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A Case of Acute Aluminium Phosphide Poisoning With ST Segment Elevation ECG Changes

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Abstract
AlP poisoning has elevated mortality rates because of cardiovascular involvement. Patient survival can be increased by prompt supportive care. In this study, we evaluated a case of aluminium phosphide intentional poisoning in an adult patient. The patient had toxic myocarditis and developed ST elevation on ECG during hospital course, raised troponins and global hypokinesia on echocardiogram. The patient was managed by trimetazidine, intravenous magnesium sulphate, n-acetylcysteine and inotropes. Follow-up echocardiogram, three months later was normal.
Keywords: *Pesticide, Aluminium phosphide, Cardiotoxicity.*

Introduction

Globally, pesticide self-poisoning account for 14-20% of suicides and are responsible for 110,000–168,000 deaths each year. In the year 2015, over 20,000 persons died from pesticide self-poisoning in India[1]. Aluminium phosphide is a fumigant rodenticide, pesticide, and grain preservative agent. It is found in every rural household of northern Inda, involved in cereal grain crop production and storage. It is cheap, easily available in the market and readily accessible at home. It is sold as tablets or pellets, granules and as powder. The toxidrome includes features specific to cardiovascular system and include hypotension, cardiogenic shock, dysrhythmias, global wall motion abnormalities and reduced ejection fraction. In addition, toxidrome includes nausea, vomiting, epigastric discomfort, retrosternal burning, diarrhea, tachypnea, cyanosis, adult respiratory distress syndrome, tender hepatomegaly, jaundice, elevated transaminases, oliguria, acute renal failure, altered sensorium, restlessness, coma, metabolic acidosis, hypomagnesemia, hypermagnesemia and hypokalemia[2].

In the present study, we are describing a case of acute aluminium phosphide poisoning with ST segment elevation in ECG, raised troponins and reversible global hypokinesia on echocardiogram. This is an uncommon presentation due to a commonly consumed pesticide with a suicidal intent. It is important to be aware of these features associated with exposure to aluminum phosphide and have knowledge of management protocols. Analyzing the pathophysiology of ALP poisoning, the use of antiplatelets, anticoagulants and thrombolytic agents for ST segment elevation is unjustified.

Case Report

A 19 years old male gave a history of ingestion of one tablet of aluminium phosphide 3-4 hours back at 1pm on 31-12-2022. He presented to emergency with history of vomiting. The vomitus was non-bilious and non-projectile. He had no significant past history, family and personal history. On examination he was conscious, oriented, and sick looking. His radial pulse was not palpable and blood pressure was unrecordable. He had cold extremities and the capillary refill time was prolonged. Rest of cardiovascular system examination was normal. Patient was started on intravenous fluids and ionotropic support. The ECG at the time of admission had right axis deviation, absent P waves and irregular ventricular response (suggestive of atrial fibrillation) and left posterior fascicular block (Figure 1). On day one of admission ECG was showing wide QRS tachycardia (Figure 1). Patient was started on intravenous magnesium sulfate (2 g intravenously stat followed by intravenous infusion of 1 g/hour for 3 h, followed by 2 g 8 hourly for next 24 hours). On second day of admission ECG had features of ST elevation in V1, V3, V4 leads (Figure 1). His high sensitivity cardiac troponin (hs-cTn) were 9.74 ng/ml (0.0 -0.06 ng/ml). 2D Echo showed global dyskinesia with ejection fraction of 30-35%. Patient was started on N-acetylcysteine (NAC) infusion at150mg/kg over one hour followed by 50 mg/kg IV over four hours, then 100 mg/kg IV over 19 hours and tablet trimetazidine (35 mg twice a day). Blood pressure normalized on day three with inotropic support. Magnesium sulfate was stopped. On day four and beyond ECG was normal (Figure1). Inotropes were discontinued on day four. The details of laboratory investigations from the day of admission till discharge are shown in Table 1. Patient was discharged in stable condition. Repeat echocardiogram after three months was normal.

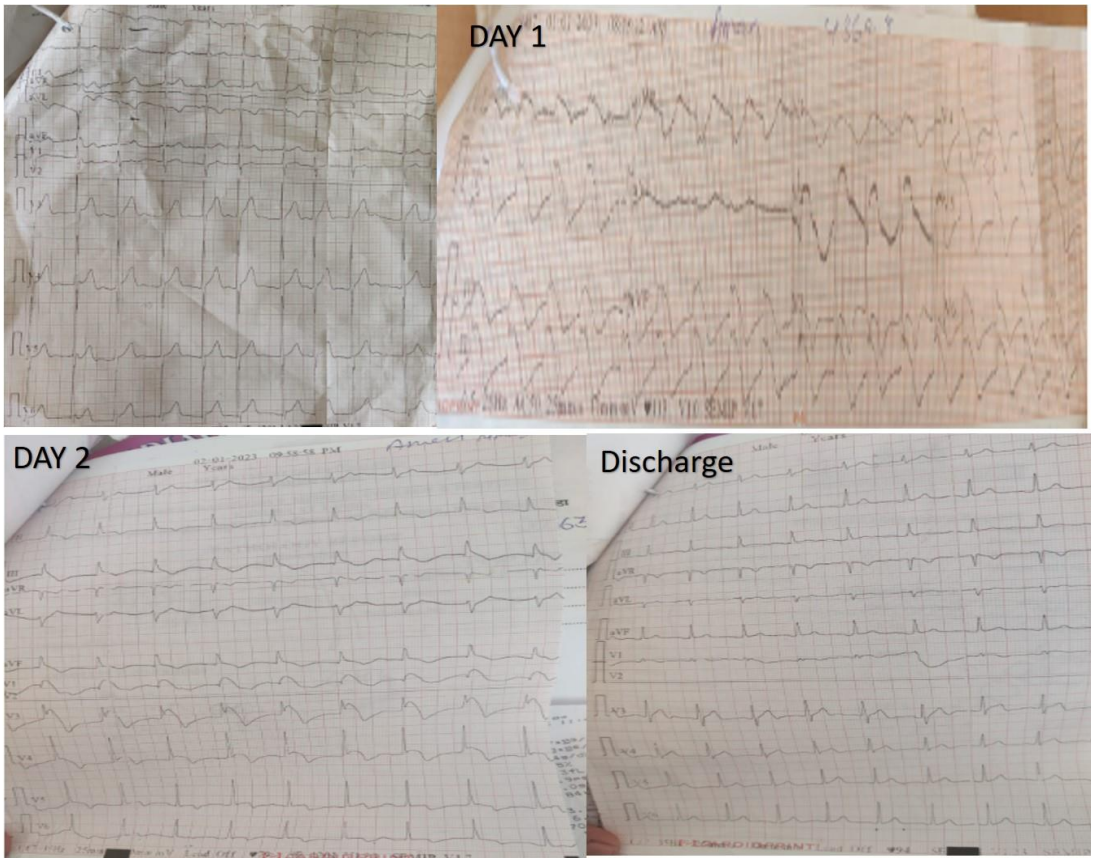

Figure 1: ECG showing right axis deviation, absent P waves and irregular ventricular response (suggestive of atrial fibrillation) and left posterior fascicular block (at admission); wide QRS tachycardia (Day 1); ST segment elevation V1,V3,V4(Day 2); normal(at discharge).

Table 1: Laboratory parameters during hospital stay

Parameter	At admission	Day 1	Day 3	Day 7	At discharge
Hb(gm/dl)	12.8	12.2	10.4	10.5	-
TLC(per µL)	14160	20800	4700	4000	-
Platelet(per µL)	132000	180000	84000	268000	-
Bilirubin(mg/dl)					
Total	1.7	3.8	4.5	0.5	0.8
Direct	0.7	1.4	2.3	0.2	0.3
AST(U/L)	13	227	190	45	43
ALT(U/L)	10	181	250	94	81
ALP(U/L)	85	96	119	108	99
Urea(mg/dl)	29	50	168	33	33
Creatinine(mg/dl)	1.0	1.9	2.3	1.3	0.9
Magnesium(mg/dl)	2.1	2.7	2.9	-	

Hb-hemoglobin; TLC-total leucocyte count; AST-aspartate transaminases; ALT-alanine transaminases; ALP- alkaline phosphatase

Discussion

Aluminium phosphide poisoning has a high mortality rate (40-80%). Early deaths are due to cardiotoxicity. shock, severe metabolic acidosis, and refractory hypotension are poor outcome predictors. The cardiovascular manifestations include shock or hypotension, tachycardia or bradycardia, cardiac arrhythmia, myocarditis, pericarditis, congestive heart failure and asystole ECG changes like ST segment dispersion and QT interval prolongation. Electrocardiographic abnormalities are very common and highly variable in aluminium phosphide poisoning and include supraventricular and ventricular ectopics, sinus bradycardia or tachycardia, supraventricular tachycardia, atrial flutter/fibrillation, ventricular tachycardia, ventricular fibrillation, ST-segment elevation/depression, T inversions and variable degrees of heart block.[3] The cardiotoxicity is the main feature of ALP poisoning.[4] Aluminium phosphide in the presence of moisture and/or gastric hydrochloric acid release phosphine gas. Phosphine inhibits the catalase, cytochrome *c* oxidase (ETC) and glucose-6-phosphate dehydrogenase (G6PD) activity in cardiac myocytes. It disrupts the mitochondrial function, decrease in overall ATP production, and reduces myocardial energy. G6PD inhibition reduces glutathione concentration. Free radicles especially ROS and oxidative stress lead to lipid peroxidation and lead to cardiac toxicity and multiorgan failure[4].

The differential diagnosis of ST segment elevation includes; myocardial ischemia/infarction, acute pericarditis, early repolarization pattern, Brugada pattern and myocarditis (infectious and non-infectious). ST segment elevation, raised troponins and reversible global hypokinesia suggest toxic myocarditis as the likely cause of the changes in our case. The most common etiology of ST segment elevation and raised troponins is acute coronary syndrome. However, these findings in the setting of ALP poisoning don't warrant use of antiplatelets, anticoagulants or thrombolytics therapy. The mechanism is not acute atherothrombosis of coronary artery but ALP induced myocardial injury as described. The management of ALP poisoning is supportive in absence of a specific antidote. Therapeutic approaches for managing cardiotoxicity includes intravenous vasopressors, intra-aortic balloon pump, digoxin, melatonin, trimetazidine, n-acetyl

cysteine, extracorporeal membrane oxygenation, liothyronine (T3), minocycline, l-carnitine, magnesium sulfate, glucose insulin potassium infusion[4]. None of the treatment strategies have shown consistent results in the prevention or treatment of cardiovascular complications of ALP poisoning.

Conclusion

Acute aluminium phosphide poisoning was associated with toxic myocarditis and developed ST elevation on ECG with global hypokinesia on ECHO which was fully reversible on follow up ECHO 3months later.

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