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Acute pancreatitis due to zinc phosphide ingestion

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Summary

The case of a young woman is described who suffered from acute pancreatitis related to the ingestion of zinc phosphide. This unusual complication was successfully managed with conservative treatment.

Keywords: pancreatitis, zinc phosphide

Zinc phosphide is a commonly used rodenticide in India. When exposed to moisture, or gastric juice hydrochloride, it liberates highly lethal phosphine gas (PH_3), producing various metabolic and non-metabolic toxic effects.^{1–3} Mortality due to PH_3 is very high (37–100%).¹ The clinical and pathological features are shown in box 1. The poisoning may also result in hypoglycaemia,² but not to date, acute pancreatitis. We report a case of acute pancreatitis due to zinc phosphide ingestion that was successfully treated.

Case report

A 21-year-old woman was hospitalised six hours after ingestion of zinc phosphide. On examination, she appeared drowsy and her vital signs were normal. Twelve hours after admission, she was found to be stuporose but irritable, febrile (37.4°C), jaundiced and hypotensive. Signs of metabolic acidosis and peripheral circulatory failure were present. Epigastric tenderness and generalised abdominal distension with absent intestinal peristaltic sounds were noted. Laboratory studies showed haemoglobin 110 g/l, hyperglycaemia (glucose 161 mmol/l), raised blood urea nitrogen 7.1 mmol/l, creatinine 124 $\mu\text{mol/l}$, hyperbilirubinaemia (bilirubin 72 $\mu\text{mol/l}$), raised hepatic enzymes (aspartate aminotransferase 706 U/l, alanine aminotransferase

617 U/l), hyperamylasaemia (2132 U/l), hyperkalaemia (6.2 mmol/l), hypoalbuminaemia (32 g/l), hypocalcaemia (1.9 mmol/l), glycosuria and ketonuria. An abdominal X-ray showed generalised ileus with air-fluid levels. Ultrasound demonstrated an oedematous, enlarged pancreas. She was treated conservatively with intravenous fluids, fasting, nasogastric suction, antibiotics, H_2 -blockers, analgesics, and crystalline insulin injections guided by blood glucose estimations.

Over the next four days, she became normotensive; cyanosis and jaundice disappeared; tenderness and abdominal distension decreased. Blood glucose and potassium were reduced to 60 mmol/l and 40 mmol/l, respectively, glycosuria and ketonuria disappeared but urinary amylase levels rose to 11873 U/l. Repeat ultrasound performed on the seventh hospital day showed marked decrease in pancreatic oedema. After 17 days serum albumin rose to 38 g/l, and calcium to 2.13 mmol/l. Blood urea nitrogen, creatinine, bilirubin, aspartate aminotransferase, and alanine aminotransferase were reduced to 4.26 mmol/l, 88.4 $\mu\text{mol/l}$, 18.1 $\mu\text{mol/l}$, 50 U/l, 50 U/l, respectively and normalised after an additional week with the exception of a slight rise in amylase to 66 U/l, and urinary amylase to 575 U/l.

The patient is well two months after discharge. Ultrasound and computed tomography showed a normal pancreas.

Discussion

PH_3 causes non-competitive inhibition of cytochrome oxidase, and insect catalase, and a change in the dichroic spectrum of haemoglobin, suggesting a valency change in haem accompanied by conformational changes in the prosthetic group.^{1,4} The exact pathogenesis of PH_3 -induced organ toxicity is not well under-

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Clinical features of PH₃ poisoning

- gastrointestinal system: nausea, vomiting, diarrhoea, retrosternal pain
- respiratory system: cough, dyspnoea, cyanosis, rales and rhonchi, respiratory failure (type-I)
- cardiovascular system: hypotension or shock, arrhythmias, electrocardiographic abnormalities, myocarditis, pericarditis, acute pulmonary oedema, congestive cardiac failure
- hepatobiliary system: jaundice, tender hepatomegaly, raised transaminases
- kidney: oliguric and nonoliguric renal failure

Box 1

stood, but appears to be hypoxic and, if recovery occurs, it is complete, without residual effects.¹

In their review article, Steinberg and Tenner⁵ described acute pancreatitis as a disorder with numerous causes which may result in anything from a mild disease to multiorgan failure and sepsis; it has an obscure pathogenesis, few effective remedies, and an often unpredictable outcome. Toxin-induced acute pancreatitis is uncommon. Only five toxins were mentioned as toxicological causes of acute pancreatitis, with ethyl alcohol the most common, accounting for 35% of cases.⁵ A report published in 1980⁶ implicated pyriminil (Vacor), a nitrosourea-derived rodenticide in its aetiology. In a recent report, Gasser *et al*⁷

Toxicological causes of acute pancreatitis

- ethyl alcohol
- methyl alcohol
- scorpion venom
- organo-phosphorus insecticides
- pyriminil (Vacor)
- pentavalent antimonial agents
- zinc phosphide

Box 2

Learning points

- toxin-induced acute pancreatitis is rare
- PH₃-induced organ toxicity appears to be hypoxic and if recovery occurs, it is complete without residual effects
- zinc phosphide may be included in the expanding list of toxicological causes of acute pancreatitis

Box 3

described pancreatitis induced by pentavalent antimonial agents during treatment of leishmaniasis (box 2).

Zinc phosphide should be included in the expanding list of identifiable, remediable toxic causes of acute pancreatitis.

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