Review

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Thrombotic thrombocytopenic purpura in pregnancy: a comprehensive review

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Abstract: Thrombotic thrombocytopenic purpura (TTP) is a rare but life-threatening hemolytic disorder with severe implications during pregnancy, characterized by microangiopathic hemolytic anemia (MAHA), severe thrombocytopenia, and systemic microvascular thrombosis. The condition arises from a severe deficiency in the ADAMTS13 enzyme, whether congenital or acquired, leading to the accumulation of ultra-large von Willebrand factor (vWF) multimers and widespread platelet aggregation. Pregnancy itself exacerbates TTP due to physiological reductions in ADAMTS13 activity, necessitating a high degree of clinical vigilance. This review addresses the etiology, diagnostic challenges, clinical presentation, and management of TTP in pregnancy, focusing on clinical relevance and emphasizing the importance of prompt therapeutic plasma exchange (TPE) and interdisciplinary care to optimize maternal and fetal outcomes.

Keywords: ADAMTS13 deficiency; therapeutic plasma exchange; microangiopathic hemolytic anemia; pregnancy-related thrombotic thrombocytopenic purpura

Introduction

Thrombotic thrombocytopenic purpura (TTP) is a life-threatening blood disorder marked by systemic microvascular thrombosis, which can lead to significant maternal and fetal complications during pregnancy. Characterized by severe thrombocytopenia and microangiopathic hemolytic anemia (MAHA), TTP results from a critical deficiency of the ADAMTS13 (a disintegrin and metalloproteinase with thrombospondin type 1 motif, member 13) enzyme. ADAMTS13 is an enzyme that cleaves ultra-large von Willebrand factor (vWF) multimers, which prevents excessive platelet aggregation. Severe ADAMTS13 deficiency leads to the accumulation of vWF, resulting in microvascular thrombosis and TTP [1, 2].

TTP has an estimated annual incidence of approximately four cases per million individuals, with a higher prevalence observed in women [3, 4]. Pregnancy is estimated to account for the presenting episode of TTP in 10–30 % of the cases [5]. During pregnancy, physiological reductions in ADAMTS13 enzyme activity further heighten the risk of TTP. As a result, pregnancy can also provoke adult-onset TTP in women with congenital ADAMTS13 deficiency, emphasizing the importance of early diagnosis and management. Moatti-Cohen et al. reported that congenital TTP (cTTP) accounts for 24 % of pregnancy-related TTP cases, a significantly higher proportion than the <5 % observed in non-pregnant populations [5]. In Saudi Arabia, the prevalence of TTP in pregnancy is similar to international rates, though recent and specific regional data is limited [6]. Managing TTP in pregnancy is particularly challenging due to overlapping clinical features with other thrombotic microangiopathies, such as preeclampsia and hemolysis, elevated liver enzymes, and low platelets (HELLP) syndrome, necessitating early recognition, accurate diagnosis, and a multidisciplinary approach to improve outcomes. Before the introduction of therapeutic plasma exchange (TPE), maternal mortality from TTP was as high as 90 %, highlighting the catastrophic nature of the disease when inadequately treated [1].

This review compiles the latest evidence and guidelines on TTP, aiming to assist healthcare providers in managing this complex condition in pregnant women.

Etiology and pathophysiology

TTP can be either congenital (cTTP) or acquired/immune (iTTP). cTTP, also known as Upshaw-Schulman syndrome, is due to mutations in the ADAMTS13 gene, resulting in a deficiency of the ADAMTS13 enzyme [7–9]. Symptoms can start in childhood but might not appear until adulthood,

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including during pregnancy. On the other hand, iTTP, which is more common, is often linked to autoimmune conditions where antibodies block ADAMTS13 activity. Triggers can include infections, medications, and certain cancers [9].

ADAMTS13 is a metalloprotease enzyme that cleaves ultra-large vWF multimers, thereby preventing excessive platelet aggregation and pathological thrombus formation. In TTP, severe ADAMTS13 deficiency, whether congenital or acquired, leads to the accumulation of ultra-large vWF multimers, promoting widespread microvascular thrombosis. This results in consumptive thrombocytopenia and MAHA, hallmarks of TTP pathophysiology [9].

ADAMTS13 activity gradually declines during a normal pregnancy, reaching approximately 50 % of its baseline levels from the second trimester through delivery. While reduced ADAMTS13 activity, typically ranging from 20 to 40 %, is also seen in conditions such as preeclampsia, eclampsia, HELLP syndrome, and hemolytic uremic syndrome (HUS) associated with pregnancy, it generally remains above detectable thresholds. In contrast, pregnancy-related TTP is defined by a marked deficiency in ADAMTS13 activity (<20 %), which often presents during the second or third trimester [3].

Clinical features and pregnancy complications

The typical triad of TTP includes MAHA, thrombocytopenia, and neurological symptoms (such as confusion, headaches, seizures, or stroke). These features often accompany renal involvement and fever, forming a pentad, although the full pentad is not always present [7].

Pregnancy-related TTP often presents with symptoms and clinical features that overlap with thrombotic microangiopathies (TMAs) pregnancy-specific conditions (e.g., preeclampsia, HELLP syndrome), making diagnosis challenging [5, 10]. Clinicians should consider TTP in cases of TMA presenting before 20 weeks of gestation or when severe neurological symptoms or cardiac injury are present.

Table 1 summarizes the maternal and fetal complications associated with a diagnosis of TTP during pregnancy. Maternal complications include severe thrombocytopenia, microangiopathic hemolytic anemia, and potential multiorgan failure, often accompanied by hypertensive disorders such as preeclampsia, which can lead to significant maternal morbidity and mortality. Fetal complications are characterized by preterm delivery, fetal distress, and, in severe cases, stillbirth or neonatal death. These adverse outcomes highlight the critical need for vigilant monitoring, prompt diagnosis, and effective management of TTP in pregnancy to optimize maternal and fetal prognoses [10].

Table 1: Maternal and fetal complications associated with a TTP diagnosis during pregnancy.

Complication	Details
Maternal	- Severe thrombocytopenia
	- Microangiopathic hemolytic anemia
	- Multiorgan failure
	- Hypertension
	- Preeclampsia
	- Maternal mortality
Fetal	- Preterm birth
	- Fetal distress
	- Stillbirth
	- Neonatal death

Differential diagnosis

Given the overlapping clinical features of TTP, HUS, disseminated intravascular coagulation (DIC), and preeclampsia/HELLP syndrome, a systematic diagnostic approach is essential for timely and accurate differentiation. TTP should be suspected in the presence of severe thrombocytopenia, MAHA, neurological symptoms, and normal coagulation tests [11]. In contrast, preeclampsia and HELLP syndrome are characterized by hypertension, liver dysfunction, and other organ involvement, with or without proteinuria [12]. DIC is more likely when coagulopathy, evidenced by prolonged prothrombin time (PT) and activated partial thromboplastin time (aPTT), low fibrinogen levels, and severe thrombocytopenia, is present [13]. HUS, distinguished from TTP by normal ADAMTS13 activity, predominantly presents with acute renal failure and may exhibit extrarenal manifestations [14].

However, immune thrombocytopenia (ITP) is characterized by isolated thrombocytopenia without hemolysis or significant organ dysfunction, necessitating the exclusion of other TMAs [15]. This structured diagnostic framework facilitates the early identification of pregnancy-associated thrombotic disorders, enabling timely intervention and improved maternal-fetal outcomes. Table 2 summarizes key clinical and laboratory differences among pregnancy-associated causes of thrombocytopenia and hemolysis, allowing clinicians to systematically narrow potential diagnoses.

Diagnosis

When TTP is suspected, an initial workup (as summarized in Table 3) should include ADAMTS13 activity testing. Pregnancy-related TTP diagnosis is supported by ADAMTS13 activity

Table 2: Differential diagnosis of thrombocytopenia in pregnancy.

Condition	Key clinical features	Key laboratory findings
TTP	MAHA, thrombocytopenia, neurological symptoms (confusion, headache, seizures), fever, renal involvement (variable)	Severe thrombocytopenia, microangiopathic hemolytic anemia (schistocytes on smear), ADAMTS13 activity <10 %
Preeclampsia/HELLP syndrome [12]	Hypertension (BP≥140/90 mmHg), proteinuria, headache, visual disturbances, right upper quadrant pain, severe cases progress to eclampsia	Elevated liver enzymes (ALT, AST), hemolysis (low haptoglobin, high LDH), thrombocytopenia, proteinuria
DIC [13]	Systemic thrombosis, secondary to obstetric complications (placental abruption, sepsis, amniotic fluid embolism)	Prolonged PT/aPTT, low fibrinogen, elevated D-dimer, thrombocytopenia, microangiopathic hemolysis
HUS [14]	Acute kidney injury (more severe than in TTP), hypertension, neurological symptoms (less common)	Severe thrombocytopenia, microangiopathic hemolytic anemia, normal ADAMTS13 activity, elevated creatinine
ITP and gestational thrombocytopenia [15]	Isolated thrombocytopenia without hemolysis, mucosal bleeding, no hypertension	Isolated thrombocytopenia, normal hemolysis parameters (LDH, bilirubin, haptoglobin)

Table 3: Comparative laboratory findings in suspected pregnancy-related TTP and differential diagnoses.

Test	Findings in TTP	Findings in differential diagnoses
Platelet count	Severe thrombocytopenia (<50,000/μL)	↓ in TTP, DIC, HUS, preeclampsia/HELLP, normal in ITP
Peripheral blood smear	Schistocytes (fragmented RBCs)	Schistocytes present in TTP, HUS, DIC, absent in ITP, preeclampsia/HELLP
Lactate dehydrogenase (LDH)	Elevated (due to hemolysis)	↑ in TTP, HUS, DIC, HELLP (normal in ITP)
Bilirubin	Elevated indirect bilirubin	↑ in TTP, HUS, DIC, HELLP, normal in ITP
Haptoglobin	Low (due to hemolysis)	↓ in TTP, HUS, DIC, HELLP, normal in ITP
ADAMTS13 activity	<10 % (Diagnostic for TTP)	Normal in HUS, DIC, preeclampsia/HELLP, ITP
Creatinine	Variable (mild elevation possible)	Markedly ↑ in HUS, variably ↑ in DIC, preeclampsia/HELLP
Coagulation tests (PT, aPTT, INR)	Normal	Prolonged in DIC, normal in TTP, ITP, HUS, preeclampsia/HELLP
D-dimer	Normal or slightly elevated	Markedly ↑ in DIC, elevated in preeclampsia/HELLP
Fibrinogen	Normal	↓ in DIC, normal in TTP, ITP, HUS, preeclampsia/HELLP

below 10 %. However, due to the extended turnaround time for the ADAMTS13 assay, plasma exchange treatment should begin immediately if TTP is suspected, without waiting for confirmatory results [9]. Once TTP is confirmed by ADAMTS13 activity below 10 %, further testing for ADAMTS13 antibodies is recommended. Patients with cTTP will not have detectable antibodies, and genetic testing should be performed to confirm cTTP when necessary [9, 16]. Genetic testing is also recommended for patients experiencing their first episode of TTP during pregnancy [11]. Patients with ADAMTS13 levels of 20 % or higher and no detectable ADAMTS13 antibodies should be evaluated for other causes of thrombocytopenia and microangiopathic hemolytic anemia, such as HUS [16].

Neurological imaging, including MRI or CT scans of the brain, is usually normal and should not be delayed if neurological symptoms are present. Similarly, renal ultrasounds are typically normal and may be used to rule out structural renal abnormalities.

When the maternal condition allows, assessing fetal well-being through ultrasound to rule out fetal growth restriction (FGR) is crucial. TTP is an acute event that typically does not involve a chronic placental process, unlike preeclampsia, where fetal FGR is more common. The presence or absence of fetal compromise, along with gestational age, may guide clinical decisions regarding intervention and delivery timing.

Management

Management of pregnant patients with TTP should involve a collaborative approach involving maternal-fetal medicine experts, hematologists, intensivists, and specialists in transfusion medicine or nephrology. The first-line treatment for TTP is TPE, which removes autoantibodies and replaces ADAMTS13. TPE should be initiated as soon as possible, ideally within 4–8 h of suspicion, even before confirming ADAMTS13 levels or genetic testing results. Daily TPE is continued until clinical remission is achieved, with normalization of platelet counts and LDH levels, followed by maintenance TPE every two weeks throughout pregnancy and the puerperium [17–19]. Prednisone or methylprednisolone is used alongside TPE to reduce autoantibody production [17–19].

Although TPE is considered life-saving, obstetrical factors must be taken into account. The most significant side

effect of TPE in pregnancy is hypotension, which can lead to fetal distress in approximately 1.1 % of cases [20]. These instances are typically transient and can be managed by maintaining maternal blood pressure with saline infusion. Ensuring adequate maternal intravascular volume and positioning the patient in a left lateral position during procedures helps prevent inferior vena cava compression by the uterus in late pregnancy [20].

During pregnancy, immunosuppressive therapy is generally limited to calcineurin inhibitors, rituximab, and azathioprine. While rituximab has been used in sporadic cases of pregnancy-related TTP without significant adverse maternal or fetal effects, it is important to note that rituximab crosses the placenta, especially in the third trimester [21, 22]. As such, the evidence supporting its safety during pregnancy remains limited, and its use should be carefully considered. Caplacizumab, an anti-von Willebrand factor nanobody, should be reserved for life-threatening, refractory cases that do not respond to standard therapies due to potential bleeding risks. [23, 24]. Delivery may become necessary in cases of refractory pregnancy-related TTP when maternal or fetal compromise occurs or when immunosuppressive therapy cannot be further delayed. Obstetric management should carefully consider the timing of delivery to balance maternal and fetal outcomes. Red blood cell transfusions should be used for severe anemia, and platelet transfusions should be avoided except when urgently needed for obstetric procedures, with plasma exchange administered concurrently to minimize risks of clotting and worsening neurological symptoms [25].

Once remission is achieved, the pregnancy can continue, provided that maternal and fetal conditions remain stable. Maternal status should be closely followed with regular clinical assessments and biochemical testing, including monthly evaluations of ADAMTS13 activity. Fetal well-being should be monitored through serial ultrasound examinations. There are currently no established international guidelines on the timing of delivery in pregnancy-related TTP. However, considering the risks of neonatal prematurity with early delivery and maternal complications such as preeclampsia with delayed delivery, early-term or near-term delivery is often deemed appropriate for cases in remission. In contrast to preeclampsia and HELLP syndrome, where delivery often improves maternal outcomes, immediate delivery is only indicated in TTP for refractory cases or significant fetal compromise.

Prognosis

Prompt and appropriate diagnosis and treatment have significantly improved the prognosis for TTP. However, maternal mortality remains a concern, particularly in resource-limited settings. The introduction of TPE and newer therapies has reduced mortality rates to less than 20 % [17, 26]. Long-term maternal outcomes may include a risk of relapse, especially during subsequent pregnancies, and the potential for chronic kidney disease in severe cases [26].

Management of subsequent pregnancies

Management of subsequent pregnancies in women who have recovered from pregnancy-related TTP remains complex and requires meticulous pre-pregnancy planning. Evidence from the Oklahoma TTP-HUS Registry, which included 16 pregnancies in 10 women with severe ADAMTS13 deficiency, demonstrated a relatively low recurrence rate of iTTP, with relapses occurring in only two pregnancies (13 %) postpartum. However, the incidence of preeclampsia was significantly elevated at 31 %, compared to the general population rate of 2.1–3.2 %, highlighting the substantial risk of hypertensive complications [27]. Other studies have documented higher relapse rates of up to 50 %, emphasizing the variability in outcomes based on patient-specific factors [19]. The risk of relapse is strongly influenced by ADAMTS13 activity levels early in pregnancy, with lower levels (<20 %) indicating a higher risk. In cTTP cases, plasma prophylaxis is essential from around 10 weeks of gestation and should be continued throughout pregnancy to maintain a normal platelet count and prevent hemolysis.

Preventive strategies are recommended, including low-dose aspirin (LDA) and low-molecular-weight heparin (LMWH) for pregnancies with a history of TTP. The UK TTP Registry documented that LDA and LMWH effectively reduce the risk of preeclampsia and thrombotic complications, contributing to improved pregnancy outcomes [19]. Additionally, observational data from 382 cases suggest that 81 % of pregnancies resulted in the birth of healthy infants, emphasizing the potential for favorable neonatal outcomes with vigilant monitoring and comprehensive management [19, 26, 27].

Pregnancy counseling should emphasize ADAMTS13 activity monitoring and individualized preventive measures. Preventive immunosuppressive therapy, such as rituximab, may be indicated in cases of acquired TTP [19, 26]. Multidisciplinary collaboration among hematologists, obstetricians, and maternal-fetal medicine specialists is crucial to optimize both maternal and fetal outcomes. Furthermore, for cTTP cases, genetic counseling is vital, especially in consanguineous marriages, to discuss the risks of disease inheritance.

Conclusions

Pregnancy-related TTP remains a complex clinical challenge, with outcomes dependent on early diagnosis, prompt therapeutic intervention, and vigilant monitoring. The risk of maternal complications, including preeclampsia, organ dysfunction, and disease relapse, underscores the need for a multidisciplinary approach to optimize both maternal and fetal outcomes. TPE remains the cornerstone of treatment, with immunosuppressive agents playing a crucial role in preventing recurrence. However, achieving a balance between maternal treatment efficacy and fetal well-being is critical, particularly in minimizing the risks of preterm delivery and FGR. Given the overlapping features of TTP and other TMAs, such as preeclampsia and HELLP syndrome, differential diagnosis remains a key priority to ensure appropriate and timely interventions.

Despite advancements in management, TTP during pregnancy necessitates individualized care, particularly in women with cTTP. LDA and LMWH have demonstrated potential in reducing thrombotic complications and improving pregnancy outcomes. However, further research is required to refine treatment protocols and develop evidence-based guidelines tailored to pregnancy. Given the rarity of the disease, establishing standardized clinical recommendations will require large, multicenter studies to overcome the inherent logistical, ethical, and financial barriers associated with studying rare pregnancy-related disorders. Additionally, the heterogeneity in disease presentation and variability in treatment response highlight the need for further investigation into risk stratification models and predictive biomarkers to guide clinical decision-making.

Optimizing maternal and fetal outcomes necessitates ongoing evaluation of prevention and treatment strategies, including the effectiveness of LDA and LMWH, refinement of TPE protocols, and the role of emerging targeted therapies such as rituximab and caplacizumab in pregnancy. Moreover, genetic counseling is crucial for women with cTTP, particularly in populations with high rates of consanguinity, to guide reproductive planning and assess inheritance risks. Advancing knowledge in this field requires international collaboration to generate high-quality data and address the challenges posed by pregnancy-related TTP.

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