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BRAVING THE STORM: SURVIVING PREECLAMPSIA, HELLP SYNDROME AND PRES: A CASE REPORT

Obstetrics and Gynaecology

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ABSTRACT

Aim And Background: Posterior Reversible Encephalopathy Syndrome (PRES) is a medical enigma that lies at the intersection of neurology and obstetrics. First described by Hinchey et al. in 1996, PRES is marked by a dramatic constellation of symptoms: severe headache, seizures, visual disturbances, and altered mental status. The pathophysiology of PRES is as complex as its presentation, often involving a breach in the blood-brain barrier precipitated by acute hypertension or endothelial dysfunction leading to characteristic edema, predominantly affecting the parieto-occipital regions of the brain. In this context, we delve into the case of a 19-year-old primigravida, whose journey through the triple challenges of Pre-Eclampsia, HELLP syndrome, and PRES exemplifies the essence of resilient motherhood. By exploring her case, we aim to shed light on the critical importance of early recognition and comprehensive management of PRES in pregnancy, ensuring better outcomes. **Case Description:** A 19 year old primigravida at 26 weeks of gestation presented with preeclampsia with severe features, with impending eclampsia with HELLP syndrome. She experienced bilateral lower limb weakness postpartum. Based on clinical examination and radiological imaging, she was diagnosed with PRES. Management included blood pressure control using intravenous and oral antihypertensives, anticonvulsant and neuroprotective agents. Supportive care was provided, including close monitoring of neurological status. The multidisciplinary approach involved obstetricians, neurologists, and intensive care specialists, ensuring comprehensive care. Recovery was uneventful with scheduled followup. **Conclusion:** This case underscores the critical importance of early detection of PRES in pregnant women with preeclampsia. Prompt diagnosis through clinical assessment and radiological imaging is vital for initiating appropriate treatment and averting serious complications. Although the development of PRES involves various mechanisms, effective management primarily focuses on controlling hypertension and addressing the root cause. With timely intervention, the prognosis is generally positive, but additional research is needed to thoroughly understand the pathophysiological processes and refine treatment protocols. **Clinical Significance:** Understanding PRES is vital because it can be reversible if treated promptly. With diverse range of symptoms, often associated with elevated blood pressure- pregnant women with preeclampsia are at risk. Early detection through clinical assessment and radiological imaging followed by proper management can prevent severe complications, significantly improving patient prognosis. Recognising PRES helps distinguish it from other neurological disorders, ensuring precise diagnosis and appropriate treatment, thus enhancing patient care quality. This case highlights the critical management of severe preeclampsia and the necessity of a coordinated multidisciplinary approach. The timely intervention was essential in managing maternal and fetal outcomes. The transient lower limb weakness post-delivery was notable, underscoring the importance of thorough neurological assessment in such cases. Severe preeclampsia with HELLP syndrome and IUGR presents significant management challenges requiring prompt and comprehensive care. This case emphasizes the importance of vigilant monitoring and multidisciplinary strategies to prevent adverse outcomes.

KEYWORDS

INTRODUCTION:

This case study illustrates a clinical triad comprising preeclampsia, HELLP syndrome, and posterior reversible encephalopathy syndrome, revealing the intricate interplay among these conditions. This unique scenario underscores the complexities of obstetric complications and highlights the necessity for nuanced management to ensure favourable patient outcomes.

Posterior reversible encephalopathy syndrome (PRES) is a clinicoradiologic entity characterised by a variable combination of consciousness impairment, seizure activity, headaches, visual abnormalities, nausea/vomiting and focal neurological signs, associated with a number of conditions such as severely high blood pressure, preeclampsia, eclampsia, renal failure, SLE and some immunosuppressive medications.[1,3]

While the exact cause of PRES is unclear, it typically involves endothelial damage and hypertension. The clinical and neuroradiological features are caused by vasogenic oedema or vasoconstriction-induced cytotoxic edema.[1]

Improved brain imaging has enhanced diagnosis. Although the illness is usually benign with reversible symptoms and temporary radiological findings, some cases have resulted in irreversible brain damage and death.[2] Thus, a prompt, multidisciplinary therapeutic response is thus recommended.

Case Description:

A 19-year-old primigravida at 26 weeks of gestation was referred to our hospital with a 3-day history of headache and a 1-day history of blurred vision. She had no significant past medical, surgical, or family history. Upon examination, she was well-oriented; PR:92 bpm; BP:160/100 mmHg; uterus corresponded to 20-22 weeks, relaxed, FHR present.

The patient received Injection Labetalol 20 mg IV (two doses), Tablet Labetalol 100 mg, and Injection MgSO4 IV according to the ZUSPAN regimen. Relevant investigations done (Table 1). Fundus examination was normal. Bedside ultrasound revealed a SLIUG of 20 weeks with fetal growth restriction and no retroplacental clots.

The diagnosis of severe preeclampsia with impending eclampsia, with HELLP syndrome, with FGR was made. The pregnancy was terminated using Foley's induction and Tablet Misoprostol as per FIGO guidelines. 18 hours post-induction, she delivered a single, deceased fetus weighing 590 grams. No retroplacental clots,

postpartum hemorrhage, or abruption were noted. Antihypertensive medications were continued to control blood pressure.

Following delivery, the patient had no imminent symptoms or bleeding. BP:140-150/90-100 mmHg. MgSO4 was discontinued after 24 hours, and antihypertensives were continued.

On PND–1, the patient showed no imminent symptoms or bleeding. BP was 110/70 mmHg. Investigations repeated (Table 1), and antihypertensives were tapered accordingly. On PND 2, the patient experienced sudden bilateral lower limb weakness and was unable to walk. On examination, PR:86 bpm, BP:120/80 mmHg, intact higher mental functions and cranial nerve sensations, normal muscle bulk, tone, and power (5/5 in all limbs), and normal deep tendon reflexes and plantar reflexes. Cerebellar signs were negative. An MRI brain was performed (Figure 2), and neuromedicine recommended Tablet Levetiracetam 500 mg BD, Injection Citicoline 500 mg IV BD, and Tablet Labetalol 100 mg BD.

By PND 4, the patient regained the ability to walk. She was discharged on PND 6 with oral antihypertensives (Tablet Labetalol 100 mg BD, Tablet Clinidipine 10 mg OD), oral anticonvulsants (Tablet Levetiracetam 500 mg BD), and oral neuroprotective agents (Tablet Citicoline 500 mg BD) to ensure continued management and prevention of recurrence. The patient had an uneventful recovery and follow-up.

DISCUSSION:

PRES, formerly called Reversible Posterior Leukoencephalopathy Syndrome, Reversible Posterior Cerebral Oedema Syndrome and Reversible Occipital Parietal Encephalopathy, is an acute neurotoxic syndrome initially described by Hinchey in 1996.[3]

Pathogenesis (Figure 1): Although the pathophysiology is not fully known, a number of theories have been put out, such as– cerebral autoregulation failure resulting in vasogenic oedema (most widely accepted theory), cerebral vasoconstriction, endothelial damage leading to blood brain barrier breakdown.[3]

When MAP remains above 150-160 mm Hg, cerebral autoregulation mechanisms are disrupted, resulting in elevated perfusion pressure and cerebral vessel injury. This leads to extravasation of fluid and proteins into the interstitial space, which ultimately results in vasogenic oedema.

Changes become irreversible when MAP exceeds 200mmHg.[3] However, the vasogenic theory does not account for PRES happening without hypertension and the correlation of extent of the oedema and the severity of hypertension, as seen in our case. Additionally, rather of showing cerebral hyperperfusion, several PET-based research has shown cerebral hypoperfusion.[3]

Another theory has linked PRES to a systemic inflammatory conditions, such as sepsis, eclampsia, transplantation, and autoimmune diseases, causing endothelial dysfunction. Increased levels of circulating cytokines promote leukocyte adhesion and interaction. Endothelial dysfunction and blood-brain barrier disruption cause fluid and macromolecules to extravasate into the interstitium. These processes can lead to reversible focal and diffuse abnormalities visible on angiographic investigations. Pre-existing endothelial dysfunction often worsens due to vasocontriction during cerebral autoregulation, leading to increased hypoxia and subsequent vasogenic oedema. While this theory explains the role of inflammation in endothelial dysfunction, it does not explain PRES cases without inflammation.[3]

Recent studies have also suggested that an increase in AVP secretion can cause PRES.[1,3]

Clinical picture: Common symptoms seen in obstetric patients are-seizures(45%),visual disturbances(34%),altered consciousness(19%) and focal deficits(4%).[3,4]

Symptoms usually develop rapidly within hours, peak within 12–48hours, and commonly resolve within a week, though in some cases, they may persist longer.[4]

Our patient experience PRES two days after delivery. Symptoms developed rapidly and resolved withing 36-48 hours.

Imaging studies: Lesions typically occur in the parietal-occipital lobe but can also affect the brainstem, cerebellum, anterior regions and basal ganglia.

Characteristic imaging patterns seen: Holo-hemispheric watershed, Superior frontal sulcus, Dominant parietal/occipital, Partial and/or asymmetric PRES. Our case had Hyperintensities in cortex and sub-cortical white matter of bilateral parietal and left frontal lobe with subtle diffusion restriction. (Figure 2)

Differential diagnosis: Severe neurological conditions such as Posterior Circulation Stroke (seizures typically absent); Reversible Cerebral Vasoconstriction Syndrome (characterised by thunder clap headache); Primary CNS vasculitis (symptoms are subtle,CSF: Abnormal-inflammatory); Encephalitis (aphasia, seizures, confusion); Status Epilepticus (mostly unilateral cerebral lesion); Intracranial Venous Thrombosis; Intoxication with Anticholinergic Drugs.[3]

Therefore, a multidisciplinary approach involving obstetricians, neurologists, intensive care specialists ensure comprehensive care. While PRES can be severe neurological disease, recovery is the rule, as it is a treatable condition with good prognosis if diagnosed early.

Management involves addressing the underlying causes (Figure 3). In pregnancies with severe preclampsia, eclampsia, or HELLP syndrome, timely termination may be necessary to mitigate risks. Stabilizing the patient includes administering fluids, correcting acidosis, and normalizing electrolyte imbalances.

Symptomatic treatment comprises using antihypertensive medications (such as labetalol, nifedipine, nicardipine) to manage blood pressure. For seizure management, anti-epileptic drugs like magnesium sulphate, benzodiazepines, phenytoin, or valproate are employed, with mannitol used for cerebral edema.

Coagulopathy and low platelet count are addressed through interventions like fresh frozen plasma and platelet transfusions to ensure adequate clotting function.[3,4]

TABLE 1: DAY WISE INVESTIGATIONS OF THE PATIENT

INVESTIGATIONS	On admission	PND 0	PND 1 (morning)	PND 1 (evening)	PND 2	PND 3	PND 4	PND 8
Hemoglobin (g/dL)	11.3	8.6	10.6	7.9	7.6	7.5	7.6	8.3
Platelet Count (cells/mm³)	1.1l	25,000	20,000	31,000	60,000	1.07	1.34	2.17
Total Leukocyte Count (cells/mm³)	19,000	17140	16120	16,600	30,560	21,400	15,620	14,930
Urine Protein	3+	4+						
Total Bilirubin (mg/dL)	1.1	3	1.4		0.4			
Direct Bilirubin (mg/dL)	0.1	1	0.5		0.2			
Indirect Bilirubin (mg/dL)	1.0	2	0.9		0.2			
AST/SGOT (U/L)	248	607	180		57			
ALT/SGPT (U/L)	117	138	87		58			
LDH (U/L)	819				630			
Serum Urea (mg/dL)	25	40	41		24	19	13	
Serum Creatinine (mg/dL)	0.5	0.5	0.7		0.5	0.5	0.5	
Serum Na+/K+/Cl- (mmol/L)	138/3.9/114	129/4.1/107	129/4.0/105		139/4.5/113	139/4.3/113	135/4.5/110	
PT/APTT/INR	13.1/29.1/0.96	16.1/32/1.23	15.8/31.4/1.21		16.8/33/1.29			
Dengue Serology		Negative						

FIGURE 1: PATHOPHYSIOLOGY OF PRES

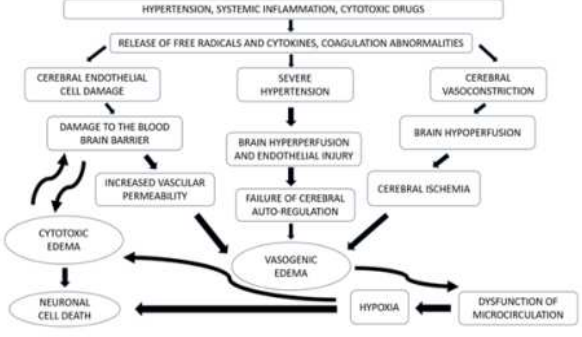


FIGURE 2: MRI FINDINGS

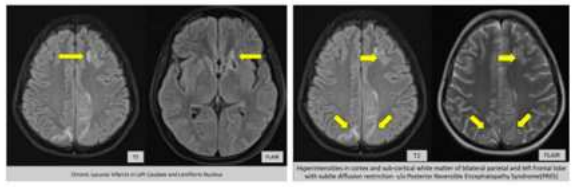
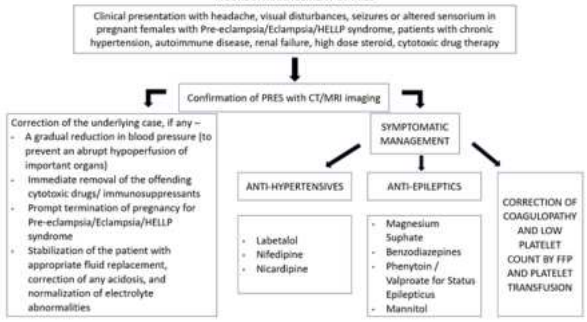


FIGURE 3: MANAGEMENT OF PRES



CONCLUSION:

In recent years, PRES has gained significant recognition, with elevated blood pressure often identified as the primary cause. Early imaging diagnosis is crucial for initiating treatment and preventing complications. Stabilization and the removal of toxic causes are essential steps in management. Treatment primarily focuses on lowering blood pressure and controlling seizures, resulting in a good prognosis when promptly administered. However, delayed or inadequate therapy can lead to poor outcomes. While PRES is commonly associated with symptoms such as headache, altered mental status, seizures, and visual disturbances, focal neurological deficits can also occur. Our patient had weakness of bilateral lower limbs, indicative of the specific regions of the brain affected by the syndrome and emphasize the importance of early and accurate diagnosis. Further research is necessary to elucidate the pathogenesis of PRES, identify diagnostic markers, and develop targeted treatment strategies. Understanding the underlying mechanisms and contributing factors will aid in the prevention and management of this complex syndrome, ultimately improving patient outcomes.

List Of Abbreviations:

PRES- Posterior reversible encephalopathy syndrome
SLE- Systemic Lupus Erythematosus
SLIUG- Single Live IntraUterine Gestation
PR- Pulse Rate
BP- Blood Pressure
FHR- Fetal Heart Rate
IV- Intravenous
$MgSO_4$- Magnesium Sulphate
HELLP- Hemolysis, Elevated Liver enzymes, Low Platelets
FGR- Fetal Growth Restriction
IGO-Internation Federation of Obstetrics and Gynaecology
PND- Post Natal Day
OD- Once daily
BD- Twice daily
MAP- Mean Arterial Pressure
AVP- Arginine VasoPressin

REFERENCES:

1. Aafreen A, Pankaj A, Pankaj A. Posterior reversible encephalopathy syndrome: a case report. Int J Reprod Contracept Obstet Gynecol 2022;11:2895-7.
2. Waghmare BV, Jajoo S, Patel DJ, Mohammad S, Muneeba S. Hemolysis, Elevated Liver Enzymes and Low Platelet Count Syndrome Complicated by Disseminated Intravascular Coagulation and Posterior Reversible Encephalopathy Syndrome: A Case Report. Cureus. 2024 Apr;16(4).
3. Hinduja A. Posterior reversible encephalopathy syndrome: clinical features and outcome. Frontiers in neurology. 2020 Feb 14;11:71.
4. Mapari SA, Shrivastava D, Bedi GN, Pradeep U. Unveiling the Hidden Culprit of Posterior Reversible Encephalopathy Syndrome Superimposing Postpartum Eclampsia: A Rare Case. Cureus. 2024 Mar;16(3).