Am J Transl Res 2025;17(11):8612-8622
www.ajtr.org /ISSN:1943-8141/AJTR0168640

Original Article

Treatment outcomes and determinants of combined hemodialysis-hemoperfusion therapy in chronic glomerulonephritis

Xiaotong Yuan[1,2], Caixiang Zhang[1,2]

[1]Department of Nephrology, Shanxi Provincial People's Hospital, Taiyuan 030012, Shanxi, China; [2]Department of Nephrology, The Fifth Clinical Medical College of Shanxi Medical University, Taiyuan 030012, Shanxi, China

Received September 7, 2025; Accepted October 31, 2025; Epub November 15, 2025; Published November 30, 2025

Abstract: Objective: To evaluate the effects of combined hemodialysis (HD) and hemoperfusion (HP) on serum parathyroid hormone (PTH), phosphorus (P), and calcium (Ca) levels in patients with chronic glomerulonephritis (CGN). Methods: A retrospective analysis was conducted on 118 CGN patients treated between May 2022 and May 2024. Based on therapeutic regimen, participants were divided into the HP group (n=52) and HD+HP group (n=66). Clinical efficacy, bone metabolism indices (PTH, P, Ca), renal function parameters (24 h urinary protein (24 h Upro), blood urea nitrogen [BUN], serum creatinine [SCr], estimated glomerular filtration rate [eGFR]), inflammatory markers (IL-17, TNF-α, hs-CRP), hemorheology, coagulation/fibrinolysis indices (tissue-type plasminogen activator [t-PA]), serum uremic toxins, adverse events, and 36-Item Short Form Health Survey (SF-36) quality-of-life scores were compared. Logistic regression was applied to identify factors associated with poor therapeutic response. Results: The HD+HP group showed a higher total effective rate than the HP group (89.39% vs. 73.08%, P=0.021). Compared with HP alone, HD+HP significantly reduced PTH, P, 24 h Upro, BUN, SCr, inflammatory markers, and uremic toxins, while increasing Ca, eGFR, t-PA, and SF-36 scores (all P<0.05). The incidence of adverse events was comparable between groups (30.30% vs. 26.92%, P=0.687). Multivariate analysis identified disease duration >5 years (OR=4.930, 95% CI: 1.339-18.147, P=0.016) and HP monotherapy (OR=3.069, 95% CI: 1.103-8.539, P=0.032) as independent predictors of poor efficacy. Conclusion: Combined HD and HP therapy yields superior therapeutic and biochemical outcomes compared with HP alone in CGN patients.

Keywords: Chronic glomerulonephritis, hemodialysis, hemoperfusion, parathyroid hormone, phosphorus, calcium

Introduction

Chronic glomerulonephritis (CGN) is a glomerular disease primarily characterized by proteinuria, hematuria, hypertension, and edema, often progressing to end-stage renal disease (ESRD) [1]. It represents a major cause of chronic kidney disease (CKD), with nearly seven million global cases and a 10.81% increase in prevalence reported in 2021 [2]. CGN progresses insidiously, presenting with hematuria, proteinuria, hypertension, edema, nocturia, fatigue, and anemia; in advanced stages, oliguria, nausea, muscle cramps, and metabolic acidosis may develop, severely compromising patients' health and quality of life [3, 4]. Pathological subtypes include membranous nephropathy, mesangial proliferative glomerulonephritis, and IgA nephropathy, arising from the interaction of immune, genetic, and environmental factors [5].

Chronic inflammation is central to CGN pathogenesis, with elevated interleukin-17 (IL-17), tumor necrosis factor-α (TNF-α), and high-sensitivity C-reactive protein (hs-CRP) playing key roles [6-8]. Progressive renal impairment leads to mineral metabolism disorders such as hyperphosphatemia and hypocalcemia, accompanied by decreased 24-hour urinary protein (24 h Upro), blood urea nitrogen (BUN), serum creatinine (SCr), and estimated glomerular filtration rate (eGFR) [9]. Parathyroid hormone (PTH) contributes to mineral dysregulation and bone abnormalities by promoting calcium release and phosphate excretion [10, 11]. CGN also

https://doi.org/10.62347/DLGA7398

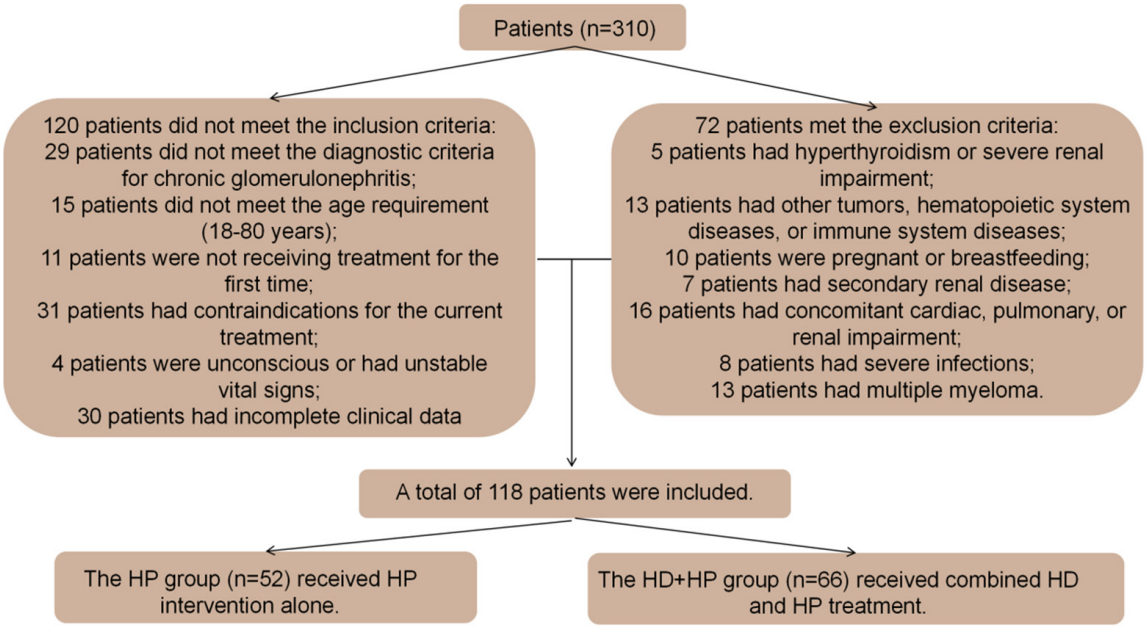

Figure 1. Flowchart of patient selection. HD, hemodialysis; HP, hemoperfusion.

involves increased blood viscosity and coagulation-fibrinolytic imbalance, reflected by elevated plasminogen activator inhibitor-1 (PAI-1) and reduced tissue-type plasminogen activator (t-PA) [12, 13].

A range of therapeutic modalities is available for CGN, including symptomatic treatment, immunosuppression, and renal replacement therapy such as hemodialysis (HD) and hemoperfusion (HP) [14-17]. However, these treatments are often limited by steroid resistance, significant drug toxicities (e.g., hypotension, circuit clotting, thrombocytopenia), low bioavailability, and inadequate targeting precision [14]. Notably, few studies have explored the impact of treatment regimens on patients' serum parathyroid hormone (PTH), phosphorus (P), and calcium (Ca) levels [15]. Specifically, HD eliminates toxins, solutes, and excess fluid via a synthetic filter to restore homeostasis, while HP achieves blood purification through adsorption by binding target molecules to adsorbent materials [16, 17]. To address these research gaps, this study focuses on investigating how the HD-HP combination therapy influences serum PTH, P, and Ca concentrations in CGN patients, aiming to provide data-driven insights for optimizing clinical management of the disease.

Materials and methods

Patient selection

Ethical approval for this retrospective study was obtained from the Ethics Committee of Shanxi Provincial People's Hospital. All patient data were anonymized before analysis. A total of 118 CGN patients admitted between May 2022 and May 2024 were enrolled. Based on treatment modality, patients were divided into the HP group (n=52) and the HD+HP group (n=66). Baseline demographic and clinical characteristics did not differ significantly between groups (P>0.05), confirming comparability (**Figure 1**).

Inclusion criteria: (1) Confirmed CGN diagnosis [18]; (2) Age 18-80 years; (3) No prior treatment; (4) Absence of contraindications; (5) Hemodynamic stability and intact neurological function; (6) Complete medical records.

Exclusion criteria: (1) Hyperthyroidism or advanced renal dysfunction; (2) Malignancy; (3) Hematologic or autoimmune disease; (4) Pregnancy or lactation; (5) Secondary nephropathies; (6) Severe cardiopulmonary or renal impairment; (7) Active severe infection; (8) Multiple myeloma.

Therapeutic protocols

All patients received a standardized pharmacotherapy regimen including oral Vitamin D_2 Calcium Hydrogen Phosphate Tablets (0.15 g, twice daily; Renqi Pharmaceutical, Jiangxi, China, H36022014) and Lanthanum Carbonate Chewable Tablets (0.5 g, once daily; Mingrui Pharmaceutical, Hunan, China, H20203039).

Sample size was calculated for comparison of two independent proportions, assuming 60% and 85% response rates for the HP and HD+HP groups, respectively (α=0.05, 1-β=0.8, δ=25%), yielding 49 cases per group. The final cohort (52 HP, 66 HD+HP) satisfied this criterion.

The HP group underwent 2-hour HP sessions using bicarbonate dialysate at 500 mL/min. The HD+HP group additionally received HD with sodium bicarbonate dialysate (Na^+ 140 mmol/L, Cl^- 140 mmol/L, K^+ 2.0 mmol/L, Ca^{2+} 1.5 mmol/L), with dialysate and blood flow rates of 500 mL/min and 200-250 mL/min, respectively. Each 4-hour session was conducted 1-2 times weekly for six 14-day cycles.

Treatment allocation was not randomized but based on each patient's clinical condition, financial status, and personal preference after full discussion with the patient and family. Written informed consent was obtained from all participants.

Data extraction

Data were retrieved from hospital electronic records. Baseline variables included sex, age, disease duration, diagnostic type, and family history. Clinical outcomes, biochemical parameters, inflammatory markers, hemorheology, coagulation/fibrinolysis indicators, adverse events, and quality-of-life scores were analyzed.

Outcome measures

Therapeutic effectiveness [19]: Outcomes were classified after 3 months as clinical control, marked effectiveness, effectiveness, or ineffectiveness. Total efficacy combined the first three categories.

Bone metabolism: Serum PTH, P, and Ca were measured pre- and post-treatment (3 months) using an automated biochemical analyzer (Tianlong, Xi'an, China, ZY-400).

Renal function: 24 h Upro, BUN, SCr, and eGFR (Cockcroft-Gault) were assessed before and 3 months after treatment.

Inflammatory biomarkers: IL-17, TNF-α, and hs-CRP were quantified via ELISA (Abbkine, Wuhan, KTE6022, KTE6032; Yipu, Wuhan, CSB-E08617h).

Hemorheology: Whole blood viscosity (WBV) under high- and low-shear conditions was measured (Jumu, Shanghai, b3513).

Coagulation/fibrinolysis: Serum PAI-1 and t-PA were determined via ELISA (Baiyixin, Wuhan, TD711259, TD711260).

Uremic toxins: Serum β_2-microglobulin (β_2-MG), homocysteine (Hcy), and advanced glycation end-products (AGEs) were analyzed by ELISA (Yipu, Wuhan, E-EL-H2188, CSB-E08895h, CSB-E09412h).

Adverse events: Hypotension, circuit clotting, thrombocytopenia, puncture-site oozing, and pruritus were recorded.

Quality of life: 36-Item Short Form Health Survey (SF-36) [20] (Physical Functioning, Bodily Pain, Social Functioning, Role-Emotional, Mental Health) were assessed pre- and post-treatment (0-100 scale).

Primary endpoints included treatment efficacy, bone metabolism, renal function, and adverse events; secondary endpoints encompassed inflammatory, hemorheological, and coagulation markers, uremic toxins, and quality-of-life outcomes.

Statistical analysis

Statistical analysis was performed using SPSS v20.0. Categorical data were expressed as n (%) and compared using χ^2 tests. Continuous data were expressed as mean ± standard error of the mean (SEM) and were analyzed using independent-samples and paired t-tests where appropriate. Variables significant at $P<0.05$ in univariate analysis were entered into stepwise multivariate logistic regression to identify inde-

Table 1. Comparison of baseline characteristics

Indicators	HP group (n=52)	HD+HP group (n=66)	χ^2/t	P
Sex			0.008	0.930
Male	28 (53.85)	35 (53.03)		
Female	24 (46.15)	31 (46.97)		
Age (years)	45.63±7.01	44.39±6.82	0.969	0.335
Illness duration (years)	5.60±2.35	5.32±2.11	0.681	0.498
Diagnostic category			0.833	0.659
Membranous nephropathy	28 (53.85)	30 (45.45)		
Mesangial proliferative glomerulonephritis	17 (32.69)	26 (36.39)		
Immunoglobulin A nephropathy	7 (13.46)	10 (15.15)		
Family medical history			0.324	0.569
No	46 (88.46)	56 (84.85)		
Yes	6 (11.54)	10 (15.15)		

Table 2. Therapeutic effectiveness assessment

Indicators	HP group (n=52)	HD+HP group (n=66)	χ^2	P
Clinical control	8 (15.38)	17 (25.76)		
Marked effectiveness	20 (38.46)	29 (43.94)		
Effectiveness	10 (19.23)	13 (19.70)		
Ineffectiveness	14 (26.92)	7 (10.61)		
Overall effectiveness	38 (73.08)	59 (89.39)	5.293	0.021

pendent predictors of treatment response. Statistical significance was set at P<0.05.

Results

Baseline patient demographics

Baseline characteristics of enrolled participants are summarized in **Table 1**. Age, sex, disease duration, diagnostic subtype, and family history were comparable between the two groups (P>0.05), confirming baseline homogeneity.

Therapeutic effectiveness

As shown in **Table 2**, the overall treatment effectiveness was significantly higher in the HD+HP group compared with the HP group (P<0.05).

Bone metabolic parameters

Changes in bone metabolism markers (PTH, P, and Ca) are illustrated in **Figure 2**. Baseline values were comparable between groups (all P>0.05). After treatment, both groups demonstrated significant reductions in PTH and P, alongside an increase in Ca (all P<0.05). The HD+HP regimen achieved greater PTH and P reductions and more pronounced Ca elevation than HP alone (all P< 0.05).

Renal function assessment

Renal function indices, including 24 h Upro, BUN, SCr, and eGFR, are shown in **Figure 3**. Baseline parameters did not differ significantly (all P>0.05). Following therapy, both groups exhibited significant improvements in all indices (all P<0.05), with the HD+HP group showing superior improvement compared to HP monotherapy (all P<0.05).

Inflammatory biomarker levels

As presented in **Table 3**, pretreatment serum levels of IL-17, TNF-α, and hs-CRP were similar between groups (all P>0.05). Post-treatment, all markers decreased significantly (all P< 0.05), with the HD+HP group demonstrating greater reductions than the HP group (all P<0.05).

Hemorheological parameters

WBV values at high and low shear rates are presented in **Table 4**. No baseline difference was noted (P>0.05). After treatment, viscosity reduced significantly in both groups, with the HD+HP therapy yielding markedly lower post-treatment WBV compared to HP alone (P<0.05).

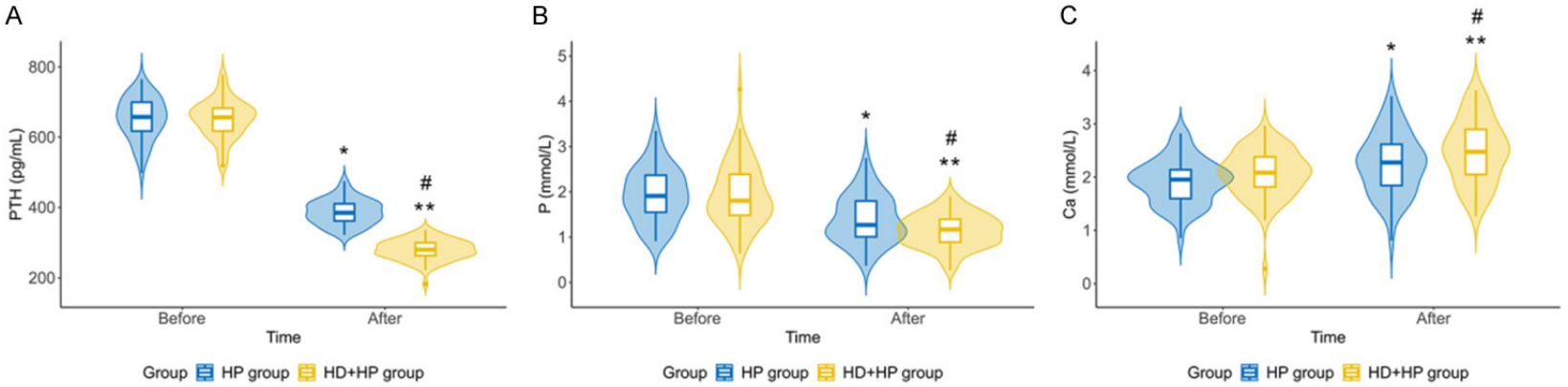

Figure 2. Bone metabolism marker profiles. A. Pre- and post-treatment assessment of parathyroid hormone (PTH). B. Phosphorus (P) levels pre- and post-treatment. C. Calcium (Ca) concentrations measured at baseline and study conclusion. Note: *P<0.05, **P<0.01 denote intragroup differences from pre-treatment; #P<0.05 indicates a significant difference compared to the HP group at the identical time point.

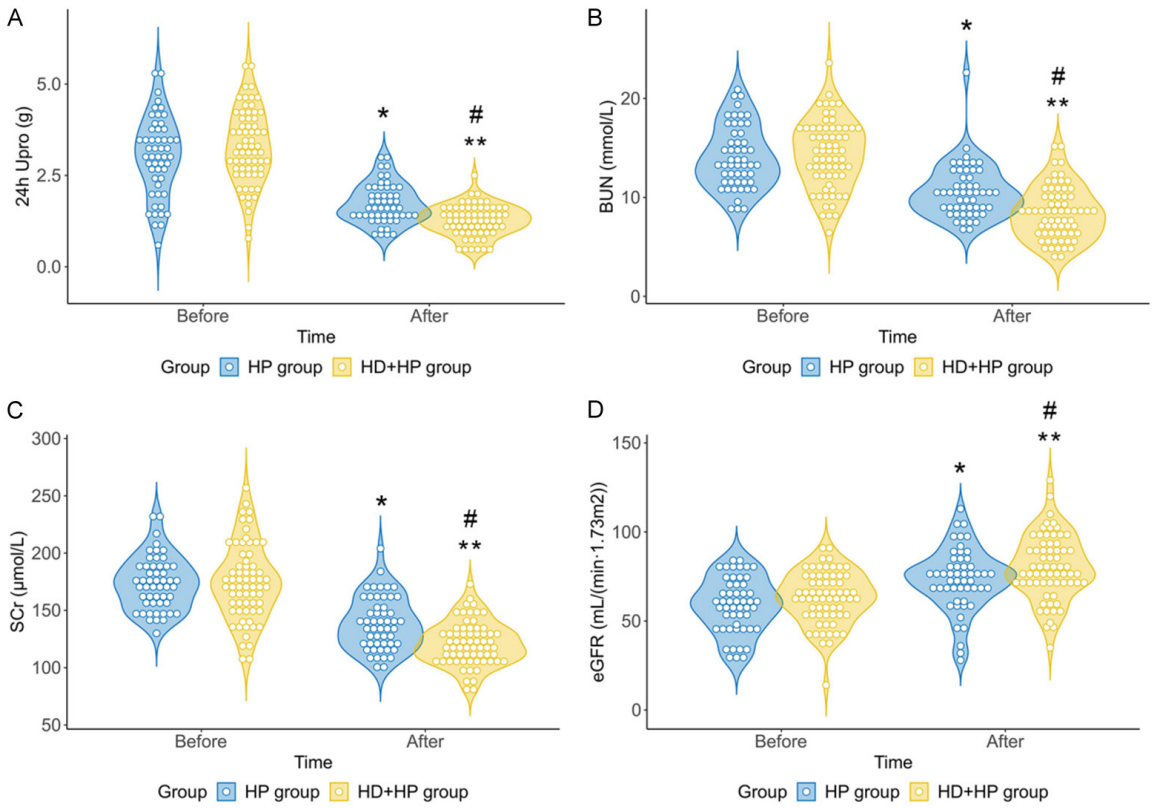

Figure 3. Renal function indicators. A. 24-hour urinary protein (24 h Upro) pre- and post-treatment. B. Blood urea nitrogen (BUN) variations before and after intervention. C. Serum creatinine (SCr) measurements prior to and following treatment. D. Estimated glomerular filtration rate (eGFR) values across both groups. Note: *P<0.05, **P<0.01 for within-group comparisons to pre-treatment; #P<0.05 for between-group comparisons to the HP group at the identical time point.

Table 3. Inflammatory biomarker concentrations

Indicators	HP group (n=52)	HD+HP group (n=66)	t	P
IL-17 (ng/L)				
Before	12.60±3.69	12.83±4.12	0.315	0.753
After	8.23±2.60*	5.14±1.84**	7.552	<0.001
TNF-α (ng/L)				
Before	38.19±9.13	38.67±6.98	0.324	0.747
After	25.31±6.06*	20.08±3.70**	4.279	<0.001
hs-CRP (mg/L)				
Before	5.15±1.69	5.83±2.15	1.870	0.064
After	3.98±1.26*	2.42±1.13**	7.076	<0.001

Note: IL-17, interleukin-17; TNF-α, tumor necrosis factor-alpha; hs-CRP, high-sensitivity C-reactive protein; *P<0.05, **P<0.01 for within-group comparisons to pre-treatment.

Coagulation/fibrinolysis markers

As shown in **Table 5**, baseline levels of PAI-1 and t-PA did not differ significantly (both P>0.05). Post-treatment, PAI-1 levels decreased while t-PA increased in both groups (both P<0.05), with more favorable changes observed in the HD+HP group (both P<0.05).

Serum uremic toxins

Table 6 details β2-MG, Hcy, and AGEs. Baseline levels were comparable (all P>0.05). After therapy, all markers decreased significantly in both groups (all P<0.05), with HD+HP treatment achieving more pronounced reductions than HP alone (all P<0.05).

Adverse events

Adverse reactions - including hypotension, circuit clotting, thrombocytopenia, puncture-site oozing, and pruritus - are summarized in

Table 4. Hemorheological parameters

Indicators	HP group (n=52)	HD+HP group (n=66)	t	P
Whole blood viscosity at high shear rate (mPa·s)				
Before	4.99±1.44	5.10±1.06	0.478	0.634
After	4.18±1.38*	3.73±1.07**	1.996	0.048
Whole blood viscosity at low shear rate (mPa·s)				
Before	9.86±2.36	9.75±2.53	0.241	0.810
After	7.96±1.62*	6.29±1.41**	5.981	<0.001

Note: *P<0.05, **P<0.01 denote intragroup differences from pre-treatment.

Table 5. Coagulation/fibrinolytic system indicators

Indicators	HP group (n=52)	HD+HP group (n=66)	t	P
PAI-1 (μmol/L)				
Before	39.75±3.87	39.97±4.44	0.283	0.778
After	35.33±4.36*	24.47±3.77**	14.497	<0.001
t-PA (μmol/L)				
Before	3.02±1.04	3.15±1.00	0.689	0.492
After	4.67±2.00*	5.99±1.52**	4.074	<0.001

Note: PAI-1, Plasminogen Activator Inhibitor-1; t-PA, tissue Plasminogen Activator; *P<0.05, **P<0.01 denote intragroup differences from pre-treatment.

Table 6. Assessment of serum uremic toxin parameters

Indices	HP group (n=52)	HD+HP group (n=66)	χ^2/t	P
β2-MG (mg/L)				
Before	44.67±8.30	43.85±11.15	0.442	0.659
After	34.56±8.41	23.97±7.08	7.424	<0.001
Hcy (μmol/L)				
Before	21.40±6.09	22.00±6.71	0.502	0.617
After	12.38±3.57	9.55±2.51	5.050	<0.001
AGEs (U/mL)				
Before	3.82±1.13	3.97±0.87	0.815	0.417
After	3.25±1.29	2.84±0.88	2.048	0.043

Note: β2-MG, β2-microglobulin; Hcy, homocysteine; AGEs, advanced glycation end-products.

Table 7. Recorded adverse reactions

Indicators	HP group (n=52)	HD+HP group (n=66)	χ^2	P
Hypotension	4 (7.69)	8 (12.12)		
Perfuser/circuit clotting	5 (9.62)	8 (12.12)		
Thrombocytopenia	6 (11.54)	10 (15.15)		
Puncture site oozing	3 (5.77)	5 (7.58)		
Pruritus	3 (5.77)	4 (6.06)		
Total	14 (26.92)	20 (30.30)	0.162	0.687

Table 7. The total incidence of adverse events was similar between groups (P>0.05), indicating comparable safety profiles.

Quality of life evaluation

Quality of life, assessed via the SF-36 questionnaire, is presented in **Table 8**. Baseline scores across domains (Physical Functioning, Bodily Pain, Social Functioning, Role-Emotional, and Mental Health) were comparable (all P>0.05). After treatment, HD+HP recipients exhibited significantly higher scores in all domains relative to HP-only patients (all P<0.05).

Determinants of treatment outcomes in CGN

Univariate analysis (**Table 9**) revealed no significant association between therapeutic efficacy and sex, age, diagnostic type, or family history (P>0.05). However, longer disease duration and treatment modality were strongly associated with treatment outcome (P<0.05).

Multivariate logistic regression (**Table 10**) identified extended disease duration (OR= 4.930, 95% CI: 1.339-18.147, P<0.05) and HP monotherapy (OR=3.069, 95% CI: 1.103-8.539, P<0.05) as independent predictors of inferior therapeutic efficacy.

Discussion

This study demonstrated that combining HD with HP yields greater therapeutic effective-

Table 8. Quality of life (SF-36) scores

Indicators	HP group (n=52)	HD+HP group (n=66)	t	P
Physical Functioning (points)				
Before	47.42±5.79	46.97±4.83	0.460	0.646
After	59.87±8.01*	71.29±6.53**	8.532	<0.001
Bodily Pain (points)				
Before	48.77±4.85	47.73±5.33	1.095	0.276
After	63.56±6.61*	70.70±7.29**	5.502	<0.001
Social functioning (points)				
Before	45.02±5.85	45.35±6.68	0.281	0.779
After	82.10±6.48*	87.45±6.47**	4.456	<0.001
Role-Emotional (points)				
Before	50.46±5.65	48.68±5.08	1.798	0.075
After	74.23±6.66*	79.14±7.46**	3.719	<0.001
Mental health (points)				
Before	49.04±5.14	50.73±6.57	1.523	0.130
After	77.67±5.90*	82.91±6.42**	4.560	<0.001

Note: *P<0.05, **P<0.01 for within-group comparisons to pre-treatment.

Table 9. Factors affecting treatment efficacy in chronic glomerulonephritis: results from univariate analysis

Indicators	Ineffective group (n=21)	Effective group (n=97)	χ^2/t	P
Sex			0.744	0.388
Male (n=63)	13 (61.90)	50 (51.55)		
Female (n=55)	8 (38.10)	47 (48.45)		
Age (years)			0.405	0.525
<45 (n=58)	9 (42.86)	49 (50.52)		
≥45 (n=60)	12 (57.14)	48 (49.48)		
Illness duration (years)			6.956	0.008
<5 (n=47)	3 (14.29)	44 (45.36)		
≥5 (n=71)	18 (85.71)	53 (54.64)		
Diagnostic category			1.702	0.427
Membranous nephropathy (n=58)	13 (61.90)	45 (46.39)		
Mesangial proliferative glomerulonephritis (n=43)	6 (28.57)	37 (38.14)		
Immunoglobulin A nephropathy (n=17)	2 (9.52)	15 (15.46)		
Family medical history			2.290	0.130
No (n=102)	16 (76.19)	86 (88.66)		
Yes (n=16)	5 (23.81)	11 (11.34)		
Treatment protocol			5.293	0.021
HP (n=52)	14 (66.67)	38 (39.18)		
HD+HP (n=66)	7 (33.33)	59 (60.82)		

Table 10. Multivariate analysis of determinants for treatment outcomes in chronic glomerulonephritis

Indicators	β	SE	Wald	P	Exp (B)	95% CI
Illness duration (years)	1.595	0.665	5.758	0.016	4.930	1.339-18.147
Treatment protocol	1.121	0.522	4.615	0.032	3.069	1.103-8.539

Note: Illness duration: Categorized as <5 years =0 and ≥5 years =1; Treatment protocol: Categorized as HD+HP =0 and HP =1; Efficacy: effective =0, ineffective =1.

ness than HP alone in managing CGN. Li J et al. [21] similarly reported that HD+HP therapy in elderly patients undergoing maintenance HD produced enhanced clinical outcomes, attenuated inflammation, and improved quality of life - findings consistent with the present study. Cheng W et al. [22], through a systematic review and meta-analysis, further confirmed that HD+HP improves survival in patients with ESRD. The enhanced efficacy likely arises from the complementary clearance mechanisms of HD and HP, which together facilitate metabolite removal, prevent HD-related complications, and improve internal homeostasis.

HD+HP therapy also favorably influenced bone metabolism (lower serum PTH and P, higher Ca), renal function (reduced 24 h Upro, BUN, and SCr; elevated eGFR), and systemic inflammation (suppressed IL-17, TNF-α, and hs-CRP). Declining renal function in CGN may result from active vitamin D deficiency and phosphate retention in residual nephrons - both potent stimulants of PTH secretion, which disrupt phosphate excretion and calcium reabsorption [23]. Elevated serum phosphorus independently accelerates IgA nephropathy progression [24]. Li W et al. [25] also reported that HD+HP increased serum Ca without compromising nutritional status, consistent with our findings. IL-17 and TNF-α act as central inflammatory mediators in glomerulonephritis [26, 27], while hs-CRP reflects renal microcirculatory disturbance [28]. The NLRP3-ASC-caspase-1 inflammasome axis also contributes to renal inflammation and fibrosis [29]. Zhao D et al. [30] observed that prolonged HD+HP use in ESRD patients alleviated pruritus and improved renal function through PTH suppression.

Furthermore, HD+HP improved hemorheological indices by lowering whole blood viscosity under both high- and low-shear conditions. High-shear viscosity reflects erythrocyte deformability, whereas low-shear viscosity correlates with red blood cell aggregation and plasma protein concentration [31]. Hyperviscosity contributes to renal hypoperfusion, ischemia, and hypoxia, thereby accelerating CGN progression [32]. Consequently, reductions in abnormal viscosity indicate improved microcirculation and therapeutic response [33].

The combination therapy also modulated coagulation and fibrinolysis by decreasing PAI-1 and increasing t-PA levels. PAI-1 inhibits fibrinolysis and promotes fibrin accumulation, while t-PA activates plasminogen to degrade fibrin [34]. Dysregulation of this balance contributes to glomerulosclerosis, and reversing it - via reduced PAI-1 and elevated t-PA - helps delay CGN progression [35]. HD+HP further reduced uremic toxins (β_2-MG, Hcy, and AGEs), which are implicated in tubulointerstitial inflammation, endothelial injury, and matrix deposition through the RAGE axis [36]. Although coagulation abnormalities are common in HD due to blood-membrane interactions [37], no increase in total adverse events was observed. Chen et al. [38] similarly reported no serious complications in maintenance HD patients treated with HD+HP. Additionally, the present study found significant improvements in quality-of-life indices among CGN patients receiving combined therapy. HD+HP has also been identified as more cost-effective than HD alone in ESRD populations [39], reinforcing its clinical utility.

Prolonged disease duration and HP monotherapy emerged as independent predictors of suboptimal response. This may reflect irreversible renal injury in long-standing cases and the limited detoxification scope of HP alone, which cannot fully restore internal homeostasis.

Several limitations should be acknowledged. First, the single-center, small-sample design may restrict generalizability. Future multicenter studies with larger, more diverse cohorts are warranted. Second, treatment costs were not assessed, and cost-effectiveness data would enhance clinical decision-making. Lastly, long-term prognostic differences between regimens were not examined; extended follow-up is needed to determine sustained benefits.

In conclusion, combined HD+HP therapy significantly improves bone metabolism, renal function, inflammatory control, and quality of life in CGN patients while maintaining an acceptable safety profile. However, therapeutic efficacy may be attenuated in those with prolonged disease duration or prior HP monotherapy.

Disclosure of conflict of interest

None.

Address correspondence to: Caixiang Zhang, Department of Nephrology, Shanxi Provincial People's Hospital, Taiyuan 030012, Shanxi, China. Tel: +86-15534023334; E-mail: Magicalove@163.com

References

[1] Wen CQ, Zou J, Li JX, Wang FJ and Ge HT. Integrating proteomics and network pharmacology to explore the relevant mechanism of Huangkui capsule in the treatment of chronic glomerulonephritis. Front Pharmacol 2025; 16: 1560420.

[2] Wang X, Liu Z, Yi N, Li L, Ma L, Yuan L and Wang X. The global burden of chronic kidney disease due to glomerulonephritis: trends and predictions. Int Urol Nephrol 2025; 57: 2613-2624.

[3] Khanna R. Clinical presentation & management of glomerular diseases: hematuria, nephritic & nephrotic syndrome. Mo Med 2011; 108: 33-36.

[4] Sharma S, Kalra D, Rashid I, Mehta S, Maity MK, Wazir K, Gupta S, Ansari SA, Alruqi OS, Khan R, Khan I and Anwar S. Assessment of health-related quality of life in chronic kidney disease patients: a hospital-based cross-sectional study. Medicina (Kaunas) 2023; 59: 1788.

[5] Keskinyan VS, Lattanza B and Reid-Adam J. Glomerulonephritis. Pediatr Rev 2023; 44: 498-512.

[6] Basile DP, Ullah MM, Collet JA and Mehrotra P. T helper 17 cells in the pathophysiology of acute and chronic kidney disease. Kidney Res Clin Pract 2021; 40: 12-28.

[7] Lousa I, Reis F, Santos-Silva A and Belo L. The signaling pathway of TNF receptors: linking animal models of renal disease to human CKD. Int J Mol Sci 2022; 23: 3284.

[8] Huang JW, Su T, Tan Y, Wang JW, Tang JW, Wang SX, Liu G, Zhao MH and Yang L. Serum anti-CRP antibodies differentiate etiology and predict relapse in acute tubulointerstitial nephritis. Clin Kidney J 2022; 15: 51-59.

[9] Nakagawa Y and Komaba H. Roles of parathyroid hormone and fibroblast growth factor 23 in advanced chronic kidney disease. Endocrinol Metab (Seoul) 2024; 39: 407-415.

[10] Komaba H and Fukagawa M. FGF23-parathyroid interaction: implications in chronic kidney disease. Kidney Int 2010; 77: 292-298.

[11] Felsenfeld AJ, Levine BS and Rodriguez M. Pathophysiology of calcium, phosphorus, and magnesium dysregulation in chronic kidney disease. Semin Dial 2015; 28: 564-577.

[12] Chapin JC and Hajjar KA. Fibrinolysis and the control of blood coagulation. Blood Rev 2015; 29: 17-24.

[13] Kitching AR, Holdsworth SR, Ploplis VA, Plow EF, Collen D, Carmeliet P and Tipping PG. Plasminogen and plasminogen activators protect against renal injury in crescentic glomerulonephritis. J Exp Med 1997; 185: 963-968.

[14] Zhan HQ, Zhang X, Chen XL, Cheng L and Wang X. Application of nanotechnology in the treatment of glomerulonephritis: current status and future perspectives. J Nanobiotechnology 2024; 22: 9.

[15] Yu Y, Xu L, Xu T, Yang C, Bu Q, Zhang W, Zhao L, Xu Y and Jiang W. Efficacy and safety of entecavir for hepatitis B virus-associated glomerulonephritis with renal insufficiency. Clin Exp Nephrol 2023; 27: 680-686.

[16] Murdeshwar HN and Anjum F. Hemodialysis. In: editors. StatPearls. Treasure Island (FL) ineligible companies. Disclosure: Fatima Anjum declares no relevant financial relationships with ineligible companies.: 2025.

[17] Ricci Z, Romagnoli S, Reis T, Bellomo R and Ronco C. Hemoperfusion in the intensive care unit. Intensive Care Med 2022; 48: 1397-1408.

[18] Romagnani P, Kitching AR, Leung N and Anders HJ. The five types of glomerulonephritis classified by pathogenesis, activity and chronicity (GN-AC). Nephrol Dial Transplant 2023; 38: ii3-ii10.

[19] Lorant T, Bengtsson M, Eich T, Eriksson BM, Winstedt L, Jarnum S, Stenberg Y, Robertson AK, Mosen K, Bjorck L, Backman L, Larsson E, Wood K, Tufveson G and Kjellman C. Safety, immunogenicity, pharmacokinetics, and efficacy of degradation of anti-HLA antibodies by IdeS (imlifidase) in chronic kidney disease patients. Am J Transplant 2018; 18: 2752-2762.

[20] Machaca-Choque D, Palomino-Guerra G, Flores-Cohaila J, Parihuana-Travezano E, Taype-Rondan A, Gomez-Colque S and Copaja-Corzo C. Quality of life and its associated factors in chronic kidney disease patients undergoing hemodialysis from a Peruvian city: a cross-sectional study. PLoS One 2024; 19: e0300280.

[21] Li J, Li H, Deng W, Meng L, Gong W and Yao H. The effect of combination use of hemodialysis and hemoperfusion on microinflammation in elderly patients with maintenance hemodialysis. Blood Purif 2022; 51: 739-746.

[22] Cheng W, Luo Y, Wang H, Qin X, Liu X, Fu Y and Ronco C. Survival outcomes of hemoperfusion and hemodialysis versus hemodialysis in patients with end-stage renal disease: a systematic review and meta-analysis. Blood Purif 2022; 51: 213-225.

[23] Kumari S, Singh PP, Kumar D, Kumar N, Kumar S and Shekhar R. Intact Parathyroid Hormone (iPTH) assay: an early approach for bone health assessment in chronic renal failure. Cureus 2024; 16: e72510.

[24] Yu G, Cheng J, Jiang Y, Li H, Li X and Chen J. Serum phosphorus and calcium levels, and kidney disease progression in immunoglobulin

A nephropathy. Clin Kidney J 2021; 14: 2108-2113.
[25] Li W, Wang J, Wang Y, Guan R, Zhao F and Zhang R. Additional hemoperfusion for patients receiving maintenance hemodialysis: a retrospective analysis. Am J Transl Res 2023; 15: 4045-4054.
[26] Ramani K and Biswas PS. Emerging roles of the Th17/IL-17-axis in glomerulonephritis. Cytokine 2016; 77: 238-244.
[27] Stefania K, Ashok KK, Geena PV, Katarina P and Isak D. TMAO enhances TNF-alpha mediated fibrosis and release of inflammatory mediators from renal fibroblasts. Sci Rep 2024; 14: 9070.
[28] Yang Y, Tang XF, Wang Y, Xu JZ, Gao PJ and Li Y. High-sensitivity C-reactive protein predicts microalbuminuria progression in essential hypertensive patients: a 3-year follow-up study. Blood Press Monit 2024; 29: 242-248.
[29] Hashemi A, Bigdeli R, Shahnazari M, Oruji F, Fattahi S, Panahnejad E, Ghadri A, Movahedi-Asl E, Mahdavi-Ourtakand M, Asgary V and Baghbani-Arani F. Evaluation of inflammasome activation in peripheral blood mononuclear cells of hemodialysis treated patients with glomerulonephritis. Iran J Pharm Res 2021; 20: 609-617.
[30] Zhao D, Wang Y, Wang Y, Jiang A, Cao N, He Y, Wang J, Guo Z, Liu W, Shi W, Hao L, Li J, Li W, Wang C, Wang J, Lin H, Shi W, Wang L, Jiang H, Ding G, Li Y, Hu W, Yue H, Liu J, Yang X, Yang Y, Liu G, Li H, Xiao Y, Wang N, Jiang G, Ma G, Wang J, Li Y, Li R, Li Q, Sun S, Jiao J, Xi C, Cai G, Sun X and Chen X. Randomized control study on hemoperfusion combined with hemodialysis versus standard hemodialysis: effects on middle-molecular-weight toxins and uremic pruritus. Blood Purif 2022; 1-11.
[31] Celik T, Yilmaz MI, Balta S, Ozturk C, Unal HU, Aparci M, Karaman M, Demir M, Yildirim AO, Saglam M, Kilic S, Eyileten T, Aydin I and Iyisoy A. The relationship between plasma whole blood viscosity and cardiovascular events in patients with chronic kidney disease. Clin Appl Thromb Hemost 2017; 23: 663-670.
[32] Celik T, Balta S, Ozturk C and Iyisoy A. Whole blood viscosity and cardiovascular diseases: a forgotten old player of the game. Med Princ Pract 2016; 25: 499-500.
[33] Kinik M, Camci S, Ari S, Ari H, Melek M and Bozat T. The effect of whole blood viscosity on contrast-induced nephropathy development in patients undergoing percutaneous coronary intervention. Postgrad Med 2022; 134: 78-84.
[34] Kwaan HC. The role of fibrinolytic system in health and disease. Int J Mol Sci 2022; 23: 5262.
[35] Medipally A, Xiao M, Biederman L, Dasgupta A, Satoskar AA, Parikh S, Ivanov I, Mikhalina G and Brodsky SV. Role of plasminogen activated inhibitor-1 in the pathogenesis of anticoagulant related nephropathy. Front Nephrol 2024; 4: 1406655.
[36] Yang Q, Liu G, Guo M, Yuan D, Huang J, Zhou Z and Li Q. The clinical efficacy evaluation of the KHA-200 hemoperfusion device in the treatment of end-stage renal disease patients undergoing blood purification therapy. Kidney Dis (Basel) 2025; 11: 270-282.
[37] Popova JA, Yadrihinskaya VN, Krylova MI, Sleptsova SS and Borisova NV. Comparison of clinical and laboratory parameters in patients with end-stage renal failure in the outcome of chronic glomerulonephritis and patients with end-stage renal failure in the outcome of other diseases. Wiad Lek 2016; 69: 739-741.
[38] Chen SJ, Jiang GR, Shan JP, Lu W, Huang HD, Ji G, Wu P, Wu GF, Wang W, Zhu C and Bian F. Combination of maintenance hemodialysis with hemoperfusion: a safe and effective model of artificial kidney. Int J Artif Organs 2011; 34: 339-347.
[39] Wang H, Jin H, Cheng W, Qin X, Luo Y, Liu X, Fu Y, Jiang G, Lu W, Jin C and Pennington M. Cost-effectiveness analysis of hemodialysis plus hemoperfusion versus hemodialysis alone in adult patients with end-stage renal disease in China. Ann Transl Med 2021; 9: 1133.