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A CASE REPORT OF TOXIN-INDUCED MYOCARDITIS IN YELLOW PHOSPHORUS POISONING

Toxicology

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ABSTRACT

Yellow phosphorus is a toxic substance, that spontaneously reacts in the air and when ingested causes renal, hepatic, cardiac, and multi-organ failure. Rat killer paste form usually contains elemental phosphorus. Ratol paste and powder is an easily available rodenticide in the market in India. We report a case of toxin-induced myocarditis due to yellow phosphorus poisoning. Three stages have been described after yellow phosphorus ingestion. The third stage requires advanced treatment to prevent mortality. There remains a challenge for peripheral hospitals to provide timely interventions due to lack of antidotes and advanced resuscitative measures such as inotropic supportive therapy and renal replacement facilities in order to prevent deaths. So, early and excellent supportive care is the key factor in reducing morbidity and mortality.

KEYWORDS

Yellow phosphorus, Toxin induced myocarditis, Rat killer paste poisoning

INTRODUCTION:

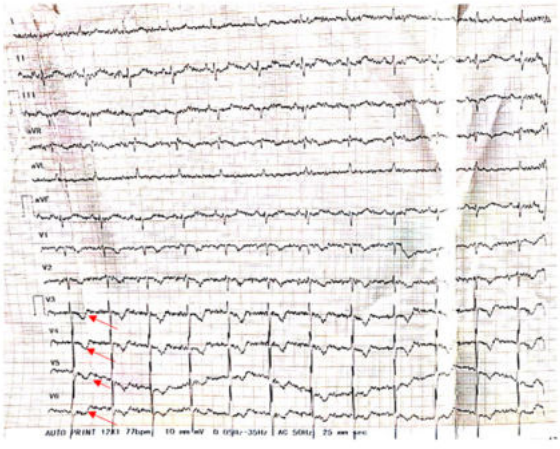
The prevention of rat-borne diseases and bites has been an important public health goal for centuries and the use of chemical rodenticides -to achieve these goals has been part of the efforts. A Rodenticide is any product commercially marketed to kill rodents, squirrels, mice, gophers, and other small animals. Ratol paste and powder is an easily available rodenticide in the market in India. Rat killer paste form usually contains elemental phosphorus. Yellow/White phosphorus is a highly cellular toxin still used in the manufacture of fireworks, fertilizer, and as a rodenticide. Red phosphorus is not absorbed and is essentially non-toxic.[1] Yellow phosphorus is a commonly used rodenticide containing 3-5% yellow phosphorus. Yellow phosphorus, a toxic substance which is a protoplasmic poison, that spontaneously reacts in the air and when ingested it causes renal, hepatic, cardiac, and multi-organ failure.[2] A dose of about 1mg/kg in adults is likely to cause significant morbidity.[1] We report a case of toxin-induced myocarditis due to yellow phosphorus poisoning.

CASE REPORT:

A 60-year-old male presented with an alleged history of consumption of rat killer paste (Ratol three tubes mixed with alcohol) poisoning on 08.09.2023. Each tube of Ratol had a net weight of 15 gm, containing 3% yellow phosphorus. The patient was initially treated in a nearby hospital for 1 day and then referred to our hospital on 09.09.2023 for further management. At the time of arrival, the patient had vomiting, loose stools, and abdominal pain. On examination, the patient was conscious, and oriented, BP:120/80mmhg PR:85/min, SpO2:99% in RA. There was no icterus or cyanosis. Systemic examination was normal. His laboratory investigations are listed in Table 1. His liver enzymes were mildly elevated with acute kidney injury. His initial electrocardiogram (ECG) was normal. The patient was treated with an injection of vitamin K, an injection of N-acetyl cysteine,4 units of Fresh frozen plasma because of a deranged coagulation profile, and also on supportive measures. USG abdomen was normal. Viral markers were negative. On day 4, the patient complained of giddiness and his extremities were cold and clammy. BP:70/40 mm hg; PR: feeble; SpO2:96% in room air. His ECG shows new onset T-wave inversion as shown in Figure 1. The patient was started on inotropes support. ECHO was taken which showed global LV hypokinesia with dilated all chambers and ejection fraction of 37%. A cardiologist's opinion was obtained. On subsequent days, the patient's condition was gradually improving and on day 8, he was weaned off from inotropes with complete recovery of deranged blood parameters. He was discharged with ACE Inhibitors and diuretics.

Table 1 Shows the Laboratory Investigation (CBC, RFT, LFT, Electrolytes, and coagulation profile) of the patient

	D-1(9/9/23)	D-4(12/9/23)	D-8(17/9/23)
Hb	12.3	14.2	13.5
TC	10,400	12,100	9800
Platelet (Lakhs)	2.22	2.16	2.39
Urea	59	97	30
S. Creatinine	1.7	1.9	0.7
Total bilirubin	1.0	0.9	0.8
Direct bilirubin	0.3	0.2	0.2
SGOT	97	69	20
SGPT	46	55	24
ALP	134	169	113
Na+	138	137	138
K+	3.8	4.7	4.3
PT	30	20	15
INR	2.5	1.5	1.0



Patient showing T wave inversion in lead V1-V5 and ST depression in V6 in figure 1.

DISCUSSION:

Yellow phosphorus is an inorganic substance also known as white phosphorus,[3] When orally ingested, it is rapidly absorbed through the gastrointestinal system and approximately 70% is accumulated in the liver within 2 to 3 hours. It accumulates to a lesser extent in the heart (12%), kidneys (4%), pancreas (0.4%), and brain (0.39%), and also leads to damage in those organs.[2]

Yellow phosphorus ingestion shows cardiac toxicity features like electrocardiographic changes, hypotension, tachycardia, arrhythmias, atrial fibrillation, and decreased ventricular contractility. Ventricular tachycardia and ventricular fibrillation have also been noted and it simulates acute myocardial infarction and persistent left ventricular dysfunction. This may be due to the direct action of the phosphorus on the myocardium and conduction fibers within the heart. It can also

occur due to secondary peripheral vascular collapse that causes a decrease in the coronary blood flow resulting in severe myocardial ischemia.[2]

Three stages have been described after yellow phosphorus ingestion. The 1st stage occurs during the 1st 24 hours in which the patient is either asymptomatic or has some signs and symptoms of Gastrointestinal irritation. The 2nd stage occurs between 24-72 hours after ingestion where the patient is asymptomatic, there may be slight increase in levels of liver enzymes and bilirubin. Sometimes patients may get discharged prematurely at this stage.[3] Nausea, protracted vomiting, diarrhea, and massive hematemesis may occur in stage 3. This stage is notably marked by clinical deterioration which encompass mainly hepatic failure, metabolic complications, CNS toxicity, and cardiovascular toxicity. Mortality is high in this stage. Death occurs usually in 4-8 days.[2] Our patient presented with stage 3 - acute kidney injury, cardiac toxicity (Dilated cardiomyopathy with Ejection fraction 37%), and coagulopathy. Even though yellow phosphorus poisoning is a common issue in India, the myocardial toxicity and ECG changes have been reported rarely.[2]

CONCLUSION:

In tropical countries, the rural population engaged in agriculture has easier access to the rat killer paste as it is available at a lower cost. There remains a challenge for peripheral hospitals to provide timely interventions due to lack of antidotes and advanced resuscitative measures such as inotropic supportive therapy and renal replacement facilities in order to prevent deaths.[4] So, early and excellent supportive care is the key factor in reducing morbidity and mortality.

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