

Letter to the editor

## IS THE USE OF CARDIOACTIVE STEROIDS APPROPRIATE IN MANAGING ALUMINIUM PHOSPHIDE POISONING-INDUCED HEART FAILURE?

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Following the publication of a review on aluminium phosphide poisoning in your journal, as well as another letter to the editor regarding this subject, we would like to add to this discussion (1, 2). Patients with acute aluminium phosphide poisoning generally suffer from multiple organ dysfunction due to diminished mitochondrial activity and oxidative phosphorylation. This is the result of the direct effect of toxins on mitochondrial performance (3, 4).

As Shadnia et al. (5) stated, this toxicity may, among other consequences, lead to congestive heart failure. Cardioactive steroids were until recently considered as a supposition that improves cardiac functioning and were used in the treatment of heart failure (6).

In fact, in the acute phase of poisoning, the clinical presentations include: hypovolemia, shock, tissue hypoxia, decreased oxygen consumption, and decreased cardiac output, as well as accumulation of serous fluid in body cavities and congestion of vital organs. Even though these symptoms resemble manifestations of heart failure, Marashi et al. (7) believe that the real reasons behind these symptoms are the transfer of intravascular fluid into the third space and the consequent hypovolemic shock that results from the breach of vascular wall integrity, therefore causing metabolic acidosis.

Similarly, other studies (8) have shown that, despite the high central venous pressure and hypokinesia of the left ventricle, volume resuscitation with excessive amounts of fluid in patients with acute aluminium phosphide poisoning cannot be associated with severe problems such as pulmonary oedema (9).

Thus, in contrast to Shadnia et al. (5), we believe that the “heart failure” associated with this toxicity is not true heart failure. In addition to the break redox activity of myocardiocyte mitochondria in the context of diminished phosphine-induced oxidative stress (3, 4), Shapiro (10) asserted that metabolic acidosis could also cause decreases to cardiac function via intracellular acidosis. Moreover, some scientists believe that the inhibition of metabolism and necrosis of the myocardiocytes may lead to the generation of reactive oxygen radicals (11). It is clear that in such conditions, the polarization of myocardiocytes is not conceivable. Thus, the inhibition of  $\text{Na}^+/\text{K}^+$ -ATPase by a cardioactive steroid does not have an important role in improving cardiac function.

Other studies (12) have indicated that cardioactive agents should not be used in the management of acutely decompensated heart failure patients. Although the use of cardioactive steroids is associated with

reducing hospitalization time in cases of heart failure, it does not have a role in reducing mortality (13).

Finally, we should keep in mind that hypomagnesaemia, another important result of aluminium phosphide poisoning, can be amplified by cardioactive steroid-induced magnesuria making the patient vulnerable to refractory arrhythmias (8, 14).

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