

Case Report Posterior Reversible Encephalopathy Syndrome with Magnesium Toxicity

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Abstract: PRES is clinic-radiological entity characterised by Headache, Visual disturbance, Altered sensorium, Seizures and White matter vasogenic edema. PRES is associated with acute renal failure, hypertension, eclampsia, vascular and autoimmune disease, immunosuppressive drugs and organ transplantation. Magnesium sulfate is drug of choice in eclampsia. Magnesium sulfate is given by intramuscular or intravascular route for prevention of and treatment of eclampsia. Magnesium toxicity can be detected by signs and symptoms like loss of deep tendon reflexes, decreased respiratory rate (less than 12), decreased urine output (less than 30ml/hr), chest pain, heart block, pulmonary edema, and respiratory distress. 24 year old 2nd gravida postpartum female referred from periphery with history of convulsions 4 hours after normal vaginal full term delivery. On admission patient was unconscious, no response to painful stimuli, absent deep tendon reflexes, absent plantar, pupils sluggishly reactive to light, and urine output was <100/24 hrs. History revealed that patient was given magnesium sulfate before and after delivery as treatment of eclampsia. MRI brain showed asymmetrical altered signals in bilateral occipital cortical region in T2 and FLAIR suggestive of PRES. Patient is diagnosed as PRES with septicemia with acute kidney injury with magnesium toxicity. Patient improved after management with iv fluids, antibiotics, antiepileptics, hemodialysis, iv calcium gluconate and blood pressure monitoring. PRES can be a major problem in pre-eclampsia, eclampsia with renal failure. Magnesium sulfate should not be given without monitoring signs and symptoms of magnesium toxicity. A high index of suspicion and prompt treatment can reduce morbidity and mortality.

Key Words: Posterior Reversible Encephalopathy Syndrome, Posterior Reversible Leukoencephalopathy Syndrome, Hypertension, Eclampsia, Acute Kidney Injury, Septicemia, Magnesium Toxicity

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I. Introduction

PRES presents with rapid onset of symptoms which includes Headache, Visual disturbance, Convulsions, Altered sensorium. PRES strongly associated with renal diseases. It is therefore important to consider PRES as differential diagnosis of patient with acute renal failure with altered neurologic symptoms. Important causes of PRES are given in table 1.

Table 1: PRES associated clinical conditions

Acute Hypertension
Acute Kidney Injury
Eclampsia
Pre-eclampsia
Sepsis And Multiorgan failure
Autoimmune disease
Immunosuppressive drugs
Organ transplantation

There is wide variation in clinical presentation of PRES, i.e. Altered consciousness can vary from mild confusion to agitation or coma. Headache, Visual disturbances and Convulsions are common. Magnesium toxicity can complicate PRES if associated with eclampsia as magnesium sulfate is drug of choice in eclampsia.

Magnesium sulfate should be used cautiously in eclampsia patients and monitoring of signs and symptoms of magnesium toxicity should be done. Serum magnesium level should be done if required. Serum magnesium level and its effect on body is shown in table 2.

Serum magnesium level(meq/L)	Effect
4-7	Controls convulsion (therapeutic range)
7-10	Loss of deep tendon reflexes
>10	Respiratory depression
>25	Cardiac arrest

Causes of PRES is usually identifiable.If identified early and treated promptly PRES usually resolves within a week.

II. Case Report

24 Year old 2nd gravida postpartum female brought to emergency room with history of two episodes of convulsions and altered sensorium. History revealed that patient has normal vaginal delivery one day prior to admission. After vaginal delivery patient starts complaining of headache and visual disturbances and ptosis followed by two episodes of convulsion.

On examination :patient was unconscious.

Temperature was normal,

Pulse:136/min

Respiratory rate: 13/min

Blood pressure:230/150 mm of hg,

CNS examination:

Cranial nerve and higher function : not possible to evaluate

Tone :decreased in all four limbs,

Power : no movement to painfull stimuli'

Deep tendon reflexes: all absent,

Plantar reflex: absent/absent

Pupils : 1-2 mm, sluggishly reactive to light,

No signs of meningeal irritation.

Respiratory system, cardiovascular system and abdominal examination shows no significant abnormality.

Patient's obstetric history indicates pre-eclampsia in ANC period. Patient was given magnesium sulphate injection before and after delivery.patients urine output was 100 ml / 24 hours.

Laboratory investigation revealed normal haemoglobin, WBC count 13600/cu.mm, impaired renal function test with serum creatinine 5.6 mg/dl, serum magnesium level was 10.5 meq/L, serum phosphorus level 5 mg/dl. Urine albumin was +3.Liver function test was normal. Peripheral smear showed microcytic hypochromic anaemia with normal coagulation profile. Serum TSH level was normal.Chest radiography, arterial blood gas analysis were normal.MRI brain shows asymmetrical altered signals in bilateral occipital cortical regionmay be indicating PRES.

Patient was admitted in medical intensive care unit and was managed with intravenous fluids, antibiotics, antiepileptics, intravenous calcium gluconate,hemodialysis and blood pressure monitoring. Patient improved symptomatically in form of normal sensorium, leucocyte count, serum creatinine, serum magnesium level and vital signs. MRI brain after 10 days shows complete reversibility of altered signals in occipital lobe which confirms diagnosis of PRES with magnesium toxicity.

III. Discussion

PRES should be considered in patients who presents with convulsion, altered sensorium, visual disturbance and headache particularly if associated with acute hypertension and acute renal failure. Pathophysiology of PRES is unclear but most acceptable theory is severe hypertension causing interruption of brain autoregulation which causes vasogenicoedema. Another theory is systemic inflammatory state causing endothelial dysfunction.

Magnesium toxicity is life threatening if not detected early. It can worsen prognosis if associated with PRES. Magnesium within therapeutic range acts as membrane stabiliser and neuroprotector. It reduces motor endplate sensitivity to acetylcholine, blocks neuronal calcium influx. So magnesium toxicity can cause neuromuscular blockage.

High index of suspicion, early diagnosis and prompt treatment in form of intravenous fluid, antibiotics, antiepileptics, control of blood pressure, renal replacement therapy in form of haemodialysis if associated with renal failure, and removal of offending agent may lead to reduction of morbidity and mortality of PRES.

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