Case Report

A case of eclampsia with cerebral hemorrhage in the postpartum period

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Abstract Intracerebral hemorrhage is an infrequent but severe complication in pregnant women with hypertension. We describe a typical case of a patient with eclampsia who developed spontaneous intracerebral hemorrhage in the postpartum period. High degree of suspicion, MRI imaging, prompt intervention and multidisciplinary management helped in full recovery of the patient.

Keywords: Eclampsia, intracerebral hemorrhage, postpartum, magnetic resonance imaging, stroke.

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INTRODUCTION

Pre-eclampsia and Eclampsia is a multi-system disorder and is associated with significant maternal morbidity and mortality. Cerebral hemorrhage though rare in young women is one of the main causes of maternal morbidity and mortality seen in eclampsia and preeclampsia. Hence early recognition and proper management may serve to avoid serious maternal complications.

CASE DESCRIPTION

A 20 years old female, primigravida with 40 weeks of gestation was admitted with four episodes of generalised tonic-clonic convulsions. Her BP was 180/100 mm of Hg with pulse rate of 92/min and 3+ proteinuria. A diagnosis of eclampsia was made and treatment was commenced with Magnesium Sulphate. She received 5g intramuscular in each buttock and 4g in 200ml of normal saline infused intravenously over 20 min and a maintenance dose of 2gm in every four hours. Blood tests were ordered for complete blood count, liver function test, coagulation profile, urea and electrolytes. The reports were normal. The patient was admitted to the obstetric high care unit for observation and stabilization. Emergency caesarean section was done for fetal distress under spinal anaesthesia. A healthy infant of birth weight 2.2kg with AGPAR score of 8 and 10 at 1 and 5 minutes respectively was delivered. Post-operatively, the patient was conscious and oriented. Her BP was 150/100 mm of Hg, Tb Depin 10 mg every 8 hourly was started and Injection $MgSO_4$ was continued for 24 hours after caesarean section. On Day 3 onwards, the patient started complaining of severe frontal headache. Her BP was 150/108 mm of Hg Tb Depin R 20 mg bd was started. On Day 4, MRI Brain with MR Venography was done.

MRI Brain study revealed:

- Streak of subdural hematomas encircling bilateral cerebral hemispheres.
- Subcortical bleed in right high parietal lobe with surrounding edema.
- White matter edema in left frontal and temporal lobes.
- Subarachnoid hemorrhages in left sylvian fissure, frontoparietal sulci.
- Hyperintense signals on T1W images in pituitary fossa, pituitary hemorrhages.

MR venography study revealed no significant abnormality.

Expert opinion of neurologist and neurosurgeon was taken. Injection mannitol 150cc iv stat and followed by 100cc iv 8 hourly, Injection phenytoin 100 mg iv 8 hourly, Injection Dexamethasone 8 mg iv 8 hourly was started. Her BP was 160/112 mm of Hg which was

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controlled by Tab Nimodipin 30 mg four hourly and Tab Atenolol 25 mg bd and sodium nitroprusside infusion. Adjuvant therapy i.e. Inj Citistar 500 mg iv 12 hourly and Inj Nootropil 500 mg iv 8 hourly was started. Blood investigation in the form of complete blood count, coagulation profile and Liver function test and serum electrolytes and serum calcium were done and were within normal limits. Satisfactory blood pressure control was achieved on Day 12 and sodium nitroprusside infusion was stopped. The patient was completely relieved of headache on Day 14. On Day 20, the patient was discharged on Tab Nimodipin 30 mg 8 hourly and Tab Phenytoin 100 mg 8 hourly. On discharge, the patient was conscious, oriented and general condition was good.

DISCUSSION

The case illustrates a classic presentation of a patient with eclampsia who develops ICH, a rare but grave complication of pregnancy. ICH accounts for 5% to 12% of all maternal deaths. Known risk factors for lCH are maternal age > 35, African-American race, tobacco dependence, substance abuse, coagulopathy or previous pre-eclampsia / eclampsia. Higher incidence of hemorrhagic stroke is reported in Asian women compared to Caucasian patients, most commonly occurring in the prepartum stage (58%). Cerebrovascular malformations are evident in 20-67% of patients with pregnancy related ICH. In our case, no underlying vascular malformation or aneurysm was found on MR venography. The vascular tissue structure of the brain, the changing of coagulation status during pregnancy and the pathological state of pre-eclampsia such as endothelial dysfunction could have contributed in our case to ICH. ICH in this case was due to the escalation in the BP. A sudden increase in BP can lead to hypertensive encephalopathy in pregnant women without a history of hypertension even with a diastolic BP around 100 mm of Hg.The current management protocols are based on elevated diastolic blood pressure. In contrast to this protocol, women with high systolic blood pressure are at high risk for hemorrhagic stroke and that antihypertensive therapy should be considered in these patients. Additional studies in patients with pre-eclampsia/eclampsia found a moderate decrease in cerebrovascular resistance together with increased cerebral blood flow velocities causing cerebral hyperperfusion, losses of auto-regulatory mechanisms and eclampsia with ICH. The most common initial sign is the sudden onset of severe headache. Multiple thunderclap headaches recurring nearly every day over 1-4 weeks are almost pathognomonic. Other symptoms include nausea, vomiting, visual disturbances, seizure and coma. This case emphasizes that the two distinct but related cerebral pathologies include gross intracranial hemorrhage due to rupture of arteries caused by severe hypertension and diffuse cerebral oedema. With improved CT scan and MRI, all women with eclampsia are found to have abnormal brain finding. Imaging studies should be advocated whenever there is a doubt and in cases of eclampsia. Expert opinion of neurologists and neurosurgeons should be taken depending on the imaging findings. Control of blood pressure is of utmost importance in cases of severe pre-eclampsia to prevent CNS complication. With sudden blood pressure elevation, vasogenic edema worsens and consideration should be given for treatment with mannitol and dexamethasone.

CONCLUSION

Even short time hypertension should be treated aggressively to prevent ICH. Even though eclampsia can cause seizures with no ICH, obstetrician should be suspicious about ICH. The prompt intervention of a multi-disciplinary team (obstetric, neurologist, neurosurgeon and anesthesiologist) is required to ameliorate the devastating effects of eclampsia and ICH.

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