Successful Treatment of Severe Metabolic Acidosis Due to Acute Aluminum Phosphide Poisoning With Peritoneal Dialysis

A Report of 2 Cases

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Keywords. aluminum phosphide, poisoning, peritoneal dialysis

Aluminum phosphide poisoning is common in our region. It can cause severe metabolic acidosis and persistent hypotension, which lead to cardiogenic shock and subsequently mortality. Oliguric or anuric acute kidney injury is seen in almost all patients with aluminum phosphide poisoning. Renal replacement therapies are recommended in these patients to improve metabolic acidosis and increase the rate of survival. We report 2 cases of severe acute aluminum phosphide poisoning treated successfully with peritoneal dialysis.

IJKD 2017;11:165-7
www.ijkd.org

INTRODUCTION

There is an increased report of self poisoning with aluminum phosphide tablets from Iran in recent years.[1] Aluminum phosphide poisoning has a high mortality rate due to the absence of a specific antidote.[2] Cardiogenic shock is the most important clinical feature, which is caused by direct myocardial toxicity of phosphine gas and severe metabolic acidosis.[2-4] Different treatments are recommended in order to decrease the toxin absorption and to alkalize blood pH to prevent or improve the inevitable metabolic acidosis.[5]

Aggressive correction of metabolic acidosis could result in significant improvement in the patients' outcome.[6] Continuous renal replacement therapy is reported as an effective method of treating metabolic acidosis and increasing the survival.[7] However, peritoneal dialysis (PD) is not reported as a treatment modality for these cases. Here, we report successful treatment of 2 cases of intentional aluminum phosphide ingestion with PD.

CASE REPORT

Two women aged 23 and 40 years old were admitted to the Emergency Department of Imam Khomeini Hospital of Ardabil after 30 minutes and 3 hours ingestion of 1 tablet of 3 g of aluminum phosphide. The first patient had several vomiting episodes and the second patient presented with abdominal pain and multiple vomiting episodes. Both patients were tachypenic and hypotensive and had tachycardia with mild metabolic acidosis (Table). Severe metabolic acidosis occurred in the following hours and 19 and 10 vials of sodium bicarbonate were infused during the admission.

We used an aggressive correction of acidosis protocol introduced by Jaiswal and colleagues[6] to improve metabolic acidosis. During the treatment, the first patient had ventricular fibrillation and DC shock was given and later had ventricular tachycardia which was treated with amiodarone. Due to the severe metabolic acidosis and persistent hypotension regardless of fluid resuscitation and vasopressors in both cases, PD was initiated. We defined a new continuous PD protocol with the following setting: On the first day, 1000 mL every 2 hours; on the second day, 1000 mL every 3 hours; and on the third to the last day of PD, 1000 mL

Case Report

Arterial Blood Gas Changes During Hospital Stay

Parameter	Admission	Peritoneal Dialysis						Discharge
		Initiation	2 Hours	4 Hours	6 Hours	12 Hours	18 Hours	
Patient 1								
pH	7.32	7.04	7.19	7.26	7.38	7.40	7.46	7.49
Carbon dioxide pressure	18.0	57	25.2	19.9	18.0	28.0	41.7	37.2
Bicarbonate	9.2	15	9.7	9.1	10.8	17.4	29.0	28.1
Base excess	-14.4	-16.1	-18.7	-18.1	-14.5	-5.8	6.1	6.1
Patient 2								
pH	7.23	6.98	7.13	7.23	7.28	7.30	7.42	7.37
Carbon dioxide pressure	25	49	19.6	22.3	25.5	32.2	27.6	41.2
Bicarbonate	10.2	11.1	10.1	16.2	19.5	15.2	21.5	24.4
Base excess	-19.6	-18.1	-17.5	-9.9	-6.2	3.5	4.7	-1

every 4 hours. Heparin, 500 units, intraperitoneal, was injected on the first day to the second day in every change and 1 time on the third day to the last day of PD. The PD was discontinued if kidney function, metabolic acidosis, and hemodynamic state were improved. Lactate-base peritoneal fluid was used for both patients. The PD fluid leaks or any complications were not seen during the PD.

After 24 hours of successful PD in the first case, the metabolic acidosis was improved and the patient had normal ABG (Table) and normal hemodynamic state. On day 3, PD was stopped and the patient was discharged on day 5. The status of second case was improved in the first 8 hours of PD and during the next days, she had stable hemodynamics, normal blood pressure, and normal arterial blood gas, and the patient was discharged 4 days after PD.

DISCUSSION

There is no potent antidote for aluminum phosphide poisoning and supportive and critical care to balance blood pH, electrolytes, and arterial blood pressure is the main treatment approach.[5] Most of the patients have low blood pressure refractory to inotropic support.[6] Lower bicarbonate levels may require sodium bicarbonate intravenously in order to increase the bicarbonate level to 18 mEq/L to 20 mEq/L.[8] It is shown that the correction of metabolic acidosis improves outcomes in aluminum phosphide poisoning.[6] Dialysis may be required for severe acidosis and acute kidney failure.[8]

We used PD as the main treatment along with conservative and routine treatments of aluminum phosphide poisoning in the treatment of severe metabolic acidosis and observed significant improvement in pH and bicarbonate levels of these patients. Both cases survived and were discharged from the hospital. We intended to treat both metabolic acidosis due to mitochondria dysfunction and lactic acidosis (hemodynamic impairment) by using PD.

There are few theories on how PD is effective in treating severe metabolic acidosis. Main physiopathology of aluminum phosphide poisoning is cellular ischemia that results in multi-organ failure, and persistent hypotension is the consequence of this process.[8] Cellular ischemia, vasodilator, and free radicals release can expedite this process and lead to death. Peritoneal dialysis by improving acidosis and hypothetically removing these toxins may lead to restoration of various organ functions.

The phosphine gas is absorbed by the gastrointestinal tract by simple diffusion and is mainly excreted by the kidneys and lungs.[9] Removal of phosphine gas from the gastrointestinal tract especially before its absorption could be helpful. Although the levels of phosphine or its metabolites were not measured in the dialysate fluid in our cases, we also hypothesize that PD could remove phosphine gas, possibly through the diffusion of phosphine from the intestinal membrane to the dialysate fluid.

CONFLICT OF INTEREST

None declared.

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Received May 2016
Revised August 2016
Accepted September 2016