

Case report

Successful treatment of cardiogenic shock with an intra-aortic balloon pump following aluminium phosphide poisoning

Omid Mehrpour^{1,2,3}, Ahmad Amouzeschi^{4,5}, Bita Dadpour³, Zohreh Oghabian⁶, Nasim Zamani⁷, Shahram Amini⁸, and Robert S. Hoffman⁹

Atherosclerosis and Coronary Artery Research Center¹, Medical Toxicology and Drug Abuse Research Center (MTDRC)², Department of Cardiac Surgery⁴, Birjand University of Medical Sciences, Birjand, Addiction Research Centre³, Department of Cardiac Surgery⁵, Department of Anesthesiology⁸, Mashhad University of Medical Toxicology Sciences, Mashhad, Department of Clinical Toxicology, Kerman University of Medical Sciences, Kerman⁶, Department of Clinical Toxicology, Loghman Hakim Hospital, Shahid Beheshti University of Medical Sciences, Tehran⁷, Iran, Division of Medical Toxicology, Department of Emergency Medicine, NYU School of Medicine, Bellevue Hospital Center, NY, USA⁹

Received in May 2013
CrossChecked in May 2013
Accepted in September 2013

Aluminium phosphide (AIP) is a highly toxic pesticide that inhibits cytochrome oxidase c and causes oxidative stress. Death results from refractory cardiogenic shock due to myocardial dysfunction. There is very little information regarding extracorporeal life support in severe AIP poisoning. Although several therapies are available, none are curative. We report on the use of an intra-aortic balloon pump (IABP) in a 24-year-old woman brought to our hospital after an intentional ingestion of a tablet of AIP (3 g), which caused refractory AIP-induced cardiogenic shock and acute respiratory distress syndrome (ARDS). The patient underwent gastric lavage with potassium permanganate, received sodium bicarbonate intravenously, and was admitted to the intensive care unit. Echocardiography at 36 h post ingestion showed a left ventricular ejection fraction (LVEF) of <20 %. An IABP was inserted and the patient's vital signs stabilised. After eight days, the IABP was removed and on day 20, the patient's LVEF increased to 50 %. IABP was successfully used and may improve future prognoses for severely poisoned AIP patients with refractory cardiogenic shock. We encourage clinical toxicologists to examine this new treatment.

KEY WORDS: *emergency medical treatment; extracorporeal life support; phosphine gas poisoning*

Aluminium phosphide (AIP) is an extremely efficient substance used as both an outdoor and indoor pesticide (1-5). It is easily available in Asian countries such as India and Iran, where it is also known as "rice tablet" because of its frequent use in the protection of stored rice and grains from rodents and other household pests (6). Many cases of suicide attempts

have been reported with rice tablets in these countries (3, 5). In fact, AIP is generally the most commonly used fatal intentional poison in Iran and India (3, 7, 8). AIP poisoning leads to multiorgan failure (9, 10), but death following this poisoning is most often caused by refractory hypotension and cardiogenic shock in the form of toxic chemical myocarditis, which leads

to severe left ventricular dysfunction (8, 11, 12). The exact mechanism of its toxicity is unclear, but the release of phosphine gas as well as cytochrome oxidase inhibition, oxidative stress, and free oxygen radicals have been suggested as the main causes of toxicity (8, 13). Unfortunately, there is no specific antidote and all treatments, such as gastric lavage with potassium permanganate, administration of coconut oil (14), etc., are only supportive procedures. Refractory hypotension is generally managed by early resuscitation with fluid and vasoactive agents (norepinephrine or phenylephrine, dopamine, and dobutamine). Moreover, Vitamins C and E (3), hyperbaric oxygenation (15), N-acetyl cysteine (16), digoxin (11), and recently intra-aortic balloon pump (IABP) (12) have been used to treat toxic myocarditis due to rice tablet poisoning.

AIP is highly reactive and any contact with moisture will result in decomposition to phosphine gas. Phosphine poisoning has a mortality rate as high as 70-100 % (9, 10, 17). Its main mechanism of toxicity is the inhibition of cytochrome oxidase c and oxidative stress (8, 13, 18). As a result, AIP poisoning can cause refractory cardiogenic shock and metabolic acidosis (8). Currently, there is no specific antidote for AIP poisoning and treatment is only supportive. This paper reports a successful treatment of AIP-induced cardiogenic shock using an IABP in a 24-year-old woman.

Case report

The Imam Reza hospital in Mashhad, Iran is the referral hospital for poisoned patients in Eastern Iran and one of the largest toxicology hospitals in the world (19, 20). A 24-year-old woman who had intentionally ingested one three-gram tablet of aluminium phosphide (AIP) was referred to our hospital and admitted to the Toxicology Emergency Department. She ingested the tablet with four glasses of water, vomited immediately, and was presented fully conscious to a local hospital complaining of weakness, cold extremities, and thirst. The patient's initial vital signs were as follows: blood pressure 90/50 mm Hg; pulse rate 120 bpm; respiratory rate 20 per minute; and body temperature 37.1 °C, with an oxygen saturation of 96 % at room air. Initial blood gas analysis showed a pH of 7.39; PCO_2 21.2 mm Hg; PaO_2 59 mm Hg; HCO_3^- 12.9 mmol L⁻¹; and a base excess (BE) of -9.1 mmol L⁻¹. The patient underwent gastric lavage with potassium permanganate (1/10000) and was given intravenous sodium bicarbonate

(NaHCO_3) 44 mEq every 15 min. The patient was then admitted to our Centre (about three hours post-ingestion of AIP). Vital signs on arrival were: blood pressure 100/70 mm Hg; pulse 130 bpm; respiratory rate 18 per minute; and body temperature 37 °C with an oxygen saturation of 96 %. Electrocardiography (ECG) revealed sinus tachycardia with no ST-T changes, but serial ECGs showed non-specific ST and T-wave changes and interventricular conduction delay (Figure 1). Serial vital signs and laboratory data are shown in Table 1. The patient was admitted to the intensive care unit (ICU) and received intravenous crystalloid fluid (normal saline; 150 mL h⁻¹ until her thirst was resolved), vasopressors (dopamine; 10 $\mu\text{g kg}^{-1} \text{min}^{-1}$, and norepinephrine; 4 $\mu\text{g min}^{-1}$), digoxin 0.5 mg stat followed by 0.25 mg q6h for 24 h and 0.25 mg daily, afterwards, glucagon 4 mg h⁻¹, magnesium sulphate 2 g stat followed by 1 g q6h, sodium bicarbonate (until correction of metabolic acidosis), vitamin C 500 mg per day, vitamin E 100 IU per day, heparin 5000 IU BID and N-acetylcysteine 300 mg kg⁻¹ through 24 h in three divided doses. About 24 h later, the patient experienced tachypnea (respiratory rate 45 per minute) and her O_2 saturation dropped to 70 %.

The patient also complained of retrosternal discomfort and was therefore intubated and placed on mechanical ventilation with the following parameters: synchronised intermittent mandatory ventilation (SIMV) mode of fractional inspired oxygen (FiO_2) was 50 %, tidal volume was 350 mL, and positive end-expiratory pressure (PEEP) was 7 mm H₂O. However, her O_2 saturation remained at 88-92 % and gasometry showed an arterial oxygen pressure (PaO_2) level of 50 mm Hg. A chest X-ray confirmed acute respiratory distress syndrome (ARDS) (Figure 2). ARDS was defined as diminished PaO_2 to FiO_2 ratio (P to F ratio smaller than 200). Thirty-six hours after admission, bedside echocardiography showed a left ventricular ejection fraction (LVEF) of less than 20 %, global hypokinesia of the left ventricle and mitral valve regurgitation (MR) and an IABP was inserted by a cardiac surgeon. The patient had no contraindications to the use of an IABP (e.g., sepsis, aortic aneurysms, peripheral vascular diseases, neurological deficits, aortic dissection, etc.).

On the third day of ICU admission, ventilator acquired pneumonia (VAP) occurred and the patient's temperature increased to 38.5 °C. Meropenem (1 g three times per day), amikacin (500 mg BID), and vancomycin (1 g BID) were administered. On day

Table 1 Clinical and paraclinical findings of our case of AIP poisoning

laboratory tests / time after ingestion	on admission	12 h	24 h	day 5	day 8	day 10	day 18	day 26
Blood pressure (mm Hg)	100/70	90/60	80/50	105/65	110/70	105/80	120/70	115/80
Pulse rate (bpm)	130	160	150	110	86	90	103	98
Respiratory rate (pm)	20	30	45	SIMV	SIMV	SIMV	20	15
O ₂ saturation (%)	94	86	70	91	100	100	96	95
pH	7.48	7.30	6.90	7.47	7.49	7.38	-	7.42
PaCO ₂ (mm Hg)	17.6	19.9	69	45	34.8	43.9	-	32.3
PaO ₂ (mmHg)	58	52	50	56	60	62	65	65
Serum HCO ₃ (mmol L ⁻¹)	13.1	10	15.4	32.9	28.2	26.5	-	21.4
WBC (10 ³ μL)	16.5	-	23	-	23	22.6	14.1	4.4
Hb (g dL ⁻¹)	13.7	-	14.4	-	8.4	8.7	9	10
Platelet count (10 ³ μL)	232	-	194	-	81	131	267	316
Na (meq L ⁻¹)	140	-	139	144	139	136	138	140
K (meq L ⁻¹)	4	-	3.9	2.6	4.6	4.4	4.1	3.8
Prothrombin time (s)	12.5	-	-	-	15.6	14.4	-	13.3
Creatinine (mg dL ⁻¹)	0.6	-	-	0.8	0.8	0.7	0.6	0.9
Glucose (mg dL ⁻¹)	145	-	485	45	169	155	135	109
Creatine phosphokinase (IU L ⁻¹)	219	-	-	-	648	203	-	124
AST* (IU L ⁻¹)	52	-	-	-	-	72	43	45
ALT** (IU L ⁻¹)	75	-	-	-	-	273	216	89

SIMV - synchronised intermittent mandatory ventilation

*Aspartate aminotransferase

**Alanine aminotransferase

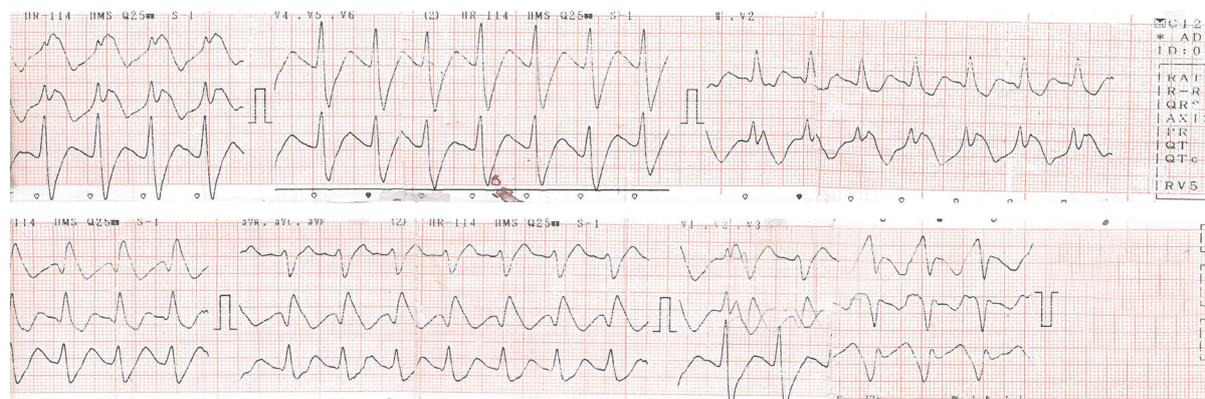


Figure 1 Electrocardiography of AIP poisoned case. Electrocardiography (ECG) revealed sinus tachycardia with no ST-T changes; but serial ECGs showed non-specific ST and T-wave changes and interventricular conduction delay

four, sudden complete cardiac arrest occurred; however, fortunately, the patient responded to resuscitation. After stabilization of vital signs, the IABP was removed (8 days after insertion). Repeated echocardiography on day 20 of admission showed an LVEF of 50 %.

Surprisingly, our patient exhibited thrombocytopenia, hypokalaemia, and variations in haemoglobin between the second and the eighth day of admission (Table 1), maybe due to receiving heparin. The administered

antibiotics were the other possible causes of acute thrombocytopenia (21, 22). Hypokalaemia in our patient was probably due to the administration of sodium bicarbonate to correct acidosis, although there is evidence that AIP can affect potassium levels in cases of poisoning (9, 23). Variations in haemoglobin were probably due to haemolysis induced by the IABP as an external device. Mean corpuscular volume amounts have been reported to be at maximum levels



Figure 2 Chest X-ray of AIP poisoned case. Acute respiratory distress syndrome (ARDS)

due to the release of unmaturation red blood cells into circulation from bone marrow hyperactivity (24).

The patient's medication (vasopressors, calcium gluconate, magnesium sulphate, hydrocortisone, vitamin C, and vitamin E) was tapered gradually. After consultation with a psychologist, the patient was discharged on day 27 in good condition and normal vital signs and laboratory tests. A follow-up visit about two months later revealed that the patient was asymptomatic with no evidence of ongoing toxicity or sequelae.

DISCUSSION

Cardiogenic shock is defined as systolic blood pressure <90 mm Hg or 30 mm Hg below the baseline during organ dysfunction (25). It is a kind of shock in which the failure of the cardiac ventricle causes inadequate circulation. It is defined by tissue hypoperfusion and hypotension and is a largely irreversible and fatal condition (26). Cardiogenic shock is commonly caused by damage to the heart muscle such as myocardial infarction, arrhythmias, cardiomyopathy, etc. Some cases of drug poisoning, such as those by β_1 adrenergic receptor antagonists (e.g. atenolol, propranolol) and calcium channel blockers, also lead to profound hypotension and cardiogenic shock; similar to those that occur with aluminium phosphide (27-29). Cardiogenic shock could also be approached by procedures such as administration of positive inotropic agents or glucagon (11, 30) and enhancing pumping capabilities, which

should cause a positive inotropic and chronotropic effect by stimulating adenosine monophosphate.

Pumping can also be improved by using an IABP, a left ventricular assist device that enhances the perfusion of coronary arteries. During an IABP procedure, a balloon is inserted via the femoral artery and placed below the left subclavian and above the renal arteries (31). It is generally used in patients with heart failure waiting for heart transplantation, further revascularization, and shock. At the end of a systole, pumping the balloon causes an increase in aortic blood pressure and increases cardiac output and coronary perfusion as well as improves central nervous system perfusion.

There have been several cases of intoxication where an IABP was applied (27-29), all of which involved refractory cardiogenic shock. Janion et al. (32) reported successful treatment of combined massive drug intoxication using mechanical ventilation and IABP, concluding that combined mechanical and pharmacological treatment may even prevent multi-organ insufficiency.

The use of an IABP for the treatment of AIP was first reported by Chacko et al. (33) in 2008, in a 25-year-old female who suffered from severe toxicity after ingesting 10 tablets of AIP. Unfortunately, it was unsuccessful. A successful treatment of AIP with an IABP was first reported by Siddaiah et al. (12) in 2009, in a 22-year old female who, suffering with cardiogenic shock due to AIP poisoning, was treated with an IABP until the effects of AIP resolved. There are no other reports in the literature on IABP in the treatment of AIP poisoning and the case presented here was the second successful one in this regard.

Our use of an IABP for treating AIP-induced cardiogenic shock yielded success in a patient who otherwise appeared to have a very poor prognosis due to refractory hypotension and ARDS (34, 35). Given our success in the case reported here, it is reasonable to question why IABP has not been used more often. We assume that this most likely relates to the unavailability of a cardiac surgeon in Iranian referral toxicological centres and probably other countries where AIP poisoning is common. Furthermore, to improve outcomes, AIP-poisoned patients need extensive ICU care after insertion of an IABP and, due to the high rate of poisoned patients in the aforementioned countries, the lack of ICU beds is another limitation that should be considered.

IABP has also been used in severe LV dysfunction in other toxic myocarditis cases (27-30), as IABP

generally reduces afterload and improves coronary artery perfusion. Despite many proposed therapies, patients with severe AIP poisoning continue to die of cardiogenic shock. We recommend the use of this treatment in other cases of refractory cardiogenic shock from AIP poisoning and encourage clinical toxicologists to report on their experiences.

Acknowledgments

The successful completion of this paper was facilitated by our balloon pump team comprising Mr Abdolali Ghazmeh and Mr Majid Heidari.

REFERENCES

1. Mehrpour O. Comment on "An update on toxicology of aluminum phosphide". *DARU* 2012;20:50. doi: 10.1186/2008-2231-20-50
2. Mehrpour O, Keyler D, Shadnia S: Comment on Aluminum and zinc phosphide poisoning. *Clin Toxicol (Phila)* 2009;47:838-9. doi: 10.1080/15563650903203684
3. Mehrpour O, Jafarzadeh M, Abdollahi M. A systematic review of aluminium phosphide poisoning. *Arh Hig Rada Toksikol* 2012;63:61-73. doi: 10.2478/10004-1254-63-2012-2182
4. Shadnia S, Mehrpour O, Soltaninejad K. A simplified acute physiology score in the prediction of acute aluminum phosphide poisoning outcome. *Indian J Med Sci* 2010;64:532-9. PMID: 21258160
5. Shadnia S, Mehrpour O, Abdollahi M. Unintentional poisoning by phosphine released from aluminum phosphide. *Hum Exp Toxicol* 2008;27:87-9. doi: 10.1177/0960327107086241
6. Mehrpour O, Singh S. Rice tablet poisoning: A major concern in Iranian population. *Hum Exp Toxicol* 2010;29:701-2. doi: 10.1177/0960327109359643
7. Singh D, Dewan I, Pandey AN, Tyagi S. Spectrum of unnatural fatalities in the Chandigarh zone of north-west India: a 25 year autopsy study from a tertiary care hospital. *J Clin Forensic Med* 2003;10:145-52. PMID: 15275009
8. Bolter CJ, Chefurka W. Extramitochondrial release of hydrogen peroxide from insect and mouse liver mitochondria using the respiratory inhibitors phosphine, myxothiazol, and antimycin and spectral analysis of inhibited cytochromes. *Arch Biochem Biophys* 1990;278:65-72. PMID: 2321971
9. Mehrpour O, Alfred S, Shadnia S, Keyler DE, Soltaninejad K, Chalaki N, Sedaghat M. Hyperglycemia in acute aluminum phosphide poisoning as a potential prognostic factor. *Hum Exp Toxicol* 2008;27:591-5. doi: 10.1177/0960327108096382
10. Mehrpour O, Dolati M, Soltaninejad K, Shadnia S, Nazparvar B. Evaluation of histopathological changes in fatal aluminum phosphide poisoning. *Indian J Forensic Med Toxicol* 2008;2:34-6.
11. Mehrpour O, Farzaneh E, Abdollahi M. Successful treatment of aluminum phosphide with digoxin: a case report and review of literature. *Int J Pharmacol* 2011;7:761-4. doi: 10.3923/ijp.2011.761.764
12. Siddaiah L, Adhyapak S, Jaydev S, Shetty G, Varghese K, Patil C, Iyengar S. Intra-aortic balloon pump in toxic myocarditis due to aluminum phosphide poisoning. *J Med Toxicol* 2009;5:80-3. PMID: 19415593
13. Price NR, Moles KA, Humphires OA. Phosphine toxicity and catalase activity in susceptible and resistant strains of lesser grain borer (*Rhyzopertha dominica*). *Comp Biochem Physiol Part C: Comp Pharmacol* 1982;73:411-5. doi: 10.1016/0306-4492(82)90144-7
14. Pajoumand A, Jalali N, Abdollahi M, Shadnia S. Survival following severe aluminum phosphide poisoning. *J Pharm Pract Res* 2002;32:297-9.
15. Saidi H, Shokraneh F, Ghafouri HB, Shojaie SJ. Effects of hyperbaric oxygenation on survival time of aluminum phosphide intoxicated rats. *J Res Med Sci* 2011;16:1306-12. PMID: 22973324
16. Tehrani H, Halvaie Z, Shadnia S, Soltaninejad K, Abdollahi M. Protective effects of N-acetylcysteine on aluminum phosphide-induced oxidative stress in acute human poisoning. *Clin Toxicol (Phila)* 2013;51:23-8. doi: 10.3109/15563650.2012.743029
17. Mehrpour O, Aghabiklooei A, Abdollahi M, Singh S. Severe hypoglycemia following acute aluminum phosphide (rice tablet) poisoning; a case report and review of the literature. *Acta Med Iran* 2012;50:568-71. PMID: 23109032
18. Zamani N, Mehrpour O. Protective role of G6PD deficiency in poisoning by aluminum phosphide; are there possible new treatments? *Eur Rev Med Pharmacol Sci* 2013;17:994-5. PMID: 23640450
19. Mehrpour O, Abdollahi M. Poison treatment centers in Iran. *Hum Exp Toxicol* 2012;31:303-4. doi: 10.1177/0960327110392086
20. Mehrpour O, Zamani N, Brent J, Abdollahi M. A tale of two systems: poisoning management in Iran and the United States. *DARU* 2013;21:42. doi: 10.1186/2008-2231-21-42
21. Ruggero MA, Abdelghany O, Topal JE. Vancomycin-induced thrombocytopenia without isolation of a drug-dependent antibody. *Pharmacotherapy* 2012;32:e321-5. doi: 10.1002/phar.1132
22. Alegre Herrera S, Quirós Valera M, Rodríguez Fernández A. Trombocitopenia aguda por imipenem/cilastatina [Imipenem/cilastatin-induced acute thrombocytopenia, in Spanish]. *Med Clin (Barc)* 2001;117:197-8. PMID: 11481089
23. Mehrpour O, Shadnia S, Soltaninejad K, Yaghmaei A. Evaluation of electrolytes and blood glucose level in aluminum phosphide poisoning. *Sci J Forensic Med* 2009;15:49-53.
24. Ficek SJ, Stammers AH, Galbraith TA, Alonso A. Hemostatic assessment of patients undergoing intraaortic balloon pump therapy. *J Extra Corpor Tech* 1997;29:78-82. PMID: 10168534
25. Worthley LIG. Shock: A review of pathophysiology and management. Part I. *Crit Care Resusc* 2000;2:55-65. PMID: 16597285
26. Sarswat N, Hollenberg SM. Cardiogenic shock. *Hosp Pract (Minneapolis)* 2010;38:74-83.
27. Melanson P, Shih RD, De Roos F. Intra-aortic balloon counterpulsation in calcium channel blocker overdose. *Vet Hum Toxicol* 1993;35:345.
28. Lane AS, Woodward AC, Goldman MR. Massive propranolol overdose poorly responsive to pharmacologic therapy: use

- of the intra-aortic balloon pump. *Ann Emerg Med* 1987;16:1381-3. PMID: 3688604
29. Gillard P, Laurent M. Dextropropoxyphene-induced cardiogenic shock: treatment with intra-aortic balloon pump and milrinone. *Intensive Care Med* 1999;25:335. PMID: 10229178
30. Hall-Boyer K, Zaloga GP, Chernow B. Glucagon: hormone or therapeutic agent? *Crit Care Med* 1984;12:584-9. PMID: 6375966
31. Wnek W. Zastosowanie kontrapulsacji wewnątrzortalnej w terapii ciężkiej niewydolności krążenia w zatruciach lekami kardiotoksycznymi [The use of intra-aortic balloon counterpulsation in the treatment of severe hemodynamic instability from myocardial depressant drug overdose, in Polish]. *Przegl Lek* 2003;60:274-6. PMID: 14569902
32. Janion M, Stepień A, Sielski J, Gutkowski W. Is the intra-aortic balloon pump a method of brain protection during cardiogenic shock after drug intoxication? *J Emerg Med* 2010;38:162-7. doi: 10.1016/j.jemermed.2007.10.037
33. Chacko J, Shivaprasad C. Fatal aluminium phosphide poisoning due to myocardial depression refractory to high dose inotropic support and intra-aortic balloon counterpulsation. *Indian J Crit Care Med* 2008;12:37-8. doi: 10.4103/0972-5229.40949
34. Siwach SB, Singh P, Ahlawat S, Dua A, Sharma D. Serum and tissue magnesium content in patients of aluminium phosphide poisoning and critical evaluation of high dose magnesium sulphate therapy in reducing mortality. *J Assoc Physicians India* 1994;42:107-10. PMID: 7860467
35. Wahab A, Zaheer M, Wahab S, Khan R. Acute aluminium phosphide poisoning: an update. *Hong Kong J Emerg Med* 2008;15:152-5.

Sažetak

Primjena intra-aortne balonske pumpe pri kardiogenom šoku uzrokovanom trovanjem aluminijevim fosfidom

Aluminijev fosfid (AIP) visoko je toksičan pesticid koji inhibira citokrom c oksidazu i uzrokuje oksidacijski stres. Smrt nastupa nakon refraktornog kardiogenog šoka uslijed miokardijalne disfunkcije. Vrlo je malo literature o izvantjelesnim sredstvima održavanja na životu nakon otrovanja AIP-om. Iako postoji određeni broj terapija, nijedna ne vodi do potpunoga izlječenja. Prikazan je slučaj uporabe intra-aortne balonske pumpe (IABP) kod 24-godišnje žene otrovane namjernim uzimanjem AIP-a (3 g), što je uzrokovalo refraktorni kardiogeni šok i akutni respiratorni distres sindrom (ARDS). Pacijentici je želudac ispran kalijevim permanganatom, intravenozno je primila natrijev bikarbonat te je zbrinuta u jedinici za intenzivnu skrb. Ehokardiografija 36 h nakon uzimanja tablete AIP-a pokazala je ejekcijsku frakciju lijevog ventrikula (LVEF) od < 20 %. Nakon umetanja IABP-a vitalni su se znakovi su se poboljšali. Pumpa je uklonjena nakon osam dana, a dvadesetoga dana LVEF je iznosio 50 %. Naši rezultati pokazuju da bi primjena IABP-a mogla poboljšati prognoze pacijenata s refraktornim kardiogenim šokom uslijed teškog otrovanja s AIP-om. Stoga preporučujemo kliničkim toksikolozima da razmotre ovaj novi tretman.

KLJUČNE RIJEČI: *hitni medicinski tretman; izvantjelesna sredstva održavanja na životu; otrovanje pesticidom*

CORRESPONDING AUTHOR:

Omid Mehrpour MD
Medical Toxicology and Drug Abuse Research Center (MTDRC)
University of Medical Sciences;
Pasdaran Avenue; Birjand; Iran
Email: omid.mehrpour@yahoo.com.au