"Alkaline Diuresis" is the Savior in 2,4-Dimethylamine Intoxication

Abstract

2,4-dimethyalmine is a herbicide commonly used by agriculturalist. Acute intoxication can cause fatal multiorgan damage. No antidote is available. We report a rarely encountered manifestation of this toxin, that is, spontaneous rhabdomyolysis, acute kidney injury and the effectiveness of alkaline diuresis as a life saving treatment in 2,4-dimethylamine intoxication.

Keywords: *2,4-dimethylamine, rhabdomyolysis, acute kidney injury, alkaline diuresis, herbicide*

Introduction

The use of herbicide 2,4-dimethylamine is common among agriculturalists. Acute intoxication is rare and often fatal, and it affects multiple organ systems. No antidote is available. Toxic effects may involve the central nervous system, heart, lungs, liver, kidney, muscles, and endocrine system. This herbicide exhibits a range of dose-dependent harmful mechanisms, including cell membrane destruction, disruption of acetyl coenzyme A metabolism, and oxidative phosphorylation uncoupling.[1,2]

This case reports the rarely encountered manifestation of this toxin, that is, spontaneous rhabdomyolysis, and acute kidney injury and the effectiveness of alkaline diuresis as a life-saving treatment in 2,4-dimethylamine intoxication.

Case Report

A 22-year-old male was admitted to our hospital with a history of consumption of 150 ml of herbicide (2,4-dimethylamine). He was taken to a nearby hospital, where he was intubated for altered consciousness and referred. He was admitted after 10 hours of consumption in our hospital. On arrival, he was on mechanical ventilation. His pulse rate was 110 beats per minute, and his blood pressure was 138/76 mmHg. The pupils were reacting to light.

His lab investigations [Table 1], showed mild leukocytosis. Serum creatinine was 2.2 mg/dl. His liver enzymes were deranged. He was given Ryle's tube lavage. CT brain was normal. He was started on isotonic saline and other supportive care. The urine output was 75-100 ml per hour. He was started on liver supportive measures (N-acetyl cysteine, antioxidants, Co-Q), and alkaline diuresis (150 mEq of sodium bicarbonate added to 1 L of 5% dextrose at the rate of 150 ml per hour) via a separate intravenous line apart from the isotonic saline to promote 2,4-dimethylamine excretion.

Serum creatinine phosphokinase (CPK) was very high (49,215 U/L) on day 2. Serum calcium was low, and serum phosphorus, uric acid, and LDH were elevated, suggestive of rhabdomyolysis. Ultrasound showed normal-sized kidneys. He was continued with adequate IV hydration and alkaline diuresis. The creatinine phosphokinase peaked at 93,182 U/L on day 4.

Table 1: Lab investigations during hospital stay

	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 10
Hb (g/dl)	14.9						
TLC (cells/cu.mm)	11,200						
Platelet count (cells/cu.mm)	4,62,000						
Urea (mg/dl)	83						
Serum creatinine (mg/dl)	2.24	2.02	1.61	1.02	0.79	0.5	0.5
Serum Na (mEq/L)	142	138	140	138	135		
Serum K (mEq/L)	3.1	3.4	3.7	3.8	4.0		
Serum calcium (mg/dl)		6.3	7.4	8.3			
Serum phosphorus (mg/dl)		6.4					
Serum uric acid		8.2					
Serum LDH (U/L)		1552					
Serum CPK (U/L)		49215	84517	3.8	45338	28361	4598
SGOT (U/L)		428			952	283	176
SGPT (U/L)		111			340	232	196
Total protein (gm/dl)		7.2					
Serum albumin (gm/dl)		4.6					
Alkaline phosphatase (U/L)		77					

Hb, hemoglobin; TLC, total leucocyte count; Na, serum sodium; K, serum potassium; LDH, lactate dehydrogenase; CPK, creatine phosphokinase; SGOT, serum glutamic-oxaloacetic transaminase; SGPT, serum glutamic-pyruvic transaminase

With the supportive measures, he showed clinical improvement. The urinary pH was maintained above 8. He gradually regained consciousness, and he was extubated on day 5. The creatinine settled at 0.5 mg/dl on day 6. There was a serial drop in CPK, and on day 10, CPK was 4598 U/L. The liver enzymes showed a declining trend.

A psychiatric opinion was obtained, and he was discharged in a stable state. He is doing well on follow-up.

Discussion

Until now, there has been no specific antidote for 2,4-dimethylamine intoxication. Being a weak acid, it gets excreted in the urine in the same form. Alkaline diuresis acts by increasing urine pH, thereby promoting renal excretion of 2,4-dimethylamine in alkaline urine.[3] For each unit increase in urine pH, the clearance of 2,4-dimethylamine by the kidney is estimated to increase nearly five-fold. A similar case report by Badu *et al*. has shown successful treatment of 2,4-dimethyl amine[4]. Hemodialysis is a treatment modality for this intoxication. However, our patient improved with alkaline diuresis. In addition to hemodialysis, there are case reports describing plasmapheresis, but there is limited evidence to support this.[4] Rhabdomyolysis due to 2,4-dimethylamine has not been reported in the literature so far. Administration of alkaline diuresis prevented acute kidney injury due to rhabdomyolysis in this case apart from eliminating 2,4-dimethyalmine via urine.

Conclusion

2,4-Dimethylamine intoxication is rare and has high morbidity and mortality. Timely management with alkaline diuresis can be life-saving, accompanied by other supportive therapies.

Declaration of patient consent

The authors declare that they have obtained all appropriate patient consent.

Conflicts of interest

There are no conflicts of interest.

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