DO LESS FOR MORE WITH ACETAZOLAMIDE POISONING

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Objectives: We describe a case report of a 5-year-old child that presented to the emergency department after an accidental ingestion of 2500mg of acetazolamide.

Case report: A previously well 5-year-old boy, weighs 22.7 kg accidentally ingests 10 x 250-mg acetazolamide. Three hours after the ingestion, he developed a headache, dizziness and lethargy. On initial assessment, the child was alert, cooperative and in no distress. Vitals signs were normal. The respiratory rate peaked at 28 breaths per min. Urinalysis showed a specific gravity of 1.01 and a pH of 8.5. Venous blood gas showed a normal anion gap metabolic acidosis (Table 1). His renal function was normal with a creatinine 32µmol/L. Urine output within the first 12 hours of arrival was in excess of 100ml/h. The child was admitted for observation and supportive care of intravenous fluid with potassium supplement. He was discharged home on third day with no complications.

Discussion: Acetazolamide is a carbonic anhydrase inhibitor, but has limited use as a diuretic due to its transient action and known development of metabolic acidosis with chronic use.[1] Other indications for acetazolamide are epilepsy, glaucoma, idiopathic intracranial hypertension and acute mountain sickness. There were only 2 published cases of acute acetazolamide poisoning, both cases were treated with intravenous sodium bicarbonate.[2,3] Our case demonstrates survival in acute acetazolamide toxicity without the use of intravenous sodium bicarbonate.

The main pharmacological effect in acetazolamide acute toxicity is the inhibition of carbonic anhydrase in the luminal membrane of the proximal convoluted tubules of the kidney, hence preventing sodium bicarbonate reabsorption, resulting in metabolic acidosis and diuresis. Sodium bicarbonate is often used to manage acetazolamide toxicity. However, the use of NaHCO3 is not without adverse effects[4]: hypokalaemia and cardiac arrhythmias, reduction in ionised calcium and subsequent decrease in cardiac contractility and catecholamine response, QT prolongation and arrhythmogenic potential and paradoxic intracellular acidosis.

Conclusions: Acute acetazolamide toxicity may be managed effectively and safely in a conservative manner without the use of sodium bicarbonate therapy.

[1] Kassamali R, Sica D. Acetazolamide: A Forgotten Diuretic Agent. Cardiology in Review 2011; 19: 276-278.
[2]Baer E, Reith DM. Acetazolamide poisoning in a toddler.J Paediatr Child Health. 2001 Aug;37(4):411-2.
[3]Altay S et al. Mortal suicidal acetazolamide intoxication in a young female.Anadolu Kardiyol Derg. 2014 Jun;14(4):408-9.
[4] Sabatini S, Kurtzman N. Bicarbonate Therapy in Severe Metabolic Acidosis. J Am Soc Nephrol April 2009; 20 (4): 692-695.

Table 1: Acid-base and electrolyte results of the child who has ingested 2500 mg acetazolamide.

Time post ingestion (h)	pH	CO2 (mmHg)	HCO3 (mmol/L)	Base Excess (mmol/L)	Lactate (mmol/L)	Sodium (mmol/L)	Potassium (mmol/L)
5	7.23	45	18.1	-8.1	1.2	140	3.7
16	7.25	38	16.2	-9.6	1.1	143	4.0
19.5	7.22	44	17.2	-9.2	1.4	139	3.5
24	7.31	30	14.8	-10.2	1.4	136	3.8
36.5	7.31	35	17.4	-7.6	1.8	139	3.8