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Case Report

Post-Partum Eclampsia Complicated with a Posterior Reversible Encephalopathy Syndrome: A Case Report

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Abstract

Acute severe hypertension occurring often in the setting of eclampsia or renal failure, may result in posterior reversible encephalopathy syndrome. An uncommon clinical and radiological entity that requires prompt multi-disciplinary management in order to avoid further complications. We share with readers a case of post-partum eclampsia followed by PRES, managed in intensive care unit with favorable outcome.

Keywords: *Posterior Reversible Encephalopathy Syndrome; Eclampsia; Post-Partum; Case Report*

Abbreviations

PRES: Posterior Reversible Encephalopathy Syndrome; BP: Blood Pressure; MRI: Magnetic Resonance Imaging

Introduction

Posterior reversible encephalopathy syndrome (PRES) is a rare clinical and radiological condition of acute neuro-sensorial symptoms along with the presence of vasogenic subcortical brain edema in neuro imaging [1]. It has often been described in the context of acute hypertension (essential, eclampsia, renal failure), but also in sepsis, autoimmune diseases or with the use of certain treatments (immunosuppressants, cytotoxics) [2].

Far apart from what the denomination may suggest, the reversibility of injuries in PRES depends on their severity and the quality of therapeutic management [2,3].

We present the case of a PRES following eclampsia with prompt and appropriate treatment, resulting in a favorable outcome with 3 years' follow-up.

Observation

Patient aged 33, right-handed, no medical history, G3P2, 2 uneventful vaginal deliveries, currently pregnant at 37 weeks' gestation, pregnancy poorly monitored, admitted to our obstetrics emergency unit for management of severe pre-eclampsia in the face of high blood pressure (170/110), positive proteinuria, neurosensorial signs of headache and visual blur. No fetal repercussions, in a patient not in labor.

The patient was conditioned with the beginning of venous anti-hypertensive treatment (Nicardipine by auto-pulsed syringe), magnesium sulfate (loading dose of 4g over 20 minutes, followed by a maintenance dose of 1g per hour) and then admitted directly to the operating room for fetal extraction. At the same time, a biological work-up was carried out, showing thrombocytopenia at 60,000/ul and LDH at 327 IU/L. the rest of blood tests was normal.

The immediate postpartum period was marked by the persistence of high blood pressure and neuro-sensorial symptoms (headaches and blurred vision) despite the continuous parenteral anti-hypertensive and magnesium sulfate treatment, leading to the occurrence of a tonic-clonic seizure resolved by 10 mg of Diazepam.

The patient was admitted to intensive care unit with close monitoring, after stabilization a cerebral MRI was performed showing bilateral occipital white matter T2/FLAIR high-signal intensities of the left posterior junctional zone and the left semi-oval center, suggestive of PRES syndrome.

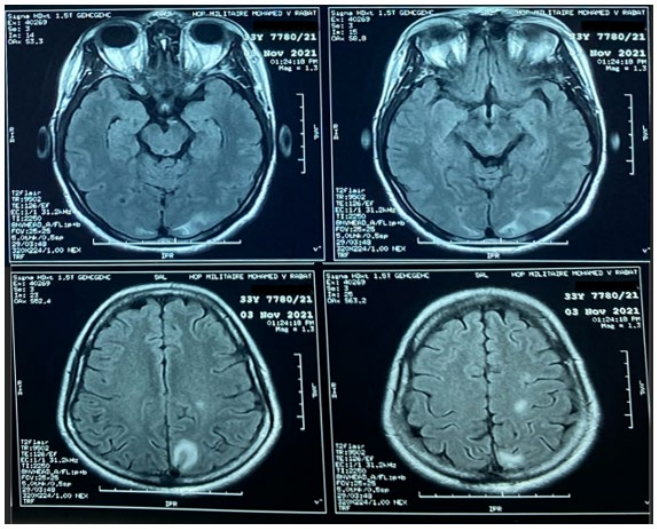

Figure 1: *Bilateral occipital white matter T2/FLAIR high signal of the posterior junctional zone.*

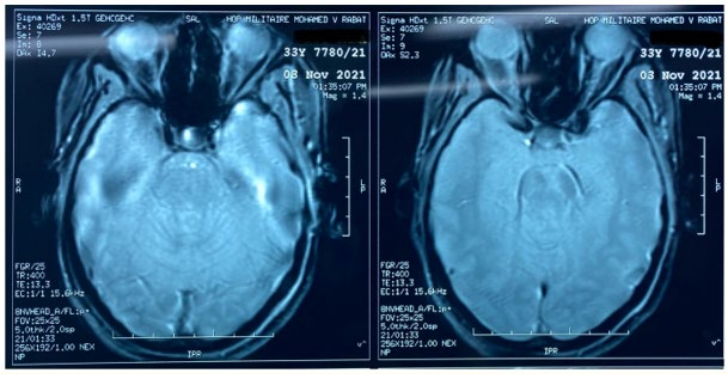

Figure 2: *White matter T2/FLAIR high signal of the left semi oval center.*

After 10 days stay in intensive care, the patient was discharged on alpha methyl dopa-based antihypertensive treatment and no recurrence was observed. A control MRI was performed 3 months later and came back strictly normal.

Discussion

PRES is a rare condition, occurring in two-thirds of cases during the first week after delivery in patients with uneventful pregnancies, or sometimes in the context of severe pre-eclampsia [4].

The pathophysiological mechanism so far accepted point to vasogenic edema linked to altered cerebro-vascular autoregulation (particularly in the posterior region, which is more vulnerable to systemic pressure variations) and/or endothelial damage [5,6] (as summed up in figure 3) [7].

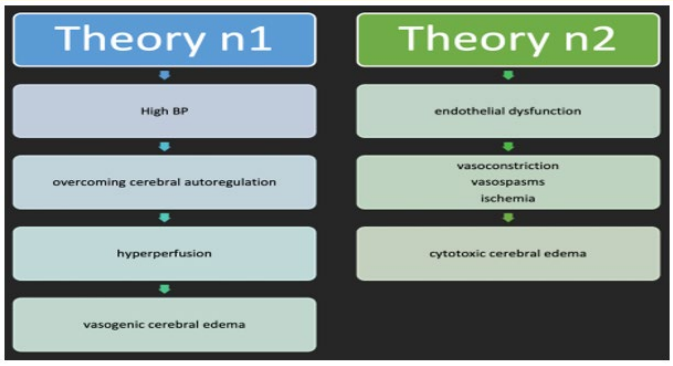

Figure 3: *Figure summarizing mechanisms of PRES.*

Pregnancy seems to be a fertile ground to such syndrome by many mechanisms such as: capillary hyperpermeability, elevation of BP, increased sensibility to endogenous vasopressor agents, reduction of prostaglandins and liberation of cytotoxic placental factors responsible for endothelial cell damage [8].

The symptoms in PRES can be developed over hours to days, they include: headaches, altered mental state, focal deficit, visual disturbance and seizures [2,5,6].

Brain imaging, especially MRI, reveals multifocal lesions in white and grey matter [9]. Corresponding to cerebral edema, predominantly in the posterior region of the cerebral hemispheres, mainly in the parieto-occipital region [10] they appear typically as iso or low signal T1 and high signal T2/FLAIR, with no enhancement after contrast injection [11]. Occasionally, contrast may indicate a rupture of the blood-brain barrier [12].

There is no standardized therapeutic approach [8,13]. However, controlling hypertension is the most important aspect of treatment [14,15]. The purpose is to maintain a mean arterial pressure between 105 and 125 mmHg, without reducing this pressure by more than 25% during the first hour [16,17]. Magnesium sulfate has a vasodilatory effect, increasing cerebral blood flow and thus preventing the onset of ischemic lesions that cause seizures [18-20]. Corticosteroids can be used to combat vasospasm and headaches.

if treated early and appropriately, PRES is reversible in 70% - 90% of patients, with complete regression of clinical and radiological abnormalities within few weeks [5]. However residual neurological deficits, such as epilepsy and motor deficits, may persist in cases with severe complications [2,3].

Conclusion

Posterior reversible encephalopathy remains little known syndrome, its prognosis can be appalling if not recognized and treated in time. It is therefore essential to be aware of it, so as to diagnose it early and provide effective treatment to prevent irreversible neurological damage and permanent after-effects.

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