A case of zinc phosphide poisoning with ST segment elevation ECG changes

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Abstract
Zinc phosphide (Rat poison) poisoning is becoming increasingly common in most parts of India. It is one of the most lethal poisons. The harmful effects of the poison have been ascribed to the release of phosphine gas which inhibits cytochrome oxidase. Aluminium phosphide, a similar toxin has been well documented to produce infarct pattern in ECG. However such changes ascribable to Zinc Phosphide have been infrequently reported in literature. We hereby report the case of young teenage male whose ECG showed ST elevation mimicking myocardial infarction pattern which resolved after appropriate management of the poisoning.

Keyword: ST elevation, Pseudoinfarct Pattern, Myocarditis, Toxic Carditis.

Introduction
Zinc phosphide (ZP) is used as a fungicide and rodenticide in India .In recent years ZP poisoning has become a common cause of suicidal poisoning in adults.(1) The most common presentation is cardiogenic shock secondary to myocarditis. Neurological, gastrointestinal and renal involvement is also common.(2) Aluminium Phosphide (which has similar physical and chemical structure) is very well known to produce ST elevation mimicking myocardial infarction in ECG.(3) Very few reports are available regarding this with respect to ZP poisoning that too mainly in adults and elderly patients. We encountered a young teenage patient with ZP poisoning whose ECG showed ST elevation pattern mimicking myocardial infarction, which reversed subsequently after 4 days.

Case Report
A young boy of 17 years was brought to the emergency with history of consumption of rat poison. He had swallowed three tablets of zinc phosphide with suicidal intention three hours prior to presentation. The patient complained of severe pain in the upper abdomen. On examination patient was conscious, PR-100b/min, BP-110/70mmhg, Spo2-99% at room air. There was no icterus or cyanosis. Cardiovascular system, Respiratory System, Per Abdomen and Central nervous System revealed no significant abnormality. ECG was normal. (Fig. 1) After initial stabilisation, Activated charcoal 100g was given, gastric lavage was done with potassium permanganate (1:10,000),(4) and he was shifted to MICU. He was started on magnesium sulphate (3 g as infusion over 3 h, followed by 6 g per 24 h for 3–5 days) and N-acetylcysteine as per protocol. Other supportive measures were also initiated.

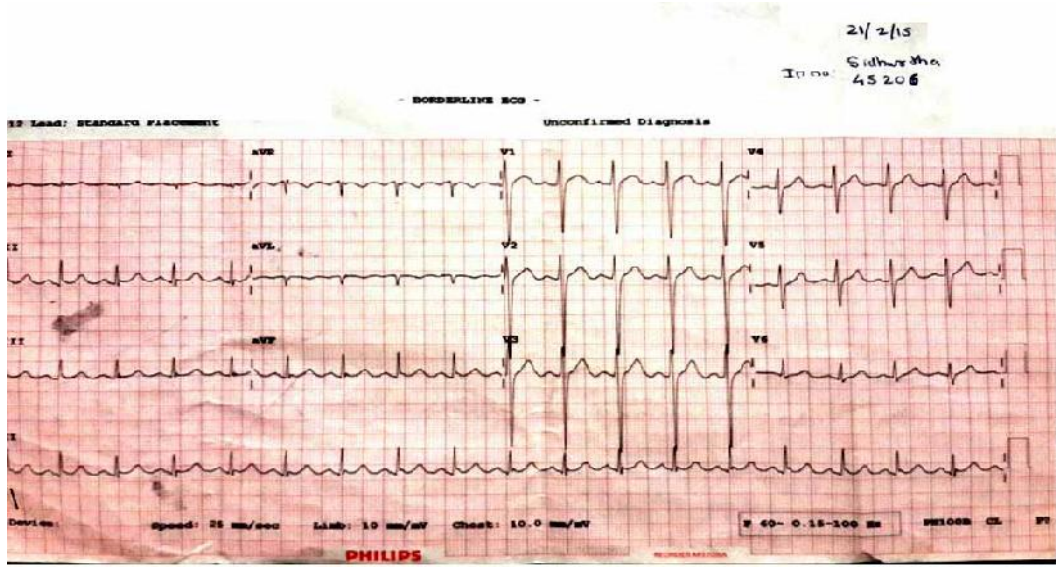

Fig. 1: Showing ECG on admission which is within normal limits L

After 12 hours, patient reported persistent palpitations. He did not have any chest pain or sweating. Monitor showed tachycardia of 140beats/min. ECG showed significant ST elevation with convexity upwards in leads 2,3,aVF and V3-V6 (Anterior-lateral and inferior leads). (Fig. 2) Cardiac enzymes were significantly elevated (Trop T-0.67, CK-MB- 124). Echocardiography showed global hypokinesia with ejection fraction of 40%. The above features of myocarditis prompted us to start steroids (Inj. Dexamethasone 4mg IV TID) which were continued for 4 more days.

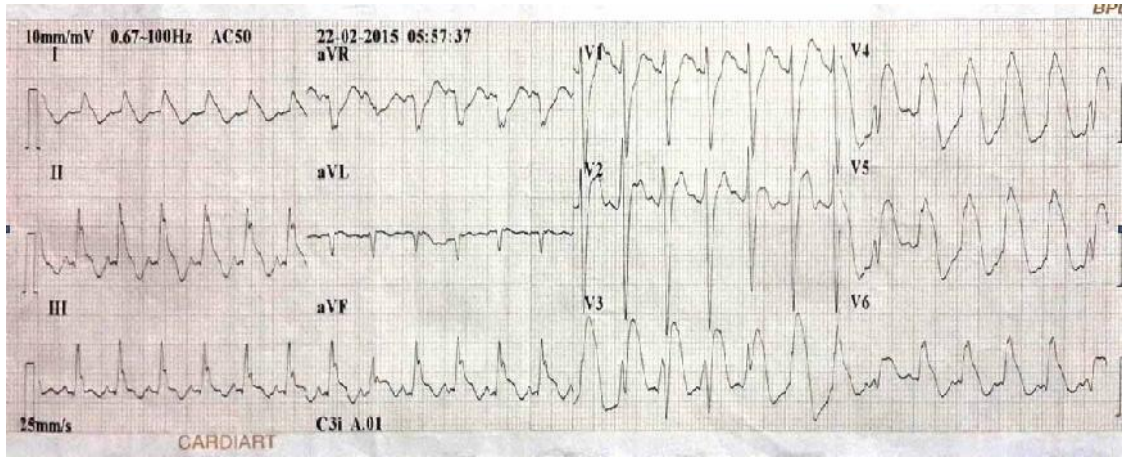

Fig. 2: ECG showing ST segment ST Elevation 12 hours after admission

On 2nd day patient was hemodynamically stable. Daily ECG showed no changes for subsequent 2 days. On day four, the above ECG changes resolved and ECG showed normalised ST segments. (Fig. 3) The patient recovered completely and was discharged in a stable condition.

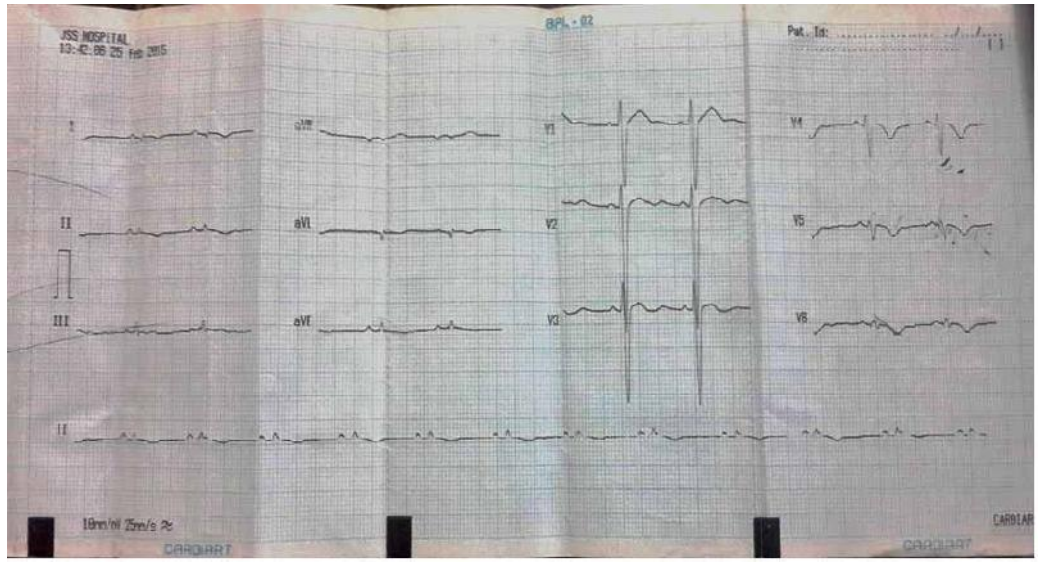

Fig. 3: ECG showing ST segment resolution on fourth day

Discussion

Zinc phosphide is similar to aluminium phosphide in releasing phosphine (PH_3) when coming in contact with moisture. PH_3 is absorbed from the gastrointestinal tract, and it disturbs the mitochondrial function by blockage of the cytochrome C oxidase once it reaches the tissues. Energy failure in cells and generation of the free radicals also result in lipid peroxidation.[5, 6] Profuse vomiting, abdominal pain, tachypnea, hyperpnoea, dyspnoea, chest tightness, tachycardia, hypotension, raised transaminases, hepatic failure and delayed onset noncardiogenic pulmonary oedema have been reported as the clinical manifestations associated with this poisoning.[6, 7]

Aluminium phosphide poisoning is well known to result in atrial fibrillation, supreventricular tachycardia and ventricular tachycardia. ST-T changes have also been reported.

Various ECG changes following Zn Phosphide poisoning have been reported. Atrial fibrillation, supreventricular tachycardia and ventricular tachycardia, non specific ST-T changes, bundle branch blocks have all been noted.[7, 8] However most of the reported cases were seen in subjects aged 45 years and above. The features of the present case of note are ST elevation changes of myocardial infarction pattern and the very young age of the subject.

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