

# STEMI in Young Befogged by Aluminum Phosphide Toxicity—Role of ECMO as Salvage Therapy and Trimetazidine and Magnesium to Suppress Arrhythmias

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## ABSTRACT

**Introduction:** Aluminum phosphide poisoning (ALP) has a high-mortality rate despite intensive care management, primarily because it causes severe myocardial depression. This case report highlights the subset of ALP patients presenting as ST elevation myocardial infarction (STEMI) with profound myocardial dysfunction and multiorgan failure and successfully treated with extracorporeal membrane oxygenation (ECMO), trimetazidine, and magnesium.

**Case description:** A 25-year-old man without any comorbidities was brought to emergency department with dyspnea and hypotension. His electrocardiograph (ECG) revealed STEMI with elevated troponin levels, arterial blood gas (ABG) showed severe metabolic acidosis, and echocardiography (echo) revealed ejection fraction 15%. He was initiated on venoarterial (VA) ECMO in view of refractory hypotension. History of consumption of three tabs of celphos was revealed later by the family members. He progressed to cardiogenic shock, arrhythmias, respiratory failure, acute kidney injury with severe lactic acidosis, liver injury, pancreatitis, and disseminated intravascular coagulation (DIC). He was successfully supported by ECMO, hemodialysis, magnesium, trimetazidine, *N*-acetyl cysteine, inotropes, and blood products. He was weaned off ECMO on day 6 and was discharged home on day 12. Despite his severe and confounding clinical presentation, he had complete normalization of end-organ dysfunction with no neurological sequela. This case demonstrates the high index of suspicion required for ALP, given the potential for rapid progression and severe multiorgan toxicity. This report also highlights the importance of early referral to a tertiary care center with ECMO capability and also the role of magnesium and trimetazidine to suppress arrhythmias.

**Conclusion:** Aluminum phosphide poisoning can present as STEMI with cardiogenic shock resulting in acute kidney injury, liver injury, pancreatitis, and DIC. Venoarterial ECMO provides an effective means of support until the recovery of organ function. Trimetazidine and magnesium are helpful in suppressing fatal arrhythmias. This report emphasizes that early recognition and early institution of ECMO can save many young lives who succumb to toxic effects of this poison.

**Keywords:** Aluminum phosphide, Extracorporeal membrane oxygenation, Magnesium, ST elevation myocardial infarction, Trimetazidine.  
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## INTRODUCTION

Aluminum phosphide is a common form of poisoning in India with no specific antidote. Refractory myocardial depression due to aluminum phosphide poisoning (ALP) toxicity carries a very high mortality rate.<sup>1,2</sup> Patients deteriorate rapidly due to the effect of phosphine gas produced when the poison comes in contact with moisture or acid in the stomach causing a cyanide-like toxicity in which mitochondria are unable to utilize oxygen due to the inhibition of the cytochrome oxidase enzyme system leading to cellular hypoxia.<sup>3</sup> The resultant anaerobic metabolism and severe lactic acidosis causes multiorgan dysfunction and death ensues.<sup>3</sup> Extracorporeal membrane oxygenation (ECMO) has shown consistently positive results by providing cardiovascular support till the poison is eliminated from the body.<sup>11</sup> Trimetazidine preserves oxidative metabolism and magnesium by stabilizing cardiac membrane and decreases arrhythmias.<sup>17,18</sup>

## CASE DESCRIPTION

A 25-year-old man without any comorbidities was brought to emergency department with sudden onset of dyspnea. He was hypotensive or arrival not responding to intravenous fluids and high-dose vasopressors. Arterial blood gas (ABG) showed severe metabolic acidosis with electrocardiograph (ECG) showing ST elevation in lateral leads. Two-dimensional (2D) echocardiography

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(echo) revealed global left ventricular dysfunction, ejection fraction of 25% along with elevation of cardiac biomarkers. His condition rapidly deteriorated with refractory hypotension and severe lactic acidosis. Initiated on venoarterial (VA) ECMO as a rescue therapy with left femoral venous access (25 Fr) and right femoral arterial access (17 Fr) with an extra 6 Fr sheath inserted distally into right femoral artery for the perfusion of distal leg cannula. Pump flow was maintained at 4.5 L/minute. Heparin-bonded circuit was used and anticoagulation was avoided due to coagulopathy. He progressed to

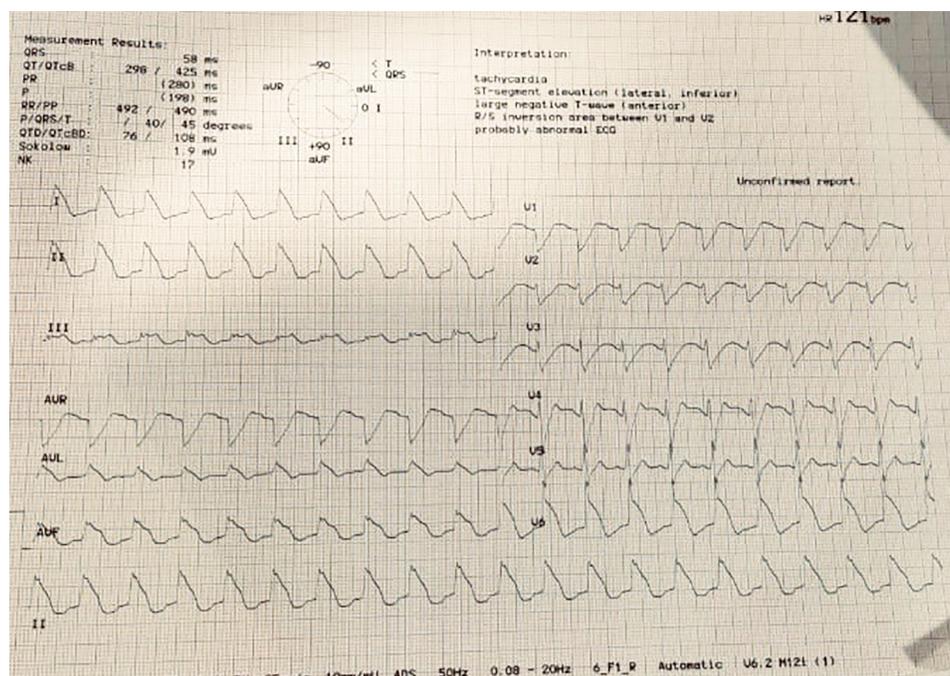


Fig. 1: ECG changes showing broad QRS complexes

acute kidney injury with worsening of lactic acidosis and developed wide QRS complexes (Fig. 1) and he was treated with magnesium and trimetazidine. He also developed severe ischemic hepatitis with disseminated intravascular coagulation (DIC) and was managed with blood product transfusion. The serum transaminases peaked at day 3 and showed a decreasing trend by day 5. His pancreatic enzymes were also elevated and a diagnostic ultrasound confirmed pancreatitis which was managed conservatively. Dobutamine was initiated as the vasopressors gradually tapered with good clearance of lactate and good peripheral perfusion. Serial 2D echo showed improvement in left ventricle (LV) function. He was continued on dialysis because of oliguria and recurrent pulmonary edema. Patient was weaned off VA ECMO successfully on day 6 and extubated onto high-flow nasal cannula on day 7. His liver and renal functions gradually recovered and coagulopathy reversed and he was discharged home on day 12 (Tables 1 and 2).

## DISCUSSION

Aluminum phosphide is a common suicidal agent in India and associated with very high mortality of about 80–90%. Aluminum phosphide poisoning can have a varied presentation and clinical course. The initial symptoms can be non-specific and include epigastric pain, vomiting, diarrhea, dizziness, and dyspnea.<sup>1,2</sup> The deadly phosphine gas that is generated as a result of its interaction with moisture or acid in the stomach results in inhibition of the cytochrome oxidase enzyme system in mitochondria due to which the body is unable to utilize oxygen for aerobic metabolism.<sup>1</sup> The ultimate dependence on anaerobic metabolism results in significant lactate accumulation and severe metabolic acidosis affecting the major organ systems. Cellular damage and hypoxia causes cardiotoxicity, resistant hypotension, lung injury, and liver failure which are the most common causes of mortality and morbidity after its poisoning. Cardiovascular complications including dysrhythmias, such as ventricular tachycardia and systolic heart failure, ranging from decreased cardiac function to complete cardiovascular

collapse are common in ALP.<sup>3–6,10,11</sup> The initial ECG for our patient showed ST elevation similar to acute presentations of myocarditis. He developed wide QRS complexes on day 2 of ECMO which was managed by magnesium, trimetazidine, and afterload reduction with nitrates.

Trimetazidine enhances glucose oxidation by inhibiting beta-oxidation of fatty acids, thus preventing further insult to the myocardium.<sup>18</sup> Lidocaine, magnesium, and amiodarone have been used to convert ventricular tachycardia to sinus rhythm in patients with ALP toxicity.<sup>4,7</sup> Magnesium acts as a cell membrane stabilizer in the myocardium and combats free radical stress due to phosphine.<sup>17</sup> Since there is no specific antidote available, ECMO has shown promise by acting as a rescue therapy providing cardiovascular support until the effect of the poison wears off and the end organs recover. ECMO has been well described for reversible cardiogenic shock in patients with ALP.<sup>3,8–11</sup> Hemodialysis has been described as treatment for acute renal failure and metabolic acidosis in ALP.<sup>13–15</sup> *N*-Acetyl cysteine, which was administered to our patient, has been described as treatment for cardiotoxicity secondary to oxidative stress in ALP and its use has been associated with a decreased hospital length of stay.<sup>12</sup> Acute pancreatitis and ischemic hepatitis are known complications and improved with cardiovascular support with ECMO. In our case, ECMO was initiated early for cardiovascular support in view of severe myocarditis and hemodynamic instability as the initial presentation was ST elevation myocardial infarction (STEMI) and severe left ventricular dysfunction. A high index of suspicion for ALP is necessary in these clinical presentation given the potential for rapid progression of multisystem organ failure, cardiovascular collapse, and death.<sup>2,10,16</sup>

## CONCLUSION

Aluminum phosphide poisoning has varied presentation and can be confused with cardiac emergency because of the ECG changes and myocardial dysfunction. There is no specific antidote for ALP and there is high risk of multiorgan failure resulting in acute kidney injury, liver injury, pancreatitis, and DIC with high mortality.

**Table 1:** Key laboratory variables with time course of illness

<i>Complete blood count</i>	<i>Day 1</i>	<i>Day 2</i>	<i>Day 3</i>	<i>Day 4</i>	<i>Day 5</i>	<i>Day 6</i>	<i>Day 7</i>	<i>Day 8</i>
Hemoglobin (g/dL)	15.9	8.8	8.3	9.1	9.8	9.0	9.2	10
Total WBC count (10 <sup>9</sup> /L)	11.7	10.5	4.7	4.4	6.6	12.2	14	13.6
Platelets (10 <sup>9</sup> /L)	324	72	89	83	65	80	85	125
<b>Renal function tests</b>								
Sodium (mEq/L)	137	144	144	143	141	138	140	138
Potassium (mEq/L)	4	4.4	4.1	4.6	4.1	4.7	4.3	3.5
Urea (mg/dL)	24	30	127	144	85	183	144	62
Creatinine (mg/dL)	1.1	1.3	5.1	5.3	3.3	6	4.8	2.1
<b>Ischemic hepatitis</b>								
Total bilirubin (mg/dL)	0.7	1.6	2.0	2.2	2.1	1.9		
Alanine transaminase (U/L)	31	1,152	7,515	5,310	1,838	441	841	76
Aspartate transaminase (U/L)	33	1,051	3,385	2,590	1,884	1,377	166	366
Prothrombin time (seconds)	14.7	24.3	22.3	19.3	15.4	13.4	15.4	20.5
International normalized ratio	1.12	1.91	1.74	1.5	1.18	1.02	1.18	1.59
Activated prothrombin time (seconds)	23.9	40.9	37.7	47.8	36	27.7	27	27
Fibrinogen (mg/dL)	270	98	110		170			293
<b>Acute pancreatitis</b>								
Amylase (IU/L)		1,120			987			
Lipase (IU/L)		174.3			289.6			

**Table 2:** Key clinical parameters with time course of illness

	<i>Day 1</i>	<i>Day 2</i>	<i>Day 3</i>	<i>Day 4</i>	<i>Day 5</i>	<i>Day 6</i>	<i>Day 7</i>	<i>Day 8</i>
Ejection fraction	25%	30%	35%		40%		50%	
Lactate levels (mmol/L)	8.8	4.6	2.5	2.8	1.7	1.4	1.6	1.2

Trimetazidine and magnesium help to suppress arrhythmia. Venoarterial ECMO provides an effective means of cardiovascular support until the poison is eliminated from the body. Our case emphasizes that early recognition and early institution of ECMO can save many young lives who succumb to toxic effects of this poison.

## CLINICAL SIGNIFICANCE

A high index of suspicion is required for ALP because of its confusing presentation similar to cardiac emergencies, given the potential for rapid progression and severe multiorgan toxicity. Fatal arrhythmias are common and can be successfully treated with trimetazidine and magnesium. Early recognition and early institution of ECMO is the key to a successful outcome.

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