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POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME IN AN ECLAMPSIA PATIENT IN LATE POSTPARTUM PERIOD: A DIAGNOSTIC CHALLENGE.

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ABSTRACT PRES is known as posterior reversible encephalopathy syndrome is a clinico-radiological syndrome characterized by headache, seizures, altered mental status & visual loss[1,2,3]. It was first well described by Hinchey et al in 1996[1]. PRES is also known as an acute hypertensive encephalopathy syndrome. If promptly recognized and treated, this clinical syndrome resolves within a week and the changes seen in magnetic resolution imaging resolves over days to weeks.[4]

We present a peculiar case with complaint of acute onset of convulsions after normal delivery at home. Patient was brought unconscious to the emergency department with GCS 8 (E2V2M4). Patient was immediately intubated and taken on ventilator support. NCCT & MRI showed infract involving parieto-occipital areas, basal ganglia & brain stem. Patient was provide with symptomatic medical management and improved significantly in few days.

KEYWORDS :

CASE HISTORY:

25 year old female presented in emergency department with unconsciousness and GCS was E2V2M4. Patient was tachypnoeic and respiratory pattern was abdomino thoracic. Patient's blood pressure was 82/54 mmHg, pulse 118/min, oxygen saturation 86 % on room air. On auscultation B/L air entry decreased, B/L crepts present in lower lung field. On CNS examination, patient responding to painful stimuli, B/L planters were extensor, B /L pupils mid dilated and reacting to light. Per-vaginal examination was within normal limit. Patient was intubated and taken on ventilator and vasopressor support. Routine blood investigation, chest x-ray and blood sugar was within normal limit. History was given by her husband.

HOPI: The patient was apparently asymptomatic 2 days back, she had delivered a male baby at home (P5L3D2). On next morning she had an episode of convulsion (not associated with loss of consciousness, urination, and defecation, frothy discharge from mouth and tongue bite) for around 5 min duration followed by severe headache. Patient was taken to local hospital. She was referred to our emergency department after symptomatic treatment .There was another episode of convulsion for 5 min during transfer of patient and had become unconscious.

Past history: History of convulsions in her first pregnancy 8 years back immediately after delivery. She became unconscious and spontaneously recovered in some minutes to hrs, without any treatment and there was no residual signs and symptoms.

Personal history: She has no history of hypertension, diabetes mellitus, tuberculosis, asthma and any other chronic illness. No h/o trauma and no family history suggestive of similar event.

Emergency call attended, patient was assessed, IV lines secured and immediately intubated and shifted to ICU and taken on ventilator. Routine blood investigations and ABG were sent immediately. Treatment started with empirical antibiotic therapy, IV fluids, aspirin, and atorvastatin and injection phenytoin and injection sodium valproate as anti-epileptics.

Once patient became hemodynamically stable chest x-ray and CT scan done.

Routine blood investigation reports were within normal limits and chest x-ray showed no pathology.

NCCT showed ill-defined non enhancing hypo dense areas seen involving right capsule ganglionic region left thalamic region and brainstem and left parieto- occipital region with minimal edema. Figure 1and figure2.

Hemodynamic Vitals are within normal limit and ABG showed all parameter within normal limit. Respiratory and neurological condition improved next day.

Her average BP was 130-150/80-90mmhg, Pulse from 80 to 100/min and spo2 99%to 100%. Neurological consultation advised.

On 1st day GCS was E2VTM4 and patient was continuously on ventilator control mode and on vasopressor.

On 2 day same treatment continued and hemodynamic vital and general condition improved, GCS was E3VTM4 and all routine blood investigation within normal limit and Kept on CPAP mode. Vasopressor weaned off.

MRI head was done: T1 showed hypo dense, T2 /flair showed hyper intense lesions in right capsule-ganglionic region, B/L parasagittal, posterior parietal and occipital lobes mid brain, pons, left postero-inferior cerebellar hemisphere showing restriction in dw1.Figure 3 and figure 4.

On CNS examination patient was conscious oriented following verbal commands, motor and sensory examination were normal and B/L pupil NSNR, superficial and deep tendon reflexes were normal and equal bilaterally, B/L planters flexors , no focal deficit was found with no complaint of visual disturbances or headache. The patient was gradually weaned off from ventilator. The patient was kept under observation. The patient then shifted to ward and observed for next 3 days and then discharged.

DISCUSSION:

Posterior reversible encephalopathy syndrome is a clinico-radiological syndrome characterized by headache, seizures, altered mental status, and visual loss. This condition is designated by a variety of names-reversible posterior leuko- encephalopathy syndrome, reversible posterior cerebral edema syndrome, and reversible occipital parietal encephalopathy[2].

PRES is typically reversible once cause is removed. However patients with severe manifestation of PRES may require admission in ICU. PRES has been reported in patients aged 4 to 90 years. Although mostly it occurs in young and middle aged adults. There is marked female predominance. Mechanical ventilation is required in 35-40% of patients with PRES for 3 to 7 days[5,6,7,8]. PRES is associated with severe hypertension in postpartum eclampsia/preeclampsia, acute glomerulonephritis, hemolytic anemia syndrome, thrombocytopenic purpura, systemic lupus erythematosus, drug toxicity as in cisplatin, cyclophosphamide, bone marrow or skin cell transplantation, sepsis, hyperammonemia[9,10]

The mechanism is not well understood but is thought to be related to integrity of blood brain barrier.

One of the dominating hypothesis is that severe hypertension exceeds the auto regulatory ability of cerebral blood vessels leading to compromise of blood brain barrier and vasogenic edema.[11,12,13]

The upper limit of acute regulation of cerebral vasculature is approximately 150-160 mmHg. This phenomenon can occur because of the rich sympathetic innervations of cerebral vasculature[14].

however because little sympathetic innervations exist in posterior fossa, the parieto occipital regions of the brain can be particularly susceptible to hyper perfusion leading to endothelial damage & vasogenic edema[15,16,17,18].

Endothelial dysfunction leads to vasoconstriction & hypo perfusion resulting in cerebral ischemia & subsequent vasogenic edema.

The most commonly affected regions, in descending order, are the parietal and occipital lobes, frontal lobes, inferior temporal-occipital junction and cerebellum[19].

Radiological finding of PRES_-three radiological pattern was found[20,21]

1. Holo – hemispheric watershed pattern
Confluent vasogenic edema extending through the frontal, parietal and occipital lobe. The topography matches the watershed zone between anterior and posterior cerebral arteries on one hand and middle cerebral artery on other.

2. Superior frontal suture pattern
Patchy edema predominant in the frontal lobe along with superior frontal sulci

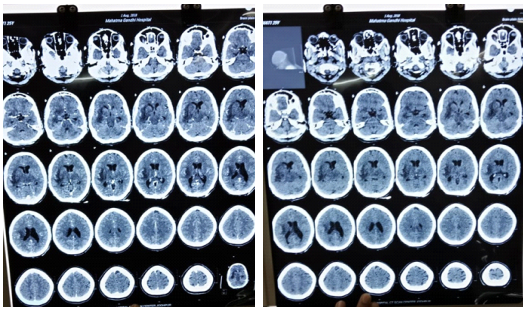
3. Dominant parietal occipital pattern
The posterior part of the parietal and occipital lobes are predominantly involved.

In computed tomography affected regions are hypo attenuating and in MRI, T1 shows hypo intensity in affected region and T2 shows hyper intensity and diffusion weighted intensity is usually normal.
Differential diagnosis

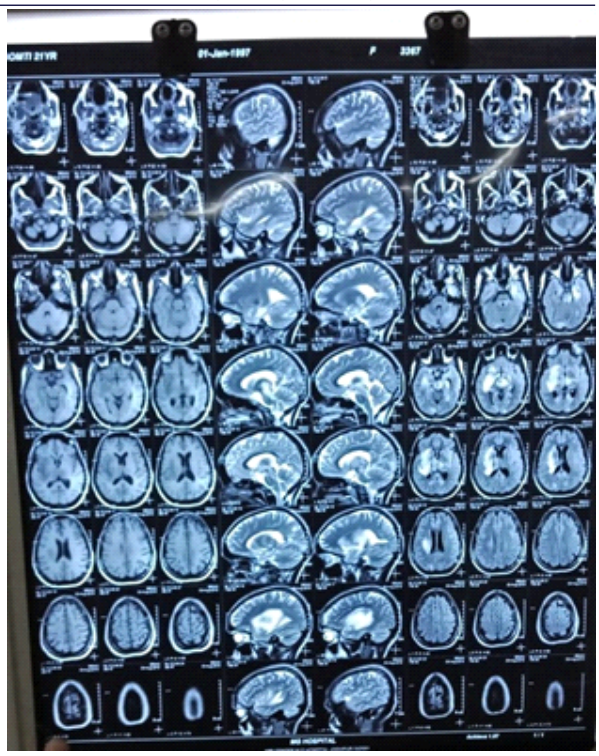
- PM – periventricular and subartrial involvement with little or no mass effect[22,23]
- Severe hypoglycemia.
- Posterior circulation infarct either occipital or cerebellar involvement. acute infarct however demonstrate restricted diffusion (PRES typically does not restrict)
- Sagittal sinus thrombosis
- Hypoxemic ischemic encephalopathy.

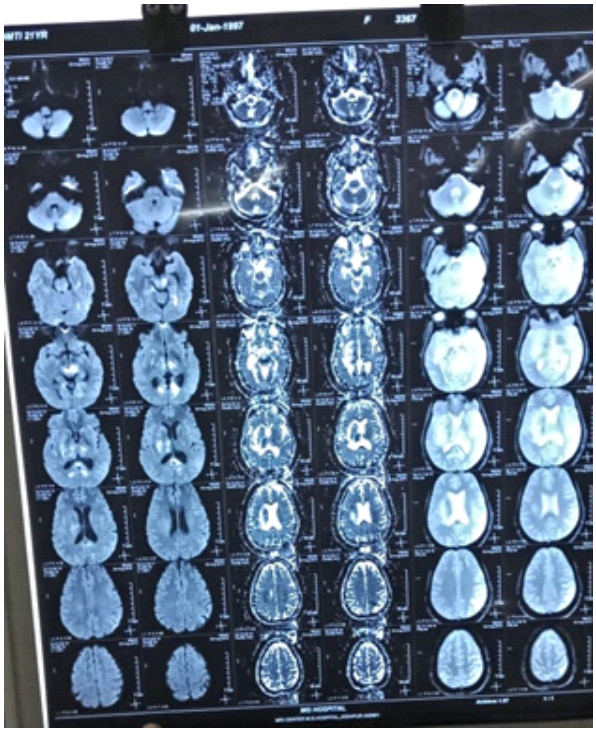
Patients with PRES require symptomatic measures usually taken in ICU for upper airway protection.PRES should also be suspected in pregnant patients with marked consciousness impairment or seizures .Hypoglycemia should be rule out and corrected. Anti-epileptic treatment should be initiated on emergency basis. Early diagnosis and prompt treatment of the cause of PRES is mainstay of the management of pres. Prompt early diagnosis and treatment can prevent complication as both clinical signs and neuro radiological pattern are reversible whereas delayed diagnosis and treatment can lead to ischemic or hemorrhagic lesions with permanent neurological damage.

Figure -CT scan



MRI





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