

# Letter to the Editor

## Protective role of G6PD deficiency in poisoning by aluminum phosphide; are there possible new treatments?

Dear Editor,

Aluminum phosphide (AIP) is an insecticide and rodenticide available in Asian markets and is a major cause of death in many countries including Iran. No definite treatment has yet been established for it. We suggest a possible treatment method for AIP poisoning based on two cases available in the literature.

A 24-year-old known case of G6PD deficiency was referred to us two days after ingestion of one AIP tablet. His poisoning had been conservatively managed. After two days, he referred again with icter with-out ingestion of any oxidant. He was conscious, febrile, icteric and had sinus tachycardia. Lab tests showed normal ABG, electrolytes, PT and PTT, negative Coombs' test results, and normal hepatobiliary ultrasonography. Normal WBC, platelet count and hemoglobin of 9.3 mg/dL dropping to 3.9 mg/L were documented. Hemoglobinuria was detected. By the next day, he improved and was discharged completely symptom-free<sup>1</sup>.

Another case of G6PD deficiency exposed to ALP<sup>2</sup> was a 22-year-old presenting with nausea, epigastric distress, vomiting and cola-coloured urine after ingestion of one ALP tablet. He denied taking any other intoxicant. On admission, he was conscious, icteric, and normotensive with mild tachycardia. Urine sample was deep brown. ABG and renal tests were normal. Liver function tests showed unconjugated hyperbilirubinaemia. A G6PD assay obtained showed deficient activity. He was conservatively managed, improved, and was discharged<sup>2</sup>.

The interesting point is that with a total mortality rate of almost 71% for rice tablet<sup>3</sup>, there are two cases of ALP poisoning presenting with definite signs and symptoms of this fatal toxicity and survive. Is it possible that these patients' common feature – G6PD deficiency – has a role in their outcome?

G6PD deficiency is the most common enzymatic disorder of RBCs in humans<sup>4</sup>. It causes sudden destruction of erythrocytes in contact with fava beans, several medications, toxins and poisons, and metabolic abnormalities<sup>5</sup>. G6PD deficiency, as known, has a protective effect against falciparum malaria attributed to the vulnerability of the parasites to the oxidative stress induced by G6PD deficiency<sup>6</sup>. It was suggested that the parasite oxidized the NADPH in RBC of the host to maintain its own glutathione (GSH) in reduced form. It was then hypothesized that G6PD-deficient erythrocytes would protect against fulminant malaria infection because utilization of NADPH by both the host erythrocyte and malaria parasite would overwhelm the limited ability of the G6PD deficient red cells to regenerate NADPH and the resultant decrement in GSH would lead to oxidant-induced hemolysis. In fact, it seems that the oxidative stress caused by a toxin in a G6PD-deficient patient lyses the RBCs and inhibits further dissemination of the parasites. Oxidation of the host RBCs does not let the parasite to keep its own glutathione in reduced form and this damages the parasite<sup>6</sup>.

On the other hand, the theory of oxidative stress and extra-mitochondrial release of free oxygen radicals has been suggested as a potential cause of ALP poisoning and death<sup>7</sup>. It seems that if we can prevent the oxidative stress caused by the phosphine gas, we can save the patient. The interesting point is that in two patients with G6PD deficiency and extensive hemolysis, the patients survived. This may mind that in these patients, the extensive RBC lysis has prevented the disseminated oxidative stress distributing by the phosphine-damaged RBCs. The other interesting idea is that can we possibly prevent further oxidative stress in ALP-poisoned patients by routes of extracorporeal elimination of the damaged RBCs? Can, for instance, blood exchange prevent the disseminated oxidative stress that is induced and distributed in the body by damaged RBCs? This may need further studies evaluating such idea in ALP-poisoned patients.

### Conflict of Interest

None.

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