

Case Report

Magnesium Toxicity Presented as Quadriparesis in Postpartum Period: Case Report

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Abstract:

Quadriparesis may result from numerous neurologic diseases. Any of the causes could occur in the postpartum period. However, some conditions have increased prevalence during periparturient period, such as cerebral venous thrombosis, eclampsia itself, or its treatment with magnesium sulfate causing neuromuscular dysfunction in case of toxicity. Herein, we report a case of magnesium toxicity in a 34-year-old mother in the early postpartum period. This case signifies the importance of magnesium toxicity in patients with decreased renal clearance.

Keywords: Hypermagnesemia, Postpartum, Preeclampsia/Eclampsia, Quadriparesis, Lancet General Hospital

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Introduction

Since the early 1900s magnesium sulfate has been used for the prevention and treatment of eclamptic seizures [1]. Treatment with magnesium sulfate rarely results in hypermagnesemia, which is diagnosed when the serum magnesium level exceeds 2.3mg/dl. Hypermagnesemia is seen in about 15 % of hospitalized patients and renal failure can increase its risk [2, 3].

The normal serum magnesium level is maintained by the renal system. As kidney function declines serum level of magnesium increases. There is no magnesium regulatory mechanism other than urinary excretion. Thus, acute kidney injury (AKI) is an important risk factor for magnesium toxicity [4, 5]. Here, we report a case of quadriparesis after magnesium sulfate administration for atypical preeclampsia in patient with acute kidney injury during early postpartum period.

Case presentation

A 34-year-old para-four mother was referred to our hospital for acute-onset generalized body weakness of three hours duration. At the referring hospital, she had severe globalized headache, blurred vision, two

episodes of vomiting, and elevated blood pressure. At the referring institution, she received a magnesium sulphate loading dose of 10 gm intramuscularly and a first maintenance dose of 4 gm for elevated blood pressure.

Three hours after her last dose of magnesium, the patient developed generalized body weakness that affected both her upper and lower extremities, making her unable to move them. Her antenatal course had been uneventful until the third trimester, when she developed a uterine rupture, resulting in a total abdominal hysterectomy. She received a blood transfusion during the surgery. With a stable immediate post-operative period, she was discharged after 48 hours of observation.

Upon examination, the patient was conscious with a blood pressure reading of 140/90, a pulse rate of 102, and normal respiration and temperature. Her neurologic examination revealed quadriparesis, with reduced muscle strength (power 4/5) in both her upper and lower extremities. Additionally, she exhibited hypotonia, reduced reflexes, an equivocal bilateral plantar reflex, and no signs of meningeal inflammation

Initial investigations showed elevated levels of urea and creatinine (urea 165 mg/dl, creatinine 10.6 mg/dl), indicating acute kidney injury. Her electrolyte levels were abnormal (sodium 129 mEq/l, potassium 5.03 mEq/l, and magnesium 8.5 mg/dl). However, complete blood cell counts and liver function tests were normal. Brain magnetic resonance imaging with magnetic resonance venography yielded normal results. Her electrocardiogram was also unremarkable. The diagnosis of quadriparesis secondary to hypermagnesemia due to magnesium toxicity, along with AKI possibly caused by ischemic acute tubular injury, was made.

Immediate treatment involved the administration of calcium gluconate and the initiation of hemodialysis to normalize serum magnesium levels. The patient showed complete neurologic recovery at the time of discharge.

Serial measurements of renal function tests and electrolytes are shown in Table 1 below.

Variable	On Admission	After 1 st dialysis session	After 2 nd dialysis session	After 3 rd dialysis session	On discharge
Creatinine (mg/dl)	10.6	7.4	3.9	2.7	1.16
Urea (mg/dl)	165	116	78	56	44
Magnesium (mg/dl)	8.5	5.6	3.8	2.4	1.9
Calcium (mmol/l)	1.1	1.1	1.0	1.1	1.2
Sodium (mEq/l)	129	131	133	136	140
Potassium (mEq/l)	5.03	4.6	4.0	3.6	3.8

Discussion

Preventing and controlling of preeclamptic/eclamptic seizures are the most common obstetric indications of magnesium sulfate administration [1].

Toxicities from hypermagnesemia correlate with the serum concentration. Non-specific symptoms occur during the early phase. Thus include nausea, dizziness, weakness, and confusion. As the serum level increases, areflexia occurs at 8.5 to 12.0 mg/dl; respiratory paralysis occurs at 12 to 16 mg/dl; cardiac conduction is altered at > 18 mg/dl; and cardiac arrest occurs at > 30 mg/dl [2, 4, 5].

The predominant neurologic manifestations are muscular weakness and paralysis. If untreated, respiratory failure results from respiratory muscle involvement. The muscle weakness is typically flaccid type, and it is caused by blockage of neuromuscular transmission, which resolves only when the magnesium level returns to normal [5, 6].

Treating physicians should maintain a high index of suspicion to consider magnesium toxicity in at-risk patients. The initial evaluation includes the determi-

nation of serum magnesium and other electrolyte levels, serum creatinine and blood urea nitrogen determinations, and arterial blood gas analysis [7].

Treatment of magnesium toxicity includes administration of calcium gluconate or chloride, administration of normal saline at a rate of 150 to 200 ml/h if renal function is normal, and hemodialysis in severe toxicities and renal failure [5, 8].

Although it is not a contraindication for the administration of magnesium sulfate, in patients with acute renal insufficiency, the magnesium sulfate dosing should be reduced according to the American College of Obstetricians and Gynecologists' recommendations [9].

Although our patient's serum creatinine level increased serially after magnesium administration, there is no evidence in the literature showing magnesium sulfate causing renal injury. Thus, worsening of the renal condition could be due to the underlying preeclampsia or hemodynamic instability from bleeding [10].

Conclusion

Maintaining a high degree of suspicion is important for the early diagnosis of quadriparesis from magnesium toxicity in at-risk patients during the postpartum

period. Initial workup should include serum magnesium and creatinine levels. To prevent toxicity from magnesium sulphate, dose adjustment should be

Reference

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