

Letter to the Editor

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Hydroxyethyl starch should not be used to manage severe aluminium phosphide poisoning

Zvonko Rumboldt

Split University School of Medicine, Split, Croatia

In the *Archives* January issue (1), I have read with interest and surprise a letter by Marashi and Nasrabadi concerning the use of hydroxyethyl starch (HES, hetastarch; best known under the registered name Plasmasteril) to treat severe aluminium phosphide (AIP) poisoning.

At the current level of scientific evidence such a claim seems unacceptable.

Aluminium phosphide poisoning (the compound is commercially available as a fumigant for stored cereals) is believed to be caused by phosphine (PH₃), as it induces mitochondrial damage and severe multi-organ failure (2). The poisoning is mostly reported from the Indian subcontinent, and is largely unknown in Europe. The management is exclusively supportive (gastric lavage with KMnO₄ solution, treatment of shock, lactic acidosis, etc.), since there is no specific antidote (2, 3). Some traditional (e.g. coconut oil) or “modern” interventions (e.g. haemodialysis) have not been adequately assessed, and rest instead on pathophysiological assumptions, anecdotal reports, and wishful thinking (2, 3).

Lactic acidosis type A, a form of high anion gap metabolic acidosis, is just one of the many aspects of AIP poisoning with fatal prognosis. Treatment with NaHCO₃ in this situation is highly controversial, as the authors (1) appropriately underscore. However, the suggested administration of colloid solutions is potentially even more dangerous. The authors corroborate their proposal by quoting just one hypothetical paper of their own on the role of HES in acute AIP poisoning (4) with no further experimental evidence. It is not clear whether they are advocating starch solution administration at arterial pH >7 (as stated in the letter) or <7, or what the dosage of 500-1000 mL of HES really means (i.e. isotonic, 6 g L⁻¹, or hypertonic, 9 g L⁻¹ solution)?

Contrary to their claim (1), colloidal solutions such as dextrans, pentastarch, polygeline, and HES in particular may worsen metabolic acidosis and shock, possibly due to

the substance’s deposition in the tissue and its interaction with fibrinogen, leading to even more severe coagulopathy and organ failure (5). Crystalloid solutions are better tolerated and equally effective plasma expanders (6).

Beware of ill-founded therapeutic enthusiasm!

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