Successful Treatment of Aluminum Phosphide Poisoning with Digoxin:
A Case Report and Review of Literature

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Abstract: Aluminum phosphide (AIP) is a pesticide which release phosphine gas when comes in contact with water or hydrochloric acid in stomach. Phosphine is a mitochondrial poison and interferes with protein and enzyme synthesis. Cardiogenic shock secondary to toxic myocarditis remains the most common cause of death in AIP poisoning. There is sable report on the use of digoxin for treatment of cardiac failure from AIP poisoning, although it has been used effectively for other causes of cardiac failure. Here an 18-years old female is introduced who referred to poisoning center with acute AIP poisoning and cardiogenic shock. Digoxin 0.5 mg was initially used and followed by 0.5 mg every 6 h during the first day. Digoxin was continued by 0.25 mg daily for management of cardiogenic shock until the effects of AIP resolved. The patient was discharged 10 days after admission, with full recovery. The conclusion is that administration of digoxin to AIP poisoning cases help manage to cardiogenic shock and prevent from death but needs to be confirmed by further studies.

Keywords: Phosphine, poisoning, phosphide, digoxin

INTRODUCTION

There are two kinds of pesticide which are used to protect grains and rice from pests and rodents in household that are known as rice tablet. One of them is an herbal product and poisoning with it, is not dangerous but the other one is aluminum phosphide (AIP), a fumigant which release phosphine gas when comes in contact with water or hydrochloric acid in stomach (Mehrpour and Abdollahi, 2010). Phosphine is a mitochondrial poison (Abdollahi et al., 2004) and interferes with protein and enzyme synthesis (Mehrpour and Singh, 2010; Shadnia et al., 2005, 2009). Impairment of oxidative phosphorylation and shock due to AIP poisoning may lead to multi organ dysfunction especially hypoxic damages (Mehrpour et al., 2008a).

Reported mortality rate due to AIP poisoning is too high (60-80%) and it is an emergency situation in emergency departments (Pajoumand et al., 2002; Shadnia et al., 2009; Mehrpour and Abdollahi, 2010). Although, some compounds have been introduced as decontaminant from gastrointestinal tract (Shadnia et al., 2005) but there is no specific antidote for AIP poisoning (Nikfar et al., 2011). Cardiogenic shock still remains the most common cause of death in AIP poisoning (Shadnia et al., 2011), so theoretically, treatment of cardiogenic shock may result in recovery because of digoxin cardiotoxic potential in this poisoning as hypothesized by Samei-Zadeh and Farajidana (2011). Here a case of severe acute AIP poisoning with major cardiotoxicity who was successfully survived by use of digoxin is reported.

CASE REPORT

An 18 years old female was admitted to emergency Department of Emam-Khomeini Hospital as the main referral hospital for poisoned patients located in Ardabil, west of Iran (Mehrpour and Singh, 2010), about 4 h post ingestion of one 3 g tablet of AIP. After intentionally ingestion of AIP at the home, she complained vomiting, epigastric pain and thirst, when she was transferred to a local hospital where received gastric lavage and 100 g charcoal. Then, the patient was referred to Emam-
Khomeini Hospital for better management. The patient did not have history of chronic disease or taking any medications or illicit drugs. At admission, she was agitated with Glasgow Consciousness Scale (GCS) of 14. Her Blood Pressure (BP) was not detected but others were as follow: pulse rate: 120 min⁻¹ and filiform, respiratory rate: 17, oral temperature: 35.8°C with cold and clammy extremities. Investigations revealed hematocrit: 40 and white blood cell counts: 11500. Arterial Blood Gas (ABG) analysis revealed ever metabolic acidosis with pH: 7.162; serum O₂ pressure (PO₂): 59 mmHg; serum CO₂ pressure (PCO₂): 31.7 mmHg; serum HCO₃: 11.1 mmol L⁻¹ and O₂ saturation of 92%. Her electrocardiogram was normal except for sinus tachycardia. Blood biochemistry revealed serum sodium (Na): 142 meq L⁻¹, serum potassium (K): 3.9 meq L⁻¹; serum calcium (Ca): 7.9 mg dL⁻¹, serum magnesium (Mg): 1.5 mg dL⁻¹; Blood Urea Nitrogen (BUN): 18 mg dL⁻¹, creatinine: 1 mg dL⁻¹ and blood glucose: 212 mg dL⁻¹ (Table 1). Electrocardiogram (ECG) showed sinus tachycardia with no ST-T changes. Troponin I (TPI) was negative. Her bedside echocardiogram showed severe Left Ventricular (LV) systolic dysfunction (EF = 25%), global hypokinesia, mitral valve regurgitation (MR), global increased mean pulmonary artery pressure (PAP = 26). End-diastolic size of LV was 55 mm and end-systolic size of LV was 45 mm. Left atrial size was 37 mm. Then when she was transferred to ICU, she received endotracheal intubation and mechanical ventilation and gastric decontamination with sodium bicarbonate (44 Meq, orally), permanganate potassium (1:10,000) and activated charcoal (1 g kg⁻¹, orally). Then she was treated with magnesium sulfate 6 g by IV infusion daily, calcium gluconate 4 g by IV infusion daily and adequate hydration. Due to severe metabolic acidosis she received 6 vials of sodium bicarbonate (44 mlq) stat that continued by 6 vials daily. Due to acute left heart failure and severe hypotension, dopamine (10 μg kg⁻¹ min⁻¹) was started. Additionally, she received digoxin 0.5 mg initially followed by 0.5 mg every 6 h during the first day. Digital continued by 0.25 mg daily. At day 2, her blood pleasure and pH significantly increased (Table 1). At day 3, INR were increased till 2. Echocardiography on day 3 was normal. At that time due to better condition of patient she was extubated. At day 4 of admission, she was transferred to the poisoning ward. At day 6, infusion of digoxin was stopped. She was discharged 10 days after admission, with full recovery.

**DISCUSSION**

AIP is a highly toxic pesticide that is often used for suicide in many countries as a cheap, easily available and effective grain fumigant and rodenticide (Proudfoot, 2009; Shadnia et al., 2005, 2008). After ingestion, it produces serious systemic effects within an hour. The toxic effects and prognosis is highly dependent on dose, freshness of tablets, immediate vomiting after the onset of poisoning, lower GCS, hypotension, acidemia, existence of abnormality in ECG, hematocrit, leukocytosis, hyperglycemia, BUN and SAPS-II at the time of admission in the hospital (Shadnia et al., 2010). The most common signs and symptoms in AIP poisoning are gastrointestinal symptoms and profound circulatory collapse which results in congestive cardiac failure and acute respiratory arrest (Shadnia et al., 2009). Profound cardiogenic shock and circulatory collapse is the result of direct effects of AIP on cardiac myocytes (Shadnia et al., 2005, Proudfoot, 2009). Supporting by previous reports, the present case showed severe hypotension, hyperglycemia, acidemia, low ejection fraction and LV systolic dysfunction confirmed by echocardiography. Echocardiographic performance in the present case showed marked LV systolic dysfunction. Follow-up of cardiac function by echocardiography in few cases of AIP poisoning revealed
dysfunction of the left ventricle (Bhasin et al., 1991; Gupta et al., 1995). In fact, cardiotoxicity of this agent varies from minor electrocardiographic abnormalities such as a single sinus tachycardia to severe cardiac contractility depression secondary to toxic myocarditis (Saneei-Zadeh and Farajidana, 2011). Other surveys on cardiotoxicity of AIP poisoning showed an increased left ventricular dimension, left ventricle hypokinesia, akinesia, low ejection fractions, severe hypotension, increased systemic venous pressure, normal pulmonary artery wedge pressure and ECG abnormalities (Gupta et al., 1995; Bhasin et al., 1991).

After treatment with digoxin in the present case, the echocardiography on day 3 became normal. It has been reported that patients who survived AIP poisoning have normal echocardiographic findings on day 5 (Gupta et al., 1995).

Till now, researchers are trying to reduce mortality rate with different agents such as N-acetylcysteine and trimetazidine (Proudfoot, 2009), but none of them have been effective yet. In addition, some surveys have tried to show efficacy of magnesium in AIP poisoning and although its efficacy in other pesticides such as organophosphate poisoning was proved (Pajoohand et al., 2004), but its advantage in AIP poisoning is in debate. Intra-aortic balloon pump has been used to mechanically support the heart in toxic myocarditis and refractory shocks in AIP poisoning (Gurjar et al., 2011; Siddaiyah et al., 2009), but these are too invasive.

Although, our cases had a severe poisoning with AIP with marked LV dysfunction and reduced capillary wedge but after using digital, she significantly improved. In systolic dysfunction, intravenous administration of digoxin increases cardiac output and reduces pulmonary capillary wedge pressure and heart rate (Saneei-Zadeh and Farajidana, 2011). Although, in non-toxic cases of acutely decompensated heart failure, alternative therapies with superior short and/or long-term safety and efficacy profiles are available (e.g., angiotensin converting enzyme inhibitors, intravenous diuretics and other intravenous inotropes), but in cases of AIP, these treatments are less effective (Gurjar et al., 2011; Siddaiyah et al., 2009), thus digoxin can be used as adjustment therapy in addition to specific treatments for the stabilization of patients with acutely decompensated heart failure. In patients with heart failure, digoxin may be the preferred drug for slowing the ventricular rate, due to an improvement in left ventricular function. Rapid intravenous digitalization can be performed by giving 0.5 mg initially followed by 0.25 mg or 0.5 mg every 6 h until digitalization is achieved. Digoxin can be continued by 0.25 mg daily.

The present patient showed hyperglycemia that is suggested as a poor prognostic factor in AIP poisoning (Mehrpoor et al., 2008b, 2009). Treatment of hyperglycemia throughout management of the poisoning should be considered within treatment, which may improve the outcome such as that of organophosphates (Rahimi and Abdollahi, 2007). The blood glucose concentration of our case became normal after treatment and continued to remain normal.

CONCLUSION

Clinical significance of digoxin in the management of AIP poisoning should be considered in further cases to optimize the management protocol.

REFERENCES


