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Cardiogenic Shock Due to Aluminum Phosphide Poisoning Treated with Intra-aortic Balloon Pump: A Report of Two Cases

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Abstract
The mortality rate from aluminum phosphide (AlP) poisoning is as high as 70–100%, with refractory hypotension and severe metabolic acidosis being the two most common presentations in this poisoning. As this poisoning has no specific antidote, treatments revolve around supportive care. Cardiogenic shock created by toxic myocarditis is considered the main cause of mortality in these patients. Meanwhile, the intra-aortic balloon pump (IABP) has been suggested for the treatment of cardiogenic shock. This article reports the successful treatment of cardiogenic shock caused by AlP poisoning in a 17-year-old man and a 21-year-old woman using the IABP procedure.

Keywords Aluminum phosphide · Phosphine · Poisoning · Cardiotoxicity · Cardiogenic shock · Intraaortic balloon pump

Introduction

Aluminum phosphide (AlP) is employed as a pesticide for both outdoor and indoor purposes [1–5]. In Asian countries, the substance is better known as the "rice tablet" as it is frequently used to protect rice and grains from rodents and other pests [6]. The tablets have been used in many suicidal attempts in these countries [3, 5], and an increase in Iran has been observed in recent years [5, 7, 8]. The mechanism of toxicity is postulated to be related to the release of phosphine gas leading to oxidative stress and free radical formation, and cytochrome oxidase inhibition [9, 10]. The mortality rate from AlP poisoning is as high as 70–100%, where refractory hypotension and severe metabolic acidosis are the two most common presentations in this poisoning [7]. AlP-associated death is mostly induced by refractory hypotension and cardiogenic shock [10–12].

Regrettably, there is no specific antidote, and treatments merely stand as supportive measures such as fluid resuscitation and vasoactive agents [13]. This case series describes the successful application of intra-aortic balloon pump (IABP) for the treatment of AlP-induced cardiogenic shock.

Case Presentation

Case 1

A 17-year-old man referred to a healthcare facility after intentionally ingesting one tablet of aluminum phosphide. Before this suicide attempt, he was healthy without any significant past medical history and did not have any cardiac problem. Upon presentation 2 h after ingestion, his vital signs included blood pressure (BP) 110/70, pulse rate (PR) 96, a saturation of peripheral oxygen (SPO_2) 97%, and Glasgow Coma Scale (GCS) 15. He was alert and oriented but had complaints of dizziness, nausea/vomiting, coughing, and muscle weakness. Electrocardiogram (ECG) showed sinus tachycardia and incomplete right bundle branch block with no ST-T changes (Fig. 1). Upon admission to the intensive care unit (ICU), the echocardiogram

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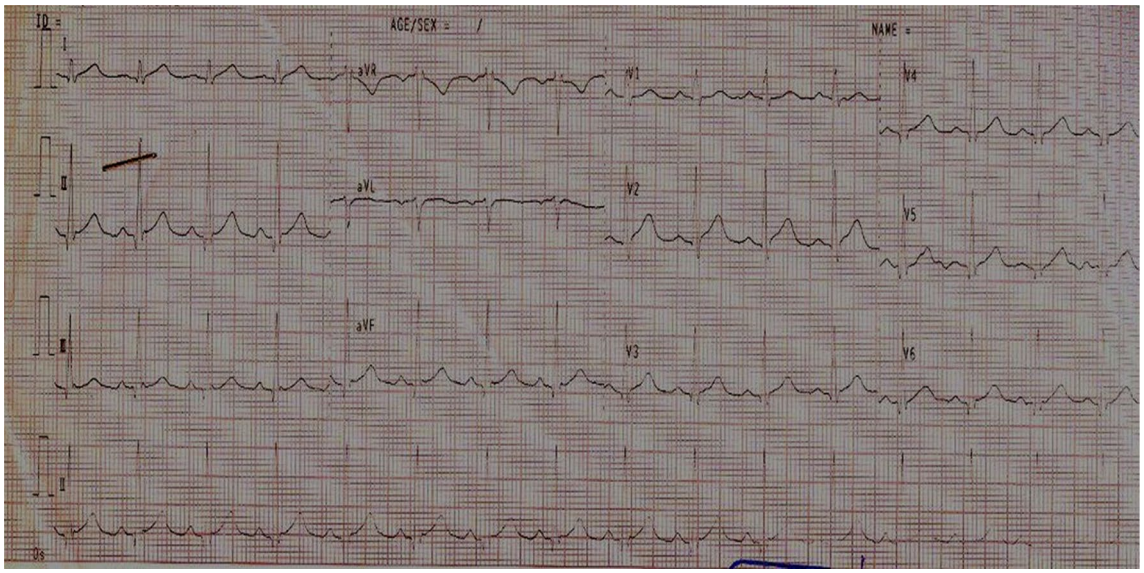

Fig. 1 Electrocardiogram (ECG) of case 1: sinus tachycardia and incomplete right bundle branch block with no ST-T change

revealed an ejection fraction (EF) of 55%. Approximately 6 h after presentation, his blood pressure began to drop despite intravenous fluid therapy (Table 1). Dopamine was initiated and titrated to 10 µg/kg/min, followed by norepinephrine 5 mg/kg/min. Hypotension persisted and repeated echocardiogram demonstrated an EF of 35–40% characterized by a normal left ventricle (LV) and right ventricle (RV) size, as well as moderate systolic dysfunction with severe hypokinesis of the lateral and inferior wall.

On hospital day 3, the patient had worsening respiratory distress whose oxygen saturation fell to 75%. Repeat ECG indicated sinus tachycardia and ST-elevation in V5–V6 as well as increased QRS duration and long QT (Fig. 2). Echocardiography was performed, where EF was 15–20% with moderate LV enlargement with severe LV dysfunction, mild RV enlargement, mild mitral and tricuspid valve failure, severe hypo-kinesis of the anterior and inferior wall and septum, severe RV dysfunction, and PAP = 25–30. Due to refractory cardiogenic shock, intra-aortic balloon pump was inserted.

During hospitalization, on day 2, there was an increase in the patient's liver enzymes aspartate aminotransferase (AST) = 170 (U/L) and alanine aminotransferase (ALT) = 146 (U/L). On day 3, patient's liver enzymes were AST = 1031 (U/L); ALT = 558 (U/L) and impaired coagulation was observed (INR = 3.08 and PT = 22.9). Also, the patient had mucosal bleeding, so he received treatment with Fresh Frozen Plasma (2 U every 6 h) and vitamin K (Table 2).

On the seventh day, the patient was normotensive. Norepinephrine was discontinued afterward.

Also, thanks to improving the blood pressure (BP): 100/70 on day 8, treatment with losartan, spironolactone, and then carvedilol was started.

On day 8, the echocardiography examination showed EF = 30%, normal LV and RV size, moderate to severe LV systolic dysfunction, and no pericardial effusion. He became gradually alert, and vital signs became stable. Hence, the patient's balloon pump removed on 8th day.

The patient had pneumonitis on this day, so antibiotic treatment was started. Also, medication for treatment of heart failure continued.

On day 10, the patient was extubated. The diet initiated and was tolerated. ECG was normal. Also, echocardiography showed EF = 45%. Mucosal bleeding was recovered on day 11.

He was transferred to the ward on the 12th day. Finally, on the 18th day, after psychiatric counseling, the patient was discharged from the hospital with medication orders of carvedilol 3.125 twice daily and losartan 12.5 mg daily. He was suggested to be visited by a cardiologist 2 weeks after discharge.

Case 2

In a self-harm attempt, a previously healthy 21-year-old woman ingested 1.5 tablets of ALP (4.5 g). Within 30 min, she experienced nausea, vomiting, drowsiness, and fatigue. Five hours after ingestion, she presented to the emergency department with hypotension and vital signs as follows: pulse rate: 112, respiratory rate: 22, blood pressure: 90/50, temperature: 37 (°C), and GCS: 15. In cardiac monitoring, wide complex tachycardia was observed.

Table 1 Clinical and laboratory finding of case 1

Laboratory tests/time after ingestion	On admission	2 h	6 h	12 h	24 h	48 h	3rd day	4th day	5th day	6th day	7th day	14th day
Blood pressure (mmHg)	110/70	96/65	83/41	91/32	90/38	101/63	80/36	116/56	104/64	112/65	132/66	
Pulse rate (bpm)	96	75	104	120	125	116	134	119	107	113	101	
Respiratory rate (pm)		17	18	25	24	SIMV	SIMV	SIMV	SIMV	SIMV	SIMV	14
O_2 saturation (%)	97%											
pH	7/417	7.37	7.29	7.20	7.25	7.38	7.42	7.42	7.45	7.41	7.42	7.57
$PaCO_2$ (mmHg)	28.1	28.3	29.3	24.6	10	20	56	48	42.4	41.3	40.3	26
PaO_2 (mmHg)	61.5	65	91.1	146.6	98.3	98.8	105.2	65	69.2	60	125.6	38
Serum HCO_3 (mmol/L)	18.1	16.3	14.1	9.7	2.1	12	36.2	31.3	29.8	26.1	26.3	23
WBC (10^3 μL)	9.8						7.6	7.5	7.5	8.9	7.5	
Hb (g/dL)							9.4	9.1	8.8	8.7	8.3	
Platelet count (10^3 μL)	255						148	90	89	88	120	
Na (meq/L)	141				150	153	153	150	152	146	146	
K (meq/L)	3.9				3.7	2.8	3.4	4.5	4.7	4.8	4.9	
Prothrombin time (s)	15				24	42	38.2	19.7	21	19.2	18.4	
PTT	26				> 120	> 120	91	55	42	46	32	
INR	1.19				1.3	6.5	5.66	1.86	2.07	1.79	1.68	
Urea (mg/dL)	35					81	79	58	47	53	67	
Creatinine (mg/dL)	1.3					1.8	1.4	1.2	1.4	1.3	1.6	
AST (IU/L)						170	1031	–	–	–	757	
ALT (IU/L)						146	558	–	–	–	604	
Glucose (mg/dL)	174						159	225	180	207	136	
Calcium (mg/dL)	8.6						7.8	7.4	9.2	8	8.4	
Magnesium (mg/dL)							2.6	2.1	2.3	2	2.6	
TSH							0.1	–	–	–		
T4							9.2	–	–	–		
T3							0.9	–	–	–		

WBC white blood cell, *Hb* hemoglobin, *PTT* partial thromboplastin time, *INR* international normalized ratio, *ALT* alanine transaminase, *AST* aspartate aminotransferase, *TSH* thyroid-stimulating hormone

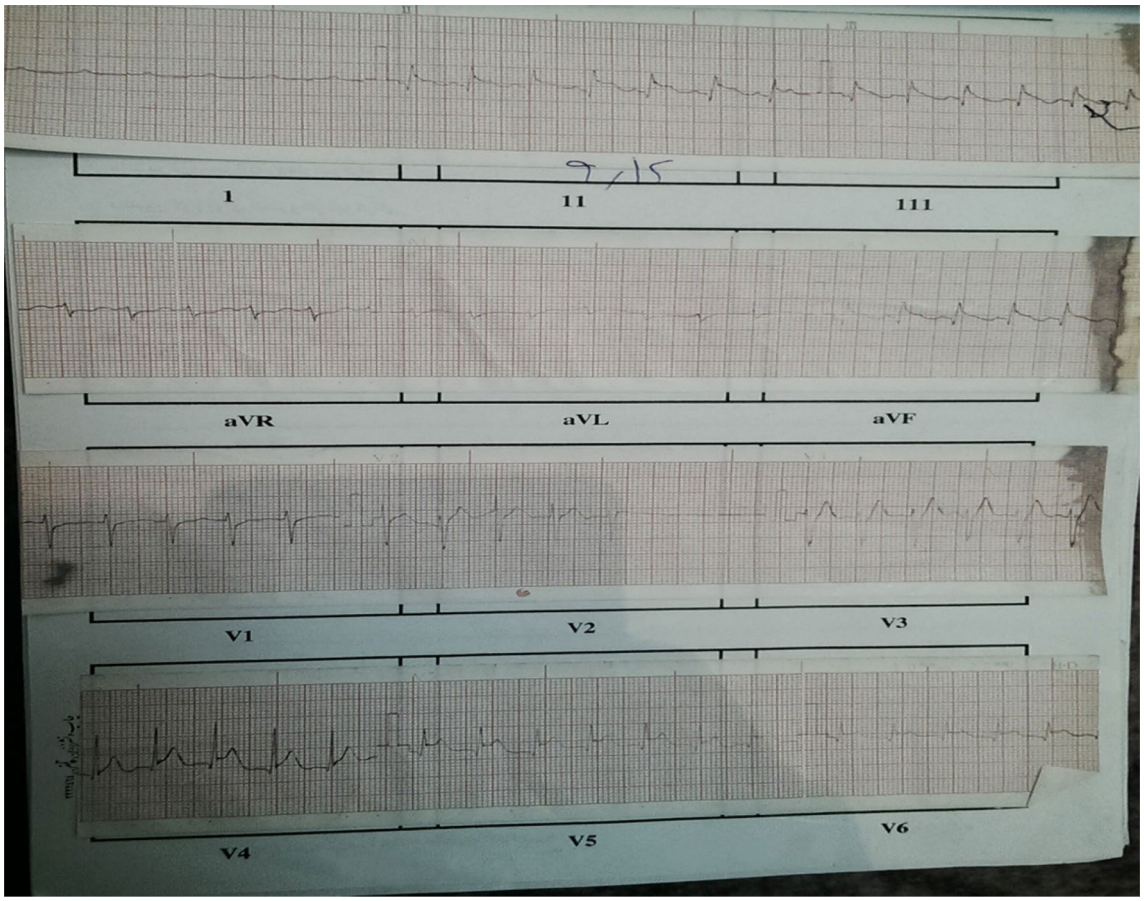

Fig. 2 Repeated ECG of case 1: sinus tachycardia and complete right bundle branch block with ST elevation in v5–v6

Blood samples were taken, and an ECG showed wide complex tachycardia with right bundle branch block pattern with ST elevation in v1–v6 and long QT (Fig. 3). Due to hypotension, the patient received 1 L of intravenous fluids. The patient was admitted to the ICU, given intravenous *N*-acetyl cysteine (9 g/1 h, 3 g/4 h and 6 g/16 h), intravenous glucagon at a dose of 5 mg/stat and 2 mg/h, 10% intravenous calcium gluconate 10 mg twice daily, and a 400 IU vitamin E twice daily. Sodium bicarbonate 7.5% was administered intravenously to resolve her metabolic acidosis.

Due to the critical condition, she was intubated.

Because of refractory hypotension, echocardiography was performed. Left ventricle ejection fraction (LVEF) was 15%. Three hours later, she experienced bradycardia, ventricular tachycardia, and premature ventricular contraction. Meanwhile, ventricular tachycardia was repeated within a few hours, which was treated by intravenous amiodarone. Intraaortic balloon pump was immediately inserted on this day.

There was an increase in the patient's liver enzymes (AST = 110 IU/L; ALT = 65 IU/L), and given her coagulation impairment, fresh frozen plasma (FFP) was transfused.

On the sixth day, the patient was alert, and the heart rhythm became sinusoidal. On this day, the patient reached a stable hemodynamic status without receiving inotropic agents.

Echocardiography results were as follows: EF = 30–35%, left ventricle (LV) enlargement with moderate to severe reduced systolic dysfunction, LV diastolic dysfunction grade II, global hypokinesis, normal right ventricle size and reduced function, and tricuspid annular plane systolic excursion (TAPSE) = 16 mm.

The seventh day was characterized by normal heart rhythm, reduced liver enzymes, and controlled fever. On the eighth day, echocardiography revealed improved cardiac function and increased EF up to 50%. IABP was removed on day 9. On the 13th day, symptoms of pneumonia disappeared, and she was discharged from the hospital with medication orders of carvedilol 3.125 twice daily and losartan 12.5 mg daily. She was also suggested to be visited by a cardiologist 2 weeks after discharge.

Table 2 Clinical and laboratory finding of case 2

Laboratory tests/time after ingestion	On admission	6 h	24 h	Day 3	Day 4	Day 12
Blood pressure (mmHg)	90/50	90/50	108/82	102/67	82/42	113/64
Pulse rate (bpm)	112	100	128	109	108	85
Respiratory rate (pm)	22	18	25	18	10	20
O_2 saturation (%)	–	74.7	91.6	97.1	98.6	97.5
pH	7.36	7.21	7.29	7.49	7.49	7.52
$PaCO_2$ (mmHg)	–	31.5	21.9	26.0	30.2	30.9
PaO_2 (mmHg)	–	49.9	68.9	85.4	119.2	89.6
Serum HCO_3 (mmol/L)	13.9	12.6	10.7	19.9	23.1	25.2
WBC (10^3 µL)	–	17.9	13.6	8.07	–	11.53
Hg (g/dL)	–	12.2	12.5	10.9	–	12.3
Platelet count (10^3 µL)	–	287	313	212	–	161
Na (meq/L)	–	138	146	140	139	–
K (meq/L)	–	3.9	4.6	4.7	4.4	–
Prothrombin time (s)	–	–	17	21.8	24.5	15.2
PTT (s)	–	–	29	20.4	26	30
INR	–	–	1.46	2.18	2.67	1.22
Blood sugar (mg/dL)	–	240	97	170	157	–
Fasting blood sugar (mg/dL)	–	–	150	–	223	–
Urea (mg/dL)	–	28	19	48	78	–
Creatinine (mg/dL)	–	1.2	1.2	1.1	1.1	–
Bilirubin direct (mg/dL)	–	–	0.8	0.5	–	–
Bilirubin total (mg/dL)	–	–	1.0	1.5	–	–
AST (IU/L)	–	–	16	110	–	31
ALT (IU/L)	–	–	21	65	–	166
Alkaline phosphatase (IU/L)	–	–	180	123	–	162
Phosphorus (mg/dL)	–	–	5.7	2.9	2.2	4.1
Calcium (mg/dL)	–	–	7.8	8.4	–	8.3
Magnesium (mg/dL)	–	–	3.2	3.3	–	2.1
LDH (IU/L)	–	–	–	–	–	–
CPK (creatine phosphokinase) (IU/L)	–	–		–	–	–
Total protein (g/dL)	–	–	4.6	–	–	–
Albumin serum (g/dL)	–	–	3.5	–	–	–

WBC white blood cell, *Hb* hemoglobin, *PTT* partial thromboplastin time, *INR* international normalized ratio, *ALT* alanine transaminase, *AST* aspartate aminotransferase, *LDH* lactate dehydrogenase

Discussion and Conclusion

We described two cases of previously healthy adults with acute ingestions of AlP who received IABP as an adjunct to support their resulting myocardial dysfunction and cardiogenic shock.

AlP is a cheaply and easily available potent pesticide in many countries, which is usually used to protect against cereal pests or to kill mice [14]. In Iran, AlP is available in 3-g pills with commercial brands of quickphos, celphos, and phostoxin. The pill contains 56% AlP and 44% alumina carbonate [15]. It produces oxidative factors in the body and leads to increased extra-mitochondrial release of oxygen radicals and thus lipid peroxidation and denaturation of the cell wall of many organs [3].

The signs and symptoms of AlP poisoning are initially manifested in the gastrointestinal system including abdominal pain, nausea, and vomiting. However, after several hours, severe cardiovascular collapse, hypotension, and respiratory failure also occur [16]. AlP poisoning is frequently reported to cause cardiovascular collapse and cardiogenic shock [11].

Similar to what happened in our patients, changes in the ECG can be seen, from sinus tachycardia which frequently occurs 3–6 h after taking the pill to ST-T changes and different types of dysrhythmia [17]. In this article, as noted above, the patients showed ST-segment disorder and tachyarrhythmia. The main cause of this cytogenetic shock is the inhibition of cytochrome oxidase and myocarditis developed by oxidation factors leading to cardiac impairment and arrest

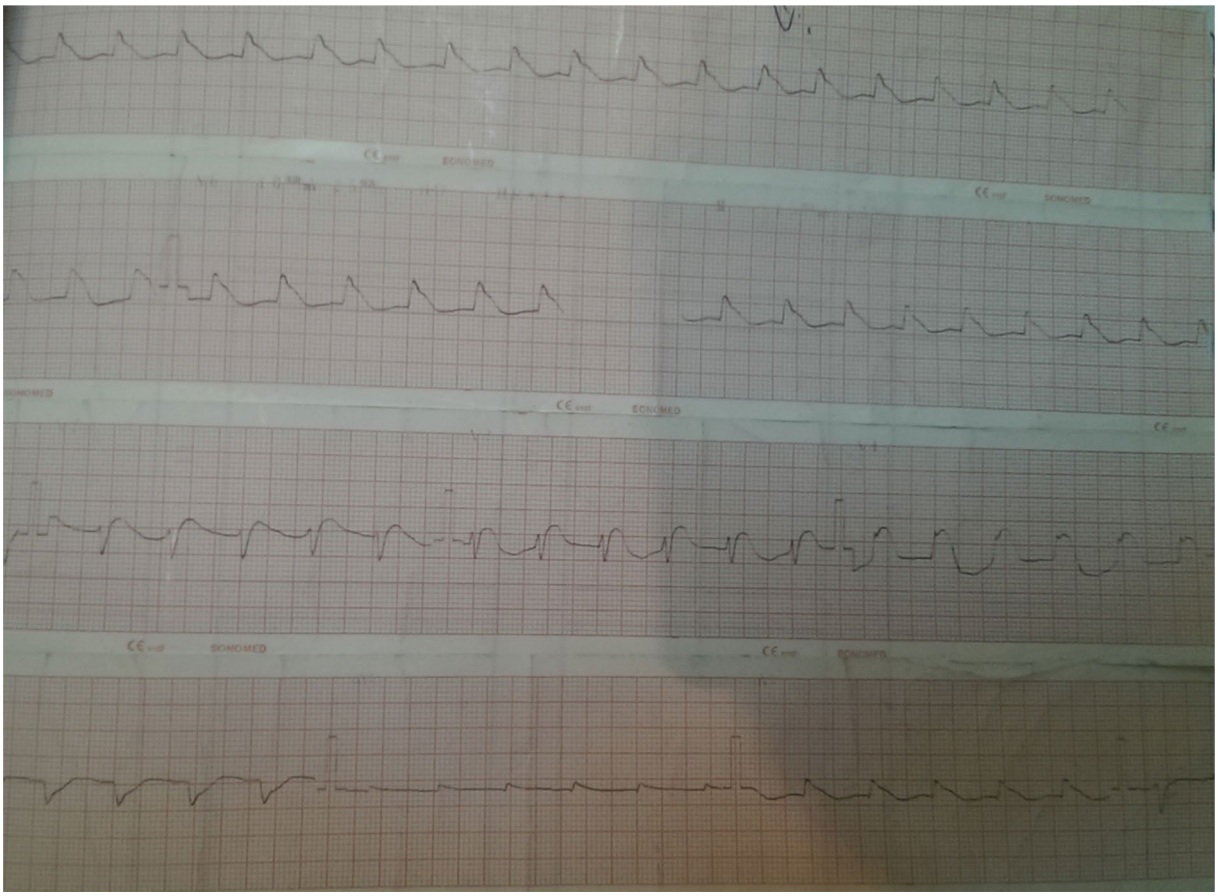

Fig. 3 Wide complex tachycardia with right bundle branch block pattern with ST elevation in v1–v6 and long QT in the ECG of patient were seen

[3]. Other pathologies that can develop include coagulopathy and hepatotoxicity [18].

Direct toxicity to the vasculature combined with coagulopathy can lead to clinical hemorrhage from various organ systems throughout the gastrointestinal tract and pulmonary tree.

Interestingly, both the study patients had symptoms of pneumonia, potentially due to indirect or direct effects of AlP on the lung, and had undergone a period of coma and intubation, which may have also caused pneumonia.

The effect of AlP on the lung has rarely been investigated in the literature. The effects of AlP on non-survived cases of AlP poisoning had already been studied. Lung microscopic assessments revealed congestion as the most common finding followed by edema, hemorrhage, collapses of alveoli, alveolar thickening, dilated capillaries, and disturbance of the alveolar wall [19, 20]. Sinha et al. found congestion and thickening of the alveolar walls, edema, dilated capillaries, and hemorrhage to be the most common observations in microscopic evaluations. Further, gray or red hepatization and round cell infiltration around bronchioles were observed in some cases [21].

Cardiogenic shock or low ejection fraction due to AlP poisoning has frequently been reported [3, 22–32].

Both of our patients had EF of about 15% plus persisted hypotension and both of them required vasopressors. So, the diagnosis of cardiogenic shock was made for the patients. Indeed, cardiogenic shock is one of the main causes of mortality in this poisoning [10]. For the diagnosis of cardiogenic shock, the suggested criteria include systolic blood pressure < 90 mmHg for more than 30 min or vasopressor requirement to reach a blood pressure ≥ 90 mmHg, elevated left ventricular filling pressures, pulmonary congestion or signs of impaired organ perfusion [33].

Patients with cardiogenic shock have the options of mechanical circulatory support such as IABP, left ventricular assist devices (LVADs), and extracorporeal membrane oxygenation (ECMO) [34, 35].

The indications reported for the IABP include: poor left ventricular failure, blood pressure problems, cardiogenic shock, critical coronary artery disease (including left main), refractory chest pain, intraoperatively, elevated left heart filling pressure, problems with weaning, supply circulation support during or after cardiac catheterization, weaning from

cardiopulmonary bypass, preoperative utility in high-risk patients and persistent unstable angina, adjunctive therapy in patients with complicated or high risk angioplasty, prophylaxis in cases with severe left main coronary arterial stenosis who are awaiting surgery, intractable myocardial ischemia awaiting further therapy, as a bridge to a further treatment in cases with profound and refractory heart failure [36, 37].

Further, IABP is relatively simple to be placed and provides a proper safety profile and fewer side effects especially vascular side effects compared with the other devices [34]. Although some vascular complications such as aortic dissection have been reported for it, these complications are significantly fewer than those associated with other devices [37]. Other adverse effects include limb ischemia and thromboembolic complications [37]. In addition, IABP provides acceptable hemodynamic support, but this support is less compared to other devices including ECMO and LVADs, though it is indeed less invasive in comparison with ECMO and LVADs.

In the IABP procedure, a balloon is inserted into the femoral artery under the left subclavian artery or above the renal artery, increasing the heart output and perfusion and improving the CNS [38]. As in the present case studies, IABP has been used to help support the cardiac function in cases of AlP toxicity [12, 39].

Both of the study cases with left ventricle failure and a low cardiac output met the indication criteria for receiving IABP. The temporary cardiogenic shock in AlP poisoning can be improved with supportive care. Fortunately, none of our patients had any adverse effect or complications related to IABP.

Other researchers proposed using IABP for the temporary support of left ventricle dysfunction; nevertheless, it is worth noting that IABP provides support only to the left ventricle and is unlikely to improve primary right ventricular systolic dysfunction [40].

IABP has also been applied on other toxicological exposures leading to cardiac failure [41–43]. Despite the successful use in our described case series, there have been other case series where IABP did not improve the clinical status of AlP ingestion [31]. Thus, it is possible that patients with huge ingestion of AlP do not benefit similarly from the IABP procedure. Further research with larger sample sizes is required to investigate the success rate of AlP poisoning treatment using IABP to determine the probable effective factors in patients' response to the treatment by IABP.

AlP toxicity can lead to multi-organ failure, severe metabolic acidosis, and cardiovascular collapse and shock. The mainstay of treatment is symptomatic and supportive care. IABP was successfully used in support of cardiogenic shock following IAP ingestion. Further studies are required to more accurately evaluate when IABP is best used for this and other toxicologic etiologies of cardiogenic shock.

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Compliance with Ethical Standards

Conflict of interest The authors declare that they have no conflict of interest.

Ethical Approval This study involved human data and was conducted in strict accordance with the Declaration of Helsinki.

Informed Consent Consent for publication of these two cases was obtained.

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