Dear Editor:

A 25-year-old woman presented to the hospital 7 to 8 hours after intentionally ingesting around 50 to 60 grams of zinc phosphide (ZnP). The patient's neurological examination was normal, but she had gastrointestinal complaints including nausea, vomiting, and mild epigastric pain. Her electrocardiogram was normal and her vital signs were stable. The results of her routine laboratory tests including coagulation profile, liver function tests, complete blood count, blood urea nitrogen (BUN), creatinine (Cr), lactate dehydrogenase (LDH), creatine phosphokinase (CPK), blood sugar, sodium, potassium, magnesium, calcium, phosphate, and venous blood gas analyses were all within normal ranges.

The patient's pulse oximetry and cardiac monitoring were normal. Her plain abdominal radiography showed radiopaque materials in the pelvis and ascending colon (Figure 1).

The patient was given 60 cc of oral castor oil every 6 hours until diarrhea occurred. Since the second day of hospital admission, the patient's liver transaminases have started to rise. Throughout her hospital stay, the peak levels of aspartate aminotransferase (AST) and alanine aminotransferase (ALT) were 586 mg/dl and 1656 mg/dl, respectively.

The enzyme-linked immunosorbent assay (ELISA) results for anti-HCV and HIV antibodies and HBs surface antigen were all negative. The patient's liver ultrasound showed no abnormalities. Silymarin (Livergol®) was started at a dosage of 140 mg orally every 6 hours. Due to the increase in AST and ALT levels, intravenous N-acetylcysteine (NAC) was initiated with a loading dose of 150 mg/kg over 4 hours followed by 300 mg/kg/day. Since the seventh day, the patient's AST and ALT levels have started to decrease. On the eleventh day, she was discharged with a prescription for Livergol® at a dosage of 140 mg orally every 12 hours. She was also scheduled for a follow-up appointment in the next two days.

Zinc phosphide is a rodenticide and its lethal dose for humans is close to 50 to 500 mg/kg. Usually, 30 to 60 percent of patients who have ingested ZnP are asymptomatic [1]. The clinical manifestations of ZnP poisoning usually appear a few days after ingestion. An asymptomatic patient should be hospitalized and monitored for 3 to 4 days [1]. Common symptoms of ZnP poisoning include nausea, vomiting, abdominal pain, confusion, diarrhea, depressed level of consciousness, headache, agitation, chest pain, weakness, dyspnea, and acute kidney injury.
Other rare manifestations may include tachypnea, hyperpnea, coughing, sweating, tachycardia, hypotension, electrocardiographic abnormalities, various dysrhythmias, seizures, acute respiratory distress syndrome, hemolysis, disseminated intravascular coagulation, pancreatitis, and liver failure. Some laboratory abnormalities such as metabolic acidosis, hypo- and hyperglycemia, hypo and hyperkalemia, hypo- and hypernatremia, coagulation disorders, an increase in liver transaminases (AST and ALT), an increase in BUN, Cr, LDH, and CPK levels have also been reported [1-4].

A study has shown that if plain abdominal radiography is negative, 90% of patients will remain asymptomatic. In contrast, if the radiography is positive, there is a high possibility of acidosis, increased liver transaminases, and kidney failure [5]. There is no antidote for ZnP poisoning, so the main treatment is symptomatic therapy. Despite controversy, it is recommended to administer oral polyethylene glycol or castor oil if abdominal radiography shows radiopaque materials. Treatment with sodium bicarbonate, calcium gluconate, antidysrhythmics, magnesium sulfate, hydrocortisone, NAC, vitamin E, pantoprazole, and tranexamic acid is also recommended [1-5].

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References


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