Aluminum Phosphide Poisoning Presenting like Acute Myocardial Infarction in a Young Girl

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Abstract
Aluminum phosphide poisoning is very common in India. It is one of the most fatal poisons. The clinical spectrum of poisoning varies depending upon the dosage and duration of consumption. The main effect of the poison is due to the release of phosphine which inhibits cytochrome oxidase and thereby hampers cellular oxygen utilization. Almost any organ can be affected by aluminum phosphide poisoning. We report a case where the heart was the predominantly affected organ. Cardiac involvement usually manifests with nonspecific ST-T changes in ECG. Here we report a case presented with ECG finding mimicking acute anteroseptal myocardial infarction which is extremely rare.

Introduction
Aluminum phosphide (ALP) poisoning (Celphos) has emerged as a common cause of accidental poisoning in children with a mortality ranging from 37-100%.¹ Since ALP is commonly used as a fungicide and rodenticide in India, many reports of accidental poisoning with severe consequences have been noted both in adults and children. However, only a few cases from outside India have been reported. The spectrum of symptoms and signs and their severity depends upon the time lag between ALP ingestion and hospitalisation. The most common presentation is shock with cold and clammy skin, a weak thready pulse and severe hypotension often refractory to vasopressors. Arrhythmias are common in ALP poisoning and are attributed to various causes including hypomagnesaemia. Neurological, gastrointestinal and renal involvement is also common and documented in many case reports. Myocardial depression is also reported in many cases. In ECG cardiac involvement usually manifests as nonspecific ST-T changes. We report a case of ALP poisoning with severe myocardial depression which manifested in the ECG as acute anteroseptal myocardial infarction which is extremely rare.

Case Report
A 19-year-old female patient presented to the hospital with a history of ingestion of 2 Celphos tablets with a suicidal intent (one tablet of Celphos - 3 gm). The time between consumption and hospital arrival was approximately 5 hours. At presentation the patient complained of epigastric pain and nausea. She was conscious and oriented, but restless with cold clammy extremities and diaphoresis. Her pulse rate was 110 beats/minute and blood pressure was 80/40 mmHg. Examination of the respiratory system was unremarkable. The oxygen saturation was 90% with pulse oximetry in room air. Routine blood examination and biochemical parameters were sent and the patient was started on rapid IV infusion with normal saline along with norepinephrine administered as per the standard dosage. Gastric lavage was performed and coconut oil given through Ryle’s tube. Investigations showed a haemoglobin of 12 gm%, total white cell count of 7000/cumm and a normal ABG. Serum total calcium was 8.8 mg/dl, serum magnesium 2.33 mg/dl, serum sodium 143 meq/L, serum potassium 5.0 meq/L and serum bicarbonate 20 mmol/L. Blood urea, serum creatinine and LFTs were normal. Electrocardiogram (ECG) recorded on arrival showed a broad QRS complex with ST elevation in mainly the anteroseptal leads mimicking anteroseptal wall myocardial infarction (Figure 1). Serum CPKMB was very high (290 U/L) and Troponin T was positive.

Echocardiography showed hypokinesia of IVS and anterior wall with a left ventricular ejection fraction of 30%. Over the next few hours the patient’s blood pressure continued to fall despite being on norepinephrine and dopamine infusion was added but the patient continued to deteriorate. Electrolytes remained except for a mild decrease in serum magnesium and hence, IV Magnesium was given. But despite our best efforts the patient succumbed due to circulatory failure.

Discussion
Aluminum phosphide (ALP) is used as a rodenticide and is a commonly used as a suicidal agent in some parts of India.¹ Refractory myocardial
depression from ALP toxicity is not uncommon and carries a mortality of up to 77% (37-100%). Easy availability and no antidote makes it an ideal suicidal poison. Upon exposure to moisture, it liberates phosphine gas, which is absorbed rapidly by inhalation or through the cutaneous or enteral routes. Phosphine resembles cyanide in that it inhibits cytochrome oxidase and thereby hampers cellular oxygen utilization. The classical presentation of ALP is epigastric pain, nausea and cardiogenic shock reflected as severe refractory hypotension and is described in many case reports. There are a few case reports of survival in case of ALP poisoning when patients were treated with vegetable oils particularly with coconut oil and hence, we tried the same. The lethal dose for a person of 70 Kg is 150-500 mg. Cardiovascular involvement is common in ALP poisoning and is manifested by hypotension, shock, bradycardia or tachycardia, CHF with toxic myocarditis and ECG abnormalities. Our patient had severe myocardial depression with ECG changes resembling myocardial infarction/myocarditis. ECG changes in Celphos poisoning have been studied in various studies. It include atrial fibrillation, supraventricular and ventricular tachycardia, ST-T changes, bundle branch blocks and AV conduction disturbances. Although ST-T depression with T-inversion is present in many cases but pure ST-segment elevation is not so commonly reported in literature. Echocardiographic findings include decreased ejection fraction, generalized hypokinesia of the left ventricle, and pericardial effusion. However regional wall motion abnormalities along a arterial territory is extremely uncommon. Our case in unique because ECG and echocardiographic finding were mimicking with ischaemic insult to left anterior descending arterial territory which is extremely unusual in ALP poisoning.

**Conclusion**

Aluminium phosphide poisoning can involve various organ systems in the body owing to its inhibition of cytochrome oxidation pathway and cause serious cardiac toxicity, circulatory failure and death. Cardiac involvement is marked by ST-T changes and bundle branch blocks in ECG and global hypokinesia with depressed left ventricular ejection fraction in Echocardiography. However ST elevation and regional wall motion abnormality as seen in our patient mimicking antero-septal myocardial infarction is extremely rare.

**References**