Original Research Paper

Volume - 7 | Issue - 7 | July - 2017 | ISSN - 2249-555X | IF : 4.894 | IC Value : 79.96

Clinical Research

A RARE CASE OF IMPENDING ECLAMPSIA WITH POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME FOR EMERGENCY LSCS

Dr. Gayatri Khanvelkar (Senior Resident),

Dr. Mary Samuel (Professor)

Dr. Adnan Qadri (Post Graduate Student)

ABSTRACT Posterior reversible encephalopathy syndrome (PRES) is a cliniconeuroradiological syndrome associated with various clinical conditions, presenting with headache, encephalopathy, seizures, cortical visual disturbances or blindness. Imaging predominantly shows parieto-occipital white matter changes, with vasogenic oedema being the most accepted pathophysiology. We report a 22-year-old primigravida who presented in term pregnancy with seizures and blindness, scheduled for emergency caesarean section. She was managed peri-operatively under general anaesthesia and shifted to intensive care unit. Postoperative MRI brain revealed ill-defined hypointensine lesions in the bilateral parietio-occipital and bilateral fronto-parietal regions involving white matter in T2 and FLAIR and hypointensie lesions on T1W1, few foci of restricted diffusion noticed on DW1. Similar foci were noted on bilateral lentiform nuclei and adjoining posterior limb of the internal capsule. Both eyeballs showed hyperintense lesions in the posterior segment on the temporal side on FLAIR: retinal detachment due to haemorrhage in the retinal space, suggestive of PRES. Clinical improvement with complete resolution of visual disturbances was observed with supportive treatment. The importance of prompt suspicion and management in preventing short- and long-term neurological deficits in reversible condition like PRES is highlighted.

KEYWORDS : Leukoencephalopathy, posterior leukoencephalopathy syndrome, posterior reversible encephalopathy syndrome, pregnancy, reversible posterior cerebral oedema syndrome, reversible posterior leukoencephalopathy syndrome

Case Details:

A 22yr old primigravida with 36.5 weeks of gestation came to the OPD with chief complaints of inablity to see since morning. She gave a history of sudden painless loss of vision, 5 hours prior to admission. She did not give any history of pain in abdomen, nausea, vomiting, headache or convulsions, bleeding diathesis or per vaginal leak/discharge/ bleeding. Her menstrual and obstetric history were within normal limits.

Patient was diagnosed as a case of pre-eclampsia 10 days ago and was started on Tab. Alpha dopa 250mg tds and Tab. Depin 5mg tds in a private hospital. She however did not have any other comorbidities.
On examination, her general condition was irritable. She was afebrile with a pulse of 90/min, BP- 170/110 mm Hg rt. Arm, left lateral position and RR- 18/min, thoracoabdominal breathing. She had pallor + and pedal oedema +++ (Grade V). On fundoscopy she had grade II hypertensive retinopathy.

On examination of the central nervous system, patient was irritable, conscious, uncooperative, disoriented to time, place and person. She had the inability to see, with no perception of light and no projection of rays but her deep tendon reflexes were present. Her other systems were within nirmal limits.

On investigations, everything was within normal limits except for Hb : 9.6 and urine ketones were +2.

Thus the patient was diagnosed as a primigravida with 36. 5 weeks of gestation, impending eclampsia (with visual disturbances). She was administered Inj. Magnesium sulphate according to the Pritchard's regimen (4gm iv plus 5gm im in each buttock) and Inj. Ranitidine 50mg iv, Inj. Metoclopramide 10mg iv and iv antibiotics were given prior to shifting the patient to the operation theatre.

Anaesthetic Management:

She was accepted for emergency LSCS under ASA III (E) under general anaesthesia. She was induced with rapid sequence induction. Airway was secured with cuffed endotracheal tube of 7mm internal diameter and anaesthesia was maintained with oxygen, nitrous oxide (1:1) and isoflurane (0.5-1%) and Inj. atracurium (20mg) i.v to facilitate muscle relaxation. Inj. Oxytocin 20 U i.v infusion was given after the delivery of the baby.1.8 kg male child was born and was shifted to the neonatal intensive care unit in view of low birth weight. Inj. Fentanyl (60 mcg) i.v. and inj. Midazolam 1mg IV was given after the delivery of the baby. During total surgery of 1 hour, her vitals remained stable and was reversed from neuromuscular blockade and was shifted to the ICU for further observation and management.

In the ICU, continous monitoring was done for vitals and for deep tendon reflexes. However she was still irritable with inability to see but was understanding verbal commands. But her blood pressure started rising up gradually after 2 hours of surgery. Hence Inj. Labetelol 10 ml/hr (20mg/hour) was started as an infusion and an additional dose of magnesium sulphate was given. In spite of this line of treatment, at the 6thpost-operative hour patient had 1 episode of general tonic clonic seizure. Inj. Midazolam 2 mg i.v. stat was given and Inj. Labetelol was titrated to a higher dose according to the blood pressure (15-20ml/hour with maximum dose not more than 200mg). Inj. Lorazepam 2mg IV and Inj. Levipril 500mg IV twice a day was started and Tab. Minipress (Amlodipine + Prazosin) was crushed and was administered via the Ryle's tube.

In view of worsening of the symptoms, urgent ophthalmology and neurophysician reference was done to rule out other possible causes of neurological symptoms. Repeat fundoscopy revealed papillodema with resolving exudative retinal detachment most probably secondary to eclampsia. MRI brain was suggestive of: Posterior reversible encephalopathy syndrome with retinal detachment due to haemorrhage in the subretinal space.

After diagnosing PRES syndrome, Inj. Mannitol was included in the treatment chart. Aggressive treatment on the same line of management was continued with strict monitoring of vitals. On the 3rd post-operative day the patient got back perception of light. On the 4th post-operative day there was further improvement in vision, decrease in the other signs of neurological irritability. Inj. Labetelol and Inj. Levipril was gradually tapered off and patient was shifted to the ward on post-operative day 8.

Discussion

PRES syndrome is also known as reversible posterior cerebral oedema syndrome, reversible posterior leukoencephalopathy syndrome (RPLS), reversible occipitoparietal encephalopathy syndromeand hypertensive encephalopathy syndrome. It was first described by: Hinchey ET. Al in 1996. The clinical symptomatology ranges from headache, nausea, encephalopathy, seizures, and cortical visual disturbances/blindness to coma. It is associated with hypertensive Encephalopathy, renal Failure, autoimmune disorders: Henoch

Schnolein Purpura, SLE, sepsis, shock and treatment with immune-suppressants and cytotoxic drugs. PRES syndrome though rare has been known to be associated with pre-eclampsia and eclampsia from 28 weeks of gestation to 13 days of postpartum period. Despite the marked neurological and radiological picture the syndrome is reversible. However delayed diagnosis can lead to cerebral ischemia, cerebral infarction and death; hence prompt intervention is essential.

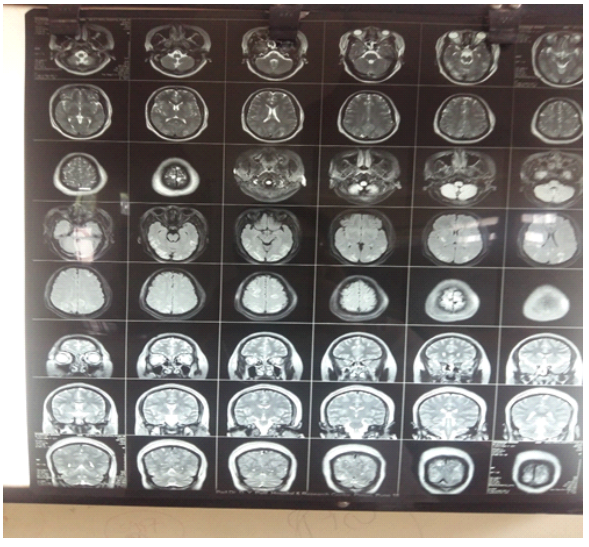
The pathogenesis lies in the loss of cerebral autoregulation and excessive arteriolar (cerebral vasoconstriction). Hyperperfusion and decreased blood flow causes breakdown of the blood brain barrier leading to ischaemia, vasogenic oedema and cytotoxic oedema. The preferential involvement of the posterior circulation has been postulated to be due to the sympathetic innervation protecting the brain from sudden increase in blood pressure being relatively less in the arterioles supplied by the vertebral- basilar system than in the anterior circulation. It is labelled as atypical PRES when there is involvement of: frontal lobes, temporal lobes, cerebellum, thalamus, brainstem.

Anaesthetic Challenges

An anaesthetist faces challeneges in such cases due to atypical presentations being often confused with other differential diagnosis. Cardiovascular instability is often associated with PRES. Increased blood pressure itself poses as a major challenge. Mild fluctuations in blood pressure during or after anaesthesia can worsen PRES. It should be treated promptly with magnesium sulphate. (Nitroglycerine is contraindicated.) Changes in S.electrolyte levels (especially magnesium) have to be checked as it can worsen PRES. Raised intracranial pressure and loss of cerebral autoregulation makes anaesthesia further complicated.

Conclusion:

This case emphasises the need for early diagnosis and prompt treatment of PRES to avert short and long term neurological sequelae. Though the association of PRES and PIH is well documented, the cause and effect relationship is unknown. The possible presence of PRES with proper preoperative neurological evaluation, vigilant intra-operative monitoring and postoperative intensive care management (SICU), should be considered in pregnant patients presenting with seizures and blindness.



References:

1. Hinchey J, Chaves C, Appignani B, Breen J, Pao L, Wang A, et al. A reversible posteriorleukoencephalopathy syndrome. Engl J Med. 1996;334:494–500. Shreepathi Krishna Achar
2. , Nanda Shetty, and Tim Thomas Joseph. Posterior reversible encephalopathy syndrome at term pregnancy. Indian J Anaesth. 2011 Jul-Aug; 55(4): 399–401.doi: 10.4103/0019-5049.84856
3. Long TR, Hein BD, Brown MJ, Rydberg CH, Wass CT. Posterior reversible encephalopathy syndrome during pregnancy: Seizures in a previously healthy parturient. J Clin Anesth. 2007;19:145–8.
4. Bartynski WS, Boardman JF. Distinct imaging patterns and lesion distribution in posterior reversible encephalopathy syndrome. AJNR Am J Neuroradiol. 2007;28:1320–7.
5. Casey SO, Sampaio RC, Michel E, Truwit CL. Posterior reversible encephalopathy syndrome: Utility of fluid-attenuated inversion recovery MR imaging in the detection of cortical and subcortical lesions. Am J Neuroradiol. 2000;21:1199–206.
6. Thackeray EM, Tielborg MC. Posterior reversible encephalopathy syndrome in a patient with severe preeclampsia. Anesth analg. 2007;105:184–6.
7. Swende TZ, Abwa T. Reversible blindness in fulminating preeclampsia. Ann Afr Med. 2009;8:189–91.
8. Finsterer J, Schlager T, Kopsa W, Wild E. Nitroglycerin-aggravated pre-eclamptic posterior reversible encephalopathy syndrome (PRES) Neurology. 2003;1:715–6.
9. Rangi PS, Partridge WJ, Newlands ES, Waldman AD. Posterior reversible encephalopathysyndrome: A possible late interaction between cytotoxic agents and general anaesthesia. Neuroradiology. 2005;47:586–90.
10. Schwartz RB, Mulkern RV, Gudbjartsson H, Jolesz F. Diffusion-weighted MR imaging in hypertensive encephalopathy: Clues topathogenesis. AJNR Am J Neuroradiol. 1998;19:859–62.
11. Pratap JN, Down JF. Posterior reversible encephalopathy syndrome: A report of a case with atypical features. Anaesthesia. 2008;63:1245–8.