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Application of Double Plasma Adsorption Combined with Plasma Exchange in Patients with Acute and Chronic Liver Failure

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Abstract: To investigate the clinical efficacy of a dual plasma molecular adsorption system combined with plasma exchange in the treatment of acute and chronic liver failure. Clinical data of 60 patients with acute and chronic liver failure were collected from January 2024 to July 2025 for study. The patients were divided into a control group (n=30) and an observation group (randomized using a random number table). The control group received plasma exchange alone, while the observation group received dual plasma adsorption plus plasma exchange. The clinical treatment outcomes, liver function, coagulation function, and inflammatory factor levels were compared between the two groups. The study data showed that after corresponding treatments, the clinical total effective rate in the observation group was significantly superior to that in the control group. In terms of liver function indicators, the levels of aspartate aminotransferase (AST), alanine aminotransferase (ALT), total bilirubin, and direct bilirubin in the observation group were significantly lower than those in the control group. Coagulation function tests revealed that the prothrombin time and activated partial thromboplastin time in the observation group were significantly prolonged compared to the control group. Additionally, in terms of inflammatory markers, the levels of C-reactive protein (CRP) and procalcitonin in the observation group were also lower than those in the control group, with statistically significant differences between the groups ($P < 0.05$). The combined treatment approach can effectively improve liver function and coagulation function, reduce inflammatory factors in the body, and enhance treatment efficacy.

1. Introduction

In the clinical management of acute and chronic liver failure, the effective clearance of accumulated pathogenic substances is a critical factor in improving prognosis. Patients with liver failure not only exhibit elevated levels of protein-bound toxins such as bilirubin and bile acids but also often experience significant accumulation of inflammatory mediators, leading to systemic inflammatory response syndrome and multiple organ dysfunction. Conventional medical treatments

often yield limited efficacy [1]. Plasma exchange (PE), as a traditional artificial liver support method, can non-selectively remove toxins through plasma exchange. However, it remains limited by high plasma demand, susceptibility to allergic reactions, and insufficient clearance efficiency for certain medium-to-large molecules and protein-bound toxins. In recent years, advancements in blood purification technologies have facilitated the application of dual plasma adsorption (DPA) systems. This technology combines plasma separation with specific adsorbent materials, enabling efficient and targeted removal of harmful substances such as bilirubin, inflammatory factors, and immune mediators [2]. Theoretically, the combined use of dual plasma adsorption and plasma exchange can achieve complementary advantages, potentially enhancing toxin clearance specificity and efficiency while rapidly stabilizing the internal environment, thereby providing more comprehensive artificial liver support for liver failure patients [3]. This study aims to investigate the clinical efficacy and safety of DPA combined with PE in treating acute and chronic liver failure, with the goal of optimizing treatment protocols for liver failure.

2. Materials and Methods

From January 2024 to July 2025, clinical data of 60 patients with acute and chronic liver failure were collected for study. The allocation method was random number table, with 30 cases in each group, designated as the observation group and the control group. The control group comprised 18 males and 12 females, aged 38-75 years (mean age: 56.52 ± 4.22 years), with a body mass index (BMI) range of 22.56-26.37 kg/m^2 (mean: 24.46 ± 1.33 kg/m^2). The observation group consisted of 16 males and 14 females, aged 36-74 years (mean age: 56.04 ± 4.62 years), with a BMI range of 22.02-27.11 kg/m^2 (mean: 25.03 ± 1.59 kg/m^2). No significant differences were observed between the groups in baseline characteristics (P> 0.05), and the study was deemed complete.

Inclusion criteria: Patients diagnosed with acute or chronic liver failure; age range of 18 to 75 years, with no gender restriction; total bilirubin (TBil) level ≥171.1 μmol/L or accompanied by severe complications; patients or their families have been informed of the detailed study content, adhering to the principle of voluntariness, and signed the informed consent form. The patients or their immediate family members voluntarily participate in this study under full informed consent and sign the written informed consent form.

Exclusion criteria: Patients with severe primary diseases or organ system failure in other systems; those with malignant tumors or immune system disorders; individuals with significant active bleeding or severe coagulation disorders; and those unable to comply with the treatment regimen and efficacy evaluation.

2.1 Method

Patients in the control group received only plasma exchange therapy: At the initiation of treatment, a vascular access was established for the patient, typically using a dual-lumen hemodialysis catheter for arteriovenous connection. After blood collection, the blood pump was activated with an initial flow rate of 50-100 mL/min. The circuit was connected when the blood approached the end of the venous tube, marking the formal entry into the treatment phase. Systemic heparinization anticoagulation was employed, with the first dose of heparin (0.5-1.0 mg/kg) administered intravenously 10 minutes prior to treatment, followed by continuous infusion at 10-20 mg/h until 30 minutes before discontinuation. During treatment, the blood flow gradually increased to 100-150 mL/min, with plasma exchange lasting approximately 2-3 hours. Throughout this process, the isolated patient plasma was discarded, and an equivalent volume of fresh frozen plasma or albumin solution was supplemented to maintain coagulation function and plasma osmotic pressure. At the end of treatment, the extracorporeal circulation blood was reinfused into the patient

using the saline return method, with protamine used to neutralize heparin if necessary.

The treatment method in the observation group was dual plasma adsorption plus plasma exchange: the operational procedures for plasma exchange were identical to those in the control group. Dual plasma adsorption integrated two technical modalities—plasma bilirubin adsorption and hemoperfusion. The treatment utilized a disposable bilirubin plasma adsorber (model KC B-350, adsorbent volume 330 mL ±5%) and a disposable hemoperfusion device (model KCM-350, adsorbent volume ≥350 mL.), manufactured by Guangzhou Koncen BioScience Co.,Ltd., in conjunction with a blood purification system and a plasma separator. At the initiation of treatment, an intravenous access was established for the patient, typically using a 11Fr × 13.5cm double-lumen hemodialysis catheter. The preflushing process was performed in steps: first, the plasma separator and tubing were flushed with 1000 mL of normal saline at a flow rate of 50 mL/min; subsequently, the bilirubin adsorber and hemoperfusion device were flushed with 2000 mL of heparinized normal saline (heparin concentration 1250-1875 U/500 mL) at the same flow rate; finally, the entire circuit was flushed with 500 mL of heparin-free normal saline to remove heparinized saline. The entire preflushing process took approximately 30-45 minutes, during which the consumables were gently tapped to eliminate air bubbles, and the clarity of the fluid was closely monitored. The treatment duration typically ranged from 2.5 to 3 hours, with a total plasma volume processed of approximately 5000-6000 mL. After treatment, a normal saline backflow method was employed, with the backflow rate controlled at 80-100 mL/min. Throughout the treatment, 20% human albumin (50-100 mL) and fresh frozen plasma (600-800 mL) were administered to maintain plasma colloid osmotic pressure and coagulation function.

The treatment parameters were set as follows: the initial total blood flow rate was 80-100 mL/min, which was gradually adjusted to 120-150 mL/min within 15 minutes based on the patient's blood pressure; the plasma separation rate was controlled at 25-35 mL/min; the serial order of the bilirubin adsorber and perfusion device was plasma separator → bilirubin adsorber → blood perfusion device. The anticoagulation strategy employed a systemic heparinization regimen, with an initial dose calculated at 0.8-1.0 mg/kg body weight, followed by continuous infusion at a rate of 8-10 mg/h, aiming to maintain the activated clotting time (ACT) between 180-220 seconds. Throughout the treatment, dynamic monitoring of arterial pressure, venous pressure, and transmembrane pressure was performed, with control targets set as follows: arterial pressure below -200 mmHg, venous pressure not exceeding 200 mmHg, and transmembrane pressure less than 100 mmHg.

2.2 Observation indicators

The efficacy evaluation criteria are as follows: Marked efficacy refers to the basic resolution of clinical symptoms in patients after treatment, with significant recovery of key laboratory indicators such as liver function; Effective efficacy refers to partial relief of clinical symptoms, with improving trends observed in major laboratory indicators; Ineffective efficacy indicates that neither clinical symptoms nor related laboratory indicators show significant improvement. The total effective rate is calculated using the formula: (Number of marked efficacy cases + Number of effective cases) / Total number of cases × 100%.

Liver function: The levels of aspartate aminotransferase (AST), alanine aminotransferase (ALT), total bilirubin, and direct bilirubin in the patient's serum were measured using a fully automated biochemical analyzer before and after treatment.

Coagulation function: Venous blood samples of 4 mL were collected from all enrolled patients before treatment and at the end of the treatment course. The plasma prothrombin time (PT) and activated partial thromboplastin time (APTT) were measured using the SF-8000 fully automated

coagulation analyzer.

Inflammatory marker testing: Serum samples were collected from patients before and after treatment, and the levels of C-reactive protein (CRP) and procalcitonin (PCT) were measured using corresponding reagent kits.

2.3 Statistical methods

SPSS software was used for data processing. Measurement data were expressed as mean ± standard deviation, and intergroup comparisons were performed using t-tests. Categorical data were presented as case numbers (percentage) [n (%)], and intergroup comparisons were conducted using chi-square tests. A P-value <0.05 was considered statistically significant.

3. Results

3.1 Therapeutic Efficacy

As shown in Table 1, the treatment efficacy rate was higher in the observation group (P<0.05).

Table 1: Differences in Treatment Efficacy among Patients (n,%)

Group	Number of cases	Excellence	Valid	of no avail	Effective percentage
Observation group	30	17(56.67)	12(40.00)	1(3.33)	29(96.67)
Control group	30	10(33.33)	13(43.33)	7(23.33)	23(76.67)
X2 value					5.192
P price					0.022

3.2 Liver Function

Post-treatment data demonstrated that patients in the observation group showed significantly better serum transaminase and bilirubin metabolic indicators compared to the control group, specifically manifested as markedly lower levels of aspartate aminotransferase (AST), alanine aminotransferase (ALT), total bilirubin, and direct bilirubin (P<0.05). (See Table 2.)

Table 2: Changes in Liver Function Parameters between the two Groups

Group	Number of cases	Aspartate Aminotransferase (U/L)		Alanine aminotransferase (U/L)		Total bilirubin (μmol/L)		Direct bilirubin (μmol/L)	
		pretherapy	post-treatment	pretherapy	post-treatment	pretherapy	post-treatment	pretherapy	post-treatment
Observation group	30	184.33±10.25	100.65±8.26	255.69±15.68	143.92±11.67	322.69±20.55	215.25±14.36	225.61±17.69	120.54±10.09
Control group	30	185.24±10.66	120.57±9.80	253.69±16.88	161.55±12.99	324.02±23.66	240.52±16.33	222.98±18.99	136.84±12.91
t price		0.337	8.512	0.475	5.529	0.232	6.364	0.555	5.448
P price		0.737	<0.001	0.636	<0.001	0.817	<0.001	0.581	<0.001

3.3 Coagulation Function

Post-treatment statistical analysis revealed that the prothrombin time (PT) and activated partial thromboplastin time (APTT) of patients in the observation group were significantly prolonged

compared to baseline values, whereas both coagulation parameters in the control group showed reduction. Inter-group comparisons demonstrated that the aforementioned parameters in the observation group were significantly longer than those in the control group (P<0.05). (See Table 3.)

Table 3: Changes in Coagulation Function Parameters between the two Groups

Group	Number of cases	PT(s)		APTT(s)	
		Pretherapy	Post-treatment	Pretherapy	Post-treatment
Observation group	30	20.05±4.29	24.50±4.42	45.77±6.39	50.24±8.26
Control group	30	20.11±4.31	18.21±4.88	45.85±6.61	40.49±9.22
t price		0.054	5.232	0.047	4.314
P price		0.957	<0.001	0.962	<0.001

3.4 Inflammatory Factors

The CRP and PCT levels in the observation group were significantly lower than those in the control group after treatment (P<0.05), as shown in Table 4.

Table 4: Changes in Inflammatory Factor Levels between the Two Groups

Group	Number of cases	CRP (mg/L)		PCT (μg/L)	
		Pretherapy	Post-treatment	Pretherapy	Post-treatment
Observation group	30	45.09±6.21	20.16±4.26	1.89±0.25	0.57±0.12
Control group	30	46.11±6.58	28.22±4.51	1.82±0.34	0.88±0.15
t price		0.617	7.116	0.908	8.839
P price		0.539	<0.001	0.367	<0.001

4. Discussion

Liver failure is a clinically severe syndrome of hepatic disease, characterized by extensive necrosis of hepatocytes, leading to significant impairment or loss of critical liver functions such as synthesis, detoxification, excretion, and biotransformation [4]. Under this pathological state, patients not only accumulate large amounts of endogenous toxins such as bilirubin, bile acids, and blood ammonia, but also develop systemic inflammatory response syndrome (SIRS), with massive release of various inflammatory mediators, forming a vicious cycle that further exacerbates hepatocyte injury and multi-organ dysfunction, resulting in a critical condition with extremely poor prognosis [5]. The artificial liver support system, as an important therapeutic intervention, aims to temporarily replace hepatic function through extracorporeal blood purification, creating opportunities for hepatocyte regeneration or buying time for liver transplantation.

The study results demonstrated superior therapeutic outcomes in the observation group. This difference was closely associated with the synergistic effect of dual plasma adsorption combined with plasma exchange. Although plasma exchange alone can rapidly reduce toxin levels, its clearance lacks selectivity, removing harmful substances while also losing a significant amount of

beneficial substances such as coagulation factors and albumin, and exhibits limited efficiency in clearing certain specific toxins [6]. In contrast, the dual plasma adsorption system employs specific adsorbent materials, enabling more targeted and efficient adsorption of medium-to-large molecules and protein-bound toxins such as bilirubin and inflammatory factors. The combination of these two technologies not only leverages the rapid and broad-spectrum clearance advantages of plasma exchange but also enhances the deep clearance capacity of key pathogenic substances through subsequent adsorption therapy, thereby more effectively blocking the sustained damage to hepatocytes caused by toxins and inflammatory responses. This is the fundamental reason for the more significant improvement in liver function indicators among patients in the observation group [7].

In terms of biochemical indicators, the key liver function parameters in the observation group showed significantly better outcomes after treatment compared to the control group. The substantial decrease in alanine aminotransferase (ALT) activity indicated that combination therapy could more effectively mitigate the degree of hepatocyte injury. The marked reduction in total bilirubin and direct bilirubin concentrations fully demonstrated the potent specific clearance capacity of dual plasma adsorption for bilirubin, effectively alleviating the toxic effects of hyperbilirubinemia on the body [8]. These results collectively suggest that combination therapy exhibits significant advantages in promoting hepatocyte functional repair and improving bile metabolism. Regarding coagulation function, the observation group exhibited a different trend compared to the control group. Prolonged coagulation parameters typically reflect coagulation dysfunction, but in this therapeutic context, they hold positive clinical significance. Patients with liver failure often experience coagulation factor deficiency due to impaired hepatic synthesis, manifested as shortened clotting time, which is actually a marker of coagulation failure rather than a normal state [9]. The prolonged clotting time post-treatment may indicate that combination therapy, after effectively clearing toxins and inflammatory mediators, alleviates the inhibition of hepatic synthesis function, allowing the body to restart the physiological synthesis and regulation of coagulation factors, reflecting a potential trend of gradual recovery in hepatic synthesis function [10]. The enhanced purifying effect of combination therapy may temporarily disrupt the homeostasis of the coagulation system, but its long-term effects may be more conducive to fundamental recovery of liver function.

Regarding inflammatory markers, the observation group exhibited significantly lower levels of all inflammatory biomarkers post-treatment compared to the control group. Patients with acute or chronic liver failure commonly present with systemic inflammatory response syndrome (SIRS), where excessive release of inflammatory mediators such as C-reactive protein (CRP) and procalcitonin not only directly damages hepatocytes but also leads to multi-organ dysfunction [11]. The adsorbent used in the combined therapy demonstrates broad-spectrum adsorption properties, effectively eliminating these inflammatory mediators and thereby significantly reducing the systemic inflammatory load. Effective control of inflammatory responses not only creates a more favorable microenvironment for hepatocyte regeneration but also contributes to lowering complication rates, which represents one of the key mechanisms underlying the superior clinical efficacy observed in the observation group [12].

In conclusion, dual plasma adsorption combined with plasma exchange demonstrates promising clinical applications in the treatment of acute and chronic liver failure. Its advantages may stem from the synergistic effects of the two technical mechanisms: plasma exchange achieves rapid initial clearance, creating conditions for subsequent adsorption therapy; while dual plasma adsorption provides efficient and specific deep clearance. These two approaches complement each other, collectively promoting hepatic function recovery and internal environment stabilization.

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