THE PERILOUS GULP

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Introduction

Cyanide is a potentially lethal tissue asphyxiant. It is present in structural fires, fire of silk, processed wool, and synthetic rubber, also in industries of electroplating, jewellery production, and polishing. It is naturally present in pits of peaches, bitter almonds, apricots, and cassava roots and also present in nitroprusside. Cyanide poisoning can occur following exposure to cyanide containing elements via inhalation, ingestion, transdermal or parenteral. Although it is uncommon, it must be recognized rapidly to initiate prompt life saving measures.[1][2][3]

Case Report

A one-year and eight-month-old boy was brought to our Emergency department on 31st January 2020, with history of ingestion of a silver polishing solution, bought from a goldsmith. The child was found alone in the kitchen by his grandmother, with a previously half-filled but now emptied bottle. Upon arrival at the Emergency department, child was unresponsive, pupils were 4mm bilaterally but sluggish, normotensive but bradycardic. Subsequently, child was intubated. Initial venous blood gas showed lactic acidosis with elevated anion gap. Full blood count showed thrombocytopenia with deranged coagulation profile, liver, and renal profiles. He was treated for accidental cyanide ingestion. Initial supportive treatments were started, oral thiamine was given and exchange transfusion was done. IV hydroxycobalamine 750mg was only able to be given on the next day. Serum cyanide level was obtained later and showed the result of 1.73 mcg/ml. However, the child had developed toxic encephalopathy, as shown in his brain MRI, and multiorgan failure. He expired on day 14 of admission.

Hb	11.7	AST	39	pH	7.19
TWC	23	LDH	317	PCO2	59.1
PLT	273	CK	162	PO2	46.4
Na	138	PT	15.9	HCO3	23.2
K	4.5	INR	1.31	BE	-4.9
Cl	105	APTT	59.4	Lac	17.2
Urea	4.1				
Creat	38				

Table 1: Blood investigation upon admission

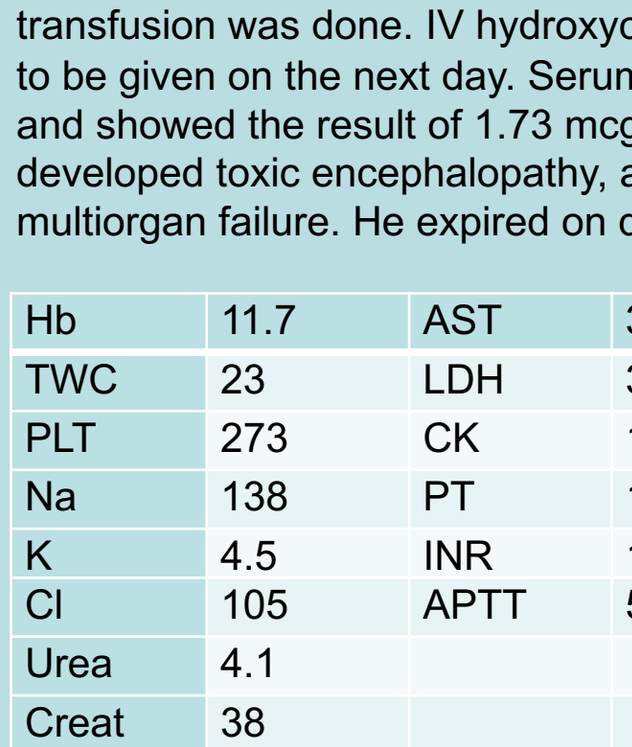

Fig 1: CXR upon admission.

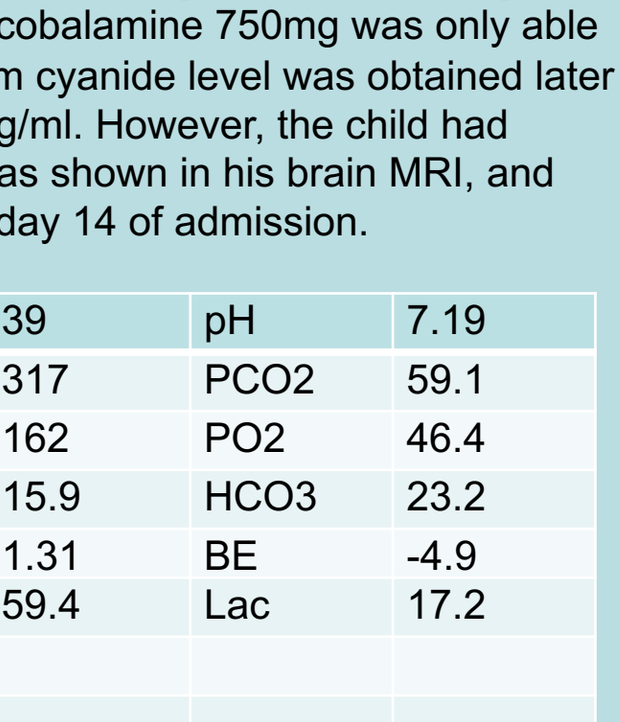

Fig 2: CXR shows pneumonitis on day 2 of admission

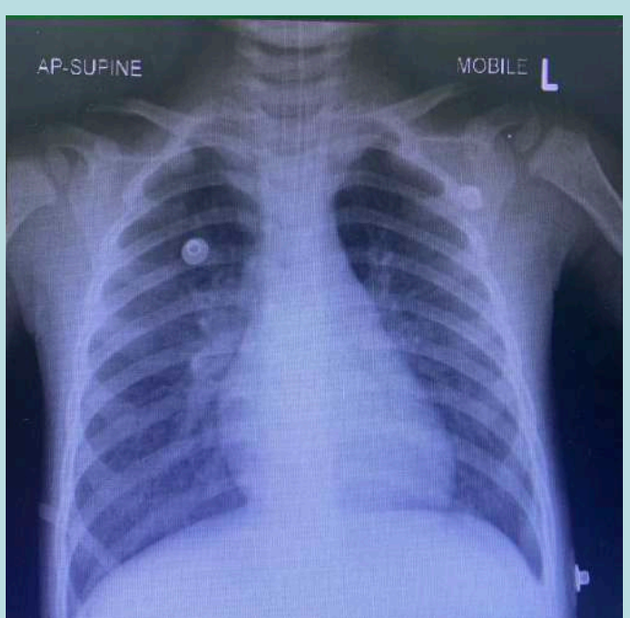

Fig 3: Multifocal infarcts at bilateral frontal lobes, midbrain and cerebelli.

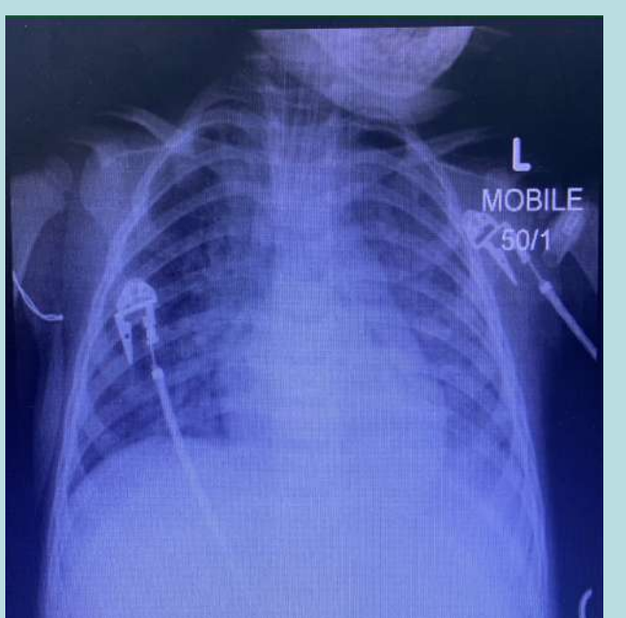

Fig 4: Moderate hydrocephalus.

Discussion

Diagnosis of cyanide poisoning might be difficult due to the lack of any specific toxidrome. The initial clinical effects are non-specific. The clinical manifestations reflect rapid dysfunction of oxygen sensitive organ particularly the central nervous system (CNS) and cardiovascular system (CVS).[4]

The CNS manifestations are typical of progressive hypoxia, including headache, anxiety, agitation, confusion, lethargy, seizure and coma. The CVS manifestations are complex, starting with an initial period of tachycardia and hypertension and ultimately bradycardia and hypotension.[5]

In this case, the solution identification was problematic since the bottle was not labeled. The child was treated for cyanide poisoning based on the Emergency physician's experience, a suggestive history, hemodynamic instability, and lactic acidosis.

For the confirmation of the diagnosis, serum cyanide level was outsourced to Department of Chemistry Malaysia in Petaling Jaya and was only available two weeks later. The delayed antidote administration was because it was not available at our center and because it had to be delivered from Sarawak General Hospital in Kuching by flight.

Fig 5: The silver polishing solution at the goldsmith

Conclusion

Physicians need to have a high level of suspicion of cyanide poisoning for an unresponsive patient with lactic acidosis and potential access to cyanide. Other challenges faced in managing this case were the lack of a readily available confirmatory test and antidote at our center.

Acknowledgement

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Declaration of conflict

The authors report there is no declaration of conflict.

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