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Posterior reversible encephalopathy syndrome in pregnancy: A case report with review literature

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Abstract

Posterior reversible encephalopathy (PRES) is an uncommon condition that causes edema in the white matter of the posterior fossa of the brain along with headache, altered consciousness, visual impairment, and occasionally seizures. The diagnosis and course of treatment for a PRES case involving a patient who was 24 weeks pregnant are discussed in this article along with relevant literature.A 21-year-old patient who was 24 weeks pregnant suddenly lost her vision, had high blood pressure, hemolysis, raised liver enzymes, low platelet count, and an emergency cesarean section was used to deliver the baby. Magnetic resonance imaging (MRI) in the early postoperative phase showed vasogenic edema in the occipito-parietal region's white matter. In addition to antihypertensive drugs and cortisol, the patient received intravenous (IV) hydration treatment. She was discharged on the 10th postoperative day with his complaints and laboratory values within normal limits. In the existence of neurological symptoms in PRES disease, strong suspicion, and brain imaging are required for early diagnosis. Treatment is available before a lasting neurological disability arises with an early diagnosis.

Keywords: posterior reversible encephalopathy, pregnancy, neurological symptom, magnetic resonance imaging

1. Introduction

Posterior reversible encephalopathy syndrome (PRES) is a rare neuroradiological clinical condition that was first described by Hinchey in 1996. This syndrome includes brain capillary leak syndrome, reversible posterior leukoencephalopathy, reversible outside syndrome, posterior cerebral edema syndrome, and hypertensive Even though it is known as encephalopathy, PRES is the term that is most frequently used. In this instance, patients who arrive complaining of headaches also have altered mental status, epileptic convulsions, altered eyesight, and radiological abnormalities that point to posterior subcortical edema. Pregnancy accounts for around 6-20% of PRES syndrome cases, however, because not all cases undergo neuroradiological imaging, the real frequency of PRES syndrome is unclear. in PRES Although its origin is unknown, thrombotic thrombocytopenic Purpura is linked to acute hypertension, encephalopathy, renal failure, sepsis, autoimmune illnesses, immunosuppressive medication, and chemotherapy, as well as HIV syndrome, blood transfusions, and electrolyte imbalances. The most prevalent definition of PRES syndrome is preeclampsia with or without eclampsia during and after pregnancy (1). Although its pathogenesis is not fully understood, endothelial dysfunction is recognized as a typical mechanism for the degradation of the blood-brain barrier (2).

PRES is reversible in individuals with isolated eclampsia and does not arise spontaneously. Delays in treatment may result in irreversible brain damage in the afflicted areas (1). Because many acute neurological disorders include PRES in their differential diagnoses, brain magnetic resonance imaging (MRI) is used to make differential diagnoses with a high level of clinical suspicion. The optimal way to make a diagnosis is to use T2/fluid-attenuated inversion recovery (FLAIR) sequences on MRI to reveal hyperintense lesions in the parieto-occipital and frontal lobes (primarily at the cortical-subcortical junction), which are typically symmetrically positioned posteriorly (3).

In this article, the diagnosis and treatment process of a 21-year-old patient with a 24-week pregnancy who was diagnosed with PRES by MRI due to acute neurological findings following preeclampsia and HELLP (hemolysis, elevated liver enzymes, low platelet) syndrome, is presented in the light of literature.

2. Case Presentation

The 21-year-old female patient, who had a child with Gravida 2, was 24 weeks pregnant based on her previous menstrual period. The patient was hospitalized at the Gynecology and Obstetrics Clinic after complaining of headache and vision

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impairment and receiving a preliminary diagnosis of HELLP syndrome.

The patient was found to have a moderate overall health status, a 9 Glasgow Coma score, and a blood pressure reading of 170/110 mmHg. The patient began receiving Nidilat (nifedipine 10 mg) and intravenous (IV) magnesium sulfate (MgSO4) loading and maintenance therapy for tuberculosis at an outside facility. In the blood tests of the patient, BUN, urea, and creatinine were normal. Other parameters were as follows: albumin: 3.3 g/dL (3.5-5.2), AST: 824 IU/L (0-32), ALT: 448 IU/L (0-33), LDH: 2312 IU/L (135-214), total bilirubin: 1.6 mg/dl (0-1.2), calcium: 6.94 mg/dl (8.6-10), sodium: 121 mmol/L (135-145), potassium: 5.42 mmol/L (3.5-5.1), magnesium: 3.47 mg/dl (1.6-2.6), fibrinogen: 148.7 mg/dl (200-400), hemoglobin 13.7 g/dL (12.5-16), hematocrit: 40.9% (37-47), platelet: 77000 (150000-400000), white blood cell (WBC) 9.5x103/uL (4-10.5), C-reactive protein (CRP): 30 mg/L (0-5), complete urinalysis (UA) 3+++ proteinuria was detected. PT, APTT, and INR were normal. The ultrasound revealed a viable fetus at 24 weeks gestation, a breech presentation, a placenta fundus location, and adequate fluid. The patient was diagnosed with HELLP syndrome, and an emergency procedure was scheduled. Under spinal anesthesia, a single live female infant, measuring 29 cm in length, 515 grams in weight, and 21 cm in circumference around the head, was born via cesarean section (C/S abdominal). For close observation, the patient was brought to the intensive care unit. Intubation of the patient was not necessary. IV fluid therapy, IV calcium, IV Dexamethasone, IV antifibrinolytic (Herajit 250 mg), IV antihypertensive furosemide amp IV, oral 12.5 mg (Carvedilol (Beta Blocker)+, cleane 0.6 1x1, MgSO4 2 grams/hour maintenance therapy, analgesic, and IV calcium was administered to the patient. Amlodipine (5 mg) was started as a treatment. The critical care unit's departments of neurology, cardiology, and infectious diseases assessed the patient. Metronidazole 500 mg three times per day IV and Cefbactam 1 g three times per infectious disease guidelines were initiated. During the neurology evaluation, speaking, comprehension, cooperation, and consciousness were normal. Words are formed with meaningful replies, despite the difficulty. The patient did not exhibit any facial asymmetry, dysarthria, lateralizing impairment, or extremity-jerking epileptic episodes. The pupils were isochoric. The patient had diffusion magnetic resonance imaging (MRI) and cranial computed tomography (CT), with a tentative diagnosis of PRES.

A cardiology consultation was also requested for the patient to control her blood pressure. BP: 135/90 mmHg; pulse rate: 125 beats per minute for the patient. The patient received Dilatrend 12.5 mg 1x1 on recommendations from the field of cardiology. On the second surgical day, the patient was transferred from the intensive care unit to the gynecology and obstetrics clinic. The patient's vital signs were clear: TA was 140/80, pulse was 95 beats per minute, and overall condition was mild. Cardiology, neurology, and infectious disease guidelines were followed for administering IV antibiotics, IV Prednol, IV hydration therapy, low-molecular-heparin, and antihypertensives (Furosemide amp; oral 12.5 mg Carvedilol (Beta Blocker) + 5 mg Amplodipine (Monovas)). The cerebral CT scan revealed no signs of bleeding (Fig. 1).

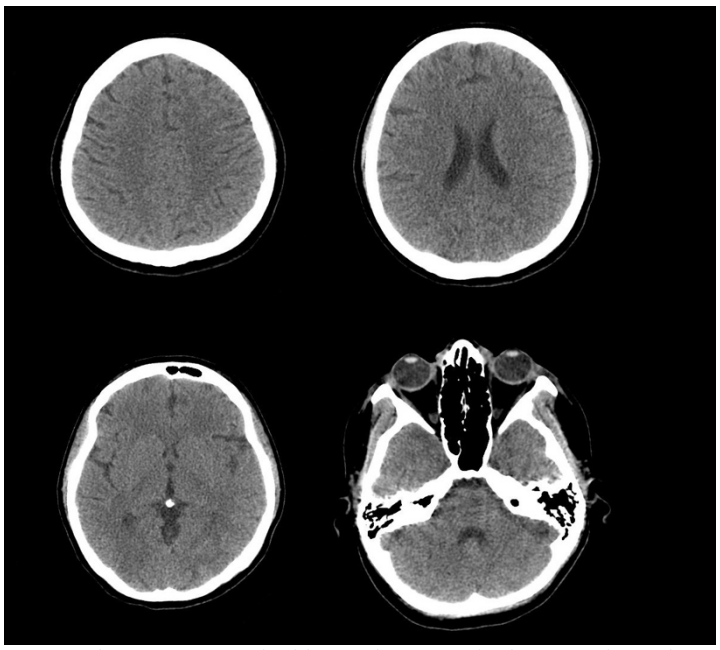

Fig. 1. There was no cerebral hemorrhage seen in the non-enhanced computed tomography scans

In cranial diffusion MRI, there were increases in the T2-WI/FLAIR signal at the parahippocampal level, more pronounced on the left, and the T2-WI/FLAIR hyperintense signal in the temporo-occipital deep white matter and the right internal capsule posterior crus (Fig. 2).

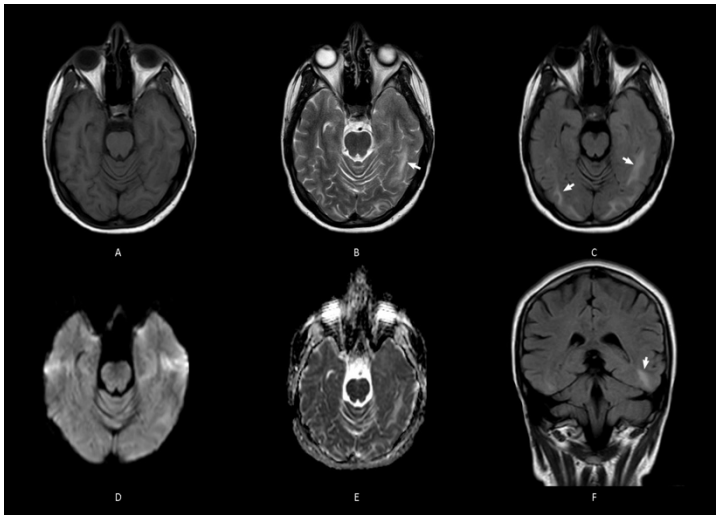

Fig. 2. Shows cranial magnetic resonance imaging demonstrating parieto-occipital region PRES findings. Axial (A), coronal (C), and axial (F) plan FLAIR sequences (arrows); (B) hyperintense signal property on T2-WI; (A) hypointense on T1-weighted imaging (WI). Diffusion weighted imaging (D) and apparent diffusion coefficient images (E) did not exhibit any diffusion restriction

Three days have passed since the surgery. Overall health was good, and consciousness was evident. It measured 135/90 mmHg for blood pressure (BP) and 95 beats per minute for pulse. Along with cranial MRI results and BP follow-ups, the patient was consulted once again with the ward's neurology and cardiology departments. It was advised to continue taking oral 12.5 mg Carvedilol, stop taking Monovas, and check BP during the cardiology visit. The neurology consultation recommended electrolyte monitoring, magnetic resonance venography (MR venography), and continuation of the existing course of treatment. Vital signs and overall health were monitored for the patient. On the fourth and fifth postoperative days, the overall condition was satisfactory. Oral intake is possible, and consciousness is evident. Following BP checks, average readings were 130/80 mmHg, the average pulse was 85 beats per minute, and room air po2 was 97. Liver enzyme levels were found to be relatively normal. As directed by neurology, the patient had an MR venography evaluation, and the results showed that the venous structures were normal. It was determined that the left transverse sinus was hypoplastic (Fig. 3).

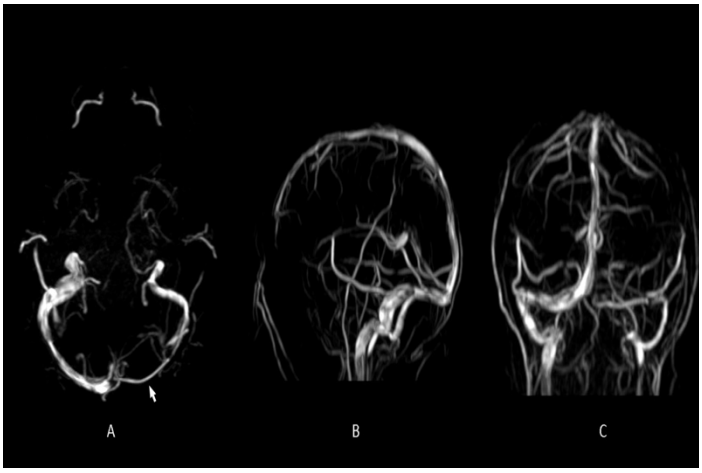

Fig. 3. (A) sagittal, (B) coronal, and (C) axial plans. The superior sagittal sinus, sinus rectus, galena vein, internal cerebral vein, right transverse sinus, bilateral sigmoid sinuses, and internal jugular veins were all shown to be patent on MR venography pictures. A hypoplastic left transverse sinus was noted (arrow)

During the follow-up with the patient, vital signs were stable and BP values were measured within normal limits. The patient's laboratory results on the 7th postoperative day were 15 IU/L for AST and 51 IU/L for ALT, respectively. The patient's laboratory results on the 7th postoperative day were 15 IU/L for AST and 51 IU/L for ALT, respectively. Fever: 34 C, BP: 140/90 mmHg, pulse: 95/min. Regarding the release, the patient had another consultation with neurology. A follow-up visit to the outpatient clinic was advised upon discharge, as neurology had no further recommendations. On the 11th postoperative day, the patient's overall condition was good; awareness was clear; BP: 130/80 mmHg; pulse: 90/min; fever: 36.9 °C; AST: 25 IU/L; ALT: 40 IU/L; and CRP: 21 mg/L were measured. Antihypertensive medication was administered to the patient. On the 11th postoperative day, the patient was discharged with her neurological problems entirely cured, and a check-up was referred to the gynecology, obstetrics, and neurology outpatient clinic.

3. Discussion

Eclampsia has been linked to several conditions, including PRES syndrome, hemolytic uremic syndrome, collagen vascular diseases, immunosuppressive medications, acute porphyria, pheochromocytoma, primary aldosteronism, thermal injury, hypercalcemia, blood transfusion, and situations involving stimulant medications like phenylpropanolamine, ephedrine, pseudoephedrine, and scorpion poisoning (4).

The neurological condition known as PRES is characterized by vasogenic edema, mainly in the parieto-occipital lobe, headache, visual abnormalities, epileptic seizures, and decreased consciousness (5). Even though pregnancy is not the main cause of PRESS, treating preeclampsia and eclampsia requires an understanding of the pathophysiology of PRESS. The pathogenesis of preeclampsia, eclampsia, and PRES syndrome share significant similarities. The pathophysiological process of PRESS has been the subject of several ideas, but it is generally associated with poor cerebrovascular autoregulation. Hyperperfusion results from rapidly increasing hypertension that surpasses the capacity of the cerebrovascular autoregulation system. Hypoperfusion is another benefit of vasospasm, which arises in hypertension to preserve brain perfusion. Endothelial damage occurs in the blood-brain barrier in both circumstances. The blood-brain barrier is disrupted by this illness, allowing plasma and macromolecules to permeate the interstitial space. Additionally, by altering the feeding arterioles' lumen diameter, certain hazardous inflammatory cytokines may indirectly compromise the blood-brain barrier. Vasogenic edema is usually the result of PRES in the parieto-occipital white matter, frontal and temporal lobes, and posterior fossa. It is unclear, therefore, why the posterior region of the brain is overly involved (1, 2). The posterior half is assumed to be more involved because of inadequate autoregulation brought on by the region's predominant vascular circulation as a result of the absence of sympathetic input. Additional theories of PRES have linked immunosuppressive therapy, particularly cyclosporine, excessive medication dosages, aluminum overload, hypomagnesemia, and hypercholesterolemia. The release of endothelin, prostacyclin, and thromboxane A2, which directly cause damage to vascular endothelial cells, are other potential pathways (4).

PRES is clinically seen with nonspecific symptoms that appear acutely within a few hours or days. Approximately 28–94% of the cases that develop encephalopathy include confusion, dizziness, and in 39%, decreased visual accuracy, diplopia, visual field disorders, cortical blindness, color

blindness, Visual symptoms such as vision abnormalities and visual hallucinations, epileptic seizures affecting 74–87% of the cases and developing within 24-48 hours, and widespread headaches are observed in approximately half of the patients. Many acute neurological conditions are included in the differential diagnosis of PRES. The key to diagnosis is high suspicion; communication between clinicians and radiologists is important (6).

Confirming the diagnosis of PRES and assessing its severity can be aided by a brain MRI scan. High-resolution CT is therefore recommended, particularly in the posterior fossa structures. However, the initial method of acute imaging is CT scanning, which is also used to diagnose PRESS. Symmetric hemisphere vasogenic edema affecting the subcortical white matter and frequently extending to the overlaying region is a characteristic seen on CT and MRI scans (6). However, unlike in the literature, it is stated that imaging methods are less sensitive than computed tomography (CT) MRI in the diagnosis of PRES, and T2/FLAIR sections are more sensitive than routine MRI sequences in detecting subcortical and cortical lesions. Similarly, Shaikh et al. stated in their study that 44% of PRES cases were diagnosed with MRI in cases with normal CT (4). In these patients, an MRI is initially useful in ruling out conditions such as cerebral hemorrhage or infarction; nevertheless, an MRI is required to confirm the diagnosis (7). The cerebral spinal fluid investigation is not required until MRI results are specific for diagnosis, and lumbar puncture should be avoided when findings indicate increased intracranial pressure (6). In the current case, the patient had simultaneous cranial CT and brain diffusion MRI scans. Although the tomography was regarded as normal, a conclusive diagnosis was confirmed using an MRI.

The treatment guidelines for PRES are primarily based on consensus, and there have been no randomized trials comparing various treatment modalities. To remove or counteract the triggering element, early identification and therapy are crucial (6). The goals of treatment for PRES syndrome are symptomatic and center around controlling or removing the underlying cause. Because plasma exchange removes systemic inflammatory mediators, it may be helpful in PRES (8). In individuals with acute hypertension, it's critical to treat the patient with IV fluids, address electrolyte imbalances, and reduce blood pressure. The ideal range for mean arterial pressure is 105–125 mmHg. First-line antihypertensive therapy medications include nimodipine, nicardipine (5–15 mg/hour), and labetalol (2–3 mg/minute). Second-line medicines include sodium nitroprusside, hydralazine, and diazoxide. Since nitroglycerin may exacerbate cerebral edema, it is not advised for PRES patients. Early delivery is crucial for people who are pregnant. Benzodiazepines, sodium valproate, levetiracetam, or phenytoin can be administered as antiepileptics in situations of status epilepsy (6, 9). In the case presented, IV fluid therapy and electrolyte therapy were started early in the patient's life.

About 75–90% of PRES cases recover completely without sequelae. Life-threatening side effects, such as hemorrhage or infarction, could, however, sporadically occur. In extreme circumstances, patients would require an intensive care unit, which could result in fatalities or long-term impairments. Three to six percent is said to be the death rate. Preventing brain damage and death by removing the etiological cause with an early diagnosis is the most crucial aspect of treating PRES. Retrospective investigations have shown that 4% of patients experience PRES recurrence, with autoimmune diseases, sickle cell crises, hypertensive episodes, and renal failure disorder being the most common causes (6,9,10).

In conclusion, PRES is a neurological disease characterized by headaches, visual disturbances, changes in consciousness, and epileptic seizures. To confirm the diagnosis in clinically suspected instances, T2-WI and FLAIR MRI sequences should be evaluated. Despite being a benign disease, there is a chance that postponing diagnosis and treatment could result in irreversible brain damage. Even though most therapies are symptomatic, it is important to begin treating the etiological cause as soon as possible

Ethical Statement

None.

Conflict of interest

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Authors' contributions

Concept: E.K.Ç, H.S., Design: E.K.Ç, H.S., Data Collection or Processing: E.K.Ç, H.S., Analysis or Interpretation: E.K.Ç, H.S., Literature Search: E.K.Ç, H.S., Writing: E.K.Ç, H.S.

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