CASE REPORT OPEN ACCESS

Massive and sustain potassium therapy saves life in barium chloride intoxication: A case report

Mohy Kadri El Masry, Walaa Gomaa Abdelhamid, Salma Ibrahim Abdelkader, Sara Ahmad Elmorsi, Sara Atef Abdelaziz

ABSTRACT

Introduction: Barium salts present real threat to patients if the radiocontrast material contains soluble barium contaminant as the chloride form which is one of the most soluble salts. Case Report: A patient 37-year-old was given 40 g barium chloride as radiocontrast for gastro-esophageal reflux imaging. It was immediately followed with severe vomiting and hematemesis. Shock, ventricular tachycardia, severe flaccid paralysis, coma and severe hypokalemia followed within the first hour. The patient responded favorably to large infusion of IV potassium, reaching 40 mmol hourly for the first three hours. Potassium infusion was continued for the first four days of treatment. A total IV potassium infusion of 560 mmol was given in the first day. Abundant IV fluids infusion contributed to the correction of severe dehydration caused by vomiting and diarrhea. Prerenal failure and ischemic hepatitis secondary to shock improved with hemodynamic correction. Conclusion: Following barium poisoning, severe hypokalemia (1.2 mmol/L in this case) is responsible for ventricular tachycardia, shock, flaccid paralysis and respiratory failure. Large IV potassium infusion should continue for the first few days and is considered the principal therapeutic guideline in barium poisoning and the mainstay for the correction of almost all vital functions.

Keywords: Barium poisoning, Hypokalemia, Ventricular tachycardia, Paralytic respiratory failure

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Mohy Kadri El Masry[1], Walaa Gomaa Abdelhamid[2], Salma Ibrahim Abdelkader[2], Sara Ahmad Elmorsi[3], Sara Atef Abdelaziz[3]

Affiliations: [1]Professor, Poison Control Center–Forensic Medicine and Clinical Toxicology Department, Faculty of Medicine, Ain Shams University, Cairo, Egypt; [2]Assistant Lecturer, Poison Control Center– Forensic Medicine and Clinical Toxicology Department, Faculty of Medicine, Ain Shams University, Cairo, Egypt; [3]Resident, Poison Control Center–Forensic Medicine and Clinical Toxicology Department, Faculty of Medicine, Ain Shams University, Cairo, Egypt.

Corresponding Author: Mohy Kadri El Masry, Department of Forensic Medicine and Clinical Toxicology, Poison Control Center Cairo, Egypt - 11591; Ph: 00201223420302; 0020226836674; Email: mohymasry@yahoo.com

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INTRODUCTION

Insoluble barium sulfate is used in its pure form for the insolubility, as an X-ray radiocontrast agent for imaging the human gastrointestinal tract. Rare cases of unintentional toxicity have been reported during radiographic procedures and include complications associated with oral administration [1] of soluble barium salts unintentionally contaminating contrast solution [2]. Soluble barium compounds like acetate, chloride, hydroxide, nitrate are poisonous due to release of the soluble barium ion, and have been used as rodenticides. The solubility of barium chloride is high and typically produces a rapid onset of life threatening

manifestations. In addition to good supportive care, the mainstay of treatment is rapid correction of hypokalemia [3].

CASE REPORT

A 37-year-old male was transferred by ambulance from a district hospital following uncompleted barium meal radioimaging procedure, during which the patient developed upper abdominal pain, recurrent vomiting, hematemesis and profuse sweating followed by hypotension, tachycardia and drowsiness. Present history revealed that the patient was requested to supply the barium for the imaging as the hospital was short of the chemical. Radiology technician realized that he gave the patient a suspension containing the equivalent of 40 g barium chloride instead of barium sulfate when he revised the container. Serum potassium was 2.2 mmol/L one hour after the accident. He was immediately given 5 g oral magnesium sulfate, 40 mmol KCl in IV fluids, 200 mg hydrocortisone and metoclopramide.

On arrival to poison control center (PCC), as evident in Table 1 he was in deep flaccid coma grade IV (Reeds classification); he had occasional fasciculations of small muscles of hands and face. Pupils were dilated and sluggishly reacting. He was in shock with systolic blood pressure of 50 mmHg, diastolic blood pressure of 30 mmHg, and barely palpable peripheral pulsations. ECG showed ventricular tachycardia.

Table 1: Progressive clinical and laboratory notes in response to advocated management

	1-2 h	**PCC 3rd h**	**4th h**	**6th h**	**8th h**	**12th h**	**24th h**	**Day 2**	**Day 3**	**Day 4**	**Day 5**
Blood Pressure (mmHg)		50/20	90/60	150/100		130/80		120/80			
ECG		VT - VF		ST- PVCs		VT		PVCs		Sinus	
Vomiting (mL)	700	300			200	200		200	50		
Diarrhea (mL)		800					500	500			
Coma (Grade)		III/IV		III			I	0			
Muscle Power		0			1		3	4	5		
PO2 mmHg		31.1	69.3					227.5	159	80.9	137.7
PCO2 mmHg		58.3	42					38.7	36.5	38	43.9
PH		7.29	7.26					7.31	7.43	7.48	7.46
SaO2		51	91.3					99.6	99.5	96.4	99.0
Serum potassium (mmol/L)	2.2	1.2	2.4		2.5	1.8	4.0	3.7	3.6	3.6	3.6
Hematocrit		55.7						54.5			51%
WBCs x (10^3/mm^3)		26.6						23.5			11
SGPT (IU/L)			37					105		97	73
SGOT (IU/L)			42					148		161	109
Urea (mg/dL			83					65	47	41	
Creatinin (mg/dL)			1.8					1.6	1.3	1.1	
Gastric lavage		+			+						
Dose KCl (mmol)	40	40	40	40	40	120	240	200	120	80	
Antiarrhythmic		Lidocaine 100 mg bolus followed by 2 mg/min			Amiodarone 150 mg IV over 20 min, then 1 mg/min (6 h) then 0.5 mg/min						
MgSO4 (gm)	5 gm	15			15						
Dopamine			5 µ/kg/min		Weaned						
IV Fluids (ml)		1500	1000	1000		6450		3400	2250	2750	
Input (+)/ Output (-)			+2150			+2950		+900	-750	- 500	
Mechanical Ventilation				Controlled				SIMV	Wean T tube	Extubat i-on	

Abbreviations: h - hour, VT - Ventricular tachycardia, VF - Ventricular fibrillations, PVC - Premature ventricular contractions, SIMV: Synchronized intermittent mandatory ventilation

Immediate cardiopulmonary resuscitaion (CPR), endotracheal intubation and respiratory support were undertaken. Initial assessment revealed respiratory failure with PaO2 31.1 mmHg, PaCO2 58.3 mmHg; SaO2 51% and serum K 1.2 mmol/L. Intravenous fluids with large doses of potassium were immediately infused with a high potassium rate reaching 40 mmol hourly in the first 3 hours and 20–30 mmol/L hourly thereafter for the first day guided by serum potassium levels. Gastric lavage with abundant tap water was performed followed by instillation of 10 g magnesium sulfate. This was repeated after three hours. Blood pressure was rapidly corrected to 90/60 mmHg in response to large IV fluid administration, and antiarrhythmic therapy.

A gradual regain of consciousness and muscle power of hands and arms was noticed after the first 12 hours of treatment and spontaneous breathing was detected during mechanical ventilation. Lidocaine used initially was discontinued and followed by amiodarone. Both were given in recommended doses. Abundant IV fluids were followed by the correction of hemodynamic status. Dopamine started initially was rapidly weaned.

Serum potassium showed fluctuations between 1.8 and 2.5 mmol/L during the first 24 hours despite large potassium infusion rates. Patient receiving 560 mmol potassium on the first day. On the second day, 200 mmol potassium, and almost 120 mmol daily thereafter were required to keep serum potassium levels within low normal values. Starting from the second day serum potassium varied between 3.6 and 4.0 mmol/L.

Patient was initially on controlled mode mechanical ventilation and was shifted to SIMV mode on day 3, weaned and extubated on day 4.

Hematemesis and vomiting continued for four days despite high doses of parenteral omeprazole, vitamin K, tranexamic acid and cold water gastric lavage.

On the first day, hematocrit was 55.7%, total proteins 9 g/dL and total leucocytic count $26x10^3/mm^3$. A mild rise of urea and creatinine was evident on the first day and decreased slightly on the second day. CPK was normal. Liver transaminases were normal on admission but rose to 105 IU/L for SGPT and 148 IU/L for SGOP to gradually decrease daily thereafter. Neurological examination was normal before discharge on day 5.

DISCUSSION

Life threatening hypokalemia is uncommon in clinical toxicology practice. Moderate to severe hypokalemia are common after deliberate insulin overdose and theophylline toxicity. In barium toxicity, potassium redistribution and sequestration occur dramatically and rapidly inside the cells. Barium reduces potassium efflux from muscle cells by blocking potassium channels. Therefore, the continued activity of the ion pump combined with blocked potassium efflux results in extracellular hypokalemia and intracellular potassium accumulation. The serum potassium level can drop quite precipitously, and so does the trans-membrane ionic diffusion potential, to less than 60 mV. At this membrane potential the muscle is unexcitable and paralysis ensues [4].

The precipitous hypokalemia occurring in our case, reaching 1.2 mmol/L, was evidently responsible for the appearance of paralytic respiratory failure, ventricular tachycardia and episodes of ventricular fibrillation and eventually shock.

Despite the rapid CPR, lidocaine infusion and mechanical ventilation, it was evident that the concomitant intravenous infusion of large doses of potassium chloride reaching 120 mmol in the first three hours, as a combination treatment, was responsible for the correction of rhythm disturbances and shock state. This was previously confirmed and recommended [5, 6]. Despite the high rate of potassium infusion, we estimate the rate of potassium infusion was slower than the ideal. Under lidocaine infusion and later amiodarone recommended dosages treatment, recurrence of non-sustained ventricular tachycardia and premature ventricular ectopy, was strongly correlated with slowing rate of potassium infusion to 20 mmol/h and relapsing life-threatening hypokalemia (1.8 mmol/L) necessitating the resumption of massive infusion of potassium greater than the dosage recommended by some authors although encouraged by others [5, 7]. This demonstrates that blocking barium effect on the potassium rectified channels and block of potassium efflux continued till the 12^{th} hour after ingestion. With potassium infusion reaching 560 mmol in the first 24 hours, serum potassium still tended to decline after initial correction to 1.8 mmol/L

A non-negligible contributing effect to hypotension and shock is the dehydration provoked by vomiting and third space fluid entrapment in the digestive tract related to barium and magnesium sulfate. Large infusion rates with positive fluid balance (+6100 mL on the first day) in our case corrected the laboratory signs of dehydration evidenced by an elevated total proteins (90 g/L) and hematocrit (55.4%) which returned to 51% on discharge. This case highlights the importance of dehydration in barium poisoning in precipitation of shock.

Ischemic hepatitis secondary to shock was responsible for the mild rise of transaminases on day 2. Elevated serum transaminases gradually returned to normal after correction of blood pressure and control of shock. Severe blood tinged vomiting persisting till day 4 of poisoning confirming hemorrhagic gastritis and esophagitis for which H2 blockers and proton pump inhibitors were essential part of our treatment strategy.

Peripheral respiratory failure secondary to serious flaccid paralysis and ultimately respiratory muscle weakness in our case, necessitated immediate and prolonged control mode mechanical ventilation. The gradual regain of muscle power by the end of the first day closely coincided with the correction of severe hypokalemia, and permitted the initiation of SIMV mode ventilation. This observation indicated the correlation of profound muscle weakness to hypokalemia rather to the barium itself.

This case represents the largest barium chloride dose (40 g) required to survive poisoning and highest IV

potassium requirements (560 mmol in the first 24 hours) so far reported in literature.

The case presents a serious form of medical malpractice related to administration of barium chloride, non-verified by the radiology technician before using for radiocontrast imaging. This life-threatening situation is caused by the lack of clear strategic guidelines for checking the safety of this procedure, deficient provision of drugs and contrast materials in hospitals and dependence on patients to purchase the materials and drugs required to accomplish the due care.

CONCLUSION

The critical communication conveyed by this case of acute barium poisoning, is the importance of early and massive potassium administration in doses even higher than recommended in literature to correct severe life-threatening recurrent ventricular arrhythmias, shock and paralytic respiratory failure. Even with toxic doses of soluble barium compounds higher than described in literature, survival is possible with early, aggressive and sustained proper management. Dehydration and ischemic hepatitis concomitant with shock, although rarely described, might complicate acute barium poisoning.

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Author Contributions

Mohy Kadri El Masry – Conception and design, Analysis and interpretation of data, Drafting the article, Revising it critically for important intellectual content, Final approval of the version to be published
Walaa Gomaa Abdelhamid – Acquisition of data, Revising it critically for important intellectual content, Final approval of the version to be published
Salma Ibrahim Abdelkader – Acquisition of data, Revising it critically for important intellectual content, Final approval of the version to be published
Sara Ahmad Elmorsi – Acquisition of data, Revising it critically for important intellectual content, Final approval of the version to be published
Sara Atef Abdelaziz – Acquisition of data, Revising it critically for important intellectual content, Final approval of the version to be published

Guarantor

The corresponding author is the guarantor of submission.

Conflict of Interest

Authors declare no conflict of interest.

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