

Case and Review

Acute Liver Failure due to Wilson's Disease and Rescue Therapy with Plasma Exchange: A Case Report and Literature Review

Sameera Hajijama^a Mahmoud Marashi^b Samuel B. Ho^{a, b}

^aMohammed Bin Rashid University of Medicine and Health Sciences, Dubai Healthcare City, Dubai, United Arab Emirates; ^bDepartment of Medicine, Mediclinic City Hospital, Dubai Healthcare City, Dubai, United Arab Emirates

Keywords

Wilson's disease · Fulminant hepatic failure · Acute liver failure · Cirrhosis · Plasmapheresis · Plasmas exchange · Liver transplantation

Abstract

Introduction: Wilson's disease (WD) is a rare genetic condition characterized by impaired copper metabolism and can rarely present as acute liver failure with an associated high mortality rate. Treatment often requires liver transplantation, and few rescue treatments have been shown to be effective by randomized trials. This is a case report and scoping literature review on plasmapheresis in acute liver failure due to WD. **Case Presentation:** We report a case of a previously undiagnosed 32-year-old female who presented with acute liver failure due to WD. Initial severity indices (MELD = 31 and RWPI - Revised Wilson Prognostic Index = 14) warranted liver transplant referral; however, no facilities were available. The patient was managed by two sessions of plasmapheresis with plasma exchange with persistent improvement in MELD score over 1 week. Subsequent ongoing chelation therapy resulted in a total reversal of clinical liver disease over 2 years. Literature review resulted in 74 patients reported to have been treated with plasmapheresis for WD and acute liver failure. Of these, 40% survived without transplant. Out of the total patients reported, 54 patients had an available NWI score, with 46 patients having a high-risk score of ≥ 11 (85%) and 8 patients had a score < 11 (15%). Of the 46 patients with a score ≥ 11 , 21 (46%) recovered without liver transplantation, 16 (35%) underwent liver transplantation, 8 (17%) died of liver failure, and 1 (~2%) died of sepsis. Of the 8 patients with a score of < 11 , 4 (50%) recovered without a liver transplantation, and 4 (50%) underwent transplantation. **Conclusion:** The outcomes observed in the literature and the favorable

outcome of our patient suggest an emphasis on the use of urgent plasmapheresis with plasma exchange as an initial intervention for acute liver failure in WD, although further randomized controlled trials are needed for determining the optimal dose and duration of treatment.

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Introduction

Wilson's disease (WD) is rare, with a global prevalence of 1/10,000–1/30,000 according to the WHO [1]. The condition may remain asymptomatic for years, and in the case of liver disease may dramatically present with acute liver failure. Fulminant WD is an uncommon presentation of hyperacute severe hepatic cellular injury and hemolysis due to copper accumulation. Management of this condition is challenging and often necessitates liver transplantation. We report a case of acute liver failure from WD that occurred during the COVID-19 pandemic with no recourse to liver transplantation. Reversal of liver failure was accomplished by early diagnosis, plasmapheresis, and initiation of chelation therapy. Subsequent long-term follow-up indicated reversal of underlying cirrhosis and signs of portal hypertension. The purpose of this report was to emphasize the potential efficacy of plasmapheresis for acute liver failure and the reversibility of severe liver disease with long-term chelation therapy.

Case Report – Fulminant WD

A previously healthy 32-year-old female presented to the emergency department with an episode of jaundice, nausea, vomiting, and unrecorded fever. She had a previous history of “stomach problems” for the 5–6 days associated with bilious vomiting, scleral icterus, nausea, and low-grade fever. She was fatigued and somnolent, and passed black stool once 2 days ago, but passed brown stool on the day of presentation, with no change in color or consistency, and no blood in the stool.

The patient was seen in a clinic the previous day, in which laboratories revealed elevated transaminases in which her total bilirubin was 177, AST 128, ALT 31, ALP 25, and her INR was 1.91. An ultrasound done at the clinic showed an enlarged liver and gallbladder with edema and sludge.

She denied any history of previous liver disease, recent travel, heavy use of alcohol, intravenous drug use, new medication, or herbal medication use. She took an appropriate dose of acetaminophen over the past 4 days for her symptoms. In review of systems, she did not report any neurological, psychiatric, cardiovascular, respiratory, dermatological, musculoskeletal, or genitourinary symptoms.

The patient's past medical history includes an uncomplicated normal vaginal delivery 7 months previously. Otherwise, there was no significant past medical or surgical history. There is no significant family history of gastrointestinal or hematological diseases. The patient denied any history of smoking or alcohol consumption.

On admission, the patient was hemodynamically stable. Her physical examination revealed significant tenderness of the right upper quadrant. She had an enlarged liver span of 14 cm, 4 cm below the right costal margin. The abdomen was distended with flank dullness. The NWI score and MELD score both indicated high-risk for fatality and were above the cut-offs for mortality without liver transplant, whereas the King's College score was below the cut-off for transplantation indication based on her initial laboratories [2].

Laboratory studies revealed a severely decreased hemoglobin, an elevated WBC, INR, AST, total bilirubin and direct bilirubin, and normal remaining liver tests (Table 1; Fig. 1). Alkaline phosphatase levels were low (<24 U/L). Urinalysis revealed mild protein and blood in the urine.

Tests for acute viral hepatitis were negative with negative hepatitis B surface antigen, hepatitis B core antibody, hepatitis C antibody, and hepatitis A IgM antibody. An MRCP was done, revealing mild to moderate hepatomegaly, with a distended gallbladder with edema, no obvious stones, and no dilation of the common bile duct or intrahepatic bile ducts. The patient did not have ascites upon admission but later developed it on day 5 of admission with associated edema. The upper endoscopy was normal, ruling out mucosal bleeding lesions and varices. The hematological manifestations of this case were ascertained due to hemolysis with a negative Direct Coombs test. Blood smear revealed spherocytes, echinocytes, and polychromasia.

To ascertain the etiology of her acute liver failure, there was a thorough investigation to rule out infectious, autoimmune, or idiopathic causes. As determined, the patient took appropriate doses of acetaminophen, denied illicit drug use, and viral markers were negative (hepatitis B surface antigen, hepatitis C antibody, hepatitis A antibody, and hepatitis E antibody). The Doppler US was normal and a triphasic CT scan revealed no indication of cholecystitis or thrombosis. Autoimmune markers were all negative (antinuclear [ANA] profile, antineutrophil cytoplasmic antibodies [ANCA], and anti-smooth muscle antibodies [ASMA]).

Due to the low alkaline phosphatase level on admission, WD was suspected. A slit-lamp examination was done on day 2 of admission, revealing Kayser-Fleisher rings. Subsequently, her serum ceruloplasmin levels were then measured and revealed to be mildly low (Table 2). Her serum copper levels were measured on the second day of admission, and 24-h urinary copper measurement was done on day 3 of admission (Table 2).

Due to the severity of liver failure and elevated MELD, WPI, and RWPI scores on admission (Table 1), urgent transfer to a liver transplant center was sought. No liver transplants were being performed in the UAE at this time due to COVID-19 related travel restrictions. The patient could not be sent to her home country of Ukraine because of the unavailability of liver transplants there. Transport to other countries was blocked due to travel restrictions, cost, and family refusal.

In terms of management, transfusions were initially given due to the severely low hemoglobin levels. Folate supplementation was given to improve bone marrow response. Vitamin K was also administered to aid in treatment. On the first day of admission, a 24-h infusion of N-acetyl cysteine was initiated. Because of the severity of the liver failure plasmapheresis was initiated in the intensive care unit on day 3 after admission, with 3 L of plasmapheresis containing 1-unit packed RBCs and 2 units fresh frozen plasma, followed by a replacement by 2.25 L saline + 750 mL of 20% albumin. The patient completed a second plasmapheresis session on day 5 of admission. Her coagulation profile was monitored, and she received heparin and hydrocortisone to prevent complications from the procedure.

Clinically, the patient's jaundice slowly improved over the initial 5 days with marked improvement following plasmapheresis that was sustained on subsequent days (Table 1). Her liver span decreased from 14 cm upon admission to 9 cm on day 6 of admission. Ascites was treated with Spironolactone and Furosemide. Her laboratories improved significantly after treatment (see Table 1); however, her phosphate levels were low, and her platelets continued to drop from 150,000 to 74,000/ μ L. The patient continued Penicillamine and improved clinically and through laboratory results. Her total bilirubin had dropped from 231 to 35.4 after the interventions. Her INR ranged from 2.1 to 3.2 over the course of the admission, then improved to 1.8–2.3 after interventions were done.

As for the patient's renal function, her initial elevated blood urea nitrogen and creatinine with mild proteinuria and hematuria on urinalysis raised suspicion for acute kidney injury (see Table 1); however, her renal function tests quickly improved following plasma exchange. After the plasmapheresis, she developed hypophosphatemia, likely due to Fanconi syndrome with

Table 1. Admission laboratory results of the reported case

	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	Day 10	Day 11
Hg, 13.5–17.5 g/dL	3.9	7.1	5.1	6.1	5.1	6.5	6.5	8.3	8.6
WBC, 4.5–11.0 × 10 ⁹ /L	18.4	16.1	15.9	15.3	10.2	9.7	6.6	3.9	4.7
Platelet, 150–400 × 10 ⁹ /L	198	173	159	164	150	125	74	57	63
Total bilirubin, 3.4–20.50 U/L	270	153	185	231	126.9	55	41	43	35.4
ALP, 1–60 U/L	<24	<24	<24	N/A	29	29	56	95	111
AST, 6–34 U/L	144	121	103	116	96	47	47	75	64
ALT, 0–55 U/L	22	23	16	N/A	39	28	32	62	62
Albumin, 35–50 g/L	27.5	23.5	20.4	20.0	19.7	34.8	29.0	37.6	38.5
INR, ≤1.1	3.0	2.6	3.2	2.2	2.1	2.7	2.6	2.0	1.8
BUN, 5–20 mg/dL	31.4	N/A	N/A	11.0	N/A	15.3	14.4	N/A	N/A
Cr, 70–133 µmol/L	10.0	99.0	90.40	65.70	64.60	58	60.90	62.10	67.10
Na, 137–142 mEq/L	136	136	134	136	134	139	140	137	138
MELD	31	26	30	25	24	22	20	18	16
WPI score	8	7	7	7	4	4	4	3	2
RWPI score	14	14	15	14	10	7	6	4	3
Treatment	2 PRBC	Folic acid	1 PRBC Vitamin K	1 PRBC	1 PRBC 2 FFP Zinc Phosphorus		Folic acid Pyridoxine Spironolactone Furosemide		Zinc Vitamin D
NAC infusion	24-h infusion started	Infusion completed							
Plasmapheresis			1st round started	1st round completed	2nd round started	2nd round completed			
D-penicillamine				Started					

Hb, hemoglobin; WBC, white blood cells; ALP, alkaline phosphatase; AST, aspartate aminotransferase; ALT, alanine aminotransferase; BUN, blood urea nitrogen; Na, sodium; MELD, model for end-stage liver disease; WPI, Wilson Prognostic Index; RWPI, Revised Wilson Prognostic Index; NAC, N-acetyl cysteine; PRBC, packed red blood cells; FFP, fresh frozen plasma. A WPI score ≥7 or an RWPI ≥11 = increased risk of mortality without liver transplant.

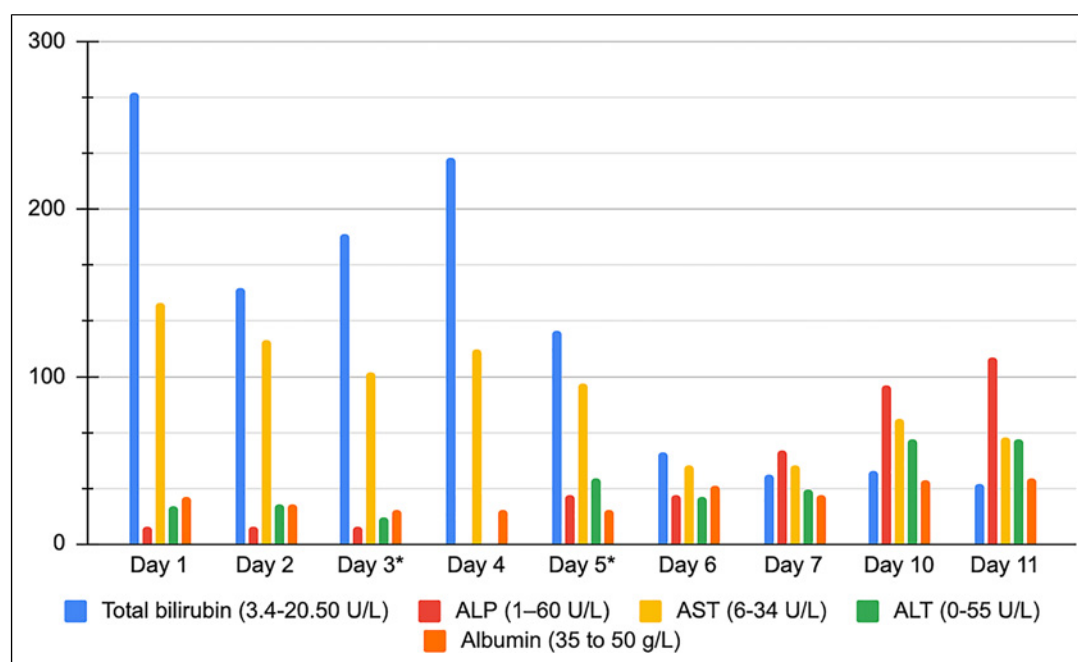


Fig. 1. Liver function test results of the reported case. The trend of liver function tests during the admission for acute liver failure due to WD. The levels were noted to improve after two sessions of plasmapheresis were done on admission days 3 and 5, respectively. *Plasmapheresis sessions performed on day 3 and day 5.

metabolic acidosis, but the levels quickly improved after phosphate supplementation was given. During the rest of her admission, her renal function was normal with adequate urine output.

An ultrasound done on her third day of admission showed nephromegaly, which remains unexplained. The possibility of infiltrative diseases such as amyloidosis was considered. Due to her clinical improvement after plasmapheresis and normal renal function tests, a biopsy of the abdominal fat pad or salivary gland was not indicated.

Her MELD score decreased from 31 on admission, to 18 after plasmapheresis treatment, further improving to 16 on the day of discharge. Her serum copper levels reduced from 138 to 67, and her urinary copper levels reduced from 12,943 to 1,050 on the day of discharge.

The patient was discharged and continued to take D-penicillamine, pyridoxine, urso-deoxycholic acid, folic acid, and vitamin D. She was followed regularly in the outpatient and remained well apart from mild, intermittent, right sided abdominal pain. Her liver span continued to decrease on physical examinations.

At 6 months of follow-up, the urinary copper decreased to 910 with resolution of anemia but persistent thrombocytopenia. The MELD score improved to 9 and an abdominal ultrasound was normal. Genetic testing subsequently revealed a pathologic mutation in the H1069Q rehz ATP7B gene. The patient was regularly followed and continued therapy with oral D-penicillamine over the last 3 years. This resulted in complete normalization of all laboratory tests and MELD score (Table 3).

Discussion: Fulminant WD – Evaluation and Prognosis

As copper accumulates systemically in WD, the manifestations may vary between asymptomatic and severely symptomatic. Copper accumulation into hepatocytes leads initially to mild asymptomatic elevation of transaminases, and then over time can evolve to cirrhosis and

Table 2. Additional pertinent laboratory results

LDH, 140–280 U/L	Reticulocyte count, 0.5%–2.5%	Absolute reticulocyte count, 50–100 × 10 ⁹ /L	Fibrinogen, 200–400 mg/dL	Ceruloplasmin, 0.20–0.60 g/L	Serum copper, 70–140 µg/dL	24-h urinary copper, 20–50 µg per 24 h	C3, 0.83–1.93	C4, 0.15–0.57	PT, 11–13.5 s	PTT, 25–35 s
776	12.9%	121.2	251	0.19	138	12,943	0.52	0.10	13.5	33.2
LDH, lactate dehydrogenase; C3, complement C3; C4, complement C4; PT, prothrombin time; PTT, partial thromboplastin time.										

Table 3. Long-term outcomes

Hb, 13.5–17.5 g/dL	WBC, 4.5–11.0 × 10 ⁹ /L	Platelets, 150–400 × 10 ⁹ /L	Total bilirubin, 3.4–20.50 U/L	INR, ≤1.1	Serum albumin, 35–50 g/L	Serum copper; 70–140 µg/dL	Urinary copper; 20–50 µg per 24 h	MELD score	WPI score	RWPI score	
At discharge	8.6	4.7	63	35.40	1.8	38.50	67	1,050	16	2	3
2 years after discharge	13.4	5.5	111	6.10	1.26	48.0	26	401.7	9	0	0
3 years after discharge	13.1	6.6	144	5.30	1.10	44.8	31	125.1 µg/L or 136.0 µg/g	7	0	1
Hb, hemoglobin; WBC, white blood cell count; MELD, model for end-stage liver disease; WPI, Wilson prognostic index; RWPI, revised Wilson prognostic index. A WPI score ≥7 or an RWPI ≥11 = increased risk of mortality without liver transplant.											

acute liver failure. Elevated transaminases need to be differentiated from viral hepatitis, NAFLD, or autoimmune hepatitis. A recent study revealed that of all patients with WD initially presenting with liver disease, 39% of patients will have cirrhosis [3]. WD accounts for around 3–5% of patients presenting to the hospital with acute liver failure [4]. Signs include jaundice, anorexia, encephalopathy, and compensated or decompensated cirrhosis with evidence of ascites. Other extrahepatic features to note include coagulopathy, Coombs's negative intravascular hemolytic anemia, Kayser-Fleisher rings, and progression to acute renal failure [5]. The clinical basis for liver transplantation includes acute hepatic failure and decompensated cirrhosis unresponsive to medical management. Extrahepatic (neuropsychiatric) manifestations are not considered a primary indication for transplant [6]. Our patient had signs primarily of acute liver failure, thus a liver transplantation as a definitive treatment was immediately considered.

Important prognosis indicators include the Wilson's Disease Prognostic Index (WPI) by Nazar et al. [7], the Revised Wilson's Prognostic Index (RWPI) also known as the New Wilson Index (NWI) by Dhawan et al. [8], and the MELD score (or PELD for pediatrics). The cut-off limits are 7, 11, and 30, respectively, for predicting fatality without a liver transplantation. In a large series of pediatric patients with liver failure due to WD, the accuracy of the MELD score, WPI, and RWPI for prediction of response to medical chelation therapy in patients with decompensated chronic WD was 0.968, 0.980, and 0.993, respectively. In this series, RWPI was proven to be the most reliable predictor of response to medical therapy with a sensitivity of 93% and a specificity of 98% (Dhawan [8]). Its use was extended for adult patients with chronic decompensated WD [9]. Note that our patient did not exceed the cut-off for high fatality risk according to the traditional Kings College Criteria for non-acetaminophen related liver failure [10]. This phenomenon has been seen in literature. In a case series of 20 patients with WD and ALF that were transplanted, only 13 fit the traditional Kings College Criteria, whereas 18 fit Naser WPI criteria, and 18 fit RWPI criteria for need for liver transplant [2]. Hence, 5 patients who did not fit the traditional Kings College Criteria still required a transplant as per the Naser WPI and Dhawan NWI criteria.

Interestingly, our patient did not manifest or have a history of neurological manifestations of WD. Neurologic symptoms include dysarthria, dysphagia, drooling, ataxia, and Parkinson-like movement disorders due to basal ganglia involvement. Finally, other systemic manifestations include hypothyroidism, amenorrhea, cardiomyopathy, osteomalacia, renal tubular dysfunction, and electrolyte abnormalities associated with Fanconi syndrome [5].

Literature Review: Fulminant WD – Treatment Options and Plasmapheresis

To determine the reported efficacy of plasmapheresis as a potential life-saving treatment for patients presenting with acute liver failure due to WD, we searched the literature using the criteria of acute liver failure, WD, plasmapheresis, and human studies. Our initial search revealed 311 articles, and after excluding duplicates, non-English articles, and articles without sufficient information, we found and reviewed 24 articles from the years 1989 to 2023.

A total of 74 patients were described in the literature with reported therapy with plasmapheresis, with an age range of 5–57 (Table 4). Most were reported in single case reports or small case series, there were no randomized controlled trials. Multiple plasmapheresis regimens were used, ranging from 2 sessions to 12 or more sessions per patient. Out of the total patients reviewed, 30 (40%) fully recovered without transplant, 32 (43%) underwent liver transplantation, and 12 (16%) died. Of the 54 patients with an available RWPI/NWI score, 46 patients had a score of ≥ 11 (85%), whereas 8 patients had a score < 11 (15%). Of the 46 patients with a score ≥ 11 , 21 (46%) recovered without liver transplantation, 16 (35%) underwent liver transplantation, 8 (17%) died of liver failure, and 1 (~2%) died of

Table 4. Literature review of plasmapheresis therapy for fulminant WD

First author [reference]	Publication year	Patients, <i>n</i>	Age, years	NWI score/ RWPI score (Dhawan [8])	WPI score (Naser [7])	Outcome (recovery/ transplant/death)	Sessions of plasma exchange	Hepatic encephalopathy	CRRT
Glover [11]	2023	1	17	N/A	7	Transplant	4 over 4 days	No	
Pawaria [12]	2021	19	<18	>11	N/A	9 recovered, 4 transplant, and 6 died	13 cases (≥3 sessions), 4 cases (2 sessions), and 2 cases (1 session)	Yes (4 cases)	
Zou [13]	2021	1	19	8 ^a	7 ^a	Transplant	N/A	No	
Proost [14]	2020	1	14	14	N/A	Recovery	5 sessions over 5 days	No	
Camarata [15]	2020	8	19, 19, 49, 57, 21, 18, 25, 20	9, 9, 13, 12, 15, 15, 17, 15	N/A	3 recovery, 5 transplant	2 sessions	No	
Damsgaard [16]	2019	1	27	16	N/A	Recovery	12 sessions	Yes	
Zhang [17]	2019	1	7	12	N/A	Transplant	2 sessions	Yes	
Kido [18]	2018	5	11, 10, 13, 6, 15	12, 9, 15, 12, 6, 15	N/A	3 Recovered, 2 Transplant	N/A	Yes (2 cases)	
Kido [19]	2017	4	6, 15, 13, 10	11, 12, 15, 8	N/A	2 Recovered, 2 Transplant	N/A	Yes (2 cases)	
Tian [20]	2016	9	7, 8, 13, 16, 17, 22, 11, 15, 21	13, 15, 12, 11, 14, 10, 16, 15, 15	N/A	6 Recovered, 2 died from Liver Failure, 1 died from <i>E. coli</i> septicemia	N/A	Yes (4 cases)	
Pham [21]	2016	10	6–30	N/A	N/A	1 Recovery, 9 Transplant	43 Sessions over 13 days	No	Yes (2 cases)
Motobayashi [22]	2014	1	10	16	N/A	Recovery	11 sessions over 16 days	Yes	

(Continued on following page)

Table 4 (continued)

First author [reference]	Publication year	Patients, n	Age, years	NWI score/ RWPI score (Dhawan [8])	WPI score (Naser [7])	Outcome (recovery/ transplant/death)	Sessions of plasma exchange	Hepatic encephalopathy	CRRT
Hilal [23]	2014	1	19	9 ^a	9 ^a	Transplant	3 sessions	Yes	Yes
Verma [24]	2014	1	5	N/A	N/A	Died	7 sessions over 10 days	Yes	
Morgan [25]	2012	1	6	N/A	N/A	Transplant	5 sessions over 11 days	No	
Akyildiz [26]	2011	1	9	16	N/A	Recovery	5 sessions	Yes	
Hursitoglu [27]	2009	1	17	N/A	N/A	Recovery	1 session	No	Yes
Jhang [28]	2007	1	21	N/A	N/A	Transplant	5 sessions over 6 days	Yes	
Asfaha [29]	2007	1	18	9 ^a	4 ^a	Recovery	7 sessions over 7 days	No	
Nagata [30]	2003	1	15	N/A	N/A	Transplant	8 sessions over 8 days	yes	
Lee [31]	1998	1	27	N/A	N/A	Died	2 sessions over 5 days	No	
Kiss [32]	1998	2	19, 16	N/A	N/A	2 Transplant	4 sessions over 4 days, 2 sessions over 2 days	Yes, No	
Enomoto [33]	1989	1	16	N/A	N/A	Died	N/A	No	
Current study	2020	1	32	14	8	Recovery	2 sessions over 4 days	No	

A WPI score ≥ 7 or an RWPI ≥ 11 = increased risk of mortality without liver transplant.CRRT, continuous renal replacement therapy. ^aCalculated as per the laboratory values available on day 1 of Admission.

E. coli septicemia. Of the 8 patients with a score of <11 , 4 (50%) underwent liver transplantation, and 4 (50%) recovered without liver transplantation.

Of the 5 patients with a WPI score available, 4 had a WPI score of ≥ 7 (80%), and 1 had a WPI score <7 (20%). Of those with an WPI score of ≥ 7 , 3 underwent liver transplantation (75%), and 1 recovered (25%). One case with the WPI score <7 resulted in recovery. In addition, 4 cases had continuous renal replacement therapy as part of the management (see Table 4).

Our study overlaps and expands on a recent literature review of previously reported cases with 63 patients from the ages of 5–30 presenting with acute liver failure from WD who were treated with various types of plasma exchange. In this series, of 37 patients with of NWI score ≥ 11 , 17 (46%) recovered without requiring liver transplantation, 12 (33%) received a transplant, and 8 (22%) died [14].

There is increasing evidence of the benefit of plasma exchange for acute liver failure of a variety of causes [34]. A recent randomized controlled trial [35] of 40 patients with non-acetaminophen acute liver failure and randomized 1:1 to either standard medical treatment (SMT) or SMT with standard-volume plasma exchange (SVPE). They found that SVPE was associated with a higher 21-day transplant-free survival (75% vs. 45%; $p = 0.04$, HR 0.30, 95% CI: 0.01–0.88). A significant decrease in levels of pro-inflammatory cytokines and an increase in anti-inflammatory cytokines were seen with SVPE [35].

Conclusion

WD is often a hidden disease and difficult to diagnose, as patients may remain asymptomatic for years and then dramatically present with acute liver failure, as seen in this case. Despite the COVID-19 pandemic preventing a liver transplant, the early diagnosis and treatment of this patient with two sessions of plasma exchange led to a marked improvement and avoidance of the need for transplant, and subsequent long-term chelation therapy led to the reversal of cirrhosis and portal hypertension. The outcomes of this case and multiple cases reported in the literature emphasize the potential benefit of plasmapheresis with plasma exchange for reversing acute decompensated liver failure in WD; however, the optimal dose and duration of therapy are yet to be established. Further randomized trials of early plasma exchange or plasmapheresis for acute or fulminant liver failure of all etiologies are warranted.

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Statement of Ethics

Written informed consent for the publication was provided by the patient and approved by the Mediclinic Institutional Review Board. This study protocol was reviewed and the need for approval was waived by Mediclinic Middle East (MCME) Clinical Research Ethics Committee in accordance with national guidelines. The CARE Checklist has been completed by the authors for this case report and is attached as online supplementary material (for all online suppl. material, see <https://doi.org/10.1159/000544927>).

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

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Author Contributions

S.H.: wrote the first draft of the manuscript and reviewed the literature. S.B.H.: participated in the clinical assessment of the patient and critically reviewed the manuscript. M.M.: critically reviewed the final manuscript, final approval of the version to be published, and provided expertise regarding the use of plasmapheresis in acute liver failure.

Data Availability Statement

All data generated or analyzed during this study are included in this article. Further inquiries can be directed to the corresponding author.

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