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Therapeutic Plasma Exchange Procedures and Outcomes in a Pediatric Intensive Care Unit: A Single-center Experience

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ABSTRACT

Objective: Therapeutic plasma exchange (TPE) is an apheresis procedure in which the patient's plasma is removed and replaced with replacement fluid. We report an 11-year single-center experience with pediatric TPE in a pediatric intensive care unit (PICU).

Methods: This retrospective, single-center, observational study included 51 pediatric patients who underwent 248 TPE procedures in the PICU over an 11-year period. The indications for TPE were grouped as follows: 1. microangiopathic hemolytic anemia, including sepsis, disseminated intravascular coagulation (DIC), thrombocytopenia-associated multiorgan failure (TAMOF), hemolytic uremic syndrome (HUS), and hepatitis; 2. hemophagocytic lymphohistiocytosis (HLH) or macrophage activation syndrome (MAS); 3. intoxications; 4. neurological diseases. The clinical and laboratory data of these patients, including organ failure and scores on the pediatric logistic organ dysfunction (PELOD) and Glasgow coma scale, were recorded.

Results: The time for TPE after admission to PICU was 2.2±5.1 days. Twenty-eight (54.9%) patients were discharged, and 23 (45.1%) died. Minor complications that can be attributed to TPE were observed in 14.9% of the patients. Earlier initiation of TPE appeared to be associated with more favorable outcomes. A significant increase in platelet count was observed in Group 1, and favorable responses were also noted in selected intoxication cases. In contrast, no clear additional benefit was demonstrated in the HLH/MAS group.

Conclusion: TPE may be considered an option in pediatric patients with sepsis, DIC, TAMOF, HUS, and hepatitis (Group 1), as well as in selected intoxication cases. In Group 1, improvements in platelet counts and PELOD scores were the principal responses to treatment.

Keywords: Therapeutic plasma exchange, pediatric, critically ill children, intensive care unit

INTRODUCTION

"Apheresis" means "forced removal" in Greek. In many diseases, the problem is not only the accumulation of harmful substances but also the depletion of other substances. In such patients, removal and replacement of the patient's plasma may be beneficial. This procedure, called therapeutic plasma exchange (TPE), is non-selective and removes both normal and pathological plasma components, including antibodies, immune complexes, and cytokines (1). Although the removal of the pathologic substances is the major mechanism of action of TPE, various immunomodulatory effects of TPE, including the shift of the TH1/TH2 balance towards TH2 and the inhibition of interleukin-2 and interferon-γ production were reported (2).

The American Society for Apheresis (ASFA) published a categorized list of indications for therapeutic apheresis using an evidence-based approach (3). ASFA guidelines are the main reference for decision-making regarding indications in pediatric and adult patients. When taking indications in consideration, it can be seen that number of diseases which can be treated with TPE has increased significantly over the years (4).

Limited data are available on the safety and efficacy of TPE in pediatric patients. We aimed to present a single-center, 11-year

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J Acad Res Med 2026;16(2):106-112

TPE experience in a pediatric intensive care unit (PICU), including indications, complications, outcomes of TPE use, and prognostic factors affecting TPE success.

METHODS

This study was a retrospective, single-center, observational study. The medical records of pediatric patients who underwent TPE in the PICU over an 11-year period were retrospectively reviewed. A total of 248 TPE procedures, performed in 51 patients, were included in the analysis. All pediatric patients who underwent at least one TPE procedure in the PICU during the study period were eligible for inclusion. Patients with insufficient medical records were excluded from the relevant analyses.

All TPE procedures were performed after written informed consent had been obtained from the parents or legal guardians.

The patients were categorized into four groups (Table 1):

Group 1: Microangiopathic hemolytic anemia group including patients with sepsis, disseminated intravascular coagulation (DIC), thrombocytopenia-associated multiorgan failure (TAMOF), HUS, and hepatitis (n=23).

Group 2: Patients with hemophagocytic lymphohistiocytosis (HLH)or macrophage activation syndrome (MAS), (n=11).

Group 3: Patients with intoxications at fatal doses with amitriptyline, carbamazepine, colchicine, mushrooms, or digoxin (n=12).

Group 4: Patients with neurological diseases, including Guillain-Barre syndrome, encephalitis, and Rasmussen encephalitis (n=5).

Complete blood count, serum liver and kidney function tests, and electrolytes were ordered before and after each TPE session for all patients.

The serum BUN and creatinine results from patients who underwent hemodialysis were excluded from the analyses. Serum ferritin, lactate dehydrogenase (LDH), triglyceride levels, aPTT, and PT were studied in Groups 1 and 2; thrombin time, plasma anti-thrombin III, and fibrinogen were studied in Groups 1-3. The clinical and laboratory data of these patients, including Pediatric Logistic Organ Dysfunction (PELOD) scores (5), organ failure scores [as recommended by Goldstein et al. (6)], and Glasgow coma scale (GCS) scores were noted (7).

Before the initiation of TPE, patients with hemoglobin levels <9 g/dL or platelet counts <50 x 10^9/L were transfused. Fresenius As. Tec 204™, COM TEC, or OPTIA devices were used for the TPE procedure. Veno-venous access was used, and either albumin or fresh-frozen plasma (FFP) was preferred as the replacement fluid. In each procedure, TPE was performed with an exchange of 1 to 1.5 plasma volumes.

The termination of TPE was decided by numerous clinical and laboratory criteria: in patients with TAMOF, TPE was ceased when organ failures remitted and platelet count >100 x10^9/L, in those with HLH, when serum LDH levels normalized and platelet count >100 x10^9/L, in patients with intoxication, when serum levels of the responsible agent decreased and intoxication signs and symptoms receded, and in those with Guillain-Barre syndrome, after the completion of five cycles.

Statistical Analyses

Statistical analyses were performed using SPSS for Windows, version 15.0 (SPSS Inc., Chicago, IL, USA). The normality of continuous variables was assessed using the Kolmogorov-Smirnov test. Continuous variables were summarized as mean ± standard deviation together with minimum and maximum values, which were considered more informative given the wide range of laboratory parameters observed in critically ill pediatric patients. Categorical variables were expressed as numbers and percentages.

Due to the non-normal distribution of several variables, paired comparisons of laboratory measurements before and after TPE [platelet count, albumin, aspartate aminotransferase (AST), alanine aminotransferase (ALT), alkaline phosphatase (ALP), LDH, prothrombin time, international normalized ratio (INR), and serum electrolyte levels] were performed using the Wilcoxon signed-rank test. Changes in PELOD and organ failure scores before and after TPE were also analyzed using the Wilcoxon signed-rank test. Comparisons between categorical variables were performed using the chi-square test or Fisher's exact test, as appropriate.

Standardized mortality rates (SMRs) were calculated using the exact Poisson method, and the total SMR was 0.73 (95% confidence interval: 0.44-1.06). A two-sided p-value of <0.05 was considered statistically significant.

The study protocol was approved by the Institutional Ethics Committee of Hacettepe University (approval number: LUT 12/92-46, date: 11.09.2012). Written informed consent for the TPE procedure was obtained from the parents or legal guardians as part of routine clinical practice.

RESULTS

The mean age of the patients was 8±5.2 (0.1-16) years and 28 (54.9%) were female. A total of 248 procedures were performed on 51 patients. The underlying indications for TPE were summarized in Table 1. The mean number of TPE cycles performed on one patient was 4.9±4.6 (1-19). The mean time of one TPE cycle was 87.5±29 minutes (24-180). The overall mean time elapsed from admission to the PICU until the initiation of TPE was 2.2±5.1 days (1-32). For groups 1-4, the mean times to TPE initiation in Groups 1-4 were 2.3±6.5 days, 2.6±4.9 days, 0.2±0.4 days, and 5±3.8 days, respectively.

The devices used for TPE were COMTEC (53.8%), OPTIA (33.3%), and Fresenius AS 204 (12.9%). The central venous sites for TPE were the jugular vein (89.7%) and the femoral vein (10.3%). The diameter of the catheters ranged from 4 to 12 Fr.

The replacement fluid was FFP in 209 (90.5%) and 5% albumin in the remainder. FFP was used in all patients in Group 1, in 94.9% of those in Group 2, and in 93.3% of those in Group 3. All patients in Group 4 underwent TPE with 5% albumin. Citrate was used as an anticoagulant throughout the procedures, and

J Acad Res Med 2026;16(2):106-112

prophylactic calcium gluconate was administered to all patients during TPE.

All patients' GCSs increased significantly after TPE compared with before TPE (p=0.038). The post-TPE values in Group 3 patients were significantly higher than those in the other groups (p=0.005). Among the survivors, the difference in pre- and post-TPE GCS values of Group 1 was significantly higher than in the other groups (p=0.043).

Of the 51 patients, 23 (45.1%) died and 28 (54.9%) were discharged. The mortality rates in Groups 1, 2, 3, and 4 were 60%, 63%, 8%, and 20%, respectively (Table 1). The overall mean time until death after admission to the PICU was 27.5±40.3 days (1-186). Invasive mechanical ventilation was used in 35 patients, of whom 22 (62.8%) died before being weaned from the ventilator. The remaining 13 patients (37.2%) were extubated. One of the extubated patients died on the 10th post-extubation day. Seventy-five percent of patients in Group 1 were successfully extubated within 48 hours (early), while all patients in Group 3 were off mechanical ventilation within 24 hours (very early). In Groups 2 and 4, early extubation was not possible.

In Group 1, the survival rates of patients who received the first TPE session within 24 hours (n=10), within 25-48 hours (n=5), and within 49-72 hours (n=6) of admission were 40%, 40%, and 50%, respectively. Those in Group 1 who had their first TPE after the 3rd day of admission (n=2) died.

The SMRs were calculated according to the PRISM scores and summarized in Table 2.

Table 1. Clinical characteristics of the patients

	Indication for TPE	Number of cycles/ patient mean ± SD	Deceased patients n (%)	Pre-TPE PELOD score mean ± SD median (25th-75th percentile)	Post-TPE PELOD score mean ± SD median (25th-75th percentile)	P-value for the difference of pre-TPE, post-TPE PELOD scores*	Pre-TPE organ failure score mean ± SD median (25th-75th percentile)	Post-TPE organ failure score mean ± SD median (25th-75th percentile)	P-value for the difference of pre-TPE, post-TPE organ failure scores*
Group 1 (n=23)	-Sepsis and TAMOF (n=10) -HUS (n=7) -DIC (n=4) -Hepatitis (n=2)	6.4 (1-19)	14 (60)	All groups 14.1±11.2 12 (10-21)	All groups 12.6±13.6 11 (1-23)	All groups p=0.234	All groups 3.7 (±1.5) 4 (2-5)	All groups 3.0 (±2.0) 3 (1-5)	All groups p=0.035
				Among survivors 9.1±7.0 10 (1.5-12)	Among survivors 2.9±4.4 1 (0-6)	Among survivors p=0.007	Among survivors 2.8±1.6 2 (2-4.5)	Among survivors 1.0±1.4 0 (0-2)	Among survivors p=0.020
Group 2 (n=11)	-MAS (n=7) [secondary to JRA (n=5) or SLE (n=2) -HLH (n=4)] [secondary to infections (n=2) or propionic acidemia (n=2)]	5.4 (1-13)	7 (63)	All groups 8.6±8.9 10 (2-12)	All groups 11.7±8.4 11 (1-23)	All groups p=0.239	All groups 2.7±1.2 2 (2-3)	All groups 3.3±1.2 3 (2-4)	All groups p=0.096
				Among survivors 4.0±6.0 1.5 (0.3-10.3)	Among survivors 2.5±0.6 2.5 (2-3)	Among survivors p=0.713	Among survivors 2.2±0.9 2.5 (1.3-3)	Among survivors 2.5±0.6 2.5 (2-3)	Among survivors p=0.317
Group 3 (n=12)	-Amitriptyline (n=5) -Carbamazepine (n=2) -Colchicine (n=2) -Mushroom (n=2) -Digoxin (n=1)	1.8 (1-5)	1 (8)	All groups 9.6±9.9 10 (1-17.8)	All groups 0.6±0.9 0 (0-1)	All groups p=0.017	All groups 1.5±1.0 2 (0.3-2)	All groups 0.4±0.6 0 (0-1)	All groups p=0.016
Group 4 (n=5)	-Guillain-Barre syndrome (n=3) -Encephalitis (n=1) -Rasmussen encephalitis (n=1)	3.6 (1-5)	1 (20)	All groups 9.0±17.8 1 (1-21)	All groups 8.8±18 1 (0.5-21)	All groups p=0.317	All groups 1.4±0.8 1 (1-2)	All groups 0.8±1.7 0 (0-2)	All groups p=0.180
Total (n=51)		4.9 (1-19)	23 (45)	All groups 11.3±11.2 10 (1-13)	All groups 9.2±12.1 2 (1-13)	All groups p=0.093	All groups 2.7±1.6 3 (2-4)	All groups 2.2±2.0 2 (0-4)	All groups p=0.012
				Among survivors 7.75±8.26 6 (1-11.8)	Among survivors 1.57±2.68 1 (0-2)	Among survivors p=0.001	Among survivors 1.89±1.34 2 (1-2.8)	Among survivors 0.82±1.18 0 (0-2)	Among survivors p=0.001

*: Wilcoxon test, TPE: Therapeutic plasma exchange, SD: Standard deviation, TAMOF: Thrombocytopenia-associated multiorgan failure, HUS: Hemolytic uremic syndrome, DIC: Disseminated intravascular coagulation, MAS: Macrophage activation syndrome, HLH: Hemophagocytic lymphohistiocytosis

J Acad Res Med 2026;16(2):106-112

Only Group 1 showed a significant increase in mean platelet count after TPE procedures compared to pre-TPE values (90435 vs. 101478 K/uL, p=0.011). The serum albumin levels significantly increased among the survivors of Group 1 after TPE (p=0.025) 3.01 vs. 3.78 g/L). The decrease in serum AST levels after TPE was associated with better survival in all patients and in Group 1 (767 vs. 152 IU/L, p=0.002 in all patients, and 442 vs. 53 IU/L, p=0.008 in Group 1); the same was observed for decreases in LDH (2149 vs. 852 IU/L, p=0.002 in all surviving patients, and 2049 vs. 934 IU/L, p=0.012 in Group 1) and in PELOD scores (7.75 vs. 1.57, p=0.001 in all patients, and 9.11 vs. 2.89, p=0.007 in survivors of Group 1). Serum ALT and ALP levels, as well as prothrombin time and INR, decreased after TPE in all patients, but none of these reductions were associated with improved survival rates. The serum electrolyte levels did not change significantly with TPE treatment in any of the patients (Table 3).

The complications attributable to TPE included rash (4.9%), electrolyte imbalances (4.4%), including hypocalcemia, hypokalemia, and hypernatremia, nausea and vomiting (1.2%), fever (1.2%), hypotension (1.2%), altered consciousness (0.8%), hypertension (0.4%), epistaxis (0.4%), and catheter occlusion (0.4%).

DISCUSSION

Indications for TPE in pediatric patients are often based on adult data, and the ASFA guidelines do not include age-specific recommendations regarding indications. Rather, they are categorized according to strength, ranging from 1 to 4 (3). Category I disorders are those in which TPE may be used as first-line therapy, either alone or in conjunction with other therapeutic options, such as thrombotic thrombocytopenic purpura (TTP), dialysis-independent or alveolar hemorrhage-associated Wegener granulomatosis or Goodpasture syndrome, recurrent focal segmental glomerulosclerosis, Guillain-Barre syndrome, chronic inflammatory demyelinating polyradiculopathy, cryoglobulinemia, hemolytic uremic syndrome related to autoantibody to factor H, pediatric autoimmune neuropsychiatric disorders associated with streptococcal infections and antibody-mediated rejection after renal transplantation (8). Many other diseases may benefit from TPE (8,9). Although peripheral access may be used in older children, central venous access is usually required. The major risks of TPE are related to catheters, including infections, bleeding, thrombosis, pneumothorax, hemothorax, or cardiac arrhythmias (10). A common problem during TPE is paresthesia due to hypocalcemia caused by citrate chelation. Citrate is used as an anticoagulant to prevent the blood from clotting in the

Table 2. The SMR of patients

	Observed mortality (n)	Expected mortality (n)	SMR (95% CI)
Group 1	14	18	0.78
Group 2	7	7	1
Group 3	1	6	0.17
Group 4	1	2	0.5
Total	23	33	0.73 (0.44-1.06)

SMR: Standardized mortality rates, CI: Confidence interval

Table 3. Changes in laboratory parameters before and after TPE

	Pre-TPE mean ± SD (min-max)	Post-TPE mean ± SD (min-max)	p-value*
AST (U/L)	1828±5510 (14-28831)	584±1866 (11-12166)	0.001
ALT (U/L)	823±2254 (8-9960)	234±588 (5-3369)	0.003
ALP (U/L)	164±131 (23-686)	103±88 (18-615)	<0.001
LDH (U/L)	2579±2763 (98-12000)	1988±3957 (425-21626)	0.007
aPTT (s)	36±10 (17.6-63.0)	32±12 (1.1-82.1)	0.011
INR (-)	2.12±1.60 (1.02-9.31)	1.50±0.76 (0.88-5.31)	0.001
Sodium (mmol/L)	137.5±6.0 (126-158)	140.4±5.5 (128-156)	0.055
Potassium (mmol/L)	3.99±0.81 (2.20-5.70)	3.66±0.91 (2.02-6.45)	0.459
Calcium, total (mg/dL)	8.58±1.55 (4.20-13.20)	9.36±1.45 (6.20-13.90)	0.258
Calcium, ionized (mmol/L)	1.06±0.22 (0.16-1.43)	1.12±0.17 (0.69-1.47)	0.414
Phosphorus (mg/dL)	4.40±1.80 (0.35-10.86)	4.27±1.69 (1.39-10.67)	0.903
Glucose (mg/dL)	133±85 (48-601)	136±60 (53-334)	0.453

*: Paired comparisons were performed using the Wilcoxon signed-rank test, TPE: Therapeutic plasma exchange, AST: Aspartate aminotransferase, ALT: Alanine aminotransferase, ALP: Alkaline phosphatase, LDH: Lactate dehydrogenase, aPTT: Activated partial thromboplastin time, INR: International normalized ratio, SD: Standard deviation

J Acad Res Med 2026;16(2):106-112

TPE device and the risk for hypocalcemia is higher in patients with underlying renal or hepatic impairment (11,12). This risk is eliminated if heparin is used instead of citrate. Another common complication during TPE is hypotension, which may be related to hypocalcemia or hypovolemia (10). The coagulation factors decrease by one volume of plasma exchange and factors VIII, IX, and vWF return to normal within 4 hours, whereas the remaining coagulation factors achieve pre-TPE levels by the 24th hour, excluding fibrinogen, which takes 72 hours to return to normal levels after one TPE session (11). Anti-coagulation factors also decrease after TPE, including anti-thrombin III, and there is data on the increased risk of thrombosis after this procedure (11). A decrease in pseudocholinesterase levels after TPE may prolong neuromuscular junction blockage (13).

The replacement fluids include either albumin in physiologic saline or plasma. Plasma poses a risk of transfusion reactions or transfusion-related infections. However, it is the only choice for patients who undergo TPE due to TTP or DIC (11,14).

Previous studies have reported that earlier initiation of TPE is associated with better survival outcomes. Qu et al. (15) analyzed the TPE results in patients with fulminant sepsis aged 8 months to 14 years and reported that all patients who received TPE within 2-10 hours survived. They also reviewed 10 studies including 60 patients with fulminant sepsis and noted that delayed TPE initiation was related to increased mortality (15). Chong et al. (16) analyzed children with critical heart diseases and a clinical diagnosis of TAMOF and reported that patients who received TPE early (within <1 day of ECMO cannulation) had significantly improved modified organ failure index scores and platelet counts compared to patients with late TPE initiation. In the study of Kawai et al. (17) on 14 children with sepsis-induced TAMOF, initiating TPE early (within <30 hours from admission) was associated with greater improvement in organ dysfunction and decreased requirement for vasoactive and/or inotropic agents. In our study, the mean time to initiation of TPE was 2.2 days. In Group 1, earlier TPE initiation appeared to be associated with more favorable outcomes; however, this observation should be interpreted cautiously because of the small sample size. In our patients with MAS and HLH, the timing of TPE was not correlated with improved survival; however, this may be attributed to the small sample size. Group 3 included patients with intoxications; all underwent TPE within 24 hours, and only one patient died. No clear association between earlier initiation and improved outcomes was observed in Group 4.

Among all our patients, there were adverse events that may be attributed to TPE in 14.9%, of which rash and electrolyte imbalance were the most common, similar to the literature (18-20). No TPE-related mortality occurred. Michon et al. (21) examined 1632 apheresis procedures performed on 186 pediatric patients and observed adverse events in 55% of procedures and 82% of patients. Hypotension and symptomatic hypocalcemia were the leading adverse events; two patients died from TPE complications. The risk factors associated with complications were low body weight, low hemoglobin level, apheresis in the PICU, and an increased number of procedures per patient (21). In a similar study, 4.3% serious adverse effects had been observed (22). In a study by Sık et al. (4), minor adverse effects were seen in 16.5% of the sessions and no patients died because of TPE. They stated that circuit clotting and access malfunction were the most frequent adverse events. In a study conducted among European pediatric nephrology units, the rate of minor adverse effects observed with plasma exchange was 6.9%, hypotension, and premature disconnection being the most common (23).

In our cohort, 35 patients required ventilation support, of whom 22 (62.8%) died before extubation. Patients in Groups 1 and 3 had the shortest intubation periods. Similarly, in a study of 48 critically ill children, Cortina et al. (24) stated that the rate of ventilated patients among the survivors and non-survivors were 42.5%, and 75%, respectively.

In a prospective study on thrombocytopenia-associated multiple organ failure, Fortenberry et al. (25) showed that 28-day all-cause mortality was lower in children treated with TPE compared with those who were not. A retrospective study conducted in 43 hospitals over 9 years reported mortality rates of 22% among TPE patients without MODS and 44.4% among those with MODS. Children receiving TPE had higher mortality because of greater comorbidities and MODS (26). In our current study, the mortality rate among patients with sepsis and TAMOF was 60,% with an SMR of 0.78. An SMR of <1 indicates lower-than-expected mortality among Group 1 patients who underwent TPE. In the HLH/MAS group, observed mortality was similar to expected mortality, and no clear survival advantage associated with TPE could be demonstrated. In a previous study from our center, we reported lifesaving TPE applications in three severe amitriptyline intoxication cases (27). Group 3 had the lowest SMR scores, and only 1 out of 12 patients (8%) who was intoxicated with a lethal dose of colchicine (>0.8 mg/kg) died. The greatest benefit of TPE for survival was observed in this group. TPE has limited benefit in colchicine intoxication because of its large volume of distribution.

The GCSs of patients in Group 3 significantly increased after TPE, indicating rapid toxin removal that led to resolution of the comatose state. PELOD scores significantly decreased after TPE in all survivors, with a larger decrease observed in Group 1. It is reasonable to observe the greatest improvement in PELOD scores among Group 1 patients, since this group also included patients who had TAMOF. The decrease in PELOD scores after TPE in survivors indicated a beneficial effect of TPE on the progression of organ failure. Similar to PELOD scores, the scoring by Goldstein et al. (6) revealed a decrease in evidence of organ failure after TPE.

Among survivors in all groups, platelet counts increased post-TPE. The significant increase in platelet counts with TPE in Group 1 patients may serve as an indicator of treatment response. Chong et al. (16) had already reported a similar trend of increase in platelet counts with TPE in critically ill cardiac children with TAMOF.

J Acad Res Med 2026;16(2):106-112

Study Limitations

One of the major strengths of this study is its reliance on a large number of TPE procedures performed in a single PICU over an 11-year period. The inclusion of different disease groups allowed for a broader perspective on the effects of TPE in various clinical presentations. The comparative analysis of clinical parameters [e.g., pre- and post-TPE organ failure scores (PELOD) and GCS] and biochemical parameters (laboratory values) offers a significant methodological advantage. Furthermore, standardization of the devices used in TPE (COMTEC, OPTIA, Fresenius AS 204) is valuable for ensuring data consistency.

However, the study has several limitations. First, its retrospective design precludes establishing a cause-and-effect relationship. Furthermore, changes in clinical practices and treatment protocols over the long period of data collection may have influenced the results. The small number of patients in some subgroups (especially Groups 2 and 4) limits the generalizability of findings. In addition, the relatively small sample size limited the study's statistical power and precluded the use of advanced modeling approaches, such as logistic or Cox regression analyses.

CONCLUSION

The clinical and laboratory responses to TPE varied by underlying indication. The most favorable responses were observed in selected patients in Group 1 and in some cases of intoxication. Among survivors, increased platelet counts and improvements in PELOD scores were the main indicators of treatment response in Group 1. Earlier initiation may be clinically important, although definitive conclusions regarding treatment timing cannot be drawn because of the retrospective design and limited sample size. No clear additional survival benefit was demonstrated in the HLH/MAS group. Prospective multicenter studies are needed to clarify patient selection, optimal timing, and clinical effectiveness.

Ethics

Ethics Committee Approval: The study protocol was approved by the Institutional Ethics Committee of Hacettepe University (approval number: LUT 12/92-46, date: 11.09.2012).

Informed Consent: Written informed consent for the TPE procedure was obtained from the parents or legal guardians as part of routine clinical practice.

Footnotes

Author Contributions: Surgical and Medical Practices - E.A.A., S.K., Ş.Ü.C., S.A., B.B.; Concept - E.A.A.; Design - E.A.A., B.B.; Data Collection and/or Processing - E.A.A.; Analysis and/or Interpretation - E.A.A., B.B.; Literature Search - E.A.A.; Writing - E.A.A.

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J Acad Res Med 2026;16(2):106-112

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