RESEARCH PAPER

Maternal Cardiorespiratory Arrest Following Standard Magnesium Sulfate Therapy for Imminent Eclampsia in a Twin Pregnancy

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Abstract

The present case report describes a woman at 30 years of age who is a first-time mother, who was diagnosed with imminent eclampsia at 28 weeks of her twin gestation and has other co-morbidities of pre-gestational diabetes and hypothyroidism. She was loaded with magnesium sulfate (MgSO4) using the standard protocol before having an urgent cesarean section followed by nearly 2 hours of recovery in the intensive care unit when she suddenly experienced bradycardia, hypotension, and pulseless electrical activity, which led to cardiac arrest. Cardiopulmonary resuscitation (CPR) and intravenous (IV) calcium gluconate were initiated immediately, resulting in return of spontaneous circulation (ROSC). Subsequent assessment following ROSC showed a CT of the floor of the brain that was normal, a serum magnesium level of 6.0 mg/dL and associated hypocalcemia and hypoalbuminemia. Due to both the pronounced clinical presentation and timing of these events, magnesium toxicity was determined to be responsible for the events described. Following her hospital course, the patient unexpectedly remained longer than anticipated, having prolonged a significant course of seizure activity, infections, and severe hypoxic-ischemic encephalopathy, eventually requiring relocation to a neuro-rehabilitation facility in a persistent vegetative state. This case highlights the potential for life-threatening toxicity from MgSO4 when used according to current medical guidelines, particularly in high-risk obstetric patients with existing risk factors for MgSO4 toxicity (i.e., hypoalbuminemia, hypocalcemia). Therefore, close monitoring of all high-risk obstetric patients and having immediate access to both resuscitation equipment and IV calcium gluconate as MgSO4's antidote, as well as correcting all electrolyte imbalances prior to administering MgSO4 are all essential to preventing MgSO4 toxicity.

Keywords: Magnesium Sulfate, Pre-Eclampsia, Eclampsia, Cardiorespiratory Arrest, Medication Toxicity, Pregnancy Complications, Intensive Care, Twin Pregnancy

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Introduction

Pre-eclampsia is a disorder that affects multiple organ systems and is unique to pregnant women. It affects approximately 2-8% of pregnancies worldwide and continues to be a leading contributor to both maternal and fetal morbidity and mortality [1]. Eclampsia, the most severe neurological manifestation of pre-eclampsia, is characterized by grand mal seizure activity in an otherwise normal-appearing pregnant woman. For more than 30 years, intravenous magnesium sulfate (MgSO4) has been the treatment of choice for the prevention and treatment of eclamptic seizures and is more effective than other medications, such as diazepam or phenytoin, at preventing and treating seizures [2]. The therapeutic effects of MgSO4 are attributed to multiple mechanisms including cerebral vasodilation, blockade of calcium influx into neurons and antagonism of excitatory neurotransmitters.

There are also risks associated with MgSO4. The narrow therapeutic window for MgSO4 means that serum magnesium needs to be maintained in a very specific range (typically between 4.8 and 8.4 mg/dL) to ensure that neuroprotection can occur while at the same time avoiding adverse systemic effects from excessive serum magnesium concentrations ([3].). The toxic effects of excessive serum magnesium concentrations are primarily related to depression of the central nervous system and cardiovascular system. The earliest indicator of magnesium toxicity is the loss of deep tendon reflexes, while progressive magnesium toxicity can lead to respiratory muscle failure, severe hypotension, bradycardia, and ultimately, cardiopulmonary arrest ([4.]). Patients that have renal insufficiency, hypocalcemia or other disease states that impair the distribution or metabolism of magnesium are at greatest risk of magnesium toxicity. We present a case of a pregnant woman who suffered an immediate cardiac arrest after receiving a standard MgSO4-loading dose for imminent eclampsia. The importance of close monitoring of patients,

ability to provide timely access to resuscitation protocols, and awareness of specific risk factors for patients are critical due to the possibility of these patients deteriorating rapidly.

Case Presentation

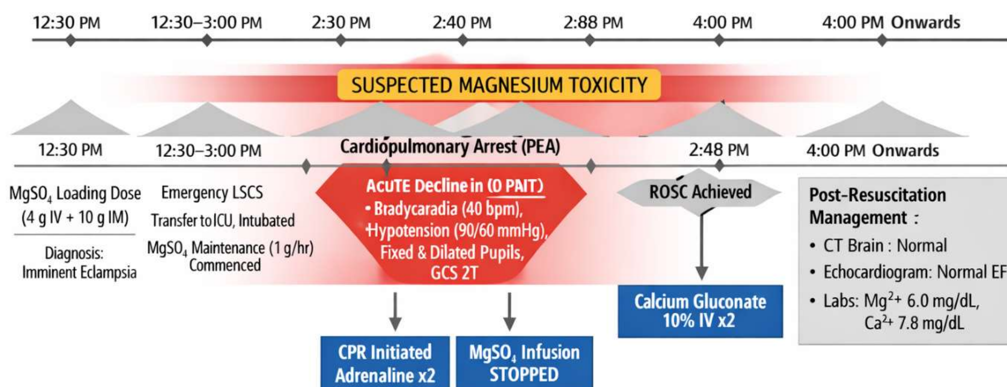
History of Patient and Initial Presentation

A 30-year-old woman, who is pregnant for the first time by in vitro fertilization (IVF), is 28 weeks since inception with twins with two placentas (dichorionic) and two amniotic sacs (diamniotic). She saw her obstetrician on January 10, and on January 11, she was diagnosed at that same appointment with worsening preeclampsia. She has preexisting diabetes (controlled with complex insulin regimen with an insulin lispro and glargine combination), gestational hypertension (on labetalol and nifedipine) and hypothyroidism (treatment with 50 mcg levothyroxine daily). Upon physical examination, she was afebrile, did not exhibit signs of palor, and demonstrated substantial bilateral pitting edema on her feet. Vital signs showed blood pressure of 130/80 mmHg and pulse of 88 beats/minute. On abdominal examination, her abdomen was consistent with that of a 30-32 week multipara with twins with reassuring fetal heart sounds. Initial urinalysis produced significant levels of proteins (2+) in urine. Therapy at that time included initiation of anti-hypertensive (labetalol with nifedipine), anti-platelet (aspirin), insulin replacement therapy, thyroid replacement (levothyroxine), and corticosteroids (dexamethasone) for speeding fetal lung development.

Deterioration and Clinical Intervention

The fourth day following admission reveals a new complaint of a severe headache and visual disturbances (i.e., "floaters"), with the possibility that the client is presenting with neurological symptoms consistent with the impending diagnosis of eclampsia. According to standard protocol, the client was given a loading dose of magnesium sulfate (Mag) = 4 grams IV over 20 minutes, followed by 10 gm IM divided into 2 doses (5grams each injected deep into each gluteal muscle). After reviewing with a multidisciplinary team that a diagnosis of imminent eclampsia is present, a decision was made to proceed with an immediate delivery of the twins. Emergency lower segment cesarean section under general anesthesia was performed at which time live, pre-term twins were delivered (twin A = 1.04 kg; twin B = 0.975 kg). Since neither twin exhibited any spontaneous respiratory effort at that time, the patient was intubated and transferred to the ICU with mechanical ventilation. The patient was initiated on an intravenous infusion of Mag at 1 gram per hour after surgery for maintenance.

Figure 1: Timeline of critical events following magnesium sulfate administration



Around two hours after she received the loading dose of medication, while she was still in the intensive care unit (ICU), the patient experienced an abrupt and dramatic deterioration in status. She developed severe bradycardia (heart rate 40 bpm) and severe hypotension (blood pressure 90/60 mmHg). Neurologically, her pupils were fixed and dilated (3mm, despite being non-reactive), and her Glasgow Coma Scale (GCS) score was 2T. Within minutes, this patient progressed to pulseless electrical activity (PEA) cardiac arrest. Cardiopulmonary resuscitation (CPR) was immediately provided and further advanced cardiac life support (ACLS) was provided according to protocols,

including administering 2 doses of 1mg intravenous epinephrine. Return of spontaneous circulation (ROSC) occurred approximately 8 minutes after CPR was initiated. Concurrent with the resuscitation effort, the $MgSO_4$ infusion was immediately discontinued. Post-ROSC, the specific antidote for Mg toxicity, intravenous calcium gluconate (10 ml of a 10% solution, given slowly), was given as soon as ROSC occurred, and subsequently given again.

Upon achieving ROSC and afterwards, the course of the patient's management was critically complicated. Initially, she developed a focal motor seizure (involving the left upper and lower extremities), which was managed using intravenous benzodiazepines (midazolam and lorazepam), along with a loading dose of levetiracetam. The patient underwent expeditious investigations in order to rule out other possible causes of her clinical deterioration. A non-contrast CT scan of the head was normal, and a transthoracic echocardiogram showed normal cardiac function with no regional wall motion defects. There were laboratory findings post-arrest which included an elevated serum magnesium level of 6.0 mg/dL (within the high end of the therapeutic range); an elevated serum calcium level of 7.8 mg/dL; significantly elevated liver transaminases (AST 931 U/L, ALT 318 U/L), which point to liver cellular damage likely from either ischemia or from severe pre-eclampsia (variant of HELLP syndrome) as contributing factors to the deterioration of this patient. During her extended stay in the ICU, she acquired pneumonia and urinary tract infections, both of which were treated with appropriately cultured antibiotics.

Table 1: Summary of Key Investigations Before and After Cardiorespiratory Arrest

Parameter	Pre-Arrest / Admission (24-28/10/24)	Post-Arrest / Critical Phase (28/10/24 onwards)	Reference Range / Clinical Significance
Vital Signs	BP: 130/80 mmHg, PR: 88 bpm, Afebrile	BP: 90/40→140/90 mmHg, PR: 40→130 bpm, GCS: 2T	Acute deterioration indicates cardiovascular collapse.
Neurological	Alert, oriented. Complaints of headache & floaters.	Pupils: 3mm, non-reactive. Post-ROSC: focal seizure (L UL/LL). GCS: E1VTM1→E4VTMM6 on discharge.	Findings consistent with profound neurological depression and subsequent hypoxic injury.
Serum Magnesium	Not measured prior to loading dose.	6.0 mg/dL (drawn post-arrest, ~2 hrs post-loading dose)	Therapeutic: 4.8–8.4 mg/dL. Level in high-therapeutic range at time of arrest.
Serum Calcium	Not measured on admission.	7.8 mg/dL (post-arrest)	Normal: 8.6–10.2 mg/dL. Concomitant hypocalcemia potentiates Mg toxicity.
Serum Albumin	Diagnosed with hypoalbuminemia.	Low (specific value not documented).	Low albumin increases free, active Mg^{2+} fraction, raising toxicity risk.
Liver Function	Normal on admission screening.	AST: 931 U/L, ALT: 318 U/L, LDH: 2111 U/L	Marked transaminitis suggests hepatocellular injury from ischemia/severe pre-eclampsia.
Renal Marker	Urine Albumin: 2+	Spot Protein:Creatinine Ratio: 17.76	Confirms significant glomerular involvement consistent with severe pre-eclampsia.
Imaging – CT Brain	Not performed.	Normal (post-arrest)	Excludes intracranial hemorrhage or infarct as cause of collapse.
Cardiac – Echocardiogram	Not performed.	EF: 55-62%, No RWMA, IVC Collapsing	Excludes peripartum cardiomyopathy; supports hypovolemia/distributive shock post-arrest.

Neurological recovery was profoundly limited. The patient remained ventilator-dependent, requiring a tracheostomy, and was maintained on enteral feeding via a nasogastric tube. She exhibited minimal consciousness, inconsistently following verbal commands. After weeks of ICU management, she was transferred to a specialized neurorehabilitation facility in a persistent vegetative state. Her medication regimen at discharge included agents for neurostimulation (Brevipill), neuroprotection (Citicoline), anticoagulation (Rivaroxaban), hypertension (Metoprolol, Telmisartan), neuropsychiatric symptoms (Quetiapine, Olanzapine), and her pre-existing endocrine conditions.

Discussion

This patient suffered a severe and life-threatening complication from a standard treatment used for an obstetrical emergency. The timing of the event, which occurred within two hours after starting MgSO4 therapy, strongly suggests that magnesium toxicity was the cause of her cardiovascular collapse, even though her serum magnesium level at the time of collapse was within the normal therapeutic range. This case illustrates an important tenet of clinical medicine; magnesium toxicity is a clinical diagnosis, and the life-threatening complications of magnesium toxicity may occur even before serum levels reach the defined laboratory toxic level due to the rate of increase in serum magnesium levels [5].

Certain characteristics of this patient may have contributed to her developing magnesium toxicity. First, the hypoalbuminemia present in this patient may have resulted in an increase in the free ionized magnesium concentration, increasing the pharmacologic and toxic effects of magnesium at a given total serum magnesium concentration [6]. Second, this patient's pre-existing hypocalcemia (serum calcium of 7.8 mg/dL) likely acted as a synergistic risk factor; calcium and magnesium are physiologic antagonists at the neuromuscular junction, and the low serum calcium level in this patient would diminish the competitive inhibition of magnesium on cardiac conduction and respiratory muscle function [4]. Finally, the immediate postpartum period is associated with significant volume shifts and variation in renal perfusion, making drug clearance and distribution unpredictable, which could lead to greater than expected tissue concentrations.In a peripartum patient with sudden cardiorespiratory collapse, a broad differential diagnosis exists; pulmonary embolism (PE), amniotic fluid embolism (AFE), complication from anesthetics, intracranial hemorrhage (ICH), and acute coronary syndromes are all examples of possible causes. Rapid exclusion of many of these diagnosis through clinical examination, normal neuroimaging, and normal echocardiography supports direct toxicological causes of this patients collapse. The immediate treatment with calcium gluconate, which reverses the competitive effects of magnesium at the motor endplate, was both a definitive and likely emergent life-saving intervention.

The understanding of approach to administration of Magesium Sulfate was reinforced by this report, as such: 1. The administration of MgSO 4 must occur in an environment where continuous monitoring of deep tendon reflexes; respiratory rate; oxygen saturation; and level of consciousness, occurs; 2. The antidote (calcium gluconate) must be readily available at the bedside; 3. In high risk patients (ie. those with hypocalcemia and hypoalbuminemia), electrolyte imbalances should be assessed and corrected prior to or together with the administration of MgSO 4 and; 4. Healthcare teams trained to identify subtle, early signs of magnesium toxicity (ie. absent patellar reflex) will be able to intervene before serious respiratory or cardiac depression occur.

Conclusion

For the treatment of eclampsia, the use of magnesium sulfate is an essential, life-saving treatment. However, medications like magnesium sulfate that have a narrow therapeutic window necessitate close clinical monitoring. This case report demonstrates how rapid cardiorespiratory collapse occurred following administration of the standard dose of magnesium sulfate due to 2 specific underlying risk factors (hypocalcemia and hypoalbuminemia). As demonstrated in this case, the delivery of protocol-based care should be individualized based on each patient's unique risk factor profile and be adapted dynamically while the patient receives care. Adequate magnesium sulfate administration along with the ability to quickly access resuscitative equipment and the specific antidote must be included as essential components of any clinical setting that uses magnesium sulfate.

Patient consent

For the purposes of this case report, informed consent for publication was obtained in writing from the patient's legally authorized representative (her husband) who stated that the information contained in this report has educational value in creating a safer environment for patients in the future.

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