Reversible encephalopathy syndrome in a patient with eclampsia at pregnancy

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Abstract

Posterior reversible encephalopathy syndrome (PRES) is a established but rare cliniconeuroradiological entity characterized by headache, altered mental status, cortical blindness, seizures and other focal neurological signs, and a diagnostic magnetic resonance imaging picture. Most commonly, these cases are seen in obstetric patients with pregnancy induced hypertension and a variety of other nonobstetric conditions with the common denominator being hypertension. The management of an antepartum eclamptic patient with PRES is a challenging one as it poses not only diagnostic dilemmas but also of a lot of patient management issues as two lives are at stake. This report is important for the anaesthesiologists practicing obstetric anaesthesia.

Keywords: Posterior reversible encephalopathy syndrome, eclampsia

Case Report

A 20 year- old, 36-week pregnant, gravida 2, para1 with breech, presented with hypertension, foetal distress in active labour. She was referred to our hospital after the loading dose of intravenous (IV) magnesium sulphate ($MgSO_4$) 4 g. The patient attainder gave history of 4-5 episodes of convulsions with loss of consciousness and history of pedal oedema since 3 months. The patient had no history of hypertension during her previous pregnancy. The patient had another episode of generalised convulsion and was given diazepam 10mg IV. On examination, patient was drowsy, disoriented and restless. Anasarca, icterus, pallor and tongue oedema and frank haematuria. Vitals were as follows heart rate 158 beats per minute (bpm), blood pressure 170/110mmHg. Biochemical values were as follows: Hb: 11.1g/dl, haematocrit: 32.3%, total white cell count: 15,500/mm^3, platelet: 3,05,000/mm^3, AST: 40.3U/L, and ALT: 21.5U/L, LDH: 103U/L, ALP: 284 U/L, blood urea: 20 mg/dl, uric acid: 3.5 mg/dl, serum creatinine: 0.8 mg/dl, albumin: 3.2 g%, globulin: 3.9 g%, A:G ratio- 0.8 and her coagulation parameters were normal. Total bilirubin: 1.7mg/dl, conjugated bilirubin: 1 mg/dl, unconjugated bilirubin: 0.7 mg/dl. Urine analysis showed traces albumin and plenty of RBC's. Bedside ultrasonography confirmed foetal heart movements with heart rate of 162 beats per minute, breech presentation.

Following pre-oxygenation, anaesthesia was induced with Propofol 60mg and succinylcholine 100mg. Patient intubated and maintained with O_2 N_2O (in 50: 50 ratio)and 0.5-0.75 MAC isoflurane. Muscle relaxation was continued with atracurium. A viable female baby in breech presentation was extracted by Caesarean section. The baby measured 2.25kg with Apgar score at 1min, 7. After the surgery, patient was extubated, conscious and responding to verbal commands following which she was shifted to intensive care unit. Fourteen hours after the surgery, patient was noted to have laboured breathing not responding to painful stimulus with heart rate 160

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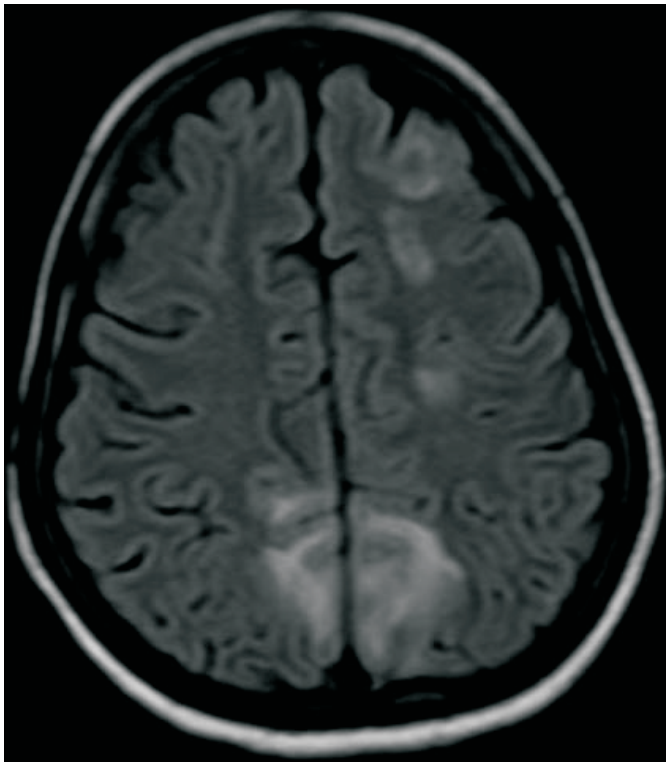
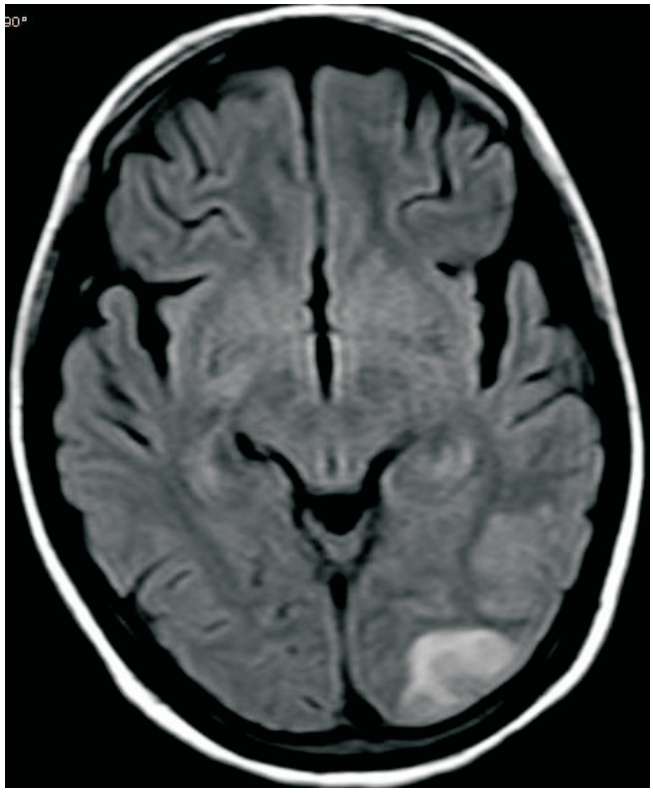
bpm, blood pressure 150/100 mmhg, sluggishly reacting pupils and on auscultation bilateral crepitations were heard. Patient was intubated with 7.0cm endotracheal tube and mechanically ventilation was initiated (SIMV, f: 12/min, FiO_2: 60, TV: 500 mL, I : E = 1 : 2). In the intensive care unit, her blood pressure tended to increase (180/110 mmHg) so nitroglycerin 10 μg/kg/h and magnesium sulfate 2 gr/h infusion was continued. The patient was administered dexamethasone 32 mg/day and an oral antihypertensive amlodipine 10 mg/day. Four hours later with return of spontaneous efforts, endotracheal tube was shortened to decrease the work of breathing and connected to T-piece with 4L/min of O_2.

On 1st Postoperative day, magnetic resonance imaging (MRI) was done which showed confluent areas of T2 or fluid-attenuated inversion recovery (FLAIR) hyperintesities in bilateral occipital lobe, parietal lobe and right frontal lobe, mainly in sub-cortical region and white matter with diffusion images (vasogenic oedema) suggesting the diagnosis of PRES. Patchy areas of diffusion restriction (cytotoxic oedema) seen in the left occipital lobe, bilateral high parietal region and left frontal lobe suggestive of infarction (figure 1 and 2). MR-venogram was normal .Chest x-ray revealed features of pulmonary oedema. Patient was extubated on 2nd postoperative day as sensorium and ventilatory parameters improved. This patient had memory disturbance.





Figures 1 and 2: MRI Brain- FLAIR images show bilateral high parietal, frontal and left occipital hyperintensities.

Discussion

PRES is a neurological condition that presents with symptoms viz unconsciousness, seizures and vision impairment. Its symptoms and imaging findings are generally reversible. Many conditions such as hypertension, eclampsia/preeclampsia, immunosuppresive drugs (cyclosporine), various antineoplastic agents, hypercalcaemia, thrombocytopenic syndromes, Henoch-Schönlein purpura, haemolytic uremic syndrome, systemic lupus erythematosus, amyloid angiopathy, and renal failure can cause PRES [1].

The eclamptic parturient presenting with altered mental status and acute focal neurologic deficit suggested a broad differential diagnosis in this case. Given the severity of the pregnancy induced hypertension and loss of vision, intracranial haemorrhage was of greatest concern. Distinguishing between thrombotic or embolic stroke and hemorrhagic stroke is paramount when considering treatment of arterial blood pressure [2]. Hinchey et al. first reported a reversible posterior leukoencephalopathy syndrome in 1996 [3]. This name was superseded by posterior reversible encephalopathy syndrome in 2000, which is now the most widely accepted terminology. Most cases of PRES are associated with hypertensive disorders, particularly those of pregnancy. In most obstetric cases, there is a history of preeclampsia/eclampsia and PRES usually develops only after delivery [4].

The pathophysiology of PRES is most likely, vasogenic oedema secondary to an acute increase in arterial blood pressure, which overcomes the autoregulatory capacity of the cerebral vasculature, causing arteriolar vasodilatation and endothelial dysfunction, leading to interstitial extravasation of fluid. Hence, PRES represents as vasogenic oedema rather than cytotoxic oedema in the majority of cases [2.5].

The most common finding on neuroimaging in these patients was confluent areas of signal abnormality seen in bilateral pattern that may be limited to the subcortical white matter but also involves the overlying cortex. High signal intensity is seen on the T2- weighted sequences usually in occipital, parietal, and posterior temporal lobes, and posterior fossa but may also involve the frontal lobes and corpus callosum. Diffusion weighted sequences may be normal or demonstrate increased diffusion in these regions, supporting the concept of increased interstitial fluid in the white matter and not ischemia. In cases with prolonged seizures or hypertension, frank ischemia or infarction may result [6]. Lesions due to vasogenic oedema are reversible. However, in some cases, lesions with reduced diffusion due to cytotoxic oedema can be determined, and these lesions generally heal leaving a sequel [1.] Our patient had patchy areas of diffusion restriction (cytotoxic oedema) in the left occipital lobe, bilateral high parietal region and left frontal lobe suggestive of infarction. Bilateral cerebral infarction may give similar appearances on neuroimaging but, sparing of calcarine and paramedian occipital lobe structures suggests PRES. A further possible CT differential diagnosis is glioma. MRI is the imaging modality of choice as it resolves the hallmark subcortical vasogenic oedema of PRES from the cytotoxic oedema of acute cerebral infarction. This is seen as increased T2 or fluid-attenuated inversion recovery (FLAIR) sequence signal [4].

'Posterior reversible encephalopathy syndrome' is a misnomer. The imaging changes and clinical features may not be limited to the posterior cerebral hemispheres as in this case. The brain stem is involved in more than half of cases and the anterior and middle cerebral artery territories are involved in over 60%. Posterior circulation changes predominate due to the relative lack of efferent sympathetic innervations in this region. Reversibility of PRES may be clinically or radiologically incomplete and the condition may be complicated by ischemic or haemorrhagic stroke [4].

The first and the most important step in the treatment of such patients is supportive treatment. Then is the withdrawal of the aetiological factor if possible. Preventing hypertension and other triggers (immunosuppressive drugs, sepsis and the like) is the key. In this case, the aetiology was thought to be acute elevation of blood pressure as a result of eclampsia, and encephalopathy is because of disturbed blood pressure autoregulation. The patient condition rapidly improved after the convulsions stopped, and her blood pressure was normalised. Oral and IV. antihypertensive agents, sedative hypnotics, and

diuretics can be used to treat the hypertension. In our patient, generalized tonic-clonic seizures occurred while she was under the treatment of magnesium sulphate, and with the addition of antihypertensives, her condition rapidly regressed. Furosemide and dexamethasone were additionally given.

To conclude, PRES is a reversible condition and it is no longer limited to posterior circulation which depends on severity of underlying condition. The diagnosis is confirmed by radioimaging studies. If mental status disorders and generalized seizures are present in the presence or absence of preeclampsia/eclampsia in the perinatal period, one must suspect the possibility of PRES and should investigate. This paper is intended to remind obstetric anaesthesiologists and critical care specialists of the aetiology and differential diagnosis of PRES. Because, early recognition and institution of supportive therapy to stabilize the haemodymanics, ensuring oxygenation are all the more important as an adverse event during this period can lead to irreversible CNS damage even though PRES is generally considered as completely reversible.

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