ORIGINAL ARTICLE

Outcome of Plasma Exchange in Acute Liver Failure due to Yellow Phosphorus Poisoning: A Single-center Experience

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Abstract

Background: Yellow phosphorus (YP) is a protoplasmic poison that causes acute liver failure (ALF) for which liver transplantation is the definitive modality. Hereby, we present our clinical data on the role of plasma exchange (PE) in ALF due to YP poisoning when liver transplantation is not readily available.

Methods: Our study is a prospective observational type, conducted between January 2017 and January 2020, which included patients with ALF due to YP poisoning requiring PE. Clinical features, quantity of poison consumed, and laboratory data before and after PE were noted, and the outcome was documented.

Results: This study had 10 patients. The mean age was 30 years. The ratio of male to female being 1.5:1. The amount of YP consumed (median) was 10 gm. Six patients consumed ≤10 gm and four consumed >10 gm. The mean of total PE sessions was 3.3. Seven patients (70%) had recovery from ALF, out of which five had consumed <10 gm of YP. Among patients who recovered after consuming YP, the mean day to get admitted to the hospital was 3.6 ± 1.81 ($p = 0.017$) and the time to start PE was 4.86 ± 1.67 days ($p = 0.033$). Three patients did not recover from ALF, of whom two expired. Peak total bilirubin (mg/dL) decreased to 2.76 from 9.29 ($p = 0.005$), serum glutamic oxaloacetic transaminase to 53.5 from 530 (IU/L) ($p = 0.005$), serum glutamic pyruvic transaminase to 54.5 from 378 (IU/L) ($p = 0.005$), international normalized ratio to 1.08 from 2.26 ($p = 0.008$), prothrombin time(s) decreased to 13.3 from 25.5 ($p = 0.013$), and activated partial thromboplastin time(s) to 24.6 from 40.8 ($p = 0.007$) post-PE sessions.

Conclusions: Our study revealed that the patient outcome depends on the quantity of poison consumed, duration of hospitalization, and time to start PE from the day of YP consumption. PE may be considered as a bridge to liver transplant in ALF patients.

Keywords: Acute liver failure, Plasma exchange, Poisoning, Yellow phosphorus.

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Introduction

Elemental phosphorus exists in two forms: yellow and red. Red phosphorus is nontoxic because it is not soluble, unabsorbable, and hence not toxic when consumed. Yellow phosphorus (YP) is the only toxic form of elemental phosphorus. It is a protoplasmic poison. It is used in the production of a chemical that kills rodents and is also used for preparing firecrackers. Rodenticides are obtained as powders and pastes having 2–5% of YP.[1–4] It affects multiple organ systems. As little as 15 mg can be lethal, although 1 mg/kg body weight is the generally accepted lethal dose.[5] It is fat-soluble and well-absorbed in the intestinal tract. It evolves phosphine in dilute acid in the digestive tract producing signs and symptoms.[5] It is directly hepatotoxic after absorption.[6]

Oral ingestion of YP produces necrobiosis of organs particularly the liver causing acute liver failure (ALF), resulting in overwhelming hepatocyte death. Damage-associated molecular patterns, systemic inflammatory responses, and various toxins are released with toll-like receptor-dependent activation of innate immune cells.[6,7] Plasma exchange (PE) tends to remove albumin-bound and unbound toxins, like ammonia, indoles, aromatic amino acids, endotoxin, and other inflammatory mediators.[8,9] Liver transplant is the definitive modality for ALF due to YP. There is no antidote to reverse hepatic damage.[10] Recent guidelines recommend PE as an essential and emergency treatment modality for critically ill patients with ALF until recovery or availability of liver transplantation.[11,8] Both high and standard volume PE have been advocated for patients with ALF.[6–7]

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Hereby, we elaborate our experience in approaching ALF due to YP poisoning, with a standard volume PE as a modality when liver transplants are not easily available and to assess the effect of PE on liver parameters and patient outcome.

Methods

This prospective observational study is done in a tertiary center between January 2017 and January 2020 in South India. All YP poisoning patients (as confirmed by observing the container containing the poison) with ALF requiring PE were included after informed written consent was obtained. Exclusion criteria included age <18 years, chronic liver disease patients, all causes of ALF other than YP, and those patients who did not give consent for

PE. All patients were treated with vitamin K and N-acetylcysteine (NAC) and all underwent standard volume PE.[8] Clinical features, comorbidities, quantity of poison consumed, days of hospital stay, days to start PE, and baseline characteristics were documented. Laboratory profiles, including complete hemogram, liver function tests (LFT), and coagulation parameters [prothrombin time (PT), international normalized ratio (INR), activated partial thromboplastin time (APTT)], were noted serially before and 12 hours after each session of PE. Patient outcomes were recorded as survival or death; liver parameter outcomes as recovered or not. Hence, we assessed the impact of PE on the parameters of liver functions. Those patients who survived were followed up till their liver functions were normalized or up to three outpatient department visits whichever is earlier.

ALF was elucidated as acute and severe hepatic injury up to a duration of 4 weeks in a patient with no preexisting liver disease, resulting in coagulation derangements with an INR of ≥1.5 and any degree of encephalopathy.[12,13] Recovery of ALF was defined as resolution of liver enzymes [serum glutamic oxaloacetic transaminase (SGOT), serum glutamic pyruvic transaminase (SGPT)] and coagulation derangements (PT/INR) to normal or a declining trend in the above parameters during the hospital course to reach at least 25% of the upper limit of normal.

PE was performed using hollow fiber membrane plasmapheresis with standard volume of approximately 3 L for each session using formula 0.065 × weight (kg) × [1 − Hct] with a duration of 2 hours done through noncuffed two-lumen temporary dialysis catheter inserted in the common femoral vein.[9,14] Equal volumes of replacement fluid and fresh frozen plasma were utilized. The PE sessions were decided by the treating physician based on liver parameters, coagulation improvements, and clinical recovery.[8]

Baseline characteristics were documented. Continuous variables were expressed as mean ± standard deviation (SD) or median and interquartile range (IQR) according to a normal distribution by using Shapiro–Wilk test. Friedman test for nonparametric variables at different points of time was applied. After adjusting for Bonferroni correction, a post hoc test was done for multiple comparisons. Independent *t*-test and χ^2 tests were used for statistical analysis wherever required. For data that did not follow a normal distribution, the significance of differences was tested using Mann–Whitney or Wilcoxon tests. Statistical analysis was carried out using the SPSS version 20.0 software. A *p*-value of <0.05 was considered statistically significant. For the post hoc test, a *p*-value of <0.016 was considered significant.

Informed written consent was obtained. Institutional ethics committee approval was obtained.

Results

Ten patients were available for analysis. Age ranged from 18–56 years (mean 30 years; median 24.5 years with IQR of 21.7–38.2). The ratio of male to female was 1.5:1. One had a history of both diabetes and hypertension and one had hypothyroid. All patients presented with icterus. Six patients had vomiting, and five had headache. Two had abdominal pain. One developed acute kidney injury requiring hemodialysis (HD). Ninety percent of patients had intentional YP consumption in the form of a paste, with each tube having 40 gm of 4% YP. Among patients who consumed ≤10 gm (*n* = 6), five recovered from ALF (83.3%) whereas only two recovered (50%) among those who consumed >10 gm (*n* = 4).

Baseline laboratory characteristics are summarized in Table 1. A majority of patients presented with vomiting (*n* = 6), fever, and headache (*n* = 5) with generalized weakness. All patients had icterus (*n* = 10). Mean admission model for end-stage liver disease (MELD) score was 20.8 ± 9.4, which peaked to 27 ± 11.6 prior to PE. Higher grades of hepatic encephalopathy (HE), higher ammonia levels, high total bilirubin, low platelets, and sodium at presentation resulted in poor recovery from ALF, whereas high MELD score, high total bilirubin, and higher grades of HE on admission resulted in poor patient survival (Table 1).

Overall, patients underwent 33 sessions of PE (mean 3.3). Out of 10 patients, 4 underwent 2 PEs, 3 underwent 5 PEs, 2 received 3 PEs, and the remaining 1 received 4 sessions. A total of seven patients (70%) recovered from ALF (22 sessions of PE with a mean of 3.1), whereas three did not recover (11 sessions of PE with a mean of 3.7). Out of those three patients, one patient was referred for liver transplantation, while the rest two succumbed. For recovery from ALF, mean time to get admitted to the hospital was 3.6 days (*p* = 0.017) and mean time to start PE was 4.86 days (*p* = 0.033) when compared to those who did not recover from ALF. The mean time to hospitalization among survivors was 3.9 days (*p* = 0.04), and mean time to initiate PE was 5.1 days (*p* = 0.044) when compared to nonsurvivors (Table 2).

Table 3 summarizes the laboratory parameters before and after PE with significant improvements in liver functions and coagulation profile. Total bilirubin declined from 9.3 before PE to 2.76 after PE (*p* = 0.005); SGOT declined from 530 to 53.5 (*p* = 0.005); SGPT from 378–54.5 (*p* = 0.005); INR to 1.08 from 2.26 (*p* = 0.008); PT declined to 13.3 from 25.5 (*p* = 0.013); and APTT to 24.6 from 40.8 (*p* = 0.007) post-PE.

Table 4 compares the data before and after PE among those who recovered from ALF with those who did not, also among the survival and nonsurvival groups. Levels of total bilirubin (*p* = 0.03) and SGOT (*p* = 0.017) were significantly lower in those recovered from the ALF group. The group that showed liver recovery had lower total bilirubin (*p* = 0.03) before PE; normalized total bilirubin (*p* = 0.033); lower INR (*p* = 0.017); and APTT (0.033) after PE when compared to the group, which showed no liver recovery. Although SGOT was higher in the recovered group prior to PE, there was significant decline post-PE (*p* = 0.017) in contrast to the patients who did not have liver recovery. Among the survivors, there was much more decline in the liver and coagulation parameters when compared to nonsurvivors (Table 4). However, multivariate logistic regression did not show significant predictors for liver recovery.

Discussion

This study analyzed the role of PE in ALF due to YP. The mean age in this study was 30 years with a median of 24.5 years. Other studies too had a majority of patients in their second or third decade.[1,2,15] The ratio of male to female was 1.5:1. Other studies too had a similar ratio of 1.1 to 1.8:1.[2,3] One study had patients presenting in the mid to late second decade with more female preponderance (1:1.7).[16] Ninety percent of YP intake in this study was deliberate with suicidal intent seen among young individuals and may reflect rising social stressors. The earliest symptoms of YP are usually nausea, abdominal pain, feeling of chilliness, and of being "cold all over." Phosphine is a highly toxic gas that is evolved on exposure of YP to gastric acids, which in turn inhibits cytochrome C oxidase and oxidative respiration.[17,18] YP is crystalline, white to yellow powder that turns dark on exposure to light.[22] YP is directly hepatotoxic

Table 1: Baseline characteristics of patients (on admission) with YP poisoning who underwent PE

		Recovery from ALF			*Patient survival*		
Variables	*All patients (n = 10)*	*Recovered (n = 7)*	*Not recovered (n = 3)*	*p value*	*Survivors (n = 8*)*	*Nonsurvivors (n = 2)*	*p value*
Age (years)	30 ± 12.6	32.29 ± 13.79	24.67 ± 9.07	0.413	32.6 ± 12.8	19.5 ± 2.12	0.204
Gender (M/F)	6/4	3/4	3/0	0.20	4/4	2/0	0.462
Hb (gm/dL)	11.9 ± 1.47	11.83 ± 1.71	11.9 ± 0.95	0.949	11.71 ± 1.62	12.4 ± 0.57	0.585
TC ($\times 10^3$/µL)	11.9 ± 5.7	11.11 ± 6.63	13.8 ± 3.1	0.531	11.02 ± 6.1	15.5 ± 1.13	0.355
Platelet ($\times 10^3$/µL)	149 ± 73	178 ± 66	84 ± 36	**0.05**	162 ± 76	101 ± 26	0.315
PT (s)	22.22 ± 6.7	21.56 ± 7.79	23.77 ± 3.5	0.658	21.33 ± 7.24	25.75 ± 0.92	0.434
INR	1.92 ± 0.56	1.87 ± 0.65	2.03 ± 0.37	0.711	1.84 ± 0.61	2.24 ± 0.014	0.396
APTT (s)	37.2 ± 16.98	35.34 ± 19.98	41.5 ± 7.76	0.629	35.11 ± 18.52	45.5 ± 4.95	0.472
Creatinine (mg/dL) (median and IQR)	0.7 (0.5–1.45)	0.6 (0.5–0.9)	1.0 (0.8–5.0)	0.117	0.6 (0.5–0.875)	3 (1–5)	0.089
Total bilirubin (mg/dL)	7.05 ± 7.18	2.85 ± 2.01	16.84 ± 3.85	0.001	4.57 ± 5.2	16.96 ± 5.44	0.017
SGOT (U/L) (Median and IQR)	308.5 (182.75–683)	349 (303–704)	203 (122–264)	0.117	331.5 (273.8–697)	162.5 (122–203)	0.178
SGPT (U/L) (Median and IQR)	328 (178.25–407.25)	244 (110–477)	381 (327–384)	0.517	286.5 (132.7–453)	355.5 (327–384)	0.711
Albumin (gm/dL)	2.6 ± 0.63	2.64 ± 0.68	2.47 ± 0.60	0.708	2.56 ± 0.68	2.75 ± 0.46	0.711
Ammonia (µmol/L)	67.56 ± 30.66	54.83 ± 18.60	97.27 ± 36.06	0.035	57.46 ± 18.78	107.9 ± 43.84	0.342
Sodium (mEq/L)	134.3 ± 2.91	135.6 ± 2.37	131.3 ± 1.52	0.015	134.8 ± 2.95	132 ± 1.41	0.231
HE grade 1/2/3 (*n*)	5/2/3	5/2/0	0/0/3	0.007	5/2/1	0/0/2	0.05
Amount of YP consumed (gm)	10 (8.75–21.25)	10 (5–15)	20 (10–22)	0.196	10 (6.25–22.5)	15 (10–24)	0.771
MELD score	20.8 ± 9.4	17.29 ± 7.3	29 ± 9.6	0.065	17.88 ± 6.95	32.5 ± 10.6	0.039

*One patient among survivors was referred for liver transplantation. *Abbreviations*: Hb, hemoglobin; TC, total count; PT, prothrombin time; INR, international normalized ratio; APTT, activated partial thromboplastin time; SGOT, serum glutamic oxaloacetic transaminase; SGPT, serum glutamic pyruvic transaminase; HE, hepatic encephalopathy; MELD, model for end-stage liver disease

Table 2: Clinical outcomes of patients with ALF with YP poisoning

		Recovery from ALF			*Patient survival*		
Variables	*All patients (n = 10)*	*Recovered (n = 7)*	*Not recovered (n = 3)*	*p value*	*Survivors (n = 8*)*	*Nonsurvivors (n = 2)*	*p value*
Mean sessions of PE (total sessions)	3.3 ± 1 (33)	3.1 ± 1.4 (22)	3.7 ± 1.1 (11)	0.517	3.4 ± 1.5 (27)	3 ± 0 (6)	1.0
Time to initiation of PE after YP consumption (mean day)	7.3 ± 5	4.86 ± 1.67	13 ± 7.21	0.033	5.13 ± 1.73	16 ± 7.07	0.044
Time to Hospital admission after YP consumption (mean day)	6 ± 5	3.6 ± 1.81	11.7 ± 7.39	0.017	3.9 ± 1.88	14.5 ± 7.78	0.044
Length of hospital stay (mean day)	13.6 ± 4.19	14.14 ± 3.13	12.33 ± 6.81	0.564	14.8 ± 3.56	8.5 ± 2.12	0.046

*One patient among survivors was referred for liver transplantation. *Abbreviations*: ALF, acute liver failure; PE, plasma exchange; YP, yellow phosphorus

Table 3: Overall effects of PE sessions on laboratory parameters of patients among YP with ALF (n = 10)

Variables (median and IQR)	*Before PE*	*After PE sessions*	*p value*
Total bilirubin (mg/dL)	9.3 (1.53–15.7)	2.76 (0.5–9.9)	0.005
SGOT (U/L)	530 (323–794)	53.5 (29.5–84.8)	0.005
SGPT (U/L)	378 (269–566)	54.5 (38–97.5)	0.005
PT (s)	25.5 (22.9–66.1)	13.3 (11.7–26)	0.013
INR	2.26 (2–3.6)	1.08 (1–1.21)	0.008
APTT (s)	40.8 (31.7–88.2)	24.6 (22.9–29.4)	0.007

Abbreviations: PT, prothrombin time; INR, international normalized ratio; APTT, activated partial thromboplastin time; SGOT, serum glutamic oxaloacetic transaminase; SGPT, serum glutamic pyruvic transaminase

after absorption.[6] YP poisoning manifests in three stages. The first stage is within a day where patients are either asymptomatic or have features of local gastrointestinal irritation. The second stage occurs between 24 and 72 hours after consumption and has no symptoms with mild elevation of liver enzymes, when a patient may be discharged. The third stage is after 72 hours till the resolution of symptoms or death.[1,19,17] In our study, all patients had icterus and the majority had vomiting. Thirty percent had grade 3 HE and 20% had abdominal pain. In one study, abdominal pain and altered sensorium were seen in 60%.[20]

The treatment of YP is aimed at the removal of the poison, prevent absorption, and protect body organs. The use of 1:1000 potassium permanganate as gastric lavage[15,21] or liquid petrolatum (mineral oil) as a purge has been found useful.[18] The fatality rate is >50% depending on the amount of phosphorus absorbed from the intestine.[16] ALF due to YP being treated with NAC is still to be validated.[2,20] A recent retrospective study has shown the benefit of NAC if given early. However, a time lag of >24 hours was a significant risk factor for mortality.[23] Many studies were reported for the beneficial effect of extracorporeal techniques in ALF caused by acetaminophen, alcohol, viruses, etc.[6,7,9] Recent guidelines recommend PE (high volume) as therapeutic first-line management in ALF.[8] The amount of YP consumed was 13.5 gm (mean) with a median of 10 gm. This was comparable with other studies.[20] There was 85.7% recovery from ALF among patients who consumed <20 gm, whereas only 14.3% recovered among those who consumed >20 gm. The mean delay in presentation to hospital after YP consumption was 6 days, which was in-line with other study.[20]

Subgroup analysis of baseline characteristics on admission showed higher grades of HE, higher ammonia levels, high total bilirubin, low platelets, and sodium at presentation resulted in poor recovery from ALF, whereas high MELD score, high total bilirubin, and higher grades of HE resulted in poor patient survival (Table 1). This is in contrast to another study, where there was no significant difference in lab values at admission among survivors and nonsurvivors.[9]

Seventy percent recovery rate from ALF was seen in our study with a mean PE session of 3.3 and a mean duration of hospital stay of 13.6 days. The mean time to get admitted to the hospital was 3.6 days ($p = 0.017$) and mean time to start of PE was 4.86 days ($p = 0.033$) after YP consumption for recovery from ALF. Among the three patients who did not recover from ALF, two expired and one was sent for liver transplantation. In other studies[7,9] where PE was done for ALF due to any cause, the mean sessions ranged from 2.4–5. Mean hospital stay in these studies was 21.9–33 days. A significant difference in various parameters of liver functions and coagulation parameters was observed in pre- and post-PE values (p <0.05) (Table 3). This was in keeping with other studies also, where bilirubin, SGOT, SGPT, and INR had significant improvement post-PE.[9] Laboratory data for univariate analysis for recovery as in Table 4 had significant difference (p <0.05) in bilirubin [before (B/F) and after (A/F) PE], SGOT (B/F and A/F PE), INR (A/F PE), and APTT (A/F PE) among recovered and not recovered. However, no significant difference was noted among the survival group. This is in-line with the other study.[9] However, multivariate logistic regression analysis did not show any significant predictors for liver recovery.

Exchange transfusions in acute YP intoxication were described as early as 1971.[15] In that study, 5 among 15 patients of HE were given exchange transfusions (one to three), resulting in 3 survivals and 2 deaths. Of those 10 patients, who were not given exchange transfusion, 7 died and 3 survived. The study did not show statistically significant conclusions. Cheng et al.[9] retrospectively studied 10 patients of Asian descent with ALF caused due to wide etiologies treated with standard volume PE. Mean PE sessions were 5 with a median length of hospital stay of 33 days with nonsignificant improvement in coagulation parameters after PE with 30% survival. A prospective study of high volume PE in Caucasian patients by Larsen et al.,[7] among 92 patients of ALF with a wide etiology showed a survival benefit of 58.7% with a mean session of PE of 2.4 and mean days of hospital admissions of 21.9 days when compared to standard medical therapy. There was a statistically significant improvement in INR and ALT post-PE. Extrapolating the results of these studies to ALF caused by only YP needs to be validated by large randomized controlled trials.

Our study had many limitations. Firstly, a small sample size in the study population was done in a single center. Secondly, those patients who presented late with significant liver damage were also included and given PE, which is a limitation. Thirdly, a poor resource setting where affordability to PE is also a big burden. Finally, confirmation of YP consumed was done by observing the container containing poison and not by chemical analysis. However, as per the best of our knowledge, it is the first observational prospective study reporting the outcome of PE in patients with ALF due to YP poisoning.

Conclusion

In conclusion, PE may be considered as a modality for ALF due to YP as a bridge to transplantation. Patient outcomes depend on the amount of YP consumed and the time to initiation of PE. Further prospective randomized multicenter studies are needed to validate these findings.

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Table 4: Effects of PE sessions among patients of YP with respect to liver recovery and patient survival

		Recovery from ALF			Patient survival		
Variables (median and IQR)	*All patients (n = 10)*	*Recovered (n = 7)*	*Not recovered (n = 3)*	*p value*	*Survivors (n = 8*)*	*Nonsurvivors (n = 2)*	*p value*
Total Bilirubin (mg/dL) (Before PE)	9.3 (1.54–13.3)	5.39 (1.5–12.5)	22.9 (13.11–25.2)	0.033	5.59 (1.51–13.17)	19 (13.11–25.2)	0.178
Total bilirubin (mg/dL) (after PE)	2.76 (0.48–7)	0.56 (0.37–3.5)	18.6 (5.6–24.5)	0.033	1.29 (0.39–6.13)	15.67 (5.63–24.5)	0.178
SGOT (U/L) (before PE)	530 (346–730)	604 (490–988)	254 (122–346)	0.017	587 (426–923)	188 (122–254)	0.044
SGOT (U/L) (after PE)	54 (31–82)	31 (25–58)	93 (82–162)	0.017	40 (26.5–63.25)	87.5 (82–93)	0.178
SGPT (U/L) (before PE)	378 (300–550)	340 (175–550)	384 (372–613)	0.517	401.5 (206–597)	378 (372–384)	1.0
SGPT (U/L) (after PE)	55 (39–96)	44 (35–56)	102 (96–135)	0.17	48.5 (36–63.5)	115.5 (96–135)	0.089
PT(s) (before PE)	25.5 (23.5–48.8)	25.8 (23.5–118)	25.1 (21–48.8)	0.833	25.15 (21.6–95.7)	36.9 (25.1–48.8)	0.711
PT(s) (after PE)	13.25 (11.8–16.1)	12 (11.2–14.6)	16.1 (13.6–55.8)	0.183	12.45 (11.35–15.72)	34.7 (13.6–55.8)	0.40
INR (before PE)	2.26 (2.1–3.08)	2.28 (2.1–3.08)	2.23 (1.77–4.94)	1.0	2.23 (1.85–2.96)	3.58 (2.23–4.94)	0.533
INR (after PE)	1.08 (1.02–1.17)	1.03 (0.96–1.11)	1.34 (1.17–4.94)	0.017	1.04 (0.97–1.11)	3.05 (1.17–4.94)	0.089
APTT(s) (before PE)	40.8 (31.8–87.3)	32.7 (31.4–87.3)	42 (41.8–91)	0.267	36.25 (31.5–75.92)	66.5 (42–91)	0.267
APTT(s) (after PE)	24.6 (23.1–28.5)	23.7 (22.5–25.3)	32 (28–91.1)	0.033	23.75 (22.65–27.7)	59.55 (28–91.1)	0.178

*One patient among survivors was referred for Liver Transplantation; *Abbreviations*: PT, prothrombin time; INR, international normalized ratio; APTT, activated partial thromboplastin time; SGOT, serum glutamic oxaloacetic transaminase; SGPT, serum glutamic pyruvic transaminase

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References

1. McCarron MM, Gaddis GP, Trotter AT. Acute yellow phosphorus poisoning from pesticide pastes. Clin Toxicol 1981;18(6):693–711. DOI: 10.3109/15563658108990295.
2. Fernandez OU, Canizares LL. Acute hepatotoxicity from ingestion of yellow phosphorus-containing fireworks. J Clin Gastroenterol 1995;21(2):139–142. DOI: 10.1097/00004836-199509000-00015.
3. Diaz-Rivera RS, Collazzo PJ, Pons ER, Torregrosa MV. Acute phosphorus poisoning in man: a study of 56 cases. Medicine (Baltimore) 1950;29(4):269–298. DOI: 10.1097/00005792-195012000-00001.
4. Simon FA, Pickering LK. Acute yellow phosphorus poisoning. "Smoking stool syndrome". JAMA 1976;235(13):1343–1344. DOI: 10.1001/jama.1976.03260390029021.
5. Konjoyan TR. White phosphorus burns: case report and literature review. Mil Med 1983;148(11):881–884. PMID: 6419161.
6. Maiwall R, Moreau R. Plasma exchange for acute on chronic liver failure: is there a light at the end of the tunnel? Hepatol Int 2016;10(3):387–389. DOI: 10.1007/s12072-016-9703-z.
7. Larsen FS, Schmidt LE, Bernsmeier C, Rasmussen A, Isoniemi H, Patel VC, et al. High-volume plasma exchange in patients with acute liver failure: An open randomised controlled trial. J Hepatol 2016;64(1):69–78. DOI: 10.1016/j.jhep.2015.08.018.
8. Padmanabhan A, Connelly-Smith L, Aqui N, Balogun RA, Klingel R, Meyer E, et al. Guidelines on the use of therapeutic apheresis in clinical practice – evidence-based approach from the writing committee of the American Society for Apheresis: the eighth special issue. J Clin Apher 2019;34(3):171–354. DOI: 10.1002/jca.21705.
9. Cheng YL, Chang CH, Chen WT, Tsai MH, Lee WC, Tu KH, et al. Prognostic factors and treatment effect of standard-volume plasma exchange for acute and acute-on-chronic liver failure: a single-center retrospective study. Transfus Apher Sci 2018;57(4):537–543. DOI: 10.1016/j.transci.2018.05.030.
10. Ates M, Dirican A, Ozgor D, Aydin C, Isik B, Ara C, et al. Living donor liver transplantation for acute liver failure in pediatric patients caused by the ingestion of fireworks containing yellow phosphorus. Liver Transpl 2011;17(11):1286–1291. DOI: 10.1002/lt.22384.
11. Sarin SK, Kedarisetty CK, Abbas Z, Amarapurkar D, Bihari C, Chan AC, et al.; APASL ACLF Working Party. Acute-on-chronic liver failure: consensus recommendations of the Asian Pacific Association for the Study of the Liver (APASL) 2014. Hepatol Int 2014;8(4):453–471. DOI: 10.1007/s12072-014-9580-2.
12. O'Grady JG, Schalm SW, Williams R. Acute liver failure: redefining the syndromes. Lancet 1993;342(8866):273–275. DOI: 10.1016/0140-6736(93)91818-7.
13. Vincent JL. Fulminant hepatic failure. Textbook of critical care. 6th ed. Elsevier Saunders; 2011. p. 771–779.
14. Kaplan AA. A simple and accurate method for prescribing plasma exchange. ASAIO Trans 1990;36(3):M597–M599. PMID: 2252761.
15. Marin GA, Montoya CA, Sierra JL, Senior JR. Evaluation of corticosteroid and exchange-transfusion treatment of acute yellow-phosphorus intoxication. N Engl J Med 1971;284(3):125–128. DOI: 10.1056/NEJM197101212840303.
16. González-Andrade F, López-Pulles R. White phosphorus poisoning by oral ingestion of firecrackers or little devils: current experience in Ecuador. Clin Toxicol (Phila) 2011;49(1):29–33. DOI: 10.3109/15563650.2010.547860.
17. Suneetha DK, Inbanathan J, Kannoth S, Reshma PK, Shashank MS. Profile of rat killer poisoning cases in a tertiary care hospital at Mysore. Int J Sci Study 2016;3(12):264–267. DOI: 10.17354/ijss/2016/162.

18. Fletcher GF, Galambos JT. Phosphorus poisoning in humans. Arch Intern Med 1963;112:846–852. DOI: 10.1001/archinte.1963.03860060084008.
19. Ravikanth R, Sandeep S, Philip B. Acute yellow phosphorus poisoning causing fulminant hepatic failure with parenchymal hemorrhages and contained duodenal perforation. Indian J Crit Care Med 2017;21(4):238–242. DOI: 10.4103/ijccm.IJCCM_410_16.
20. Mishra AK, Devakiruba NS, Jasmine S, Sathyendra S, Zachariah A, Iyadurai R. Clinical spectrum of yellow phosphorous poisoning in a tertiary care centre in South India: a case series. Trop Doct 2017;47(3):245–249. DOI: 10.1177/0049475516668986.
21. Saoji A, Lavekar AS, Salkar H, Pawde GB, Tripathi S. A case on suicidal poisoning associated with ratol and a perspective on yellow phosphorus poisoning. Int J Recent Trends Sci Technol 2014;10(2):223–225.
22. International Programme on Chemical Safety (IPCS). Yellow phosphorus fact sheet 2004 [internet]: World Health Organization (WHO) – International peer reviewed chemical safety information (INCHEM); 2004. Available from: http://www.inchem.org/documents/icsc/icsc/eics0628.htm.
23. Mark K, Hyder S, Rashid M, Chandran VP, Seshadri S, Seshadri S, et al. Survival benefits of N-Acetylcysteine in rodenticide poisoning: Retrospective Evidence from an Indian tertiary care setting. Curr Clin Pharmacol 2020. DOI: 10.2174/1574884715666200513090634. PMID: 32400335.