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Rodenticide Poisoning Presenting as Acute Flaccid Quadriparesis and Cardiac Arrthymia

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Abstract

This case report describes a case of rat poisoning presenting as acute flaccid paralysis with cardiac arrhythmia. There are wide range of toxins used in rodenticides. It contains Thallium, Sodium monofluoroacetate, Strychnine, Zinc and aluminum phosphide, Arsenic, Barium carbonate and Superwarfarins. In this barium is known to cause hypokalemia, muscle paralysis, respiratory failure and cardiac arrthymia. Our case highlights the need for a high index of suspicion for barium carbonate poison with history of rodenticide ingestion presenting with these complains.

Keywords: Rodenticide poisoning, Barium carbonate, muscle paralysis, cardiac arrthymia

Introduction

Rodenticides or "rat poisons" are mixed compounds commonly used to eradicate rodents. Barium carbonate is a compound used in rodenticides. It is a white compound and can be fatal if consumed in large amount mainly by causing muscle paralysis and cardiac arrthymia. Treatment is supportive and symptomatic. Large amount of potassium supplementation and hemodialysis may be needed for treatment.

Case History

A 44-year-old male presented to emergency six hour after ingestion of Rat poison. At presentation he had complain of 6-7 episode of vomiting, not blood stained, associated with nausea and epigastric discomfort. There was no history of shortness of breath, bleeding from any site, diarrhoea, weakness in any part of body, seizure, or loss of consciousness. There was no history of hypertension, diabetes, and coronary artery disease. On examination, the patient was conscious, oriented, and hemodynamically stable. He had respiratory rate of 18/min, pulse rate of 84/min regular, blood pressure of 170/90 mmHg and SpO2 of 98%. Cardiovascular, respiratory, abdominal, and central nervous system examination were unremarkable.

Gastric lavage was done with normal saline. On investigation he had hypokalemia (K+ =2.2) and all other investigation were normal (Table 1). ECG showed normal sinus rhythm with flattening of T wave. CXR also didn't reveled any abnormality. A provisional diagnosis of Rat poisoning with hypokalemia was made. Patient was given potassium supplementation (200-300 meq/day), inj vitamin k anti hypertensives and intravenous fluids. However, 3-4 hour after presentation patient started complaining of generalized body weakness and shortness of breath. And within 1-2 hour there was complete loss of tone and power in all 4 limbs (power 0/5). All reflexes were absent, and planters were mute. Sensory system and cranial nerve examination were with in normal limit.

Due to decrease in SpO2- 85% on room air patient was intubated and was kept on mechanical ventilation. His potassium was persistently low despite continuous potassium supplementation. Serum magnesium was also with in normal limit (2.1 mg/dl). During admission patient developed ST depression and there was serial rise in CPK T/MB from 53/9 to 420/44 µ/L for which patient was loaded with anti-platelet drugs. On day-2 there was no improvement in power of limbs and respiratory effort of patient and patient was kept on mechanical ventilation. His serum

potassium although improved from beginning as shown in Table 1 but continued to be low despite adequate supplementation (200-300 meq/day). But patient suddenly developed ventricular tachycardia and went into cardiorespiratory arrest for which resuscitation was done however patient succumbed to his illness.

Table 1

PARAMETERES	1 HOUR	6 HOUR	12 HOUR	24 HOUR
HEMOGLOBIN (gm/dl)	15.2	14.9	15	14.4
TOTAL LEUKOCYTE COUNT (cell/mm^3)	13000	12590	11560	13600
DIFFERENTIAL LEUKOCYTE COUNT	76/22/1/1	74/22/3/1	78/19/2/1	75/22/3/1
PLATELET (lakhs/mm^3)	1.9	2.2	2	2.6
BLOOD UREA (mg/dl)	14	22	19	20
SERUM CREATININE (mg/dl)	0.8	0.9	1	0.8
SODIUM (meq/dl)	134	138	141	140
POTASSIUM (meq/dl)	2.2	1.9	2.4	2.6
CALCIUM (mg/dl)	8.6	8.3	8.3	8.1
PHOSPHORUS (mg/dl)	4.2	4.1	3.8	4
MAGNESIUM (mg/dl)	2.1	2.3	2.2	2.2
TOTAL BILIRUBIN (mg/dl)	1.2	1	1.1	1
AST (U/L)	23	27	22	33
ALT (U/L)	34	30	34	24
CPK T/MB (µ/L)	53/9	420/44	512/53	546/54
INR	0.8	0.9	1	0.9
APTT (seconds)	32	30	34	32

Discussion

Rodenticides or "rat poisons" are mixed compounds commonly used to eradicate rodents. We presented a case of acute quadriparesis with respiratory failure after rodenticide ingestion. There are wide range of toxins used in rodenticides. It contains Thallium, Sodium monofluoroacetate, Strychnine, Zinc and aluminum phosphide, Arsenic, Barium carbonate and Superwarfarins (Brodifacoum, difenacoum, bromadiolone, chlorophacinone). Clinical presentation varies and depends on the type of rodenticide used.

Thallium is an odorless and tasteless powder absorbed via skin or through inhalation. It causes acute gastroenteritis, seizure, peripheral neuropathy, tremor, ataxia and distal motor weakness. Fluoroacetamide is also an odorless and tasteless white powder, symptoms starts 30 min to 20 hours after ingestion. It causes seizure, hypocalcemia, metabolic acidosis, arrhythmia, shock and organ failure. Zinc and aluminium phosphide releases phosphine gas which has rotten fish odor. When ingested it get converted to phosphine gas and absorbed into blood stream. It causes gastroenteritis, cardiac arrhythmia, acute respiratory distress syndrome, intravascular hemolysis, respiratory failure, metabolic acidosis, hepatotoxicity and renal failure. Superwarfarins (brodifacoum, difenacoum, bromadiolone, and chlorophacinone) are anticoagulant rodenticides similar to warfarin. It causes hematuria, hemoptysis, epistaxis, flank pain, easy bruising, intracranial hemorrhage.

Barium carbonate are highly toxic compound which is white powder like, odorless and tasteless which easily dissolve in water. It causes gastroenteritis, cardiac arrhythmia, hypokalemia and muscle paralysis. Our patient had the classic feature of barium intoxication and abdominal pain, hypokalemia, muscle paralysis, and arrhythmia are attributed to the direct effect of barium [1]. Barium also causes hypertension which was present in our patient due to direct effect on the smooth muscle of vasculature [2]. There is a delay before symptoms appear because barium carbonate is transformed to barium chloride in the stomach by free hydrochloric acid before being absorbed from the small intestine. Barium causes shift of potassium from extracellular to intracellular compartment. This large shift of potassium into muscle blocks the Na/K ATPase pump causing depolarization and paralysis [3]. Barium causes myocardial hyperexcitability leading to fast heart rate, elevated blood pressure, pre-excitation, ventricular fibrillation and ventricular arrest [4].

Our patient experienced the typical side effects of barium poisoning, such as nausea, vomiting, abdominal discomfort, and diarrhoea, as well as areflexic flaccid quadriplegia brought on by hypokalemia. Treatment is supportive and symptomatic. Gastric lavage and enteral infusion of sodium sulphate or magnesium

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sulphate are two emergency treatments used to limit absorption by precipitating soluble barium ions into insoluble barium sulphate. To treat the hypokalemia, large doses of intravenous potassium (up to 200–400 mEq) might be required. Hemodialysis appears to be an effective treatment for barium poisoning as it appears to shorten the serum half-life of barium [5]. Prognosis is good, as symptoms usually resolve after 24 - 48 hours of potassium supplementation and supportive care [6]. In the present case the patient suddenly developed ventricular tachycardia probably due to barium and went into cardiorespiratory arrest in spite of giving adequate potassium supplementation.

Similar type of presentation was reported by Jamshidi et al. where a 38 year old female had history of ingestion of barium carbonate poisoning and hypokalemia at presentation and developed profound muscle weakness and cardiac arrthymia and was managed conservatively with potassium supplementation, endotracheal intubation, defibrillation and continuous veno-venous haemodialysis (CVVHD) but patient was fortunate to survive the event [7]. Tao et al also reported a similar case where a 19 year old male presented to emergency with complain of nausea, vomiting, abdominal pain and weakness following ingestion of barium chloride and patient had hypokalemia, cardiac arrthymia and was managed conservatively with potassium supplementation and had good recovery without any complication [8].

Conclusion

Barium carbonate is a rare poison should be suspected in patient presenting with acute flaccid paralysis, hypokalemia and cardiac arrhythmia. Prognosis is usually good but may be fatal in case of cardiac arrhythmia. Treatment is usually supportive and large amount of potassium and hemodialysis may be need for treatment.

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