Case report

Posterior Reversible Encephalopathy Syndrome Associated with Late onset Eclampsia: A Case Report

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Abstract

We describe a case of 25 year old women who had full term pregnancy, presented with symptoms of headache, loss of vision and seizure episodes after 7th postpartum day. There were no findings of pre-eclampsia in antenatal stage. The diagnosis of posterior reversible encephalopathy syndrome was made after magnetic resonance imaging. Imaging findings include vasogenic edema of bilateral occipital lobe and parietal lobe. Hypertension and endothelial injury seems to be involved in pathophysiology of the syndrome. After appropriate management, the syndrome resolved. Early diagnosis and proper treatment plays a key role in management of posterior reversible encephalopathy syndrome.

Keywords: Eclampsia, Posterior reversible encephalopathy syndrome, Seizure

Introduction

Posterior reversible encephalopathy syndrome (PRES) is a rare neuro-radiological entity manifested as headache, vomiting, seizures, visual disturbances, altered mental status and encephalopathy in various combinations. The characteristics finding in imaging is white matter vasogenic edema of bilateral parietal and occipital lobe [1, 2]. The various risk factors for PRES include acute and severe hypertension, exposure to certain drugs like chemotherapeutic drugs, immune modifying drugs and toxins [3]. The most notable incidence of PRES is seen in pregnant women with preeclampsia or eclampsia. Several studies have shown the incidence of PRES to be 20.0% to 98.0% among those suffering from preeclampsia and eclampsia [4, 5].

This is a case report of female patient who had uneventful antenatal period but presented with sudden onset loss of vision and eclampsia after 7th day postpartum and was diagnosed to have posterior reversible encephalopathy syndrome. The main objective of our case report is to contribute valuable insights into presentation, man-
agement and prognosis of PRES that occurs in late onset eclampsia.

Case report
It was a 25 year old pregnant woman with full term pregnancy. The patient had no history of hypertension or neurological mental illness. Blood pressure was normal during the pregnancy period. She was P2L1 mother with normal vaginal delivery. Child birth was uneventful and smooth with 3 kg live male baby with APGAR score of 8/10 and 9/10 at 0 min and 5 min respectively. After child birth she had normal blood pressure of 110/70 mm Hg and pulse 72 beats/min. She was discharged the next day with iron and calcium supplement and there was no other obvious discomfort in her early postpartum days. In the 7th postpartum day after delivery, she started having headache which was present around the occipital region and along with it she also had blurry vision. So for it she visited the nearby clinic for checkup. There she was managed conservatively and was referred to eye hospital. At that time period she had complete loss of vision. While returning to home she had one episode of abnormal body movement with up-rolling of eyes and foaming at the mouth. She was brought to Nobel Medical college teaching hospital for further management. On her way to hospital she had one more episode of similar attack lasting for 10 minutes.

At our center in the emergency department, patient was assessed and her vitals were as follows: BP 140/100 mm Hg, in right arm pulse 90 beats/min, RR 21 /min, SPO2 98% in right arm and temperature 98F. There was complete loss of vision, bilaterally pupils were round with normal size and the light reflex was normal. Bilaterally lung fields had equal air entry with normal vesicular breath sound, heart sound was normal with no added murmur. Per abdomen was soft and non-tender.

While at emergency patient suddenly had seizure at 7:46 pm, showing up-rolling of eyes, convulsions, foaming at the mouth with SPO2 98%. The provisional diagnosis of postpartum eclampsia was considered and immediate rescue measures with resuscitation were adopted as follows: oxygen administration, magnesium sulphate 5 g intravenous bolus followed by continue intravenous drip magnesium sulphate 1.5g/hour to control seizure, IV fluids, IV midazolam 5 ml in bolus. Patient seizure lasted for about 1 minute which was generalized tonic-clonic and then she went to sleep. Emergency laboratory test showed Hb 11.4 gm%, WBC count 13600, neutrophils 83%, lymphocytes 13%, ESR 35 mm/hr, slightly low potassium (3.2 mmol/L) and LDH 207 U/L. Her Renal function test, liver function test, red cell count, platelet count, blood coagulation and blood glucose were within normal range. She also had similar history 8 years back after her first child birth which was stillbirth. Similar history of raised blood pressure in her 7th post-partum day with seizure (lasted for short period and only 1 episode).

Brain CT was done which showed normal findings. Immediate Neurology, gynecology and ophthalmology consultation was done. During examination, patient was uncooperative, bilateral pupil diameter 2.0 mm, light reflex existed. Neurological examination showed bilateral Babinski’s sign negative, Chaddock’s sign negative, normal limb tendon reflex in her postictal phase.

She was shifted to general ICU for further management. In the ICU stay she had similar loss of vision with vision of perception of light. In ICU patient was started with IV Levetiracetam 500 mg iv stat and then twice daily despite magnesium sulphate to control seizure, IV fluids and midazolam was continued. Brain MRI was planned for the next day.

Brain MRI revealed, T2W1/FLAIR showing high signal involving the subcortical white matter of bilateral occipital lobes, bilateral parietal lobes and left temporal lobe. No diffusion restriction, blood products or abnormal enhancement was seen. Rest of the neuro-imaging showed normal morphology (Figure B-F). Provisional diagnosis of posterior reversible encephalopathy syndrome due to late onset eclampsia was made. After initiation of afore mentioned treatment, the patient’s vision improved to counting fingers on bilateral eye by following day evening. On the 3rd day of stay her vision improved to 6/6 with persistent mild headache. The patient was recovering well and shifted to neuro-medicine ward. Patient was admitted for 5 days and was discharged on oral Levetiracetam 500 mg BD. With the improvement of signs and symptoms, analyzing neuro-imaging (MRI) and laboratory findings, the final diagnosis of this patient that was made was late onset postpartum eclampsia associated with posterior reversible encephalopathy syndrome (PRES).
postpartum eclampsia of late onset [6]. Preeclampsia is a systemic syndrome in pregnancy that develops after 20 weeks of gestation and is characterized by high blood pressure and proteinuria. Presence of seizure of unexplained etiology when occurs together with preeclampsia establishes the diagnosis of eclampsia. Preeclampsia is known to precipitate PRES due to hypertension, endothelial dysfunction and vasospasm [7]. The secondary data analysis of WHO multicountry survey by Abalos et al. showed the prevalence of preeclampsia for Asian regions as 2.07% in China, 1.19% in Japan, 2.22% in Thailand and 0.59% in Nepal and the prevalence of eclampsia globally to be 0.3% [8]. Zhang et al. and Masai et al. found similar cases of PRES in late onset eclampsia [9, 10].

Seizure and headache was the most prominent finding in our case. Headache was present on occipital region. Headache in PRES is generally dull and diffuse but can also progress to severe headache. There was typical three episodes of seizures in our case. We managed our case with levetiracetam and midazolam. These drugs work by enhancing the effect of gama-aminobutyric acid (GABA) in the brain. Seizure may progress to status epilepticus in upto 18 % of the cases [11]. Loss of vision was also one of the major findings in our case which lasted for three days and was gradually reversed. Loss of vision is known to cause by the swelling and compression of brain tissue. Since visual cortex is present in back of the brain, vision generally gets affected. The visual changes may manifest as diminished acuity, visual field-deficits, visual hallucinations and can also manifest as blindness. Similar to our case visual disturbance was found as prominent findings by different authors [12, 13].

Magnetic resonance imaging (MRI) in our case showed vasogenic edema of white matter in bilateral occipital lobes, bilateral parietal lobes and left temporal lobe. Initially CT scan was done which showed no changes (Fig. A-F). Neurological examination was also normal, but plan to shift to MRI was recommended because when available it is always preferred over CT as fluid-attenuated inversion recovery and T2 weighted sequences are more sensitive to vasogenic edema. Involvement of posterior occipital and parietal region simultaneously, as shown in our case has been reported by reviewing authors [2]. The typical involvement of occipital lobes is attributed to less sympathetic innervation in posterior cerebral region rendering it more susceptible to injury with change in blood pressure [14]. There was almost complete reversal of sign and

**Discussion**

Posterior reversible encephalopathy syndrome (PRES) is an acute neurological disorder characterized by a range of symptoms, including headaches, seizures, altered consciousness, and visual disturbances. It is typically associated with rapidly rising blood pressure, certain medical conditions, or use of certain medications.

Preeclampsia and eclampsia are one of the major cause of PRES. In our case there was no noted finding of preeclampsia in antenatal check-up but presented with eclampsia after 7th postpartum day. Patient presented to us with three episodes of seizure and blood pressure of 140/100 mm Hg. The condition is called as
symptoms in the patient and with supportive care and treatment at neuromedicine ICU and ward. After 5 days patient could be discharged with oral medications. PRES due to eclampsia is generally found to have good outcome with appropriate treatment in contrast to other cause.

Conclusion
PRES as a manifestation of late onset eclampsia is a rare finding. Headache, seizure and visual disturbances are common findings. Hypertension, endothelial dysfunction and vasospasm are known to contribute to the disease. Prompt diagnosis by clinical awareness and imaging and appropriate treatment along with supportive care can reverse the disease uneventfully.

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References