

Magnesium Sulphate Toxicity In Eclamptic Patient

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Abstract: A case of MgSo4 toxicity in a case of eclampsia after normal delivery is reported. The patient was treated with magnesium sulphate as per 'Pritchard regimen'. Post administration of the drug, she developed respiratory depression and was placed on ventilator. History revealed that she was inadvertently given higher dose of magnesium sulphate. Her creatinine levels were also high suggestive of slow excretion of the drug. MgSo4 toxicity was suspected and she was successfully treated with calcium gluconate.

Keywords: Eclampsia, MgSo4 toxicity, Respiratory distress with MgSo4, Calcium gluconate antidote, MgSo4 toxicity.

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I. Introduction:

Hypertensive disorders of pregnancy, including pre-eclampsia and eclampsia, complicate 10% of pregnancies worldwide. [1] Pre-eclampsia is defined clinically as hypertension and proteinuria, with or without pathological edema that occurs after 20 weeks of gestation. It can present up to 4 to 6 weeks postpartum. Pre-eclampsia complicated by generalized tonic clonic convulsions is referred as eclampsia. The convulsions in eclampsia are prevented or arrested very effectively by standard 'Pritchard regimen'. Generally, this regimen is safe. Toxicity though rare, could be monitored by checking patellar reflex, urine output and respiration rate. At times higher doses are administered inadvertently leading to life threatening complications. A prompt diagnosis and treatment can save the patient. One such case is reported here.

II. Case Report:

A 19-year-old woman presented to MGM hospital, Navi Mumbai, on Post-natal day two of full term normal vaginal delivery with relatives giving history of two episodes of convulsions at home. She was first taken to local hospital and was advised to take the patient to a tertiary care center for further treatment. On arrival in casualty, patient had another episode of convulsions. Her pulse rate of 120/minute, blood pressure of 160/110mmHg. In view of convulsions within 24 hours after delivery and high blood pressure, diagnosis of Eclampsia was made, and the patient was placed on MgSo4 as per 'Pritchard regimen'. She was given '4g of magnesium sulphate as a 20% solution intravenous, followed by 10 g of 50% magnesium sulphate solution, injected deeply intramuscular in upper outer quadrant of each buttock'. There were no further convulsions after administration of MgSo4 and only then the patient could be examined in detail.

On physical examination, patient was drowsy and disoriented, respiratory rate was 9/minute, patellar reflexes depressed both sides. Multiple tongue bites were noticed. Urine output 15 ml in last 30 minutes. Her oxygen saturation was varying between 70-90%. In view of low O2 saturation, the patient was intubated and placed on ventilator with 100% oxygen.

Laboratory findings showed normal hemogram, urine protein 2+, serum aspartate aminotransaminase (AST) 98 IU/L, serum alanine aminotransaminase (ALT) 102 IU/L, serum creatinine 1.5 mg/dL. The coagulation profile, ECG and chest X-ray were normal.

In the absence of any history of cardiac or respiratory ailment, low respiratory rate and low O2 saturation was worrisome. Suddenly it occurred to the treating gynecologist that it could be a case of MgSo4 toxicity in view of low respiratory rate, absent knee jerk and urine output at lower limit of normal. The patient also had high serum creatinine suggestive of compromised renal function which could have been responsible for slow excretion of MgSo4. No further doses of MgSo4 were given. Meanwhile the patient's relatives brought the referral note which was not shown to the duty doctor earlier. The referral letter showed that patient was already given Inj. Magnesium sulphate 5 gm on each buttock at the local hospital before arrival at our hospital. This further added to the possibility of magnesium toxicity. Blood level of magnesium could have further confirmed the toxicity, but the facility was not immediately available. Since respiratory distress is a known complication of

MgSo₄ toxicity, decision was taken to give the patient calcium gluconate as antidote for MgSo₄. The Inj. Calcium gluconate 10 mg IV in 10 cc normal saline over 10 minutes was administered to her. Her condition started improving slowly and her respiratory rate and O₂ saturation became normal in 2-3 hours after giving Calcium gluconate. She was taken off the ventilator. The patient made uneventful recovery and was discharged from hospital after 7 days of admission with normal renal function.

III. Discussion:

Eclampsia is an important cause of maternal mortality. The eclampsia related mortality can be reduced by early referral and effective anticonvulsant therapy.^[2] In modern day practice, magnesium sulphate, administered parenterally has become standard form of treatment for eclampsia. Most of the hospitals follow 'Pritchard regimen' which is considered safe. This patient was inadvertently given higher dose of MgSo₄. Higher dose with compromised renal function could have been the cause of MgSo₄ toxicity. Respiratory distress is not a common complication of MgSo₄ administration. Smith et al^[3] reviewed the toxicity of MgSo₄, found respiratory distress in 1.3 % of the patients.

Blood levels of MgSo₄ could have been measured to confirm the toxicity levels. However, there is no consensus on correlation between blood MgSo₄ levels and clinical picture of the patient. It is only free magnesium levels which are responsible for suppressing neuronal electrical activity, not the total MgSo₄ levels which is measured by most of the labs. Clinical assessment of the patient remains main stay of monitoring the patient for MgSo₄ toxicity. The parameters to be monitored are- Patellar reflex, respiratory rate, urine output. Tardy or absent patellar reflexes is because of curare like action and is an impending sign for magnesium toxicity. At blood MgSo₄ levels of 12 mEq/L or more, there is respiratory paralysis followed by respiratory arrest.^[4] Treatment is with calcium gluconate, 1 gm intravenously, with discontinuation of further doses of magnesium sulphate. This treatment usually reverses mild to moderate respiratory depression. Smith et al found Calcium gluconate administration in 0.2 % cases.^[3]

There are always questions about the correct dose of MgSo₄ for our patients. Attempts have been made to reduce this dose of magnesium sulphate in Indian patients because of their small body mass. Dhaka Regimen is a low dose regimen which has been found effective among south Asian patients may be more suitable for our patients than 'Pritchard' regimen which has been formulated keeping western population in mind.^[5] Creatinine levels need to be measured to assess renal function. Normal creatinine levels in pregnancy are less than 1mg/ml. Among the patients with highly compromised renal function only loading dose of MgSo₄ may be given.

This case, patient had reduced respiratory rate, absent patellar reflexes which was suggestive of magnesium toxicity. In addition, she received higher dose of MgSo₄ than recommended due to lack of communication. She also had high creatinine levels suggestive of compromised renal function leading to decreased excretion of magnesium sulphate. Timely diagnosis, intubation and ventilation, stopping the further doses of MgSo₄ and administration of calcium gluconate saved the patient.

IV. Conclusion

Eclampsia along with hypertensive disorders in pregnancy is responsible for almost one third of maternal mortality in our country. The introduction of MgSo₄ for treatment of eclampsia has brought down the mortality considerably all around the world. However, though the MgSo₄ treatment protocol for eclampsia is generally considered safe, we do get a patient with MgSo₄ toxicity occasionally. Careful monitoring of the patient for respiratory distress, decrease urine output and absence of patellar reflex may help in diagnosis of MgSo₄ toxicity and the patients can be treated successfully with IV calcium gluconate.

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