POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME IN PREGNANCY- A CASE REPORT
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ABSTRACT
We report the case of a 19 year old pregnant patient presented to us with hypertension, seizures and stroke. A number of clinical scenarios can present with similar symptomatology, which poses a challenge to treating clinicians. Here we report a similar case which on evaluation turned out as posterior reversible encephalopathy syndrome (PRES).

Keywords: Posterior Reversible Encephalopathy Syndrome

INTRODUCTION
Posterior reversible encephalopathy syndrome (PRES), first described by Hinchey et al., in 1996, is a clinical condition presenting with neurological symptoms including headache, seizures, altered sensorium, and loss of vision, and accompanied by characteristic magnetic resonance imaging (MRI) findings which are potentially reversible. MRI is the investigation of choice. MRI shows symmetrical white matter edema in posterior cerebral hemispheres, particularly in parieto-occipital regions. With treatment, resolution of findings occurs within days to weeks.

CASES
A 19 year old primigravida at 34 weeks of gestation presented to the emergency labour room with history of convulsions. She was having regular antenatal check ups from the local hospital and it was uneventful till then. At 34 weeks of gestation, she gave history of severe headache and vomiting for which she did not seek any medical help. On the same day after 4 hours, she had five episodes of generalised tonic clonic seizures and consulted in the local hospital and from there referred to our institution.

On the time of admission, patient was drowsy, vitals stable with blood pressure of 150/90 mms of Hg. Obstetric examination revealed single live fetus of 34 weeks growth cephalic presentation and on vaginal examination cervix was unfavourable. Investigations - Urine albumin was absent, renal and liver functions were normal.

She was treated with general supportive measures and magnesium sulphate infusion. Emergency caesarean section was done as the cervix was unfavourable and delivered female baby of 1.5 kg.

Post operative period, patient continued to be in an irritable state and complained of decreased movement of her right upper and lower limbs and visual disturbances. Emergency CT head showed abnormal patchy ill defined hypodensities involving bilateral frontoparietal and pareito occipital subcortical white matter and basal ganglia.

MRI showed areas of high signal throughout the white matter particularly involving the parietal regions with some extension on the right to the frontal lobe and inferiorly bilaterally to involve the occipital and right temporal lobes with hypo perfusion infarct along border zone suggestive of Posterior Reversible Encephalopathy Syndrome. No mass-effect is present. High signal on diffusion is appreciated focally in the parietal regions at the grey-white junction. There was mild mass-effect, without hemorrhage or midline shift.

MRI Venogram showed no significant thrombosis. She was treated with antiedema measures and antihypertensives. She responded well and was discharged on 10th post operative day.
MR Brain: Areas of high signal throughout the white matter particularly involving the parietal regions with some extension on the right to the frontal lobe and inferiorly bilaterally to involve the occipital and right temporal lobes. No mass-effect is present. High signal on diffusion is appreciated focally in the parietal regions at the gray-white junction. There is mild mass-effect, without hemorrhage or midline shift.

**DISCUSSION**

PRES is a complex multifactorial syndrome and can be diagnosed with reversible hyper intensities on T2-weighted cranial MR-images. It predominantly affects the posterior circulation territory and the clinical hallmarks are headache, confusion, seizures, cortical visual disturbances or blindness and other neurological signs. (Fitzgerald-Hines, 2006)

There are three theories for PRES. The earliest theory suggested that overreaction of brain autoregulation results in reversible vasospasm, which in turn results in potentially reversible ischemia to the brain, especially in vascular borderzone territories (Hagemann, 2004). The newer theory suggested 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 the increased systemic blood pressure, resulting in brain hyperperfusion. This increased perfusion pressure is sufficient to overcome the blood-brain barrier, allowing extravasation of fluid, macromolecules, and even red blood cells into the brain parenchyma. So, P.R.E.S. represents vasogenic rather than cytotoxic oedema in the majority of cases (Kahana, 2005). The last theory suggests that at intravascular pressures just below those that could rupture the capillary wall, permeability through the endothelium increased markedly, which was most likely due to increased pinocytotic activity through the capillary wall. The active passage of fluid through the capillary wall may act to relieve intravascular pressure, forestalling the development of large haemorrhages (Schwartz, 2002). So in this case, P.R.E.S. is not an example of cytotoxic or vasogenic oedema but hydrostatic oedema.

However the pathogenesis is attributed to a failure of cerebral autoregulation that is probably facilitated in posterior brain regions due to a sparse sympathetic innervation of the vertebrobasilar vascular system. In our patient, presentation of seizures in pregnancy with high blood pressure and stroke suggested eclampsia. But urine albumin was nil. Another possibility in peripartum period is cortical venous thrombosis but MRvenogram was not suggestive. MRI picture was suggestive of PRES.
REFERENCES


