Cassava (Ubi Kayu) Poisoning in Children

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Summary

Three sisters with cassava poisoning are described. A review of the toxic properties of cassava is presented together with discussion on the methods of its preparation, its adverse effects on man, its detoxification in the body, and the treatment of its poisoning.

Key-words: cassava poisoning in children; cyanide.

Introduction

Cassava (Tapioca, "ubi kayu", manihot utilissima) is commonly cultivated and consumed. It is important that doctors are aware of poisoning caused by it. We report here three sisters with accidental poisoning after eating tapioca.

Case Report

NSI, a six year old Malay girl, was admitted to Hospital Universiti Sains Malaysia (HUSM) on 11.12.88 at 8.30 a.m.. with complaints of one episode of vomiting and diarrhoea at 5.00 a.m. that morning, after eating three to five blocks of tapioca at 3.30 p.m. the previous day. The tapioca had been grown near her house and was dug out eight days before being eaten. Her mother had peeled the tubers which were then chopped into blocks and boiled for half an hour. On admission, NSI appeared in good condition. Her vital signs were as follows:- heart rate 110/min, blood pressure 100/60mmHg, respiratory rate 30/min, temperature 37°C. She was not drowsy nor acidotic. Her pupils were equal and reactive, and the rest of the examination was normal. Investigation revealed that her blood contained 4 ug cyanide/ml; her gastric aspirate did not have cyanide or thiocyanate, and her urine was free of thiocyanate. Her haemoglobin was 12.2 g/dl, total white count 20.7 x 10^3 (P81%, L15%, E3%, atypical lymphocytes 1%), platelet count 543 x 10^3/L, blood urea 4.4 mmol/L, serum sodium 136 mmol/L, serum potassium 4.4 mmol/L, serum calcium 2.4 mmol/L, and blood glucose 7.1 mmol/L. Her liver function tests, urine and stool for microscopic examination and culture were normal. Analysis of the uncooked tapioca tuber revealed 181 mg/kg (181 ppm) of hydrocyanic acid (HCN); it was negative for aflatoxin. Her blood pressure, pulse and respiratory rates, intake/output were closely monitored; they were all normal. Supportive treatment given were stomach washout, oxygen and intravenous dextrose-saline. She did not have further episodes of diarrhoea and vomiting. Her pupils remained equal and reactive to light. She made an uneventful recovery and was discharged well.

Her younger sister, NZ (11/2 years old), also ate the tapioca at the same time. At 10.00 p.m. on that same night, she developed abdominal cramp and nausea followed by diarrhoea and vomiting. She then

became drowsy, and was sent to HUSM, but died on the way. An elder sister, WSI (8 years old), and their mother who also ate the tapioca only had vomiting and had no symptoms respectively. An investigation of food poisoning outbreak was carried out. Analysis of food specific attack rate incriminated tapioca as the culprit. Food poisoning was limited to this family; there were no other reports in the area.

Discussion

This is an attempt to report local experiences of 'ubi kayu' poisoning as there are very few reports of this kind being published. There are serious discrepancies between the blood concentrations of cyanide (4 ug/ml = 4 mg/L) and the clinical presentation of the patient. A blood cyanide concentration of 4 mg/l is usually classified as severe poisoning and patients at this level would normally present with coma, dilated unreactive pupils and deteriorating cardio-pulmonary function. Classically the clinical presentations of the 6 year old child described would be consistent with a blood cyanide level of < 2 mg/L. It must be emphasized that blood sample for cyanide level have to be collected before administration of antidotes because results of analysis become unreliable afterwards. No antidote was given in the 6 year old girl before blood sample was taken. Immediate analysis of plasma is imperative because of the pronounced instability of cyanide in plasma. During storage of blood there may be important changes in cyanide concentrations. At deep freeze temperatures (-20°C) the cyanide concentration rises immediately and after three days the concentration is more than doubled. At room temperatures (+20°C) the concentration falls gradually. In the refrigerator (+4°C) cyanide concentrations rise slightly but after four hours the concentrations remain unchanged[1]. It is more likely that the laboratory result for cyanide content was erroneous (otherwise one had to assume that the child was extremely resistant to the effects of cyanide).

Tapioca tubers contain several alkaloids, but the poisonous substance is HCN. The cyanide in tubers occurs in either free form or combined as glycoside called limarin. The tubers contain an enzyme, linase, which is capable of releasing HCN from limarin (a process held in check in nature, but occurs once tubers are dug out). The content of HCN in tapioca varies with different parts of the plant. Thus the fresh leaf contains a mean of 752 mg/kg HCN (range 328 – 1334 mg/kg), the fresh flesh (peeled tuber) contains a mean of 34 mg/kg HCN (range 15 – 56 mg/kg) and the fresh skin (rind) contains a mean 184 mg/kg HCN (range 165 – 198 mg/kg)[2]. The amount of cyanide in tapioca also varies with: (a) types of tapioca (most important), (b) parts of tuber (e.g. the lower portion has twice the cyanide content of the upper), (c) site of cultivation (some species of tapioca is sweet i.e. has lower HCN, when grown in one soil but bitter in another soil or in a different climate), (d) duration of storage (the longer the tuber is dug out and stored dry, the higher the cyanide content), (e) size of tuber (smaller tuber contains more cyanide). The lethal dose in man is 50 to 60 mg of HCN for an adult of 50 kg weight, [3] but is much less in children.

In this country the leaves and the tubers of tapioca are consumed. Some local methods of preparing tapioca are as follows: (a) young leaves are washed, pounded and fried with "ikan bilis", (b) leaves are dipped in boiling water or steamed, and (c) peeled tubers are ground into paste and steamed, or fried into cakes. Cooking removed cyanide by destroying the enzyme linase. However short boiling (e.g. half an hour) is inadequate, although the water (containing cyanide) after boiling is usually discarded. Roasting is the most effective method of destroying the enzyme. After pounding, the juice extracted is also discarded. The above methods of preparation probably accounts for the low incidence of poisoning in this country.

On ingesting tapioca, limarin is hydrolysed by gastric acid to liberate free HCN. Both free HCN from tapioca and those liberated by hydrolysis are absorbed in the stomach. The cyanide radical causes harm

by combining with cytochrome oxidase system. The cytochrome oxidase is vital for electron transfer, which is responsible for releasing energy during aerobic glycolysis. Blockage of electron transfer results in tissue asphyxia e.g. in brain and spinal cord. Tapioca cyanide intoxication occurs in two forms: (a) acute-presenting as nausea, vomiting, abdominal distension, respiratory difficulty (cyanosis, apnoea), apathy, collapse and death; and (b) chronic-presenting as optic atrophy, perceptive deafness and sensory ataxia.

The HCN content in this family's uncooked tuber was 181 mg/kg tapioca, which would place it in the severely poisonous category when based on the following classification: the non-poisonous variety has an HCN content of less than 50 mg/kg wt., the moderately poisonous 50 – 100 mg/kg wt. and the severely poisonous more than 100 mg/kg wt.[4]

The mother did not develop symptoms of poisoning like her children probably because: (a) adults are more resistant to tapioca poisoning (thus in 190 patients, Tumbelaka[4] found 8/9 fatalities were children of less than 5 years old), (b) she had consumed parts of tapioca with lower HCN content, (c) gastric hyperacidity in children might have also caused increased hydrolysis of limarin and hence predispose them to poisoning.

The detoxification of cyanide in the body is shown in Fig. 1. Treatment of tapioca poisoning is treatment of cyanide ingestion. Thus gastric lavage is performed to remove as much ingested poison as possible. Vomiting is induced e.g. by ipecac. But induction of vomiting in semi-comatose or comatose patients is contraindicated. Acidosis is corrected with sodium bicarbonate and 100% oxygen is given to maintain high oxygen tension. Lastly, specific antidotes can also be used, namely cobalt edetate, sodium nitrite and thiocyanate.

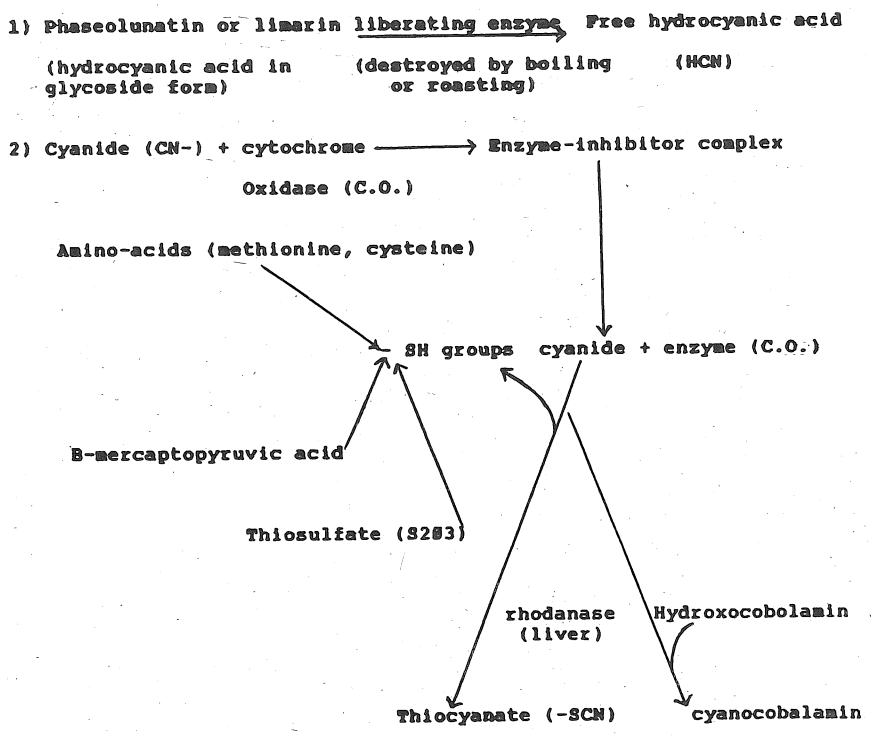

Fig. 1 : Detoxification of cyanide in the body[2]

The antidote in itself is toxic, therefore its use is only recommended for severe cases[5]. In moderately severe cases (blood cyanide concentrations between 2 – 3 mg/L) and in unclear severe cases only sodium thiosulphate solution is given. In definite or clear severe cyanide poisoning cases (blood cyanide > 4 mg/L), administer amyl nitrite via Ambu bag immediately followed by either 3% sodium nitrite solution I.V. over 5 – 20 minutes or 5% 4-dimethylaminophenol (4-DMAP) or 40% hydroxocablamin solution over 20 minutes and 25% sodium thiosulphate solution I.V. over 10 minutes. If the patient fails to respond, doses of hydroxobalamin and thiosulfate may be repeated, but expert advice is required before repeating a dose of any other specific antidote[6]. It must be stressed that specific antidote administration is effective only if it is given early (< 2 hours). Sodium nitrite itself also can cause severe and prolonged hypotension and thus need to be administered with care[5]. A relatively new antidote is dicobalt acetate[5]. It is effectively given by repeated I.V. bolus or by I.V. infusion. Given by nasogastric tube, it retards absorption of ingested cyanide. This antidote is reserved for severe poisoning when the amount of sodium nitrite and thiosulphate required would themselves be lethal. Side effects of dicobalt acetate includes sweating, angina, ventricular ectopics, severe nausea, vomiting and transient maculo-popular rash. Successful treatment with hyperbaric oxygen up to 3 atmospheres absolute combined with amyl nitrite and sodium thiosulphate has been described in a 30 year old man[7].

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