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Anesthetic management of parturient with undiagnosed posterior reversible encephalopathy syndrome at term: A case report

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Abstract

Use of prompt and efficient anesthetic management of an actively convulsing gravid to term female in the OPD. In this case report, we present a successful anesthetic induction and management in a case of gravid to term female with active convulsions and complaints of vomiting and headache, later diagnosed as Posterior Reversible Encephalopathy Syndrome under General anesthesia.

Keywords: PRES, eclampsia, hypertension in pregnancy, leukoencephalopathy, obstetric anaesthesia

Introduction

PRES is an increasingly recognised disorder with a wide spectrum of symptoms and aggravating factors. Most of these patients are hypertensive, however a significant proportion have normal to mildly raised BP^[1], presenting with rapid onset of symptoms, including headache, seizures, altered consciousness & visual disturbances^[2]. On a T2 weighted FLAIR MRI, white matter lesions in occipital, posterior parietal and posterior temporal lobes, in the typical order are classical findings^[1]. With rapid onset of symptoms, if promptly recognised & treated, it resolves within a week^[3]. MRI changes resolve in days to weeks^[3]. Rapid diagnosis is of utmost importance.



Fig 1: Trolley prepared for an emergency case. Includes Functioning Laryngoscopes with MAC Blades, Guedel's Airways, Emergency i/v drug tray and Vapourier with Inhaled anaesthetic agent.

A 25 year old female came to MGM Women & Children Hospital, Kalamboli OPD in a semiconscious state, primigravida (By date 37.1 weeks, by scan 37.3 weeks). With history of fall at home earlier the day, complaints of headache (sudden in onset and moderate grade) and 3 episodes of vomiting (projectile, non-blood stained, containing food particles), followed by 1 episode of convulsion (abnormal movement of limbs, with up-rolling of eyeballs, frothing from the mouth) at 7 am on 4th of January 2021. Her Blood pressure was 160/100 mmHg, pulse-98/min, SpO₂- 98% on room air with clear chest on auscultation and bilateral pedal edema on physical examination. The urine tested positive for the presence of albumin (+2). Preoperatively, Inj. Labetalol 20 mg IV Stat was given with loading dose of Magnesium

Sulphate as per the regimen (Intravenous bolus 4 gm loading dose, 5 gm intramuscularly into each buttock followed by a maintenance dose of 5 gm intramuscular every 4 hour), followed by infusion at 20ml/hr, in 24h after the last seizure. Patient had 2 more episodes of convulsions in the OPD, following which a bite block was inserted, preventing the tongue bite. Patient was given Inj. Lorazepam 2 mg i/v and Inj. Labetalol 20 mg IV stat with vitals of BP-150/100mmHg, Pulse – 118/min and SpO₂-98% on room air. In addition to this, the patient was provided with supplemental oxygen via Hudson mask. The chest was clear and urine output was 50 ml. The patient previously had Antenatal checkups which were normal, and no abnormality was detected.



Fig 2: An Anaesthetic Workstation and Ventilator Machine



Fig 3: Intraoperative image.

With preoperative Haemoglobin of 13gm%, TLC of 9000/mm³ and platelet count 1.23 lacs, the patient was shifted to the Operating Room and taken up for emergency LSCS under GA. The operating room was prepared for difficult airway and emergency drugs loaded for necessary resuscitation measures.

Pre-medicated with Inj. Glycopyrrolate 0.2 mg, Inj. Loxicard 3 cc, patient was induced with Inj. Propofol 140 mg, and

Scoline 50 mg intravenously. Endotracheal intubation with Tube size 7.0 under vision using direct laryngoscopy was attempted. Under bougie assistance, the tube was slid over it, owing to Cormack-Lehane Grading IV. With successful intubation in ramp position, further emergency LSCS was proceeded with.

After the baby was delivered out, inj. Pitocin in 20IU I/M and 20IU I/V was administered, along with Inj. Fentanyl 50mcg i/v. Further, Inj. Levitracetam 2g IV and 1 g of Inj. Tranexamic acid was given slowly over 30minutes. Intraoperatively, 40 mg of Inj. Atracurium was administered. The patient was maintained on Sevoflurane, oxygen and air. In addition to this, the patient was transfused two units of Packed Cell Volume (PCV), in view of blood loss intraoperatively.

Intraoperative extubation wasn't planned since any intracranial pathology hadn't been ruled out.

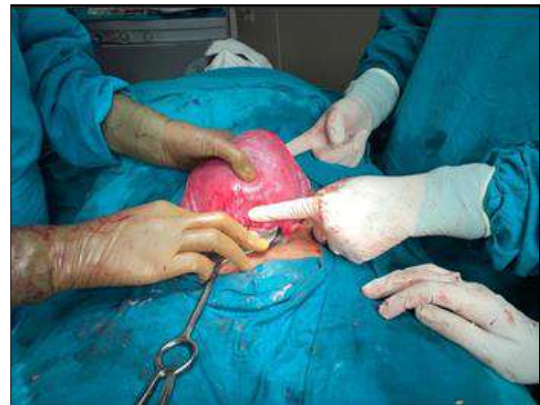


Fig 4: Intraoperative image of a uterus post baby out.

The patient was shifted to the Medical ICU with ETT in situ under close observation and with due precautions post operatively. Post op vitals: BP-120/90mmHg, pulse-90/min, spo₂-98%. Eventually, her haemoglobin dropped to 10gm%, TLC raised to 19,240 and platelet count of 1.25 lacs. However, further the same day, the haemoglobin drastically dropped to 6.7 gm% and platelets to 7.9 lacs. Keeping the presence of blood clots without an active bleed, patient was transfused 2 units of PCV, 2 units of Fresh frozen plasma (FFP) and 2 units of Cryoprecipitate in the MICU, in order to prevent disseminated intravascular coagulation (DIC) or any further complications.

CT Scan Brain revealed few hypodense areas in cortical and subcortical region of right parietal lobe and right occipital lobe with premature mild cerebral atrophy and mega cisterns magna. 2D echo report was normal with Left ventricular ejection fraction of 50-55%. With intravenous Midazolam infusion, the patient was sedated until evening when the sedation was finally weaned off. She was conscious, with responsiveness to voice commands and movement of all 4 limbs.

Under neurology reference, the patient was started on Inj. Levetiracetam 500 mg i/v BD and Inj. Phenytoin 100 mg i.v TDS. With due considerations, the patient was put on tab. Nicardipine – sustained release 20 mg R/T QID and Inj. mgSo₄ as per the regimen. Later, 2 units of cryoprecipitate were transfused. Further ruling out all the causes of altered sensorium, blood pressure was controlled, with a successful weaning trial, the patient was safely extubated and put on oxygen support.

The next day (day 2), the patient had bilateral blurring of vision with perception of light. Later, with improving vision, she could follow voice commands, finger movements and able to localise pain. Neurology reference later impressed on the probable diagnosis of PRES; Posterior Reversible Encephalopathy Syndrome with recurrent seizures and vision abnormality, ticking all the symptoms for diagnosis. Day 3 examination showed improved vitals and normal investigations. With full restoration of vision, normal investigations, the patient was mobilised on day 4. The patient was transferred to wards on day 5, further discharged on day 11.

Discussion

Preeclampsia is an insidious disease occurring in over 4-7% of all pregnancies. One of the serious complications of preeclampsia is PRES; Posterior Reversible Leuko-Encephalopathy Syndrome. Evolving over a matter of hours, it manifests as headache, visual abnormalities, seizures, with nausea and altered mental state or confusion^[4]. Typical signs of PRES are best detected by T2 – weighted and fluid attenuated inversion recovery (FLAIR) MRI, the gold standard investigation^[5]. CT scan only reveal 50% of the lesions. Typical findings on MRI include symmetric edema involving the white matter of the posterior regions of the cerebral hemispheres. White matter lesions in the occipital lobes, posterior parietal lobes and posterior temporal lobes, in the typical order are classical findings^[6].

The visual symptoms may vary ranging from blurred vision, homonymous hemianopia or even cortical blindness. Mental status may be altered, ranging from mild confusion, agitation to comatose state. Seizures and status epilepticus (SE) are common, where non-convulsive SE is more frequent than generalised SE^[7]. In patients with prolonged states of altered consciousness, one must suspect non convulsive status with clinical signs being stereotypic movements such as staring, eye blinking or head turning. Post-ictal confusion may last for hours, however both PRES and non-convulsive state can persist for several days, and can be mistaken for psychosis, drug intoxication or psychogenic states^[8]. Often, PRES is associated with acute hypertension. In addition to this, PRES shows strong association with co-existing conditions like renal disorders, vascular and autoimmune diseases, exposure to autoimmune drugs and organ transplantation^[9].

Various theories have explained the multifactorial pathophysiology of PRES. In PRES, the immune response is highly activated with increased levels of cytokines, a degree of renal dysfunction, vasoconstriction, coagulation system alterations and endothelial dysfunction. The hyper-perfusion theory, also called the vasogenic theory, is the most likely and accepted cause of PRES, suggesting that due to errors in the CNS Blood pressure auto regulation (usually regulated by dilatation and constriction of vessels to maintain adequate tissue perfusion) and lack of sympathetic innervation of vessels emanating from basilar and vertebral arteries, blood flow in the CNS increases. Further, it elevates the capillary filtration pressure and damages the capillary wall, eventually leading to increased blood brain barrier permeability. The uncontrolled hypertension leads to hyper-perfusion and cerebral vessel damage, resulting in interstitial extravasation of proteins and fluids, causing vasogenic cerebral edema.

However, in those with chronic hypertension have hypertrophied artery walls including those in the CNS, causing reduced permeability of the blood brain barrier.

Those with pre-eclampsia lack this compensatory effect and even a small increase in blood pressure can cause them to respond with increased permeability of the blood brain barrier^[10].

Delayed recognition of PRES leads to secondary complications like status epilepticus, intracranial haemorrhage and ischemic infarction^[11], thus making it essential to find the cause triggering the seizures and minimise number of seizures as far as possible, irrespective of the underlying cause. Our main aim should be to stabilise the patient in the primary stage and prevent the occurrence of further seizures. The emphasis should be on this since PRES is a reversible condition, if managed promptly, can be well treated.

Conclusion

In conclusion, prompt use of anti-epileptics and anti-hypertensives with airway management plays an integral role in the stabilisation of the patient's clinical condition. Early recognition of symptoms, and effective use of anti-epileptics plays a pivotal role, in such cases. Quick anaesthetic induction with efficiency proved to be life-saving in such a case.

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Conflict of interest

Nil

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