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CASE REPORT

Hypermagnesemia Complicated by Acute Flaccid Paralysis, Case Report

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Abstract: Hypermagnesemia is a rare electrolyte abnormality observed in the clinical setting. Patients with this condition can present with a variety of symptoms, depending on the level of serum magnesium. Hypermagnesemia is rarely complicated by weakness of the extremities. We report the case of 32 years old female patient who presented with bilateral upper- and lower-extremity weakness due to hypermagnesemia. Following correction of the serum magnesium level, extremity weakness subsided, and the patient was discharged with improvement. Here, we discuss the delay in the diagnosis and management of this type of clinical presentation.

Keywords: hypermagnesemia, extremity weakness, flaccid paralysis

Introduction

Magnesium is one of the most important cations in the body, and it is the second most dominant cation within cells.[1] It is essential to control a wide range of physiological processes, including neuromuscular junction and nervous signal transmission.[2]

There are several mechanisms that maintain magnesium homeostasis, including those within the kidneys, intestines, and bones; however, overall, the kidneys play a major role.[3] It is important to understand that hypermagnesemia is a rare and iatrogenic condition that can be caused by the administration of magnesium-containing drugs, such as laxatives used for constipation or MgSO4, which are used in obstetrics during pregnancy induced hypertension, namely pre-eclampsia and eclampsia.[4]

Patients may exhibit a wide range of clinical manifestations depending on their serum magnesium levels. These include nonspecific symptoms such as fatigue, nausea, and altered sensorium at slightly elevated levels or severe neurological manifestations such as headache, depressed reflexes, weakness, and visual changes at levels above 8.5 mg/dl.[5,6]

Hypermagnesemia can be life-threatening if left untreated; hence, prevention of morbidity and mortality can be achieved with Intravenous administration of calcium gluconate and fluids, and in severe cases with hemodialysis.[3] To the best of our knowledge, the association between hypermagnesemia and acute flaccid paralysis is rare. Here, we present the case of 32 years old Para 2 woman on her 5th post-partum day, who was diagnosed with hypermagnesemia complicated by acute flaccid paralysis after presenting with bilateral upper and lower extremity weakness for five days.

Case Presentation

A 32-year-old Para 2, Right-handed woman presented to Tikur Anbessa Specialized Hospital on her 5th post-partum day with bilateral upper and lower extremity weakness for 5 days. She received antenatal care at a nearby hospital and was delivered vaginally at 37 weeks of gestation.

One day prior to delivery, she complained of severe headache, epigastric pain, and blurred vision, which worsened during the postpartum period. Following this, a diagnosis of preeclampsia with severe features was made, and she was administered magnesium sulfate. She received 24 g of magnesium sulfate for both loading and maintenance doses. In addition, she complained of generalized body swelling, which started on her face and progressively involved the whole

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body, decreased urine volume, and shortness of breath. Further investigation did not reveal any history of hypertension or renal disease. She also denied any similar history in the past and had no history of substance abuse.

On presentation, the patient's morning axillary temperature was 36.5 degree Celsius, pulse rate was 80 bpm, she has elevated blood pressure of 160/100 mmHg, and respiratory rate was 14 breaths/min. Neurological examination revealed a GCS score of 15/15 and normal cranial nerve examination. Furthermore, bilateral deep tendon reflexes (knee, ankle, biceps, and triceps) were absent, plantar reflexes were up going, and the muscle strength was 3/5 in both lower extremities and 4+/5 in both upper extremities (Figure 1). Coordination or sensory deficits were not observed.

Laboratory investigations revealed mild hyperkalemia (K: 5.2 mg/dl) and normal serum calcium level (Total Ca: 10.2mg/dl). Renal function test results revealed a serum creatinine of 7.2 mg/dl. Further investigations using brain computed tomography (CT) and nerve conduction tests showed no abnormalities.

She was managed for acute kidney injury due to preeclampsia using magnesium sulfate, furosemide, and amlodipine. Despite this management, there was no improvement in the extremity weakness.

Subsequently, the serum magnesium level was determined, and the patient was found to have severe hypermagnesemia, with a peak serum magnesium level of 6.4 mg/dl (normal laboratory level of serum magnesium: 1.7–2.2mg/dL) (Figure 2). Subsequently, Intravenous calcium gluconate was administered, and over four days, serum magnesium levels normalized, extremity weakness improved, and the patient was discharged.

The patient was followed up 2 weeks later and was in good condition with no extremity weakness.

Discussion

Hypermagnesemia is a relatively rare electrolyte abnormality in clinical setting.

Among the few studies on the prevalence of hypermagnesemia, one study found the prevalence to be approximately 3% and more common in males than female.[7] Pregnant women with preeclampsia are at a high risk of hypermagnesemia because magnesium is frequently used to prevent and treat eclampsia.[8] Hypermagnesemia is relatively common in hospital settings compared to that in the general population. Its prevalence among hospitalized patients ranges from 5.7% to 9.3%, and approximately 10–15% of hospitalized patients with renal failure develop hypermagnesemia.[9]

Hypermagnesemia commonly occurs in patients with renal failure and rarely occurs in those with normal kidney function. Other less common causes of hypermagnesemia occur when patients are administered large doses of

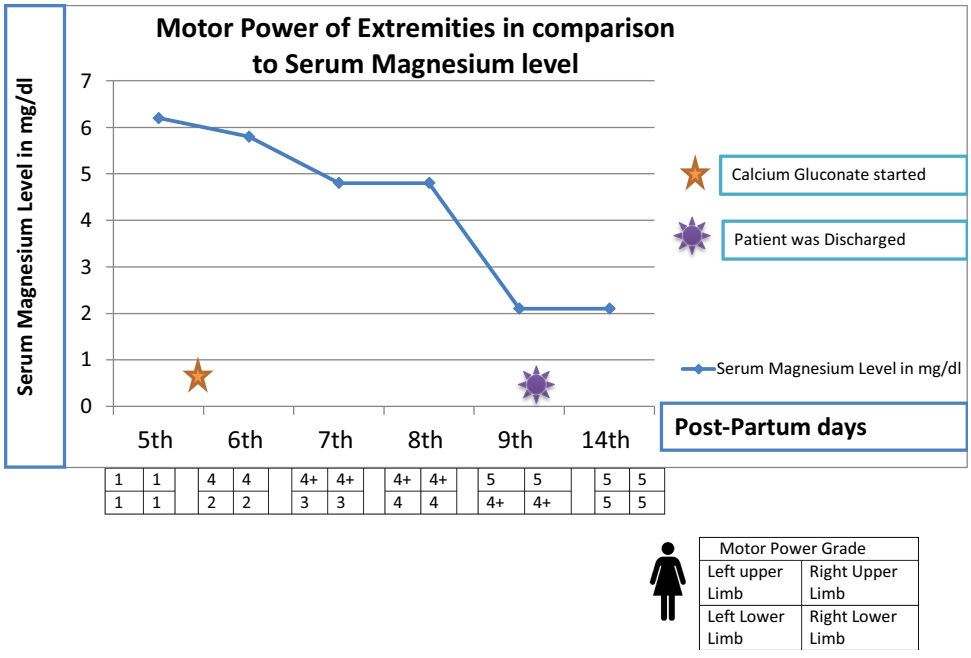

Figure 1 A graph depicting motor power of extremities in relation to serum magnesium level.

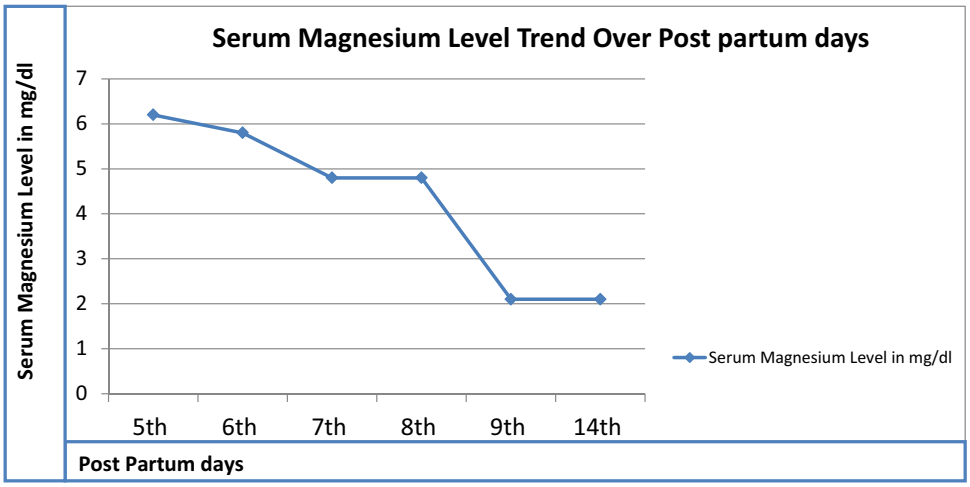

Figure 2 A graph depicting the serum magnesium level over the post-partum days.

magnesium, either intravenous or orally, and increased absorption of magnesium from the gastrointestinal tract in patients with constipation, colitis, gastritis, and gastric ulcer disease.[10,11]

Patients with hypermagnesemia can present with a wide range of symptoms, depending on the level of serum magnesium. Mild elevation in serum magnesium levels can be asymptomatic or present with nonspecific symptoms.[12] While, a serum magnesium level increased to more than 3.5 mmol/L present with worsening confusion, drowsiness, depressed reflexes, headache, blurring of vision, urinary incontinence and gastro-intestinal symptoms.[13] More serious manifestations can occur if the serum magnesium level increases to more than 6.5 mmol/L and include somnolence, loss of deep tendon reflex, muscle paralysis, decreased respiration, and apnea.[14]

Our patient's pregnancy was complicated by pre- eclampsia, and subsequently she developed acute kidney injury. Despite multiple investigations, including imaging studies such as Brain CT, which were unremarkable, the cause of the weakness was not identified. When the serum magnesium level was determined on the post-partum day 5, it was 6.4 mg/dl, and flaccid paralysis due to hypermagnesemia was considered.

There are multiple factors that can explain the hypermagnesemia in our patient, including reduced renal function and several doses of magnesium sulfate administered as part of the management for Pre- eclampsia.

Following the diagnosis of hypermagnesemia, the patient was administered Intravenous calcium gluconate, and the serum magnesium level dropped to 3.2 mg/dl over 4 days. Subsequently, the patient showed improvement in extremity weakness. After four days of calcium gluconate therapy, the serum magnesium level became 2.5 mg/dl, and the extremity weakness subsided.

The patient was discharged on postpartum day 14. She was followed up 2 weeks after discharge and was in good condition, with no extremity weakness.

Conclusion

Our case illustrates that hypermagnesemia is rarely complicated by acute extremity flaccid paralysis and the diagnosis may be delayed or missed. Physicians evaluating patients with weakness should consider that extremity weakness may be due to hypermagnesemia. Prompt detection and correction of serum Mg levels will eliminate the need for further imaging studies and delayed management.

Ethical Approval

The authors' institution does not require ethical approval for publication of single case report.

Consent for Publication

The patient provided written informed consent for publication of the case report.

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Disclosure

The authors report no conflicts of interest in this work.

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