

Research Paper

Medical Science

Late Onset Postpartum Eclampsia Presented with Posterior Reversible Encephalopathy Syndrome (PRES)

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ABSTRACT

Posterior Reversible Encephalopathy Syndrome (PRES) refers to a clinico-radiological diagnosis. Clinically it is Characterized by non specific symptoms such as headache, confusion, visual disturbances and seizures. The radiological findings in PRES are thought to be due to vasogenic oedema, predominantly in the posterior cerebral

hemispheres, and are reversible with appropriate management. We report a case of posterior reversible encephalopathy syndrome diagnosed by MRI scan showing hyper intense areas involving right basi occipital region, right temporal lobe involving hippocampus, right Cerebellar hemisphere and bilateral occipital parasagittal region of the brain.

KEYWORDS: PRES; Vasogenic edema; Postpartum; normotensive.

Introduction:

Posterior reversible encephalopathy syndrome (PRES) is a well-recognized, clinical and neuro-radiological entity first described in 1996 by Hinchey et al.(1)This is characterized by headache, vomiting, confusion, seizures, visual abnormalities and motor signs. These transitory neurological disturbances are thought to be due to cerebral vasospasm causing ischemia of the involved territory. The ensuing cerebral ischemia has a characteristic imaging pattern on MRI scan. It is associated with a variety of underlying conditions including hypertensive encephalopathy, preeclampsia, haemolysis, elevated liver enzymes, low platelets (HELLP) syndromes, systemic lupus erythematosus, thrombotic thrombocytopenia purpura, sepsis, septic shock, treatment with immunosuppressant, renal failure and central nervous system infections.

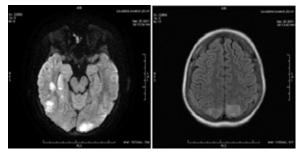
Importantly, these changes appear to be completely reversible if the underlying cause is treated or the precipitating drug withdrawn early in the clinical course.(1)

We report a case of Posterior reversible encephalopathy syndrome, occurring in the post partum period managed successfully.

Case Report

A 25 years old multi-parous lady underwent full term normal delivery conducted outside. Patient was referred to civil hospital on third postpartum day with complained of bifrontal headache along with visual disturbances followed by generalised tonic and clonic convulsions and postictal confusion. Her blood pressure was 190/110 mm Hg which was previously normal. A provisional diagnosis of postpartum eclampsia was thought off. Blood pressure could not be controlled even after starting her on magnesium sulphate and calcium channel blockers. She was suspected post partum cerebral venous thrombo-embolism. Her investigations including serum electrolytes, serum calcium, serum magnesium, Serum uric acid, serum creatinine, blood urea and coagulation profile were within normal limits. There was no evidence of urinary proteinuria. Antinuclear antibody evaluation was negative. Fundoscopy did not reveal any papilloedema. CT brain and MRI venogram were normal. Liver enzyme suggestive of increased SGPT (387.72 U/L) and SGOT(590 U/L) ,total bilirubin 5.25 mg/dl, direct bilirubin 0.88 mg/dl, indirect bilirubin 4.37 mg/dl. Her HB 11.2 gm/dl, WBC 19,400 cu/ mm, platelet 1.18 lac /cumm. Brain MRI scan showing hyper intense areas involving right basi occipital region, right temporal lobe involving hippocampus, right cerebellar hemisphere and bilateral occipital parasagittal region of the brain.

She was started on anti-hypertensive, anticonvulsants, anti edema measures, proton pump inhibitors and antibiotics. The patient stabilized by the 5th post partum day. Her neurological symptoms completely resolved by 10 days. She was discharged on anti-hypertensive and anti-convulsive medications.



Discussion:

The differential diagnosis for seizures in the late post-partum period includes eclampsia, subarachnoid haemorrhage, intracerebral haemorrhage, thrombotic phenomena, intracranial neoplasm, head trauma, idiopathic epilepsy, infection (meningo-encephalitis), amniotic fluid embolism, postpartum angiopathy.(1,2)

There was no past history of epilepsy or head injury. Infection was a possibility, the total count was 19,400 cells per cu mm. MRI brain with venogram ruled out intracranial bleed, ischemia secondary to thromboembolism, vasospasm or space occupying lesion. Amniotic fluid embolism rarely occurs after 48 hours post partum and generally presents with cardiopulmonary collapse and coagulopathy which was not seen in our patient. Possibility of systemic lupus erythematosis was ruled out as the Anti-nuclear antibody evaluation was negative. Renal failure was ruled out as serum creatinine was normal.

An alternative explanation is the possibility of post-partum angiopathy.

This diagnosis should be considered in a post-partum patient with hypertension and headache but no proteinuria as it was in this case. Post-partum angiopathy is a form of reversible cerebral segmental vasoconstriction characterised by severe "thunderclap" headaches, seizures, focal neurological deficits and segmental narrowing and dilatation of large and medium sized arteries. Typically, scanning reveals ischemic lesions but MRI findings consistent with reversible posterior leucoencephalopathy syndrome have been reported. (1)

Posterior reversible encephalopathy syndrome mostly secondary to late post partum eclampsia was suggested, as she presented with headache, followed by seizures, with raised WBC count(19,400 cumm), hypertension without protienuria, mostly secondary to hypertensive encephalopathy itself .MRI was typically consistent with vasogenic oedema, which was involving the areas like right basi occipital region, right temporal lobe involving hippocampus, right cerebellar hemisphere and bilateral occipital parasagittal region. Usually it involves the posterior occipito-parietal lobes and hence the nomenclature posterior

reversible encephalopathy syndrome.(3) PRES is still an under recognised and untreated condition. The incidence in the peripartum setting is not known.

in a normotensive multiparous lady following full term normal delivery, presenting as posterior reversible encephalopathy syndrome involving areas like right basi occipital region, right temporal lobe involving hippocampus, right cerebellar hemisphere and bilateral occipital parasagittal region, accompanied with late postpartum eclampsia or hypertensive encephalopathy. Due to prompt intervention and supportive therapy, this woman recovered within a 10 days and did not have any neurologic deficits at the time of discharge. Postpartum posterior reversible encephalopathy syndrome

(PRES) following a full term normal delivery is a very rare clinical entity. Recognition at the earliest and prompt initiation of the supportive measures can prevent permanent neurologic damage and thereby the associated morbidity. Multidisciplinary care forms the corner stone to achieve a safe motherhood in these women.

Conclusion:

In our case report etiology most commonly are septicaemia, postpartum eclampsia or hypertension encephalopathy and patient was present with headache, seizures, and altered mental status, and MRI brain suggestive of possibility of posterior reversible encephalopathy syndrome.

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