CASE REPORT

Severe reversible myocardial injury associated with aluminium phosphide toxicity: A case report and review of literature

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Aluminium phosphide is commonly used as an insecticide and can be toxic to humans at the cellular level by interfering with mitochondrial energy metabolism. We report on three cases of severe aluminium phosphide cardiotoxicity, resulting in severe decrease in both ventricular heart functions. The first case succumbed to intractable ventricular arrhythmias complicated by multi-organ failure before she died; while the other two cases required invasive hemodynamic support and eventually improved over the course of 10–14 days. We describe our experience and the challenges faced while managing one of them.

Keywords: Cardiomyopathy, Extra corporal membrane oxygenation (ECMO), Intra-aortic balloon pump, Aluminium phosphide toxicity

Established facts

Established fact 1: Aluminium phosphide toxicity causes cardiac toxicity.

Established fact 2: No known antidote for this type of poisoning at present.

Novel insights

Novel addition 1: Aluminium phosphide causes reversible cardiac toxicity whose effects may last 10–14 days. According to our local experience, optimal aggressive hemodynamic support throughout the injury period results in better outcomes.

Novel addition 2: To our knowledge, this is one of the first reported cases of aluminium phosphide poisoning treated using a combination of intra-aortic balloon pump and extra corporeal membrane oxygenation to treat severe prolonged cardiogenic shock.

Novel addition 3: Drugs which work at the mitochondrial level to improve the metabolism of cardiac muscle cells may be a useful adjunct to invasive hemodynamic support in such severe cases of poisoning.

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Case report

A 45-year-old woman, accompanied by her seven-year-old son and 11-year-old daughter, presented to the emergency department with vague symptoms of fatigue, nausea and recurrent vomiting. They were initially treated with intravenous normal saline, anti-emetics and discharged home after several hours. The next day, they presented with severe deterioration in clinical status as evidenced by hypotension, tachypnea and tachycardia (See Fig. 1).

The 11-year-old daughter followed a steeply declining clinical course. Within 12 h she was intubated, developed intractable ventricular tachycardia twice, requiring prolonged CPR and multiple DC shocks, followed by persistent hypotension and anuria despite multiple inotropes. She succumbed to her fate and died within 36 h.

The 6-year-old son had a bedside echo showing left ventricular ejection fraction (LVEF) of 35% and was started on milrinone infusion 0.25/kg/min. He was later moved to another facility for further treatment which included extra corporeal membrane oxygenation (ECMO) (See Fig. 2).

Our index patient had been initially started on triple inotropes (Dobutamine 20 μg/kg/min, Dopamine 20 μg/kg/min, Noradrenaline 8 μg/h), which did not deter her hemodynamic deterioration. Her initial bedside echocardiogram showed severe biventricular systolic dysfunction, and LVEF of 25–30%. No valvular abnormality, no pericardial effusion. On the second day, she required intubation, and an intra-aortic balloon pump (IABP) was inserted. Six hours later, she continued to be anuric with high oxygen requirement and low blood pressure, and she was placed on ECMO via right femoral vein and artery access, while her IABP was kept via left femoral artery access. Cardiac output was set at 3.7 l/min. Unfractionated heparin was initiated by bolus 1000 units followed by 800 units/h, with target ACT of 150–180. She was started on 8 L/min oxygen. The patient also required CRRT during the initial two days due to acute kidney injury and anuria (See Fig. 3).

Medication wise, the patient was started early on high dose L-carnitine 1 g three times a day for 10 days along with broad spectrum antibiotics. Initial hypo-magnesemia was treated intravenously by supplemental magnesium sulphate (2 gm) daily for four days. She was also started on Candesartan 2 mg bid, Spironolactone 12.5 mg daily, and Carvedilol 3.125 mg bid (See Fig. 4).

Over the next day, the patient was weaned off inotropes. Daily bedside echo via sub-costal window showed initial deterioration of overall cardiac function up to day 3; in the form of severe global myocardial hypokinesia/akinesia. LVEF had reached a nadir of 10%; right ventricular dysfunction was evidently moderately severe. Slow improvement ensued over the next 10 days with LVEF rising up to 45–50%. Initial improvement had commenced in the lateral and inferior left ventricular walls, while last walls to improve were the septal and anterior walls. Initial echo pictures had also shown a mild increase of wall thickness involving myocardial mid and distal segments (up until the apex). As heart function improved, this regressed. A repeat echo performed 3 weeks after index event confirmed the almost complete resolution of systolic dysfunction in all wall segments except distal apex. There was resolution of all the myocardial increased thickness except at the distal lateral wall segment where it persisted.

ECG at presentation showed normal sinus rhythm, normal PR interval and partial left bundle branch block with QRS duration of 94 ms. Over

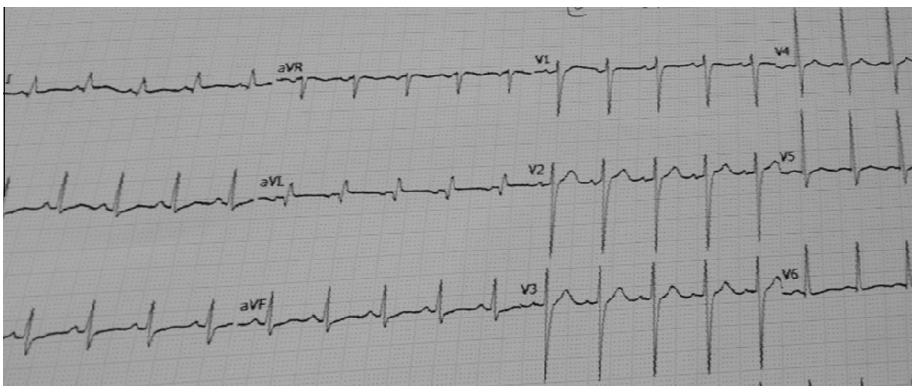

Figure 1a. Initial ECG showing minimal QRS prolongation and non-specific ST and T wave changes.