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Case Report

Stridor: a rare complication of magnesium sulfate therapy in a pregnant patient

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ABSTRACT

Magnesium sulfate is frequently used in severe preeclampsia and eclampsia for the prevention and reoccurrence of seizure activity. Their adverse effects of magnesium sulfate are minor but it cause respiratory depression and tetany. We report a case of stridor due to laryngospasm as result of hypocalcemia in a pregnant patient on magnesium sulfate therapy. A 30 year old gravida5 para 4 had severe preeclampsia started on magnesium sulfate therapy and to control her hypertension she was on labetalol and nifedipine. One hour after the lower segment cesarean section, she developed severe laryngospasm and stridor, no upper airway secretion but found to have hypocalcemia. She responded to immediate intravenous calcium chloride with dramatic clinical improvement. Patients on magnesium sulfate can have life threatening hypocalcemia and stridor. The calcium channel blockers may augment the hypocalcaemic effect of magnesium sulfate.

Keywords: Calcium channel blockers, Eclampsia, Hypocalcemia, Laryngospasm, Magnesium sulfate, Preeclampsia and stridor

INTRODUCTION

Preeclampsia and eclampsia is a life threatening disorder affecting 2-8% of pregnancies. Magnesium sulfate is commonly used in Preeclampsia and eclampsia for prevention of seizure activity.¹

But this therapy is not free from complications, usually minor adverse effects such as nausea, vomiting, muscle weakness, dizziness but can be major side effects such as respiratory depression and tetany.¹

Rarely the hypocalcemia caused by magnesium sulfate therapy can lead to laryngospasm, which is not reported in the literature. We report a case of stridor due to hypocalcemia caused by the magnesium sulfate therapy in a pregnant patient.

CASE REPORT

A 30 year South African black lady, gravida 5, para 3+1, presented with severe preeclampsia (BP 215/145 mmHg, proteinuria +2), and preterm premature rupture of membrane at 33 weeks of gestation. Her cardiovascular and respiratory examination was unremarkable. Past medical history she was known case of HIV, CD4 was unknown, she wasn't on ARVS therapy.

Up on presentation, she was given 10 grams magnesium sulfate (Mg. Sulfate) intramuscular, followed by 2 grams/hour intravenous Mg sulfate as a maintenance, as well as antibiotic and course of Betamethasone.

Blood pressure was dropped to 145/95 mmHg, the investigations (FBC, urea, creatinine, electrolytes, LFT, coagulation screen) were done, it showed raised serum creatinine 115 mmol/l, and liver enzymes LDH 1433

IU/l, ALP 162 IU/l hemoglobin 10 grams/dl other investigations were unremarkable.

After 3 hours of admission, blood pressure raised to 186/124 mmHg, controlled with Labetalol infusion and Nifedipine (10 mg orally). After 6 hours of admission patient had emergency cesarean section (LSCS) due to fetal distress. Patient underwent LSCS under spinal anesthesia.

After 1 hour of Cesarean section she was dyspnic, distressed, and had stridor, her blood pressure was 111/70 mmHg, heart rate 76 beat/min, Glasgow Coma Score (GCS) 15/15, she had carpobedal spasm (Trousseau's sign), although there was no masseter muscle spasm (Chvostek's sign).

Table 1: Laboratory findings.

	At presentation	Pre-operative 13:00 hours	Post-operative 21:00 hours
Hemoglobin (gram/dl)	13.7	13.3	11.7
WBC ($\times 10^3/\text{mm}^3$)	8.9	12.1	14.4
Platelet ($\times 10^3$)	197	197	161
Sodium (mmol/l)	135	134	136
Potassium (mmol/l)	3.5	3.5	4.1
Chloride (mmol/l)	109		110
Urea (mmol/l)	3.9	4.2	3.8
Creatinine (mmol)	101	115	117
Albumin (gram/l)	21	22	19
Bilirubin (mmol/l)	9	11	5
ALP (IU/L)	150	162	131
LDH (IU/L)	1287	1433	
ALT (IU/L)		35	28
GGT (IU/L)	23	27	20
INR (International Normal Ratio)	0.82	0.82	
Calcium (mmol/l)			1.78
Phosphate (mmol/l)			1.55
Magnesium (mmol/l)			2.74

Magnesium sulfate was stopped, 40% O₂ was given via face mask, 10 ml of 10% calcium chloride was given over 10 minutes; she improved dramatically after 10% calcium chloride. Arterial blood gas, FBC, urea and

electrolytes were sent, it showed low ionized calcium 0.89 mmol/l, magnesium level was 2.74 mmol/l (Table 1).

DISCUSSION

Worldwide the occurrence of Preeclampsia is about 2-8% of all pregnancies, most frequently it occurs after 32 weeks of gestation, some cases experience Preeclampsia at 20 weeks of gestation. Preeclampsia is one of leading cause of maternal death and perinatal morbidity and mortality.² Eclampsia is one of major complication of Preeclampsia, affects 0.56 per 1000 pregnant women in developed countries.

Magnesium sulfate is the treatment of choice for prevention of convulsion in severe preeclampsia patient, also for prevention of repetitive seizures in eclamptic patient as it is confirmed by 2 clinical trials. The eclampsia trial collaborative group concluded that the Magnesium sulfate reduced the hazard of frequent convulsions in eclamptic patient by 52% when compared with diazepam.² Magpic trial reported that the Preeclampsia patient had 58% lower risk of getting eclampsia than the placebo group.²

The serum magnesium level should be between 4-7 meq/l to prevent eclampsia. Magnesium acts via a number of mechanisms, such as decreasing calcium influx by blocking N-methyl D-aspartate receptors causing vasodilatation of cerebral blood vessels and reduces the cerebral vasospasm.^{2,3}

Magnesium sulfate can cause several side effects such as flushing, nausea muscle weakness, respiratory depression, hypotension, hypocalcaemia, especially in patient with impaired kidneys function. Magnesium causes hypocalcaemia by inhibiting parathyroid hormone secretion and increases urinary excretion of calcium.^{2,3}

Few case reports of symptomatic hypocalcaemia secondary to magnesium sulfate were published, one of the cases was critically ill eclamptic patient treated with large dose of magnesium sulphate and she had tetany when her serum magnesium was 10 mg/dl and her serum calcium was 6.7 mg/dl.

Another case was preeclamptic patient, she received magnesium sulfate developed Chvostek's and Trousseau's signs 2 days after discontinuation of Magnesium sulfate, her serum calcium was 4.9 mg/dl.⁴

Calcium ion is one of most important ions in our body; it's involved in most of essential biological functions, such as muscle contraction, blood coagulation, release of hormones and neurotransmitters, and bone metabolism.

It's not surprising that abnormalities in calcium balance leads to significant physiological disturbance, the severity of these disturbances depend on severity of hypocalcaemia. Mild hypocalcaemia can lead to increase neuromuscular irritability, which may present with

paresthesia of the feet and hand, circumoral numbness, muscular cramps. Severe hypocalcaemia leads to focal or generalized tonic muscle cramps, convulsions, arrhythmia, and laryngospasm and stridor which is common in pediatric population although has rarely been reported in adult age group.⁵

The most common causes of stridor in adults are swelling of the upper airway, pharyngeal and laryngeal edema, tumor and paralysis of the vocal cord.

In spite of strong evidences for effectiveness of magnesium sulfate concern has been expressed regarding the safety especially in situations where the capacity for patient monitoring is limited, patient with impaired kidneys function, concomitant use of magnesium sulfate and nifedipine as both of them affect calcium metabolism.^{2,4} Nifedipine alone does not appear to cause hypocalcaemia, but nifedipine may make hypocalcemic patient symptomatic.⁴

Many studies have advocated that nifedipine and magnesium can be used safely together (Magpie trial 30% of patient received Nifedipine after they included in the study and no side effects were reported.

CONCLUSION

This case report should serve to remained clinicians of possibility of major complications of hypocalcaemia such as stridor especially if patients are receiving nifedipine with magnesium sulfate.

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