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Can sodium bicarbonate really help in treating metabolic acidosis caused by aluminium phosphide poisoning?

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Management of aluminium phosphide (AlP) poisoning seems to have seen some changes over the recent years (1). We believe that one of the reasons was the review article (2) and letters to the editor (3-5) published in your journal between 2012 and 2013. However, we still face problems in treating severe cases, who generally present with refractory metabolic acidosis and whose prognosis is poor (6). To combat this life-threatening complication, some recommend administration of large amounts of sodium bicarbonate ($NaHCO_3$) (7), but our experience is that this strategy does not address the problem.

Even though controversy continues about the exact mechanism of AlP toxicity and metabolic acidosis, all agree that AlP toxicity leads to H^+ ion efflux from the cell and, when H^+ diffuses outside the cells, to metabolic acidosis (2, 8-11).

When administered, sodium bicarbonate splits into Na^+ and HCO_3^- ions in the extracellular compartment. However, as the cell membrane prevents HCO_3^- ions to enter the cell, the increase in extracellular pH does not counter intracellular acidosis. Moreover, bicarbonate and H^+ ions react together in the acidic medium and produce carbonic acid, which dissociates to H_2O and CO_2, and CO_2 can readily diffuse across the cell membrane. The only defence against increased CO_2 is hyperventilation, which usually is not sufficient to prevent CO_2 from accumulating within the cell. At high enough concentrations, CO_2 will react with H_2O to produce carbonic acid (10) and intensify intracellular acidosis.

Graf et al. (12) tested the treatment of lactic acidosis with sodium bicarbonate in dogs. They have found that visceral, intracellular, and arterial pH drops, presumably due to increased lactate production and CO_2 load. They also observed a continuous decrease in visceral blood flow rate. It seems that the sole benefit of sodium bicarbonate administration in acidosis is that it may improve heart preload and increase the tonicity of the intravascular fluid. Furthermore, Cooper et al. (13) reported no beneficial haemodynamic effect of sodium bicarbonate in patients with severe lactic acidosis.

The conservative opinion about acidosis treatment is *not* to discourage the use of sodium bicarbonate as a routine medication (11, 14, 15). Dellinger et al. (16) found no evidence supporting routine use of bicarbonate therapy in severe sepsis and consequently hypoperfusion-induced lactic acidosis. However, they do not disagree with the use of sodium bicarbonate in patients whose pH is lower than 7.15. A survey conducted by Kraut and Kurtz (17) has indicated that two-thirds of critical care and nephrology physicians decide to administrate a base (mostly sodium bicarbonate) when acidosis becomes severe (pH below 7.00). They generally believe the initial efforts must concentrate on correcting the underlying disease.

Our experience is that patients with AlP poisoning and induced shock with arterial pH >7.00 require administration of 500 to 1000 mL of hydroxyethyl starch, which can effectively resolve symptoms of shock and significantly increase arterial pH without additional administration of sodium bicarbonate.

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