Case Report

Immune thrombocytopenic purpura in late pregnancy complicated by premature rupture of membranes and cesarean delivery: a case report and review of the literature

Abstract

Thrombocytopenia represents a common hematological abnormality during pregnancy, affecting up to 10% of pregnant women, ranging from the clinically benign to processes that can threaten both mother and fetus.

While gestational thrombocytopenia accounts for the majority of cases, immune thrombocytopenic purpura (ITP) represents a less frequent but clinically significant condition due to potential maternal hemorrhagic complications, particularly during delivery.

Among pathological causes, immune thrombocytopenia (ITP) consists relatively rare, occurring in fewer than 0.1% of pregnancies, yet it remains the most common cause of thrombocytopenia in the first trimester.

ITP is an autoimmune disorder characterized by IgG-mediated platelet destruction and impaired platelet production. Although many affected women are asymptomatic, severe thrombocytopenia may increase the risk of maternal bleeding, particularly during delivery and the postpartum period, making management essential. In recent years, significant advances have been made in the treatment of ITP, but management in pregnancy is further complicated by the potential impact on the fetus, as maternal antiplatelet antibodies may cross the placenta.

Therefore, any proposed intervention must carefully balance maternal benefit against potential fetal risk. Given these complexities, optimal care requires close multidisciplinary collaboration among hematologists, obstetricians, and anesthesiologists to enhance diagnostic clarity, individualize treatment strategies, and ensure safe peripartum management for both mother and neonate.

Aim of our study consists presentation of pregnancy surveillance accompanied with ITP, in order to depict optimal maternal and fetal outcome.

Keywords: pregnancy, immune thrombocytopenic purpura, thrombocytopenia

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Introduction

Immune thrombocytopenic purpura (ITP) is an autoimmune hematologic disorder characterized by antibody-mediated destruction of platelets. In pregnancy, ITP represents one of the most common causes of isolated thrombocytopenia, accounting for approximately 3–5% of cases of thrombocytopenia in pregnant women, second only to gestational thrombocytopenia.[1,2]

The diagnosis of ITP during pregnancy is primarily one of exclusion and relies on the presence of isolated thrombocytopenia (platelet count <150,000/μL) in the absence of other secondary causes. Management becomes particularly challenging in late pregnancy and the peripartum period, where platelet thresholds influence decisions regarding mode of delivery, neuraxial anesthesia, and hemorrhage prevention strategies.[3]

Although vaginal delivery is generally preferred, cesarean section may be required for obstetric indications. The presence of severe thrombocytopenia increases the risk of postpartum hemorrhage, surgical bleeding, and complications related to anesthesia.[4] Despite the availability of international guidelines, management remains individualized, especially in cases presenting with severe thrombocytopenia at term.[5] We present a case of late-pregnancy ITP complicated by premature rupture of membranes and failure of labor progression, followed by a review of current evidence regarding maternal management.

Pathophysiology of ITP in pregnancy

ITP is mediated by IgG autoantibodies targeting platelet membrane glycoproteins, leading to splenic clearance and impaired thrombopoiesis.[6] Pregnancy-induced immune modulation may influence disease activity, resulting in variable platelet trends throughout gestation.[7] The differential diagnosis of thrombocytopenia in pregnancy includes gestational thrombocytopenia, hypertensive disorders, HELLP syndrome, and thrombotic microangiopathies.[8,9] In this case, early-onset thrombocytopenia, severity, prior diagnosis, and family history favored ITP.

Case presentation

A 21-year-old primigravida (G1P0) at 36 weeks of gestation presented to the obstetric emergency department with spontaneous rupture of membranes. On admission, laboratory evaluation revealed a platelet count of 51,000/μL, mildly reduced hemoglobin, and an elevated white blood cell count, consistent with pregnancy-

related leukocytosis. Coagulation parameters were otherwise within normal limits, and there was no clinical evidence of disseminated intravascular coagulation, preeclampsia, HELLP syndrome, or infection. The pregnancy had been monitored regularly and was reportedly uncomplicated. The patient had a known diagnosis of idiopathic thrombocytopenic purpura but was unaware of her current platelet levels at presentation. Her medical history included long-term corticosteroid therapy (Aldecort), iron, and magnesium supplementation during pregnancy, and a positive family history of ITP in her father.

Patient underwent every ten days' hematologic evaluation with measuring of platelet levels. Multidisciplinary approach regarding assiduous cooperation of gynecologist, pathologist and hematologist succeeded platelet levels approximately 100,000-110.000/mL, levels capable of labor induction. Every ten days was performed laboratory examinations in order reach optimal therapeutic mapping.

Labor was initially managed expectantly. Epidural analgesia was performed without any further complications.

Despite adequate uterine contractions, there was failure of labor progression. Given the obstetric indication and the presence of severe thrombocytopenia, a decision was made to proceed with cesarean section. The procedure was performed without complications. After placental delivery, 2g of tranexamic acid was administered prophylactically, followed by maintenance dosing of 1 g every 6 hours**,** as prophylaxis against postpartum hemorrhage to minimize the risk of postpartum hemorrhage. Intraoperative blood loss was within normal limits. The postoperative course was uneventful. The patient remained hemodynamically stable, with no signs of excessive bleeding.

Patient delivered via caesarian section a female fetus 2680gr and Apgar score 9 in the first minute and 10 in the fifth minute.

Forty-eight hours postpartum, oral corticosteroid therapy with prednisolone (two tablets three times daily) was initiated to address suspected immune-mediated thrombocytopenia. Anticoagulant prophylaxis was administered according to institutional protocol. The patient was discharged in good condition with instructions for close hematological follow-up.

Hopefully patient responded to cortisone agents without use of immunoglobulin or other pharmaceutic agents. Our Clinic in cooperation with the Hematology Department are following useful guidelines (Table 1).

Table 1 2013 practice guide in thrombocytopenia in pregnancy

First line therapy	Oral corticosteroids-initial response 2-14 days' peak response 4-28
	IVIg-initial response 1-3 days' peak response 2-7 days
Second line therapy	Combined corticosteroids and IVIg
(for refractory ITP)	Splenectomy (second trimester)
Third line therapy	
Relatively contraindicated	Anti-D Immunoglobulin
	Azathioprine
Not recommended but use in pregnancy	Cyclosporine A
	Dapsone
	Thrombopoietin receptor agonists
	Campath-1H
	Rituximab
Contraindicated	Mycophenolate mofetil
	Cyclophsphamide
	Vinca alkaloids
	Danazol

Discussion

Severe thrombocytopenia during pregnancy presents a diagnostic and treatment challenge. Gestational thrombocytopenia is usually mild and discovered incidentally, while platelet counts below 70,000/μL indicate conditions like ITP or other underlying issues.[2,5,10] This case underscores the importance of routine lab monitoring and thorough family history assessment, especially when antenatal diagnosis is not available.[11]

Managing ITP in pregnancy requires a personalized approach, balancing the risk of maternal bleeding with potential treatment side effects. Corticosteroids remain the first-line treatment, especially for symptomatic patients or those with platelet counts below 50,000/μL near delivery.

Prednisolone is preferred due to limited placental transfer. In this case, increasing corticosteroid therapy postpartum was appropriate to prevent further platelet decline.[10,12,13]

Tranexamic acid plays a proven role in reducing postpartum hemorrhage risk and was correctly used here. Although not specific to ITP, ant fibrinolytic therapy decreases blood loss without raising thrombotic risk when used carefully.[14,15]

Performance of a cesarean section should be based on obstetric indications rather than platelet count alone. Current evidence indicates that vaginal delivery is generally safe in most ITP cases, provided platelet levels are adequate. However, in this patient, failure to progress justified surgical intervention. Interestingly, ITP patients can still be at risk of thromboembolic events, especially postpartum and during corticosteroid therapy.[16] Prophylactic anticoagulation should be tailored individually, weighing the risks of bleeding and thrombosis risks.[8,17–19]

In our opinion, the management of immune thrombocytopenic purpura during pregnancy should prioritize dynamic clinical assessment rather than rigid adherence to platelet thresholds alone**.** This case demonstrates that favorable maternal outcomes can be

achieved even in the presence of significant thrombocytopenia when management is timely, multidisciplinary, and individualized.

Early involvement of hematology specialists is essential, particularly in patients with known ITP who approach term.

Regular monitoring of platelet counts in late pregnancy may prevent unexpected findings at delivery and allow for proactive therapeutic adjustments.

We believe that tranexamic acid should be considered more systematically in high-risk obstetric patients with ITP**,** especially following cesarean delivery, given its demonstrated safety and efficacy in reducing postpartum hemorrhage. Furthermore, the postpartum period should not be underestimated. Escalation of immunosuppressive therapy, combined with careful thromboembolic risk assessment, is crucial to prevent both bleeding and thrombotic complications (Table 2).[20–22]

Table 2 ITP in pregnancy: Diagnostics and therapeutic in 2024. Hematology Am Soc Hematol Educ Program 2024

	Trimester 1	Trimester 2	Trimester 3	Delivery	Fetal/Neonatal Considerations
When to treat	* Platelet <20,000/μL * Clinical bleeding * Surgery/Procedures, platelet <50,000/μL		* Platelet <20,000/μL * Clinical bleeding * Preparing for delivery when platelets are < target (usually starting at week 35/36)	**Platelet Targets:** Minimum (any mode of delivery) – 50,000/μL Epidural – 70,000/μL	Fetal thrombocytopenia is possible and may require active management after delivery * vaginal delivery is possible * goal to avoid forceps/vacuum assisted delivery * measure neonate platelet count at delivery
CBC Monitoring	Every 4 weeks (or with clinical bleeding)		Every 1-2 weeks (or with clinical bleeding)	Presentation for labor	
First line therapy	IVIG	Prednisone, IVIG	Prednisone, IVIG (Second line therapies *may be required pending clinical response*)	* Plan to taper active therapies as possible once postpartum *Platelet transfusion if clinically significant bleeding	 * Persistent neonatal thrombocytopenia is possible in nursing mothers with chronic ITP * Consider the potential for drugs used to treat maternal ITP to be expressed in breast milk

Conclusion

ITP consists a relative rare obstetric condition requiring constant surveillance. Multidisciplinary approach is mandatory in order to ensure optimal maternal and fetal outcome.

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Conflicts of interest

All authors declare any financial interest with respect to this manuscript.

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