

Aluminium phosphide induced acute kidney injury

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Aluminium phosphide is one of the most common agricultural poisons being consumed in north India. Consumption of a fresh tablet is lethal as no antidote is available. Acute intoxication primarily presents with cardiovascular collapse due to myocardial toxicity. We report here a case of acute severe poisoning along with cardiovascular collapse and oliguria. The patient developed acute kidney injury during the illness (a rare entity in aluminium phosphide poisoning), which completely resolved following prompt conservative treatment.

Keywords:

acute kidney injury, aluminium phosphide poisoning, oliguria

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Introduction

Poisoning oneself with aluminium phosphide is a common method of suicide in the agricultural community in northern India [1]. Pesticide poisoning is an important cause of morbidity and mortality in India. Aluminium phosphide (celphos) is emerging as a poison of suicidal deaths, as this pesticide is freely available in the market with no effective antidote [2]. The trade name of the fumigant is celphos, and it comes in the form of dark grey tablets of 3 g each, consisting of aluminium phosphide (56%) and aluminium carbamate (44%). Aluminium phosphide is highly toxic, of low cost and easily accessible.

Symptoms of celphos poisoning appear within the first few hours following exposure. Celphos poisoning is a real medical emergency demanding early and adequate management. Despite the progress achieved in the fields of toxicology, celphos poisoning is still responsible for a high rate of mortality as there is no specific antidote [3,4]. The objectives of this study were to emphasize the importance of rapid diagnosis and to prompt initiation of supportive treatment in patients with celphos poisoning, which could be life saving.

Case report

A previously well 32-year-old married man was presented to the Emergency Department 3 h after ingestion of a tablet of celphos with complaints of altered sensorium. On examination, he had cold clammy extremities, peripheral pulses were not palpable, blood pressure was not recordable, respiratory rate was 34/min, heart rate was 136/min, axillary temperature was 98.6°F, peripheral cyanosis was present and SpO₂ was 86%. Glasgow coma scale score of the patient was 9/15. Cardiac examination revealed tachycardia and normal first and second heart sounds. Chest examination

showed bilateral normal vesicular breathing with no added sounds. In view of hypotension, an urgent ECG was performed, which revealed sinus tachycardia and generalized ST-T changes. Arterial blood gas analysis revealed severe metabolic acidosis, with a pH of 7.11, PCO₂ of 23.4, PO₂ of 86.5, HCO₃ of 11.3 mEq/l and base excess of -14.2.

Prompt gastric lavage was performed with potassium permanganate (1 : 10 000) until the lavage fluid was negative for rotten fish smell (~8 h), and Foley's catheterization was performed. Resuscitation was initiated with intravenous crystalloids, noradrenaline at 1.0 mcg/kg/min, dopamine infusion at 5 mcg/kg/min and magnesium sulphate at 1 g intravenous stat, hydrocortisone at 200 mg intravenous stat, pantoprazole at 40 mg intravenous stat, 100 ml of soda bicarbonate (7.5%) as loading dose followed by 25 ml intravenous three times daily, and a third-generation cephalosporin.

Investigations revealed the following: haemoglobin, 10.3 g/dl; total leukocyte count, 9800/mm³; differential leukocyte count, N₆₁E₀₂L₃₄M₀₃B₀%; red blood cell, 3.73 ml/ μ l; haematocrit, 32.7%; mean corpuscular volume, 88.6 fl; mean corpuscular haemoglobin, 29.8 pg; mean corpuscular haemoglobin concentration, 33.7 g/dl; platelet count, 154/mm³; bleeding time, 1 min 45 s; clotting time, 4 min 10 s; activated partial thromboplastin time, 25.7 s; partial thromboplastin time, 25.3 s; prothrombin time (control), 12.4 s; prothrombin time (test), 14.4 s; international normalized ratio, 1.16; total bilirubin, 0.8 mg/dl (direct, 0.43 mg/dl; indirect, 0.37 mg/dl); total protein, 5.6 g/dl (albumin, 2.9 g/dl; globulin, 2.7 g/dl) with an albumin/globulin ratio of 1 : 1; aspartate aminotransferase, 24 U/l; alanine aminotransferase, 16 U/l; alkaline phosphatase, 43 U/l; lactate dehydrogenase, 21 U/l; blood urea, 68 mg/dl; serum creatinine, -2.2 mg/dl; Na⁺, 141 mmol/l; K⁺,

4.8 mmol/l; calcium, 8.9 mg/dl; magnesium, 2.1 mg/dl; creatine phosphokinase, 78 U/l; creatine kinase-muscle brain, 09 IU/l; and random blood sugar, 145 mg/dl.

Two hours later, dose of noradrenaline was increased to 2 mcg/kg/min, as his systolic blood pressure was still below 70 mmHg. After 6 h, his systolic blood pressure was noted to be 90 mmHg, and after 10 h the pulse was recorded to be 98/min, blood pressure was 100/60 mmHg and oxygen saturation was 94%. Magnesium sulphate 1 g in 100 ml normal saline was administered every hour for three consecutive hours and then 8 hourly, and hydrocortisone 200 mg was administered 6 hourly. Calcium gluconate (10%) 10 ml slow intravenous was administered 6 hourly for the first 48 h. Ceftriaxone 1 g was administered 12 hourly. During Magnesium sulphate therapy, urine output, deep tendon reflexes and respiratory effort were monitored closely. The tapering of dose of ionotropes was started after haemodynamic stabilization and was discontinued 24 h after admission. His vitals, blood investigations and serial ECGs were duly monitored in ICU for the next 2 days. Initially, the urine output was 400 ml/24 h with serum creatinine level of 6.1 mg/dl. After 2 days it was 800 ml/24 h, and in the next 3 days it increased to 1500 ml/24 h. Meanwhile, the serum creatinine level decreased to 2.2 mg/dl during the following 3 days. Ultrasound examination revealed bilateral normal renal echotexture with no evidence of renal parenchymal disease. Two-dimensional echocardiography did not reveal any evidence of regional wall motion abnormality, low ejection fraction or pericardial fluid. Radiograph of the chest was normal. The patient continued to improve physiologically and biochemically over the next 5 days and was discharged in a stable condition after 7 days of hospital stay.

Discussion

Celphos is a highly toxic, low cost and freely available pesticide. Fatal dose for a 70 kg man is 0.5 g [5]. On exposure to moisture, it liberates phosphine gas, which is absorbed rapidly through inhalational, dermal, or gastrointestinal route. The gaseous nature of phosphine has potential for contamination of the emergency service personnel exposed to victims [6], and when celphos tablets are swallowed, contamination of body tissues results [7]. Toxicity of phosphine is related to oxidant free radicals and associated inhibition of enzymes of metabolism, such as cytochrome c oxidase [8]. The average time interval between poisoning and death is 3 h, with a range of 1–48 h. As many as 95% of fatalities occur within the first 24 h. Common modes of death are ventricular fibrillation, acute respiratory distress

syndrome, hepatic failure, acidosis and electrolyte imbalance. Metabolic acidosis resulted, probably due to lactic acidosis, which was caused by the blocking of oxidation phosphorylation, which is similar to the effect of cyanide [9].

Toxic myocarditis produces extreme haemodynamic effects manifesting as myocardial pump failure, ischaemia and arrhythmias, causing severe hypotension and pulmonary oedema. Circulatory collapse, arrhythmias, conduction defects and peripheral vascular leakage make hypotension resistant to fluid therapy and inotropes, and is the major cause of death initially [10].

Widespread capillary damage leads to bleeding diathesis, disseminated intravascular coagulation (DIC) and acute tubular necrosis. Shock and DIC lead to terminal renal failure [11].

ECG changes seen in celphos poisoning cases include spectrum of atrial fibrillation, supraventricular tachycardia, premature ventricular contractions and ST-T changes. Of these, the ST-T changes with T wave inversion are by far the most common (which were seen in this patient). These changes are attributed to focal myocardial necrosis and changes in action membrane potential as a result of the alteration in the permeability of Na⁺, Mg⁺⁺ and Ca⁺⁺ ions [12].

Organ toxicity is reversed with magnesium sulphate, as it has antioxidant, antiarrhythmic and antihypoxic properties [13].

This case has been reported for the following reasons:

- (1) Survival after 'unexposed' celphos poisoning is very rare.
- (2) Our case developed acute kidney injury early without evidence of DIC, but we successfully managed the case conservatively.

Conclusion

Aluminium phosphide has no specific antidote; thus, favourable outcome correlates best with the severity of vomiting and the promptness of the initiation of treatment after toxicity. In conclusion, the main guiding principles of management are early aggressive lavage with potassium permanganate and treatment of hypotension and shock. Other appropriate supportive measures, which are tailored to the requirements of the patient, complete the management of celphos poisoning.

Acknowledgements

Conflicts of interest

There are no conflicts of interest.

References

- 1 Siwach SB, Yadav DR, Arora B, Dalal S, Jagdish. Acute aluminum phosphide poisoning – an epidemiological, clinical and histo-pathological study. *J Assoc Physicians India* 1988; 36:594–596.
- 2 Singh D, Dewan I, Pandey AN, Tyagi S. Spectrum of unnatural fatalities in the Chandigarh zone of north-west India – a 25 year autopsy study from a tertiary care hospital. *J Clin Forensic Med* 2003; 10:145–152.
- 3 Hajouji Idrissi M, Oualili L, Abidi K, Abouqal R, Kerkeb O, Zeggwagh AA. Severity factors of aluminium phosphide poisoning (Phostoxin). *Ann Fr Anesth Reanim* 2006; 25:382–385.
- 4 Bajwa SJS, Kaur SKB, Kaur J, Singh K, Panda A. Management of celphos poisoning with a novel intervention: a ray of hope in the darkest of clouds. *Anaesth Essays Res* 2010; 4:20–24.
- 5 Wahab A, Zaheer MS, Wahab S, Khan RA. Acute aluminium phosphide poisoning: an update. *Hong Kong J Emerg Med* 2008; 3:152–155.
- 6 Bogle RG, Theron P, Brooks P, Dargan PI, Redhead J. Aluminium phosphide poisoning. *Emerg Med J* 2006; 23:e3.
- 7 Nocera A, Levitin HW, Hilton JM. Dangerous bodies: a case of fatal aluminium phosphide poisoning. *Med J Aust* 2000; 173:133–135.
- 8 Chefurka W, Kashi KP, Bond EJ. The effect of phosphine on electron transport in mitochondria. *Pestic Biochem Physiol* 1976; 6:65–84.
- 9 Graham DL, Laman D, Theodore J, Robin ED. Acute cyanide poisoning complicated by lactic acidosis and pulmonary edema. *Arch Intern Med* 1977; 137:1051–1055.
- 10 Singh S, Singh D, Wig N, *et al.* Aluminum phosphide ingestion: a clinico-pathologic study. *J Toxicol Clin Toxicol* 1996; 34:703–706.
- 11 Gupta MS, Mehta L, Chugh SN, Malhotra KC. Aluminium phosphide poisoning. Two cases with rare presentation. *J Assoc Physicians India* 1990; 38:509–510.
- 12 Jain SM, Bharani A, Sepha GC. Electrocardiographic changes in aluminium phosphide poisoning. *J Assoc Physc India* 1985; 33:406–409.
- 13 Chugh SN, Kumar P, Aggarwal HK, Sharma A, Mahajan SK, Malhotra KC. Efficacy of magnesium sulphate in aluminium phosphide poisoning – comparison of two different dose schedules. *J Assoc Physicians India* 1994; 42:373–375.