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Letter to the Editor

A new concept against the priority of vasoactive agents in the management of severe hypotension associated with aluminum phosphide poisoning

Dear Editor,

Acute aluminum phosphide toxicity can create havoc for clinical toxicologist. Despite advances in the management of critically ill patients, the survival rate of this toxic substance remains very low[1]. So some questions regarding the safety and efficacy of the current treatment protocols were formed and novel ideas against the conventional unsuccessful managements were developed[2-8].

It is a common knowledge that phosphine liberation from the mother compound is responsible for symptoms. Even though, several mechanisms were proposed, some, including oxidative stress, cytochrome-C oxidase inactivation, heart failure, and disruption of vascular integrity in scientific literature, without any evidence on human case studies[1,9]. It has been mentioned that, the metal ingredients can contribute to poisoning. Even though very high concentrations were found in many corpses, the significance of these metal ingredients is still unclear[1].

One of the most important features is hemodynamic instability which response poorly to massive intravenous crystalloids. In fact, excess transudation of fluid to third space is a constant finding in autopsy reports[8,9]. This can accentuate the theory of fluid loss, which leads to refractory hypotension. It is clear that lactic acidosis is a consequence of inappropriate tissue perfusion and oxygenation[2,9-11].

However, the current opinion is that "myocardial injury is a consequence of direct effect of phosphine on myocardiocytes". There is some evidence that if a patient survives, normal cardiac function will return on the fifth day[12-15]. We recently had argued that impaired metabolism due to tissue hypoperfusion is the main reason[2,8] which can explain normalization of systolic and diastolic function after acute phase.

There have been some successful case reports, using novel treatment protocols such as: intra-aortic balloon pump (IABP), which mechanically supports the heart function[16]; cardioactive steroids, which probably exert its effects by improving the neurohormonal profile[17-19]; the hydroxyethyl starch, which utilizes its effects by relief of vascular wall integrity disruption[20]. However, none of these were evaluated in any structured clinical trial. Therefore, the current accepted strategy to overcome severe hypotension includes prompt resuscitation with intravenous crystalloids and vasoactive agents[21].

We should take in consideration that the general scenario after ingestion of ALP is a refractory hypotension, which develops within the first few hours and usually does not respond to massive intravenous crystalloids, hence, administration of vasoactive agents in the second step is considered. Despite the fact that some provisional improvement occurs, cardiac dysrhythmias are common which leads to fatality[22]. The administration of vasoactive agents is a routine protocol for hypotension management, but it has not been proven in any study for ALP toxicity patients. In addition, there is no acceptable guideline on the management of hypotension for these patients.

The most important issue is that the receptor selectivity of vasoactive agents is dosage dependent. Logically we can expect varying effects on adrenergic as well as dopaminergic receptors with high dose treatment for patients suffering from severe hypotension[23]. Contrary to the theoretical positive effect of vasoactive agents on hemodynamic stabilization by stimulation of adrenergic receptors, it can lead to increase in myocardial oxygen consumption as well as decrease in oxygen delivery due to induction of tachycardia and coronary vasoconstriction. So, they can induce myocardial ischemia which leads to cardiac arrhythmias[11,20].

Therefore, the idea is that the priority in using vasoactive agents, especially those with more β-receptor stimulant properties, need to be revised and should be considered as the last alternative choice. It should be only restricted to the patients who are fully resistant to treatment with digoxin or colloidal, and crystalloid volume expanders. Where using advanced measures, such as intra-aortic balloon pump (IABP), is technically unavailable.

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Conflict of Interest

The Authors declare that they have no conflict of interests.

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