Cureus
Part of SPRINGER NATURE

Open Access Case Report

Sequential Plasma Exchange and Tacrolimus for Probable Autoimmune Hepatitis Presenting As Acute-on-Chronic Liver Failure

Review began 10/02/2026
Review ended 10/19/2026
Published 10/24/2026

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DOI: 10.7759/cureus.118492

Mehdi Zouaoui [1, 2], Mounir El Bied [1, 2], Naima Belasri [1, 2], Mbarek Azouaoui [1, 2], Youssef Hnach [3, 2], Nourdin Aqodad [1, 2]

1. Department of Hepato-Gastroenterology, Mohammed VI University Hospital Center, Agadir, MAR 2. Faculty of Medicine and Pharmacy of Agadir, Ibn Zohr University, Agadir, MAR 3. Department of Hepato-Gastroenterology, Oued Eddahab Military Hospital, Agadir, MAR

Corresponding author: Mounir El Bied, mounirelbied27061998@gmail.com

Abstract

Autoimmune hepatitis (AIH) is an immune-mediated inflammatory liver disease that may first become apparent through severe decompensation of previously unrecognized cirrhosis. We report a 46-year-old woman with probable AIH (simplified diagnostic score 6), culture-negative spontaneous bacterial peritonitis, marked jaundice, and subsequent West Haven grade III hepatic encephalopathy despite corticosteroid therapy. Liver and cerebral failure were consistent with European Association for the Study of the Liver-Chronic Liver Failure grade 2 acute-on-chronic liver failure, conditional on the clinical diagnosis of cirrhosis. Liver transplantation was unavailable locally. Five therapeutic plasma exchange sessions produced an initial improvement in bilirubin and neurological status, but asterixis and hyperbilirubinemia recurred after an approximately nine-day interval. Three further exchanges were followed by tacrolimus at 1 mg twice daily. At the last follow-up, encephalopathy had not recurred, and aminotransferases had improved, although marked cholestasis and mild ascites persisted. Plasma exchange may provide temporary support while further treatment is established, but this observation cannot isolate its effect from concurrent immunosuppression and infection management.

Categories: Therapeutics, Internal Medicine, Gastroenterology
Keywords: acute-on-chronic liver failure, autoimmune hepatitis, corticosteroid therapy, hepatic encephalopathy, liver failure, liver transplantation, tacrolimus, therapeutic plasma exchange

Introduction

Autoimmune hepatitis (AIH) is a chronic immune-mediated inflammatory liver disease characterized by hepatocellular injury, elevated serum immunoglobulin G (IgG), and circulating autoantibodies. Loss of immune tolerance to hepatic antigens produces persistent inflammation, which may lead to progressive fibrosis when untreated [1]. Diagnosis rests on the combination of biochemical findings, autoantibodies, IgG elevation, exclusion of competing causes, and histology when a biopsy can be obtained [1,2]. Clinical presentations range from incidental biochemical abnormalities to cirrhosis and severe hepatic decompensation. In a patient with cirrhosis, acute decompensation accompanied by organ failure may meet the definition of acute-on-chronic liver failure (ACLF) [3].

Corticosteroids remain the cornerstone of initial treatment for AIH, while early assessment of treatment response and timely evaluation for liver transplantation are essential in severe presentations [1]. Tacrolimus, a calcineurin inhibitor, represents an alternative immunosuppressive option in selected patients with an inadequate response or intolerance to conventional therapy, although its role in severe AIH complicated by liver failure remains insufficiently established [1].

Therapeutic plasma exchange (TPE) removes the patient's plasma and replaces it with donor plasma or another replacement fluid, facilitating the removal of circulating bilirubin, inflammatory mediators, and other protein-bound substances while replenishing plasma proteins and coagulation factors. In severe hepatic failure, TPE may provide temporary neurological and biochemical stabilization and may be considered as a supportive rescue or bridging strategy while liver transplantation is being evaluated or remains unavailable. However, TPE does not directly control the underlying autoimmune process, and its beneficial effects may therefore be transient. Furthermore, evidence supporting its use specifically in AIH-related ACLF remains limited [4-7].

We report a case of probable AIH presenting as ACLF in which TPE was associated with initial but transient clinical and biochemical improvement, followed by the introduction of tacrolimus for ongoing immunosuppressive management in a setting where timely liver transplantation was unavailable.

Case Presentation

A 46-year-old woman with no previously known liver disease, no history of malignancy, and no reported alcohol or tobacco use presented with approximately three weeks of progressive cholestatic jaundice, generalized pruritus, dark urine, pale stools, and deterioration in her general condition. No clinical evidence of active bleeding, including gastrointestinal bleeding, was reported. On hospital day 1, she was conscious but exhibited asterixis, marked jaundice, and moderate ascites. Abdominal ultrasonography revealed a dysmorphic liver with segment I hypertrophy, splenomegaly (14 cm), and ascites, suggesting previously unrecognized cirrhosis with portal hypertension. Diagnostic paracentesis revealed 900 leukocytes/mm^3, including 90% neutrophils (approximately 810 neutrophils/mm^3), with negative ascitic fluid cultures, consistent with culture-negative spontaneous bacterial peritonitis.

Initial laboratory investigations demonstrated hepatocellular injury, cholestasis, impaired hepatic synthetic function, thrombocytopenia, and marked hypergammaglobulinemia. The principal admission laboratory values are summarized in Table *1*. The initial hemoglobin concentration was 10.5 g/dL, and serum ferritin was 150 µg/L. The etiological workup included serum ceruloplasmin (0.32 g/L), total serum copper (89 µg/dL), and 24-hour urinary copper excretion (25 µg/24 hours), all within the respective reference ranges. Slit-lamp examination revealed no Kayser-Fleischer rings. HIV serology was negative. Antismooth muscle antibody results were unavailable, alpha-1 antitrypsin testing was not performed, and no hemolysis workup results were available.

Parameter	Patient value	Unit	Reference range
Alanine aminotransferase	355	U/L	7-35
Aspartate aminotransferase	281	U/L	8-35
Alkaline phosphatase	527	U/L	35-104
Gamma-glutamyl transferase	160	U/L	5-36
Total bilirubin	>25	mg/dL	0.3-1.2
Albumin	25	g/L	35-50
Prothrombin activity	34	%	70-100
Platelet count	80	×10^9/L	150-400
Hemoglobin	10.5	g/dL	12-16
Urea	0.2	g/L	0.15-0.45
Serum creatinine	0.8	mg/dL	0.6-1.1
Immunoglobulin G	33.7	g/L	7.67-15.90
Serum ferritin	150	µg/L	15-150
Serum ceruloplasmin	0.32	g/L	0.20-0.40
Total serum copper	89	µg/dL	70-140
24-hour urinary copper excretion	25	µg/24 hours	<40
Antinuclear antibodies	Positive, 1:80	Titer	Negative <1:80*

TABLE 1: Initial laboratory findings at admission (hospital day 1)

*ANA reference values and positivity cutoffs are assay-dependent

ANA: antinuclear antibodies

Prothrombin activity was 34% at admission, and renal function was preserved. The estimated Child-Pugh score was 14 (class C) [8], based on bilirubin, albumin, moderate ascites, hepatic encephalopathy, and impaired coagulation, as reflected by prothrombin activity.

Serological tests for hepatitis A, B, C, and E, cytomegalovirus, herpes simplex virus, Epstein-Barr virus, and human immunodeficiency virus were negative. Serum IgG was elevated at 33.7 g/L (laboratory upper limit, 12 g/L), antinuclear antibodies were positive at a titer of 1:80, and antisoluble liver antigen antibodies were positive. No medication or herbal exposure was reported. Abdominal ultrasonography showed no biliary

obstruction, with patent hepatic veins and inferior vena cava. Percutaneous liver biopsy was deferred because of coagulopathy and the associated bleeding risk, while transjugular liver biopsy was unavailable at our center. The nonhistological components of the simplified International Autoimmune Hepatitis Group score totaled 6 points (antisoluble liver antigen antibodies, 2 points; elevated IgG, 2 points; and exclusion of viral hepatitis, 2 points), supporting a diagnosis of probable AIH in the absence of histological confirmation [2]. Liver biopsy remained desirable once the patient's clinical condition and procedural safety permitted.

Spontaneous bacterial peritonitis was treated with intravenous ceftriaxone 2 g/day for five days, starting on hospital day 1, together with intravenous albumin at 1.5 g/kg (90 g) on hospital day 1 and 1 g/kg (60 g) on hospital day 3, based on a body weight of 60 kg. Repeat diagnostic paracentesis after 48 hours showed a decrease in ascitic neutrophils to approximately 230/mm³, representing a reduction of approximately 72% from baseline and supporting a favorable response to antibiotic treatment. Intravenous methylprednisolone was initiated on hospital day 3 at 60 mg/day (1 mg/kg/day), following this reassessment of the ascitic infection.

After approximately one week of corticosteroid therapy, the patient developed worsening neurological manifestations, including severe confusion, somnolence, and asterixis, consistent with West Haven grade III hepatic encephalopathy [9]. Her Glasgow Coma Scale (GCS) score was 13/15 [10]. Brain computed tomography performed without and with intravenous contrast showed no significant abnormalities, and serum electrolyte levels were within normal limits. At this stage, total bilirubin was 21.6 mg/dL, and prothrombin activity was 48%. Given the neurological deterioration and insufficient clinical response to corticosteroid therapy, liver transplantation was considered but was not available locally.

TPE was initiated approximately eight days after the introduction of corticosteroids as a supportive rescue intervention in the setting of worsening hepatic encephalopathy and persistent hepatic dysfunction. The estimated plasma volume (PV) was calculated using the following formula: $\mathrm{PV\ (L)} = 0.07 \times \mathrm{Body\ Weight\ (kg)} \times (1 - \mathrm{Hematocrit})$. For a body weight of 60 kg and a pretreatment hematocrit of 45%, the estimated PV was 2.31 L. Each session involved administering thirteen 250-mL units of fresh frozen plasma, corresponding to a replacement volume of 3.25 L (approximately 1.41 estimated PVs). The total replacement volume across eight sessions was 26 L. Plasma exchange was performed through a transjugular central venous catheter. According to the procedural records, anticoagulation consisted of unfractionated heparin at an initial dose of 30 IU/kg, followed by an infusion of 10 IU/hour. No procedure-related adverse events were reported.

An initial course of five TPE sessions was performed over approximately one week. Total bilirubin decreased from 21.6 mg/dL before the first session to 5.3 mg/dL after the fourth session, while prothrombin activity increased from 48% to 65%, with a postexchange international normalized ratio (INR) of 1.27. Bilirubin subsequently reached 4.1 mg/dL, accompanied by improvement in neurological status, with the patient becoming conscious after the initial course. These findings indicated initial clinical and biochemical improvement associated with TPE, although the contribution of concurrent treatments could not be excluded.

Approximately nine days after the fifth TPE session, asterixis recurred, accompanied by an increase in total bilirubin to 6.9 mg/dL, an INR of 1.32, and prothrombin activity of 60%. At the time of this neurological recurrence, the white blood cell count was 5,430/mm³, and C-reactive protein was 18 mg/L. Although there was no leukocytosis, these findings alone could not definitively exclude an infectious precipitant. Three additional TPE sessions were subsequently performed, after which total bilirubin decreased to 3.6 mg/dL and prothrombin activity was 61%. The recurrence following the initial course highlighted the transient nature of the improvement observed with plasma exchange.

Tacrolimus was subsequently introduced at 1 mg twice daily (2 mg/day) as an alternative immunosuppressive treatment and was maintained at this dose. Around the time of its introduction, total bilirubin was 5.1 mg/dL, and prothrombin activity was 53%. Twelve-hour predose tacrolimus trough concentrations measured approximately one, three, and five days after treatment initiation were 6.0, 6.1, and 6.3 ng/mL, respectively. Tacrolimus was intended to provide ongoing immunosuppression rather than the immediate supportive effect sought with TPE.

The patient was discharged approximately two months after admission, conscious and clinically stable, although total bilirubin remained elevated at approximately 8.8 mg/dL near discharge, with persistent cholestatic liver test abnormalities.

At the most recent follow-up, hepatic encephalopathy had not recurred, although mild conjunctival icterus and small-volume ascites persisted. Laboratory investigations showed aspartate transaminase 64 U/L, alanine aminotransferase 66 U/L, gamma-glutamyl transferase (GGT) 1,305 U/L, total bilirubin 2.4 mg/dL, direct bilirubin 0.715 mg/dL, albumin 25 g/L, prothrombin activity 99%, sodium 136 mmol/L, urea 0.4 g/L, and serum creatinine 0.6 mg/dL. The tacrolimus trough concentration was 6.1 ng/mL. The estimated Child-Pugh score was 9 (class B) [8]. Although aminotransferase levels and overall clinical status had improved, significant cholestasis and residual ascites persisted. Further etiological assessment, including liver biopsy

when feasible, remained important to confirm the underlying diagnosis and investigate the persistent cholestatic abnormalities.

Table 2 summarizes the clinical and biochemical course; Figure 1 shows changes in total bilirubin activity, and Figure 2 shows changes in total prothrombin activity.

Time point	Clinical findings	Test	Patient value	Reference/target value
Admission (hospital day 1)	Conscious with asterixis; moderate ascites; jaundice; spontaneous bacterial peritonitis	Total bilirubin	>25 mg/dL	0.3-1.2 mg/dL
		ALT	355 U/L	7-35 U/L
		AST	281 U/L	8-35 U/L
		Albumin	25 g/L	35-50 g/L
		Prothrombin activity	34%	70%-100%
		Platelet count	80 × 10^9/L	150-400 × 10^9/L
		IgG	33.7 g/L	7.67-15.90 g/L
After approximately one week of corticosteroids	Confusion, somnolence, and asterixis; West Haven grade III encephalopathy; GCS 13/15	Total bilirubin	21.6 mg/dL	0.3-1.2 mg/dL
		Prothrombin activity	48%	70%-100%
After the fourth TPE session	Neurological improvement during the first TPE course	Total bilirubin	5.3 mg/dL	0.3-1.2 mg/dL
		Prothrombin activity	65%	70%-100%
		INR	1.27	0.8-1.2
After the first TPE course (five sessions)	Conscious; initial neurological improvement	Total bilirubin	4.1 mg/dL	0.3-1.2 mg/dL
		Prothrombin activity	51%	70%-100%
Approximately nine days after the fifth TPE session	Recurrent asterixis and neurological deterioration	Total bilirubin	6.9 mg/dL	0.3-1.2 mg/dL
		Prothrombin activity	60%	70%-100%
		INR	1.32	0.8-1.2
		White blood cell count	5,430/µL	4,000-10,000/µL
		C-reactive protein	18 mg/L	<5 mg/L
After three additional TPE sessions	Conscious after the second TPE course	Total bilirubin	3.6 mg/dL	0.3-1.2 mg/dL
		Prothrombin activity	61%	70%-100%
At tacrolimus initiation	Tacrolimus started at 1 mg twice daily	Total bilirubin	5.1 mg/dL	0.3-1.2 mg/dL
		Prothrombin activity	53%	70%-100%
		Tacrolimus	6.0	

		trough (day 1)	ng/mL	Target ≈6 ng/mL*
After tacrolimus initiation	Twelve-hour predose trough concentrations	Tacrolimus trough (day 3)	6.1 ng/mL	Target ≈6 ng/mL*
		Tacrolimus trough (day 5)	6.3 ng/mL	Target ≈6 ng/mL*
Final follow-up	No recurrent encephalopathy; mild icterus and small-volume ascites	Total bilirubin	2.4 mg/dL	0.3-1.2 mg/dL
		Direct bilirubin	0.715 mg/dL	0-0.2 mg/dL
		ALT	66 U/L	7-35 U/L
		AST	64 U/L	8-35 U/L
		GGT	1,305 U/L	5-36 U/L
		Albumin	25 g/L	35-50 g/L
		Prothrombin activity	99%	70%-100%
		Sodium	136 mmol/L	135-145 mmol/L
		Urea	0.4 g/L	0.15-0.45 g/L
		Serum creatinine	0.6 mg/dL	0.6-1.1 mg/dL
		Tacrolimus trough	6.1 ng/mL	Target ≈6 ng/mL*

TABLE 2: Clinical and biochemical course during treatment and follow-up

*Tacrolimus concentrations represent 12-hour predose trough levels; for autoimmune hepatitis, the therapeutic target concentration is approximately 6 ng/mL rather than a laboratory reference range [1]

ALT: alanine aminotransferase; AST: aspartate aminotransferase; GGT: gamma-glutamyl transferase; GCS: Glasgow Coma Scale; IgG: immunoglobulin G; INR: international normalized ratio; TPE: therapeutic plasma exchange

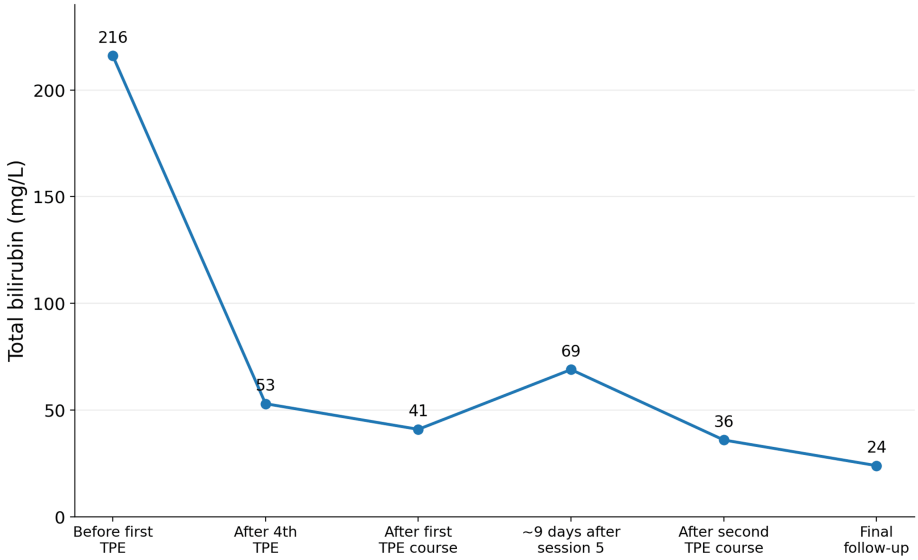


FIGURE 1: Evolution of total bilirubin during treatment and follow-up

Total bilirubin levels at selected clinical checkpoints before and after TPE. Bilirubin decreased from 216 mg/L before the first TPE session to 53 mg/L after the fourth session and subsequently to 41 mg/L after the first TPE course. An increase to 69 mg/L was observed approximately nine days after the fifth session, followed by a decrease to 36 mg/L after the second TPE course and to 24 mg/L at the final follow-up. The admission value (>250 mg/L) is not displayed. Time points are equally spaced for graphical representation and do not represent equal elapsed time

TPE: therapeutic plasma exchange

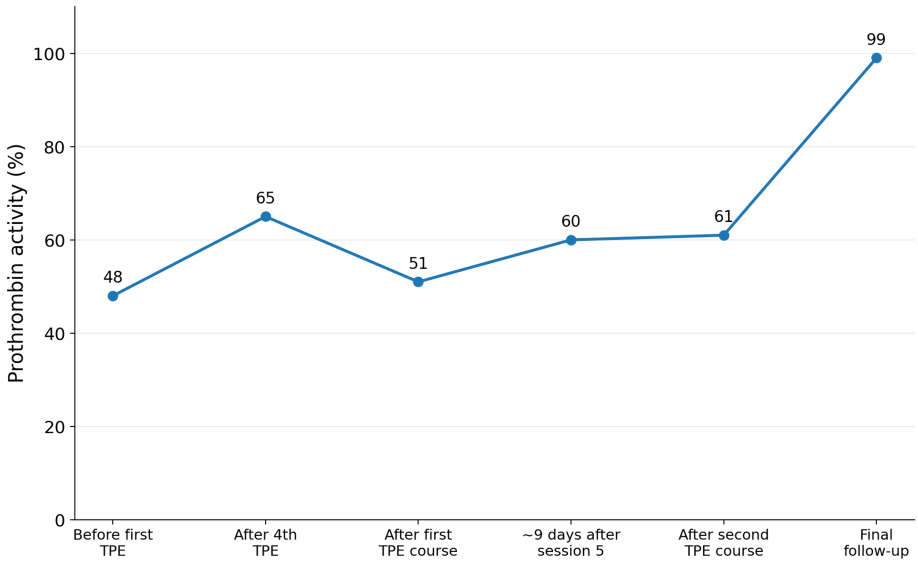


FIGURE 2: Evolution of prothrombin activity during treatment and follow-up

Prothrombin activity at selected clinical checkpoints before and after TPE. Prothrombin activity increased from 48% before the first TPE session to 65% after the fourth session, with subsequent values of 51% after the first TPE course, 60% approximately nine days after the fifth session, and 61% after the second TPE course. At the final follow-up, prothrombin activity reached 99%. Time points are equally spaced for graphical representation and do not represent equal elapsed time

TPE: therapeutic plasma exchange

Persistent cholestasis raised the possibility of an associated cholestatic liver disease, including primary

biliary cholangitis (PBC) and a potential AIH-PBC overlap syndrome. Anti-M2, anti-gp210, and anti-sp100 antibodies were negative. Liver biopsy should be considered when clinically feasible and safe to provide histological confirmation of AIH and evaluate for bile duct lesions suggestive of PBC.

Discussion

This case combines an acute inflammatory presentation compatible with probable AIH and clinical evidence of previously unrecognized cirrhosis. The dysmorphic liver, caudate hypertrophy, splenomegaly, thrombocytopenia, and ascites favor advanced chronic liver disease, but no prior records or histology establish its duration. The score of 6 from the available nonhistological simplified diagnostic items supports AIH; the absence of biopsy and the prominent cholestatic component warrant continued diagnostic reassessment [1,2]. Ascitic infection and autoimmune activity may both have contributed to the acute deterioration.

The subsequent grade III encephalopathy and bilirubin of 21.6 mg/dL indicate cerebral and hepatic organ failure, respectively. On the clinical assumption of cirrhosis, two failures meet the European Association for the Study of the Liver-Chronic Liver Failure (EASL-CLIF) definition of ACLF grade 2 [3]. Admission INR was unavailable, so coagulation failure and a numerical Model for End-Stage Liver Disease score cannot be established. Asterixis while conscious on admission does not itself establish grade III encephalopathy at that time. The transient GCS score of 13/15 and clinical West Haven classification should be interpreted in their bedside context [9,10].

TPE is a supportive intervention rather than a treatment of the underlying autoimmune inflammation. In acute liver failure of mixed etiologies, randomized trials of high-volume and standard-volume TPE have reported improved short-term outcomes [4,5]. These acute liver failure populations differ from patients with cirrhosis and ACLF. In ACLF, an updated meta-analysis and a subsequent randomized trial suggest a short-term survival benefit, while the latter did not show lower 90-day mortality [6,7]. Conversely, an observational cohort in acute liver failure found no overall survival benefit [11]. These findings do not establish the efficacy of a specific exchange protocol for AIH-related ACLF.

AIH-specific evidence remains sparse. A pediatric report described improvement following plasma exchange in three children with autoimmune-related acute liver failure [12], whereas an adult case progressed despite corticosteroids and plasma exchange and ultimately required transplantation [13]. In our patient, neurological and biochemical improvement after five exchanges was followed by a rebound after an approximately nine-day interval; three additional exchanges preceded another bilirubin decline. This temporal association is consistent with short-term stabilization. Fresh frozen plasma replacement can directly influence prothrombin activity and INR, and the concurrent corticosteroid and antibiotic treatment prevents causal attribution to TPE alone.

Tacrolimus was introduced after the second course because transplantation was unavailable locally, and continuing immune-mediated activity remained a concern. Calcineurin inhibitors are considered in selected difficult-to-treat AIH cases under specialist monitoring [1]. The subsequent absence of encephalopathy and improvement in aminotransferases are encouraging, but the bilirubin and GGT remained elevated, mild ascites persisted, and follow-up IgG was not available. Complete biochemical remission and the independent contribution of tacrolimus cannot be established. Persistent cholestasis merits continued evaluation for alternative or additional causes.

The main limitations are the absence of histology and prior liver assessments, missing admission INR and machine-recorded removed PVs, uncertainty around the detailed neurological classification, and incomplete follow-up IgG. This single case cannot establish efficacy or safety of the treatment sequence in other patients. Transplant evaluation remains important when it is feasible [1,3].

Conclusions

Probable AIH was associated with severe decompensation of clinically suspected cirrhosis, complicated by spontaneous bacterial peritonitis and subsequent hepatic and cerebral organ failure consistent with EASL-CLIF ACLF grade 2. TPE was associated with initial neurological and biochemical improvement, but recurrence after the first course highlighted the transient nature of this response. Tacrolimus was subsequently introduced as an alternative immunosuppressive treatment when timely liver transplantation was unavailable.

At the last follow-up, hepatic encephalopathy had not recurred, and aminotransferase levels had improved, although significant cholestasis and mild ascites persisted. This case illustrates the potential role of TPE as temporary supportive therapy while longer term immunosuppressive management is established. However, the individual contributions of TPE, corticosteroids, infection treatment, and tacrolimus cannot be determined from a single observation, and no definitive conclusions regarding treatment efficacy can be drawn.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Mounir El Bied, Mehdi Zouaoui, Naima Belasri

Acquisition, analysis, or interpretation of data: Mounir El Bied, Mbarek Azouaoui, Mehdi Zouaoui, Nourdin Aqodad, Youssef Hnach, Naima Belasri

Drafting of the manuscript: Mounir El Bied, Mehdi Zouaoui, Naima Belasri

Critical review of the manuscript for important intellectual content: Mounir El Bied, Mbarek Azouaoui, Mehdi Zouaoui, Nourdin Aqodad, Youssef Hnach

Supervision: Mehdi Zouaoui, Nourdin Aqodad

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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