A Case on Suicidal Poisoning Associated with Ratol and a Perspective on Yellow Phosphorus Poisoning

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Case Report

Abstract: Ratol, commonly known rodenticide, contains yellow phosphorus which if ingested results in necrobiosis, characteristically manifested in liver.

Keywords: Ratol; rodenticide; yellow phosphorus; suicidal death; poisoning.

Introduction

Ratol, a commonly known rodenticide, has been recently reported to be responsible for numerous intoxications and deaths. It is used in the management of pest populations in homes, and a suicidal ratol tablet overdose is the most common cause of ratol poisoning [1]. Accidental poisoning with same is becoming common as the product in the paste form is applied to bread to enable ingestion by rodents, making it appealing to children as well [2]. Ratol contains yellow phosphorus (YP), a severe local and systemic toxin causing damage to gastrointestinal (GI), hepatic, cardiovascular, and renal systems. When ingested, phosphorus is rapidly absorbed from the GI tract and is primarily metabolized by the liver in a first-order process. It results in necrobiosis which is characteristically manifested in the liver [3]. The clinical manifestations of YP poisoning are present in usually two phases. The primary phase is immediate within two to six hours and is characterized by irritation in GI tract with symptoms such as garlic like taste, burning in the mouth, throat, retrosternal area and epigastrium. These are followed by nausea, vomiting and sometimes diarrhoea. A peculiar feature of this kind of poisoning is that the features above abate temporarily. However, after a gap of 2-6 days, during the second stage of intoxication, original symptoms reappear in addition to jaundice. Life-threatening complications occur such as acute renal failure, oliguria and albuminuria. Nervous system complications develop in the later stages and these may include headache, restlessness, tinnitus, deafness, impaired vision and coma [3]. The potential of direct and indirect exposures and illicit use of ratol on human population have recently raised serious concerns. We report a case of an alleged ratol poisoning and describe as how potential pharmacological activity of YP, main constituent in these compound and can present difficulties for the emergency physician in management of intoxicated patients.

Case report

A 21-year-old woman presented to the emergency department with the history of alleged consumption of 2-3 pinches of rat killer poison (Ratol) poison (around 1gm) four days prior to admission in our hospital. Two hours after the ingestion, she started complaining of nausea and repeated vomiting for which she was at first admitted to a peripheral hospital. There she was provided symptomatic treatment in the form of stomach wash (gastric lavage) and antimetics. She became haemodynamically stable and symptomatically better and was discharged after one day observation. After four days of poisoning, she again developed intractable vomiting, altered level consciousness, irritability for which she was referred to our hospital. On examination her general condition was not satisfactory and she was stuporous. She had a temperature of 101.5oF degree Fahrenheit, tachycardia (pulse of 150 counts/min), a respiratory rate of 30 counts/min and a blood pressure of 100/70 mmHg in the supine position. Glasgow coma scale score was 8. The patient revealed deep icterus (over skin and sclera) and multiple ecchymotic patches of varying sizes ranging from 0.5-5 cm, were seen over extremities. Additionally, there was bilateral periorbital swelling with a chemosed conjunctiva. Nasogatric tube (No. 14) was inserted and the aspirate revealed presence of blood (75 ml). The remaining physical examination was unremarkable. Her liver function test revealed serum bilirubin levels of 9.0 mg/dl (direct 5.2mg/dl) and transaminase levels (ALT and AST) of 569.1U/L and 527 U/L (reference range < 40 U/L) She had marked elevation of serum alkaline phosphatase of 1998 mg/dl. Her kidney function tests and electrolytes were normal. Prothrombin time was prolonged 26.9 seconds and INR was 2.18. Serum total protein was 5.1 mg /dl, albumin was 3.2 mg /dl. Her haemoglobin was 8.4 gm/dl, total leukocyte count 4000

/cmm. Urine examination showed presence of 48-50 RBC's and 8-10 pus cells per high power field. HRP2 was negative. Arterial blood gases revealed pH 7.11, PCO2 20.6, PO2 75.4 and HCO3 6.3 suggesting metabolic acidosis. Sonographic imaging of the abdomen revealed fatty liver with peri gallbladder oedema with mild ascites and bilateral pleural effusion. The chest X-ray was normal. Based on the above findings, a diagnosis of acute fulminant hepatic failure (FHF) secondary to toxic hepatitis following a rodenticide poisoning was made. Patient was transfused four fresh frozen plasma and two whole blood and managed with gastric lavage ,intravenous (IV) fluids, vitamin K, She was also started on antibiotics (ceftriaxone + sulbactam), antiemetics (pantoprazole, ondensetron), tranexamic acid, N acetyl cysytein and syrup lactulose. Intake and output was strictly monitored and blood glucose was measured six-hourly. Metabolic acidosis was corrected by sodium bicarbonate infusion. Central line insertion and endotracheal intubation was done. The patient was put on ventilatory support. Despite initiation of treatment for hepatic failure, the girl progressed to stage 3 encephalopathy on day 3 and had malena for which she was administered fresh frozen plasma and packed red cells again. Her falling blood pressure was managed with intravenous fluids, vasopressors and inotropes. And despite of all available measures taken, the patient died after two days of admission. Autopsy findings revealed presence of enlarged fatty liver and gastric contents were fluorescent when examined under UV rays. It also revealed congested and inflamed skin along with subcutaneous haemorrhages.

Discussion

The presented patient allegedly ingested ratol, which is used as a rodenticide in households and agricultural farms. It is evident that large scale use of other anticoagulant rodenticide like warfarin has led to the development of resistance among rodents which lead the use of ratol more prominent. However unlike ratol, warfarin has less significant adverse effects, unless taken in significant numbers [4]. The symptoms manifested by the patient in this case report are partially consistent with the known pharmacological effects of YP. In our patient, the first manifestation of intoxication was restlessness, irritability, drowsiness, along with primary GI symptoms. McCarron MM et al., 1981 in their study have revealed high mortality of patients who initially had either central nervous system (CNS) symptoms or a combination GI and CNS symptoms as compared to GI symptoms alone [5]. Everything from mild gastroenterocolitis to fulminant failure followed by death could therefore be expected in our patient. Another important aspect in YP poisoning is dose and time interval between ingestion and treatment, which play a significant role in the survival of patient. According to published data GI symptoms are usually observed at doses less than 1mg/ kg and doses greater than 1mg/ kg or 1.5mg/dl are almost invariably fatal [6,7]. Kafeel SD et al., 2012 subsequently reported that in patients presenting late (> 24 hours) after consumption of lethal dose (>1mg/kg) developed FHF with mortality of100% [6]. Additionally, a general symptomatic treatment received during the initial phase of intoxication could invariably turn harmful for the patient as it suddenly gives impression of improvement; however, otherwise it leads to delay and impairment in proper treatment, leading to fatal outcome, as it happened in our case. Patients with YP poisoning mainly present with acute hepatic failure, coagulopathy, and deranged liver function [2, 8] and it was witnessed in our patient. Similar to the reports of Fernandez OU and Canizares LL, 1995, early elevations in transaminase, alkaline phosphatise, derangement in prothrombin time, metabolic acidosis associated were significantly associated with mortality [9]. However, phosphorus intoxication may not be limited to liver intoxication alone. Acute pulmonary oedema [10], bone marrow intoxication [11], myocardial injury [7] are other possible fatal outcomes which a clinician must be aware of while treating YP poisoning case. The current report provides evidence in line with other published reports that it is difficult to manage patients intoxicated by this compound and mainly due to two reasons. First, property of phosphorus itself, as it gets rapidly absorbed and remains stable in gut for longer period. Second, there is no specific antidote for YP and treatment is directed at removal of the poison and supportive therapy [2]. This is indeed an alarming situation and needs to be addressed on a priority basis. Prevention strategies by restricting access to this poison can be the one of the best method to avoid complications. Public as well clinicians should be made aware of lethality of inorganic phosphorus in miniscule quantities. Clinicians providing care to patients with acute hepatitis of unclear etiology should be aware that the ingestion of YP might cause acute liver failure and require more than just primary care. Moreover, this fatal poison is freely available over the counter in toxic doses at very cheaper rates (3 Rupees for 2 grams powder sachet) and there are no authentic guidelines regarding proper disposal of the same. Restricted use in public and appropriate disposal procedures should be developed in the peripheral centres based upon high incidence of YP poisoning, and death rates.

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