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Therapeutic Plasma Exchange as a Life-Saving Intervention in Severe Acetaminophen Toxicity: A Case Report

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Abstract: ***Background**: Paracetamol (acetaminophen) overdose is one of the most common causes of acute liver failure worldwide. Due to its over-the-counter availability and widespread use as an analgesic and antipyretic, it is frequently implicated in intentional self-harm. The standard treatment is N-acetylcysteine (NAC); however, patients who progress to acute liver failure with worsening coagulopathy and hepatic encephalopathy may require advanced supportive therapies and liver transplantation. Therapeutic plasma exchange (TPE) has emerged as a potential adjunct in such severe cases. **Case Presentation**: An 18-year-old female presented following intentional ingestion of approximately 20 g of paracetamol. Despite early initiation of N-Acetyl Cysteine (NAC) therapy, she developed acute liver injury characterized by markedly elevated transaminases, progressive coagulopathy and drowsiness. She was admitted to the intensive care unit and managed with standard supportive care along with six sessions of therapeutic plasma exchange. Following TPE, significant improvement was observed in liver function tests, coagulation parameters, and neurological status, leading to clinical recovery without the need for liver transplantation. **Conclusion**: Therapeutic plasma exchange may serve as an effective adjunctive treatment in severe paracetamol-induced acute liver failure. It can improve biochemical and clinical outcomes and may help bridge patients to recovery, particularly in settings where liver transplantation is not readily available.*

Keywords: Paracetamol overdose, Acute liver failure, Therapeutic plasma exchange, N-acetylcysteine, Liver transplantation

1. Case Report

Introduction:

Paracetamol (acetaminophen) toxicity remains one of the most common causes of drug-induced acute liver failure globally. In India and worldwide, it is a major cause of emergency admissions in young adults. Toxicity occurs due to accumulation of the hepatotoxic metabolite N-acetyl-p-benzoquinone imine (NAPQI), particularly when hepatic glutathione stores are depleted. Early administration of N-acetylcysteine (NAC) significantly reduces morbidity and mortality. However, in severe cases progressing to acute liver failure (ALF), supportive therapies including extracorporeal liver support techniques have been explored. Therapeutic plasma exchange (TPE) has been proposed as an adjunctive modality in ALF to remove circulating toxins, inflammatory mediators, and to correct coagulopathy by replacing deficient clotting factors. We present a case of severe paracetamol overdose managed with six sessions of TPE with favorable clinical outcome.

Description of the case:

An 18-year-old female with no known comorbidities presented to the emergency department approximately 8 hours after intentional ingestion of 20 g of paracetamol for deliberate self-harm. She had no history of chronic liver disease, alcohol use, or prior medication intake.

On presentation, she was conscious but drowsy, with complaints of nausea, one episode of vomiting, and giddiness. There was no history of abdominal pain, chest pain, palpitations, seizures, or loss of consciousness. Her vital signs were stable (BP 110/70 mmHg, HR 81/min, RR 14/min, SpO_2 99%, CBG 108 mg/dL). On examination, she was oriented and responsive (GCS 15/15), with no significant abnormalities in respiratory, cardiovascular, or abdominal systems.

Initial management included gastric lavage with 50 g activated charcoal and initiation of N-acetylcysteine (NAC). She was subsequently shifted to the intensive care unit. Due to poor cooperation, relatives were counseled regarding the risk of liver injury and possible need for transplantation. After 24 hours of admission her condition deteriorated with development of severe metabolic acidosis, markedly elevated liver enzymes, mild hyperbilirubinemia, coagulopathy, and altered sensorium suggestive of hepatic encephalopathy. She was intubated and supported with ventilator.

2. Laboratory Investigations

Ultrasound abdomen revealed normal liver size with no chronic liver disease features.

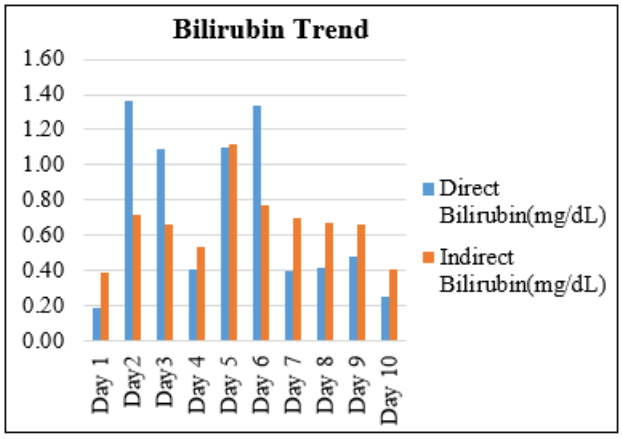


Figure 1: Trend of direct (conjugated) and indirect (unconjugated) bilirubin.

- This figure 1 shows the trend of direct (conjugated) and indirect (unconjugated) bilirubin over 10 days. There are peaks around Day 2 and Day 6, especially for direct bilirubin. Later days (Day 7–10) show a gradual decline, suggesting improvement.

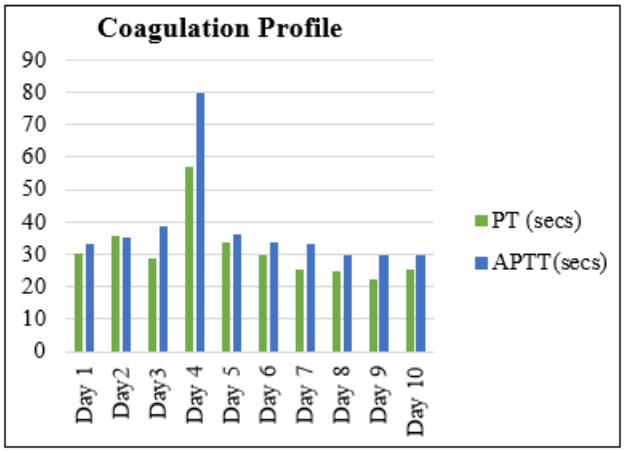


Figure 2: Trend of coagulation parameters- PT (Prothrombin Time) and APTT (Activated Partial Thromboplastin Time)

This figure 2 shows the trend of coagulation parameters- PT (Prothrombin Time) and APTT (Activated Partial Thromboplastin Time) over 10 days. Both PT and APTT are elevated early, with a marked spike on Day 4. After Day 4, there is a steady decline toward near-normal values. This indicates transient coagulation dysfunction followed by recovery.

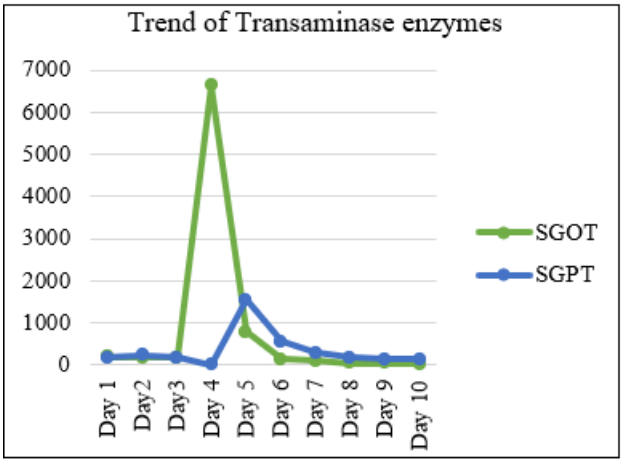
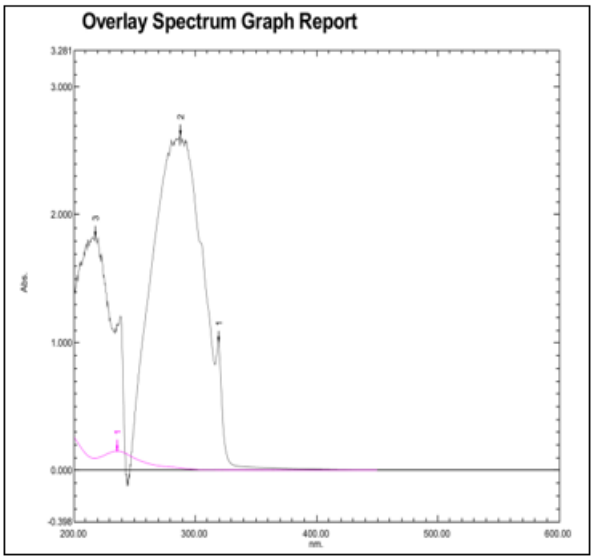


Figure 3: Trend of transaminase enzymes (SGOT/AST and SGPT/ALT)

This figure 3 shows the trend of transaminase enzymes (SGOT/AST and SGPT/ALT) levels over 10 days. Both SGOT (AST) and SGPT (ALT) are near normal or mildly elevated in first 3 days. SGOT increased dramatically in day 4 and SCPT increased in day 5 then both enzymes gradually decrease toward normal



Graph Depicting Standard Value of PCM and Patient PCM values Post 6-TPE Sessions. Method- UV- Vis Spectrophotometry.

Serum Paracetamol on Day 2 of admission (s/p first session of TPE)- 18 µg/ml

Serum Paracetamol on Day 8 of admission (s/p 6 sessions of TPE) - Negligible

3. Discussion

Paracetamol (acetaminophen) toxicity is one of the leading causes of acute liver failure (ALF) worldwide. Hepatic injury results from the accumulation of the toxic metabolite *N-acetyl-p-benzoquinone imine (NAPQI)*, which induces oxidative stress and centrilobular necrosis when hepatic glutathione stores are depleted. Although early administration of N-acetylcysteine (NAC) remains the cornerstone of management, its efficacy may be limited in

patients presenting late or with established ALF (1).

Therapeutic plasma exchange (TPE) has gained attention as a supportive modality in ALF. Its proposed mechanisms include removal of circulating toxins and ammonia, attenuation of systemic inflammation through cytokine clearance, correction of coagulopathy via replacement of clotting factors, and improvement in hemodynamic stability (2). These combined effects may promote hepatic recovery or serve as a bridge to liver transplantation.

In the present case, early initiation of therapeutic plasma exchange (TPE) was associated with significant clinical and biochemical improvement. A progressive reduction in PT, INR, and APTT was observed, along with a downward trend in transaminase levels and marked improvement in sensorium. The patient was successfully extubated on the fourth day of hospitalization and subsequently achieved complete recovery without the need for liver transplantation.

Serum paracetamol levels measured after the first TPE session (day 2 of hospitalization) were 18 µg/mL. TPE was performed on hospital days 2, 4, 5, 6, 7, and 8. Following six cycles of TPE, serum paracetamol levels assessed by UV–visible spectrophotometry were negligible. The patient demonstrated complete clinical recovery.

These findings highlight the potential role of TPE in enhancing toxin clearance and improving transplant-free survival in selected patients with severe acute liver failure.

The role of TPE in ALF is still evolving. According to the American Society for Apheresis (ASFA) 2023 guidelines, TPE for ALF is classified as Category III, Grade 2B, indicating that its optimal role is not yet established and decisions should be individualized (2)**.** However, emerging evidence supports its use. A case reported by Gowtham Kishore C G *et al*(3), he successfully used plasma exchange in a refractory case of Drug induced liver injury and patient had a complete recovery (Transplant Free survival). He concluded that use of extracorporeal therapies like plasma exchange could be considered early in such cases leading to successful outcomes and this report supports our case study.

Larsen et al. demonstrated that high-volume plasma exchange significantly improved transplant-free survival in patients with ALF, likely through modulation of innate immune responses and reduction of multi-organ dysfunction (4). Similarly, Maiwall et al. reported that plasma exchange is safe and may improve survival by reducing cytokine storm and serum ammonia levels (5).

In contrast, Burke et al., in a large cohort study from the United Kingdom, found no significant difference in overall survival or transplant-free survival between patients treated with plasma exchange and those receiving standard medical therapy, although improvements in hemodynamic parameters were noted (6). These conflicting findings underscore the need for further well-designed randomized controlled trials to define the precise role, timing, and patient selection criteria for TPE in ALF.

In our patient, early initiation of TPE before the onset of irreversible multi-organ failure likely contributed to the favorable outcome. This supports the concept that TPE may be most beneficial when instituted early in the disease course as a bridging or rescue therapy**.**

4. Conclusion

The potential role of therapeutic plasma exchange as an effective adjunct in severe paracetamol-induced acute liver failure. Early initiation of TPE may improve clinical outcomes, facilitate recovery, and reduce the need for liver transplantation. Further studies are warranted to better define its indications, timing, and optimal protocols.

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