Magnesium sulfate poisoning treated with hemodialysis: a case report

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INTRODUCTION

Magnesium (Mg) is the fourth most plentiful mineral in the body, having the role of cofactor for enzymatic reactions (1-3). The normal range of Mg concertation of serum in humans is 1.8-2.3 (4). Magnesium sulfate is the drug of choice for the prevention and treatment, and recurrence management of convulsions in eclampsia and pre-eclampsia and it is an FDA-approved indication in hypomagnesemia management (5-7). Considering the elevated use of Mg in the management of different conditions, the safety of therapy needs more research. However, electrocardiographic (ECG) and electrophysiological outcomes of intravenous (IV) Mg injection have been assessed in animal research (8). We report a case of Mg Sulfate toxicity managed with hemodialysis, with clinical improvement and hospital discharge.

CASE REPORT

Case history

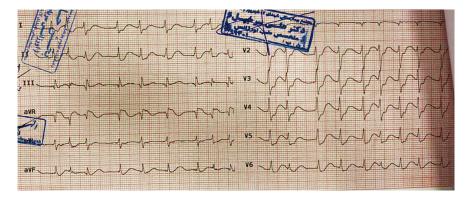
A 50-year-old married woman after a family struggle was referred to Shaheed Moddares Hospital in Save. Following the struggle, the patient got extremely agitated and the emergency medical service (EMS) reached and checked her blood sugar (BS) by finger and her BS was 65mg/dl, so the EMS injected two vials of 50ml Mg Sulfate 50% (25-gram of Mg sulfate each) instead of Dextrose 50% to the patient by mistake. Immediately after the injection, the patient got flushed and then she lost consciousness, so the EMS took her to the emergency ward of Shaheed Moddares Hospital. At the entrance to the emergency ward before any assessment, she became pulseless, and cardiac pulmonary resuscitation (CPR) was initiated for 10 minutes and was intubated, and her pulse came back, and then supportive care and diagnostic processes like hydration, ECG, and laboratory tests initiated. The patient's companion mentioned no past medical history or kidney disease and no drug or supplement for usual consumption. On examination, the skin was flushed, no sweating, pupils were symmetric and dilated, heart, lung, and intestine sounds were normal, and the deep tendon reflexes (DTR) were hypoactive. Her blood pressure was 100/70 mmHg and her pulse rate was 110 beats per minute.

The primary laboratory test result was BS 484mg/dl, serum sodium 128 mEq/L, serum potassium 2.2 mEq/L, calcium 9.1mg/dl, phosphorus 3.2mg/dl, magnesium 4.9mEq/L Urea 38 mg/dl, Creatinine 1.33 mg/dl, Aspartate transaminase (AST) 85units/liter, Alanine transaminase (ALT) 107units/liter, Lactate dehydrogenase (LDH) 528units/liter, Troponin qualitative negative, CK-MB 21 IU/L, venous blood gas was PH: 7.14 pco2: 46.2 mmHg hco3: 15.4 mmHg.

Differential diagnosis

Internal medicine and cardiology consultation were requested in the emergency ward considering the laboratory result and ECG, which were hypermagnesemia, hypokalemia, and hyponatremia. the ECG rhythm was irregular (160 bpm), the P wave was flattered, ST-depression in V2-V6, I, and II, and ST-elevation in aVR was obvious (image 1).



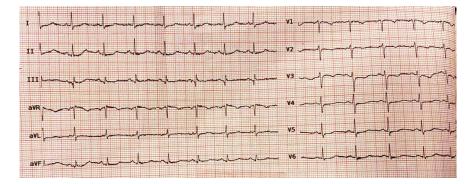


We considered acute coronary syndrome (ACS) as one of the potential causes of ECG issues such as ST changes. However, as the patient did not experience any chest pain or dyspnea and had no prior history of cardiac symptoms or risk factors for ACS, we instead suspected hypermagnesemia as the underlying cause for the ECG changes. Additionally, the patient's family history did not indicate any coronary cardiac problems.

Treatment

The patient had no clinical changes, her Glasgow Coma scale (GCS) score was 4/15. After receiving supportive care with intravenous isotonic fluids and calcium gluconate to prevent cardiac complications, it was decided to do hemodialysis for the patient. The decision to undergo hemodialysis was based on Micromedex toxicology reference, which recommends hemodialysis for severe magnesium toxicity. Then, after 4 hours of hemodialysis, serum Mg concentration became 3.1 mEq/L, calcium 6 mg/dl, and phosphorus 0.6 mg/dl, and her consciousness became better with a GCS score of 7/15. The patient's condition significantly improved, as evidenced by clinical observations, laboratory tests, and ECG, after spending 24 hours in the intensive care unit (ICU) in intubated condition. The day after, the patient was extubated, and she had normal respiratory rate and O2 saturation (image 2) (table 1).

Image 2 – ECG after hemodialysis



Two doses of 10ml of calcium gluconate 10% three times a day due to her hypocalcemia, tab phosphate Sandoz 500mg three times a day due to her hypophosphatemia, heparin 5000-unit ampules twice a day,

ampule pantoprazole 40 mg daily, and serum normal saline 0.9% 2 liters daily were given during ICU admission. After 4 days of admission, she was discharged with a normal laboratory and clinical situation.

Table 1- laboratory result

Time after							
admission/ Lab test U	pon arrival	7 hour	9 hour	15 hour	21 hour	24 hour	48 hour
PH 7.	14	7.21		7.34	7.36		7.42
PCO2 46	5.2	54.3		35.2	42.6		33.2
(mmHg)							
HCO3 15	5.4	21.2		18.7	23.5		22.5
(mmHg)							
Blood 48	84	122	111				
sugar(mg/dl)							
UREA 38	8		21	8	37		43
(mg/dl)							
Creatinine(mg/dl	.33		1.04	1.01	0.87		0.93
Magnesium(mE4,	/9L)		3.1	2.4	2.7	2.5	2
Calcium(mg/dl)9.	1	7.5	9	6.7	8.6	7.9	7.9
Phosphorus(mg30	担)		0.6	2	2.2	2.1	2.1
Sodium(mEq/L)2	28	137	141	142	140	142	138
Potassium(mEq2	(2)	3	4.6	3.1	4.6	4.4	4
White		28.1				15.6	10.5
blood							
$\operatorname{count}(103/\mathrm{mm}^3)$							
Hemoglobin(g/dl)		12.5				8.9	8.2
$Platelet(10^3/mm^3)$		166				131	110
AST 85	5						
(SGOT)							
(U/L)							
ALT 10)7						
(SGPT)							
(U/L)							
Alkaline 14	42						
phos-							
phatase							
(U/L)							
Lactate 52							
dehydrogenase(U							
Creatine 80)						
kinase(U/L)	_						
Ck-mb 28	3						
(U/L)							
Troponin(Qualita	0)						
Uric acid 6.	7						
(mg/dl)							

DISCUSSION

Magnesium sulfate is frequently utilized to treat eclampsia and pre-eclampsia during pregnancy as an anticon-

vulsant [9]. Magnesium sulfate extensively influences heart, lung, and brain function [10-12]. Predominantly hypermagnesemia occurs when renal function decreases or a large quantity of magnesium is loaded [13,14]. Clinical presentation of hypermagnesemia is associated with the serum magnesium concentration. Nausea, vomiting, bradycardia, and hypotension take place at the magnesium serum level of 4-7 mEq/L, moreover, loss of deep tendon reflexes and increased QT interval duration take place at the serum level of 8-10 mEq/L. Also, comatose, muscle paralysis, complete AV block, and cardiac arrest take place at serum levels greater than 12 mEq/L [15,16].

The management of patients with hypermagnesemia would be needed to eliminate magnesium through renal excretion by high-volume normal saline infusion and loop diuretic consumption, Because of the specificity of the loop diuretics which inhibits tubular reabsorption of magnesium in the thick ascending part of Henle's loop. For patients with impaired kidneys or who have the clinical symptoms of hypermagnesemia, hemodialysis should be considered. In patients with symptoms of hypermagnesemia, it should be managed with calcium to prevent the neuromuscular and cardiovascular adverse effects of hypermagnesemia [17].

a large number of cases of hypermagnesemia were reported in Japan due to magnesium oxide (MgO) prescription as a laxative in elderly patients with constipation, most of whom had chronic kidney disease (CKD). In addition, some of the cases had dementia or cerebrovascular events and couldn't express their symptoms, and the magnesium serum concentration was not examined. All of the cases were treated with fluid infusion (normal saline) and diuretics except one case that was managed by continuous hemodiafiltration (CHDF) and after 4 days died at the hospital [18-23].

In Akbar MIA *et al's* study, they reported 19 Mg intoxication patients in preeclampsia with severe features in women treated with magnesium sulfate and it was significantly associated with prenatal death and low Apgar score at 1 and 5 minutes. All of the Mg intoxications were treated with calcium gluconate immediately in line with Indonesian national protocol. 3 patients died, whereas, it was not due to hypermagnesemia events [24].

Another case was reported in 2021, a 34-year-old man reached the emergency ward after he was found unresponsive in a restaurant, and an empty bottle of magnesium supplement and ibuprofen was with him. He was hypotensive and hypothermic. His serum magnesium concentration was 11.7 mEq/L. he was admitted to the intensive care unit and intubated and intravenous calcium was initiated. Continuous renal replacement therapy (CRRT) was started for him and serum magnesium level lowered. His complications in the hospital were extensive. despite various vasopressors utilized, he was in shock. Abdominal compartment syndrome needed for bedside laparotomy, aspiration pneumonia, acute respiratory distress, and disseminated intravascular coagulation (DIC) led to his family's decision to transmit him to comfort care, and he died on the 4th day [25].

Our case was an iatrogenic and EMS mistake magnesium sulfate overdose which was performed continuously for 4 hours of hemodialysis. calcium gluconate was administrated to protect against cardiac complications. After hemodialysis, the patients recovered and then extubated. during treatment in the hospital, she showed hypocalcemia, hypophosphatemia, and hypokalemia which were managed appropriately.

CONCLUSION

Mg serum concentration is not measured routinely in every country so most patients come to the emergency ward due to their clinical symptoms. Magnesium toxicity is an uncommon but life-threatening issue that exhibits itself by clinical symptoms and changing consciousness levels. Utilizing intensive procedures like hemodialysis can be effective and save the patient's life.

ETHICAL STATEMENT

This study was conducted by the Declaration of Helsinki of the World Medical Association for Experiments involving Humans.

CONFLICT OF INTEREST

The authors declare that they have no competing interests.

FUNDING INFORMATION

No funds, grants, or other support were received from any organization.

CONSENT

Written informed consent was obtained from the patient to publish this report by the journal's patient consent policy.

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AUTHOR CONTRIBUTIONS

Mahdie Miri: Data curation, Project administration.Farhad Esmailsorkh: Writing – original draft, Conceptualization, Investigation. Ehsan Farhadi: Data curation, Project administration. Mitra Rahimi: Supervision, Writing – review & editing, Validation.

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