Aluminium Phosphide Poisoning - A Rare Case of Survival

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INTRODUCTION

Aluminium phosphide (ALP) is a widely available pesticide. It is highly toxic when ingested, and can lead to refractory hypotension, acute respiratory distress syndrome (ARDS) and fatal arrhythmias.[1] Management of aluminium phosphide toxicity is mainly supportive due to the fact that there is no specific antidote, so mortality with aluminium phosphide poisoning is very high, ranging from 37 % to 100 %.[2]

PRESENTATION OF A CASE

A 40-year-old male patient was admitted to the emergency ward with alleged history of consumption of 2 fresh tablets of aluminium phosphide (56 %) of 3 gram each an hour back, following ingestion of which patient developed nausea and vomiting 10 - 12 times. Vitals at admission and subsequent days has been charted in Table 1.

After placement of nasogastric tube, gastric lavage was done and initiated inotropes in view of hypotension then patient was shifted to medical intensive care unit (ICU) with guarded prognosis. Routine blood parameters including cardiac biomarkers imaging, echocardiography, electrocardiography were normal. The day after the admission, patient was awake, alert and blood pressure (BP) was normal with inotropic support.

On the 3rd day patient's condition deteriorated. Patient needed O2 support in the form of non-rebreather mask (NRBM) 15 litres to maintain saturation and needed escalation of inotropes for blood pressure maintenance. Patient developed bradycardia. Cardiac biomarker, echo, electrocardiogram (ECG) revealed toxin induced myocarditis and X-ray suggestive of early ARDS. Tab isoprenaline 10 mg BD was stared after cardiology consultation. With supportive measures and treatment as shown in the table 3, patients clinical condition improved and was taken off from inotropes and O2 supplementation. The patient responded to supportive measures over the next 4 days. On day 7, blood parameters, imaging studies, echo, ECG were repeated and were in decreasing trend.

On day 11 patient was discharged after clinical stabilization and psychiatry counselling.

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	D1	D4	D7	D11
PR	94	78	55	50
SpO2	98	90	92	99
BP	70/50	110/70 on NA	120/80	118/80
RR	16	24	20	17
GRBS	105	121	147	128
Hb	15.4	15	15.2	14.9
Total counts	2000	2100	2500	4500
Platelet	1.22 lakh	75000	81000	1.5 lakh
Blood urea	50	80	39	30
Serum creatinine	1.1	1.3	1.2	1.0
Sodium	140	136	132	139
Potassium	6.5	4.3	4.2	3.9
PT	14.08	12.1	13.2	12.8
INR	1.08	1.04	1.02	1.0
Total bilirubin	1.2	1.3	1.2	1.4
Direct bilirubin	0.4	0.2	0.3	0.2
AST	40	62	42	40
ALT	33	38	40	35
Troponin I	0.466	1.2	0.8	0.3
Chest Xray	Normal	Early ARDS	Near normal	Normal
ABG	pH-7.39 pCO2-44 mmHg HCO3-25 mEq/L			pH-7.36 pCO2-42 mmHg HCO3-22 mEq/L
Echo	EF-60 % Normal study	EF-40 % Globally hypokinetic LV Moderate TR PASP-60 %	EF-50 %	EF- 60 % normal study
ECG	normal	Sinus bradycardia	Sinus bradycardia	Sinus rhythm

Table 1. Vitals and Investigations during the Course in the Hospital

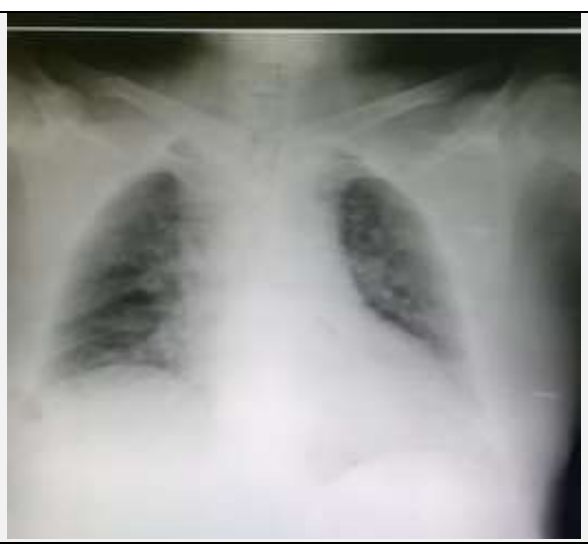

Figure 1. CXR on the Day of Admission

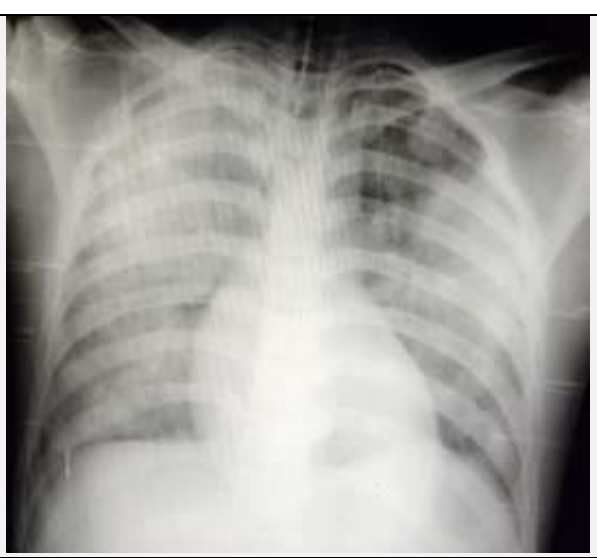

Figure 2. CXR on D4

DISCUSSION

Aluminium phosphide is highly effective insecticide and rodenticide. Acute toxicity can occur due to ingestion of products containing aluminium phosphide or by inhalation of phosphine gas generated during its use.[3] Aluminium phosphide poisoning is lethal with mortality rate about 70 %.[4] The mechanism of toxicity occurs by: a) Inhibition of cytochrome c oxidase. b) It rapidly perturbs mitochondrial morphology, inhibits oxidative respiration by 70 %, and causes a severe drop in mitochondrial membrane potential. c) It can interact with hydrogen peroxide forming reactive hydroxyl radical leading to lipid peroxidation d) It inhibits peroxidase and catalase There is no specific antidote for aluminium phosphide toxicity and the key to treatment lies in rapid decontamination and institution of resuscitative measures.[4,5] The specified fatal dose in human is 0.15 - 0.5 gm.[6] and the mortality rate is high (37 – 100 %).[2]

Figure 3. Aluminium Phosphide Compound Shown By Patient Attenders.

The risk of aluminium phosphide compound reduces in the following conditions:[6]

- When the patient consumes outdated or expired aluminium compound
- When aluminium phosphide is dissolved in water before use
- When the patient experiences immediate vomiting.

The clinical manifestations of ALP poisoning include gastrointestinal symptoms such as abdominal pain, vomiting and loose stools. It causes profound circulatory collapse, congestive heart failure and fatal arrhythmias. The patient may have arrhythmias and ischaemic changes secondary to myocyte depression. Electrocardiogram changes include heart blocks, ST changes, QRS prolongation and ventricular fibrillation. Drowsiness, delirium, and coma may result from cerebral anoxia. Other complications reported were pulmonary oedema, hepatitis, disseminated intravascular coagulation, gastrointestinal haemorrhage, and renal failure.[1,7] A positive history of ingestion is the basis of diagnosis in most of the cases. The presence of typical clinical features, garlic odour from the mouth and highly variable arrhythmias in a young patient with shock and no previous history of cardiac disease points towards aluminium phosphide poisoning.[6]

Decontamination with gastric lavage is probably best with potassium permanganate (1:10,000) to reduce absorption of phosphine. Permanganate oxidizes PH3 to form non-toxic phosphate.[6]

Supportive care is the most important factor is resuscitation of shock and institution of supportive measures as soon as possible. Oxygen is given for hypoxia. Acute respiratory distress syndrome requires intensive care monitoring and mechanical ventilation. The blood glucose concentration should be measured in every case and hypoglycaemia corrected if found.[6]

Magnesium administration appeared to terminate atrial fibrillation in many cases of aluminium poisoning[8,6] and supra ventricular tachycardia and ventricular tachycardia.[6,9]

Treatment	Experimental Model	Dose/ Route of AlP	Dose/ Route	Main Findings
Magnesium sulphate	Human study	-	-	Improve oxidative stress status
Coconut oil	Human study	12g	Oral	Increased survival rate
N-acetyl cysteine	Human study	-	140 mg/kg infusion (loading dose) followed by 70 mg/kg/iv infusion every 4th hourly	Reduction of the duration of hospitalization and mechanical ventilation. Decrement of mortality rate
Vitamin E	Case report	3g	400 units IM	Decrement of mechanical ventilation duration and reduction of the mortality
Liothyronine	Human study	-	50 mcg oral	Amelioration of cardiac complication and stress
Vasopressin	Animal study (rats)	12,5mg/ kg	2 IU/kg	Cardio protective effects

Table 2. Antidotes Studied for the Treatment of ALP Intoxication[10]

CONCLUSION

ALP is a very lethal poisoning with no specific antidote. Early detection and elimination of the toxin and intensive supportive care are the mainstay of the treatment. Despite high mortality rates, this case survived and was discharged in 11 days making it as an important rare case and unusual case.

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REFERENCES

[1] Muhamad NAN, Hawari R, Shafie H. A case report of aluminium phosphide poisoning. Med J Malaysia 2016;71(4):213-214.
[2] Ramadan N. Aluminum phosphide poisoning: a case of survival. Asia Pacific Journal of Medical Toxicology 2019;8(1):28-29.
[3] Anand R, Binukumar BK, Kiran DG. Aluminum phosphide poisoning an unsolved riddle. J Appl Tox 2011;31(6):499-505.
[4] Abder-Rahman H. Effect of aluminium phosphide on blood glucose level. Vet Hum Toxicol 1999;41(1):31-32.
[5] Proudfoot AT. Aluminium and zinc phosphide poisoning. Clinic Toxicol (Phila) 2009;47(2):89-100.
[6] Khlaif AT. Aluminum phosphide poisoning-case study. Global J Adv Res 2017;4(9):256-260.
[7] Singh S, Singh D, Wig N, et al. Aluminium phosphide ingestion--a clinico-pathologic study. J Toxicol Clin Toxicol 1996;34(6):703-706.
[8] Chugh SN, Chugh K, Ram S, et al. Electrocardiographic abnormalities in aluminium phosphide poisoning with special reference to its incidence, pathogenesis, mortality and histopathology. J Indian Med Assoc 1991;89(2):32-35.
[9] Chugh SN, Aggarwal HK, Mahajan SK. Zinc phosphide intoxication symptoms: analysis of 20 cases. Int J Clin Pharmacol Ther 1998;36(7):406-407.
[10] Karimani A, Mohammadpour AH, Zirak MR, et al. Antidotes for aluminum phosphide poisoning - an update. Toxicology Reports 2018;5:1053-1059.