



A Case of Rodenticide Poisoning with Acute Fulminant Hepatic Failure: A Case Report

Authors

Dr Harshitha. R, Dr Anil Kumar. H

RRMCH

Abstract

Rodenticides are a heterogeneous group of compounds that exhibit markedly different toxicities to humans and rodents. They are among the most toxic substances regularly found in homes. Predominant rodenticide exposure is anticoagulant rodenticides, zinc phosphide, thallium, barium carbonate and phosphorous. Ratol is a rodenticide available in most of the households and is a common suicidal or accidental poisoning. Ratol contains 3% whitephosphorous (yellow phosphorous). White phosphorous is a general protoplasmic poison causing multiorgan failure at lethal doses. Liver injury is common; however, most victims of phosphorous intoxication die before overt lesions develop. White phosphorous lethal dose for humans is around 60 mg (1mg/kg). When consumed at lethal doses patients present late (>72 hours of ingestion) with multiorgan dysfunction with high mortality rate (100%). We hereby present a 34-year-old female, came with history of alleged history of consumption of around 2g of Ratol paste 2 days prior to presenting to hospital with complains of nausea and abdominal pain. Her vitals were stable having mild scleral icterus and epigastric tenderness. Liver function tests revealed elevated bilirubin levels and deranged coagulation profile. Other parameters were normal. She was given supportive treatment. Her liver enzymes and coagulation profile improved over next 10 days.

Introduction

Ratol is a rodenticide available in most of the households. It is a common suicidal or accidental poisoning¹. Ratol contains 3% white phosphorous (yellow phosphorous)². White phosphorous is a highly combustible luminescent compound³. When consumed at lethal doses patients present late (>72 hours of ingestion) with multiorgan dysfunction, with a high mortality rate (100%)¹.

Case Report

We hereby present a 34-year-old female, who was brought to emergency ward with alleged history of consumption of around 2g of Ratol paste 2 days

prior to coming to hospital, with 1-day history of nausea, 5-6 episodes of vomiting, abdominal discomfort and generalized weakness. There was no history of yellowish discoloration of skin or mucus membranes or bleeding manifestations. On examination her vitals were within normal limits and had mild scleral icterus. Her icterus increased and developed ascites by 3rd day of admission. She developed flapping tremors by 4th day of admission.

Investigations

Liver function tests revealed elevated bilirubin levels, elevated liver enzymes and deranged

coagulation profile. Other parameters were normal. Her liver enzymes which was initially high (>2000 IU) reached to normal range by day 10 of admission while her bilirubin levels

increased day by day and peaked by day 10 of admission. Her INR initially was 4.0 initially, increased to >10 by 2nd to 3rd day of admission reached to near normal by day 10 of admission.

Trend of Liver Function Test in the patient

Day	1	2	3	4	5	6	7	8	10
TB(mg/dl)	5.6	7.1	7.7	9.6	10.8	13.4	13.3	12.5	16.1
DB(mg/dl)	2.8	3.1	3.0	6.6	8.9	12.0	8.3	8.3	9.2
SGOT(U/L)	2595	2019	890	226	160	201	198	136	56
SGPT(U/L)	850	828	691	378	258	263	217	163	64
ALP(U/L)	185	477	489	439	411	479	420	411	113
TP(g/dl)	5.6	4.8	4.7	4.7	4.9	5.2	5.4	5.2	6.0

Trend of Coagulation profile in the patient

Days	1	2	3	4	5	6	9
PT(sec)	40.8	122	102	65.8	41.5	32.7	14.5
INR	4.11	>10	10.0	7.37	4.3	3.14	1.17
aPTT(sec)	90.2	45.0	45.7	43.2	68.2	38.6	33.0

Treatment

She was treated conservatively with intravenous fluids and intravenous antibiotics. She received vitamin-K injections 10mg intra-venous once daily according to protocol and received 16 pints of fresh frozen plasma over 4 days. She was started on mannitol infusion and other encephalopathy measures for a brief period when she started to develop early Hepatic encephalopathy features. With this treatment she recovered completely over a period of 10 days.

Discussion

Rodenticides are a heterogeneous group of compounds that exhibit markedly different toxicities to humans and rodents³. They are among the most toxic substances regularly found in homes. Predominant rodenticides include anticoagulant rodenticides and phosphides (aluminium and zinc phosphide)³. Nowadays, white phosphorous is also being used as a rodenticide extensively. Lethal dose of white phosphorous for humans is 60 mg (1mg/kg)^{1,3,4}. It has been stated that as little as 15 mg can cause symptoms⁴.

Mortality rate for patients who present early with symptoms of nausea and vomiting is 25%, nearly 50% when both gastrointestinal and CNS symptoms are present and almost 75% when first manifestation is restlessness, irritability, drowsiness, stupor, or coma⁵. The difference in survival rates most likely reflects the interval between time of ingestion and treatment⁵.

White phosphorous is a general protoplasmic poison causing cardiac, hepatic, renal and multiorgan failure at lethal doses². It is a potent hepatotoxin. Large doses cause shock and cardiovascular collapse³. Fulminant toxicity occurs with massive doses of about >1-2g where death occurs within 12-24 hrs with peripheral circulatory collapse without signs of hepatic or renal damage³.

Acute poisoning has 3 stages²:

Stage 1: up to 24 hrs- asymptomatic or present with local gastric irritation features².

Stage 2: 24 – 72 hrs- symptom free, mild increase in liver enzymes².

Stage 3: after 72 hrs- resolution to death, present with deranged liver functions, acute fulminant failure, coagulopathy, confusion/psychosis/hallucination/coma, hypotension/tachycardia/300

arrhythmia/cardiogenic shock, acute renal failure². Liver injury is common; however, most victims of phosphorous intoxication die before overt manifestations¹. Phosphorous causes periportal hepatic necrosis⁴.



Fig. 1: H&E staining of liver biopsy illustrating periportal hepatic necrosis

It is difficult to manage patients with white phosphorous poisoning because it gets rapidly absorbed and remains stable in gut for longer period and there is no specific antidote for yellow phosphorous¹. Treatment is directed at removal of the poison and supportive therapy¹.

Conclusion

Anticoagulant rodenticides and phosphides are the most common rodenticide poisoning. Patients present with derangement in coagulation profile or with hemodynamic instability. With increasing focus on aluminium and zinc phosphide, the significance of inorganic phosphorus as a rat poison is often disregarded⁶. Clinicians should be aware that the ingestion of white phosphorous might cause acute liver failure and require more than just primary care. As symptoms occur late (>72 hrs), early detection and treatment of deranged liver function and coagulation profile improves the outcome. Patients must be followed up for 1 week with liver function tests due to late onset fulminant hepatitis.

References

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