Case Report

Acute aluminium phosphide poisoning: A case report of rare survival with cardiac, metabolic, hepatic, and renal complications

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Abstract

Poisoning is a very common way to commit suicide. It is more common in low- and middle-income countries. Aluminium phosphide is a very commonly available pesticide in such countries including India. Aluminium phosphide is a very toxic compound. Ingestion of aluminium phosphide can cause severe toxicity to various cells, and mortality is very high. We are presenting a case of rare survival of acute aluminium phosphide poisoning, who presented with signs and symptoms of severe toxicity including metabolic acidosis and shock. During hospitalisation, he developed ventricular tachycardia, acute kidney, and liver failure.

Keywords: Aluminium phosphide poisoning, cardiotoxic poisoning, celphos poisoning, pesticide

Introduction

The World Health Organisation estimated that more than 7,00,000 people die every year because of suicide. 77% of all suicides occur in low- and middle-income countries.[1] Pesticide ingestion is a common means of suicide. The commonly available pesticides are organophosphates, organochlorine, and aluminium phosphide (AlP). AlP, a dark-grey-coloured tablet, has brand names of Celphos, Alphos, Phostoxin, Quickphos, Phosfume, and Synfume.[2-4] AlP is a highly toxic agent; on exposure to moisture, it liberates phosphine (PH_3) gas.[5-7]

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PH_3 gas is colourless, odourless gas; on exposure to air, it produces garlicky odour. PH_3 is rapidly absorbed by the lungs or gut, which causes systemic toxic effects, which manifests as cardiac arrhythmias, shock, metabolic acidosis, and pulmonary oedema by free-radical injury and inhibiting cytochrome C oxidase enzyme. Early signs of toxicity are manifested by shock and circulatory failure. Acute AlP poisoning is very common amongst all acute poisoning in India (68%), and mortality is also very high (60%).[4-8] Mortality within 24 hours is because of cardiac arrhythmias, whereas after 24 hours, it is because of refractory shock, metabolic acidosis, acute respiratory distress syndrome (ARDS), and arrhythmias.[9]

We are presenting a case of rare survival of acute aluminium phosphide (AlP/Celphos) poisoning who presented with shock, cardiac arrythmia, and metabolic acidosis; later during hospital stay, he developed acute kidney and liver failure.

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Case Presentation

A 28-year-old male farmer presented with ingestion of two fresh tablets of Celphos (AlP) 4 hours prior to admission. He complained of vomiting with 4–5 episodes and pain in the abdomen. O/E shows that he was agitated, anxious, and irritable. The pulse rate (PR) was not palpable the blood pressure (BP) was not recordable, the heart rate (HR) was 110/minutes, the respiratory rate (RR) was 28/min, and the oxygen saturation (SPO_2) was 95%. Electrocardiogram (ECG) shows sinus tachycardia, T wave inversion in lead 3 [Figure 1]. He was managed with intravenous (IV) crystalloids, IV magnesium sulphate 1 gm stat and 8 hourly, IV calcium gluconate 8 hourly, IV hydrocortisone 100 mg stat and 12 hourly, IV dopamine, and noradrenaline. Immediate gastric lavage was performed with potassium permanganate (KMnO4), and activated charcoal was kept in the stomach, repeated 8 hourly. Routine investigations were normal at admission.

Arterial blood gas (ABG) analysis showed a metabolic acidosis pH of 7.2, HCO_3 of 8 mmol/L, and PCO_2 of 33 mmol/L; metabolic acidosis correction was performed as per the protocol. After starting first aid, he was intubated and shifted to an intensive care unit (ICU).

At the sixth hour, the BP was 60–80 mmHg (systolic), the PR was 120/min, and the RR was 20/min. The input/output was 3L/300 ml over the next 24 hours. On the second day, he developed monomorphic ventricular tachycardia (VT) [Figure 2], which was converted to normal sinus rhythm (NSR) by the DC cardio-version of synchronised 150 J [Figure 3]. IV amiodarone 150 mg bolus and infusion continued till the next 48 hours. Normal sinus rhythm was maintained till discharge. Cardiac bio-markers also raised on the second day, which normalised by the 14th day.

On the fourth day, ABG showed a pH of 7.4, HCO_3 of 18 mmol/L, and CO_2 of 42 mmol/L, and the BP was ranging from 70/50 mmHg to 90/60 mmHg till the fourth day. Blood urea was 100 mg/dl, serum creatinine was 4.5 mg/dl, the urine output was 400 ml/24 hours, aspartate amino-transferase/alanine amino-transferase (AST/ALT) was 80/90 IU/L, alkaline phosphatase (ALP) was 200 U/L, and S bilirubin was 2.5 mg/dl, suggestive of liver and kidney involvement [Figures 4 and 5].

On the fifth day, although the BP was 100/70 mmHg, the PR was 110/min, the RR was 22/min, the total leucocyte count (TLC) was 14000/mm^3 with polymorphonuclear leucocytosis, Urea was 200 mg/dl, S. creatinine was 7.5 mg/dl, the urine output was 400 ml/24 hours, ABG showed re-appearance of acidosis, the pH was 7.1, and HCO_3 was 10 mmol/L [Figure 6]. Haemodialysis was performed on the fifth day and seventh day. His blood urea after haemodialysis on the seventh day was 70 mg/dl, creatinine was 4.0 mg/dl, and the urine output improved to around 1200 ml/24 hours [Figure 7]. He was extubated on the sixth day and shifted to a step down unit (SDU) on the eighth day. Vitals were normal. Liver and kidney functions were improving and became near normal by the 14th day at discharge.

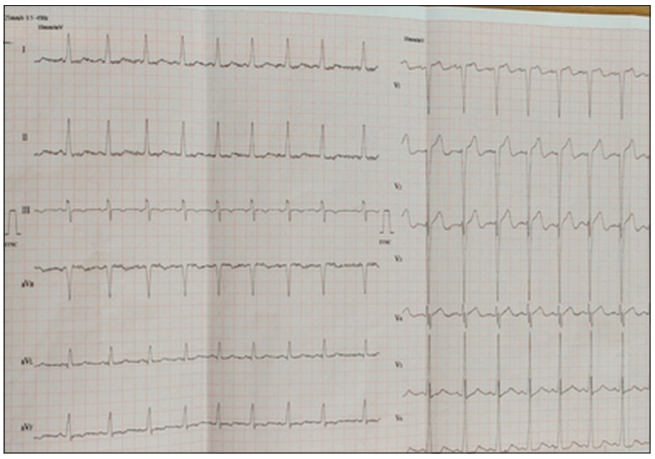

Figure 1: ECG recording at admission showing NSR, sinus tachycardia, and T-wave inverted in lead 3

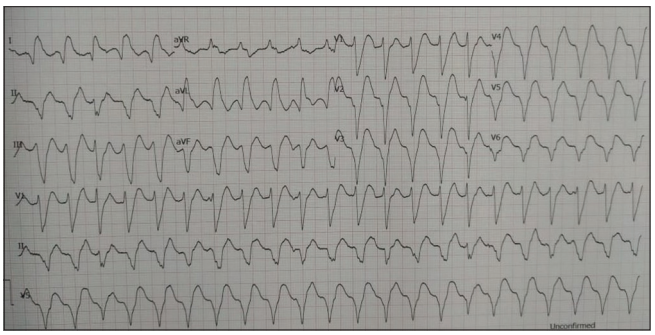

Figure 2: ECG recording on the second day showing monomorphic ventricular tachycardia (VT)

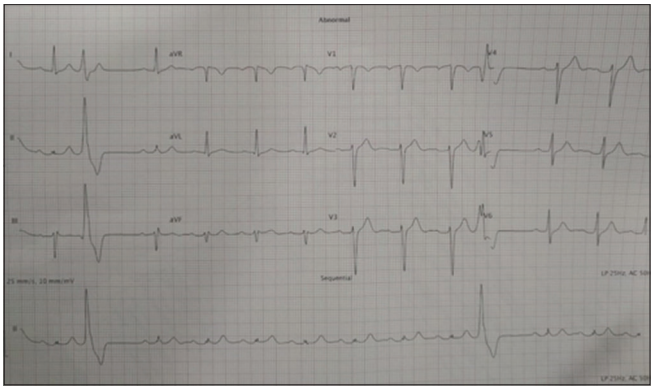

Figure 3: ECG recording normal sinus rhythm (NSR) with ventricular ectopic beat after the DC cardio-version

Discussion

The patient presented with early signs of severe toxicity with cardio-vascular shock and metabolic acidosis. Mortality of such patients is very high. The mortality ranges from 40 to 77%. One survey showed that a mortality of 55% occurred within

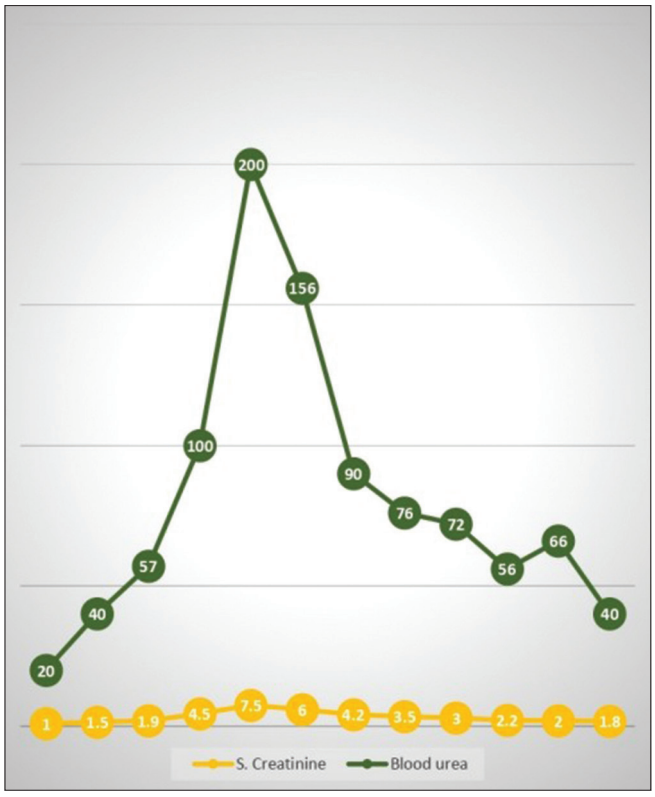

Figure 4: Renal function test (Day 1 to Day 14): Reference normal range: Serum creatinine = 0.74–1.35 mg/dl, blood urea = 5–20 mg/dl

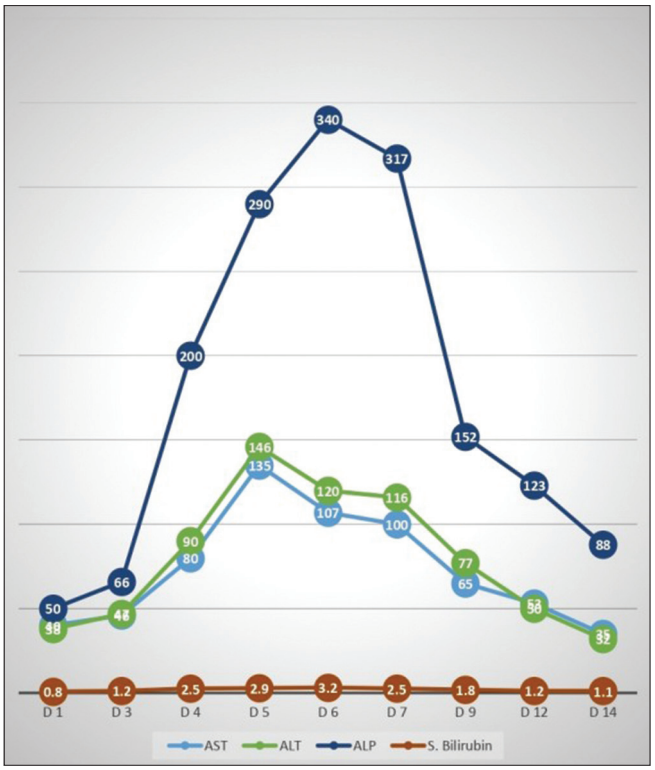

Figure 5: Liver function test charting (day 1 to day 14): Reference normal range: Aspartate amino-transferase (AST) = 10–40 IU/L, alanine amino-transferase (ALT) = 10–40 IU/L, alkaline phosphatase (ALP) = 40–112 U/L, serum bilirubin (total) = 0–0.8 mg/dl

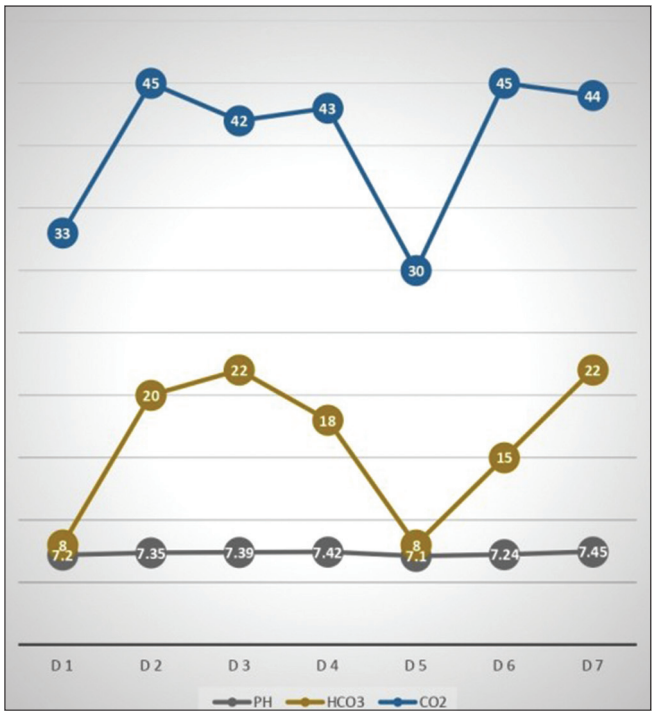

Figure 6: Arterial blood gas analysis report (day 1 to day 7): Reference normal range: pH = 7.35–7.45, HCO_3 = 22–28 meq/L, PCO_2 = 35–45 mmHg

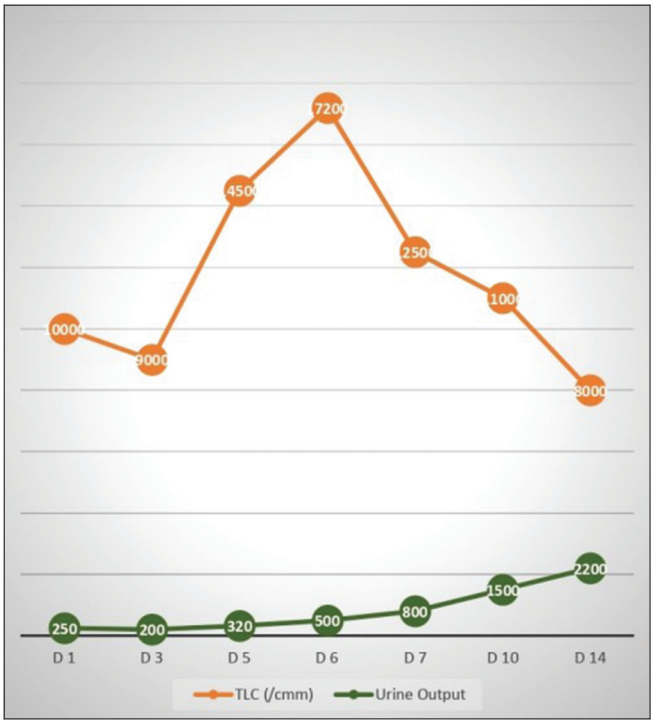

Figure 7: Total leucocyte count (TLC): Reference normal range = 4000–11000/mm³ and urine output in ml/day (day 1 to day 14)

12 hours of ingestion and 91% mortality occurred with 24 hours of ingestion.[2,4,10]

After ingestion of AlP, PH_3 gas absorption produces early manifestations of toxicity. Some absorbed AlP is metabolised in

liver and releases PH_3 slowly causing delayed toxicity. AlP causes cellular hypoxia, which leads to multiple organ failure, mainly renal, liver, and cardiac failure, appearance of cardiac arrhythmias, bundle branch block, ventricular tachycardia, or ventricular fibrillation.[5,9]

Our patient developed complications after initial response. On the second day, ECG showed VT and cardio-vascular collapse, although it was managed with DC shock and anti-arrhythmic drugs. He again showed signs of deterioration with appearance of kidney and liver failure and metabolic acidosis and septicaemia, which was managed appropriately.

Agrawal V K *et al.* mentioned that cardiac arrhythmia, respiratory failure, and requirement of mechanical ventilation are associated with poor prognosis. The presence of refractory shock, aspiration pneumonitis, metabolic acidosis, electrolyte imbalance, severe hypoxia, and gastro-intestinal bleeding indicates poor prognosis. Outcome correlates best with severity of hypotension and the number of vomiting episodes the patient gets after ingestion.[6,9,11]

Mathai A *et al.* mentioned that patients with high-base-line serum creatinine corelates best with increased mortality. They also showed that pH in ABG at admission is also a good indicator of prognosis. Survivors had statistically significant ($P = 0.015$) higher pH (7.284 ± 0.151) than non-survivors (7.148 ± 0.120).[11]

Our patient needed vasoactive drugs, a mechanical ventilator, low bicarbonate, and low pH and developed cardiac arrhythmia (VT). The presence of all these complications indicates high mortality. We discharged him on the 14th day.

Conclusion

AlP poisoning is very common in low- and middle-income countries. It is particularly common is South-East Asia and African countries, where farmers commonly use Celphos/AlP tablets as preservatives of rice and wheat. Our case highlights the importance of proper timely supportive measures, and management of complications in the absence of an antidote can prevent death in AlP poisoning. This case report can be useful as guidance for primary care physicians, who usually treat poisoning cases initially. We need to have regulatory and advisory guidelines to prevent or decrease the availability of such compounds in high-risk countries.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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