupt hormonal withdrawal (i.e., the sudden drop in HCG following abortion) induces necrosis of hormonally active ectopic endometrial glands in adenomyosis. This may resemble a "tumor lysis-like" event, resulting in a systemic inflammatory surge and release of procoagulant and complement-activating mediators. In patients with preexisting endothelial vulnerability or subtle complement regulatory defects, this cascade may precipitate full-blown TMA. This aligns with existing models of atypical HUS in pregnancy, where systemic triggers (such as preeclampsia, HELLP syndrome, or sepsis) unmask underlying complement dysregulation [30-33].

This case also highlights the diagnostic challenges in differentiating pHUS from other obstetric complications. The patient initially presented with symptoms that could mimic infection or disseminated intravascular coagulation (DIC). However, the lack of fever, negative cultures, and the constellation of laboratory findings (i.e.,

elevated LDH, low haptoglobin, schistocytes on peripheral smear, thrombocytopenia, and worsening renal function) strongly favored TMA. The dynamic changes in coagulation markers and kidney function, as shown in serial graphs, further reinforce the diagnosis.

Importantly, this report draws attention to a potentially overlooked population at risk—women with adenomyosis undergoing pregnancy termination. Although adenomyosis has been associated with obstetric complications such as miscarriage and preterm labor, its systemic implications have not been adequately explored in the context of thrombotic disease. This case suggests that adenomyosis may not merely be a local uterine pathology but may predispose certain individuals to systemic endothelial injury under physiologic stress.

Several clinical implications arise from this case:

1) Early recognition of pHUS in post-abortion settings is essential, especially in patients with unexplained anemia, thrombocytopenia, and renal dysfunction.
2) Gynecological comorbidities such as adenomyosis should be considered as potential systemic risk factors for TMA in reproductive-aged women.
3) Pre-abortion counseling and risk assessment should include coagulation profiles and inflammation markers in patients with known adenomyosis or other chronic inflammatory gynecologic conditions.
4) Multidisciplinary care involving nephrology, hematology, and gynecology is key for timely diagnosis and treatment initiation.

This case-based mechanistic hypothesis has limitations. It represents a single case, and causality cannot be definitively established without genetic screening for complement mutations or a larger cohort study. Nevertheless, it underscores a novel and plausible biological pathway that warrants further investigation through translational models and registry-based studies.

Conclusion

This case highlights the importance of recognizing pHUS as a rare but potentially life-threatening complication following induced abortion, particularly in patients with preexisting adenomyosis. The presence of unexplained anemia, thrombocytopenia, and acute kidney injury in the post-abortion setting should prompt early evaluation for thrombotic microangiopathy. Timely diagnosis and multidisciplinary intervention are essential to improve patient outcomes. Moreover, comprehensive pre-abortion counseling and long-term management of adenomyosis are critical to reducing the likelihood of such severe complications in women with no immediate fertility intentions.

Declarations

Ethics approval

Informed consent was obtained from the patient for this study and she has signed the informed consent form. The research was approved by the Ethics Committee of Shandong Provincial Maternity and Child Health Hospital, Affiliated to Qingdao University (SWYX: NO.2025-016).

Consent for Publication

All authors consent to publication of the present manuscript.

Data sharing statement

The datasets used during the current study are available from the corresponding author on reasonable request.

Competing Interests

The authors declare that they have no competing interests.

Funding

This research was supported by the the Shandong Province Maternal and Child Health Youth Science and Technology Innovation Project (SFYZXJJ-2024032), and Shandong Province Postdoctoral Innovation Project (SDCX-ZG-202503168).

Acknowledgments

Not applicable.

Author Contribution

Pengjuan He Resources, Writing – original draft

Fangfang Lang Formal analysis, Supervision

Shan Li: Writing – original draft

Kun Geng: Data curation

Wei Tian: Funding acquisition, Writing – review & editing, Project administration

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