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THERAPEUTIC PLASMA EXCHANGE IN YELLOW PHOSPHORUS POISONING: A GAME CHANGER ? EXPERIENCE FROM A SINGLE-CENTER STUDY

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ABSTRACT We managed two cases of yellow phosphorus poisoning that resulted in acute liver failure, with severely deranged liver enzymes and coagulation profiles and MELD scores above 30, highlighting the urgent need for liver transplantation. In the absence of a specific antidote, we considered liver transplantation while initiating therapeutic plasma exchange (TPE) as bridging therapy. TPE was administered over three consecutive days with patient consent, using fresh frozen plasma (FFP) and 20% albumin to address electrolyte imbalances. Gradual improvement in liver enzymes and INR normalization was observed. Both patients were discharged after 13 days of hospitalization, showing favorable outcomes at follow-up. TPE appears to be an effective, safe, and cost-efficient alternative to liver transplantation in managing yellow phosphorus-induced acute liver failure, thereby transforming critical care management.

KEYWORDS :

BACKGROUND

Yellow phosphorus poisoning (YPP) leads to acute liver failure (ALF) through metabolite accumulation and cytokine-mediated injury, causing hepatic encephalopathy (HE), progressive jaundice, and multiorgan dysfunction. Mortality exceeds 50%, primarily due to the absence of specific antidotes and delays in liver transplantation.

Extracorporeal support systems like the molecular absorbent recirculatory system (MARS) and plasma exchange (PE) have been employed to stabilize such patients by removing accumulated toxins. Standard-volume plasma exchange (1–1.5 L/day) has been shown to improve transplant-free survival in YPP-induced ALF by reducing inflammatory cytokines, modulating immune responses, and aiding toxin clearance.

Research indicates that standard volume plasma exchange (1–1.5 L/day) improves transplant-free survival rates in YPP-induced ALF. It effectively reduces inflammatory cytokine levels, modulates adaptive immunity, and may decrease susceptibility to infections and the burden of albumin-bound and water-bound toxins.

We present clinical outcomes of two patients with ALF secondary to YPP where liver transplantation was not feasible, analysed to assess the efficacy of these interventions.

CASE PRESENTATION

Case I

A teenage boy presented to our emergency department with nausea, vomiting, and abdominal pain three days after ingesting RATOL (2% Yellow Phosphorus). He was alert, oriented, and hemodynamically stable. Vital signs included HR 98 bpm, BP 110/82 mmHg, temperature 98°F, SpO_2 98% on room air, and blood glucose 109 mg/dL.

Initial investigations, including ECG, chest X-ray, ABG, POCUS, and serology, were normal, except for abnormal coagulation profiles: PT 36.2 sec, aPTT 52.8 sec, INR 3.1.

He was admitted to the ICU and started on intravenous N-acetylcysteine, vitamin K, pantoprazole, ondansetron, ceftriaxone, oral ursodeoxycholic acid, syrup silymarin, and IV L-ornithine L-aspartate.

By day 2, worsening liver function confirmed ALF with coagulopathy. With a MELD score of 39 (mortality risk ~52.6%) and no antidote available, therapeutic plasma exchange (TPE) was initiated on day 3 with caregiver consent.

TPE was performed daily for three days using FFP and 20% albumin. Electrolytes were optimized before each session. The patient remained stable throughout all sessions. Post-TPE, liver function parameters improved steadily, indicating a favorable response.

Each TPE session, performed over two hours using a heparin-free method with a hollow fibre membrane, exchanged approximately 3 litres of plasma with an ultrafiltrate removal of 2500 ml. The patient remained hemodynamically stable throughout all procedures. By the completion of three TPE sessions, there was a noted improvement in liver function parameters, indicating a positive response to treatment.

Recovery from acute liver failure (ALF) is defined as achieving either the normalization of liver enzymes (SGOT, SGPT) and coagulation parameters (PT/INR) or a declining trend in these values during the hospitalization, reaching at least 25% of the upper limit of normal.

Case II

A teenage male with yellow phosphorus poisoning (RATOL 3%, 2 packets in fruit juice) presented the next day with abdominal pain and vomiting. He had received gastric lavage at a local hospital. He was conscious, oriented, and vitals were stable (SpO_2 98%, BP 100/60 mmHg, HR 94 bpm, RBS 112 mg/dL). ECG, chest X-ray, and abdominal USG were unremarkable. The patient was admitted to the ICU, and treatment was initiated similarly to the first case.

He was managed with a treatment regimen similar to Case I. By day 3, worsening LFTs indicated ALF (MELD score 38). TPE

was initiated on day 5 with consent. Following two sessions, LFTs began improving.

However, the patient developed lower respiratory tract infection and hepatic encephalopathy (serum ammonia 120 μmol/L). He was treated with piperacillin-tazobactam, clarithromycin, high-volume bowel washes, rifaximin, ursodeoxycholic acid, L-ornithine L-aspartate, syrup silymarin, and lactulose.

By day 7, there was consistent improvement in liver markers. He was discharged after 13 days with uneventful follow-up.

Case I — Clinical Parameters (Days 1–12)

Days	D1	D2	D3	D4	D5	D6	D7	D8	D9	D10	D11	D12
TB	2.6	3.9	5.5	14.	17.5	16.4	14.5	14.3	12.9	11.5	10.2	8.4
DB	2	2.4	4.4	8	11.9	12.9	12.5	13.3	11.8	10.7	9.4	8.1
SGOT	240	1630	2048	251	216	215	188	175	145	127	104	80
SGPT	79	498	664	251	216	198	174	180	174	179	159	125
ALP	119	129	168	134	134	124	144	166	178	184	182	164
TP	6.2	7	6.9	4.8	5.7	4.5	5.3	5.8	6	6.2	6.6	6.2
S.ALB	3.8	4.2	4.1	3.2	3.2	3	3.5	3.5	3.6	3.5	3.6	3.4
GGT	16	36	44	24	18	20	34	60	77	80	108	102

The table provides a chronological progression of clinical parameters over twelve days, detailing the biochemical profile of the patient undergoing treatment for acute liver failure (ALF) due to yellow phosphorus poisoning. Total bilirubin (TB) and direct bilirubin (DB) levels show an initial increase from day 1 to day 3, peaking on day 4, followed by a gradual decline thereafter.

This trend reflects the severity of liver dysfunction and subsequent improvement in bilirubin metabolism and clearance. Serum levels of liver enzymes such as serum glutamic oxaloacetic transaminase (SGOT) and serum glutamic pyruvic transaminase (SGPT) exhibit a dramatic rise on day 2, indicative of acute hepatocellular injury, which steadily improves over the following days. Alkaline phosphatase (ALP), gamma-glutamyl transferase (GGT), total protein (TP), and serum albumin (S.ALB) levels show variable fluctuations, likely influenced by ongoing hepatocellular damage and the body's compensatory mechanisms.

Overall, the data suggest a challenging initial phase followed by a progressive recovery trajectory, highlighting the effectiveness of therapeutic interventions aimed at mitigating liver damage and supporting hepatic function restoration.

Case II — Clinical Parameters (Days 1–12)

Days	D1	D2	D3	D4	D5	D6	D7	D8	D9	D10	D11	D12
TB	1.2	6.1	5.4	5.5	8.9	16.2	18.6	17.3	16.5	15.6	16.2	15.5
DB	0.6	5	4.4	4.7	7.8	13.3	14.8	15	14.4	13.9	14.2	13.8
SGOT	21	1283	5256	957	162	126	121	105	100	111	88	78
SGPT	15	984	2194	854	461	421	375	300	212	181	139	119
ALP	43	83	182	160	144	147	133	123	173	197	138	160
TP	6.3	6.9	5	4.5	4.3	4.2	4.3	4.3	4.9	4.5	4.8	4.9
S.ALB	4.1	4.5	4.1	3,3	3.2	2.9	4.3	2.4	2.8	2.4	2.7	2.7
GGT	11	46	59	50	33	52	69	93	113	95	76	61

The table displays a progression of clinical parameters over twelve days, likely reflecting a patient's treatment course for acute liver injury. Total bilirubin (TB) and direct bilirubin (DB) levels show fluctuations, peaking on days 6 to 7 and gradually decreasing thereafter, indicating ongoing liver function recovery or stabilization.

Serum levels of liver enzymes, including serum glutamic oxaloacetic transaminase (SGOT) and serum glutamic pyruvic transaminase (SGPT), exhibit significant elevations on days 2 to 3, suggesting acute hepatocellular injury, followed by a declining trend as treatment progresses. Alkaline phosphatase (ALP), gamma-glutamyl transferase (GGT), total protein (TP), and serum albumin (S.ALB) levels vary throughout the observation period, likely reflecting dynamic responses to therapeutic interventions and the patient's overall clinical status.

The data underscores the complex nature of liver injury management and the importance of monitoring biochemical markers to guide treatment and assess recovery.

DISCUSSION

Yellow phosphorus (YP), available commercially as rodenticide in 2% and 5% strengths, poses a significant health risk when ingested, whether accidentally or intentionally. Rapid absorption in the gastrointestinal tract results in approximately 70% accumulation in the liver within 2–3 hours, with lesser amounts found in other organs like the heart, kidneys, pancreas, and brain. This distribution contributes to severe outcomes including acute liver failure (ALF), cardiac arrhythmias, toxic myocarditis, acute tubular necrosis, circulatory collapse, and hepatic encephalopathy.

YP exerts toxic effects on cellular structures such as the endoplasmic reticulum and mitochondria, impairing processes like apolipoprotein synthesis for VLDL, ATP production, and fatty acid oxidation. These disruptions lead to lipid accumulation, cellular damage, and ultimately, depletion of glutathione stores and necrosis.

Treatment for YP poisoning primarily involves supportive care and early decontamination with 1:5000 potassium permanganate for gastric lavage followed by activated charcoal. Early administration of N-acetylcysteine (NAC) is crucial as it enhances glutathione synthesis and transferase activity, improving survival rates significantly when given within the first 24–48 hours.

Clinical presentation typically progresses through three stages: Stage I (0–24 hours) with mild gastrointestinal symptoms, Stage II (24–72 hours) characterized by jaundice and abnormal liver enzymes, and Stage III (>72 hours) featuring hepatic encephalopathy, worsening liver function, and potential mortality.

Hospital management involves admission to the High Dependency Unit (HDU) or Intensive Care Unit (ICU) upon presentation, continuous monitoring of consciousness levels, vital signs, electrolyte balance, and urine output. Treatment protocols include prophylactic intravenous antibiotics, vitamin K, proton pump inhibitors, correction of electrolyte imbalances, intravenous fluids, and vasopressors to maintain adequate blood pressure. NAC therapy is administered according to a regimen tailored to mitigate liver damage, while severe cases of encephalopathy may necessitate ventilatory support.

This structured approach aims to stabilize patients, mitigate organ damage, and improve outcomes in cases of severe YP poisoning.

Current Guidelines (2019)

The American Society for Apheresis (ASFA) recommends high-volume therapeutic plasma exchange (HV-TPE) as the primary treatment for acute liver failure (ALF), emphasizing its use until liver transplantation or recovery occurs. Exchange transfusions for yellow phosphorus (YP) intoxication were first described in 1971.

Cheng et al. retrospectively studied 10 ALF patients from various causes treated with standard-volume plasma exchange (PE), averaging 5 sessions per patient with a median hospital stay of 33 days. They reported non-

significant improvements in coagulation profiles and a 30% survival rate.

In contrast, Larsen et al.'s prospective study of 92 ALF patients demonstrated a 58.7% survival rate with high-volume PE sessions averaging 2.4, and a mean hospital stay of 21.9 days. Significant improvements in INR and ALT levels were observed post-TPE. However, these findings need validation in large randomized controlled trials, particularly for ALF caused by YP.

Studies by Leena Thomas and Jolly Chandran on children with rodenticide hepatotoxicity treated with low-volume plasma exchange showed promising outcomes, with 28 out of 32 children surviving at one month, suggesting TPE as an effective non-transplant treatment option in this population.

Similarly, Krishnamurthy Radhakrishnan's study on 43 ALF patients due to YP poisoning reported a 79.06% survival rate with TPE, concluding its effectiveness in YP-induced ALF for patients not meeting criteria for immediate liver transplantation.

In our cases, standard-volume TPE procedures were performed daily for three days using a regimen of 4 units of fresh frozen plasma (FFP) plus 20% albumin over two and a half hours, with 2500 ml of ultrafiltrate removed each session. Starting from day 5, both patients exhibited significant improvements in liver function tests (LFTs) with stable hemodynamics, and any electrolyte imbalances were promptly corrected. Post-discharge follow-up showed uneventful recoveries with normal LFTs and coagulation profiles.

LEARNING POINTS

- Plasma exchange can serve as an effective bridging therapy in patients requiring urgent liver transplantation following rodenticide poisoning.
- Therapeutic plasma exchange improves both biochemical and clinical parameters, supporting liver regeneration.
- TPE is a safe, cost-effective alternative with minimal adverse effects compared to liver transplantation.
- Multi-center studies are needed to validate the role of TPE as a substitute for liver transplantation in poisoning-induced ALF.

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