RESEARCH ARTICLE

Venoarterial Extracorporeal Membrane Oxygenation for Cardiogenic Shock in Aluminum Phosphide Toxicity

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Abstract

Introduction: Aluminum phosphide (ALP) toxicity causes high mortality, often implicated for suicidal purposes since it has no specific antidote. Release of phosphine upon ingestion leads to refractory cardiogenic shock and multiorgan failure, which are the highest predictors of mortality. Compared with conventional treatment, extracorporeal membrane oxygenation (ECMO) plays a significant role in such unsolved problems.

Materials and methods: Retrospective study of 9 cases from 2021 to 2022 with an undetermined number of tablets consumed. On examination, all patients had arrhythmias, hypotension, ejection fraction (EF 10–20%), and severe metabolic acidosis. We divided our patients into groups A and B based on the time taken for initiating ECMO. In group A (4 patients) VA-ECMO was initiated within 6 hours. Group B (5 patients) patients had a delay in arrival and the late decision worsened their condition and ECMO was initiated after 8 hours of ingestion.

Conclusion: Early initiation of ECMO seems to improve survival rates in ALP toxicity.

Keywords: Aluminum phosphide, Cardiogenic shock, Extracorporeal membrane oxygenation, Mortality, Toxicity.

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Introduction

Aluminum phosphide (ALP) is often implicated in suicidal purposes and has high mortality of 80–100%. Release of phosphine upon ingestion leads to refractory cardiogenic shock and end up with multiorgan failure.[1] Poor outcome is largely due to delay in arrival, diagnosis, comorbidities, doubtfulness in the minds of clinicians, and patient relations for decision-making.[2,3] Our single-center retrospective study found that conventional treatment for ALP poisoning can be supported by ECMO in earlier toxic periods, which may reduce mortality.

Materials and Methods

A Retrospective study of 9 cases from 2021 to 2022 with no known psychiatric illness admitted to our emergency department after alleged ingestion of ALP with suicidal intent. The number of tablets consumed, the time interval between arrival and ECMO initiation, total duration, the severity of preexisting comorbidities, and their complications were analyzed. On comparative time interval between earlier ECMO initiation, cohorts were divided into two groups respectively (Table 1).

Observation

On examination, all patients had mean arterial pressure (MAP <60 mm Hg), heart rate >110 b/min, saturation ≤95%, ECG of the patients shows ventricular arrhythmia and fibrillation, echocardiography revealed EF 10–15% with global hypokinesia. Arterial blood gas analysis showed severe metabolic acidosis with PH ≤7.0 and lactate ≥80 mg/dL. Inotropic supports like adrenaline, noradrenaline, and vasopressin were started along with magnesium sulfate, sodium bicarbonate, potassium chloride, and xylocard infusion. Upon early resuscitation in group A, VA-ECMO was initiated within 6 hours of ingestion. Femoral artery and vein were percutaneously cannulated and an 8Fr sheath was placed for distal limb perfusion. Inotropes were titrated for maintaining target pressure. Bedside echo and doppler scan were done in alternate hour for monitoring EF and peripheral blood flow. Three patients developed acute kidney injury (AKI) due to toxic severity and required sustained low-efficiency dialysis (SLED) (Fig. 1) with or without fluid removal based on volume status and negative pressure of the access line. One patient had undergone a left heart vent via right superior pulmonary vein (RSPV) for myocardial stunning (Table 2). Despite the intensive treatment, one patient died because of irreversible brainstem injury.

In group B, delay in arrival and late decision worsened the condition and VA-ECMO was initiated after 8 hours of ingestion. Most of them required intermittent cardioversion (upto 40 hours) during ECMO support, In this, one patient was ECMO retrieval and required LV vent for myocardial distension. Arrhythmia

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Table 1: HTN – hypertension, DM – diabetes mellitus, AKI-acute kidney injury

	Group A (n = 4)	*Group B (n = 5)*
Tablets	Fatal dose	Fatal dose
Age	29–45 years	35–60 years
Gender	Male	Male
ECMO initiation	Within 6 hours	>8 hours
Suspected mortality	60%	80%
EF	10–20%	10–15%
Past history	HTN	HTN, DM, AKI

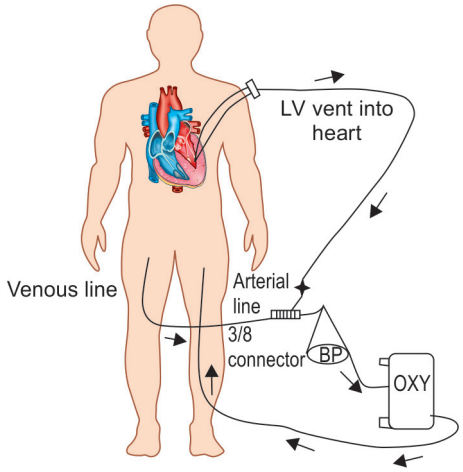

Fig. 1: LV vent diagrammatic representation
BP, centrifugal pump; OXY, oxygenator

Table 2: Special intervention

	Group A	*Group B*
LV vent	1 patient	1 patient
SLED	3 patients	4 patients
Retrieval	–	1 patient

and ventricular fibrillation settled gradually and decannulated within 3 days, and discharged at 28th day of his admission. Four patients required SLED for renal failure (Table 2) but succumbed subsequently. ECMO was discontinued in one patient on insistence of family after poor prognosis and critical condition was explained. Multiple complications arose in both groups and all were treated as per protocol. After 6–12 months of follow-up range all the survivors of both groups did well with normal organ function.

Discussion

Aluminum phosphide is the most common poison in India owing to easy availability and cheaper cost. Phosphine is a protoplasmic poison, which disrupts mitochondrial oxidative phosphorylation, inhibit cytochrome C oxidase, is lethal to cell enzymes and leads to cellular hypoxia.[4,6] The clear mechanism is multifactorial and includes myocardial damage, peripheral vasodilation, fluid loss, cellular energy, or cytokine-mediated dysfunction.[1,5] Since there is no specific antidote, treatment was based on general poison management like early resuscitation, gastric lavage with coconut oil, fluid administration, and inotropic supports to maintain normal hemodynamics[7] (Table 3).

Table 3: Target maintenance in ECMO

	Target
Pulse pressure	>10 mm Hg
MAP	>60 mm Hg
Cerebral saturation	Not less than 50%

In case of refractory cardiogenic shock VA-ECMO was initiated with intent of avoiding organ damage and providing adequate time for heart to rest and recover.[8,9] All the patients were under strict monitoring including near-infrared spectroscopy (NIRS) and supports were titrated accordingly. Phosphine causes damage to renal tubules, parenchymal cells which result in renal dysfunction.[10] Most patients developed oliguria (output <5 mL/hour) and required SLED with or without fluid removal for several days even after decannulation till the toxic metabolites are excreted. One patient from each group had severe LV distension with EF <15% multidisciplinary team inputs were obtained and patient shifted to OT immediately for insertion of LV vent, 16Fr vent catheter was placed through RSPV and an hourly drain was noted. Within a few days, left ventricular function started improving with EF >25% and normal aortic valve opening, then gradually weaned from ECMO with minimal inotropic support and decannulated uneventfully within 5 days, and finally discharged with normal organ function.[11] Despite intensive treatment, multiple complications aroused, and one patient from group A, underwent fasciotomy for compartment syndrome and succumbed due to irreversible shock and septicemia. Three patients of group B with preexisting comorbidities further exacerbated the toxicity and died because of severe lung consolidation and septicemia (Table 4). This led us to believe that earlier initiation of ECMO might mitigate cardiovascular and other organ adverse effects, thereby assist in early recovery and favorable survival rates in this highly mortal condition.

Limitations

This is a single-center retrospective study and has a small sample size. Thus, it is likely to have biased outcome. Patients who were not reverted even after receiving multiple shock at initial management and financially not affordable for further treatment and ECMO support were excluded in this study. Further research with large number of patients is required to validate this result.

Conclusion

There are no definitive guidelines indicating appropriate timing for ECMO initiation in poisoning cases. Decision-making depends upon the family's willingness and the clinical judgement of the multidisciplinary team concerment.

It is important to understand that ECMO should be initiated before irreversible end organ damage occurs. Earlier ECMO as bridge to recovery, a timely intervention for the refractory cardiogenic shock seems to increase survival rate in aluminum phosphide toxicity cases.

Table 4: Comparison of outcome between groups

	Group A		*Group B*	
ECMO initiation	<6 hours		>8 hours	
Mortality	Upto 70%		>80%	
Total ECMO hours	≤80 hours		125 hours	
Comorbidities	30%		30%	
ICU stay	<10 days		16 days	
Hospital stay	18 days		28 days	
Discharge alive	3 patients		1 patient	
Survival rate	80%		30%	
Post-ECMO complications	–		Bilateral watershed infarcts – left hemiparesis	
	Admission	*Discharge*	*Admission*	*Discharge*
EF	10–20%	40%	10–15%	>35%
Arterial blood gas and electrolytes	HCO_3^-: <15	HCO_3^-: ≥25	HCO_3^-: <10	HCO_3^-: 23
	lactate: >75	Lactate: <17	Lactate: >90	Lactate: 18
	K^+: upto 5.5	K^+: upto 4	K^+: >5.0	K^+: 3.9
	Cr:>1.5	Cr: 1.3	Cr: >2.0	Cr: 1.5

Cr, creatinine (mg/dL); EF, ejection fraction; HCO_3^-; Lactate (mg/dL); K^+, Potassium (mmol/L); Sodium bicarbonate (mmol/L)

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