J Nepal Health Res Counc 2014 Sep-Dec;12(28):200-2

Successfully Managed Case Of Celphos Poisoning: A Case Report and Review

Pangeni R,[1, 2] KC RK[1, 3]

[1]Patan Acadmy of Health Sciences, [2] Department of Medicine, Intensive Care Unit, [3]Oxfrod University Clinical Research Unit, Patan Hospital, Lalitpur, Nepal.

.

ABSTRACT

Aluminium phosphide poisoning is one of the major causes of suicidal deaths in developing countries like Nepal. It is one of the common fumigant available and is within easy access. It is primarily used for crop protection. However, it is one of the misused chemicals to commit suicide. In this case, the patient had hyperglycaemia, atrial fibrillation, severe metabolic acidosis, and shock and yet survived. The key to her survival was aggressive supportive management. Therefore, knowing all the prognostic factors along with aggressive management can be life saving in conditions when no antidotes are available.

Keywords: aluminium phosphide; fetal poisoning; fumigant; suicide.

INTRODUCTION

Aluminium phosphide (AlP) is the most commonly used rodenticide in developing countries. It is cheap, easily available and highly efficacious in protecting grains by killing insects of all stages without affecting the viability of the seeds.[1] However, AlP is increasingly misused to commit suicide either by ingestion or inhalation. It liberates phosphine gas (PH_3) on reacting with gastric acid, which results death in about 80% of cases.[2] It has no specific antidote and death rate is high even in our setup. This is the first reported case from Nepal, which was successfully managed with aggressive supportive treatment.

CASE REPORT

The patient presented to the Emergency Room with an alleged history of ingestion of three tablets of fresh aluminium phosphide, later confirmed by the container brought by the family. She was brought to hospital four hours post ingestion. At the time of presentation, she was disoriented and talking irrelevantly. She had a hypotension and systolic blood pressure was only about 60 mmHg measurable only by palpation, heart rate of 129 per minute; she had cold clammy extremities with oxygen saturation of 87% on oxygen at 6 Litres per minute. Activated charcoal was given along with rapid boluses of intravenous fluids followed by nor-adrenaline, which elevated the blood pressure to 98/68 mmHg. Arterial blood gas analysis showed severe metabolic acidosis (pH 7.15, HCO3-13.4), Na+ 137, K+ 5.8, pCO2-40. Routine investigation showed WBC count of 2.2 X 103/mm3 (N 83, L 17), HCT 34%, platelet count- 198, RBS - 241 mg/100ml, normal serum transaminase with increased K+ level (5.8 mmol/dl). Initial ECG showed paroxysmal atrial fibrillation [Figure 1: ECG of the patient], with normal serum troponin and CPK-MB level. Her serum lactate was 7 mmol/L.

Because of her poor cardiopulmonary status and low mentation, she was intubated and transferred to the Intensive care unit where she received injection hydrocortisone and $MgSO_4$ infusion. Blood sugar was controlled with insulin infusion.

Correspondence: Dr Radheshyam Krishna Kc, Patan Academy of Health Sciences, Patan Hospital, Lagankhel-5, Lalitpur, Neapal. Email: kc_radheshyam@yahoo.com, Phone: +977 9849319530.

On the next day, she had persistent lactic acidosis with lactate level of 8.5 mmol/L (normal range: 0.7 - 2.1 mmol/L), complicated by brief episodes of wide complex tachycardia ranging from sinus tachycardia, variable bundle branch block pattern to short run of ventricular tachycardia. On the third day, she developed persistent ventricular tachycardia, requiring cardiopulmonary resuscitation and DC shock, which could maintain sinus rhythm only for few seconds to minutes. Injection amiodarone was given as per the Advanced Cardiac Life Support protocol and maintained on infusion. Despite amiodarone and lignocaine, VT episodes were unremitting. The bedside echocardiography showed globally hypokinetic ventricles with poor ejection fraction of 15%. After 10 DC shocks, we administered iv metoprolol, titrated to the heart rate below 100 per minute, which stopped the recurrence of the VT episodes probably suggesting rate dependent malignant arrhythmia. The level of CPK-MB and troponin I, sent before the shocks, were elevated indicating myocardial injury. Her SGPT/SGOT were 2562/999 showing hepatic injury. Her blood sugar and K+ were controlled. Injection $MgSO_4$ and amiodarone were discontinued and post cardiac arrest care was started. Her GCS was low, with dilated and non-reactive pupils, flaccid limbs and mute planters. She began to show signs of improvement after 48 hours of the event and extubated after 11 days of hospital admission. Her ICU stay was complicated by ventilator-associated pneumonia with pleural effusion, and several episodes of flash pulmonary edema, which were managed by antibiotics, inotropes including dobutamine and diuretics. The patient improved and we were able to transfer her to medical ward 5 days after extubation for further management. She was discharged with stable vitals on day 22 of hospital admission. The bedside ECHO showed gradual improvement in ventricular contractility and on discharge, her echocardiography was essentially normal.

DISCUSSION

This patient presented with initial symptoms of retrosternal pain, vomiting and loose motion. The former two are the usual symptoms whereas diarrhoea is less common. It was followed by the development of shock, which is the cardinal feature of toxicity. Apart from these there can be other associated symptoms; restlessness, tachypnea and altered sensorium from confusion to disorientation followed by death.[3]

The patient had hypotension and atrial fibrillation, and Soltaninejad et al. (2012) reported in their study that both of these have worse prognosis,[4] which was once reported to have no effects.[5] These changes are found to be due to the action of PH3 (Phosphine gas). It affects the heart by many ways, in one hand it inhibits cytochrome c oxidase and affects mitochondrial oxidative respiration by 70% and results in myocardial energy depletion, similar to that occurs with ischemia.[6,7] In other hand, it blocks the endogenous antioxidant and increases an activity of superoxide dismutase results in generation of reactive oxygen species.[8] These reactive oxygen species bring lipid peroxidation [6, 7] and distorts intracellular homeostasis. Decreased ATP production and lipid peroxidation cause severe drop in membrane potential resulting in alteration of Na+, Mg++ &Ca++ ions permeability finally leading to cellular death. Hence, changes on cardiac rhythm followed by cardiogenic shock are the usual presentation of AlP poisoning. Magnesium Sulphate administered based on the documented evidence of its membrane stabilizing action. However, the rational use of Magnesium Sulphate has to be guided by serum Magnesium levels, as there have been reports of occurrence of hypomagnesaemia.[9] But due to the lack of facility magnesium level was not measured in this patient.

AlP also causes derangement of glucose homeostasis and hyperglycaemia is one of its effects. Toxic injury of the pituitary adrenal axis and acute pancreatic injury are proposed to be the reason for hyperglycaemia.[10] According to Mehrpour et al. (2008), the risk of mortality increases when blood sugar raises above 140 mg/dl and most of the non-survivor of AlP poisoning had blood sugar level 222 mg/dl.[10] In her case, her blood glucose level was 241 mg/dl. Therefore, she was at high risk.

Circulatory failure ensues decreased blood supply leading to tissue hypoxia. This favours anaerobic respiration leading to lactate production, which is reflected in this case by raised serum lactate level i.e. 8.5 mmol/L indicating metabolic acidosis.

Pesticides/rodenticides are the most common suicidal agents in the part of world where agriculture is the main profession. Use of such agents lead to severe complications that mostly results in death. The patient had atrial fibrillation followed by metabolic derangements to ventricular arrhythmia and asystole, however due to aggressive treatment patient treated successfully. This is the result of understanding all the prognostic factors and providing all supportive measures along with continuous intensive treatment and monitoring for the complications.

ACKNOWLEDGEMENTS

We would like to thank all the staff of the emergency department and Intensive care unit of Patan Hospital for their effort to save the patient.

REFERENCES

1. Wahab A, Rabbani MU, Wahab S, Khan RA. Spontaneous self-ignition in a case of acute aluminium phosphide poisoning. Am J Emerg Med. 2009 Jul;27(6):752 e5-6.

2. Mehrpour O, Abdollahi M. Poison treatment centers in Iran. Hum Exp Toxicol. 2012 Mar;31(3):303-4.

3. Bogle RG, Theron P, Brooks P, Dargan PI, Redhead J. Aluminium phosphide poisoning. Emerg Med J. 2006 Jan;23(1):e3.

4. Soltaninejad K, Beyranvand MR, Momenzadeh SA, Shadnia S. Electrocardiographic findings and cardiac manifestations in acute aluminum phosphide poisoning. J Forensic Leg Med. 2012 Jul;19(5):291-3.

5. Chugh SN, Chugh K, Ram S, Malhotra KC. Electrocardiographic abnormalities in aluminium phosphide poisoning with special reference to its incidence, pathogenesis, mortality and histopathology. J Indian Med Assoc. 1991 Feb;89(2):32-5.

6. Singh S, Bhalla A, Verma SK, Kaur A, Gill K. Cytochrome-c oxidase inhibition in 26 aluminum phosphide poisoned patients. Clin Toxicol (Phila). 2006;44(2):155-8.

7. Proudfoot AT. Aluminium and zinc phosphide poisoning. Clin Toxicol (Phila). 2009 Feb;47(2):89-100.

8. Kariman H, Heydari K, Fakhri M, Shahrami A, Dolatabadi AA, Mohammadi HA, et al. Aluminium phosphide poisoning and oxidative stress: serum biomarker assessment. J Med Toxicol. 2012 Sep;8(3):281-4.

9. Singh S, Singh D, Wig N, Jit I, Sharma BK. Aluminum phosphide ingestion--a clinico-pathologic study. J Toxicol Clin Toxicol. 1996;34(6):703-6.

10. Mehrpour O, Alfred S, Shadnia S, Keyler DE, Soltaninejad K, Chalaki N, et al. Hyperglycemia in acute aluminum phosphide poisoning as a potential prognostic factor. Hum Exp Toxicol. 2008 Jul;27(7):591-5.