Letters to the Editor

Transient hyperglycemia in zinc phosphide poisoning

Sir,
Zinc phosphide (ZnP) is an inorganic chemical used to control rats, mice, ground squirrels, muskrats, and rabbits. There are documented cases of death from massive doses of the phosphide (4–5 g) although patients may survive after acute exposure if vomiting occurs early after consumption.[1]

A 16-year-old young female presented to us after an alleged history of consumption of rat poison and vomiting. There was no history of pain in abdomen, hematemesis, bleeding, unconsciousness and breathlessness. She was not a diabetic and no family history of diabetes was present. On examination, the patient was conscious with stable vitals and bilateral constricted pupils. Other systemic examination was normal. There was no evidence of hemolysis on peripheral smear. The patient was promptly treated by gastric lavage with 1 in 1000 potassium permanganate solution. As there is no specific antidote available, she was started with symptomatic treatment. Laboratory parameters like serum electrolytes (sodium, potassium, and calcium), random blood sugar, liver function tests, and kidney function tests were normal. On the second day of admission, the patient developed fever (38.4°C) with altered sensorium. Her computed tomography (CT) scan head and other blood investigations were within normal limits. She was treated with antibiotics to which her fever responded and she regained her consciousness. On day 3, she developed hyperglycemia with her blood glucose values ranging from 450 to 500 mg% (without any intravenous dextrose infusion). She was started on subcutaneous injection insulin with 2 hourly blood glucose monitoring. There was no hyperamylasemia, ketonuria and acidosis. Her glycosylated hemoglobin (Hb1AC) and serum lipase were found to be within normal limits. Ultrasound abdomen did not reveal any pancreatic abnormality. Hyperglycemia persisted for 5–6 days and she had an uneventful recovery. At the time of discharge, her fasting and postprandial blood glucose were 70 mg% and 119 mg%, respectively. On follow-up visits after 3 months and 6 months, the patient was asymptomatic with normal fasting and postprandial blood glucose levels, without any antidiabetic therapy.

ZnP has a high mammalian toxicity as it is readily absorbed from the gastrointestinal tract and liberates phosphine gas in stomach. A dose of 5 g is dangerous and can cause death, with a latent period of about 60 minutes following ingestion.[1] Early symptoms of poisoning within 1 hour of ingestion of poison are usually nausea, vomiting, abdominal pain, chest tightness, excitement, agitation and a feeling of cold. Later symptoms may include shock, dyspnea, increased thirst, kidney failure, convulsions and coma. Purpura and asymptomatic thrombocytopenia have also been observed rarely. Early deaths may occur from cardiac damage leading to pulmonary edema.[2,3]

Hypoglycemia has been extensively described with ZnP poisoning and first reported in two cases. This hypoglycemia is hypothesized to occur due to impairment of glycogenolysis and gluconeogenesis and may be prolonged in duration.[4] A case of a young woman has been described, who suffered from acute pancreatitis related to the ingestion of ZnP.[5] The evidence of hyperglycemia as one of the presenting features has been documented in animals. Till date, after extensive literature search, we could not find a case of hyperglycemia after ZnP ingestion by humans. It may be hypothesised that it may be due to pancreatic involvement in ZnP poisoning. The treatment remains the same as for hypoglycemia. We hereby highlight that hyperglycemia can be a complication in a case of ZnP poisoning. It should be looked for in all ZnP poisoning patients, and actively treated if present.

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