Unexpected acute respiratory failure following administration of rocuronium bromide during cesarean delivery in a severely preeclamptic parturient treated with magnesium sulfate*

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ABSTRACT

Magnesium sulfate has been a mainstay in the management of preeclampsia and is associated with a decreased incidence of morbidity and mortality. The hypertensive disorder has an unpredictable course, sometimes rapidly evolving to full-blown disease. In patients with deteriorating status, it is indicated to terminate the pregnancy via cesarean section. The anesthesiologists would prefer to have the procedure done under regional anesthesia; however, there may be cases when neuraxial anesthesia is contraindicated, or a general anesthesia would permit prompt delivery of the fetus.

A patient with severe preeclampsia was given magnesium sulfate intrapartum, wherein a primary cesarean section was indicated for arrest in cervical dilatation, and was performed under general anesthesia. The patient developed acute respiratory failure and the causes of this occurrence were investigated in this report. It was later found out that neither the hypermagnesemia nor the muscle relaxant alone caused the patient's condition but the interaction between the two. The patient was managed expectantly at the intensive care unit (ICU) and was eventually extubated during the first post-operative day. Knowledge of this drug interaction would allow obstetricians to advise their patients and their family about the possibility of prolonged intubation and ICU admission. This would also bring to the anesthesiologists' attention the need to decrease the dose of muscle relaxant and to prepare drugs for immediate decurarisation.

Keywords: Magnesium sulfate, preeclampsia, eclampsia, preeclampsia with severe features, rocuronium

INTRODUCTION

Hypertensive disorders of pregnancy complicate approximately 10% of pregnancies worldwide.[1] In particular, preeclampsia affects about 3% of pregnancies in the United States but is believed to be higher in underdeveloped countries[2] such as the Philippines. The incidence of hypertensive disorders in pregnancy is increasing and is associated with maternal and perinatal morbidity and mortality.[3] Worldwide, 10% to 15% of direct maternal deaths are associated with preeclampsia and eclampsia. Where overall maternal mortality is high, most deaths are associated with eclampsia rather than pre-eclampsia. Perinatal mortality is high following preeclampsia, and even higher following eclampsia.[4] In the United States, there is approximately one maternal death due to preeclampsia-eclampsia per 100,000 live births, with a case fatality rate of 6.4 deaths per 10,000 cases.[5] The incidence of eclampsia has been relatively stable at 1.6 to 10 cases per 10,000 deliveries in developed countries.[6,7] In developing countries, however, the incidence is as high as 157 cases per 10,000 deliveries.[8]

Despite the relatively low incidence of eclampsia, the condition remains strongly associated with serious adverse consequences. In a study by Liu et al in 2011, they found out that eclampsia was associated with increased risks of maternal death, assisted ventilation, respiratory distress syndrome, acute renal failure, obstetric embolism, and other complications. Adverse neonatal outcomes associated with eclampsia included neonatal death, respiratory distress syndrome, and small-for-gestational age birth.[9]

An eclamptic seizure occurs in 2 to 3 percent of severely preeclamptic woman not receiving anti-seizure prophylaxis, whereas the seizure rate is estimated to be between 0 and 0.6 percent in women with preeclampsia without severe features.[10] Onset of eclamptic convulsions can be antepartum (38-53%), intrapartum (18-36%), or postpartum (11-44%). [11]

Several studies have compared the efficacy of several medications in the prevention of eclampsia. In a multicenter trial by Belfort et al, magnesium sulfate was found to be more effective than nimodipine for prophylaxis against seizures in women with severe preeclampsia.[12] On the other hand, Lucas et al. compared magnesium sulfate with phenytoin in preventing eclamptic seizures, with evidence that the former is superior.[13] Indeed, magnesium sulfate therapy for prevention of eclampsia is cited as one of the essential evidence-based interventions that could potentially eliminate the untimely deaths of 358,000 women in low- and middle-income countries.[14] In

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the Magpie Trial (2002), women given magnesium sulfate had a 58% lower risk of eclampsia than those allocated placebo. Maternal mortality was also lower among women allocated magnesium sulfate.[15]

Termination of pregnancy by cesarean section is usually indicated for uncontrolled hypertension along with the complications that occur with the syndrome. Despite the inherent advantages of neuraxial anesthesia for cesarean section in parturients with preeclampsia or eclampsia, threatened coagulopathy and risks for hemodynamic instability often compel anesthesiologists to proceed with urgent general anesthesia. We present here a case of a parturient with severe preeclampsia treated with magnesium sulfate and delivered by cesarean section under general anesthesia, eventually developing acute respiratory failure during the immediate post-operative period.

CASE

The patient is a 33-year old gravida 2 para 0, Filipino woman who presented at 39 weeks age of gestation with elevated blood pressure. She was diagnosed with hyperthyroidism in 2001, presenting as palpitations and tremors. She underwent radioactive iodine ablation in 2005 after consult with an endocrinologist and was maintained on Levothyroxine 75 mcg once daily during pregnancy. Thyroid function tests during pregnancy were normal. There were no known food or drug allergies, and the family history was unremarkable. She was working as a customer service representative prior to current pregnancy. Her first pregnancy was a spontaneous abortion and the cause of which was not investigated. She had her first prenatal check up at 8 weeks, and had an unremarkable prenatal course until 38 weeks age of gestation when she started having blood pressure elevations at 140/90 mmHg, for which she was started on Methyldopa 250 mg thrice daily. Blood pressure was then maintained at 120/80 mmHg. She underwent routine urinalysis at that time, which showed absence of proteinuria. Assessment during that time was gestational hypertension.

On the day of admission, patient had a blood pressure of 180/110 mmHg. There was no associated headache, dizziness, blurring of vision, chest pain, palpitations, shortness of breath, difficulty of breathing, or abdominal pain. The patient was ambulatory and not cardiorespiratory distress. She had no pallor or neck vein distension. Breath sounds were clear on all lung fields; heart rate and rhythm were normal and regular, respectively. She had a gravid abdomen; with symphysis-to-fundus height measuring 31 cm. Grade 2 pedal edema was documented. Admitting cardiotocogram was reactive with a baseline fetal heart rate of 135, with moderate variability and accelerations, and no decelerations. One moderate uterine contraction was recorded in 20 minutes (Figure 1). Internal examination revealed a cervix

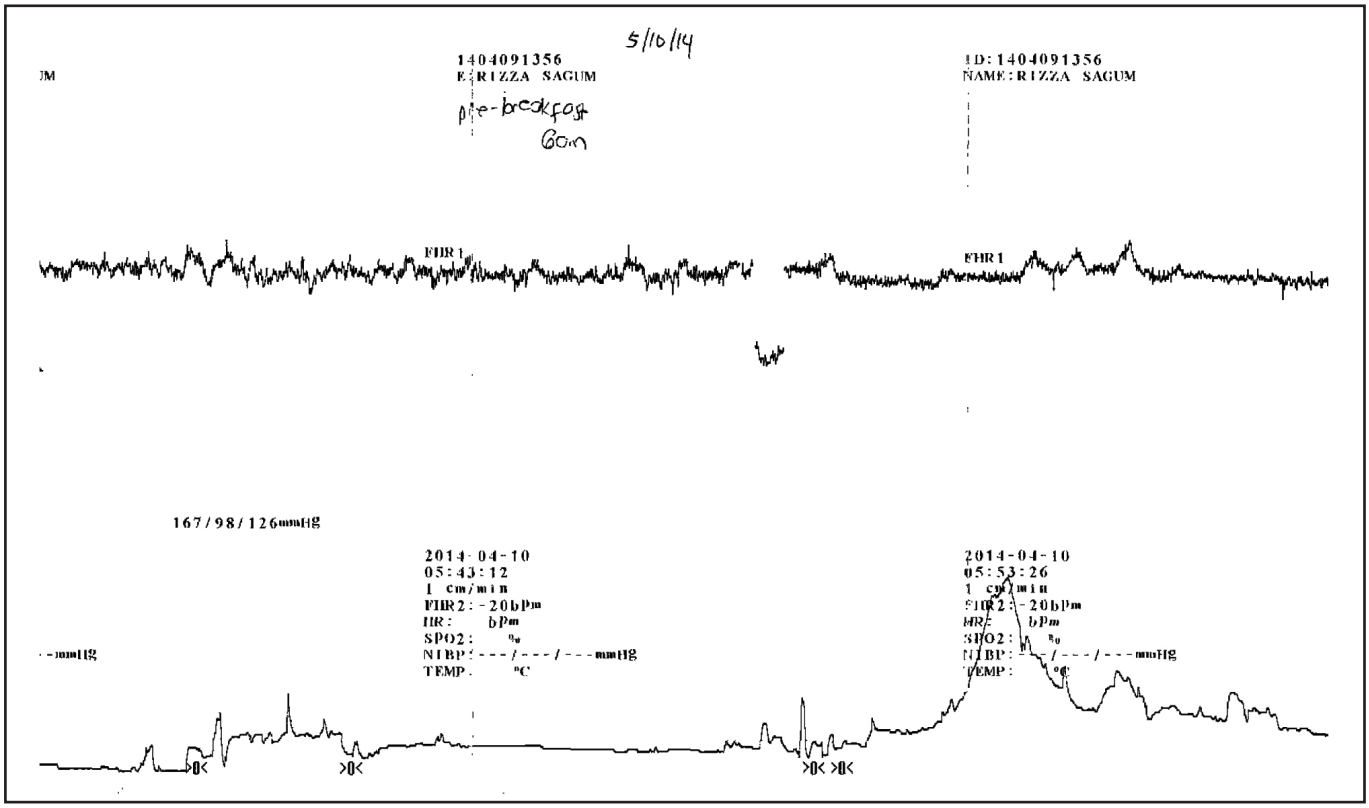

Figure 1.

that was 1 cm dilated but uneffaced, presenting part at station -2, and with intact bag of waters. Patient was then admitted for blood pressure control, hypertensive work-up and labor induction. Admitting impression was that of a G2P0 (0010) pregnancy uterine 39 weeks AOG, cephalic, in beginning labor, gestational hypertension rule out preeclampsia. Hydralazine 5 mg/IV was initially given, with a goal of maintaining blood pressure of 153/83 mmHg. Blood pressure increased to 190/110 mmHg after the first dose of Hydralazine, hence a 10mg/IV dose was given, which lowered the blood pressure to 120/70. She was also maintained on Methyldopa 250mg/tab thrice daily. Cervix was primed using Evening Primrose Oil 1000mg soft gel capsules intravaginally.

With the highest blood pressure recorded at 190/110 mmHg and a 2+ proteinuria, the patient was managed as a case of preeclampsia with severe features; hence seizure prophylaxis with magnesium sulfate was started. Baseline deep tendon reflexes were 2+ on all extremities. An indwelling Foley catheter was inserted for accurate urine output monitoring. Magnesium sulfate was given as a loading dose of 4g/IV and maintained on magnesium sulfate infusion at 2g/hour. Fetal heart rate continued to have moderate variability with accelerations and no decelerations and blood pressure was maintained at 120-130/70-80 mmHg. The rest of the baseline hypertensive work-up may be found in Table 1.

On the 5th hour after initiating magnesium sulfate, patient had an episode of vomiting. Patient denied having headache, blurring of vision, dyspnea or shortness of breath. Blood pressure at that time was 120/80 mmHg, breath sounds were clear on all lung fields, fetal heart beat at 130 bpm, uterine contractions were mild occurring every 8 minutes, urine output remained clear and adequate and deep tendon reflexes were 2+ on all extremities. Serum magnesium level was determined on the 6th hour of magnesium sulfate drip, which was 6.6 mg/dL or 5.5 meq/L (within therapeutic level of 4 to 7 meq/L). On the 7th hour of magnesium sulfate drip, patient started to complain of bitemporal headache, with no other associated symptoms. Blood pressure was 160/90 mmHg, for which she was given a Paracetamol 500mg/tab and a stat dose of Hydralazine 5mg/IV, which lowered the blood pressure to 130/70 mmHg.

On the 17th hour of magnesium sulfate drip, patient was noted to be awake, with no recurrence of vomiting or headache. However, urine output was noted to be blood-tinged but adequate. Internal examination revealed a cervix that was 2 cm dilated, 50% effaced, presenting part at station -2, with intact bag of waters. Cardiotocogram remained reactive, with mild uterine contractions every 10 minutes. A repeat serum magnesium level was requested, and was found to be 7.6 mg/dL or 6.3 meq/L.

Table 1. Baseline hypertensive work-up

Test	Value	Interpretation
CBC		
Hemoglobin	14.3 g/dl	Normal
Hematocrit	41.9%	Normal
RBC	4.88 mil/mm^3	Normal
WBC	9800 mm^3	Normal
Segmenters	70%	Normal
Platelets	385,000/mm^3	Normal
Creatinine	0.41 mg/dL	Normal
BUN	6 mg/dL	Normal
ALT	32 U/L	Normal
AST	34 U/L	Normal
LDH	255 U/L	Normal
Sodium	138 mmol/L	Normal
Potassium	4.1 mmol/L	Normal
Urinalysis		
Glucose	Negative	Proteinuria
Ketone	Negative	
Specific Gravity	1.003	
pH	6.5	
Protein	100 mg/dL (2+)	
Nitrites	Negative	
Blood	Negative	
Leukocytes	Negative	
RBC	1/hpf	
WBC	1/hpf	
Epithelial cells	1/hpf	
Bacteria	1/hpf	

On the 20th hour of magnesium sulfate drip, patient complained of difficulty of breathing and headache, with blood pressure of 180/100 mmHg. She was tachypneic at 30 cycles per minute but breath sounds remained clear on all lung fields; urine output remained blood-tinged but adequate. Oxygen saturation was 98-99% on room air. Fetal heart rate tracing was reassuring, with accelerations, and no decelerations recorded. Uterine contractions remained mild every 10 minutes. Deep tendon reflexes were still 2+ on all extremities. She was given supplemental oxygen, placed on left lateral decubitus position, and magnesium sulfate drip was then discontinued due to the possibility of magnesium toxicity. However, test results showed that the serum magnesium level was still within the therapeutic range (Table 2).

Table 2. Work-up on the 20th hour of labor augmentation

Test	Value	p-value
CBC		
Hemoglobin	14.6 g/dl	Normal
Hematocrit	44.5%	Normal
RBC	5.09 mil/mm3	Normal
WBC	18000 mm3	Normal
Segmenters	90%	Normal
Platelets	405,000/mm3	Normal
Creatinine	0.45 mg/dL	Normal
ALT	35 U/L	Normal
AST	96 U/L	Normal
LDH	378 U/L	Normal
Magnesium	6.2 mg/dL or 5.1 meq/L	Within therapeutic range
PTT	32.6 sec	Normal
Protime		Normal
Control	12.2 sec	
Test	11.0 sec	
INR	0.92	

Blood pressure and progress of labor were closely monitored thereafter. Magnesium sulfate drip was not restarted. There was spontaneous rupture of membranes, and internal examination revealed a cervix that is 4 cm dilated, 60% effaced, station -2. Uterine contractions were mild occurring every 8 minutes; hence labor was augmented with oxytocin. There were no decelerations noted intrapartum. Blood pressure remained at 150/80-90 mmHg. There was no progress in labor after 3 hours; therefore, a primary cesarean section was done for arrest in cervical dilatation at 4 cm. Due to complaints of difficulty of breathing presumed to be premonitory of respiratory failure secondary to hypermagnesemia, the anesthesiologist opted for the patient to undergo the procedure under general endotracheal anesthesia. Patient was given Propofol 100 mg/IV, Fentanyl 25 mcg/IV and Midazolam 2.5 mg/IV for induction; and Rocuronium 30 mg/IV as muscle relaxant. Controlled mechanical ventilation was instituted and anesthesia was maintained with Sevoflurane. The baby was delivered within 4 minutes from cutting time and the procedure lasted for 44 minutes, with a total operative blood loss of 380 ml. Intraoperatively, blood pressure ranged from 90/50 mmHg to 200/120 mmHg and a total of 10 mg Hydralazine was given. The patient delivered to a live baby girl with an Apgar score of 7 and 8 at 1 and 5 minutes, respectively.

During emergence from general anesthesia, patient was noted to have shallow respirations and unarousable. End tidal carbon dioxide concentration ranged from 44 to 61 mmHg despite continuous positive pressure ventilation, but oxygen saturation was maintained from 96-99%. Blood pressure was 160/100mmHg; heart rate and rhythm were normal, with no murmurs noted; but breath sounds were decreased on bilateral lung fields. Referral to cardiology and pulmonology services were made and work-up for probable causes of the patient's condition showed that the serum magnesium level was still within therapeutic range. Cardiac causes were also ruled out (Table 3).

One hour after the procedure, there was still no spontaneous respiration and end tidal carbon dioxide was 61 mmHg. Patient was then transferred to the intensive care unit for closer monitoring. Mechanical ventilator was maintained on AC mode, with a tidal volume of 400cc, and peak flow of 40. Working impression upon transfer to the ICU was acute respiratory failure type II secondary to hypermagnesemia, hence 10 mL of 10% calcium gluconate was administered. However, no improvement in patient's condition was achieved.

On the 5th post-operative hour, patient was already arousable with spontaneous breathing. Blood pressure ranged from 100-140/60-90 mmHg. There were no episodes of desaturations. A repeat arterial blood gas was done 2 hours after hooking to mechanical ventilation, which showed uncompensated metabolic acidosis with underlying respiratory acidosis.

On the 1st post-operative day, the patient has sustained wakefulness and with no complaints of dyspnea or shortness of breath. Blood pressure was maintained at 110-130/70-90 mmHg, and there were no episodes of desaturations. Serum electrolytes were serially determined showing improvement of serum magnesium and calcium levels (Table 4).

Hypocalcemia noted on the 1st operative day was corrected with a single cycle of 1g calcium gluconate in 250 cc PNSS. Correction of the hypocalcemia eventually resulted to a decline in the serum magnesium level.

Patient continued to have stable neurologic status and vital signs on the 2nd post-operative day. Weaning from the mechanical ventilator was started. Arterial blood gas determined during weaning with T-piece, which showed correction of the initial respiratory acidosis. Patient was able to tolerate weaning from mechanical ventilator, was eventually extubated and was given supplemental oxygen via nasal cannula. There were no episodes of dyspnea, shortness of breath or desaturations after extubation. Blood pressure was managed with Amlodipine 10 mg once daily and was maintained at 130-150/80-90 mmHg. Patient's condition continued to improve; hence, she was cleared for transfer to regular room. Patient's course in the wards remained unremarkable, with no episodes

Table 3. Work-up during the immediate postpartum

Test	Value	Interpretation
CBC		
Hemoglobin	14.1 g/dl	Normal
Hematocrit	41.3%	Normal
RBC	4.78 mil/mm3	Normal
WBC	23860 mm3	Normal
Segmenters	90%	Normal
Platelets	407,000/mm3	Normal
Sodium	134 mmol/L	Low
Potassium	3.7 mmol/L	Normal
Magnesium	5.0 mg/dL or 4.2 meq/L	Within therapeutic range
Ionized calcium	0.90 mmol/L	Low
Creatinine	0.78 mg/dL	Normal
BUN	6 mg/dL	Normal
Arterial Blood Gas		
pH	7.063	Combined respiratory and metabolic acidosis
pCO_2	78.7 mmHg	
pO_2	284.8 mmHg	
HCO_3	21.4 mmol/L	
O_2 saturation	97.8%	
O_2 content	21.2 mL/dL	
Chest X-ray	Consider pulmonary congestion with pleural effusion on the left; cardiomegaly	
ECG	Sinus rhythm with prolonged QT interval (45 sec)	
2D Echo with Doppler	Normal left and right ventricular dimensions with adequate wall motion and contractility Normal left and right atrial diameters Thickened anterior mitral leaflet without restriction of motion; structurally normal aortic, tricuspid and pulmonic valves Mitral, tricuspid and pulmonic regurgitation, mild	

Table 4. Serial electrolyte monitoring

Test	Day 1	Day 2
Magnesium	3.9 mg/dL or 3.25 mmol/L	2.0 mg/dL or 1.67 mmol/L
Ionized calcium	0.87 mmol/L	1.10 mmol/L

of dyspnea, shortness of breath, or weakness, with vital signs stable. Patient was eventually discharged on the 6th hospital day; with the impression of acute respiratory failure type II secondary to prolonged relaxant effect of rocuronium bromide by hypermagnesemia, resolved. Patient is now on her 14th month post-operative period, with blood pressure maintained between 100-120/80-90 mmHg.

DISCUSSION

Our patient was diagnosed as a case of preeclampsia with severe features when the highest blood pressure was recorded at 190/110 mmHg associated with proteinuria detected in urine dipstick. She was given magnesium sulfate prophylaxis as recommended in the literature: a 4g intravenous loading dose followed by continuous infusion at 2g/hour. This continuous intravenous regimen is deemed adequate in achieving the recommended therapeutic level

of 4 to 7 meq/L at which eclamptic seizures are prevented.[4]

The precise site of action of magnesium sulfate in eclampsia is not known. It has been shown to block the NMDA subtype of glutamate channel through which calcium channel enters the cell and causes neuronal damage through cerebral ischemia.[16] Ischemia causes lowering of the transmembrane potential allowing calcium ion influx across the membrane and from the endoplasmic reticulum and mitochondria. This leads to further calcium influx as activated enzymes hydrolyze membrane phospholipids. Magnesium blocks calcium at intracellular sites in addition to the outer lipid membrane.[14]

Despite the clinical usefulness of magnesium sulfate, the drug is without adverse effects. Investigators have related the pharmacological and toxic effects of magnesium to its concentration in the plasma.[14] By far, the most common side effect was flushing,[4] which was not seen in our patient. The first warning sign of impending toxicity in the mother is loss of patellar reflex at plasma concentrations between 7 to 10 meq/L.[19] During the course of magnesium infusion in our patient, there were no episodes of hyporeflexia, providing security for the continued administration of the medication. Serial serum magnesium level determination done on our patient revealed that serum magnesium levels were within the therapeutic range.

More severe adverse effects of magnesium toxicity are respiratory paralysis and cardiac arrest, which occur at serum levels greater than 10 and 25 meq/L, respectively.[17] Hence, when the patient complained of difficulty of breathing on the 20th hour of magnesium infusion, the medication was discontinued and a serum magnesium level was again determined, which remained to be within the therapeutic range. It may be recalled from above that at about the same time, there was lack of progress of labor; therefore, a primary cesarean section was planned for arrest in cervical dilatation, and that the procedure was performed under general endotracheal anesthesia for more controlled ventilation.

There were no complications encountered until during emergence from anesthesia, when the patient was noted to have shallow breathing, with signs of carbon dioxide retention. Cardiac causes of the patient's condition were ruled out with a normal 2D echocardiogram, and chest X-ray findings were presumed to be part of the syndrome that is preeclampsia. Therefore, the case was believed to be secondary to hypermagnesemia; hence, calcium gluconate IV bolus was given.

Independently, hypermagnesemia is hypothesized to cause respiratory depression through its action on end-plate potentials by competing with calcium ions. The ions antagonized each other with high magnesium concentrations inhibiting the release of acetylcholine and high calcium concentrations increasing the release of the same neurotransmitter from the presynaptic nerve terminal. Aside from its action on presynaptic nerve terminal, magnesium has also been shown to have inhibitory effects on postsynaptic potentials, with a net decrease in muscle fiber membrane excitability.[18] However, in a review of literature of the side effects related to the use of magnesium sulfate for preeclampsia and eclampsia management published by Smith et al, the incidence of respiratory depression among 9,556 women is only 1.3%, with 77 women needed to be treated to experience one incidence.[20] In a randomized trial published by Charoenvidhya and Manotaya, they concluded that significantly more women reached a therapeutic level of serum magnesium at 2 hours and 4 hours, and that no clinical magnesium toxicity was observed using an intravenous loading dose of 5g.[21]

An intravenous loading dose of 4g causes an immediate but transient increase in plasma concentrations to 4.2 to 7.6 mEq/L, which declines to 2.6 to 3.4 mEq/L within 60 minutes. An infusion rate of 2g/h such as that used in the patient yields a steady-state concentration of 4.4 mEq/L at 6 to 8 hours; and, at a constant infusion rate, serum concentrations reach a plateau when the rate of urinary excretion of magnesium equals rate of infusion. Urinary excretion is very rapid and increases 20-fold during magnesium sulfate infusion. At the end of 4 hours, from 28 to 53% of the total injected magnesium has been excreted. In patients like ours who have normal renal function, the magnesium clearance increases as a roughly linear function of the serum magnesium concentration. Furthermore, the half-life of magnesium sulfate in patients with normal renal function is as short at 4 hours.[14] These data support the improbability for magnesium to solely cause our patient's symptoms.

Although succinylcholine is the only agent that satisfies the requirements imposed by the constraints on intubation in pregnant women, this agent was not used due to the risk of developing hyperkalemia secondary to magnesium sulfate.[22] In these situations, the agent of choice for induction is rocuronium.[23] Aside from this, rocuronium has the advantage of not causing residual paralysis in the neonate.[24]

Rocuronium is a member of the non-depolarizing neuromuscular blocking agent family that antagonizes the action of acetylcholine in a competitive manner at the postsynaptic nicotinic receptor. It has a rapid onset (75 seconds) and intermediate duration of action (33 minutes). These parameters were comparable to observations in the non-obstetrical population. The only metabolite detected in the plasma (17-desacetylrocuronium) is 20 times less potent than the parent drug and not likely to contribute to neuromuscular block.[25] Therefore; rocuronium alone

could have not caused the patient's condition.

In a randomized trial published by Czarnetzki et al in 2010, it has been found that magnesium sulfate prolonged the duration of neuromuscular blockade by 27-34%.[26] This was supported by a later publication by Kim et al in 2012, wherein data showed a 30% increase in the duration of blockade when magnesium sulfate was used with rocuronium.[27]

Yoshida et al also reported the same observation in 2006 in a patient with severe preeclampsia necessitating cesarean section under general anesthesia. In their case, the parturient was also given magnesium sulfate for seizure prophylaxis, and vecuronium, a non-depolarizing neuromuscular agent, prior to tracheal intubation. In their anticipation of prolonged neuromuscular blockade in the presence of magnesium sulfate, the dose of vecuronium used was reduced. The procedure was also unremarkable until emergence from anesthesia, when the patient was noted to have insufficient breathing. Atropine and neostigmine were given for decurarisation, which initially reversed blockade to up to 90%. Endotracheal tube was successfully removed thereafter, but the patient again complained of dyspnea with shallow and frequent breathing. She was re-intubated and transferred to the ICU as well. Their patient also had serum magnesium level within the therapeutic range. It was only until the 5th post-operative hour that their patient fully recovered from neuromuscular blockade,[28] comparable to what happened to our patient.

The interaction of non-depolarizing neuromuscular blocking agents and magnesium involves several mechanisms. In the presence of high concentrations of magnesium, there is: (1) decreased pre-junctional release of acetylcholine via the inhibition of voltage-dependent calcium channels; (2) reduced sensitivity of the endplate to acetylcholine; and (3) attenuated direct excitability of muscle fibers, presumably by altering the electrical threshold of the muscle membrane.[27]

The possibility of prolonged neuromuscular blockade should have been anticipated in this case. Although there is good evidence that quantitative neuromuscular monitoring will decrease the risk of residual paralysis, many clinicians still do not monitor routinely in daily practice. A study done in the US showed that up to 40% of patients were not monitored for residual paralysis. Also, despite the use of neuromuscular blocking agents, only 43% of clinicians used neuromuscular monitoring post-operatively. Quantitative monitoring may be accomplished using the gold standard mechanomyography.[29] However, in a low-resource setting such as the Philippines, this type of monitoring may not be readily available. Hence, anesthesiologists and obstetricians alike should be vigilant in identifying this rare but real anesthetic complication.

One method that may be employed to prevent this rare occurrence is to decrease the amount of rocuronium used in endotracheal intubation. The usual dose used is 0.6-mg/kg-body weight, the dose used in our patient. If this is done, the duration of neuromuscular blockade in those whom magnesium is concurrently administered (43 minutes) is not likely to have substantially greater clinical implications than the duration of action in those who were not pre-treated with magnesium.[27]

However, when this complication is encountered, using cholinesterase inhibitors (neostigmine, edrophonium, or pyridostigmine) or calcium may reverse the potentiation of non-depolarizing relaxants by magnesium. However, these agents do not seem to be the optimal choice because of their transient effects. Cholinesterase inhibitors are not capable of reversing deeper levels of neuromuscular blockade because the amount of acetylcholine that will compete with the neuromuscular blocking agent at the receptor site is limited. Sugammadex, a selective relaxant binding agent has been designed to reverse the effects of rocuronium. The mechanism of action of this novel drug is through the encapsulation of rocuronium molecule, resulting in a rapid decrease in free rocuronium in the plasma, which results in a rapid migration of rocuronium away from the synaptic cleft.[30] Had this drug been available, we could have avoided prolonged mechanical ventilation and admission at the ICU.

A suggested alternative includes continuous management of the airway and breathing until the relaxants are metabolized or excreted. However, this approach may alarm patients and families if proper informed consent is not secured. Therefore, it may be prudent to advise patients treated with magnesium sulfate, and their family of the possibility of delayed extubation and possibility of being admitted to the ICU after general anesthesia when the decision for cesarean section is made, whether it is elective or urgent.[28]

CONCLUSION

Hypertensive disorders are still one of the most common medical complications encountered in pregnancy. Their occurrence carries risk not only to the pregnant woman but also to the fetus. Magnesium sulfate has been one of the revolutionary medications in the field of obstetrics, and its use in the management of hypertensive disorders in pregnancy improved the maternal and neonatal outcomes.

There has been a consensus among anesthesiologists that regional anesthesia is the type of anesthesia preferred during delivery of patients complicated with preeclampsia/eclampsia, may it be via vaginal or abdominal route. However, there are instances when a choice has to be

made to perform a cesarean section under general anesthesia even among patients with hypermagnesemia.

Magnesium interacts with non-depolarizing muscle relaxants such as rocuronium, and prolongs its relaxant effects. This relationship between these substances should be realized from the time a decision to use magnesium sulfate is made, so that obstetricians may give proper patient counseling regarding the need for prolonged intubation and the possibility of ICU admission, should a cesarean section be performed under general anesthesia. Anticipation of this drug interaction would also allow the anesthesiologists to plan his actions by either reducing the dose of muscle relaxant used prior to intubation, or preparing medications for immediate decurarisation.

Realization of this drug interaction should also make obstetricians, anesthesiologists, even the nurses more vigilant during the post-operative period, since the prolonged relaxant effect may not immediately manifest. ■

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