

Case

A 3.5-year-old boy was found unconscious after ingesting an uncertain amount of a colourless and odourless liquid from a plastic bottle that he found in his mother's bag. The mother knew that the bottle contained X and immediately took the child to hospital. X was obtained from a jewellery factory in China, and was usually used for electroplating. The child was unconscious, and muscle twitching was noted. His Glasgow Coma Scale (GCS) score was 5/15 (eye, 3; verbal, 1; motor, 1), heart rate was 60 beats/min, oxygen saturation (SaO₂) was 95 to 97% with 100% oxygen supplementation, and blood pressure was undetectable. He developed cardiac arrest and required cardiac massage en route to a regional trauma centre. In the emergency room, intravenous adrenaline, Y and Z, were promptly given. His GCS score was 3/15, pupils 3 mm and reactive, and SaO₂ was 90%. Laboratory tests showed that his blood lactate level was 19.7 mmol/L (reference range, 0.5-2 mmol/L), oxyhaemoglobin fraction was 79% (90-95%), carboxyhaemoglobin fraction was 0% (0.5-1.5%), and methaemoglobin fraction was 12.2% (reference level, <2%). The child was intubated, and transferred to the paediatric intensive care unit (PICU). When intravenous W was administered, the urine turned red, indicating the adequacy of treatment (Fig). In the PICU, his pH was 6.98 with base excess of -22 mmol/L. His initial blood cyanide level was 0.75 mg/L. Electroencephalography showed no electrographic seizure. Computed tomography (CT) of the brain showed no cerebral oedema, but magnetic resonance imaging showed oedema bilaterally over the basal ganglia and cerebellum. The child was extubated on the same day, and transferred to the general ward for further care 2 days later. He was noted to have dystonia and fluctuation of mood and behaviour. Subsequently, he developed generalised spasticity and seizure disorder, and CT scan revealed diffuse ischaemic changes.

What were W, X, Y, and Z?

Discussion

X was potassium cyanide, Y was sodium thiosulphate, Z was sodium nitrite, and W was hydroxocobalamin.

Many cyanide-containing compounds are colourless, odourless, and highly toxic.¹ The binding of cyanide to the enzyme cytochrome c oxidase prevents transport of electrons from cytochrome c oxidase to oxygen.² Elevated oxygen content of



FIG. (a) The characteristic purple-red-coloured urine following treatment with W for X poisoning. (b) The digits remained pink despite intracellular hypoxia

venous blood is often present in cyanide poisoning.^{1,2} In the setting of poisoning with compounds that produce cyanohaemoglobin, methaemoglobin, or

sulphaemoglobin, the pulse oximeter is fooled by the mixture of haemoglobins. The only way to know the oxygen saturation or blood oxygen content is to measure these values with a co-oximeter able to distinguish between the multiple haemoglobins present.

Antidotes to cyanide poisoning include hydroxocobalamin and sodium nitrite, which release the cyanide from the cytochrome system so that cyanide can combine with thiosulphate to produce the comparatively harmless thiocyanate.¹ Hydroxocobalamin detoxifies cyanide by binding it to form cyanocobalamin, a non-toxic compound excreted in the urine.² Transient reddish-brown discolouration of the urine and mucous membranes are common and are attributed to the red colour of the hydroxocobalamin molecule.

Workplace substances containing cyanide or cyanogenic compounds are potential sources

of paediatric poisoning. Plastic bottles of common proprietary drinks are often used to store dangerous chemicals at home, sometimes even in the refrigerator.³⁻⁵ These authors have previously reported a boy who became comatose after drinking methadone from a plastic bottle in the refrigerator.³ Children could easily mistake these bottles for their favourite drinks. Parents with young children must be warned about this exceedingly dangerous practice.

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References

1. Geller RJ, Barthold C, Saiers JA, Hall AH. Pediatric cyanide poisoning: causes, manifestations, management, and unmet needs. *Pediatrics* 2006;118:2146-58.
2. Mégarbane B, Delahaye A, Goldgran-Tolédano D, Baud FJ. Antidotal treatment of cyanide poisoning. *J Chin Med Assoc* 2003;66:193-203.
3. Hon KL, Ho JK, Hung EC, Cheung KL, Ng PC. Poisoning necessitating pediatric ICU admissions: size of pupils does matter. *J Natl Med Assoc* 2008;100:952-6.
4. Hon KL, Leung TF, Hung CW, Cheung KL, Leung AK. Ingestion-associated adverse events necessitating pediatric ICU admissions. *Indian J Pediatr* 2009;76:283-6.
5. Hon KL, Leung TF, Cheung KL, et al. Severe childhood injuries and poisoning in a densely populated city: where do they occur and what type? *J Crit Care* 2010;25:175.e7-12.