Posterior Reversible Encephalopathy Syndrome due to Eclampsia in Term Pregnancy

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We report a 30 years primigravidae presented in term pregnancy with eclampsia with intrauterine foetal death and underwent instrumental delivery. Peripartum management of eclampsia is always challenging for anaesthesiologist and obstretrician. The case was managed under general anaesthesia and kept on mechanical ventilation for three days. Postpartum cranial computed tomography revealed low attenuated area in right basal ganglia. Low attenuated area in bilateral frontal and parietal lobes with subtle gyral high density in bilateral frontal lobes. Report was suggestive of posterior reversible encephalopathy syndrome. Clinical improvement was observed with supportive treatment and extubated on the third postpartum day. Posterior reversible encephalopathy syndrome is a cliniconeuroradiological syndrome associated with the various conditions including severe hypertension and seizures. Eclampsia is one of the most important causes of posterior reversible encephalopathy syndrome.

Keywords: eclampsia; posterior reversible encephalopathy syndrome; preeclampsia.

INTRODUCTION

Preeclampsia is a multi-organ system disorder that occurs after the 20th week of gestation and is characterised by hypertension and proteinuria with or without oedema.[1] When the diastolic blood pressure is more than 110 mmHg and protein is above 3 gm/day, the condition is called severe preeclampsia. Preeclampsia and its variants affect approximately 5% of the pregnancies and remain the leading causes of both maternal and foetal morbidity and mortality worldwide.[2] The incidence of eclampsia occurs in approximately 0.5% of the patients with mild preeclampsia and 2% to 3% of those with severe preeclampsia. Depending on the systemic involvement, several other symptoms such as coagulopathy, renal or liver failure, HELLP (haemolysis, elevated liver enzymes and low platelets) syndrome also complicate the clinical picture and recent entity of posterior reversible encephalopathy syndrome (PRES).

PRES was first described in 1996 by Hinchey et al[3] and was named as reversible posterior leukoencephalopathy syndrome. It is a transient clinicoradiological syndrome, first noted in patients with hypertensive encephalopathy. PRES has been associated with many conditions including eclampsia, severe hypertension, autoimmune disease, immunosuppressive agents and sepsis. It can be reversible with prompt diagnosis and treatment without any residual neurological deficit. Delay in diagnosis and management can result in permanent damage to brain tissues. The incidence of PRES in association with eclampsia is still unknown. We presented one case of term pregnancy associated with eclampsia with PRES and completely recovered without any neurological deficits.

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CASE

A 30 years primigravidae was referred from Jiri Hospital, with the diagnosis of term pregnancy with eclampsia. She was managed there conservatively with loading dose of magnesium sulphate 4 gm intravenous slowly and 5 gm intramuscular in each buttock, paracetamol 300 mg intramuscular, diazepam 5 mg intravenous, nifedepine 10 mg per oral and referred to our hospital for better management on the same day. She had severe headache for three days and multiple episodes of loss of consciousness followed by generalised seizures, initially in every 2-3 hours, later every half hourly and lasted for one minute on average. Her personal and past history was not significant.

On our emergency room, patient's general condition was poor, pulse rate was 110 beats/min, blood

pressure was 80/40 mmHg, and respiratory rate was 20/min. On neurological examination, her Glasgow Coma Scale was 4/15 (E1M2V1), pupil was bilaterally equal and reacting to light, and bilateral plantar reflexes were absent. On chest auscultation, she had bilateral basal crepitation. On abdominal examination, fundal height was term size, fetal heart sound was not heard, and on per vagina examination cervix was fully dilated. Other systemic examinations were within normal limits.

Blood investigation reports were within normal limits except uric acid was 9.9 mg%, urea- 83 mg%, creatinine- 2 mg%, lactate dehydrogenase- 1392 U/L and urine analysis revealed 2+ proteinuria. A provisional diagnosis of primigravidae with term pregnancy in the second stage of labour with eclampsia with intrauterine foetal death (IUFD) with hypoxic brain injury was made and she was immediately transferred to the operation theatre for an emergency instrumental delivery under general anaesthesia.

In the operation theatre, preoxygenation was done and premedicated with metoclopramide 10 mg, ranitidine 50 mg and pethidine 30 mg IV. Rapid sequence induction with cricoid pressure was done with ketamine 50 mg, propofol 50 mg and suxamethonium 100 mg and intubated with 7.0 mm internal diameter endotracheal cuffed tube and maintained with oxygen, isoflurane, and vecuronium. Patient was kept on intermittent positive pressure ventilation. After forceps delivery, oxytocin 3 international units, hydrocortisone 100 mg and tranexamic acid 1 gm were given intravenously. The baby was 2800 gm male with Apgar scores 0/10 and 0/10 at 1 and 5 minutes respectively. Intraoperative period was uneventful. Although patient's spontaneous respiration was noticed, she did not gain her consciousness. So, we transferred her to the maternal intensive care unit (MICU) with endotracheal tube *in situ* and maintained on mechanical ventilation on synchronized intermittent mechanical ventilation mode. In MICU, midazolam 1 mg and morphine 1 mg were given via syringe pump every hour. Amlodipine 5 mg twice daily and enalapril 2.5 mg once daily via nasogastric tube were started and continued along with antibiotics and heparin 5000 units subcutaneously twice daily. On the first day, endotracheal tube was changed due to partial blockage. As she regained consciousness gradually and improved spontaneous breathing, she was extubated on the third day. On the second postpartum day, CT scan of the head was done and the report showed low attenuated area in right basal ganglia, frontal and posterior parietal lobes and subtle high-density area was noted in gyral part of both frontal lobes. With supporting CT scan report and clinical presentation, the patient was diagnosed as posterior reversible encephalopathy syndrome due to eclampsia. She was discharged on the fifteenth postpartum day without any residual neurological deficits.

COMMENT

The onset and intensity of PRES varies and are often nonspecific. It is characterised by variable associations of seizure activity, consciousness impairment, headaches, cortical visual abnormalities/blindness, nausea/vomiting and focal neurological signs. Headaches are typically constant, dull, non localised, and unrelieved by medications but resolve as blood pressure is normalised.[4] Mental status changes may vary from general malaise to confusion, decreased level of consciousness and coma. Generalised tonic clonic seizures are often the presenting symptom. A single seizure is infrequent; multiple seizures are more frequently reported. In our patient, we did not find any visual abnormalities, but had severe headache associated with vomiting and followed by multiple episodes of seizure. Hinchey et al[3] described association between eclampsia and PRES, three out of fifteen patients were associated with eclampsia and other etiologies included hypertensive encephalopathy and immunosuppressive medications. Preeclampsia/eclampsia is one of the common causes of PRES and most cases are managed without neuroimaging and the incidence remains unknown. However, it is uncertain whether a cause and effect relationship truly exists between the two or if these represent independent processes with some element of clinical overlap.[5] Fujiwara et al[6] and Mackinney et al[7] have reported that the cause of PRES was eclampsia in 5.5% of patients.

The lesions are seen mainly in the posterior regions of the cerebral hemispheres.[8] These abnormalities partially or completely resolve on follow up scanning, thereby suggesting subcortical oedema without infarction. Our patient had low attenuated area in right basal ganglia and bilaterally low attenuated area in frontal and posterior parietal lobes on CT scan. It

is important to note that neuroimaging usually reveals sparing of the calcarine and paramedian occipital lobe structures, a fact that distinguishes PRES from bilateral infarction of the posterior cerebral artery territory. Although MRI yields higher resolution and may show small, focal abnormalities beyond resolution of CT, it is not mandatory for diagnosis of PRES. [3]

Differential diagnosis of PRES like demyelinating diseases, basilar artery embolism and venous sinus thrombosis should be ruled out. In our patient, etiology was eclampsia because of altered blood pressure autoregulation. Her condition was improved after seizures stopped and blood pressure was normalised with delivery of the foetus.

Recurrent attacks of PRES are mainly related with eclampsia, and their incidence is proportional to recurrent eclampsia.[9,10] With early diagnosis and treatment, patients can recover clinically in a few weeks without any neurological deficit. If not treated on time, the condition can get worse, resulting in cerebral ischaemia, infarcts and even death. As there are no clinically specific signs for the syndrome, it can often be confused with other clinical conditions, leading to mismanagement.[11]

Magnesium sulphate should be initiated as soon as eclampsia is suspected to control seizures. However, during general anaesthesia, in patients receiving standard doses of magnesium sulphate, the effect of neuromuscular blocking agents can be more potential, and their duration can be prolonged.[12,13] Drugs with low biotransformation rates like isoflurane, low renal clearance, short half-life, and low active metabolites like atracurium should be chosen for general anaesthesia. Nitroglycerin and magnesium sulphate are suggested to prevent hypertensive attacks that can occur during the induction of anaesthesia.[14,15] For induction we gave ketamine and propofol combination because her blood pressure was low and she was critically ill. Cohen et al[16] concluded that the use of ketamine did not have any sustained changes in intracranial pressure or cerebral perfusion pressure, which adversely affect patient outcomes, including mortality and neurologic outcome. For maintenance of anaesthesia, isoflurane and vecuronium as a neuromuscular blocking agent were used.

Limitation of our study is that we could not provide her CT scan film as well as could not do follow-up scan. However, we examined her on first follow up after two weeks of discharge. She had no neurological deficits and for that we regularly contacted her through phone calls. She can do everything by her own and other daily activities.

CONCLUSIONS

In emergency room, this patient was diagnosed as primigravidae at term pregnancy in second stage of labour with eclampsia with intrauterine foetal death with hypoxic brain injury and supportive management was done accordingly. However, postoperative CT helped us to diagnose posterior reversible encephalopathy syndrome. Thus, this case report emphasises the need for early diagnosis and prompt treatment of it to decrease long term neurological sequelae.

DISCLOSURE

The authors report no conflicts of interest in this work.

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