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POSTPARTUM ATYPICAL POSTERIOR REVERSIBLE ENCEPHALOPHATHY SYNDROME, DIAGNOSTIC CHALLENGES: A CASE REPORT

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ABSTRACT

Seizures in pregnant women are often linked to eclampsia, but in 12% of cases, other pathology of cerebral origin may be the cause such as PRES syndrome. The clinical case that we report highlights an atypical PRES syndrome occurring in postpartum patient. Atypical PRES presents with variations in clinical features, imaging patterns, or course of recovery making its diagnosis challenging. Here we report a case of 25 year old woman with no medical history, who had undergone C section for fetal distress and developed generalised tonic clonic seizures on day six of post-operative day at home. She had no records of previous high blood pressure or any neurological manifestations. After initial examination a brain CT scan was performed which failed to identify any abnormalities suggestive of PRES. Subsequently, MRI revealed focal T2/FLAIR, hyperintensity in subcortical region of right high frontal lobe with no diffusion restriction, likely indicating vasogenic edema. She was managed in ICU with magnesium sulphate, levitaracetam and mannitol. There was a complete remission in three days and she was discharged on tablet levitaracetam. The mechanism of genesis of PRES is still based on the hypothesis not yet confirmed. Understanding the atypical presentation of PRES is crucial for early diagnosis and appropriate management, ensuring better outcome despite its potential complexities.

INTRODUCTION

Posterior reversible encephalophathy syndrome (PRES) is a syndrome affecting the CNS with a range of clinical presentations, most often including headache, altered mental status, seizures, and visual loss.[1] Typical imaging features include vasogenic edema involving predominantly bilateral occipital and parietal lobes.[2,3,4] It is a clinico-radiological entity associated with various conditions such as pre-eclampsia, eclampsia, autoimmune disorders etc. Seizures in pregnant women are often linked to eclampsia, but in 12% of cases, other pathology of cerebral origin may be the cause such as PRES syndrome.[5] Being a newly diagnosed entity, its mechanism of genesis is still based on the hypothesis not yet confirmed. Atypical PRES presents with variations in clinical features, imaging patterns, or course of recovery making its diagnosis challenging. However, during pregnancy, PRES syndrome often occurs with severe pre-eclampsia or eclampsia; hence, the great interest of our clinical case, through which we report the case of an isolated PRES syndrome occuring in postpartum and outside the context of preeclampsia.

CASE REPORT

A 25 year old woman on POD-8 following EmLSCS presented to our emergency at Dr RPGMC Tanda with history of generalised tonic clonic seizures since 1-2 hours. She -had 3 episodes of similar abnormal body movements of all 4 limbs in last 2 days and was associated with uprolling of eyes, frothing from mouth and post ictal confusion; however there was no history of fecal/urine incontinence, deviation of mouth and weakness of any limb. Her antenatal period was uneventfull with normal BP records. She went into spontaneous labour delivered by EmLSCS i/v/o MSL with AFD. Her immediate postpartum period was uneventfull and was discharged on POD-4. There is no histrory of any neurological pathology neither autoimmune nor antecedent of transplantation of organs and without notion of particular drug taking. On general

physical examination she was drowsy with GCS-E2V1M5, BP-104/60 mmHg, PR-88/min, Spo2-98%in RA, no headache or visual disturbances, absence of edema in lower limbs. Neurologic examination was unremarkable with normal tendon reflexes., Biochemistry also did not revealed any abnormality. After initial examination a brain CT was performed which failed to identify any abnormalities. Subsequently, MRI revealed multiple well defined punctate T2/FLAIR hyperintensities in bilateral periventricular white matter region and subcortical white matter of bilateral FRONTOPARIETAL lobes with no diffusion restriction –VASOGENIC EDEMA. She was shifted in ICU for monitoring and was started on magnesium sulphate, Inj levitaracetam, and mannitol. Patient recovered clinically and was discharged after 5 days on tab levitaracetam.

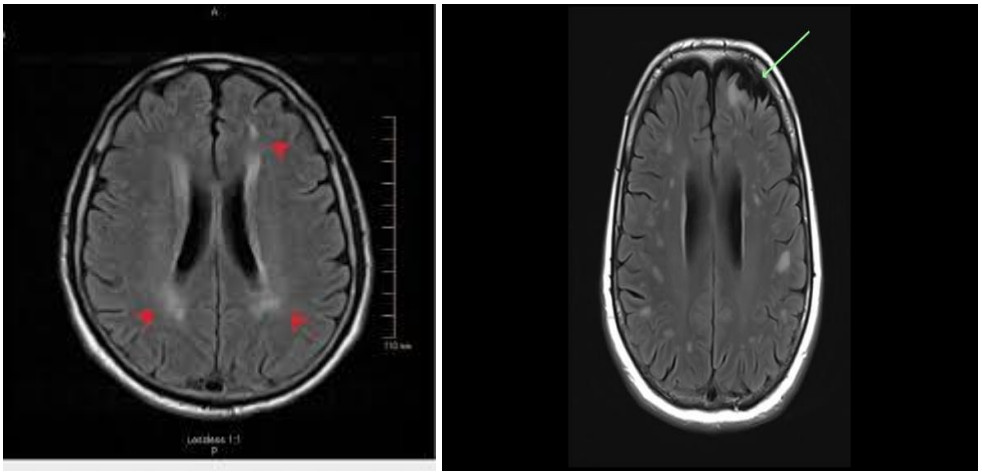

Figure 1: MRI revealed multiple well defined punctate T2/FLAIR hyperintensities in bilateral periventricular white matter region and subcortical white matter of bilateral FRONTOPARIETAL lobes with no diffusion restriction –VASOGENIC EDEMA.

DISCUSSION

Posterior reversible encephalopathy syndrome (PRES) is a rare yet critical neurological condition that presents with acute neurological symptoms such as seizures, altered mental status, visual disturbances, and headache. While PRES is commonly associated with hypertensive disorders of pregnancy, particularly preeclampsia and eclampsia, emerging reports have documented atypical cases of postpartum PRES occurring in normotensive patients.[6,7] This raises important questions regarding the pathophysiological mechanisms underlying PRES beyond the conventional association with hypertensive crises.

The pathogenesis of PRES remains an area of active investigation, with two dominant theories being widely discussed. The first theory postulates that PRES results from a failure of cerebral autoregulation, leading to vasogenic edema due to excessive perfusion pressure in the brain's posterior circulation. The second theory suggests that endothelial dysfunction, triggered by systemic inflammation or toxic exposure, disrupts the blood-brain barrier, leading to fluid extravasation and brain edema.[8] In postpartum cases, even in the absence of hypertension, endothelial dysfunction may play a significant role, possibly due to pregnancy-related physiological changes.

Diagnostic challenges arise due to the varied clinical presentation and the overlap of PRES symptoms with other obstetric and neurological emergencies. Radiological imaging, particularly magnetic resonance imaging (MRI), remains the gold standard for diagnosis, with characteristic findings of vasogenic edema predominantly in the posterior parieto-occipital lobes. However, atypical imaging patterns involving the basal ganglia, brainstem, or cerebellum have been documented in postpartum cases.[9] This underscores the need for heightened clinical suspicion and comprehensive imaging evaluations when diagnosing PRES in postpartum patients.

The management of PRES primarily focuses on addressing the underlying cause and supportive care. In cases where PRES is associated with preeclampsia or eclampsia, aggressive blood pressure control and magnesium sulfate administration are crucial. However, in normotensive postpartum PRES cases, therapeutic strategies shift towards managing potential endothelial dysfunction, optimizing cerebral perfusion, and monitoring for associated complications such as intracranial hemorrhage or persistent neurological deficits.[10] The majority of patients exhibit complete neurological recovery if prompt diagnosis and treatment are instituted, reinforcing the importance of early recognition.

The increasing number of reported atypical postpartum PRES cases challenges the conventional understanding of its etiology and calls for further research to explore non-hypertensive risk factors. Future studies should aim to elucidate the role of endothelial injury, immune dysregulation, and other systemic stressors in triggering PRES in postpartum patients.

CONCLUSION

This case underscores the significance of recognising atypical presentation of PRES, which can complicate diagnosis. Significant fact related to PRES is the possible development of this disorder with no remarkable elevation of blood pressure. Early detection and intervention can reverse the clinical and radiological features of PRES. Knowing about this rare and atypical clinico-radiological presentation of PRES can help improve the diagnosis and treatment of these patients.

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