College of Human Medicine
MICHIGAN STATE UNIVERSITY

Triggered by Pressure: Hypertension as the Likely Cause of Postpartum Thrombotic Microangiopathy

Max Scholl, MS3 & David Calderhead, MD
Department of Internal Medicine, Corewell Health Grand Rapids, Michigan

Introduction

Thrombotic microangiopathy (TMA) is a syndrome of microvascular injury characterized by endothelial damage, microthrombi, microangiopathic hemolytic anemia, thrombocytopenia, and organ dysfunction.

Primary TMA can be caused by thrombotic thrombocytopenic purpura (TTP) in the setting of ADAMTS13 deficiency or atypical hemolytic uremic syndrome (aHUS) triggered by stressors such as postpartum state. However, a secondary cause such as hypertension in the setting of chronic kidney disease (CKD) is an important etiology to consider in patients without ADAMTS13 deficiency such as this patient.[1]

Case Description (CD)

This case involves a two-week postpartum 31-year-old female with history of chronic kidney disease (CKD) stage 4, heart failure with preserved ejection fraction, poorly controlled hypertension with prior kidney biopsy showing evidence of TMA who presented to the emergency department (ED) with uncontrolled hypertension.

Initial blood pressures were in the 180s/100s which was treated with intravenous Labetalol. She had accompanied stable dyspnea on exertion along with intermittent headaches. Laboratory results supported hypertension-driven TMA as there was evidence of acute kidney injury on CKD with a creatinine of 3.38 from baseline of 2.51, elevated lactate dehydrogenase of 337 reflecting microangiopathic hemolysis, and elevated urine protein/creatinine ratio of 1.12 suggesting hypertensive kidney injury. Furthermore, AST/ALT, and bilirubin were normal which argues against HELLP syndrome.

CD Continued

Following treatment with Labetalol and Nifedipine, her blood pressure returned to baseline and her AKI resolved with subsequent discharge home.

Clinical Diagram

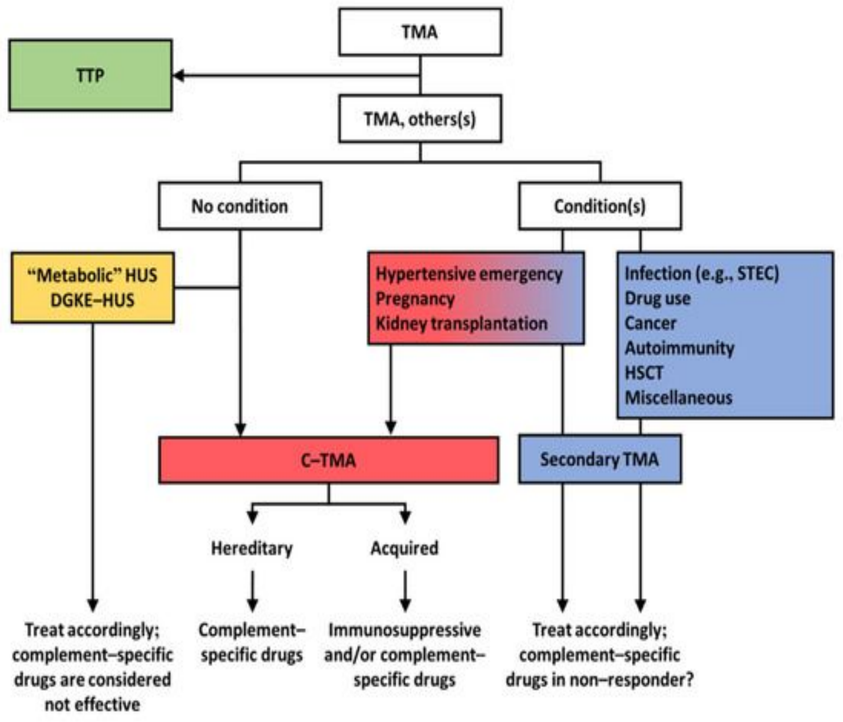


Figure 1: Approach to TMA[2]

Discussion

This case highlights the importance of distinguishing between hypertension-induced TMA from other postpartum TMAs such as TTP, aHUS, or HEELP syndrome.

Although there are overlapping features of these conditions, several key findings in this case suggest hypertensive-driven TMA. HEELP syndrome was less likely in the setting of normal AST, ALT, and bilirubin. aHUS may occur in the postpartum state. However, this patient lacked the characteristic progressive renal failure and hemolysis. TTP was less likely in the setting of normal ADAMTS13 and no evidence of significant thrombocytopenia or neurological symptoms.[3] Her rapid improvement with blood pressure management argues against primary TMA.

In this case, the patient had poorly controlled hypertension in the setting of CKD along with laboratory evidence of microangiopathic hemolysis with improvement with antihypertensive treatment suggesting hypertension-driven TMA.[4] This exemplifies the importance of integrating history, laboratory patterns, and response to therapy in differentiating postpartum TMAs to guide appropriate management while avoiding unnecessary treatment.

References

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4. Thomas MV, Alvarez MC, Acosta MP, et al. Malignant hypertension-induced thrombotic microangiopathy mimicking atypical hemolytic uremic syndrome and thrombotic thrombocytopenic purpura. *JAAC Journals*. 2025; 31 (1).