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Poster Number: EP 373 Name: MONIKA

Title: FROM CRISIS TO CURE: NAVIGATING THROMBOTIC THROMBOCYTOPENIC PURPURA IN PREGNANCY

INTRODUCTION

ADAMTS13, a liver-produced protease, cleaves von Willebrand factor , preventing accumulation of ultra-large multimers, spontaneous platelet aggregation, microvascular thrombosis, thrombocytopenia, and microangiopathic haemolytic anaemia.

CASE

24 year old, Mrs. XYZ, G2P1D1 with 20 weeks of gestation, with diagnosed case of TTP in previous pregnancy in 2019, came with a fresh CBC report of (Hb-6.9 g/dl, TLC- 10* 10^9/L, Platelet-43* 10^9/L) as per advice of local physician, with no clinical signs and symptoms.

The patient had a history of multiple FFP transfusions and an emergency C-section in a previous pregnancy due to meconium-stained fluid and fetal distress; the infant died of meconium aspiration syndrome. In this pregnancy, she was admitted, transfused with 1 PRC and 8 FFP, started on T. Thyroxine 75ug, T. Prednisolone 20mg OD, T. Azathioprine 50mg OD. Routine antenatal investigations and TIFFA scans were normal.

WEEKS OF GESTATION	CLINICAL SIGNS AND SYMPTOMS	CBC	VALUES	TRANSFUSION RECEIVED
23 WEEKS	Bleeding gums	Platelets (g/L)	40* 10^9	12 Pint FFP
27 WEEKS	None	Platelets (g/L)	39* 10^9	10 Pint FFP
32 WEEKS	None	Platelets (g/L)	39* 10^9	4 Pint FFP
36 WEEKS	Petechiae	Platelets (g/L)	15* 10^9	13 Pint FFP

	VALUES	NORMAL RANGE
ADAMTS13 ACTIVITY	37%	50-150%
IgG AUTOANTIBODY LEVEL	12.5g/L	<9.6g/L
LDH	1110U/L	140-280U/L
HAPTOGLOBIN	<8g/L	50-220g/L
SCHISTOCYTES	3%	<0.5%

DISCUSSION

Thrombocytopenia develops in 5-10% of pregnancies, primarily due to gestational thrombocytopenia (70-80%), hypertensive disorders like preeclampsia (20-40%), or immune thrombocytopenia. Rarely, Thrombotic Thrombocytopenic Purpura (TTP) occurs (estimated incidence1 in 25,000 pregnancies), a life-threatening condition with thrombocytopenia, haemolytic anaemia, and microvascular thrombosis caused by ADAMTS13 deficiency.

She received monthly plasma infusions and serial fetal monitoring with Doppler scans. At 37.6 weeks, she underwent an elective C-section, delivering a healthy 2840-gram baby. Postpartum, she received 4 FFP transfusions.

Most TTP cases are acquired, with autoantibodies inhibiting ADAMTS13 activity, while a minority are congenital, confirmed by genetic mutations. Pregnancy frequently triggers acute TTP episodes due to increased procoagulant factors, reduced fibrinolytic activity, loss of endothelial cell thrombomodulin, and decreased ADAMTS13. For Congenital TTP (ADAMTS13 <10%), confirm diagnosis and administer plasma infusions (10–15 mL/kg) starting fortnightly, increasing to weekly or more in later trimesters. Monitor for worsening (platelet drop, LDH rise); PEX may be required for severe cases. Induce vaginal delivery at 36–37 weeks, continuing plasma infusions for 4–6 weeks postpartum.

For Acquired TTP, use immunosuppressants during episodes and consider Rituximab before pregnancy (6–12 months remission advised). Monitor CBC monthly and ADAMTS13 levels once per trimester. Delivery is near term if stable; worsening may require premature termination. For ADAMTS13 <20%-25%, use corticosteroids or PEX if needed. For ADAMTS13 >20%-25%, monitor closely. Vaginal delivery at term for both, with postpartum CBC and ADAMTS13 checks. Low-dose aspirin or thromboprophylaxis is reserved for prior placental complications or thrombotic risk. Diagnosis during pregnancy is challenging as it mimics pre-eclampsia, HELLP, eclampsia, or haemolytic uraemic syndrome.