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Case Study

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MANAGEMENT OF POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME IN ICU: A CASE REPORT

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ABSTRACT

Acute neurological emergencies in pregnant and postpartum women presenting as headache, visual disturbances, seizures, and elevated blood pressure are usually attributed to preeclampsia and eclampsia which are considered as the most common causes of PRES. Posterior Reversible Encephalopathy Syndrome (PRES) is a clinico-radiological syndrome characterized by a headache, seizures, altered mental status and visual loss and characterized by white matter vasogenic edema affecting the posterior occipital and parietal lobes of the brain predominantly. We present here a middle aged woman with headache, generalised tonic–clonic seizures and blurring of vision in late antepartum stage. Reversibility of the symptoms and characteristic imaging findings led us to a diagnosis of PRES in our patient.

KEYWORDS: Posterior Reversible Encephalopathy Syndrome (PRES), Preeclampsia, Generalised tonic clonic seizures.

INTRODUCTION

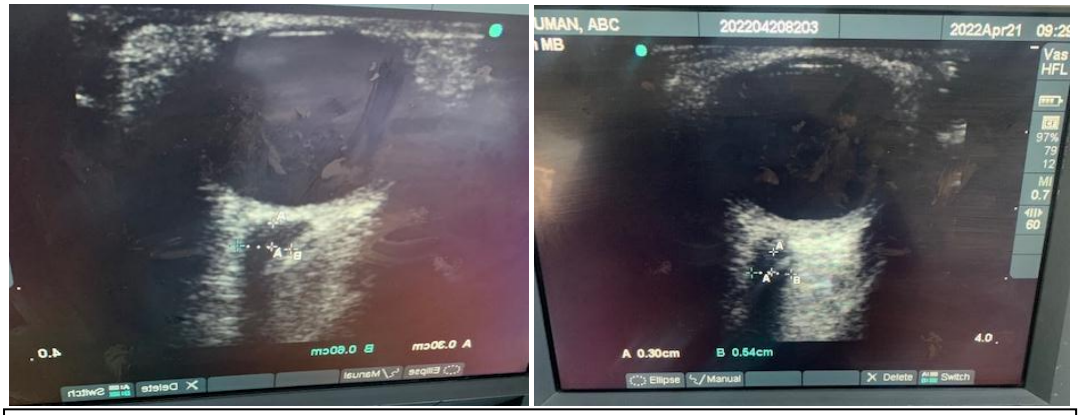
Posterior reversible encephalopathy syndrome (PRES) is a syndrome characterized by subacute neurologic and radiographic findings.[1,2] It was first described by Hinchey et al[3] in 1996. It is commonly but not always associated with acute hypertension.[4] More than 70% of patients with PRES are hypertensive, though a significant proportion have normal or mildly raised blood pressure. Patients often present with headaches, seizures, visual changes, or altered mental status hours to months after the inciting insult.[2,5] Diagnosis of PRES relies on history, clinical examination, and radiologic findings of symmetric bilateral hyper-intensities on T2-weighted magnetic resonance imagings (MRIs) representing vasogenic edema commonly affecting the posterior occipital and parietal lobes but may be seen throughout the frontal and temporal lobes, cerebellum, or brainstem[1,2,5] This condition is typically reversible once the underlying cause is removed which could be malignant hypertension, renal dysfunction, and immunosuppressant use such as cisplatin and gemcitabine.[2,6] It has been reported in patients of all age groups but It appears to have a female predominance, even after exclusion of patients with eclampsia with most cases occurring in young-aged to middle-aged adults.[7] Seizures and status epilepticus are common, and nonconvulsive status epilepticus may be more frequent than generalized status epilepticus. Signs of nonconvulsive status include stereotypic movements such as staring, eye blinking, or head turning. Management of PRES includes removal of any offending agents, blood pressure, and seizure management and temporary renal replacement therapy (hemodialysis/peritoneal dialysis) if required.[1]

We report a case of a 34-year-old woman who developed late antepartum eclampsia complicated by the development of PRES.

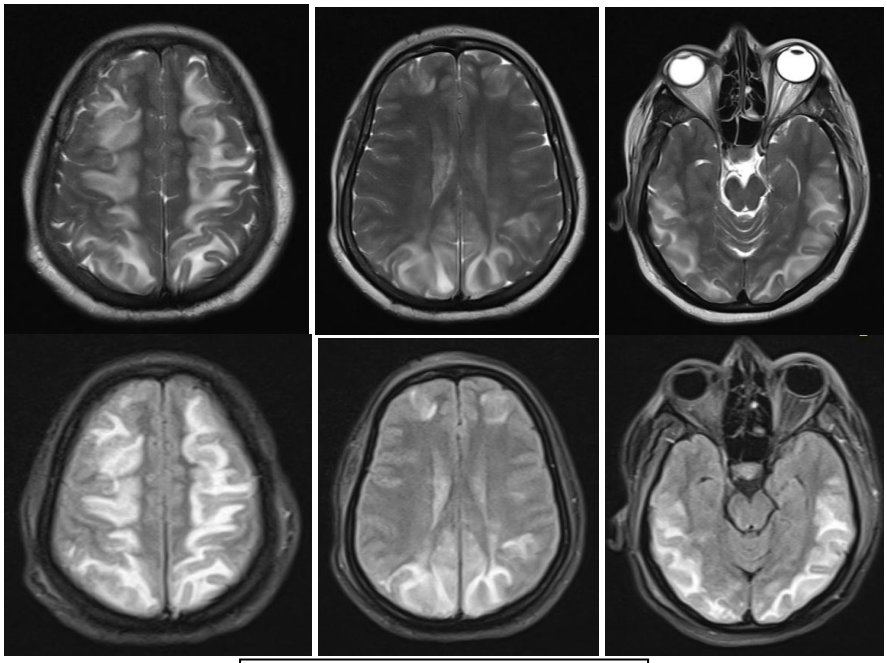
CASE REPORT

A 34-year-old woman, gravida 2 para 2, with an unremarkable medical history presented to the emergency department with symptoms of abdominal pain, distension and blurring of vision since 1 day where she was recorded to have high blood pressure recordings. She had an otherwise uneventful pregnancy and all her previous antenatal blood pressure recordings were within normal limits. Her past obstetric history was non significant. The patient developed two episodes of witnessed generalised tonic–clonic seizures. Shortly after resolution of seizures, she was found to have an elevated BP of 180/100 mm Hg. Hypertension was managed with IV labetalol and 2 doses of MgSo4 (5gm IM and 2 gm IV). The patient started developing altered sensorium with deterioration of GCS to E2V3M3. Bilateral pupils were normally reactive to light. The patient was immediately taken up for emergency LSCS under GA. Due to poor mentation of the patient preoperatively,

patient was shifted to the ICU intubated. Laboratory investigations revealed normal hemoglobin (12.8 g/dL), neutrophilic leukocytosis (16,000/dL), thrombocytopenia with deranged liver function tests and renal function tests. Chest radiography and arterial blood gas analysis were normal. The patient underwent hemodialysis twice in view of oliguria and increasing serum urea and creatinine. Brain magnetic resonance imaging was planned to rule out the cause of her non improving sensorium. MRI showed bilateral occipital, parietal and frontal cortex T2/fluid-attenuated inversion recovery (FLAIR) hyperintensities suggestive of PRES. Patient was managed with intravenous fluids, antibiotics, antiepileptics and monitoring of blood pressure. Serial ONSD were done and it was 0.60cm and when the patient became concious, it was 0.54cm. Patient improved symptomatically in form of normal sensorium, leukocyte counts and vital signs within 2 weeks of ICU admission. She was extubated and discharged after 2 weeks of admission in the ICU at request.



OPTIC NERVE SHEATH DIAMETER MEASUREMENT BY USG



IMAGES OF BRAIN MRI

DISCUSSION

PRES is a reversible neurological entity characterised by the presence of white matter oedema affecting the occipital and parietal lobes. The exact incidence of PRES is unknown.[8] The exact pathophysiological mechanism of PRES is still unclear.[9] Three hypotheses have been proposed till now, which include 1) cerebral vasoconstriction causing subsequent infarcts in the brain, 2) failure of cerebral auto-regulation with vasogenic edema, and 3) endothelial damage with blood-brain barrier disruption further leading to fluid and protein transudation in the brain.[9,10] It can occur at any age and most commonly affects females. A variety of clinical conditions are associated with the development of PRES where the commonly reported ones include hypertensive emergency, renal disease, pre-eclampsia/eclampsia and immunosuppressive agents.[11] Other reported causes include sepsis, autoimmune diseases such as systemic

lupus erythematosus, systemic sclerosis, tumour lysis syndrome, Guillain-Barres syndrome, AIDS, thrombotic thrombocytopenic purpura and acute intermittent porphyria.[7,12] Clinically, PRES presents with headache, seizures, encephalopathy, visual disturbances and focal neurological symptoms.[13] However, some patients with severe manifestations of PRES, such as coma and/or status epilepticus, may require admission to the intensive care unit.[14] Early imaging is crucial to make the distinction of PRES from other differential diagnosis of stroke, meningoencephalitis, demyelinating lesions of the brain and cerebral venous thrombosis. MRI is the imaging modality of choice.[15] Diffusion-weighted MRI helps to distinguish the vasogenic oedema from cytotoxic oedema, which is characteristic of this disease.[11] The management of PRES involves early diagnosis, treatment of symptomatology and correction of the causative factor. As indicated by its name, appropriate treatment is expected to ensure a full recovery. However, recurrence of symptoms has been observed in 8% of the cases.[8]

REFERENCES

1. Granata G, Greco A, Iannella G, Granata M, Manno A, Savastano E, Magliulo G. Posterior reversible encephalopathy syndrome—insight into pathogenesis, clinical variants and treatment approaches. Autoimmunity reviews, 2015; 14(9): 830-6.
2. Russell MT, Nassif AS, Cacayorin ED, Awwad E, Perman W, Dunphy F. Gemcitabine-associated posterior reversible encephalopathy syndrome: MR imaging and MR spectroscopy findings. Magnetic resonance imaging, 2001; 19(1): 129-32.
3. 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. New England Journal of Medicine, 1996; 334(8): 494-500.
4. McKinney AM, Short J, Truwit CL, McKinney ZJ, Kozak OS, SantaCruz KS, Teksam M. Posterior reversible encephalopathy syndrome: incidence of atypical regions of involvement and imaging findings. American Journal of Roentgenology, 2007; 189(4): 904-12.
5. Marrone LC, Marrone BF, de la Puerta Raya J, Gadonski G, da Costa JC. Gemcitabine monotherapy associated with posterior reversible encephalopathy syndrome. Case Reports in Oncology, 2011; 4(1): 82-7.
6. Cioffi P, Laudadio L, Nuzzo A, Belfiglio M, Petrelli F, Grappasonni I. Gemcitabine-induced posterior reversible encephalopathy syndrome: a case report. Journal of Oncology Pharmacy Practice, 2012; 18(2): 299-302.
7. Legriel S, Pico F, Azoulay E. Understanding posterior reversible encephalopathy syndrome. InAnnual update in intensive care and emergency medicine, 2011; (631-653). Springer, Berlin, Heidelberg.
8. Roth C, Ferbert A. Posterior reversible encephalopathy syndrome: long-term follow-up. Journal of Neurology, Neurosurgery & Psychiatry, 2010; 81(7): 773-7.
9. Vaughan CJ, Delanty N. Hypertensive emergencies. The Lancet, 2000; 356(9227): 411-7.
10. Shin KC, Choi HJ, Bae YD, Lee JC, Lee EB, Song YW. Reversible posterior leukoencephalopathy syndrome in systemic lupus erythematosus with thrombocytopenia treated with cyclosporine. JCR: Journal of Clinical Rheumatology, 2005; 11(3): 164-6.
11. Chambers KA, Cain TW. Postpartum blindness: two cases. Annals of emergency medicine, 2004; 43(2): 243-6.
12. Domínguez-Fuentes B, Garcia-Gil D, Romero-Palacios A, Sánchez-Crespo JM, García-Arjona R, Navarro-Navarro J. Posterior reversible leukoencephalopathy in a patient with postpartum eclampsia. Medicina Intensiva, 2008; 32(7): 361-3.
13. Ekawa Y, Shiota M, Tobiume T, Shimaoka M, Tsuritani M, Kotani Y, Mizuno Y, Hoshiai H. Reversible posterior leukoencephalopathy syndrome accompanying eclampsia: correct diagnosis using preoperative MRI. The Tohoku journal of experimental medicine, 2012; 226(1): 55-8.
14. Kozak OS, Wijdicks EF, Manno EM, Miley JT, Rabinstein AA. Status epilepticus as initial manifestation of posterior reversible encephalopathy syndrome. Neurology, 2007; 69(9): 894-7.
15. Ay H, Buonanno FS, Schaefer PW, Le DA, Wang B, Gonzalez RA, Koroshetz WJ. Posterior leukoencephalopathy without severe hypertension: utility of diffusion-weighted MRI. Neurology, 1998; 51(5): 1369-76.