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Case Report

Internal Medicine Section

Liver Transplant in an Unusual Case of Acute Fulminant Hepatic Failure: A Case Report

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ABSTRACT

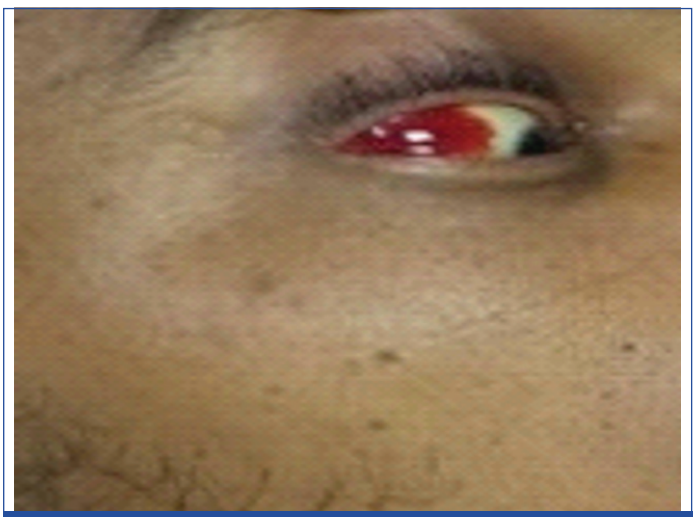
Yellow phosphorus ingestion causing acute fulminant hepatic failure is a serious condition. The establishment of health measures to monitor and prevent yellow phosphorus poisoning is of utmost importance because there is no known cure for the condition. To prevent phosphorus poisoning, it is crucial to raise awareness among the general population about its potential fatality, educate primary care physicians about the delayed onset toxidrome, and take precautions to ensure careful monitoring and reporting. Herein, we present a case of a 33-year-old male who consumed 30 grams of rat poison {Yellow Phosphorus (YP)} while under the influence of alcohol. He presented to us on day 5 with icterus and bilateral subconjunctival haemorrhage. Laboratory investigations revealed severely deranged liver function tests and raised Prothrombin Time (PT)/International Normalised Ratio (INR), indicating acute fulminant hepatic failure. The patient was started on N-acetyl cysteine, vitamin K, and received fresh frozen plasma transfusion. Despite treatment, on day 3, the patient developed grade 4 Hepatic Encephalopathy (HE) and the laboratory parameters worsened. Close monitoring revealed further deterioration, leading to the decision for emergency orthotopic Liver Transplantation (LT) on day 7. Following the transplant, the patient's liver function tests showed improvement. The subconjunctival haemorrhage and icterus resolved completely on day 3 post-transplantation. The patient was extubated on day 3 and discharged on day 5 post-transplantation. Regular follow-up appointments were uneventful. In conclusion, the present case report highlights the successful management of a patient with acute fulminant hepatic failure through early hepatic transplantation. It emphasises the importance of prompt recognition, appropriate treatment, and timely transplantation in improving patient outcomes.

Keywords: Alcohol, Liver failure, Liver transplantation, Rat poisoning, Yellow phosphorus

CASE REPORT

A 33-year-old male shopkeeper with no significant past medical history was brought to the casualty with complaints of pain in the epigastric region for five days. The pain was sudden in onset, non progressive, and dull aching. The patient also experienced vomiting (6-7 episodes per day) and loose stools (4-5 episodes per day) for three days. The patient admitted to consuming 30 grams of yellow phosphorous while under the influence of alcohol five days prior. He received treatment (unknown) at a local hospital before being brought to our hospital.

On examination, the patient's Pulse Rate (PR) was 110 bpm, Blood Pressure (BP) was 110/70 mmHg, Respiratory Rate (RR) was 18 bpm, and Oxygen Saturation (SpO_2) was 98% on room air. The patient had icterus (jaundice) and bilateral subconjunctival haemorrhage [Table/Fig-1]. Epigastric tenderness was noted upon palpation, while the rest of the examination was within normal limits.

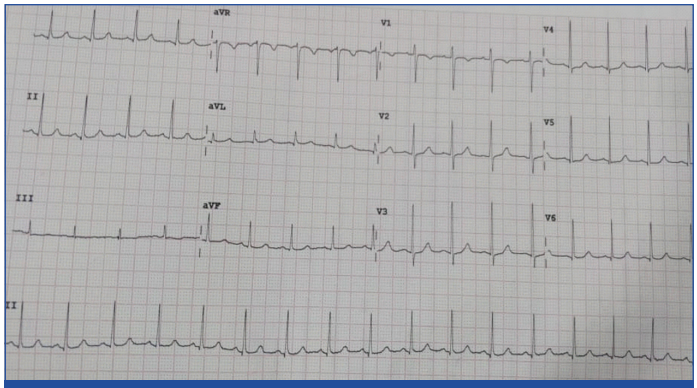


[Table/Fig-1]: Showing subconjunctival haemorrhage in right eye.

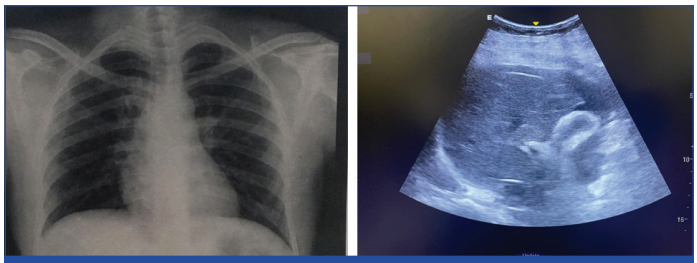
Laboratory investigations revealed bicytopenia with deranged liver function tests, elevated PT/INR, and elevated D-dimer [Table/Fig-2]. The electrocardiogram showed sinus tachycardia [Table/Fig-3]. The chest radiograph showed no obvious abnormalities [Table/Fig-4]. Abdominal ultrasonography showed mild coarse echotexture of the liver and pseudocholecystic wall thickening [Table/Fig-5]. On day 1, the patient was started on intravenous N-acetyl cysteine at a dose of 150 mg/kg in 200 mL D5 over one hour, followed by 50 mg/kg in 500 mL D5 over four hours, and finally 100 mg/kg in 1000 mL D5 over 16 hours. Additionally, the patient received a stat dose of 30 mg of vitamin K, 1 gm of meropenem three times daily, and

Investigations	Day 1	Day 3
Haemoglobin (gm%)	14.4	14.4
Total leucocyte count cells/cumm	700	7510
Platelets (/mm^3)	52000	81000
Total bilirubin (mg/dL)	7.5	25
Direct bilirubin (mg/dL)	4.7	15
Aspartate transaminase (U/L)	4950	353
Alanine transaminase (U/L)	1127	418
Alkaline phosphatase (U/L)	167	172
Urea (mg/dL)	20	20
Creatinine (mg/dL)	0.9	0.9
Serum sodium (mEq/L)	140	140
Serum potassium (mEq/L)	3.7	4.3
Prothrombin time (in seconds)	38.20	22.2
International normalised ratio	3.53	2.9
D-dimer	>10000	>10000
Urine routine microscopy Proteins RBCs, pus cells	Normal Nil Nil	Normal Nil Nil

[Table/Fig-2]: Laboratory investigations on day one and day three.
RBC: Red blood cells



[Table/Fig-3]: Electrocardiography shows sinus tachycardia.



[Table/Fig-4]: Chest radiograph posterior-anterior view shows no obvious abnormality.
[Table/Fig-5]: Ultrasonography shows mild coarse echotexture of liver, pseudocholecystic wall thickening. (Images from left to right)

transfusions of fresh frozen plasma and random donor platelets due to the subconjunctival haemorrhages.

On day 2, the patient developed grade 2 HE and the Glasgow Coma Scale (GCS) dropped to E2V2M3, leading to the patient's intubation. The same treatment was continued. On day 3, the patient developed grade 4 HE, and continuous high-grade fever was noted. The patient's liver function tests worsened [Table/Fig-2]. The PT/INR and complete blood count were closely monitored for the next couple of days. Despite treatment, the patient's liver function tests progressively worsened, leading to the decision to perform emergency orthotopic LT on day 7 due to fulminant hepatic failure.

On postoperative day 3, the patient's GCS improved to E4VtM6, resulting in extubation. Liver function tests gradually improved following transplantation. The patient made a full recovery and was discharged on the 5th day of post-transplantation. Upon discharge, the patient was advised to have regular follow-up, which was uneventful.

DISCUSSION

Rat poison sold under the name "Ratol" is highly popular in India. It contains 2%-5% (by weight) of YP [1] and has gained significant traction among farmers. YP is also used in fertilisers, fire matchsticks, and fireworks. However, YP can have fatal effects when inhaled or consumed. Acute Liver Failure (ALF) and circulatory collapse are the leading causes of death after consuming a lethal dose of YP, which is 1 mg/kg [1]. Its acute ingestion affects various organs such as the liver, kidneys, haematological system, brain, gastrointestinal tract, and heart. YP is rapidly absorbed and primarily accumulates in the liver, reaching peak concentration within 2 to 3 hours [2].

In cases of yellow phosphorus consumption, patients can present in any of the three stages [1]. In the first stage, they experience burning pain, vomiting, diarrhoea, and abdominal pain. They may also have hematemesis and a garlicky odour of breath. The vomit and stools may glow in the dark, and there may be faint fumes emanating from the stools (known as smoky stool syndrome) [3]. The index patient presented in the second stage, which occurs 5-7 days after the resolution of the first stage. This period is often treacherous and is characterised by systemic effects of absorbed phosphorus. Digestive symptoms return with increased severity around 3-4 days after the onset of the second stage. In the present case, patient presented with tender haepatomegaly, jaundice, pruritus, bleeding from multiple sites, and eventually hepatic encephalopathy (manifesting as drowsiness, confusion, asterixis, stupor, and coma) [1].

In the third stage, cardiac damage can occur, leading to tachycardia, ST and T wave changes, QTc prolongation, low voltage QRS, and arrhythmias [2]. Supportive care, cardiac monitoring, and correction of electrolyte abnormalities are typically the treatments for YP poisoning [3]. Gastric lavage with potassium permanganate is advised to convert phosphorus into comparatively safer oxides [2]. Risk factors associated with significant mortality include renal failure, acidosis, elevated alkaline phosphatase, and isolated thrombocytopenia [4].

The LT has been successfully performed in cases of ALF caused by YP poisoning in various countries, with good results in both adult and paediatric patients. The success rates of LT for ALF due to YP rodenticide poisoning are promising, as long as there are no contraindications to transplant. In some cases, plasma exchange, Renal Replacement Therapy (RRT), or cytosorb can be used as a bridge to transplant [5]. According to a case series, 43% of patients who satisfied the Kings College criteria for transplant survived spontaneously. Transplantation may be considered in the absence of hepatic encephalopathy but with indicators of severe acute liver injury such as PT-INR > 6.0, model for end-stage liver disease score > 37, consistently increased blood lactate despite resuscitation, and plasmapheresis PT-INR >2.5 atleast 12 hours after the second cycle [6]. The authors proposed that patients with grade 3 or more HE should be considered for transplant [6].

[Table/Fig-6] shows the comparison between various studies regarding treatment and outcomes. This table concludes that LT is a better treatment modality for most patients with ALF.

Type of study	Year of study	Authors name	Place of study	Age (in years)	Sex	Quantity ingested	Major treatment	Outcome
Case report (1)	2017	Ravikanth R et al.,	Karnataka, India	25	Female	2 gm	Injection vitamin k, fresh frozen plasma and N-acetyl cysteine infusion	Recovered
Case series of 19 cases [6]	2021	Mohanka R et al.,	Mumbai, India	32.0±9.6	Male-7 Female -12	21.3±11.2 gm	14- injection vitamin k, fresh frozen plasma, and N-acetyl cysteine infusion 5- Liver Transplantation (LT)	7 succumbed to death out of 14 patients who took conservative management. All the 5 liver transplant patients got recovered
Case series of 3 cases [7]	2021	Dawra S et al.,	Pune, India	1) 20 2) 19 3) 22	Female Male Female	10 gms 5 gms Not specified	Injection vitamin k, fresh frozen plasma and N-acetyl cysteine infusion	Recovered Recovered Succumbed to death
Case report [8]	2019	Yerra P et al.,	Coimbatore, India	24	Female	20 gms	Liver transplantation	Recovered
Case report [9]	2020	Soni JP et al.,	Madhya Pradesh, India	30	Female	12 gms	N-acetyl cysteine infusion	Succumbed to death
Case report	2023	Present study	Pune, India	33	Male	30 gms	Liver transplant	Recovered

[Table/Fig-6]: Various comparative studies in view of treatment and outcomes.

CONCLUSION(S)

Rat poisoning resulting in fulminant liver failure is a potentially lethal condition. While some patients may respond well to supportive therapy and experience remission of liver failure, the option of LT should be considered for those with significant hepatic damage indicated by biochemical and clinical factors, as observed in the present case. Early transfer to a specialised centre with all the necessary facilities can improve the chances of spontaneous recovery and offer a good prognosis, as well as facilitate prompt LT when needed.

Since YP poisoning is becoming a growing public health concern, particularly among younger individuals, as in the present case, it is important to take action to establish and maintain a database of all cases. The manufacturing sector should prioritise measures such as safeguarding storage, restricting access, and closely monitoring stock levels. Additionally, raising awareness among the general population about the potential fatality of YP poisoning is crucial. It is also important to educate primary care physicians about the delayed onset toxidrome associated with this condition.

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