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Case Report
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Severe Postpartum Thrombotic Microangiopathy Presenting with Purpura, Acute Kidney Injury, and Hepatic Dysfunction

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Abstract

Background: Severe thrombocytopenia in the postpartum period is a life-threatening condition requiring urgent differentiation between thrombotic thrombocytopenic purpura (TTP), disseminated intravascular coagulation (DIC), and HELLP syndrome. Early recognition significantly impacts survival.

Case Presentation: A 20-year-old previously healthy primiparous woman presented 7 days after spontaneous vaginal delivery with dyspnea, fatigue, abdominal pain, jaundice, and progressive purpuric lesions over both lower extremities. Laboratory evaluation showed severe thrombocytopenia (12,000/μL), anemia (Hb 9 g/dL), leukocytosis (22.83 $\times 10^3$/μL), acute kidney injury (creatinine 4.85 mg/dL), elevated liver enzymes (AST 233 U/L, ALT 84 U/L), hyperbilirubinemia (4.57 mg/dL), and near-normal coagulation parameters (INR 1.19). Urinalysis revealed hematuria. Ultrasound demonstrated mild–moderate ascites and increased renal parenchymal echogenicity. The constellation of findings was most consistent with postpartum thrombotic microangiopathy, highly suggestive of TTP.

Conclusion: Postpartum thrombotic microangiopathy should be suspected in patients presenting with severe thrombocytopenia, renal failure, and purpura in the absence of overt coagulopathy. Immediate plasma exchange is critical to reduce mortality.

Keywords: Postpartum thrombocytopenia, Thrombotic thrombocytopenic purpura, Thrombotic microangiopathy, Acute kidney injury, Purpura

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Introduction

The postpartum period is associated with marked hemostatic activation and endothelial changes, predisposing patients to thrombotic and hematologic complications. Severe thrombocytopenia occurring after delivery represents a medical emergency. The principal differential diagnoses include:

- Thrombotic thrombocytopenic purpura (TTP).
- HELLP syndrome (Hemolysis, Elevated Liver enzymes, Low Platelet count).
- Disseminated intravascular coagulation (DIC).
- Atypical hemolytic uremic syndrome (aHUS).

Distinguishing these entities is essential because therapeutic approaches differ significantly and delay in treatment, particularly in TTP, dramatically increases mortality.

Case Presentation

A 20-year-old primiparous woman from Eastern Ethiopia presented 7 days after an uncomplicated spontaneous vaginal delivery with: She had no prior chronic illness, hypertension, bleeding disorder, or similar previous episode.

Physical Examination

Ill-appearing, icteric.

Multiple purpuric and ecchymotic lesions over bilateral lower extremities.

Figure 1:

Figure 2:

Mild lower abdominal tenderness.

No active bleeding.

Peripheral pulses intact.

No focal neurological deficit documented.

See Figure 1 and 2.

See Table 1.

Urinalysis: Hematuria without significant proteinuria.

Ultrasound:

Mild to moderate ascites with internal echoes.

Increased renal cortical echogenicity.

Clinical Interpretation

The patient exhibited:

- Severe thrombocytopenia.
- Microangiopathic hemolytic features (anemia + indirect hyperbilirubinemia suspected).
- Acute kidney injury.
- Mild hepatic dysfunction.

Table 1:

Parameter	Result
WBC	22.83 ×10³/μL
Hemoglobin	9 g/dL
Platelets	12,000/μL
Creatinine	4.85 mg/dL
Urea	180.59 mg/dL
AST	233.65 U/L
ALT	84.85 U/L
Total bilirubin	4.57 mg/dL
INR	1.19

- Normal coagulation profile.

The preserved INR argues against overt DIC. The absence of severe hypertension and only moderate transaminase elevation makes classic HELLP syndrome less likely. The clinical constellation strongly supports **postpartum thrombotic microangiopathy**, most consistent with TTP.

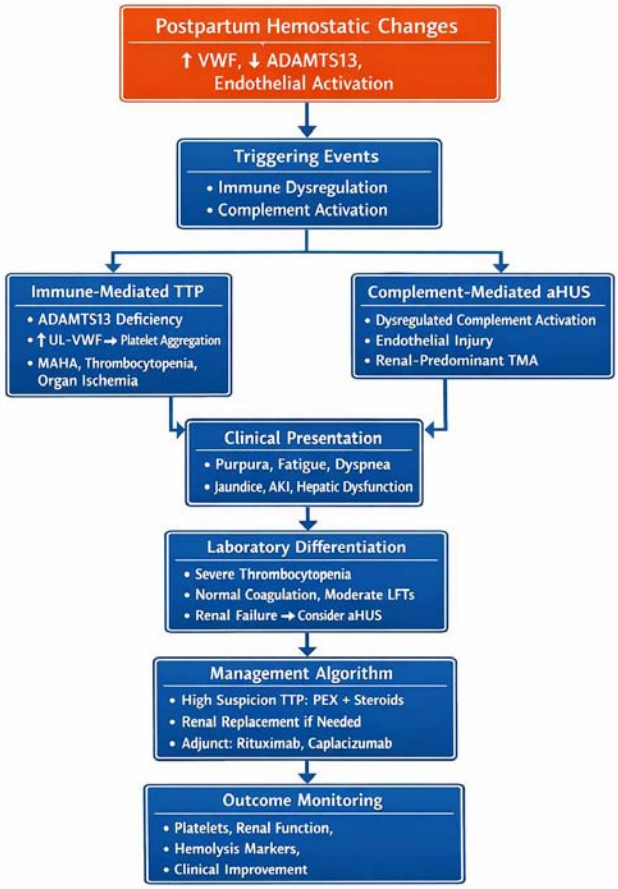
Discussion

Thrombotic microangiopathy (TMA) represents a clinicopathologic syndrome characterized by endothelial injury, platelet-rich microvascular thrombosis, microangiopathic hemolytic anemia (MAHA), and consumptive thrombocytopenia. In the postpartum period, the principal diagnostic considerations include immunemediated thrombotic thrombocytopenic purpura (iTTP), complement-mediated atypical hemolytic uremic syndrome (aHUS), HELLP syndrome, and disseminated intravascular coagulation (DIC). Distinguishing these entities is essential, as pathophysiology and treatment differ substantially.

Pathophysiologic Considerations

In immune-mediated TTP, severe deficiency of ADAMTS13 activity (<10%)—most commonly due to inhibitory autoantibodies—leads to impaired cleavage of ultra-large von Willebrand factor (UL-VWF) multimers released from activated endothelial cells. The persistence of UL-VWF promotes spontaneous platelet aggregation under high shear stress conditions in the microcirculation, particularly in arterioles and capillaries. This results in disseminated platelet-rich thrombi, mechanical red blood cell fragmentation (schistocyte formation), and end-organ ischemia. Pregnancy and the postpartum state are recognized triggers for iTTP. Physiologically, pregnancy is associated with progressive increases in VWF levels and a relative reduction in ADAMTS13 activity. In susceptible individuals, this shift in the VWF–ADAMTS13 balance may cross a critical threshold, precipitating overt microvascular thrombosis. Additionally, immune dysregulation in the peripartum period may promote the formation or expansion of anti-ADAMTS13 autoantibodies.

Complement-mediated aHUS represents a mechanistically distinct entity characterized by dysregulation of the alternative complement pathway, often due to inherited or acquired abnormalities in complement regulatory proteins (e.g., factor H, factor I, MCP). Pregnancy can act as a complement-amplifying condition, leading to uncontrolled complement activation, endothelial injury, and secondary platelet activation. Compared with TTP, aHUS more commonly present with predominant renal involvement and less severe thrombocytopenia.



Flow Chart: Demostrating Pathophysiology of Ttp.

See Flow Chart 1.

Diagnostic Differentiation in the Postpartum Setting

The differentiation between TTP, HELLP syndrome, and DIC remains challenging due to overlapping laboratory features such as thrombocytopenia, elevated lactate dehydrogenase, and hemolysis. However, several clinical and laboratory characteristics assist in discrimination:

Severity of thrombocytopenia: Profound thrombocytopenia (<30,000/μL) strongly favors TTP.

Timing: Persistence or worsening of thrombocytopenia and hemolysis after delivery argues against HELLP syndrome, which typically improves postpartum.

Coagulation profile: Normal or near-normal PT and aPTT support TTP, whereas DIC is associated with prolonged coagulation parameters and hypofibrinogenemia.

Neurologic involvement: Fluctuating neurologic symptoms are more characteristic of TTP.

Renal predominance: Severe renal failure with moderate thrombocytopenia may suggest aHUS.

In high-resource settings, ADAMTS13 activity testing confirms TTP. However, in many regions, including our setting, such assays are unavailable or delayed. Therefore, clinical prediction tools (e.g., PLASMIC score) and early empiric therapy remain critical to reducing mortality.

Therapeutic Implications

The mortality of untreated TTP historically exceeded 90%. Plasma exchange (PEX) revolutionized outcomes by removing circulating anti-ADAMTS13 autoantibodies and replenishing functional ADAMTS13. Early initiation of PEX—prior to confirmatory testing—is recommended when clinical suspicion is high. Adjunctive corticosteroids suppress autoantibody production, and in refractory or relapsing cases, rituximab targets B-cell–mediated autoimmunity. Caplacizumab, a nanobody targeting the A1 domain of VWF, inhibits platelet–VWF interaction and reduces time to platelet normalization, though availability is limited in many low-resource settings. In contrast, complement inhibition with eculizumab is the cornerstone therapy for aHUS. Therefore, accurate differentiation has direct therapeutic implications.

Clinical and Global Health Relevance

This case underscores the importance of recognizing postpartum TMA as a hematologic emergency requiring immediate intervention. In resource-limited environments where ADAMTS13 testing and complement studies are unavailable, clinicians must rely on pathophysiologic reasoning and clinical pattern recognition. The postpartum period should be viewed as a prothrombotic and immune-modulating state capable of unmasking latent susceptibility to TTP.

Early empiric plasma therapy based on clinical suspicion may be life-saving and should not be delayed pending confirmatory diagnostics. Increased awareness among internists, hematologists, and obstetricians is essential to reduce preventable maternal morbidity and mortality.

Differential Diagnosis Considerations

HELLP syndrome

Usually associated with hypertension or preeclampsia More marked transaminase elevation Typically occurs antepartum or immediately postpartum.

DIC

Prolonged PT/INR.

Low fibrinogen.

Widespread bleeding tendency.

aHUS

Prominent renal failure.

Complement dysregulation.

Less severe thrombocytopenia compared to TTP.

Given the laboratory profile and timing, TTP remains the most probable diagnosis.

Management Considerations

In suspected TTP, treatment should not be delayed for ADAMTS13 confirmation.

- Recommended interventions include:
- Immediate plasma exchange (PEX).
- High-dose corticosteroids.
- Platelet transfusion only if life-threatening bleeding.
- Renal replacement therapy if indicated.

Early intervention is life-saving.

Conclusion

This case highlights the importance of recognizing postpartum thrombotic microangiopathy in patients presenting with severe thrombocytopenia and acute kidney injury in the absence of coagulopathy. In resource-limited settings, clinical suspicion alone must guide urgent initiation of plasma exchange. Early recognition significantly improves survival.

Declarations

Ethical Approval and Consent: Informed consent was obtained from the patient for publication.

Conflict of Interest: None

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