Post -partum Posterior reversible encephalopathy syndrome

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Abstract

Posterior Reversible Encephalopathy Syndrome (PRES) is a recently recognized disorder, it is often, but not always associated with high blood pressure and the prevalence is unknown. Typical clinical symptoms include persistent headache, visual disturbances, seizures and encephalopathy. The characteristic radiological findings involve mainly white/grey matter of the parietal and occipital lobes. It is associated with conditions that induce endothelial damage causing vasogenic cerebral edema that can be observed in magnetic resonance imaging scans. A rare case of PRES is presented here in a Gravida three and Para two with two living siblings in a cesarean patient on post-operative day one without known causative factors and underlying disease such as hypertension, preeclampsia and vasculitis.

Keywords: Posterior reversible encephalopathy syndrome, Postpartum, Seizures, Magnetic resonance imaging.

Introduction

Posterior reversible encephalopathy syndrome (PRES) is a rare complication of various clinical entities. Global incidence is unknown having been reported in wide range of age from 4 to 90 years with a Male/Female ratio of 0.8/1. Most cases occur in young to middle aged adults and death has been reported. PRES is a clinical radiographic syndrome that is often characterized by headache, altered consciousness, visual disturbances and seizures, in association with typical radiologic finding of vasogenic edema involving bilateral parietal/ occipital lobe[1]. However the syndrome is not always reversible and it is not confined to either the white matter or the posterior regions of the brain. Since the diagnosis of PRES requires a high clinical and imaging suspicion with the subsequent establishment of early treatment for a favourable outcome. Delay in treatment may result in permanent neurological damage or death. PRES during postpartum is a recently diagnosed disorder and prevalence is unknown. It is often but not always associated with high blood pressure. There are several known causes of PRES such as eclampia, preeclampsia, lupus erythematosus, thrombotic thrombocytopenic purpura and long term use of immunosuppressive drugs, renal diseases and uremic syndrome[2].But none were present in this case. This patient had seizures twice before subjecting to radio imaging for diagnosis. This case report highlights the importance of awareness, prompt diagnosis and early initiation of treatment to improve the outcome in this life threatening but reversible condition.

Case Report

A 32 year old parturient, gravida three, para two, with two living siblings now with full term pregnancy was scheduled for elective lower segment casarean section (LSCS). Pre-anesthetic check up was done night before surgery. Patient had no history of hypertension or epilepsy during the present and previous pregnancies. Vital signs were unremarkable. Laboratory tests were within normal limits. Patient underwent elective LSCS at 7.00 am, it was an uneventful surgery.

On post operative day one, on usual rounds at 7.00 am, the mother and baby were doing fine. At 10 am the obstetrician was alerted by the duty nurse about the vague behavior of the patient, the obstetrician rushed immediately to the patient and noticed the agitated, restlessness and irritable behaviour and patient started complaining of headache and blurring of vision, and all

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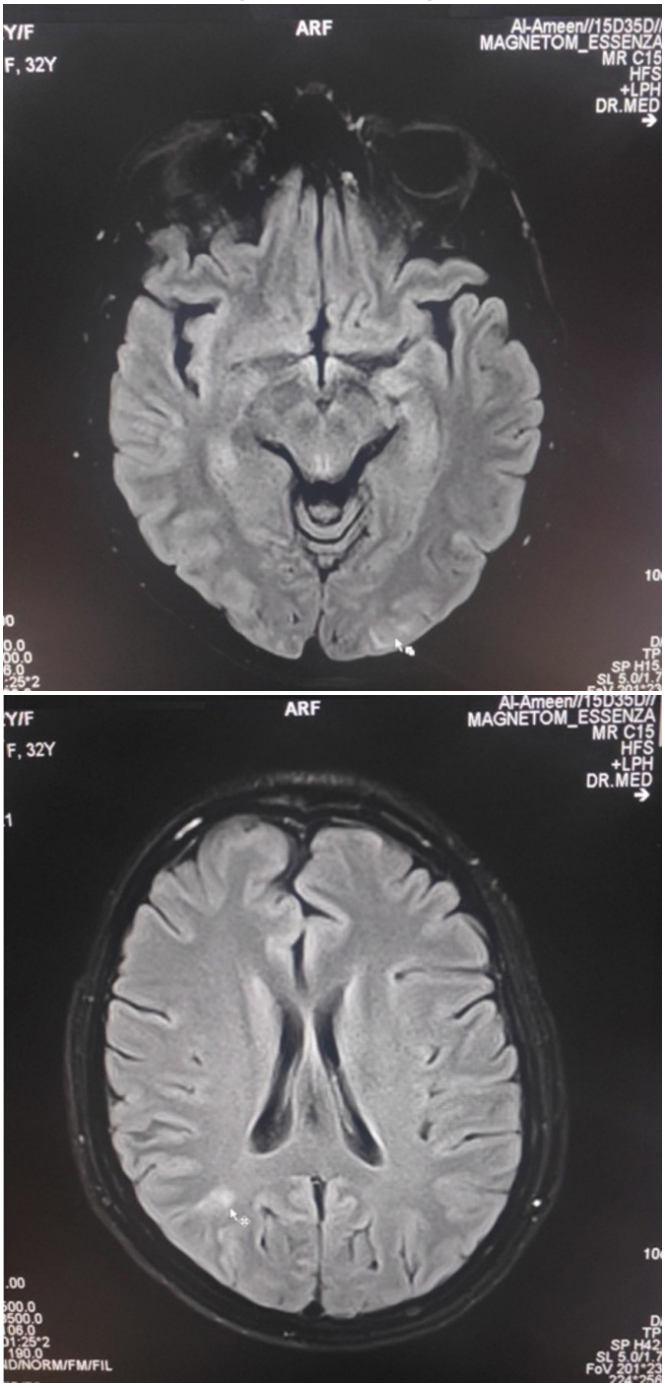
of a sudden patient threw convulsions, immediately O2 supplementation was given at 5 liters/min, inj Magnesium sulphate 4 grams given intravenously and 1 gram added to the drip to run for an hour. Inj phenytoin sodium 100 mg given slow iv, seizures were controlled, but patient was drowsy because of drug effect. On auscultation bilateral ronchi was heard for which inj deriphyllin and furosemide 10 milligram was given iv. It was decided to shift the patient to higher centre for advance care. In tertiary care centre, patient was seen by physician. Respiratory, Cardiovascular, and Gastrointestinal system examination was found to be normal. Central nervous system examination was incomplete as the patient was drowsy and confused but co-operative. There was flexor plantar response. Ophthalmic screening was done. Pupillary and corneal reflexes were normal, fundoscopic examination did not reveal any abnormalities, physician advised to repeat laboratory investigations namely, complete blood count, liver enzymes, blood sugar, serum electrolytes, The patient was closely observed. Results were made available and reviewed by the physician. All the lab investigation were found to be within normal limits. Then decision was taken to have MRI of brain at 6.00 pm in the evening. The patient was brought to MRI suit, all of a sudden patient threw convulsions and was treated with anticonvulsants and oxygen supplementation, patient was brought back to ICU and was monitored. Blood pressure was 130/80, heart rate 92/min, O2 saturation was 96%.Patient was then closely monitored till morning. As the patient was conscious, responsive and co-operative the patient was taken for MRI at 7.00 am Patient co-operated throughout the procedure. Results of MRI revealed (Fig-1), multiple ill-defined T, hypo intense and T2/ FLAIR hyper intense areas in cortical and subcortical region of bilateral occipital and right parietal region of which were showing high intensity on apparent diffusion coefficient (DWI) possibly of posterior reversible encephalopathy syndrome (PRES).

Fig-1: MRI findings



Multiple ill defined T1 hypointense areas noted in cortical and subcortical region of bilateral occipital and right parietal region which are showing high signal intensity on ADC and no restriction on DWI. Possibility of posterior reversible encephalopathy syndrome (PRES). MR Venography of brain reveals no significant abnormality

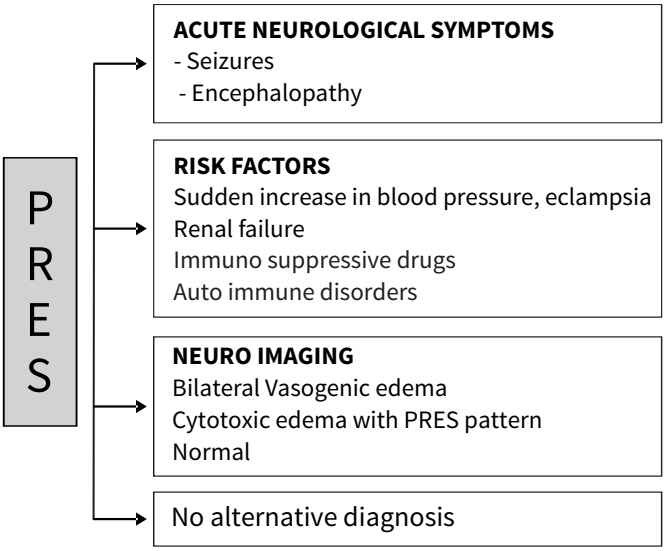
Discussion

The first description of the PRES was made in 1996 by Hinchey et al[3]. The knowledge of several aspects of this entity has been broadened. The first fifteen cases reported occurred in patients with hypertensive encephalopathy, eclampsia, or immuno suppressive treatment[4]. It has also been observed as a complication of other entities such as sepsis, renal failure, connective tissue disorder; Therefore it is currently known that the risk factors that cause endothelial dysfunction are key for development of PRES[5].

Based on the forgoing figure[2] shows the algorithm proposed by Fugate et al for the diagnosis of PRES which aims to identify even the atypical cases. The underlying pathophysiology of PRES remains elusive. Several theories have been proposed. The two main theories regarding the mechanism of disease process, one is the auto regulatory failure due to uncontrolled persistent hypertension, this failure causes cerebral vasodilatation and subsequently increased capillary hydrostatic pressure leading to vasognic edema, the second theory is that excessive arteriolar vasoconstriction (Vasospasm) results in decreased blood flow, ischemia and cytotoxic edema[6]. The preferential involvement of the parietal and occipital lobes is thought to be due to relatively poor sympathetic innervations of the posterior circulation. A notable point of this syndrome is that edema is present without infraction. A leading hypothesis suggests a crucial role for endothelial dysfunction and activation in PRES pathogenesis[7].

PRES is characterised by neurological signs including headache, visual changes, seizures and altered sensorium. Cortical blindness is considered a typical and characteristic symptom of this syndrome. However the symptoms are reversible with aggressive treatment but if appropriate treatment is delayed there are chances of permanent neurological damage secondary to cerebral infraction, hemorrhage and transtentorial herniation resulting in death. Early recognition of symptoms and timely diagnosis is the key for survival of the patient. As reported in the literature cerebral Magnetic Resonance Imaging (MRI) is the Gold standard diagnostic tool. MRI performed in this patient correlate well with the authors[8]. In this patient with normal blood pressure, symptoms were recognised early and treatment initiated on time with close monitoring of vital parameters and MRI for the accurate diagnosis of the syndrome helped us to successfully treat the patient and prevent neurological sequelae. This may occur rarely after normotensive and uneventful pregnancies which is described as atypical in literature[9,10]. In this case MRI of brain showed an increased leptomeningeal enhancement in fluid attenuated inverse recovery (FLAIR) sequence. Agarwal et al analysed MRI imaging in 20 pts suffering from PRES and they found an increasing leptomeningeal enhancement in 35% of these patients[11]. The increased enhancement is the result of an endothelial injury and an increase in microvascular permeability.

Fig-2: Proposed Algorithm



Conclusion

PRES is a life threatening, yet reversible condition. This is to be differentiated with subarachnoid hemorrhage, intravascular catastrophes such as cerebral thrombosis, sinus thrombosis (typically there would be motor deficits) HELLP syndrome and migraine as possible differential diagnoses[12].

Typical clinico-radiographic findings described should alert PRES as a rare possible cause during pregnancy and post postpartum. Early detection, good obstetric care, and judicious proplylactic/therapeutic use of drugs helps in preventing some of its devastating sequelae such as permanent vision loss.

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