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ARTICLE

Clinical features and therapeutic plasma exchange in postpartum hemorrhage-associated thrombotic microangiopathy following massive transfusion

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Abstract

Pregnancy-associated thrombotic microangiopathy (TMA) is a rare but life-threatening emergency, the diagnosis of which is particularly challenging when it emerges following postpartum hemorrhage (PPH), massive transfusion, disseminated intravascular coagulation (DIC), or HELLP (hemolysis, elevated liver enzymes, low platelets)-like laboratory abnormalities. We use two institutional cases to anchor recent literature and highlight bedside clues that should prompt suspicion of TMA after obstetric bleeding. In both patients, active hemorrhage was initially controlled, yet severe thrombocytopenia, Coombs-negative microangiopathic hemolytic anemia with schistocytes, markedly elevated lactate dehydrogenase (LDH), and renal dysfunction persisted or progressed. These features prompted the initiation of therapeutic plasma exchange (TPE), after which both patients showed clinical and biochemical improvement.

Recent literature from 2021 to 2025 reinforces three messages. First, postpartum TMA should be suspected when hematologic and renal abnormalities continue after hemostasis rather than resolving with routine obstetric care. Second, immune thrombotic thrombocytopenic purpura (TTP) must be promptly considered, as delayed treatment can be catastrophic; therefore, early empiric plasma-based therapy remains appropriate when TTP cannot be confidently excluded. Third, PPH-associated TMA is biologically heterogeneous, and some patients may ultimately prove to have complement-mediated disease rather than classic TTP, which has implications for complement evaluation, targeted short-term complement inhibition, and follow-up strategies.

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We conclude that severe hemorrhage and transfusion support do not preclude the presence of TMA; in some patients they may obscure it. A pragmatic, multidisciplinary approach incorporating repeated blood-smear review, rapid hemolysis assessment, ADAMTS13 (a disintegrin and metalloprotease with thrombospondin type 1 motif, member 13) testing when available, complement assessment when clinically indicated, and timely initiation of TPE can improve early decision-making in this uncommon yet high-risk setting.

Keywords

postpartum hemorrhage; thrombotic microangiopathy; therapeutic plasma exchange; pregnancy; massive transfusion

Highlights

- Postpartum hemorrhage-associated thrombotic microangiopathy (TMA) should be suspected when thrombocytopenia, schistocytic hemolysis, elevated lactate dehydrogenase (LDH), and renal dysfunction persist after adequate hemostasis and transfusion support.
- Massive transfusion and disseminated intravascular coagulation (DIC) do not exclude underlying TMA; in some patients they may mask it, and failure of hematologic parameters to normalize after resuscitation is a key diagnostic clue.
- Therapeutic plasma exchange (TPE) remains central to management whenever thrombotic thrombocytopenic purpura (TTP) cannot be promptly excluded, and both illustrative cases showed clinical and biochemical improvement after early initiation of plasma exchange.
- ADAMTS13 activity should be interpreted in the full clinical context: severe deficiency (< 10%) supports immune TTP, while intermediate values do not rule out complement-mediated TMA or other pregnancy-associated TMA subtypes.
- A pragmatic, multidisciplinary approach combining serial laboratory surveillance, early ADAMTS13, and complement testing when available, and a low threshold for empiric plasma exchange may improve outcomes in this rare but life-threatening postpartum emergency.

1. INTRODUCTION

Thrombotic microangiopathy (TMA) in pregnancy and the postpartum period represents a diagnostic and therapeutic emergency, as maternal deterioration may develop within hours, yet the syndrome frequently overlaps with more common obstetric disorders. The defining triad of thrombocytopenia, microangiopathic hemolytic anemia, and end-organ injury can mimic or coexist with severe preeclampsia, HELLP (hemolysis, elevated liver enzymes, low platelets) syndrome, disseminated intravascular coagulation (DIC), acute fatty liver of pregnancy, and major blood loss [1–3]. Among these scenarios, postpartum hemorrhage (PPH) is particularly challenging because clinicians understandably prioritize hemostasis, transfusion support, and immediate patient survival. Persistent TMA may therefore be recognized late, often after the onset of renal injury, neurologic involvement, or refractory hemolysis has already evolved [1, 2, 5].

Recent literature has refined the contemporary understanding of pregnancy-associated TMA. Hematology reviews published since 2021 emphasize that failure of hematologic parameters to normalize following delivery or hemorrhage control should prompt renewed diagnostic scrutiny rather than reassurance [1–3]. Concurrently, nephrology and obstetric literature now suggests that PPH-associated TMA may not represent a single disease entity. Some patients present with immune thrombocytopenic purpura (TTP) necessitating urgent plasma exchange, while others likely have complement-mediated TMA triggered by obstetric stress, tissue injury, or renal cortical ischemia [5–8].

Since the original material available to us was prepared as a stand-alone case report, the present manuscript has been developed as a case-based clinical article centered on two illustrative cases from our institution. Our aim is not to claim a definitive mechanistic classification for these patients, but rather to provide a clinically useful analysis of diagnostic clues, the role of therapeutic plasma exchange (TPE), and recent evidence relevant to PPH-associated TMA.

2. METHODS

2.1 Case Selection and Data Extraction

This article was developed from two archived institutional cases of PPH-associated TMA managed at Beijing Friendship Hospital. Clinical details were extracted from the archived case materials

and accompanying treatment-course figures, with emphasis on the obstetric context, transfusion support, platelet counts, hemolysis markers, renal function, ADAMTS13 (a disintegrin and metalloprotease with thrombospondin type 1 motif, member 13) activity, TPE timing, and early clinical outcomes.

2.2 Literature Context and Search Strategy

For transparency, the literature context for this article was informed by a targeted search of PubMed/MEDLINE and Google Scholar for publications from January 2021 to December 2025. The search terms included "pregnancy-associated thrombotic microangiopathy", "postpartum hemorrhage", "therapeutic plasma exchange", "ADAMTS13", "thrombotic thrombocytopenic purpura", and "complement-mediated thrombotic microangiopathy". Priority was given to recent reviews in hematology, nephrology, obstetric, and transfusion-medicine, as well as cohort studies and clinically relevant case reports addressing postpartum or pregnancy-associated TMA.

3. RESULTS

3.1 Illustrative Cases

3.1.1 Case 1

A 36-year-old woman at 35 weeks of gestation underwent an emergency cesarean delivery due to fetal distress and placental abruption. Intraoperative blood loss exceeded 4000 mL within 2 hours. She required massive transfusion support, including 16 units of red blood cells (RBCs), 1600 mL of fresh frozen plasma, 34 g of fibrinogen, 3000 IU of prothrombin complex concentrate (PCC), and adjunctive hemostatic therapy. Although surgical hemostasis was achieved and no major ongoing active pelvic bleeding was identified, the postoperative course remained concerning.

The patient developed persistent thrombocytopenia with a nadir platelet count of 25 × 10^9/L, hemoglobin nadir of 55 g/L, reticulocytosis, schistocytes up to 7.0% on peripheral smear, peak lactate dehydrogenase (LDH) of 1640 U/L, hyperbilirubinemia, and worsening renal function. Direct antiglobulin testing was negative. ADAMTS13 activity was 23%, which did not establish classic severe TTP but did not definitively exclude a TTP-spectrum process in a critically ill postpartum patient. Given the pattern of microangiopathic hemolysis, thrombocytopenia, and organ injury, serial TPE was initiated. The patient's clinical condition stabilized, accompanied by subsequent improvements in hematologic and biochemical parameters biochemical parameters (***Figure 1***).

3.1.2 Case 2

A 30-year-old woman at 16 weeks and 6 days of gestation was admitted with intermittent vaginal bleeding and subsequently experienced inevitable abortion, postpartum bleeding, uterine atony, and coagulopathy. The estimated blood loss was 2970 mL. Resuscitation included the administration of 8 units of RBCs, 800 mL of plasma, 2 therapeutic doses of platelets, 15 g of fibrinogen, and 1000 IU of PCC.

After abortion and initial hemostatic management, active bleeding was no longer the dominant clinical concern, yet thrombocytopenia and renal injury progressively deteriorated. Platelet nadir was 15 × 10^9/L, hemoglobin nadir was 59 g/L, schistocytes reached 6.5%, LDH peaked at 3162 U/L, and peak serum creatinine reached 334.2 μmol/L. Direct antiglobulin testing was negative. ADAMTS13 activity was 66%. Despite this value being inconsistent with severe TTP, the overall pattern remained highly concerning for postpartum TMA. TPE was initiated because progressive microangiopathic hemolysis, thrombocytopenia, and renal dysfunction could not be explained by resolved obstetric bleeding alone. The patient subsequently showed improvements in platelet count, hemolysis markers, and renal function and renal function (***Figure 2***).

3.2 Shared Clinical Signals

These two cases underscore a recurring bedside pattern a recurring bedside pattern (***Table 1***). In both women, the initial emergency was hemorrhagic; however, the subsequent clinical

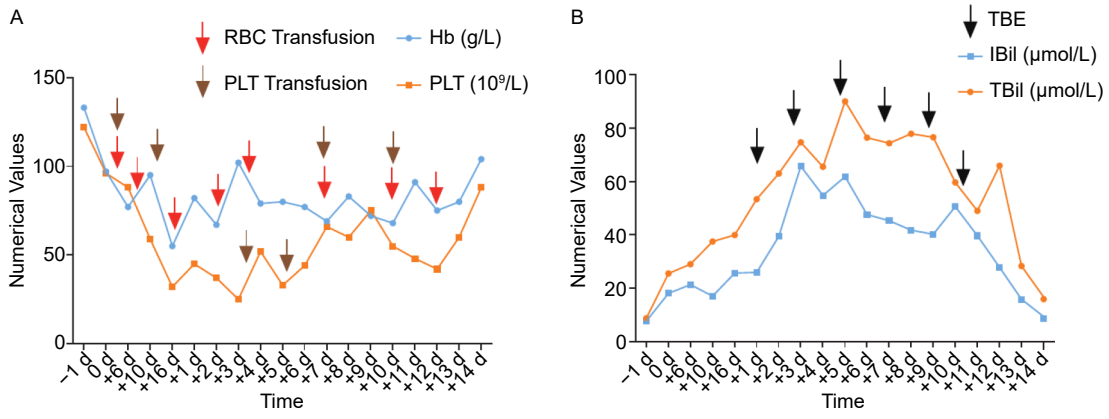

FIGURE 1 Treatment course of Case 1. (A) Serial hemoglobin (Hb, g/L) and platelet count (PLT, ×10^9/L) with red blood cell (RBC) and platelet transfusion markers. (B) Serial indirect bilirubin (IBil, μmol/L) and total bilirubin (TBil, μmol/L) with therapeutic plasma exchange (TPE) timing markers.

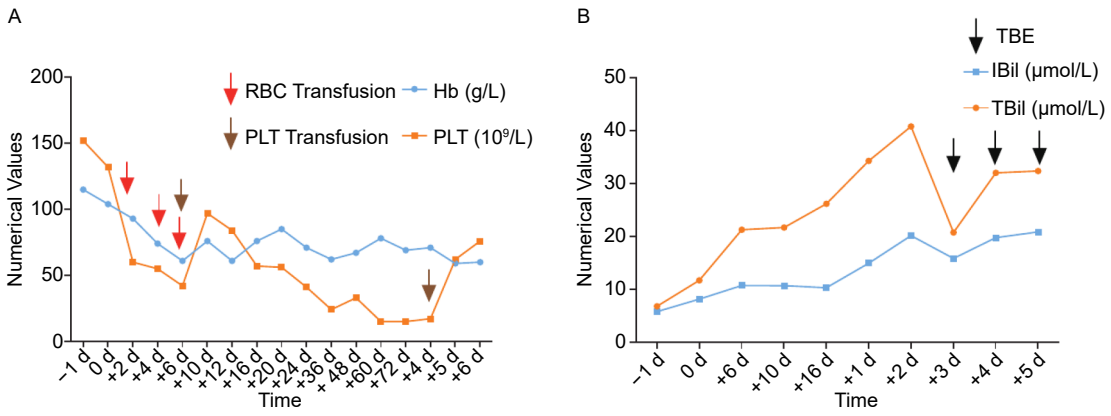

FIGURE 2 Treatment course of Case 2. (A) Serial hemoglobin (Hb, g/L) and platelet count (PLT, $\times 10^9$/L) with RBC and platelet transfusion markers. (B) Serial indirect bilirubin (IBil, μmol/L) and total bilirubin (TBil, μmol/L) with therapeutic plasma exchange (TPE) timing markers.

TABLE 1 Key clinical and laboratory features of the two illustrative patients with PPH-associated TMA.

Variable	Case 1	Case 2
Obstetric context	35-week pregnancy, emergency cesarean section for fetal distress and placental abruption	16+6-week pregnancy, inevitable abortion with postpartum bleeding
Estimated blood loss	> 4000 mL within 2 hours	2970 mL
Main transfusion support	16 U RBC, 1600 mL plasma, 34 g fibrinogen, 3000 IU PCC	8 U RBC, 800 mL plasma, 2 platelet doses, 15 g fibrinogen, 1000 IU PCC
Platelet nadir	25 × 10^9/L	15 × 10^9/L
Hemoglobin nadir	55 g/L	59 g/L
Peak schistocytes	7.0%	6.5%
Peak LDH	1640 U/L	3162 U/L
Peak serum creatinine	118.8 μmol/L	334.2 μmol/L
Direct Coombs test	Negative	Negative
ADAMTS13 activity	23%	66%
Early outcome after TPE	Clinical stabilization with hematologic improvement	Improvement in platelet count, hemolysis markers, and renal function

PPH, postpartum hemorrhage; TMA, thrombotic microangiopathy; LDH, lactate dehydrogenase; TPE, therapeutic plasma exchange; RBC, red blood cell; PCC, prothrombin complex concentrate.

course was characterized predominantly by persistent microangiopathic hemolysis, severe thrombocytopenia, and renal impairment following hemorrhage control. That combination, rather than hemorrhage itself, was the signal that shifted clinical reasoning toward pregnancy-associated TMA and justified early TPE.

4. DISCUSSION

4.1 When to Suspect TMA After PPH

A key practical insight from recent literature is that postpartum TMA should be suspected when laboratory abnormalities persist after the expected obstetric turning point. Perez Botero J *et al.* emphasized that consulting hematologists should look beyond the isolated platelet count and assess whether the overall picture is consistent with true microangiopathic hemolysis, especially when schistocytes, high LDH, low hemoglobin, and organ dysfunction develop concurrently [1]. Scully M further stressed that pregnancy-related TMA syndromes should be reconsidered whenever clinical deterioration continues after delivery, as ordinary pregnancy-associated hypertensive microangiopathy generally begins to improve once the pregnancy trigger has been removed [2].

Our two cases exemplify this teaching. In both cases, acute hemorrhage was the predominant event at presentation; however, the subsequent combination of schistocytosis, Coombs-negative hemolysis, severe thrombocytopenia, and kidney injury was disproportionate to controlled bleeding alone. This is precisely the

type of mismatch that should prompt urgent evaluation of the peripheral smear, hemolysis panel, renal indices, ADAMTS13 testing, and complement evaluation when clinically available when clinically available (***Table 2***).

TABLE 2 Practical diagnostic clues that should prompt suspicion of PPH-associated TMA.

Clinical or laboratory clue	Practical interpretation
Persistent thrombocytopenia after hemostasis	Platelet recovery should be reassessed once active bleeding is controlled; continued decline or failure to recover should prompt TMA evaluation.
Coombs-negative microangiopathic hemolysis	Falling hemoglobin with schistocytes, elevated LDH, and indirect hyperbilirubinemia suggests hemolysis beyond blood loss alone.
Renal dysfunction or oliguria	Renal-predominant injury is common in pregnancy-associated TMA and should raise concern for complement-mediated disease when severe ADAMTS13 deficiency is absent.
ADAMTS13 testing	Severe deficiency supports immune TTP; intermediate values require cautious interpretation in critically ill postpartum patients.
Complement evaluation	When available, C3, C4, soluble C5b-9, factor H/factor I-related testing, anti-factor H antibodies, and genetic analysis may support etiologic refinement.
Treatment threshold	When TTP cannot be confidently excluded, empiric plasma-based therapy and urgent multidisciplinary review should not wait for complete etiologic certainty.

PPH, postpartum hemorrhage; TMA, thrombotic microangiopathy; LDH, lactate dehydrogenase; TTP, thrombocytopenic purpura.

4.2 Differential Diagnosis in the Postpartum Setting

The postpartum differential diagnosis is broad and clinically unforgiving. DIC, HELLP syndrome, preeclampsia with severe features, and acute blood loss may initially resemble TMA, yet they do not fully account for every patient with persistent hemolysis and renal injury [2, 3]. The 2023 hematology review on labor and delivery microangiopathic emergencies emphasizes that common obstetric disorders and rare TMAs should be evaluated in parallel rather than sequentially when the patient is unstable [3]. This is particularly critical, as delayed hematology involvement can defer the initiation of life-saving therapy.

Recent nephrology reviews add another layer to the differential. Urra M *et al*. described pregnancy-associated TMA as a spectrum in which PPH, shock, hypertensive disorders, and complement activation may intersect [5]. Frimat M *et al*. similarly framed pregnancy as a susceptible biologic state in which endothelial stress, placental pathology, and complement dysregulation can converge over a short time interval [6]. This indicates that a woman with PPH may still have TTP, complement-mediated TMA, or a mixed postpartum endothelial injury syndrome; therefore, the presence of hemorrhage does not settle the diagnosis.

4.3 Interpreting ADAMTS13 and Complement Testing

ADAMTS13 activity should be interpreted in the full clinical context. Severe ADAMTS13 deficiency, particularly when activity falls below 10% with an inhibitor, strongly supports immune TTP and should trigger urgent disease-directed therapy. Intermediate ADAMTS13 activity, however, does not reliably classify postpartum TMA in critically ill patients. Inflammation, liver dysfunction, recent plasma-containing transfusion, assay timing, and pregnancy-related physiologic changes may all influence measured activity. Thus, values exceeding the severe-deficiency range reduce the likelihood of classic immune TTP but should not by themselves delay empiric TPE when the bedside pattern remains strongly suggestive of a TTP-spectrum emergency [2, 4, 9].

Complement-mediated TMA should be considered in cases of prominent renal injury, absence of severe ADAMTS13 deficiency, persistence of TMA features following hemostasis and delivery, or incomplete response to plasma-based therapy. If available, early complement assessment may include C3, C4, soluble C5b-9, factor H/factor I-related testing, anti-factor H antibodies, and complement genetic analysis. These studies are best viewed as tools for etiologic refinement and follow-up planning rather than as prerequisites for urgent treatment, because normal screening complement levels do not exclude complement-mediated disease [5–7].

4.4 Where TPE Still Fits

TPE remains central whenever TTP cannot be excluded promptly. Modern reviews continue to recommend urgent empiric plasma-based therapy in critically ill pregnant or postpartum women with suspected TTP-spectrum disease, given that delayed treatment may lead to catastrophic outcomes [2, 4, 9]. This principle remained relevant in our cases. Although neither ADAMTS13 result established classic severe TTP, both women were sufficiently ill, and the hematologic pattern was sufficiently concerning, that waiting for etiologic certainty would have been difficult to justify.

At the same time, recent literature has clarified that a response to TPE does not confirm a single pathogenesis. Kaufeld JK *et al*. studied a cohort of pregnancy-associated atypical hemolytic uremic syndrome cases and showed that PPH-associated cases may display features suggestive of complement-mediated disease, including renal cortical injury and potential benefit from short-term complement inhibition in selected patients [7]. Their work is important because it explains why postpartum TMA may initially appear TTP-like, yet later follow a renal-predominant course more consistent with complement-mediated disease. In settings where

complement inhibitors are not immediately available, TPE remains a reasonable early bridge, particularly when diagnosis is still evolving [7, 8].

4.5 What Recent Literature Adds

Recent case-based literature supports the practical value of early plasma exchange in this setting. Okawa A *et al*. reported a 2024 PPH-associated TMA case in which TPE was used successfully after the syndrome emerged from a background of severe obstetric bleeding [8]. Their report is valuable not because a single case can define practice, but because it closely resembles the clinical ambiguity encountered in real-world postpartum care: the patient initially presented with a hemorrhagic or obstetric critical-care problem, yet the later laboratory course suggested microangiopathy.

Broader review literature now reinforces the same bedside message. The 2025 comprehensive review by Alsulmi E highlighted that pregnancy itself lowers ADAMTS13 reserve and magnifies vulnerability to TTP-spectrum disease, necessitating greater reliance by clinicians on pattern recognition and rapid treatment decisions than on complete diagnostic certainty during the first evaluation [9]. Béranger N *et al*., drawing on the French experience of pregnancy-onset TTP, also demonstrated that pregnancy-related TTP is clinically heterogeneous and requires careful long-term follow-up, relapse counseling, and multidisciplinary planning in future pregnancies [10].

4.6 Clinical Implications for Transfusion and Obstetric Teams

For transfusion medicine, obstetrics, nephrology, and hematology teams, the practical algorithm is straightforward. First, achieve hemostasis and resuscitate aggressively. Second, if thrombocytopenia, hemolysis, and renal dysfunction persist following the control of bleeding, reconsider the diagnosis immediately rather than attributing all symptoms solely to blood loss. Third, obtain a peripheral smear review, repeat LDH and bilirubin, assess kidney function and urine findings, send ADAMTS13 testing if available, and consider baseline complement studies when renal-predominant or persistent TMA is suspected. Fourth, if clinical suspicion for TTP-spectrum disease remains high, do not delay empiric TPE while awaiting full clarification. Finally, once the patient is stabilized, reassess whether the course is more consistent with immune TTP, congenital TTP, complement-mediated TMA, or overlap with other postpartum syndromes. Future prospective registry studies are necessary to clarify the true proportion of complement-mediated TMA among PPH-associated TMA cases and its impact on early response to TPE.

The present article has certain limitations. It is not a formal systematic review, and the two institutional cases are retrospective and incompletely phenotyped from a mechanistic standpoint. Complement studies, long-term renal follow-up, and standardized outcome reporting were not available from the archived source material. Nevertheless, this case-based article is clinically useful as it addresses a recognizable diagnostic trap: assuming that severe hemorrhage excludes TMA.

5. CONCLUSION

PPH-associated TMA should be suspected when thrombocytopenia, schistocytic hemolysis, elevated LDH, and renal dysfunction persist after hemostasis. In this context, massive transfusion and hemorrhagic shock may obscure TMA rather than explain it completely. Recent literature supports a pragmatic approach in which repeated clinical reassessment, urgent exclusion of TTP, careful interpretation of ADAMTS13 activity, complement evaluation when clinically indicated, and timely initiation of TPE remain central to early management, while complement-mediated disease is considered during subsequent etiologic refinement and follow-up. This case-based analysis, supported by the two illustrative cases herein, may help clinicians recognize this uncommon yet dangerous syndrome earlier.

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None.

AUTHOR CONTRIBUTIONS

Jiyun Zhang and Yiming Ma reviewed the archived clinical material and drafted the manuscript. Xiaofei Li conceived the manuscript framework and supervised the work. Hui Zhang and Xiaofei Li critically revised the manuscript and served as corresponding authors. All authors approved the final manuscript.

ETHICS STATEMENT

The study was conducted according to the guidelines of the Declaration of Helsinki and approved by the Ethics Committee of Beijing Friendship Hospital (Approval No. 2021-P2-377).

INFORMED CONSENT STATEMENT

Informed consent was obtained from all subjects involved in the study.

CONFLICT OF INTEREST STATEMENT

The authors declare no conflict of interest.

DATA AVAILABILITY STATEMENT

The clinical details summarized in this article are derived from archived manuscript materials and figures supplied by the authors. Additional inquiries can be directed to the corresponding author.

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