Original Research Paper

Volume - 16 | Issue - 02 | February - 2026 | PRINT ISSN No. 2249 - 555X | DOI : 10.36106/ijar

Obstetrics & Gynaecology

POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME; RARE MANIFESTATION OF ECLAMPSIA: A CASE REPORT

Dr Gagan Lata* Department of Obstetrics and Gynecology, Adesh medical College & Hospital, Shahbad, Kurukshetra. *Corresponding Author

Dr Vartika Wadhwa Department of Obstetrics and Gynecology, Adesh medical College & Hospital, Shahbad, Kurukshetra.

ABSTRACT Posterior reversible encephalopathy syndrome (PRES) refers to white matter vasogenic edema primarily affecting the brain's posterior occipital and parietal lobes, causing acute neurological symptoms like headaches, visual symptoms, seizures, and altered mental status. We present the case of a 20-year-old pregnant female with 9 months amenorrhoea with twin pregnancy who presented with altered mental status with severe hypertension. She underwent emergency cesarean section. Postoperatively, she was diagnosed to have posterior reversible encephalopathy syndrome on MRI. She was managed with intensive care and had complete recovery.

KEYWORDS : PRES, vasogenic odema, hypertension

INTRODUCTION

Posterior reversible encephalopathy syndrome (PRES) is a neurological condition that can present acutely or subacutely with a variety of symptoms such as altered mental status , drowsiness, visual disturbances (e.g., visual hallucinations, cortical blindness, hemianopia, quadrantanopia, and diplopia), seizures (focal or general tonic-clonic), and headaches.[1]

Often, the presentation occurs in the context of acute uncontrolled hypertension, with systolic blood pressures ranging between 160 to 190 mmHg.[2] Other risk factors are preeclampsia; kidney disease (such as nephrotic syndrome, which can lead to hypovolemia and secondary hypertension via activation of the renin-angiotensin system; renal failure; liver disease; various chemotherapy agents like platinum-containing medications, gemcitabine, CHOP/R-CHOP, exposure to cytotoxic medications or immunosuppressants such as tacrolimus, sirolimus, interferon therapies as well as various immunotherapies and monoclonal antibodies like bevacizumab, pazopanib, sorafenib, and sunitinib.[3,4,5] In addition, autoimmune disorders (such as hemolytic uremic syndrome, thrombotic thrombocytopenic purpura, eosinophilic granulomatosis with polyangiitis, and systemic lupus erythematosus); or sepsis.[6][7] Of these etiologies or triggers, uncontrolled hypertension is the most common.

Based on several reports and studies, PRES affects people of all ages but is more common in middle-aged females.[8]

It is an uncommon syndrome and management and prognosis varies. Hereby, we are presenting a case of posterior reversible encephalopathy syndrome with management where patient recovered completely although recovery took more than 2 weeks.

CASE REPORT

Twenty years old primigravida presented to casuality with unknown period of amenorrhea with involuntary movements of limbs with up rolling of eyeballs and frothing from mouth and labour pains since 5 hours. As patient was disoriented and agitated, history was provided by relatives. She was totally unbooked and supervised with 9 months of amenorrhea and no documents or USG was available. She had no history of epilepsy. She was married since 1 year and had spontaneous conception. On examination, her pulse rate was 98bpm and blood pressure was 170/110 mm of Hg, RR was 22/min and sPO2 was 98% at room air. Pallor was present. Anterior abdominal wall odema and pedal odema was present. Per abdomen height of uterus was corresponding to 36 weeks with multiple fetal parts palpable. Moderate contractions were present and fetal heart could not be localised with doppler as well as on ultrasound. On per vaginum examination, Bishop score was not favourable. While examination she was repeatedly throwing the fits. Immediately all the investigations were sent, blood was arranged and patient catheterised. Her hemoglobin was 6.8g/dl, total leucocyte count was 12,000 cumm and platelet count was $111x10^9$ /L. LDH was 384 U/L. Liver function tests , renal function tests were normal and coagulation profile was normal. Urine was 2+ for albumin. She was given loading dose of MgSO4 according to Pritchard's regimen and continued till 24 hours after delivery. Injection Labetalol 20 mg was given IV. She underwent emergency cesarean section under general anaesthesia.

First twin extracted as breech, dead female foetus weighing 1.63 kg and second dead female foetus was extracted as cephalic weighing 810 grams. Liquor was thick meconium stained. One unit of PRBC was transfused in view of severe anemia. Patient was already intubated and kept in ICU care.

On POD 1, she was unconscious and not responding to pain stimuli. MRI was done on postoperative day 1, which showed posterior reversible encephalopathy syndrome. She had dilated pupils. Neurology, nephrology and ophthalmology opinion was taken. Fundus examination showed normal disc and vessels.Total bilirubin was raised to 1.2mg/dl, direct bilirubin was 0.8mg/dl. SGOT was 278 IU/L and SGPT was 74 IU/L.

POD 2 minimal response to pain stimulus was present. Serum Magnesium was raised to 9.6 mg/dl for which calcium gluconate was given although deep tendon reflexes were present and urine output was adequate.TLC raised to 30,000 cumm for which she was shifted on higher antibiotics, Meropenam and Clindamycin. She had gaseous abdominal distension. USG whole abdomen did not show any significant findings. Injection furosemide 20 mg was started 12 hourly. On POD 3, she started responding to verbal commands. Tab cabergoline 1mg was given on POD 3 by ryle's tube. Blood culture and urine culture came out to be sterile.

Patient was extubated on POD 4. She started taking orally. She started doing well and was was discharged on 18th postoperative day with complete recovery.

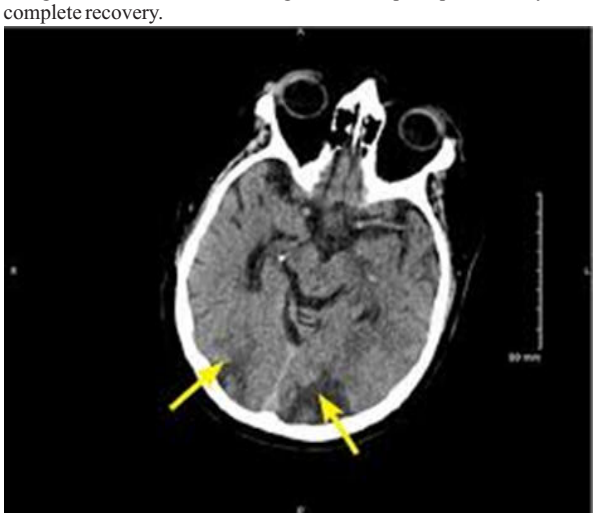

Fig. 1 MRI Showing Odema In Posterior Part Of Brain Indicative Of PRES

DISCUSSION

PRES is a rare cliniconeuroradiological condition introduced by Hinchey et al in 1996.

The name is designated to its clinical and radiographic syndrome that is inspired by (1) radiographic findings of white matter edema (i.e., hyperintense T2 signal or hypointense T1 signal on magnetic resonance imaging (MRI)), typically found in the posterior cerebrum in a symmetric fashion (although asymmetric presentations are possible); and (2) the fact that symptoms are reversible, provided that the syndrome is recognized and treated promptly. However, the name used to describe the syndrome can be misleading as the edema is not always localized necessarily to the posterior cerebral white matter as it can occur in watershed zones other than parietal-occipital regions thalamus, and sometimes in the anterior circulation.[9,10] Moreover, the syndrome is not always reversible. Some individuals can develop life-threatening complications, such as transforaminal cerebellar herniation and focal neurologic deficits, especially if prompt treatment is not initiated.[11]

PRES is a disorder of dysregulated perfusion of the brain, resulting in reversible vasogenic oedema. Severe hypertension leads to impaired cerebral autoregulation causing cerebral hyperperfusion and endothelial dysfunction, causing intravascular fluid to extravasate to the surrounding brain tissue, leading to edema.The posterior circulation is more vulnerable to hyperperfusion as it has less sympathetic innervation to counter reflex parasympathetic vasodilation. Patients with PRES commonly have acute fluctuations in blood pressure, but it is not known whether these are the cause or a secondary effect of the syndrome. Endothelial dysfunction may result, either directly or as a downstream effect, from the secretion of cytokines, including tumour necrosis factor alpha, interleukin-1, Interferon gamma and vascular endothelial growth factor.[12]

Imaging remains the gold standard in the diagnosis of PRES as it cannot be made based on clinical symptoms alone.[13] CT scan head is critical to assess neurologic emergencies that can explain an acute onset of altered mentation, headache, and seizures, such as intracranial hemorrhage. An MRI of the brain without intravenous (IV) contrast is the imaging modality of choice, looking for the presence of vasogenic edema that will appear as a hyperintense signal on T2 with the fluid-attenuated inversion recovery (FLAIR) sequences helping to improve sensitivity. This is most commonly seen in the parieto-occipital lobes—although other areas can be involved, such as the temporal lobe, frontal lobe (superior frontal gyrus), cerebellum, brainstem, and deep white matter.[14]

Obtaining blood work can help evaluate electrolyte imbalances like uremia and hypomagnesemia, hypoalbuminemia, protein deficiency, and CSF studies for underlying infection (for example, herpes simplex virus type 1 or 2, which can cause herpes simplex encephalitis), and autoimmune etiologies. Blood work can also help exclude severe hypoglycemia as a potential diagnosis. A lumbar puncture is sometimes done in immunocompromised individuals or others with clinical suspicion of infection in order to assess for the presence of encephalitis.

Obtaining an electroencephalogram (EEG) in people with PRES can be helpful. If there is persistently altered mentation in the absence of tonic, clonic, or tonic-clonic movements, an individual may have subclinical seizure activity that can be captured on EEG.[15]

Identifying, treating, and managing the underlying etiology, in addition to careful treatment of hypertension, is crucial for the management of PRES. Treatment is recommended when the blood pressure exceeds 160 mmHg/110 mmHg, with a goal of 130 to 150 mmHg/80 to 100 mmHg.[16]

A sudden or drastic reduction of blood pressure can lead to cerebral hypoperfusion and increase the risk of developing ischemia; thus, blood pressure should not be reduced by more than 25% of the initial presenting blood pressure in the first six hours.[16] Such careful acute blood pressure management may warrant a need for admission to the intensive care unit until a stable blood pressure goal is reached with the use of titratable IV antihypertensives like nicardipine, clevidipine, and labetalol.

Sometimes individuals with PRES develop life-threatening complications, such as status epilepticus or coma, and treatment and management of such complications can be done with common medications such as levetiracetam and phenytoin .[17]

Corticosteroids can improve the symptoms of PRES by reducing vasogenic edema, but can be a trigger for causing PRES in some patients taking it for asthma.[18] If PRES is attributed to an immunosuppressant like bevacizumab, pazopanib, sorafenib, and sunitinib, a reduction of the dose or substitution of the agent is recommended.[19]

The prognosis of PRES is typically favorable if recognized and treated early, with symptom improvement or resolution in a few days to several weeks. Visual symptoms often completely resolve, especially with early treatment of PRES, although residual visual deficits can remain at 3 to 4 months after onset.[20]

CONCLUSION

Posterior reversible encephalopathy syndrome is rare manifestation of eclampsia. Although reversible yet can lead to mortality.Early recognition of PRES with imaging modalities can improve the treatment thus preventing lethal complications.

REFERENCES

1. Fischer M, Schmutzhard E. Posterior reversible encephalopathy syndrome. J Neurol. 2017 Aug;264(8):1608-1616.
2. Hobson EV, Craven I, Blank SC. Posterior reversible encephalopathy syndrome: a truly treatable neurologic illness. Perit Dial Int. 2012 Nov-Dec;32(6):590-4.
3. Ito Y, Arahata Y, Goto Y, Hirayama M, Nagamutsu M, Yasuda T, Yanagi T, Sobue G. Cisplatin neurotoxicity presenting as reversible posterior leukoencephalopathy syndrome. AJNR Am J Neuroradiol. 1998 Mar;19(3):415-7.
4. Ozcan C, Wong SJ, Hari P. Reversible posterior leukoencephalopathy syndrome and bevacizumab. N Engl J Med. 2006 Mar 02;354(9):980-2; discussion 980-2.
5. Allen JA, Adlakha A, Bergethon PR. Reversible posterior leukoencephalopathy syndrome after bevacizumab/FOLFIRI regimen for metastatic colon cancer. Arch Neurol. 2006 Oct;63(10):1475-8.
6. Bodkin CL, Eidelman BH. Sirolimus-induced posterior reversible encephalopathy. Neurology. 2007 Jun 05;68(23):2039-40.
7. Garner O, Ramirez A, Iardino A. A case of posterior reversible encephalopathy syndrome associated with sepsis. BMJ Case Rep. 2018 Jul 10;2018
8. Sundin CS, Johnson ML. Posterior Reversible Encephalopathy Syndrome. MCN Am J Matern Child Nurs. 2018 Mar/Apr;43(2):77-82.
9. Hinchey J, Chaves C, Appignani B, Breen J, Pao L, Wang A, Pessin MS, Lamy C, Mas JL, Caplan LR. A reversible posterior leukoencephalopathy syndrome. N Engl J Med. 1996 Feb 22;334(8):494-500.
10. Fugate JE, Claassen DO, Cloft HJ, Kallmes DF, Kozak OS, Rabinstein AA. Posterior reversible encephalopathy syndrome: associated clinical and radiologic findings. Mayo Clin Proc. 2010 May;85(5):427-32.
11. Cordelli DM, Masetti R, Ricci E, Toni F, Zama D, Maffei M, Gentili A, Parmeggiani A, Pession A, Franzoni E. Life-threatening complications of posterior reversible encephalopathy syndrome in children. Eur J Paediatr Neurol. 2014 Sep;18(5):632-40.
12. Marra A, Vargas M, Striano P, *et al*. Posterior reversible encephalopathy syndrome: the endothelial hypotheses. *Med Hypotheses* 2014;82:619–22.
13. Zou LP, Liu LY, Li H, Wang YY, Liu Y, Chen J, Hu LY, Liu MJ, Zhang MN, Lu Q, Ma SF. Establishment and utility assessment of posterior reversible encephalopathy syndrome early warning scoring (PEWS) scale establishment and utility assessment of PEWS scale. BMC Neurol. 2019 Feb 21;19(1):30.
14. Fugate JE, Claassen DO, Cloft HJ, Kallmes DF, Kozak OS, Rabinstein AA. Posterior reversible encephalopathy syndrome: associated clinical and radiologic findings. Mayo Clin Proc. 2010 May;85(5):427-32.
15. Murray K, Amin U, Maciver S, Benbadis SR. EEG Findings in Posterior Reversible Encephalopathy Syndrome. Clin EEG Neurosci. 2019 Sep;50(5):366-369.
16. Vandenbossche G, Maquet J, Vroonen P. A reversible posterior leucoencephalopathy syndrome including blindness caused by preeclampsia. Facts Views Vis Obgyn. 2016 Sep;8(3):173-177.
17. Strother R, Wong H, Miller NE. Posterior Reversible Encephalopathy Syndrome Secondary to Hypertensive Encephalopathy Brought on by a MAO Inhibitor: A Case Report. J Prim Care Community Health. 2019 Jan-Dec;10:2150132719869539.
18. Roth C, Ferbert A. The posterior reversible encephalopathy syndrome: what's certain, what's new? Pract Neurol. 2011 Jun;11(3):136-44.
19. Gijtenbeek JM, van den Bent MJ, Vecht CJ. Cyclosporine neurotoxicity: a review. J Neurol. 1999 May;246(5):339-46.
20. Lifson N, Pasquale A, Salloum G, Alpert S. Ophthalmic Manifestations of Posterior Reversible Encephalopathy Syndrome. Neuroophthalmology. 2019 Jun;43(3):180-184.