INTRODUCTION

Posterior Reversible Encephalopathy Syndrome (PRES) is a clinicoradiological syndrome, with diverse clinical presentation and characteristic Computed Tomography (CT) and Magnetic Resonance Imaging (MRI) features. It describes a usually reversible non-specific neurologic symptoms include headache, confusion, visual disturbances, seizures, altered mental status, paresis, generalized seizures and coma.(1) The lesions in PRES are thought to be due to vasogenic edema, predominantly in the posterior cerebral hemispheres and are reversible with appropriate management. It may accrue in diverse situations, including hypertension, eclampsia, pre-eclampsia, allogenic bone marrow transplantation, in patients taking cyclosporine (and occasionally tacrolimus), in patients with autoimmune disease, renal failure, infection, sepsis (gram positive organisms predominate) and shock.(2,3) The imaging of PRES, CT or MRI shows occipital and parietal bilateral edema affecting both the cortex and subcortical white matter of the brain. Other common sites for PRES include the frontal lobes, the inferior temporal-occipital junction, and the cerebellum.(4) The treatment of PRES depends on the underlying cause. For instance, if the main problem is high blood pressure, blood pressure control will accelerate the resolution of the abnormalities. If the likely cause is medication, the withdrawal of the drug in question is needed.(5)

CASE REPORT

A 19-year-old female, primaparous, with negative past medical history, admitted in intensive care department for two episodes of generalized tonic-clonic seizures, accompanied by sphincter relaxation, vomiting, tongue bitten, hypertension and leg edema. She presented these symptoms after spontaneous vaginal delivery, two days before. Clinical examination: pallor of skin, afebrile, legs edema; blood pressure was–168/110 mmHg. Neurologic examination—revealed absence of neck stiffness, absence motor deficits, normal pupillary reflex, deep tendons reflexes brisk, bilateral Babinski sign, she was drowsy and confused. Emergency cerebral CT scan—revealed temporal and capsular bilateral hypodensities areas (figure no.1). All these changes are suggestive of posterior reversible encephalopathy syndrome (PRES).

Figure no. 1. Native cerebral CT scan, revealed bilateral hypodense lesions in temporal and capsular areas, suggestive of oedema
The patient had no known history of migraine, or seizures.

At the admission in emergency unit, the laboratory examination (complete blood picture, liver enzymes, kidney function tests, clotting parameters, serum electrolytes-sodium, potassium) were in normal range. There was no evidence of urinary proteinuria.

The chest X-ray examination was normal. Abdominal examination was normal.

A lumbar puncture for CSF analysis to rule out meningoencephalitis was not performed as the patient was afebrile with no signs of meningeal irritation.

Electrocardiographic (ECG) study was unremarkable. Although cerebral CT scan may show abnormalities in 50% of patients, MRI is the imaging modalities of choice for the diagnosis of PRES.(7) Non-enhanced head CT (NECT) can detect PRES when subcortical areas of low density are visible, however areas of limited can be missed.(7)

In our case report, images presented at non-enhanced head CT, (figure no 1.) was suggestive for PRESS in clinic context.

In the pregnancy period, there was a fluid accumulation in extracellular spaces and this generated hemoconcentration condition. During puerperium, there was fluid shift back to intravascular space; this has accentuated the tendency to develop brain oedema.

Our patient was treated in intensive care unit, with magnesium sulphate, intravenous infusion, 2 g/hour (3 days), calcium channel blockers (amlodipine 5 mg daily, 5 days), benzodiazepines (midazolam, 2.5 mg-intravenous /one dose, at admission) and cerebral depletes IV mannitol 20%, (100 ml intravenous infusion every 6 hours, 5 days).

In intensive care unit, the patient had blood pressure controlled permanently.

Since depletive and anti-hypertensive treatment has been introduced, the patient has not had any seizure and the blood pressure has reached a normal level. Also, the leg edema has remitted.

10th days after admission, the patient was discharged with a blood pressure of 110/70 mmHg and seizure free, in stable condition, with normal general and neurologic examination. Follow-up, non-enhanced head CT imaging also stable condition, with normal general and neurologic examination. Follow-up, non-enhanced head CT imaging also been introduced, the patient has not had any seizure and the blood pressure has reached a normal level. Also, the leg edema has remitted.

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10th days after admission, the patient was discharged with a blood pressure of 110/70 mmHg and seizure free, in stable condition, with normal general and neurologic examination. Follow-up, non-enhanced head CT imaging also supported the resolution of abnormal signals.

Clinical signs, radiologic imaging and evolution sustained diagnostic of PRES in our case.

**DISCUSSIONS**

Rapid diagnosis of PRES is essential for initiating treatment, to prevent complications such as infarction and haemorrhagic stroke.(8) PRES was originally thought to only present in the occipital lobes. Although a significant number of patients have some occipital lobe involvement, lesions can appear anywhere in the white matter with edema extending to the cortex. The pathogenesis of PRES is not completely understood. Vasogenic theory i.e. hypertension with loss of autoregulation remains a widely accepted theory for the development of brain oedema.(6)

Endothelial dysfunction/injury, hyperperfusion, and vasoconstriction may lead to altered integrity of the blood-brain barrier (BBB). Although water has previously been thought to transit across BBB easily, this is now known to be mediated by specific cannel, aquaporin-4. Work in animals suggests that water transport across the BBB is regulated in large part by aquaporin-4 and recent work has identified aquaporin-4 as a possible agent in development of PRES.(4) In most cases, vasogenic oedema in PRES usually resolves completely within a week.

CSF usually normal or may be elevated protein. The differential diagnosis of PRES includes, infarcts including “top of basilar syndrome”, venous thrombosis, infections-meningitis, encephalitis, post infectious encephalomyelitis and vasculitis. Management is supportive and includes control of blood pressure, seizure and to reduce brain oedema. The aim of pharmacological treatment is to maintain the blood pressure systolic between 140 and 160 mmHg and the diastolic one between 90-105 mmHg.

**CONCLUSIONS**

We have presented the case of a young primi-parous female, with negative medical history, who developed early post-partum, recurrent seizures and hypertension. Prompt recognition of the signs and symptoms of PRES, particularly identifying and treating high blood pressure, can prevent permanent neurologic disability.

PRES is a reversible condition, if the arterial hypertension is promptly treated. The exact time interval between the diagnosis, control blood pressure and permanent damage has not been studied.

Imagistic particularity in this case refer to the damage of the subcortical brain regions (temporal and capsular area), presented at non-enhanced head CT, which was complete reversible after treatment.

**REFERENCES**


