



Acute Flaccid Quadripareisis caused by hypermagnesemia

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Abstract :

We report a case of Acute Flaccid Quadripareisis due to hypermagnesemia resulting from the unsupervised use of high doses of magnesium sulphate administered as a part of Pitchards regimen to a woman to control postpartum seizures. The treatment was given in a peripheral hospital for the seizures following the delivery by LSCS. She was known to have Pregnancy Induced Hypertension (PIH) during antenatal period. Magnesium level was not monitored and also the reflexes were not periodically checked during the magnesium therapy. Hypermagnesemia is an uncommon but serious side effect of the use of Magnesium containing compounds. Magnesium therapy is usually safe if used in appropriate dosages and with normal renal function. Here the renal status of the patient was not known and liberal use of magnesium led to the deterioration of the level of conscious and weakness of all four limbs and referred to the tertiary hospital. She was found to have hypermagnesemia due to renal failure and after haemodialysis she recovered well. This case report serves to illustrate the characteristic

pathophysiologic changes of severe hypermagnesemia an entity rarely seen in obstetric practice. More importantly it alerts obstetricians to be aware of complications of magnesium therapy and importance of monitoring magnesium level.

Keyword : Hypermagnesemia renal failure toxicity

Introduction: Acute Flaccid Quadriplegia is a life threatening neurological disorder. Various causes are metabolic, infectious, autoimmune disorders etc . Here a rare case of acute flaccid quadriplegia is presented.

Case report: 28 yr old woman G2P1L1 previous caesarean section had PIH in present pregnancy, admitted for safe confinement. She delivered an alive male baby by LSCS. Postoperatively her BP was 160/100 mm of Hg. After 4 hours of delivery she developed one episode of seizure involving all the four limbs with loss of consciousness. Patient was treated with MgSO₄ (Pitchard's regimen) and on the same day she developed weakness of all four limbs. She was referred to our hospital. She was not a known case of Diabetes,

Hypertension, seizure disorder or kidney disease. Her vitals at that time were pulse 60/min, BP 160/100 mm of Hg., Resp rate 20/min, SPO2 97 %, Urine output 200ml/2 hours.

On day 1 she was pallor, afebrile, drowsy, not arousable, pupils bilateral 2mm reacting to light, not moving limbs to pain, tone decreased in all four limbs. Deep tendon reflexes are absent. Plantar bilateral no response.

On day 2 patient conscious, oriented, communicating, weakness of all four limbs, hypotonia and hyporeflexia.

She was investigated and found to have :

Hb 8.2 g, Tc 11,400 cells/cu mm, Dc P 66

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Lakhs, Blood Urea 101 mg/dL,

Serum Creatinine 3.0 mg/dL. Urine analysis Sug nil, alb + Deposit 10-12 Pus cells, Rbcs Nil. Serum

Electrolytes Na 141 K 4.5.

Serum Calcium 8mg/dL. LFT Normal. ECG Sinus Bradycardia. Echo: Normal study. Serum Magnesium on 1st Day 7.1 mg/dL. USG Abdomen Normal study. CT Brain Normal.

Thyroid profile Normal.

Fundus Normal. Patient was treated with Inj. Calcium gluconate. There is no improvement in muscle power. Haemodialysis two cycles done. Serum Magnesium and renal parameters came down. Serum Magnesium after dialysis 3.5 and 2mg within 2 days apart. Patient's power improved and she was able to walk without support.

Discussion :

Uncommon but particularly profound pathophysiologic abnormalities should alert the neurophysician to the possibility of side effects of Magnesium therapy as one of the cause of acute flaccid quadriplegia.

In our patient the rarely seen condition of severe hypermagnesemia resulted from the overdose of an otherwise safe compound Magnesium Sulphate. Hypermagnesemia has been rarely reported in medical practice. Cases usually result from large intravenous doses of magnesium or from excessive oral intake of magnesium containing cathartics by patients with renal insufficiency. However several cases of hypermagnesemia from enteral magnesium intake in patients with normal renal function also have been reported. Magnesium is primarily absorbed in the small intestine and with normal renal function excess magnesium efficiently eliminated in the urine. Clinically hypermagnesemia occurs when the capacity for renal magnesium elimination is exceeded. Excess magnesium is known to have direct and indirect cardiovascular effects. Magnesium has been described as "nature's physiologic Calcium Blocker" and cardiovascular effects seen in hypermagnesemia may be caused by disruption of calcium action. Electrocardiographic observations in humans and animals have shown an increase in the P-R interval at concentration of 6 to 12 mg/dL which may progress to heartblock and asystole at levels greater than 18 mg/dL. Bradycardia may in part be a result of sympathetic blockade. Our patient also had Sinus bradycardia which may be secondary to magnesium toxicity. The effects of magnesium on the peripheral and autonomic nervous system may explain the decreased arousability, apparent drowsiness and pupillary constriction in our patient. Magnesium blocks the neuromuscular junction by antagonizing calcium effects, suppressing acetylcholine release and diminishing postsynaptic membrane responsiveness.

Deep tendon reflexes are depressed at serum Magnesium levels above 6mg/dl and are absent at levels above 12mg/dL. Severe muscle weakness is seen at levels greater than 12mg/dL with the potential for respiratory muscle paralysis. Here our patient had magnesium level of 7.1 mg/dL and presented with severe weakness and areflexia. The metabolic abnormalities seen in our patient can be attributed to altered renal handling of calcium induced by excess magnesium. Hypermagnesemia is associated with calciuria attributable to inhibition of tubular resorption of calcium. Hypermagnesemia inhibits parathyroid hormone secretion which may further exacerbate calcium loss because parathyroid hormone enhances tubular resorption of calcium. Alternatively the effects of calcium metabolism may involve effects of elevated serum magnesium on the calcium sensing receptor present on the parathyroid gland and on the ascending limb of the loop of Henle. Magnesium may bind these receptors and lead to an inhibition of tubular resorption of calcium. Here our patient was having the calcium value lower limit of the normal and also she was treated with calcium gluconate. The treatment for severe hypermagnesemia is aggressive supportive care, including airway protection and mechanical ventilation if needed. Maintenance of intravascular volume and cessation of magnesium administration are essential. Intravenous calcium administration may be beneficial. For life threatening Hypermagnesemia, definitive therapy is the removal of excess magnesium with peritoneal or haemodialysis. Here this patient not responding to intravenous calcium recovered well after haemodialysis.

Conclusion:

Serum Magnesium level should be monitored and should be assessed clinically by deep tendon reflexes and the renal status also be evaluated during Magnesium therapy as hypermagnesemia may lead to severe neurological complications.

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