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CASE REPORT

Posterior Reversible Encephalopathy Syndrome in Pregnancy

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ABSTRACT

Posterior reversible encephalopathy syndrome (PRES) is a cliniconeuroradiological entity characterized by several symptoms of varied etiologies. The common symptoms are headache, confusion, seizures, cortical visual disturbances or blindness. Here, we report a 22 years old lady with 32 weeks of gestation who presented with complaints of acute onset of headache, sudden loss of vision with elevated blood pressure (BP), whose magnetic resonance venogram (MR venogram) revealed bilateral occipital T2 hyperintensity with restriction of diffusion suggestive of PRES. Early identification and treatment usually results in complete reversal of the deficits and delayed diagnosis and improper management can lead to irreversible sequelae.

Keywords: Posterior reversible encephalopathy syndrome, Pregnant.

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INTRODUCTION

Posterior reversible encephalopathy syndrome (PRES) is a cliniconeuroradiological entity characterized by several symptoms of different etiologies.[1,2] It is also called by several other names like reversible posterior leukoencephalopathy syndrome (RPLS). The most important symptoms are headache, confusion, seizures, cortical visual disturbances or blindness.[5] Multiple clinical associations for PRES have been described. Common clinical conditions associated with PRES include eclampsia, pre-eclampsia, autoimmune disorders and treatment with immune suppressants or cytotoxic medications. Uncommon clinical conditions include acute intermittent porphyria and cryoglobulinemia.[3] Low magnesium levels can augment PRES. High level of suspicion is necessary to recognize and prevent long-term sequelae of reversible condition like PRES.

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CASE REPORT

A 22 years old G2P1L1 with 32 weeks of gestation presented with complaints of acute onset of headache and sudden onset of loss of vision. She had history of elevated BP recordings 3 days prior to admission and was on tab. Methyldopa 500 mg TID and tab. Labetalol 100 mg BD. She was referred to our hospital in view of sudden complete loss of vision.

Patient was well oriented; BP 160/90 mm Hg, pulse rate of 88/min, respiratory rate of 18/min and chest was clear on auscultation. Room air saturation was 97%. Pupils were reacting to light, examination of other systems was normal. Complete blood picture showed normocytic normochromic anemia, platelet count 2.1 lakhs, coagulation profile, serum creatinine and LFT were normal. Urine analysis revealed proteinuria (2+). The provisional diagnosis was G2P1L1 with 32 weeks of gestation with previous LSCS, pre-eclampsia with PRES or CVT. Neurologist opinion was taken and magnetic resonance venogram (MR Venogram) revealed bilateral occipital T2 hyperintensity with restriction of diffusion suggestive of PRES as shown in Figure 1. In spite of tocolysis therapy, patient had preterm vaginal delivery of a female baby weighing 2.3 kg on 6/4/2015. Two hours after delivery, patient complained of diplopia and, 10 hours later, there was complete recovery of vision. On day 5, patient was discharged.

DISCUSSION

Sudden loss of vision in a pregnant woman poses a diagnostic dilemma as it can be due to cerebrovascular accidents complicating pregnancy, pre-eclampsia and clinical syndrome like PRES4. Posterior reversible encephalopathy syndrome is recently described syndrome which is characterized by headache, altered mental status, seizure and visual loss.[2] Altered mental status could

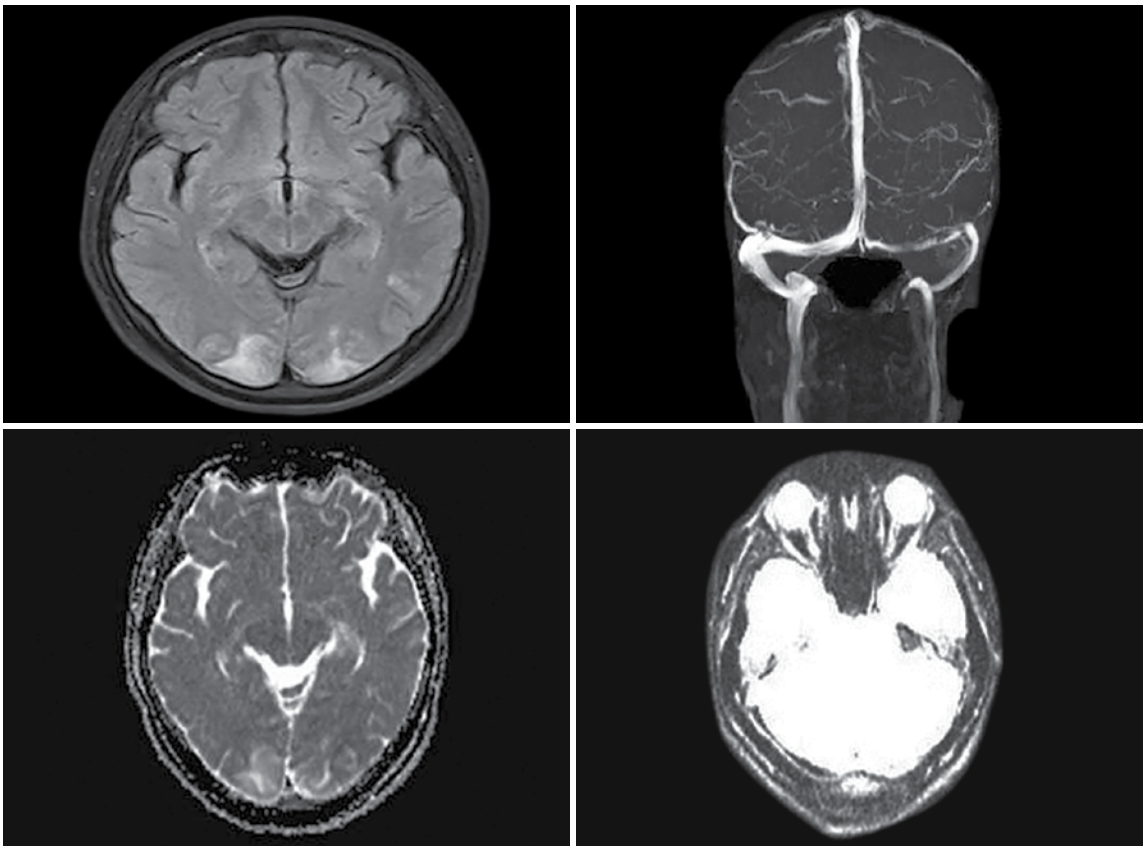

Fig. 1: Magnetic resonance venogram of brain showing bilateral occipital T2 hyperintensity with restriction of diffusion suggestive of PRES

range from lethargy, somnolence, restlessness, agitation, confusion, stupor and coma.[3] The three most common visual complications of pre-eclampsia and eclampsia are hypertensive retinopathy, exudative retinal detachment and cortical blindness.[6] Cortical blindness occurring in women with severe pre-eclampsia is a clinical syndrome characterized by intact papillary reflexes and normal fundoscopy findings. The loss of vision is usually transient and recovers within 4 hours to 8 days.[6]

Pathophysiology of PRES is elusive. Three theories have been proposed, the earliest theory suggests that breakdown in cerebral autoregulation results in reversible vasospasm and ischemia especially in vascular borderzone territories.[1] The new theory suggests that autoregulation maintains a constant blood flow to the brain, despite systemic blood pressure alterations by means of arteriolar constriction and dilatation. So the constricted arterioles are forced to dilate because of increased systemic blood pressure resulting in brain hyper-perfusion resulting in protein and fluid extravasation resulting in edema.[7] The last theory suggests endothelial dysfunction. However, pathogenesis is attributed to the failure of cerebral autoregulation that is probably facilitated in posterior brain regions due to sparse sympathetic innervations of the vertebra basilar vascular system. Clinical improvement is always seen with the treatments of the cause. Magnesium therapy is the choice of drug in cases of PRES secondary to eclampsia.

CONCLUSION

The cause of PRES is multifactorial. Early treatment usually results in complete reversal of the deficits over few days to several weeks; however, delayed diagnosis and improper management can lead to irreversible sequelae.

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