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| RESEARCH ARTICLE

From Headache to PRES: Early Recognition of Preeclampsia in a Pregnant Patient

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| ABSTRACT

Early neuroimaging should be strongly considered in pregnant patients presenting with any neurological symptoms, even when symptoms appear minor, such as headache, as this may enable timely detection of serious underlying conditions. Delaying intervention until the onset of seizures risks missing early indicators of life-threatening complications such as Posterior Reversible Encephalopathy Syndrome (PRES), which can serve as a premonitory sign of advancing eclampsia, particularly in the setting of preeclampsia. This case report illustrates that clinical principle through the presentation of a 29-year-old Saudi woman, gravida 2 para 1, at 33+4 weeks of gestation, who arrived at obstetric triage with a three-day history of progressively worsening fronto-occipital headache. Her presentation was complicated by neurological findings including confusion and bilateral ankle clonus—hallmarks of neurological irritability—later confirmed as PRES via classic MRI features. Management involved urgent blood pressure control, administration of magnesium sulfate for seizure prophylaxis, and expedited delivery due to both high-risk maternal status and signs of severe fetal compromise on Doppler and non-stress testing. This case is unique in demonstrating how subtle early neurological symptoms can unmask severe pathology, and it underscores the critical importance of multidisciplinary vigilance in preventing maternal and fetal morbidity; it offers valuable clinical insights into the timely recognition and management of PRES in the context of hypertensive disorders of pregnancy.

| KEYWORDS

Re-eclampsia, Eclampsia, Hypertension in Pregnancy, Headache, Posterior Reversible Encephalopathy Syndrome.

| ARTICLE INFORMATION

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1. Introduction

Posterior Reversible Encephalopathy Syndrome (PRES), when occurring in the context of hypertensive disorders of pregnancy, represents a complex and multifactorial neurological complication that lies at the intersection of obstetrics, internal medicine, and neurology. Although originally described over two decades ago, PRES continues to pose significant diagnostic and therapeutic challenges in clinical practice. The overall reported incidence of PRES among hospitalized patients is approximately 0.4%; however, this incidence increases substantially in the obstetric population, particularly in cases of preeclampsia and eclampsia, where it has been estimated to affect 20–40% of severe presentations [1]. This underscores the critical need for heightened clinical vigilance and a lower diagnostic threshold in pregnant patients presenting with even subtle neurological symptoms such as persistent headache. Maternal neurological complications remain among the leading causes of maternal

mortality worldwide, and the occurrence of PRES often reflects extensive cerebral involvement, frequently coexisting with HELLP syndrome and correlating with adverse fetal outcomes, including fetal demise rates of up to 12–15% in some series [4]. PRES is classically defined as a clinicoradiological syndrome characterized by the acute onset of neurological symptoms—such as headache, visual disturbances, seizures, or altered mental status—in conjunction with MRI findings of bilateral, symmetric hyperintensities predominantly involving the parieto-occipital regions [3]. Pathophysiologically, PRES is believed to be primarily mediated by vasogenic cerebral edema, which preferentially affects the posterior circulation due to its relatively sparse sympathetic innervation, rendering it more vulnerable to triggers such as hypertensive crises, sepsis, or endothelial dysfunction [4]. In early stages, computed tomography (CT) imaging may be unremarkable; therefore, magnetic resonance imaging (MRI) is essential for differentiating PRES from other critical intracranial pathologies such as ischemic stroke or hemorrhage—particularly in pregnancy, where CT is often avoided due to concerns regarding fetal radiation exposure [3]. Despite the potential for severe complications, PRES is generally reversible with timely recognition and appropriate intervention; however, delayed diagnosis may result in permanent neurological deficits or even death in up to 15% of cases [4]. In the obstetric population, the most commonly identified risk factors for PRES include severe preeclampsia, eclampsia, HELLP syndrome, rapid fluctuations in blood pressure, and exposure to immunosuppressive agents [5]. Notably, the velocity and abruptness of blood pressure elevations, rather than absolute measurements alone, are considered the most predictive indicators of PRES development [5]. Timely diagnosis remains challenging, as initial symptoms—such as headache, blurred vision, or nausea—are often nonspecific and may be overlooked, contributing to diagnostic delays in approximately 30% of cases [7]. Furthermore, the limited availability of MRI in certain settings may hinder early confirmation [7]. While the classic imaging pattern involves the parieto-occipital lobes, atypical variants have been described in a minority of cases, involving regions such as the frontal lobes, basal ganglia, or brainstem. These atypical distributions are more frequently observed in pregnancy-related PRES and can mimic cerebrovascular accidents, thus complicating the diagnostic process [7]. Seizures are a prominent clinical feature, occurring in approximately 60–75% of patients and often representing one of the earliest neurological manifestations [2]. Fetal outcomes are closely tied to the severity of PRES and the timing of delivery; studies have reported preterm birth rates ranging from 60–80%, with neonatal intensive care unit (NICU) admission rates exceeding 50% and perinatal mortality rates of 12–15% in severe cases [4,6]. Given the central role of the placenta in the pathogenesis of preeclampsia, HELLP syndrome, and PRES, delivery remains the only definitive intervention; however, it does not invariably lead to immediate resolution of hypertension or neurological symptoms. Ongoing postpartum monitoring is therefore essential, as clinical deterioration may persist if endothelial dysfunction and cerebral injury remain unresolved [6]. Hypertensive disorders of pregnancy account for approximately 14% of maternal deaths globally, with neurological complications—particularly eclampsia and PRES—constituting a significant proportion of this mortality. As such, improved recognition and management of PRES are integral to advancing maternal health and achieving the World Health Organization's maternal mortality reduction targets.

2. Case Presentation

2.1 Patient's history and Physical Examination

This case report describes a 29-year-old Saudi female, gravida 2 para 1, at 33 weeks and 4 days of gestation, who presented to the obstetric triage unit with a three-day history of progressively worsening fronto-occipital headache. She characterized the headache as severe, dull, and aching in nature, with no relief from over-the-counter analgesics. Over the preceding 24 hours, she had developed blurred vision, particularly involving peripheral visual fields, accompanied by episodes of photopsia. Additionally, she reported the onset of nausea and vomiting on the morning of presentation. According to her husband, she had appeared lethargic and intermittently confused, prompting immediate evaluation in the emergency department. The patient denied fever, chills, neck stiffness, focal neurological deficits, dysarthria, seizure activity, recent infections, or head trauma. She also reported decreased fetal movements over the past 12 hours, associated with intermittent non-radiating right upper abdominal pain. She explicitly denied any vaginal bleeding or fluid leakage. Her obstetric history included one prior full-term, uncomplicated vaginal delivery, and she had no personal history of preeclampsia. Her past medical history was unremarkable for chronic hypertension, diabetes mellitus, renal pathology, or autoimmune disease. There was no history suggestive of migraine or seizure disorders, and she did not smoke or use alcohol or illicit substances. Her only medication was prenatal vitamins, and her family history was non-contributory. On physical examination, the patient appeared drowsy yet easily arousable, with noticeable anxiety. Vital signs revealed a temperature of 37°C, blood pressure of 176/112 mmHg, heart rate of 102 beats per minute, respiratory rate of 22 breaths per minute, and oxygen saturation of 98% on ambient air. Meningeal signs were absent on neck examination; however, fundoscopic evaluation revealed bilateral papilledema with blurring of disc margins and mild retinal hemorrhages. Cardiopulmonary and abdominal examinations were largely unremarkable. Pelvic examination was consistent with a gravid uterus measuring appropriately for 34 weeks' gestation, and there was mild right upper quadrant tenderness without guarding or rebound. Neurological assessment demonstrated a Glasgow Coma Scale (GCS) score of 14, brisk deep tendon reflexes, bilateral ankle clonus, and no focal sensory deficits. Fetal heart rate was recorded at 170 bpm with decreased variability on Doppler ultrasound. The initial clinical impression was that of severe hypertension with neurological manifestations—including headache, visual disturbances, clonus, and altered mental status—in conjunction with right upper quadrant pain, raising strong

suspicion for severe preeclampsia with impending eclampsia, complicated by possible Posterior Reversible Encephalopathy Syndrome (PRES).

2.2 Investigations:

Laboratory investigations demonstrated findings consistent with HELLP syndrome (Hemolysis, Elevated Liver enzymes, and Low Platelet count), as detailed in Table 1. Neuroimaging via magnetic resonance imaging (MRI) of the brain was performed, revealing bilateral, symmetric hyperintensities predominantly involving the parietal and occipital white matter, with additional extension into the frontal lobes. Notably, there was no evidence of restricted diffusion or intracranial hemorrhage. These radiologic features were characteristic of Posterior Reversible Encephalopathy Syndrome (PRES). Fetal assessment included a non-reactive Non-Stress Test (NST), which demonstrated minimal baseline variability and intermittent decelerations—findings suggestive of fetal compromise. Complementary Doppler ultrasound evaluation of fetal circulation revealed diminished end-diastolic flow in the umbilical artery, further indicating placental insufficiency and compromised fetal well-being.

Test	Result	Normal Range
Hemoglobin	10.8	12-16 g\dL
WBC	$8.1x10^9$	$4.0\text{-}11x10^9$\L
Platelets	$73x10^9$	$150\text{-}450x10^9$\L
Sodium	140	135-145 mmol\L
Potassium	4.5	3.5-5.0 mmol\L
Creatinine	1.4	0.5-1 mg\dL
LDH	720	140-280 U\L
AST	112	<40 U\L
ALT	92	<40 U\L
Urine protein	+3	Negative

Table 1: results of relevant laboratory investigations.

2.3 Management course

Initial stabilization was promptly prioritized, encompassing the administration of supplemental oxygen at 2 L/min via nasal cannula, establishment of intravenous access, and initiation of continuous maternal and fetal monitoring. An intravenous bolus of 20 mg labetalol was administered, followed by titrated doses totaling up to 220 mg over a 24-hour period, with the therapeutic objective of maintaining systolic blood pressure below 160 mmHg and diastolic pressure above 90 mmHg, thereby preserving adequate cerebral perfusion. Seizure prophylaxis was initiated with intravenous magnesium sulfate, consisting of a 4 g loading dose infused over 15 minutes, followed by a continuous maintenance infusion at 1 g/hour. Neurological and systemic monitoring was appropriately conducted, including regular assessment of deep tendon reflexes, respiratory rate, and urinary output. Fluid administration was judiciously restricted to 80 mL/hour to mitigate the risk of pulmonary edema associated with fluid overload. Definitive management entailed expedited delivery via cesarean section, indicated due to the presence of severe preeclampsia complicated by Posterior Reversible Encephalopathy Syndrome (PRES), HELLP syndrome, and a non-reassuring fetal status. Given the patient's thrombocytopenia, general anesthesia was selected as the safest approach. A live female neonate was delivered, with APGAR scores of 4 and 7 at one and five minutes, respectively. Neonatal intensive care unit (NICU) admission was warranted due to prematurity. Postpartum management included continuation of magnesium sulfate infusion for an additional 24 hours, initiation of oral nifedipine at a dose of 30 mg daily, meticulous neurological surveillance, and a follow-up MRI on postpartum day five, which demonstrated significant resolution of the PRES-related radiologic abnormalities.

3. Discussion

The patient's progressively worsening headache, in conjunction with visual disturbances suggestive of posterior circulation involvement and altered mental status in the setting of markedly elevated blood pressure, represents a constellation of classic red flags for Posterior Reversible Encephalopathy Syndrome (PRES), and should immediately raise clinical suspicion. Neurological examination findings of hyperreflexia and bilateral ankle clonus, elicited by the consulting neurologist, further supported the diagnosis of severe preeclampsia with evolving eclampsia, reflecting heightened neuromuscular excitability. The absence of meningeal signs such as neck stiffness, alongside the lack of fever, made infectious etiologies like meningitis considerably less likely. While non-contrast head computed tomography (CT) may be helpful for ruling out acute hemorrhage or mass effect, magnetic resonance imaging (MRI) remains the preferred modality for detecting PRES lesions, particularly given that CT may miss up to 20% of early-stage cases and carries the added concern of fetal radiation exposure [3,7]. Other differential diagnoses were systematically excluded: migraine with aura was deemed improbable given the presence of objective neurological findings such as clonus and critically elevated blood pressure; cerebral venous sinus thrombosis, intracranial hemorrhage, and ischemic stroke were ruled out based on the absence of venous filling defects, hemorrhagic signal changes, or infarcts on imaging [3]. The presence of HELLP syndrome, known to exacerbate both preeclampsia and PRES, increased the urgency of delivery due to the

risk of rapid deterioration into disseminated intravascular coagulation (DIC), necessitating the preoperative preparation of blood products and correction of coagulopathy if present [6]. In this case, epidural anesthesia was contraindicated due to thrombocytopenia, and general anesthesia was appropriately selected to avoid the risk of epidural hematoma [6]. Evidence-based guidelines for acute blood pressure control recommend avoiding excessive reductions below 140/90 mmHg, in order to preserve maternal cerebral and uteroplacental perfusion, both of which may have undergone autoregulatory adaptation to chronic hypertension. Rapid reductions in blood pressure can paradoxically precipitate cerebral hypoperfusion and ischemia [4]. Magnesium sulfate remains the agent of choice for seizure prophylaxis and neuroprotection in pregnant patients at risk of eclampsia, with the landmark Magpie Trial demonstrating a 58% reduction in seizure incidence and a 45% reduction in maternal mortality compared to placebo, owing to magnesium's stabilizing effect on cerebral vasculature and its role in reducing vasogenic edema—the central pathophysiological mechanism of PRES [4]. Nonetheless, vigilant monitoring of deep tendon reflexes, respiratory rate, and urinary output is essential during magnesium therapy, and intravenous calcium gluconate (10 mL of 10%) must be readily available for prompt reversal in cases of toxicity [6]. The decision to proceed with emergency cesarean delivery was based on a combination of maternal factors—namely, HELLP syndrome and altered mental status—and fetal indicators including a non-reactive non-stress test, late decelerations, and absent end-diastolic flow on Doppler ultrasound, all of which pointed to severe fetal compromise [4]. The definitive management of both preeclampsia and PRES remains delivery of the placenta, which is central to the pathogenesis of systemic endothelial dysfunction and cerebral involvement [6]. In this case, early MRI imaging facilitated the clinical decision to prioritize blood pressure stabilization over interventions such as thrombolysis or anticoagulation, reaffirming the pivotal role of neuroimaging in establishing diagnosis, directing treatment, and prognosticating recovery. Follow-up MRI on day five postpartum demonstrated marked resolution of the previously noted lesions, confirming the reversibility of PRES when recognized and managed in a timely fashion [7]. In general, more than 90% of patients with PRES achieve complete neurological and radiological recovery within 1–2 weeks [3,4]. Poor outcomes, observed in 10–15% of cases, are typically associated with complications such as cerebral infarction, intracranial hemorrhage, or delayed diagnosis. Prognosis is significantly worse when initial blood pressure exceeds 180/120 mmHg, HELLP syndrome is present, or onset occurs during the postpartum period [7]. Postpartum monitoring remains critical, as approximately 30% of PRES cases either emerge or worsen after delivery, largely due to rapid hemodynamic shifts and fluid redistribution resulting in hypertensive surges [7]. Future recurrence must also be anticipated, as preeclampsia recurs in 15–25% of subsequent pregnancies. This reinforces the importance of prophylactic interventions such as low-dose aspirin initiated before 16 weeks of gestation, which has been shown to reduce recurrence risk by up to 60% [4]. Compared to PRES in non-pregnant populations, pregnancy-associated PRES tends to affect younger women, typically in their 20s to early 30s, and is associated with a higher likelihood of full recovery due to its rapidly reversible nature [8,9]. In contrast, non-pregnancy-related PRES is more commonly triggered by conditions such as sepsis, malignant hypertension, or renal failure, and carries a greater risk of irreversible injury, including infarction, hemorrhage, and long-term neurological deficits [8,9]. Notably, important gaps persist in the current literature, particularly regarding long-term neurological outcomes in pregnancy-associated PRES, as well as the lack of validated biomarkers to predict the progression of preeclampsia to PRES.

4. Conclusion

Maintaining a low threshold for neuroimaging in pregnant patients exhibiting any neurological symptoms—even as subtle as a headache—may prove to be lifesaving. Clinical intervention should not be postponed until seizures manifest, as Posterior Reversible Encephalopathy Syndrome (PRES) frequently represents an early warning sign of evolving eclampsia in individuals with preeclampsia, and its timely recognition can significantly influence maternal and fetal outcomes. While computed tomography (CT) plays a vital role in the rapid exclusion of acute cerebrovascular events such as hemorrhage or ischemic stroke, magnetic resonance imaging (MRI) remains indispensable for the accurate identification of PRES and for distinguishing it from other intracranial pathologies. Despite advancements in medical management, delivery continues to be the definitive therapeutic intervention for preeclampsia; however, the decision regarding its timing must delicately balance the risks to maternal health with those posed to the fetus, particularly in the context of prematurity. Crucially, the management of preeclampsia does not culminate with delivery, as a substantial proportion of complications—including hypertensive crises, seizures, and organ dysfunction—can peak during the postpartum period, necessitating vigilant follow-up and continued monitoring. A multidisciplinary approach, involving obstetricians, neurologists, anesthesiologists, and neonatologists, is essential to ensure comprehensive care and to optimize outcomes for both mother and neonate.

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