Hydroxyethyl Starch, Golden Remedy in Acute Aluminum Phosphide Poisoning Treatment: A Case Report

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Abstract

High mortality rate in acute aluminum phosphide toxicity is now a threat in developing countries. After exposure to air or acidic medium of stomach, phosphine gas released. This toxin rapidly causes a multisystem toxicity. Severe hypotension, cardiac dysrhythmias, thirst, tachypnea, and severe metabolic acidosis are significant symptoms. Autopsy examinations, point to extravasation of fluid into the third space. Besides to its consequences as intravascular volume expanders, hydroxyethyl starch solution is a colloid volume expander and reduces the extra vascular leakage of albumin and fluids. As sever hypotension and cardiovascular collapse, are common causes of death in this context, hydroxyethyl starch can be resuscitative in this toxicity.

Case Presentation

A 26-year-old man was referred to our hospital due to ingestion about 3 grams of "Rice Tablet" (the common name of aluminum phosphide in Iranian druggers) for suicide. He was conscious, but he was nauseating, he feels excessive thirst and he had air hunger. In the routine examination, his respiratory and pulse rates were 18 breathes and 80 pulses per minute respectively. His blood pressure was 70/45 mmHg. In the toxicity emergency unit, potassium permanganate (1:10 000 solution) was given through a nasogastric tube to reduce phosphine gas production. Arterial blood gas examination revealed sever metabolic acidosis, with a bicarbonate level of 12 meq/lit. Therefore 100 meq of sodium bicarbonate was administered intravenously within 5 minutes. This patient spotted for poor prognosis and admitted to the ICU for close observation. Treatment with glucagone, dopamine and norepinephrine was considered as well as intravascular fluid resuscitation based on the cardiologist consult to maintain systolic blood pressure above 80 mmHg.

After two hours of admission and treatment in the ICU the second assessment was revealed deterioration of cardiac pump activity and systolic blood pressure. The further arterial blood gas examination showing progression of severe metabolic acidosis with a bicarbonate level of 8 meq/lit. Subsequently, hydroxyethyl starch (500 ml solution) was given parenterally over 6 hours therewith previous agents. Afterwards the patient was assessed again. Fortunately, systolic blood pressure became higher and reached 75 mmHg and bicarbonate level reached 15 meq/lit. Treatment was continued with a repeated dose of 500 ml hydroxyethyl starch solution within 12 hours, and the patient became stable. After 3 days he was discharged from the toxicity intensive care unit and referred to the psychiatric department for more evaluation.

Discussion

Aluminum phosphide is used as pest control in grain storage (1). Acute aluminum phosphide poisoning occurs in developing countries usually with a suicidal attempt. Phosphine gas released after contact to the acidic content of the stomach (2). Symptoms include retrosternal burning, vomiting, agitation, refractory hypotension and severe metabolic acidosis. Capillary dysfunction causes leakage of fluids to extravascular space and is responsible for progressing hypotension and tissue hypoxia. Administration of intravenous fluids combined with dopamine and dobutamine infusion considered to provide systolic blood pressure above 80 mmHg (3). There is no specific antidote and the treatment is supportive but this toxicity has a high mortality rate without being affected by the critical care progression (4). Sever toxicity usually happens, if more than 1.5 grams swallowed, within 30 minutes that rapidly progress and in less than 6 hours the patient will die (5,6). Systemic organ dysfunction due to cellular hypoxia occurs rapidly after overt toxicity. For several years it believed that inhibition of the cytochrome oxidase in the mitochondria is the causal mechanism (7, 8).
Nowadays a novel hypothesis express that reduction in organ function is triggered by declination in oxidative phosphorylation of the mitochondria, to reduced cellular metabolism and increase the possibility of cell survival. Another hypothesis express that the main event in phosphine toxicity is acute metabolic response. Leakage of fluid to the extravascular space as a result of capillary dysfunction is responsible for severe hypotension and consequently makes tissue hypoxia and metabolic acidosis. So inhibition of the enzyme cytochrome oxidase of the mitochondria mentioned as the protective mechanism not the mechanism of cell poisoning. Therefore successful fluid resuscitation might be lifesaving (9,10).

Hydroxyethyl starch, with its potency to lessen microvascular oozing from plugs created in the endothelium during conditions such as sepsis and burns, could be used as the secondary fluid of resuscitation in this toxicity (9,11,12,13).

In our case treatment with hydroxyethyl starch in a severely toxic patient with profound hypotension improved cardiac output, oxygen delivery, and lowered blood lactate levels.

We concluded that therapy with this golden remedy was his salvation.

References

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