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Cardiac and Metabolic Profile of High-Risk Phosphine Intoxicated Patients Supported by Extracorporeal Membrane Oxygenation (ECMO): Cairo University Hospitals Experience.

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Abstract

Background: *Phosphide poisoning is highly fatal with no antidotes available to date. It causes profound, yet reversible, cardiogenic and circulatory collapse that may be refractory to conventional therapy. Veno-arterial extracorporeal membrane oxygenation (V-A ECMO) may temporarily bridge these patients to recovery with good but limited evidence.*

Patients and Methods: *This is a tertiary-care, single-center, observational study examining critically ill phosphide intoxicated patients receiving ECMO support in Cairo University from May 2022 to August 2023.*

Results: *We included 21 patients during the study period; 20 with aluminum phosphide and one with zinc phosphide intoxication. They had a mean age of 27.4 ± 10.52 years and no major comorbidities. Despite supportive therapy, they had a mean arterial pressure of 47.8 ± 11.7 mmHg and 57% suffered cardiac arrest before ECMO cannulation and extracorporeal cardiopulmonary resuscitation was deployed in 33% of the patients. Marked myocardial dysfunction dominated before ECMO with a mean left ventricular ejection fraction of 22.4% ± 14.2, which reached 49.37% ± 7.69 by decannulation and normalized on discharge. After ECMO runs lasting 93.6 ± 35.5 hours, 15 patients (71%) survived to hospital discharge. Anemia (76%), thrombocytopenia (95%), and cannula site bleeding (33.3%) were the most reported ECMO-related complications. Septic shock, multiorgan failure, and severe neurologic dysfunction were the causes of death.*

Conclusion: *In light of severely deranged cardiac and hemodynamic profiles of phosphine intoxicated patients, ECMO can provide a sufficient temporary support till recovery.*

Keywords: *Extracorporeal membrane oxygenation, cardiogenic shock, aluminum phosphide poisoning.*

1. INTRODUCTION

Aluminum and zinc phosphides, commonly utilized for pest and rodent control in many developing countries, have paradoxically emerged as agents of significant public health concern due to their association with deliberate self-poisoning leading to alarmingly high mortality rates between 40% and 91%. [1] These compounds, upon ingestion, release phosphine gas, which rapidly leads to multiorgan failure. [2] While the specific toxicological mechanisms of phosphine are not fully mapped out, existing theories suggest mitochondrial dysfunction, vascular integrity disruption, hemolysis, and direct cardiac toxicity as key factors. [2–4]

In clinical settings, the primary fatal complications of aluminum and zinc phosphide intoxication are cardiovascular in nature, including circulatory collapse, metabolic acidosis, cardiogenic shock, and severe arrhythmias. [1, 5, 6] Treatment approaches are predominantly supportive, focusing on stabilizing hemodynamics with fluids, vasopressors, and inotropes, supplemented by agents such as magnesium sulfate, sodium bicarbonate, and various antioxidants despite limited robust evidence. [7]

Emerging evidence points to the potential life-saving role of veno-arterial extracorporeal membrane oxygenation (V-A ECMO) in managing severe cases of aluminum phosphide poisoning. [8–13] By providing circulatory support and enhancing tissue perfusion, ECMO can offer a critical window for the patient's body to metabolize and excrete the toxin, thereby improving survival outcomes. However, ECMO is not without its risks, including bleeding, limb ischemia, and infections, which complicate its application in clinical practice. [14, 15]

Given that these patients are often young and previously healthy, ECMO presents a particularly valuable therapeutic option. However, clear guidelines for the optimal timing of ECMO initiation based on

clinical, metabolic, and echocardiographic parameters are lacking, and pre-ECMO prognostication remains a challenge.

This study aims to explore the role of ECMO in the treatment of high-risk phosphine-intoxicated patients, analyzing the procedural aspects, patient characteristics, outcomes, and complications associated with its use within the critical care department of Cairo University.

2. METHODOLOGY

Study Design and Setting: This is a tertiary care, single-center observational study conducted at the Critical Care Department of Cairo University, covering the period from May 2022 to August 2023. The study was driven by the high mortality rates of aluminum phosphide (ALP) intoxicated patients and involved collaboration with the National Environmental and Clinical Toxicological Research center (NECTR) at Cairo University to provide ECMO support for high-risk patients.

Ethics Approval and Consent: The study received approval from the Ethical Committee Review Board of the Faculty of Medicine, Cairo University. Informed written consent was obtained from patients' relatives before enrollment.

Inclusion and exclusion criteria:

All phosphide-intoxicated patients receiving ECMO support during the study period were included. V-A ECMO was offered to high-risk patients meeting criteria such as severe lactic acidosis unresponsive to fluid resuscitation, MAP < 65 mmHg with increasing doses of vasopressors, impaired cardiac contractility (LVEF ≤ 35%), life-threatening cardiac arrhythmias, and refractory cardiac arrest (no return of spontaneous circulation after >10 minutes of cardio-pulmonary resuscitation (CPR)). ECMO support was not provided to patients with extremes of age, prolonged CPR without adequate perfusion, irreversible multi-organ dysfunction, or non-recoverable comorbidities.

Data collection

Data collection encompassed toxin-related information (type and dose, mode of administration, time to medical contact and ECMO support), baseline hemodynamic, laboratory, electrocardiographic, and echocardiographic parameters at cannulation, and disease severity scores. Regular follow-up of hemodynamic and cardiac parameters was conducted. The primary outcome was mortality, while secondary outcomes included ECMO run and hospital stay durations and recorded complications.

Management

Initial supportive care at the toxicology center involved gastric lavage, maintaining adequate perfusion through fluid resuscitation and vasopressors, sodium bicarbonate, electrolyte correction, magnesium sulfate, and acetylcysteine. High-risk patients were promptly referred to the ECMO unit at the Critical Care department for timely ECMO support. Percutaneous cannulation was performed using the Seldinger technique, predominantly via the femoro-femoral approach. Left ventricular venting with intra-aortic balloon pump was opted for whenever signs of overload were detected and upgrading to hybrid support was done in case of severe respiratory failure. Systemic anticoagulation was instituted targeting an aPTT of 50-70 seconds.

Vasopressors were gradually weaned post-ECMO initiation while maintaining adequate perfusion pressure. ECMO support was gradually weaned upon achieving satisfactory parameters, followed by surgical decannulation. Post-decannulation, patients were monitored for complications and received psychiatric assessments. Once cleared from toxicological and medical perspectives, patients were discharged.

Statistical analysis

The data with continuous variables were expressed as mean with standard deviations if normally distributed and median with minimum and maximum if not. The categorical variables were described as percentages. If the continuous data followed a normal distribution, then the mean was compared using Student's t-tests while if it was skewed then the medians were compared using Mann–Whitney U-test. For categorical variables, either Fisher's exact tests (if the cell value is <5) or chi square test was used.

Correlations between quantitative variables were done using Spearman correlation coefficient. All probability values were 2-sided, and differences with p values of <0.05 were considered statistically significant.

3 RESULTS

We included 21 patients with a mean age 27.4 ± 10.52 years, no major comorbidities, and 52.4% were males. All patients were intoxicated with suicidal intention using aluminum phosphide (ALP) in 20 patients with a mean dose of 2.56 ± 1.17 grams and zinc phosphide (ZnP) in only one patient with unidentified dose. At time of ECMO cannulation, patients had a mean MAP of 47.8 ± 11.7 mmHg despite receiving two vasopressors/inotropes in 13 patients (62%). Other than severe metabolic acidosis, patients showed mild elevations in INR, sodium, and CK with mild hypokalemia before cannulation. Most patients developed pulmonary congestion before ECMO (15, 71.4%) and had mild renal impairment (14, 66.7%). Ten patients had impaired liver functions before ECMO.

Outcomes

Out of 21 patients, 15 (71%) survived to ICU/hospital discharge without major disabilities while 5 died during the ECMO run and 1 died after decannulation. The causes of death were septic shock, multiorgan failure, and severe neurological dysfunction.

Non-survivors had a significantly lower mean arterial pressure (MAP), required higher noradrenaline doses, had cardiac arrest for significantly longer durations before ECMO, and a greater proportion of them were cannulated as ECPR (5; 83.3%). Neurological dysfunction and severe AKI were more common among non-survivors.

Survivors had a significantly longer ICU stay duration (14.53 ± 9.16 days vs. 3.92 ± 1.96 days; p = <0.001). ECMO runs lasted 93.57 ± 35.5 hours with no significant difference between survivors and non-survivors. The duration of the ECMO run only correlated significantly with the development of ventricular arrhythmias during ECMO, ST segment elevation, and the duration of mechanical ventilation.

Metabolic and lactic acidosis

The majority of patients recorded lactate >15 mmol/L (17, 80.9%). Lactic acidosis preceded myocardial dysfunction where it started resolving in some patients when cardiac affection peaked. That was more prominent with smaller ingested doses. Metabolic acidosis resolved after 14.1 ± 9.4 hours after ECMO initiation with only one patient that died with persistent metabolic acidosis. It resolved earlier among survivors but not statistically significant. Lactate level normalized 36.4 ± 17.58 hours after ECMO initiation, yet it remained elevated in 4 patients.

Cardiac parameters

Long QTc was the most common ECG finding (20; 95.2%). ST segment changes, either elevation or depression, were present in all patients, yet only 13 (61.9%) had ST segment elevation and it resolved earlier than ST segment depression. Tachyarrhythmias were more common than bradyarrhythmia (95% vs. 14.3%), and their frequency declined with cardiac function improvement. Cardiac arrest frequency on ECMO was similar to before ECMO but with longer duration.

Patients suffered severe cardiac dysfunction before ECMO initiation, yet right ventricular affection was less pronounced. They showed marked improvement in terms of both right and left ventricular functions with EF = 49.37% ± 7.69, and TAPSE = 19.35 ± 2.9 mm at time of decannulation. All patients had normal left and right ventricular dimensions during the run and only one patient developed moderate mitral regurgitation. Echocardiographic parameters returned to nearly normal levels on discharge except for a mild pericardial effusion in 10 (66.7%) patients.

CK was only mildly elevated at time of cannulation, and it showed a delayed peak level compared to echocardiographic parameters with 9/17 patients reaching a peak level on day 3 from ingestion. Nevertheless, fluctuations due to limb ischemia and rhabdomyolysis affected its levels. ProBNP results were more accurate in the latter cases, but it wasn't routinely measured for all cases.

IABP was inserted for two patients on day 4 and 5 of the ECMO run for LV unloading due to delayed myocardial recovery. ECMO was removed 48 hours and 12 hours respectively after IABP insertion while keeping IABP in place. Only the first patient survived.

Seven (33.3%) patients received levosimendan 38.1 ± 43.17 hours after ECMO cannulation due to severe myocardial depression as a means to enhance recovery and facilitate ECMO weaning. The time from cannulation to levosimendan administration did not correlate with the duration of the ECMO run (p=0.071). In fact, the use of levosimendan was associated with statistically significantly longer ECMO runs, and that only remained true for the survivors subgroup.

Other organ dysfunction parameters and ECMO-related complications:

Pneumonia occurred in 14 (66.7%) patients, and it led to ARDS in 8 (38.1%) patients. Only 2 (9.5%) patients developed pulmonary embolism. Four patients developed irreversible central neurological damage during the ECMO run due to hemorrhagic stroke, subarachnoid hemorrhage, and prolonged cerebral hypoperfusion. One patient suffered unilateral lower limb weakness after decannulation which resolved spontaneously.

Renal impairment was mostly mild (10; 47.6%) whereas severe AKI only occurred in 5 patients (23.8%) and only one patient required hemodialysis due to rhabdomyolysis for 14 days with gradual recovery of renal functions thereafter. Liver impairment was noted in 18 (80.9%) of patients during the ECMO run with liver enzymes peaking after 2.5 ± 1.42 days.

Bleeding during cannulation occurred in 5 patients, vascular injury occurred in 2 patients, and cannula malposition in 3 patients. Two patients suffered limb ischemia where the limb was saved by urgent exploration in one patient and the other was amputated. Thrombocytopenia occurred in all but one patient; it preceded ECMO cannulation in two of them and it occurred on the first day of the run in 13 patients. Three patients suffered severe bleeding due to sutures disruption after decannulation and they required surgical intervention for ligation, repair, or bypass grafting.

Toxin dose-dependent disease severity

Patients intoxicated with smaller ALP doses had a slower disease progression and required ECMO support at a later time compared to higher doses. These findings, however, did not reach statistical significance. Higher doses led to worse hemodynamics and echocardiographic parameters but only arrhythmia duration before ECMO and ECPR were statistically significant.

Table 1: Baseline characteristics, hemodynamic and echocardiographic parameters

Characteristics	Total (*n* = 21)	Survivors (*n* = 15)	Non-survivors (*n* = 6)	P value
Age (years)	27.4 ± 10.5	27.3 ± 11.04	27.5 ± 10.07	0.85
Male sex *n* (%)	11 (52.4%)	9 (60%)	2 (33.3%)	0.361
ALP dose (gm)	2.56 ± 1.17	2.31 ± 0.9	3.3 ± 1.64	0.26
Time to first medical contact (hrs)	3.71 ± 2.22	3.53 ± 1.81	4.17 ± 3.19	0.910
Decontamination	17 (80.9%)	14 (93.3%)	3 (50%)	0.053
Time to decontamination (hrs)	4.28 ± 2.02	4.20 ± 1.90	4.67 ± 3.06	0.912
Time to admission in our ICU (hrs)	11.81 ± 5.9	12.53 ± 6.73	10.00 ± 2.61	0.850
Time to ECMO (hrs)	16.31 ± 8.23	17.37 ± 9.21	13.67 ± 4.63	0.569
APACHE II total score	15.57 ± 6.2	14.27 ± 5.64	18.83 ± 6.85	0.178
SOFA score	7.86 ± 2.35	7.47 ± 2.5	8.83 ± 1.72	0.235
SAVE score	-0.52 ± 5.5	0.2 ± 6.01	-2.33 ± 3.78	0.47
Vasoactive-inotropic score	167.6 ± 105.33	144.07 ± 105	226.5 ± 87.67	0.055
MAP (mmHg)	47.8 ± 11.7	51.9 ± 9.35	37.5 ± 11.29	0.011

HR (bpm)	124.8 ± 27.2	132.5 ± 15.84	105.83 ± 40.67	0.055
Noradrenaline dose (mcg/kg/min)	1.36 ± 0.72	1.12 ± 0.69	1.94 ± 0.39	0.014
Cardiac arrest *n* (%)	12 (57.1%)	7 (46.6%)	5 (83.3%)	0.178
ECPR *n* (%)	7 (33.3%)	2 (13.3%)	5 (83.3%)	0.006
LVEF (%)	22.43 ± 14.23	27.13 ± 12.42	10.67 ± 12.06	0.003
TAPSE (mm)	12.62 ± 5.86	14.27 ± 6.01	8.52 ± 2.81	0.023
pH	7.22 ± 0.1	7.24 ± 0.1	7.16 ± 0.08	0.055
HCO3 (mmol/L)	12.77 ± 3.76	13.84 ± 3.66	10.08 ± 2.66	0.029

Data presented as mean ± SD or n (%).

Table 2: Organ dysfunction and ECMO-related complications

Complications	**Total (*n* = 21)**	**Survivors (*n* = 15)**	**Non-survivors (*n* = 6)**	**P value**
Complications during ECMO run				
CVA	4 (19%)	0	4 (66.7%)	0.003
Severe AKI	5 (23.8%)	1 (6.7%)	4 (66.7%)	0.011
GIT Bleeding	1 (4.7%)	0	1 (16.7%)	0.286
Cerebral bleeding	1 (4.7%)	0	1 (16.7%)	0.286
Cannula site bleeding	7 (33.3%)	3 (20%)	4 (66.7%)	0.12
Cannula site infection	0	0	0	
Limb ischemia	2 (9.5%)	1 (6.7%)	1 (16.7%)	0.5
Anemia requiring blood transfusion	16 (76.2%)	12 (80%)	4 (66.7%)	0.598
Thrombocytopenia	20 (95.2%)	14 (93.3%)	6 (100%)	1
Change of circuit	0	0	0	
Complications after decannulation				
Cannula site bleeding	3 (14.3%)	3 (20%)	0	0.526
Cannula site infection	6 (28.6%)	6 (40%)	0	0.123
DVT	4 (19%)	4 (26.7%)	0	0.281

Figure 1: Time to normalization of lactate levels

4.DISCUSSION

V-A ECMO use in acute intoxication has been rising with good outcomes with a systematic review recording 68.8% survival to hospital discharge which drops to 50.9% when ECPR was the initial indication. [17] Out of 475 V-A ECMO patients included in that systematic review, 45 patients received ECMO support secondary to ALP intoxication with only 31% mortality. Apart from one comparative study including 35 patients, the rest of the available literature is limited to case reports with all positive outcomes. That raises suspicion for a high risk of publication bias.

Our study is a single-center retrospective study that included 20 aluminum phosphide and one zinc phosphide intoxicated patients who received ECMO support where 71% of patients survived to hospital discharge. That is comparable to the rates reported by Maier et al. for ALP and all cardiotoxic agents.[17] In the largest study published to date comparing conventional therapy alone and in addition to ECMO for high-risk ALP intoxicated patients, mortality rates dropped from 84% to 40% with ECMO.[8] That high-risk group was defined by having (1) LVEF ≤ 40% and (2) severe metabolic acidosis (pH ≤ 7) and/or persistent shock despite inotropic support.

In our study as well as in Mohan et al., all ECMO-receiving patients were young, had severe metabolic acidosis and severely depressed cardiac function with comparable numbers between both studies [8]. Blood pH was only numerically different between survivors and non-survivors in both studies, yet in ours, serum HCO3 reached statistical significance.

In our study, pre-ECMO hemodynamic and echocardiographic parameters predicted mortality. Non-survivors had significantly lower MAP, were on higher doses of norepinephrine, and had significantly lower LVEF and TAPSE at time of cannulation. Interestingly, non-survivors had lower heart rates at time of cannulation, yet that didn't reach statistical significance. In Mohan et al., however, LVEF was only numerically lower among non-survivors, and they did not report hemodynamics.

These derangements and their effect on mortality may be explained by the fact that 83% of the non-survivors had prolonged cardiac arrest before ECMO and received ECPR compared to only 13% of the survivors. The duration of cardiac arrest also correlated well with mortality.

In our study, none of the disease severity scores were good predictors of mortality unlike Mohan et al. where SOFA score was significantly higher among non-survivors. That could be attributed to their use of the worst score calculated during the ICU stay which would be higher with disease progression and developing complications resulting in death.

ALP lethal dose is as low as 150 – 500 mg with subsequent increase in disease severity and mortality with higher ingested doses and therefore the amount of phosphine gas released. In our study as well as in Mohan et al., ingested doses were comparable and did not affect mortality on ECMO. However, we found that ALP dose significantly correlated with developing cardiac arrest before ECMO and the duration of arrhythmias after cannulation. Nonetheless, the small number of patients and having mostly similar doses limited the significance of more correlations.

The relation between ALP ingested dose and the clinical response and rate of deterioration is not always proportional with several intervening factors. These may include using tablets that were pre-exposed to humidity, vomiting, drinking water, and using appropriate decontamination measures and resuscitation within a short period after exposure. Though the delay in seeking medical advice was found to predict mortality in some studies, it wasn't statistically significant after ECMO application.[18, 19]

Following ECMO application, severe neurological and renal complications and non-resolving lactate were associated with mortality. In Mohan et al. as well, non-survivors were more likely to develop severe renal impairment though with higher incidence than our study (51% vs. 24% for the total ECMO population).

Though ALP causes severe hemodynamic and cardiac dysfunction, these effects are short-lived. Most of our patients had a straightforward course with gradual improvement of hemodynamics and cardiac functions till decannulation. LVEF recovered to an average of 49% by the time of decannulation and normalized by hospital discharge.

In addition to vasopressors and inotropes, seven patients received levosimendan to assist cardiac recovery. Levosimendan has proven beneficial facilitating V-A ECMO weaning,[20] yet its role in ALP intoxication has only been studied in rats.[21] In our patients, levosimendan use was safe, yet it did not affect mortality and was, in fact, associated with longer ECMO runs. That is probably due to its use in some cases as a rescue therapy because of delayed cardiac recovery. Given the reversible nature of ALP toxicity, linking cardiac recovery to levosimendan would be difficult and needs further studying. Additionally, with the generally short runs, the benefit of levosimendan may be limited to complicated cases.

We used IABP for two of our patients to assist LV venting and decrease afterload with successful outcome in only one of them. Dukhin et al. also reported successfully using IABP in addition to ECMO for one patient while Mehrpour et al. managed to use it independently without ECMO for another patient with ALP intoxication.[13, 22] Levosimendan and IABP are cheaper options compared to ECMO and using them in order to avoid and limit ECMO use should be studied further.

Our patients had ECMO runs of 93.5 ± 35.5 hours which is comparable to the general intoxicated population on V-A ECMO in the systematic review by Maier et al.,[17] but is longer than in Mohan et al. (42.1 and 67 hours for survivors and non-survivors, respectively).[8]

Determining which patients need ECMO and the proper timing to initiate support would be subject to great debate. In general, early ECMO application was associated with better results,[23] but whether that applies to intoxicated patients remains unclear. Weighing the potential benefits of ECMO and associated risks and complications is a must. Defining delayed and irreversible cases as well needs good judgement and further studying so as not to waste resources.

5.STUDY LIMITATIONS

This was a single-center observational study with no matched controls which would account for several confounding factors and prevents data generalizability. Additionally, our experience managing these patients grew exponentially with each patient which could have led to some changes in the management protocols over time.

6.CONCLUSION

Metal phosphide intoxications were effectively managed by ECMO with complete recovery in most cases. However, weighing the potential risks of ECMO is a must. Further studies are still needed to explore the optimal initiation timing and to better validate the results with controlled groups.

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