Posterior Reversible Encephalopathy Syndrome in an Eclamptic Patient After Cardiac Arrest; Case Report and Literature Review

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Posterior reversible encephalopathy (PRES) is a disorder characterized by hypertension, headache, seizures and visual impairment. Causes of PRES include; severe hypertension, pre-eclampsia or eclampsia, sepsis, history of renal and autoimmune diseases and use of immunosuppressive or cytotoxic agents. Diagnosis of the syndrome can be difficult. For this reason clinical and radiological findings should be evaluated together. In this report, a 19-year old, 32 week pregnant eclamptic woman, who had been diagnosed with PRES, is presented with a discussion of the relevant literature.

Key Words: Posterior reversible encephalopathy syndrome, eclampsia, pregnancy

Introduction

Posterior reversible encephalopathy syndrome (PRES) was first defined by Hinchey et al. (1) in 1996. The syndrome is clinically characterized by lethargy, nausea, seizures and visual impairment. Cranial imaging shows symmetrical oedema in the subcortical white matter and occasionally within the cortex of occipital and parietal lobes (1). Although many conditions have been reported to cause PRES in the literature, the pathogenesis is yet unclear. Preeclampsia, eclampsia and HELLP syndrome are the obstetric causes of PRES. Hypertensive encephalopathy, sepsis, use of immunosuppressive drugs, history of renal and autoimmune diseases, HIV syndrome, acute intermittent porphyria and organ transplantation are among the other causes of PRES (1-4).

Rapid diagnosis and treatment are important in the prevention of secondary complications such as intracranial bleeding, status epilepticus and cerebral infarction that may lead to mortality and morbidity (5).

In this paper, we present the management of cardiac arrest and PRES in a pregnant patient at 32 weeks of gestation with eclampsia, along with review of the relevant literature.

Case Presentation

The patient presented here gave consent to disclosure of her clinical information in a scientific journal. A 19-year-old pregnant woman at 32 weeks of gestation, who was gravida 1, para 0 was brought to the emergency room by her relatives due to headache, convulsion and altered state of consciousness. On admission to the emergency department, her blood pressure was 150/100 mmHg and the initial physical examination was normal except pretibial oedema (3+). Urinalysis showed proteinuria (4+) and laboratory assessment revealed normal liver and kidney function tests. After about half an hour of her admission to the hospital, the patient had a seizure and went into cardiac arrest. After two minutes of cardiopulmonary resuscitation, spontaneous circulation returned. As the bedside ultrasound examination revealed foetal bradycardia, a decision to perform urgent caesarean section was taken by the department of obstetrics and gynaecology. The patient was given an intravenous bolus of magnesium sulphate (MgSO₄) at a dose of 4 g.

The patient was agitated and non-cooperative as she was taken to the operating room. Standard monitoring including electrocardiogram, pulse oxymetry and non-invasive blood pressure was performed. Her blood pressure was 162/100 mmHg, heart rate was 105 bpm and peripheral oxygen saturation was 97%. Anaesthesia was induced using propofol at a dose of 2 mg

kg⁻¹ and succinylcholine at a dose of 0.6 mg kg⁻¹. Intubation was performed at the first attempt using a 7 mm endotracheal tube. Correct placement of the endotracheal tube was confirmed by auscultation and end-tidal CO₂ monitoring. A baby girl 1750 g in weight was born with an APGAR score of 5-6. The patient was given 40 IU of intravenous Synpitan. Maintenance of anaesthesia was performed with 1 MAC of sevoflurane in a 50%/50% N₂O/oxygen mixture. Invasive arterial blood pressure monitoring was performed. As arterial blood gas analysis showed metabolic acidosis, the patient received intravenous infusion of 4 ampoules of sodium bicarbonate. MgSO₄ infusion at 2 g hour⁻¹ was started to avoid postoperative complications. At the end of the operation, the patient was transferred to the intensive care unit, placed on mechanical ventilation and ventilated in synchronised intermittent mandatory ventilation mode (SIMV; f: 12/min, FiO₂: 60, TV: 500 mL, I: E ratio: ½). The patient's blood pressure remained stable in the intensive care unit, and after spontaneous respiration started and she regained consciousness, she was extubated. Laboratory findings were as follows; haemoglobin: 8.6 g dL⁻¹, haematocrit: 26.6%, platelet count: 69.000/μL, AST: 98 U/L, ALT: 35 U/L, and LDH: 458 U/L. Her coagulation tests and cardiac enzymes were normal. As serum magnesium level was 2.8 mEq/l (normal range, 4-6 mEq/l), MgSO₄ infusion was continued. Consultant cardiologist recommended 10 mg of amlodipine in case of increased blood pressure.

The patient developed confusion and anisocoria on the second postoperative day and cranial magnetic resonance imaging (MRI) was performed on the advice of the neurologist. Cranial MRI showed high signal intensity on T2-weighted FLAIR sequences in cortical and subcortical areas and low signal intensity on T1-weighted sequences in the frontal and bilateral parietal-occipital lobes (Figure 1). In addition, lesion areas showing high signal intensity on T2-weighted FLAIR sequences and low signal intensity on T1-weighted sequences were observed in the posterior regions of the cerebellar hemispheres (Figure 2). After neurology and radiology consultation, the patient was diagnosed with PRES. By close monitoring of serum magnesium levels, MgSO₄ infusion was continued until the third postoperative day. Then, the patient was started on an antiepileptic medication, levetiracetam (Keppra® 2x500 mg, UCB Pharma). Laboratory examinations showed increased liver enzymes (AST: 168 U/L, ALT: 53 U/L) (Table 1). The lesions showed regression on the cranial computed tomography performed on the fourth postop-

erative day. As her vital signs remained stable and the elevated liver enzymes decreased, the patient was transferred to the obstetrics and gynaecology ward.

As the patient developed ptosis and confusion on the sixth postoperative day during the ward stay, readmission to the intensive care unit was advised. However, as there was no intensive care bed available in our hospital, the patient was

Figure 1. Cranial MRI showing high signal intensity on T2-weighted and FLAIR sequences in the cortical and subcortical areas and low signal intensity on T1-weighted sequences in frontal and bilateral parietal-occipital lobes

Figure 2. MRI scan of the brain demonstrating areas of involvement showing high signal intensity in the posterior region of the cerebellar hemispheres

Table 1. Laboratory values before and after the operation

	Hb (gr dL ⁻¹)	Htc (%)	Plt (μL)	ALT (U/L)	AST (U/L)	LDH (U/L)	Mg (mEq/l)
Intraoperative period	8.6	26.6	69.000	35	98	458	2.8
The 1 st postoperative day	10.2	31.4	60.000	53	168	776	4.72
The 2 nd postoperative day	10.5	32.7	66.000	30	80	724	3.95

Hb: haemoglobin; Htc: haematocrit; Plt: platelet count; ALT: alanine aminotransferase; AST: aspartate aminotransferase; LDH: lactate dehydrogenase; Mg: magnesium

transferred to another hospital. We learnt that the patient had received intravenous infusion of MgSO_4 during her stay in the intensive care unit, and as her blood pressure remained stable, she had been transferred to the obstetrics and gynaecology department after a day's stay in the intensive care unit. We've also been informed that MRI taken on the 10th post-operative day showed regression in the initial lesions whereas there were radiographic signs of PRES. As her general condition was stable, the patient had been discharged from the hospital and had been advised to come to follow-up visits in the neurology outpatient clinic.

Discussion

The diagnosis of posterior reversible encephalopathy syndrome is based on clinical and radiological findings, and subcortical oedema may be accompanied with headache, confusion, visual impairment and seizures (2, 6). Although the pathogenesis of PRES still remains unknown, the theory generally accepted is the hypoperfusion theory (7). According to this theory, the arterioles, which constrict when spontaneous increase in blood pressure exceeds the known threshold for cerebral autoregulation, begin to dilate in response to increased blood pressure. As a result, fluid, macromolecules and erythrocytes leak into brain parenchyma. Due to the lower sympathetic innervation in posterior cerebral circulation than that in anterior circulation, the involvement of posterior areas of the brain is more pronounced. After the properties of the blood-brain barrier returns to normal, oedema involving the white matter gradually resolves (7). Li et al. (8) explained the development of PRES in two ways. The first explanation was that hypertension, if severe, exceeds the limit of autoregulation and causes hyperperfusion and vasogenic oedema. Patients in this group respond to antihypertensive treatment. The second explanation was endothelial function impairment secondary to systemic toxicity which occurred due to immunosuppressive treatment, eclampsia, and sepsis. Intensive cytokine response involving IL-1, IL-6 and TNF causes endothelial cell damage. Clinical and radiographic findings were compared between the two groups and no difference was found.

MRI and CT may be used as imaging modalities in the diagnosis of PRES. CT shows symmetrically decreased white matter intensity in bilateral parietal-occipital regions. The observation of high signal intensity on T2-weighted and FLAIR sequences in cortical and subcortical areas of bilateral posterior cerebral regions on MRI helps to diagnose the condition (9, 10). The lesions are usually symmetrical but cases with asymmetric involvement have also been reported. Frontal, inferior, temporal-occipital junction and cerebellum are the other commonly involved regions. No correlation was found between the severity of the clinical picture and the extent of the lesions (11).

Our patient had a history of hypertension, proteinuria and convulsion. She had complaints of headache and altered state of consciousness. Cranial MRI showed non-specific high sig-

nal intensity on T2-weighted and FLAIR sequences in bilateral posterior cerebellar hemispheres and frontal and parietal regions. Based on clinical and radiological findings, the patient was diagnosed with PRES, and eclampsia was considered as the triggering factor.

The treatment of eclampsia, and hence that of the PRES is achieved by administering MgSO_4 (12). The therapeutic effect of MgSO_4 in eclampsia is based on many factors. MgSO_4 acts by decreasing the peripheral vascular resistance and preventing vasoconstriction. It also preserves blood-brain barrier and prevents the development of cerebral oedema owing to its central anticonvulsant activity. Deep tendon reflexes, respiratory rate, heart rate, blood pressure and urinary output of the patient should be monitored during treatment. In addition, it may potentiate the effects of neuromuscular blockers that are used in general anaesthesia. Use of antihypertensive drugs, such as labetalol, hydralazine and nicardipine, is recommended in patients with systolic blood pressure over 160 mmHg and diastolic blood pressure over 105-110 mmHg (13). Intravenous dexamethasone is also being used especially in patients with intracranial masses because of its lower mineralocorticoid activity, longer half-life and because it decreases vasogenic peritumoral oedema (14).

Brewer et al. (15) made a comparison between the methods used in eclampsia treatment and found that the recovery period was not affected whether or not MgSO_4 was given in combination with antihypertensive drugs. As our patient was considered to have eclampsia, intravenous infusion of MgSO_4 was used for treatment and as her blood pressure remained stable, no additional antihypertensive therapy was required.

Acute cerebrovascular accident, encephalitis, demyelinating diseases, sinus vein thrombosis, hyponatremia and hypoglycaemia should be kept in mind in the differential diagnosis of PRES. Clinical examination and cranial imaging are helpful in diagnosis.

Secondary complications such as status epilepticus, coma and cerebral haemorrhage may occur due to PRES. Li et al. (8) reported that the rate of recurrence was 14%. The development of confusion and thereafter ptosis on the sixth postoperative day was considered as recurrence in our patient.

Conclusion

Posterior reversible encephalopathy syndrome is a condition caused by many etiologic reasons; it may have different clinical presentations and recurrence is possible. Considering PRES in the differential diagnosis of a pregnant patient with altered state of consciousness and seizures allows early diagnosis of the condition, contributing to the reduction of mortality and morbidity.

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