Utility of Point-of-Care Ultrasound (POCUS) for predicting risk of magnesium toxicity in critically ill pre-eclamptic patients

INTRODUCTION

Seizures in pregnancy could be caused by varied aetiologies such as epilepsy, structural lesions, metabolic causes, tropical infections, apart from pregnancy-related diseases like eclampsia.\(^1\)\(^2\) When the first episode of seizure occurs during the third trimester of pregnancy, possible pregnancy-associated conditions need to be ruled out very quickly, and most of the times, the clinical management of conditions like eclampsia is initiated, awaiting laboratory reports. In this context, it is to be noted that point-of-care ultrasound (POCUS) is gaining value as a promising bedside tool aiding in the diagnosis and procedural management of patients in emergency and intensive care units.\(^3\)\(^-\)\(^5\) This index case has been described to highlight the role of screening by ultrasonography in the emergent management of provisionally diagnosed eclamptic women.

CASE PRESENTATION

A 29-year-old primigravida belonging to a family of low socioeconomic status presented at 35 weeks’ gestation to a primary health center (PHC) in northern India, with bilateral lower limb oedema of 4 weeks’ duration, abdominal pain with vomiting since 2 days and altered sensorium with seizures on the day of presentation. There was no history of fever, any past history of seizures, or any systemic disorder as per the history obtained from her husband, and she had not been compliant in her antenatal visits. Upon admission, she was actively convulsing with a pulse rate of 128 beats per minute (bpm), right arm blood pressure was 156/92 mm Hg and oxygen saturation (Sp\(_{\text{O}_2}\)) of 95% on pulse oximetry. Foetal heart sounds were diminished. With a provisional diagnosis of eclampsia, she was given intravenous (IV) magnesium sulphate (MgSO\(_4\)) 4 g followed by 5 g intramuscular (IM) in each buttock. As the seizures subsided, she was immediately referred to a tertiary care hospital, where she was intubated for airway protection due to low Glasgow coma scale (GCS) score of 8/15 and hypoxaemic respiratory failure (Sp\(_{\text{O}_2}\) 85% on oxygen therapy with respiratory rate: 30 bpm). Provisional diagnosis of eclampsia with aspiration pneumonia was made and empirical antimicrobials along with MgSO\(_4\) 2 g/hour IV infusion was continued in the emergency room. Intrauterine death was diagnosed and it was medically terminated by the obstetrician with misoprostol administration per vaginum with around 600–800 ml of estimated blood loss during the procedure. The patient was referred to the intensive care unit (ICU), without further neurological evaluation in the emergency room. On examination in the ICU, the GCS was E\(_1\) V\(_T\) M\(_1\), pupils were bilaterally reacting to light, muscle tone was flaccid, deep tendon reflexes were absent and she had bilateral extensor plantar reflexes. She was haemodynamically stable with a urine output of 40–50 ml/hour. Bedside POCUS revealed absence of the left kidney, a right kidney sized 10.8 × 6.5 cm, grade I echogenicity and moderate hydroureteronephrosis [Figure 1]. After this screening report, deranged renal function was suspected, with possible hypermagnesemia in view of the suggestive clinical picture, and based on the combination of history, physical examination and ultrasonographic findings, the ongoing magnesium sulfate infusion was stopped. Her investigations on admission received 2 hours later revealed haemoglobin: 5.5 g/dL; total leucocyte count: 65,000/cmm; platelet count: 85000/µL; blood urea/creatinine (in mg/dL): 170/3.0; serum sodium/potassium (in mmol/L): 131/3.0; serum magnesium: 12 mg/dL, serum lactate dehydrogenase: 1761 IU/L; alanine transaminase: 247 IU/L and aspartate transaminase: 258 IU/L. The patient was transfused with two units of packed red blood cells and the hypermagnesemia was further
managed with hydration, loop diuretics and IV calcium gluconate as an antagonist. Secondary rise of blood pressure noted from day 3 of ICU stay was managed with oral clonidine 300 µg in divided doses and amlodipine 5 mg. By the fourth day of ICU stay, sensorium and muscle tone improved, she was weaned and extubated from ventilatory support. Serum magnesium, leucocyte counts, platelet counts and transaminases normalised by day 5 of ICU stay. A non-contrast computed tomographic study of the abdomen revealed an empty left renal fossa and a single right kidney with compensatory hypertrophy and moderate hydroureteronephrosis (thought to be due to compression of gravid uterus on the kidney) [Figure 1]. She was discharged home on day 10 of ICU stay with mildly elevated renal parameters (blood urea 70 mg/dL, serum creatinine 1.4 mg/dL) on antihypertensive therapy. Three months after discharge, the patient remains hypertensive, and has chronic kidney disease (CKD), with serum creatinine of 1.4 mg/dL and an estimated glomerular filtration rate of 50.6 ml/min/1.73 m².

DISCUSSION

Through this case, we wish to highlight two issues that could prevent iatrogenic hypermagnesemia. Suspecting hypermagnesemia in a critically ill patient who would require transfer to a tertiary care unit based on clinical examination alone is a tough challenge. Magnesium is a drug with narrow therapeutic index, which is predominantly eliminated by the kidneys, the organs that are commonly compromised in preeclampsia. A study by Nagaria et al. demonstrated the successful use of single loading low dose bolus (4 gm IV followed by 4 gm IM in alternative buttocks), without any maintenance dose to prevent toxicity as many patients in the study were lean and malnourished. Expert committees of regional and national societies need to identify regimens for use in patients at a greater risk of magnesium toxicity, including women with acute or chronic kidney disease, instead of blindly applying the most popular regimens such as Pritchard’s and Zuspan’s regimens. Apart from logistic constraints, paucity of trained staff for meticulous clinical monitoring of patients receiving parenteral magnesium (like respiratory rate, deep tendon reflexes and urine output), and lack of availability of serum magnesium monitoring at the level of PHCs pose a challenge.

Secondly, we wish to highlight the role of POCUS, wherein the diagnosis of a single kidney with hydroureteronephrosis led to the decision of stopping the magnesium infusion suspecting hypermagnesemia, prior to laboratory confirmation, and in line with clinical examination findings suggestive of hypotonia and absent deep tendon reflexes. This index case may have had CKD due to congenital abnormality of kidney and urinary tract (CAKUT) anomaly with superimposed preeclampsia. Although CAKUT is unlikely to be causative to the acute kidney injury, which ultimately led to hypermagnesemia, its pick-up could definitely alert the obstetrician and the intensivist prior to laboratory confirmation. Although POCUS may not be feasible at PHCs, it would be prudent to train doctors, especially intensivists and obstetricians in such screening ultrasonographic techniques to increase their level of clinical proficiency and confidence, until the final confirmation of report comes from the radiologists.

CONCLUSION

There is a need to establish safe and effective protocols for the prescription of parenteral magnesium sulphate in the treatment of severe pre-eclampsia and eclampsia. Such protocols should keep in mind the patient build and the level of monitoring available in the hospital. Screening by ultrasound/POCUS can guide in valuable clinical decision-making in the management of such cases.

Declaration of patient consent
The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest
There are no conflicts of interest.

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