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Successful Resuscitation Following Aluminum Phosphide (Celphos) Poisoning Complicated by Ventricular Tachycardia and Cardiogenic Shock: A Case Report

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ABSTRACT

Background: Aluminum Phosphide Poisoning is a highly lethal form of pesticide poisoning commonly seen in developing countries. It leads to severe metabolic toxicity, refractory shock, malignant arrhythmias, and high mortality. Survival following cardiac arrest is rare.

Case Presentation: We report a case of a 30-year-old male who presented with aluminum phosphide ingestion, severe hypotension, and altered sensorium. During hospitalization, the patient developed pulseless ventricular tachycardia requiring cardiopulmonary resuscitation and defibrillation. With aggressive intensive care management including ventilatory support, vasopressors, and supportive therapy, the patient survived and was discharged in stable condition.

Conclusion: Early aggressive resuscitation, timely defibrillation, and multidisciplinary critical care support can improve survival in severe aluminum phosphide poisoning complicated by cardiac arrest.

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1. INTRODUCTION

Aluminum Phosphide Poisoning is one of the most fatal pesticide toxicities in India with mortality rates ranging from 40–80%. The toxicity occurs due to release of phosphine gas, which inhibits mitochondrial oxidative phosphorylation leading to:

1. severe metabolic acidosis
2. cardiogenic shock
3. malignant arrhythmias

Cardiac complications such as Ventricular Tachycardia, Myocardial Depression, and Cardiac Arrest are common causes of mortality.

We present a rare case of survival following severe poisoning complicated by cardiac arrest.

2. CASE PRESENTATION

A 30-year-old male was admitted to the emergency department with a history of ingestion of Celphos (aluminum phosphide).

Initial Presentation

The patient presented with:

1. altered sensorium
2. recurrent vomiting
3. severe hypotension
4. Vital parameters at admission:
5. Blood Pressure: 72/52 mmHg
6. Glasgow Coma Scale: E2V1M2
7. bilateral sluggish pupils

8. Arterial blood gas analysis was performed, and due to poor neurological status and risk of airway compromise, the patient was intubated and mechanically ventilated.

Initial Management

Immediate management included:

1. gastric lavage with 3 liters normal saline
2. invasive monitoring
3. mechanical ventilation (volume control mode)
4. fluid resuscitation
5. Clinical Deterioration

On Day 2 of hospitalization, the patient suddenly developed:

1. cardiovascular collapse
2. loss of consciousness
3. Cardiac monitoring revealed pulseless ventricular tachycardia (~240/min).

Immediate DC cardioversion (150 J) was performed. However, the arrhythmia was not sustained, and cardiopulmonary resuscitation was initiated as per Advanced Cardiac Life Support protocol.

The patient received:

high-quality chest compressions
1 mg intravenous adrenaline
Return of spontaneous circulation was achieved.
Investigations
Laboratory Findings

Initial investigations showed:

Leukocytosis: TLC 20.61
Elevated liver enzymes:
AST 254
ALT 165
Procalcitonin: 1.79
Renal function remained relatively preserved during hospitalization.
Cardiac Evaluation

2D echocardiography revealed:

severe left ventricular systolic dysfunction
LVEF approximately 20–25%
mild mitral regurgitation
There was no evidence of intracardiac thrombus, vegetation, or pericardial effusion.
Radiological Findings

HRCT thorax showed:

inter/intralobularseptal thickening
patchy areas of consolidation
mild bilateral pleural effusion

Ultrasound abdomen showed:

mild ascites
mild hepatomegaly
Treatment
The patient was managed in the intensive care unit with:
mechanical ventilation
intravenous fluids
broad spectrum antibiotics
Meropenem
Metronidazole

Additional supportive therapy included:

antioxidants (Glutathione)

anticoagulants
proton pump inhibitors
Gradually, the patient's hemodynamic status improved.
Outcome
The patient was successfully extubated after stabilization and remained hemodynamically stable.
He was later shifted to the ward and discharged in stable condition.

3. DISCUSSION

Aluminum Phosphide Poisoning causes toxicity primarily through inhibition of mitochondrial respiration and generation of reactive oxygen species.
Major complications include:
Cardiogenic Shock
Ventricular Tachycardia
Metabolic Acidosis
Mortality remains high due to lack of a specific antidote. Management is largely supportive.
In this case, early airway control, rapid defibrillation, and adherence to ACLS protocols likely contributed to survival.

4. CONCLUSION

This case highlights that aggressive critical care management and prompt resuscitation can lead to survival even in severe aluminum phosphide poisoning complicated by cardiac arrest.

DECLARATIONS:

Conflicts of interest: There is no any conflict of interest associated with this study

Consent to participate: There is consent to participate.

Consent for publication: There is consent for the publication of this paper.

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