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Post-partum Eclampsia Complicated by Cerebral Venous Thrombosis: Case Report

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Abstract

Cerebral Vein Thrombosis associated with preeclampsia is a rare phenomenon that is not fully understood and presents a potentially challenging situation for treatment and future management. This case presents a 25-year old female with a history of gestational diabetes and hypertriglyceridemia presenting three days post-partum for an eclamptic seizure complicated by cerebral vein thrombosis and HELLP syndrome. The patient's presenting symptoms were alleviated by eclampsia procedure, anti-anxiety medication, and two different types of anticoagulation. Thrombectomy was not indicated, as imaging did not suggest any large vein clotting. Future check-ins, as well as careful considerations in the event of future pregnancies, should be pursued by the physicians. Testing and gaining a better understanding of the pathophysiology behind cerebral vein thrombosis proves to be difficult as the occurrence is rare, but looking to previous literature to further an understanding of eclampsia and how it progresses in the body may help to provide a greater insight into similar cases and the approach that should be used in said cases.

Keywords

Cerebral Thromboembolism, Preeclampsia, HELLP Syndrome, Case Report

Abbreviations

HELLP: Haemolysis, Elevated Liver Enzymes, Low Platelet Count; CMP: Complete Metabolic Panel; ED: Emergency Department; MRA: Magnetic Resonance Angiogram; MRV: Magnetic Resonance Venography

Introduction

Preeclampsia is a hypertensive disorder occurring in pregnancy. It is most commonly seen after 20 weeks with rare cases occurring after delivery [1]. It affects 5-7% of pregnant women globally and is responsible for the deaths of 70,000 women and 500,000 babies

yearly. In the US, it is the leading cause of maternal death, maternal ICU admission, C-sections, and premature births. This condition is characterized by hypertension and proteinuria with possible thrombocytopenia and elevated liver enzymes. Preeclampsia can be complicated by the development of serious complications including progression to eclampsia characterized by the onset of seizures or renal and cardiovascular complications. Common risk factors for the development of preeclampsia include history of previous preeclampsia, chronic hypertension, pregestational diabetes mellitus, antiphospholipid syndrome, and obesity [2]. This case explores the diagnosis and acute management of postpartum preeclampsia with progression to eclampsia and the development of rare complications including HELLP Syndrome and cerebral venous thrombosis.

Case Presentation

25-year-old Hispanic female three days postpartum presents to the to the emergency department (ED) for complaints of worsening headaches, nausea and vomiting preceding a suspected tonic clonic seizure characterized as less than a minute of unresponsiveness, foaming at the mouth and arm and leg movement without tongue biting or incontinence. Her pregnancy and delivery were complicated by gestational diabetes, 3 episodes of elevated BP during delivery with the highest noted to be 146/87, and a headache following delivery relieved by blood patch. Patient exhibited confusion on arrival to the ED and had a second tonic clonic seizure with mouth foaming soon after admittance witnessed by ER staff. Glasgow coma score was not noted and the patient has no known preexisting seizure disorder. Physical exam revealed HR: 118, RR: 21, BP: 144/80, SpO2: 98% RA, Wt: 78.4kg. Pertinent neurological findings included confusion, mild tremors, and suspected todd Paralysis presenting as weakness in the left upper extremity. Weakness and confusion resolved completely after seizure with the patient noted to be subsequently alert and oriented x 4. Other findings included nausea, vomiting, sinus tachycardia and tachypnea. Initial finger stick showed the patient to be normoglycemic. Eclampsia protocol was initiated. Patient was administered lorazepam, 0.9% Sodium chloride, Magnesium sulfate intravenously as part of eclampsia protocol and empiric dose of ceftriaxone in case of sepsis. Initial labs ordered in ER included blood cultures, complete blood count with differential (CBC with Diff), complete metabolic panel (CMP), lactic acid reflex screen, urinalysis, computed tomography scan without contrast (CT). Significant findings from initial lab work revealed mild hyperglycemia (136), lactic acidosis (12.7), transaminitis (AST 71, ALT 91) and elevated alkaline phosphatase (227) (**Table-1**). Given the mentioned lab findings, seizures, and history of episodic elevated intrapartum BP and headaches the primary differential was Eclampsia. Physicians debated the necessity of CT due to the witnessed eclamptic seizure but decided to proceed. Initial review of the CT was noted to have findings of unclear significance. ER staff determined there was no need for immediate neurosurgical consultation.

On day two of treatment radiology and neurology review of CT revealed a 15-20mm of hyperdense region consistent with hemorrhage located in the posterior frontal white matter with no midline shift. Magnetic resonance angiogram (MRA) and Magnetic resonance venography (MRV) were ordered to rule out complications including dural venous thrombosis or aneurysm. The patient was noted to have a headache localized to the occipital region that worsened with movement and sitting up with no loss of vision, tingling and no Lhermitte's sign. Vitals were within normal limits with BP 124/70 and HR 94 with the exception of low-grade fever with maximum temperature of 38.2^{o}C. Neurologic exam from neurological consult revealed the patient to be alert, oriented with no speech deficits and grossly intact cranial nerve functions. No abnormal findings present upon motor, coordination reflex sensory and gait testing. Patient did not experience additional seizures due to magnesium sulfate infusion but neurology consult recommended continuing MG sulfate for 48 hours and initiated Maxalt (Rizatriptan) with a loading dose followed by 2g/h to prevent future seizures. Urine test was ordered to check for possible UTI. MRV results obtained on day 2 of treatment revealed right cortical vein thrombosis in the parietal area and a small infarction in the posterior frontal area with 6mm focus of hemorrhage indicative of stroke with no midline shift.

Table-1: Significant lab results are given below. Only readings outside of normal ranges or identified by physicians as clinically significant are shown

Test	Lab Values at Admittance	Lab Values at Transfer Day 3	Reference Ranges
WBC	19.3	12.6	4.5 to 11.0 × 10^9/L
RBC	2.95	2.8	4.2 to 5.4 x 10^6 cells/mcL
Hgb	8.8	8.3	12.0 to 15.5 g/dcL
MCV	91.3	89.1	80 to 100 fL
Platelets	284	272	150 - 400 x 10^9/L
Abs neutrophils	12.6	2.8	cells/microL
Abs lymphocytes	5.2	0.8	cells/microL
Abs monocytes	1.4	0.1	cells/microL
Abs basophils	0.1	0	cells/microL
PTT	20.5	-	25-40 seconds
PTT ratio	0.7	-	-
CO_2	10	25	23 to 29 mEq/L
Anion gap	28	15	16 ± 4 mEq/L
glucose	136	97	70-100 mg/dL
eGFR Non African American	85	117	>60 mL/min/1.73m^2
Magnesium	2	6.3	1.5-2.0 mEq/L
Uric acid	6	-	3.0-8.2 mg/dL
Lactic Acid	12.7	0.9	4.5 to 19.8 mg/dL
ALT	91	74	4 to 36 U/L
AST	71	34	8-20 U/L
ALK phos	227	200	20-70 U/L
LDH	401	-	140-280 U/L
UA Protein	3+	-	<0.15 g/24 h
UA RBC	3 - 5	-	4 RBC/HPF

Physician assessment indicated the primary diagnosis as seizures and cerebral vein thrombosis secondary to preeclampsia with HELLP syndrome with severe features based on MRV imaging, history of seizure and hypertensive episodes, and ALT 91, AST 71 Platelets 284. The treatment plan for continued care included: transfer to a tertiary facility for low molecular weight heparin therapy, initiation of Keppra 500mg 2x daily, iron 2x a day for anemia and kept on magnesium sulfate as precaution against further seizures with urine output monitored via foley catheter. Urine output was noted as sufficient throughout magnesium sulfate treatment. Patient was not a candidate for thrombectomy due to small vein involvement. Vitals prior to transfer were: Temp PO 36.9°C, Temp Axillary: 37.9°C, HR 80, RR 18, BP 114/75, O2: 97% on RA. No abnormal physical findings were noted on the exam.

On day three, follow up with the patient post-transfer revealed improvement after anticoagulation therapy of a full dose of Enoxaparin (30 mg IV bolus once plus 1 mg/kg SC) in the ICU and then a low molecular weight heparin drip later that same day. Follow-up noted mild fever, light sensitivity, orthostatic headache without vision loss, weakness or

numbness and issues pumping or other pain. Light vaginal bleeding was present but otherwise the patient reported feeling better. CT revealed propagation of the clot, with a slightly smaller region of hemorrhage and slightly larger region of edema in the frontal lobe, which could exacerbate or worsen the headaches, seizures, and any other neurologic symptoms. Previously ordered urine tests were positive for gram negative rods and leukocytosis was present indicating a concurrent UTI. No proteinuria was noted on urine analysis. Physicians noted general improvement with headaches still present when the patient's upper body is elevated by 30^{o} or more. On day four, patient remained on foley catheter and maintained that headaches still occurred when elevated, patient however states she was feeling better. On day five, the patient was discharged with follow-up appointments scheduled at 1-2 weeks and then again at 6 weeks. Patients Final outcome is unknown.

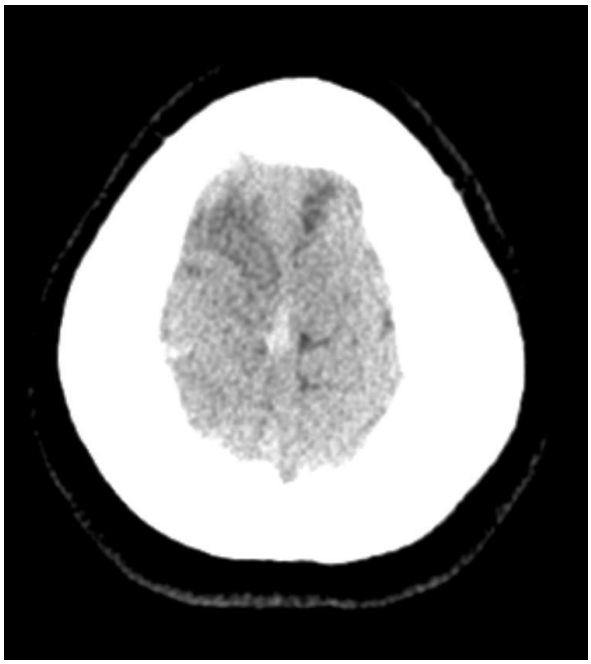

Fig-2: Axial CT showing Thrombosis in the superior sagittal sinus and surrounding cerebral vein.

Radiology Images

Day-3:

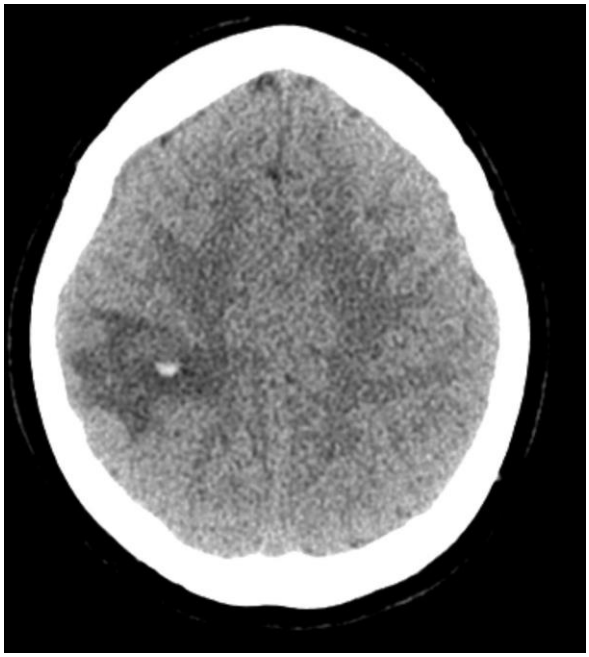

Fig-1: Axial CT showing a parenchymal focal 6mm hemorrhage w/ hyperdense region in the frontoparietal area with surrounding vasogenic edema.

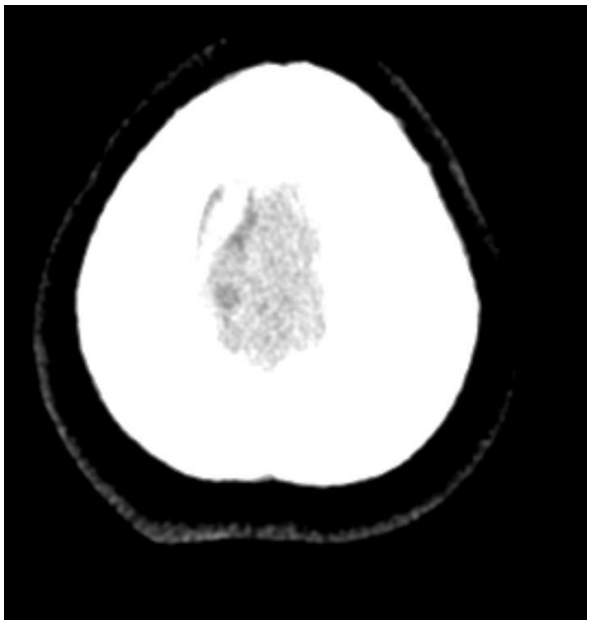

Fig-3: Axial CT showing Superior Cerebral Vein Thrombosis.

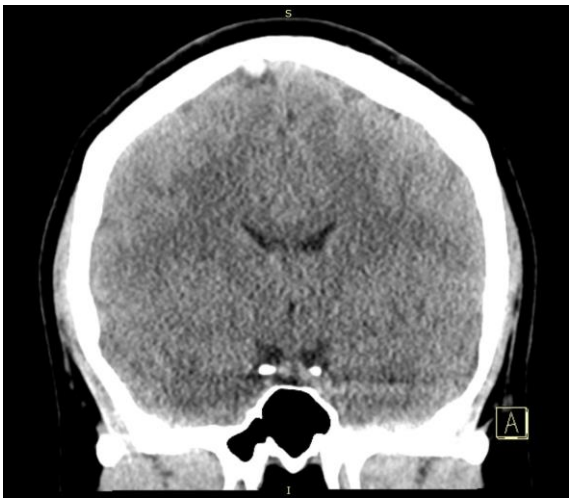

Fig-4: Coronal CT w/ Superior Sagittal Sinus Thrombosis

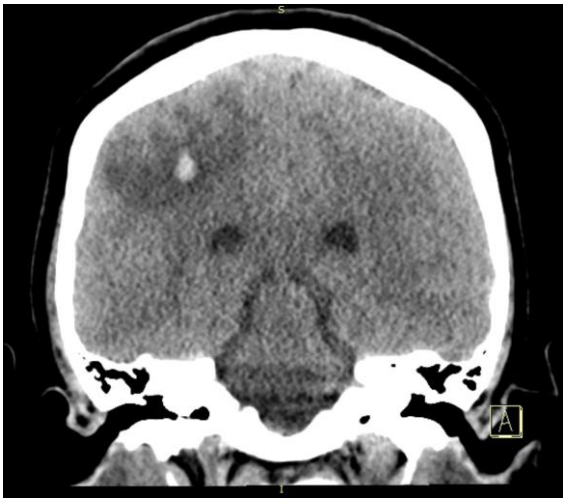

Fig-5: Coronal CT w/ Focal 6mm Hemorrhage with surrounding vasogenic edema and thrombosis in surrounding Cerebral Veins.

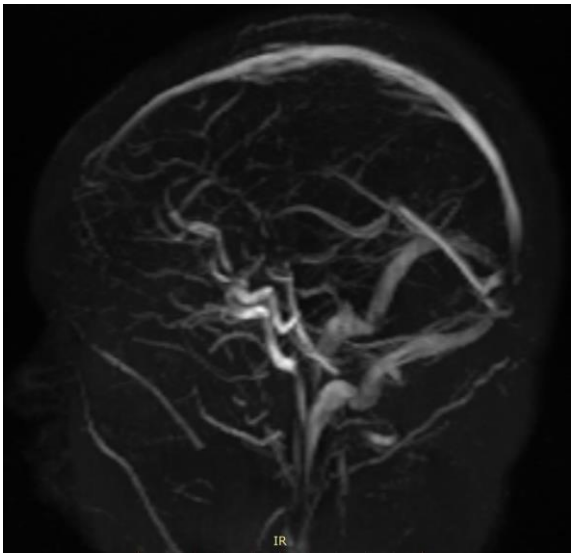

Fig-6: MR Venogram w/ Superior Sagittal Sinus Thrombosis

Discussion

Eclampsia Protocol:

There is a specific protocol in the event of an eclampsia seizure. First priority is maintaining an open airway and preventing aspiration to avoid maternal hypoxia. The patient should be rolled onto their left side to prevent aspiration and provided supplemental oxygen via a nonrebreathing face mask [3]. To prevent stroke, antihypertensive therapy is typically administered. In this case, however, due to the patient's systolic blood pressure staying in normal range, careful observation rather than immediate antihypertensives was opted for. In the event of hypertension, a β-blocker (such as Labetalol) or a vasodilator (such as Hydralazine) is typically used. [4]. Magnesium sulfate was administered to prevent further recurrence of seizures. Careful monitoring of a patient on magnesium sulfate is important as an optimal regimen has not been found [3]. 4 mg of magnesium sulfate per 100ml was administered at a rate of 300ml/hr IV x1. This is the accepted protocol for eclampsia occurrence, but patient and situational variability necessitates protocol flexibility to allow for adaptations to emergent situations as they arise such as the 1 mg of IV Lorazepam (Ativan) given for anxiety relief in this case.

Complications of Preeclampsia:

Preeclampsia is associated with complications that can occur both during and after pregnancy. In the short term, patients with preeclampsia are at risk for seizures. After the onset of seizures the condition is considered to have progressed to eclampsia. Preeclampsia is also associated with long term complications, including increased risk of various conditions including postpartum hypertension 48+ hours post-delivery, peripartum cardiomyopathy, chronic heart failure, peripheral arterial disease, coronary artery disease, cerebrovascular disease, congestive heart failure, vascular dementia, and stroke [2].

Cerebrovascular Disease in Eclampsia:

Women with preeclampsia have been found to be at increased risk of cerebrovascular dysfunction in addition to cardiovascular disease, leading to cerebral edema, seizures, stroke and maternal mortality.

Cerebrovascular involvement has been identified as a direct cause for up to 40% of maternal fatalities in pregnancy and can also result in long term cognitive changes and increased risk for further cerebrovascular accidents and long term white matter lesions. Hypertension secondary to preeclampsia may overwhelm cerebral autoregulatory processes, leading to increased blood brain barrier permeability, cerebral edema, and thus the clinical neurologic manifestations of eclampsia [5].

In imaging studies, radiographic hyperintense regions indicative of small vessel disease is often found in the cortex of patients with preeclampsia. In addition, pregnancy with preeclampsia has 4-5 times greater risk of stroke than normotensive pregnancies, and 89% of these strokes are hemorrhagic. Considering ischemic strokes are more common in general populations, preeclampsia may be associated with increased risk of hemorrhagic stroke [5].

HELLP Syndrome:

Hemolysis, elevated liver enzymes (AST, ALT, and LDH), and low platelet syndrome, also known as HELLP syndrome, is a high risk complication of preeclampsia. It can be seen in 0.17-0.85% of all pregnancies and between 2-30% of preeclamptic pregnancies. The majority of cases present between weeks 27-37; however, approximately 20% occur within 2 days of delivery. The Diagnosis of HELLP syndrome is confirmed with laboratory evidence confirming hemolysis such as the presence of schistocytes, haptoglobin under 25mg/dL or elevated LDH more than 2x reference ranges, or the presence of severe anemia. Other findings required for the diagnosis of hellp syndrome include AST or ALT elevated over 2x reference ranges and platelets under 100,000 cells/ microL [6]. It is important to note that while the clinical presentation, elevated liver enzymes and anemia present suggests HELLP syndrome her platelet counts do not fit the diagnostic criteria. Pregnancies with preeclampsia and HELLP syndrome have an increased risk of a variety of conditions for both the mother and the fetus. Maternal risks include pulmonary edema, renal failure, disseminated intravascular coagulation (DIC), hepatic failure, abruptio placenta and intracerebral hemorrhage. Fetal complications include growth restriction, intrauterine fetal death, premature birth, and low birth weight. HELLP was found to have 14% maternal mortality, with 45% of mortality attributed to intracranial hemorrhage [7]. Management of hypertension and bed rest can help lessen risk of complications. IV infused magnesium sulfate is vital for the prevention of eclamptic seizures and corresponding cerebral involvement. Patients further require close monitoring for DIC and may require interventions, including intubation and mechanical ventilation, antihypertensive therapy, plasmapheresis, and anticoagulative therapy over the course of treatment [7,8].

Right Cortical Vein Thrombosis:

The CT scans and MR Venograms from the case show this right cortical vein thrombosis, with thrombosis in a multivascular distribution along with hypodense focal hemorrhage and vasogenic edema. The CVT may be associated with the increased levels of clotting factors present during pregnancy which leads to hypercoagulability and the potential for endothelial damage thus fulfilling Virchow's Triad but exact mechanisms are still not clear [9,10]. The clinical manifestations of CVT are often symptoms such as seizures, headaches, and fevers among others. Based on the fact that there was small vein involvement, the patient was not a candidate for thrombectomy as per the attending physician, but periodic follow-ups were suggested to monitor propagation of the thrombus as well as the cerebral edema.

Conclusion

This case represents a fairly rare case of postpartum preeclampsia with HELLP syndrome that progressed to a right cortical vein thrombosis with subsequent seizures. This case illustrates the importance of providers being cognizant of postpartum preeclampsia. It also highlights the effectiveness and importance of an adaptable eclampsia protocol, timely magnesium sulfate administration for seizures, and ongoing patient monitoring as well as management to avoid ongoing complications. The decision to have the initial CT completed despite the high clinical suspicion that the patient's seizures were fully due a progression of eclampsia was essential to the diagnosis and prompt

treatment of the right cortical vein thrombosis.

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Conflict of Interest

The authors have read and approved the final version of the manuscript. The authors have no conflicts of interest to declare.

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