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Intravascular Hemolysis Due to Glucose-6-Phosphate Dehydrogenase Deficiency in a Patient with Aluminium Phosphide Poisoning

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Abstract

Aluminium Phosphide poisoning and glucose-6-phosphate dehydrogenase deficiency are two commonly seen clinical presentations in Iran. However, hemolysis associated with Aluminium Phosphide poisoning is very rare. We report a case of concurrent Aluminium Phosphide poisoning and glucose-6-phosphate dehydrogenase deficiency in a 24 year old man presenting with intravascular hemolysis.

Key words: Glucosephosphate dehydrogenase deficiency, Aluminum Phosphide, hemolysis, poisoning

Introduction

Glucose 6-phosphate dehydrogenase (G6PD) deficiency, an X-linked disorder, is the most common enzymatic disorder of the red blood cells in humans [1]. Affected patients are asymptomatic in the steady state; however, sudden destruction of erythrocytes can be triggered by drugs having a high redox potential, toxins (Methylene blue, Naphthalene (mothballs, henna)), infections, fava beans and metabolic abnormalities (eg, diabetic ketoacidosis) [2].

Aluminium and Zinc Phosphides are highly effective insecticides and rodenticides used widely to protect grain in stores and during transportation. Acute poisoning with these compounds may happen directly by ingestion of the salts or indirectly from accidental inhalation of phosphine gas generated during their use [3]. Aluminium Phosphide (ALP), known as rice tablet in Iran, is commonly available as an insecticide. ALP is highly toxic, low cost and easily accessible as a potent mole pesticide. It has emerged as a major cause of suicidal death in some Asian countries such as India and Sri Lanka. Phosphine gas (PH3) is formed from phosphides after contact with water, particularly if acidic. Phosphine gas disrupts mitochondrial function by blocking cytochrome C oxidase. In addition to producing energy failure in cells, free radical generation increases, resulting in lipid peroxidation. ALP ingestions over 500 mg are often fatal and the product is sold in 3g tablets. Phosphides have a rotten fish odor, although the resultant phosphine gas may have a garlic odor. Phosphides rapidly produce toxicity, generally within 30 minutes of ingestion; and death may follow in less than 6 hours. Phosphides are potent gastric irritants and profuse vomiting and abdominal pain are often the first symptoms. Respiratory signs and symptoms include tachypnea, hyperpnea, dyspnea, pulmonary edema and chest tightness that may progress to acute lung injury over days. Tachycardia, hypotension, refractory shock, acute renal failure, metabolic acidosis and dysrhythmias may also develop. Phosphine-induced dysrhythmias include atrial fibrillation and flutter, heart block, ventricular tachycardia and fibrillation. Central nervous system toxicity includes coma, seizures, and delirium. Other uncommon features include disseminated intravascular coagulation (DIC), and hepatic necrosis. Treatment is only supportive with no known antidote or specific therapy, although

the use of antioxidant and N-acetyl-L-cysteine (NAC) therapy warrant further study [4].

Report of the case

A 24 year old man who was a known case of G6PD deficiency was referred to Loghman-Hakim poison center, Tehran, Iran, 2 days after intentional ingestion of one ALP tablet. He had frequent episodes of vomiting soon after ingestion and was referred to this center after initial stabilization including gastric washing with potassium permanganate and fluid therapy in an emergency department. He denied any ingestion of other drugs or fava beans or contact with other toxins. On physical examination the patient was fully conscious, and icteric. He was febrile; his blood pressure was 110/70 mm Hg, his respiratory rate was 22/min, and his pulse rate was100/min. He became icteric and febrile 12 hours before referral to this center, and his urine changed to dark brown. The results of other general physical examinations were unremarkable, except mild suprapubic tenderness with abdominal pain. Laboratory tests on admission showed normal ABG, electrolytes, normal PT and PTT, negative Coombs' test, normal urinary tract and normal hepatobiliary system sonography. His electrocardiogram showed only sinus tachycardia. His CBC indicated normal WBC and platelet count,

With Hg=9.3 gr/dl and Hct=29.3. Liver function tests indicated ALT=50, AST=134, Total Bil=12.5, D Bil=0.5, and in urine analysis hemoglobinuria (4+) was detected. His hemoglobin dropped to 3.9 gr/dl thus 2 units of packed cell was transfused and he was hydrated. Within the next day, the urine cleared, showing only 1 to 2 red blood cells per high power field and no changes in CBC, electrolytes or kidney function tests. His urine culture was negative. He improved clinically and was discharged.

Discussion

The hematologic complications caused by Aluminium Phosphide poisoning are not common [5]. Phosphide which acts by liberating Phosphine gas (an oxidant) can cause hemolysis, however hemolysis associated with ALP poisoning is very rare, reported in only few cases in the literature. Aluminium Phosphide has been reported to produce intravascular hemolysis in one patient who also had concomitant G-6-PD deficiency in India [6]. Microangiopathic hemolytic anemia (MAHA) and methemoglobinemia have been reported with Aluminum Phosphide poisoning and hemolysis and methemoglobinemia may complicate the course of Phosphine poisoning [7, 8]. Aggarwal et al. have reported the occurrence of intravascular hemolysis following Aluminium Phosphide poisoning in a patient with normal G-6-PDlevels [9].

Conclusion

This case illustrates the potential of Aluminium Phosphide poisoning to trigger hemolysis in patients with G6PD deficiency. Although hemolysis is a rare presentation in ALP poisoning, detection of associated G6PD deficiency is imperative and contact with this chemicals in G6PD deficient patients should be avoided.

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