RESEARCH PAPER

Serum Albumin and C-Reactive Protein as Predictors of Intradialytic Hypotension in Patients with Chronic Kidney Disease on Maintenance Hemodialysis: A Hospital-Based Case-Control Study

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ABSTRACT

Background:

Intradialytic hypotension (IDH) remains the most prevalent acute complication of maintenance hemodialysis (MHD), affecting 10–40% of dialysis sessions and contributing substantially to cardiovascular morbidity and mortality in patients with end-stage renal disease (ESRD). Hypoalbuminemia and systemic inflammation, as reflected by elevated C-reactive protein (CRP), are prevalent in this patient population; however, their combined relationship with IDH remains incompletely characterized.

Methods:

A hospital-based case-control study was conducted at a tertiary care center in Rajahmundry. Eighty-four ESRD patients on MHD (three sessions per week, four hours per session, for at least three months) were enrolled: 42 cases (patients who developed IDH) and 42 controls (patients without IDH). IDH was defined per KDOQI guidelines as a reduction in systolic blood pressure ≥20 mmHg or a fall in mean arterial pressure ≥10 mmHg. Serum albumin and CRP were measured using standard enzymatic colorimetric methods. Hypoalbuminemia was defined as albumin <3.5 g/dL. Statistical analysis was performed using SPSS v22; chi-square test, independent t-test, and Mann-Whitney U test were applied as appropriate.

Results:

Mean serum albumin in cases was significantly lower than in controls (2.73 ± 0.65 g/dL vs. 3.62 ± 0.68 g/dL; p <0.001). Hypoalbuminemia was present in 66.7% of cases versus 19.0% of controls (p <0.001). Mean CRP was significantly higher in cases compared to controls (8.21 ± 6.02 mg/dL vs. 2.53 ± 6.08 mg/dL; p <0.001). Elevated CRP was identified in 97.6% of cases versus 33.3% of controls (p <0.001). Both groups were comparable in age, sex, serum creatinine, and comorbidities.

Conclusions:

Low serum albumin and elevated CRP are significantly associated with IDH in ESRD patients on MHD. Routine monitoring of these biomarkers may facilitate early risk stratification and guide preventive strategies to reduce IDH-related morbidity.

Keywords: End-stage renal disease; Intradialytic hypotension; Hemodialysis; Serum albumin; C-reactive protein.

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INTRODUCTION

Chronic kidney disease (CKD) is a major global public health burden, affecting approximately 13.4% of the adult population worldwide, with a mortality burden exceeding 1.2 million deaths annually.[1,2] In India, estimates suggest a crude incidence of end-stage renal disease (ESRD) of approximately 152 per million population per year.[3] Globally, the incidence of CKD increased by 89%, its prevalence by 87%, CKD-attributed deaths by 98%, and dialysis utilization by 62% between 1990 and 2016.[4] Patients with CKD carry a disproportionately high risk of cardiovascular disease (CVD) and all-cause mortality, which

escalates sharply as renal function declines toward ESRD.[5]

Hemodialysis (HD) remains the predominant modality of renal replacement therapy for ESRD globally. Despite being a life-sustaining intervention, hemodialysis is associated with a range of acute and chronic complications. Among acute complications, intradialytic hypotension (IDH) is the most frequently encountered, occurring in 10–40% of all dialysis sessions depending on the case mix and the definition applied.[6,7] IDH is broadly defined by the Kidney Disease Outcomes Quality Initiative (KDOQI) and the European Best Practice Guidelines as a fall in systolic blood pressure ≥20 mmHg or a decline in mean arterial pressure (MAP) ≥10 mmHg during or immediately following a dialysis session.[8]

The hemodynamic pathophysiology of IDH is multifactorial, involving an imbalance between the rate of ultrafiltration (UFR) and the adequacy of hemodynamic compensatory responses, including reflex vasoconstriction, sympathoadrenal activation, and splanchnic blood flow redistribution.[9] When these mechanisms fail, IDH ensues. Repeated episodes of IDH result in recurrent end-organ hypoperfusion affecting the heart, brain, kidneys, and gastrointestinal tract, leading to accelerated cardiovascular mortality, ischemic brain injury, and loss of residual renal function.[10,11]

Serum albumin, the most abundant plasma protein, constitutes 55–60% of total plasma proteins. Its principal physiological functions include maintenance of plasma oncotic pressure, modulation of intravascular volume, and transport of endogenous and exogenous ligands.[12] Hypoalbuminemia (serum albumin <3.5 g/dL) leads to reduced plasma oncotic pressure, impaired intravascular volume refilling during ultrafiltration, and a consequent premature fall in blood pressure, predisposing patients to IDH.13 Additionally, hypoalbuminemia is an established predictor of cardiovascular morbidity, left ventricular hypertrophy, and all-cause mortality in ESRD patients.[14]

C-reactive protein (CRP) is an acute-phase pentameric protein synthesized by the liver in response to interleukin-6 (IL-6) secreted by activated macrophages and adipocytes. It is a well-validated biomarker of systemic inflammation, atherosclerosis, and chronic immune activation.[15] CRP concentrations are persistently elevated in ESRD, driven by ongoing systemic inflammation, repeated membrane-blood contact during dialysis, infections, and uremic milieu.[16] Immune activation mediated by IL-6, CRP, and other cytokines has been implicated in the pathogenesis of IDH, with circulating CRP levels correlating significantly with the magnitude of intradialytic blood pressure reduction.[17]

Despite several independent studies demonstrating relationships between hypoalbuminemia and IDH [13,18,19] and between elevated CRP and IDH [17,20] relatively few studies have examined both biomarkers together as predictors of IDH. Furthermore, limited data exist from the Indian to subcontinent, where the burden of ESRD is rising rapidly and patients often present with more severe nutritional and inflammatory derangements. Given that serum albumin and CRP are low-cost, universally available laboratory tests, establishing their utility in IDH prediction would have substantial clinical and resource implications.

The present study was therefore designed to evaluate the relationship between serum albumin levels, CRP levels, and the occurrence of IDH in patients with CKD on regular MHD. The findings are expected to provide evidence supporting the inclusion of routine albumin and CRP monitoring as part of a hemodialysis risk stratification protocol.

MATERIALS AND METHODS

Study Design and Setting

A hospital-based case-control study was conducted in the Departments of General Medicine and Nephrology at GSL Medical College and General Hospital,Rajahmundry,Andhra Pradesh, India. The study was conducted over a period of 18 months from April 2025 to July 2026.

Study Population

Adult patients with ESRD who were receiving MHD (three sessions per week, each of four hours duration, for a minimum of three months) were eligible for enrollment. Patients were classified into two groups based on the occurrence of IDH during monitored hemodialysis sessions.

Inclusion Criteria

Patients were included if they: (1) had a confirmed diagnosis of ESRD and were on MHD for at least three months, three times weekly, each session lasting four hours; (2) were aged between 18 and 75 years; and (3) provided written informed consent.

Exclusion Criteria

Patients were excluded if they had: (1) decompensated chronic liver disease; (2) heart failure or established coronary artery disease; (3) underlying malignancy or neuroendocrine tumor; (4) clinical fluid overload at baseline; (5) hemoglobin <9 g/dL; or (6) severe malnutrition defined as body mass index (BMI) <18.5 kg/m².

Sample Size Calculation

The sample size was estimated based on the difference in proportions of hypoalbuminemia between IDH and non-IDH groups, as reported by Prasad et al. (70% vs. 36.7%).18 Using the standard formula $N = 2\sigma^2(Z_{1-\alpha/2} + Z_{1-\beta})^2 / d^2$, with $\sigma = 0.49$, $Z_{1-\alpha/2} = 1.96$ at 95% confidence, $Z_{1-\beta} = 0.84$ at 80% power, and $d = 0.3$, the

minimum required sample size was 42 patients per group (total N = 84).

Case and Control Definition

Cases were defined as patients who developed IDH during MHD sessions. Controls were patients undergoing the same MHD regimen who did not develop IDH during the monitoring period. IDH was defined per KDOQI and European Best Practice Guidelines as a reduction in systolic blood pressure ≥20 mmHg or a reduction in mean arterial pressure ≥10 mmHg during or at the end of a hemodialysis session.8

Data Collection

After obtaining institutional ethics committee approval and written informed consent from all participants, a structured case proforma was used to record demographic details (age, sex), anthropometric measurements (height, weight, BMI), comorbidities (hypertension, diabetes mellitus, hypothyroidism), and detailed medication history. Blood pressure was measured with a calibrated automated sphygmomanometer at baseline (pre-dialysis) and every 30 minutes during each dialysis session.

Laboratory and Clinical Investigations

The following investigations were performed in all study participants: complete blood count (CBC), blood urea, serum creatinine, serum albumin, CRP, total protein, liver function tests (total bilirubin, direct bilirubin, AST, ALT, ALP), lipid profile, urine microalbumin-to-creatinine ratio, and estimated glomerular filtration rate (eGFR) calculated using the Modification of Diet in Renal Disease (MDRD) formula. Imaging investigations included chest X-ray, ultrasonography (USG) of the abdomen, and two-dimensional echocardiography (2D Echo) including assessment of ejection fraction and ventricular geometry.

Serum albumin was analyzed using the enzymatic colorimetric (bromocresol green) method on an automated analyzer. Hypoalbuminemia was defined as serum albumin <3.5 g/dL in accordance with NKF-KDOQI guidelines. CRP was measured using a high-sensitivity immunoturbidimetric assay; a value >1 mg/dL was considered elevated.

Statistical Analysis

Data were entered and analyzed using IBM SPSS Statistics, version 22 (Somers, NY, USA). Categorical variables were expressed as frequencies and proportions. Continuous variables were expressed as mean ±standard deviation (SD). The chi-square test or Fisher's exact test (for 2×2 tables) was used for categorical data. The independent t-test or Mann-Whitney U test was used as appropriate for continuous variables. A p-value <0.05 was considered statistically significant.

Ethical Considerations

The study was approved by the Institutional Ethics Committee of GSL Medical College and General Hospital,Rajahmundry prior to the commencement of patient enrollment. Written informed consent was obtained from all participants. Patient confidentiality was maintained throughout the study.

RESULTS

Demographic and Baseline Characteristics

A total of 84 ESRD patients on MHD were enrolled: 42 cases and 42 controls. The demographic and baseline clinical profiles of both groups are presented in Tables 1–3. Mean age in the cases group was 51.29 ±14.71 years and in the controls group was 50.88 ± 11.29 years; the difference was not statistically significant (p = 0.888) (Table 1). Age distribution across categories did not differ significantly between the two groups ($\chi^2 = 5.17$, df = 4, p = 0.27) (Table 2). In the cases group, 64.29% were male and 35.71% were female; in the controls group, 76.19% were male and 23.81% were female. Sex distribution was comparable between groups ($\chi^2 = 1.424$, df = 1, p = 0.233) (Table 3). Mean duration of renal failure at the time of enrollment was significantly shorter in the cases group (3.45 ±1.87 years) compared to controls (5.05 ±2.27 years; p = 0.001).

Comorbidities

Hypertension was significantly more prevalent in the controls group (95.24%) compared to cases (54.76%; p <0.001) (Table 4). Diabetes mellitus was present in 83.33% of cases versus 66.67% of controls, with no statistically significant difference ($\chi^2 = 3.111$, p = 0.078). Hypothyroidism was observed only in the cases group (4.76%), with no cases in the controls group (p = 0.359).

Vital Parameters and Renal Profile

Mean pre-dialysis systolic blood pressure (SBP) was significantly higher in the cases group (142.95 ± 18.37 mmHg) compared to controls (128.86 ±16.73 mmHg; p <0.001) (Table 5). Mean diastolic blood pressure (84.38 ±8.23 vs. 83.57 ±14.27 mmHg; p = 0.751) and heart rate (92.6 ±8.52 vs. 92.98 ±14.94 beats/min; p = 0.886) did not differ significantly between groups. Mean blood urea was significantly lower in cases (71.68 ±54.69 mg/dL) compared to controls (111.01 ±61.78 mg/dL; p = 0.003), while serum creatinine was comparable between groups (6.63 ±2.82 vs. 6.22 ±5.23 mg/dL; p = 0.652).

Serum Albumin and Total Protein

Mean serum albumin was significantly lower in the cases group (2.73 ±0.65 g/dL) compared to controls (3.62 ±0.68 g/dL; p <0.001) (Table 6). Hypoalbuminemia was present in 66.7% of cases and only 19.0% of controls (p <0.001). Mean total protein

was significantly higher in the cases group (6.45 ± 0.72 g/dL) than in controls (5.92 ±0.78 g/dL; p = 0.002). Serum alkaline phosphatase (ALP) was significantly elevated in cases (126.76 ±71.14 U/L) versus controls (74.48 ±38.04 U/L; p <0.001). There were no significant differences in total bilirubin, direct bilirubin, AST, or ALT between the two groups.

C-Reactive Protein

Mean CRP was significantly elevated in the cases group (8.21 ±6.02 mg/dL) compared to the controls group (2.53 ±6.08 mg/dL; p <0.001) (Table 6). Elevated CRP (>1 mg/dL) was present in 97.6% of cases versus only 33.3% of controls (p <0.001). Among controls, 66.7% had low CRP (<1 mg/dL).

Hemoglobin and White Blood Cell Count

Mean hemoglobin was significantly lower in cases (10.03 ±0.96 g/dL) compared to controls (10.85 ± 1.00 g/dL; p <0.001) (Table 6). Mean white blood cell count was comparable between groups (7.76 ±2.34 × 10^3/µL in cases vs. 7.35 ±2.39 × 10^3/µL in controls; p = 0.426).

Imaging and Echocardiographic Findings

Chest X-ray findings differed significantly between groups (χ^2 = 11.842, p = 0.037), with clear lung fields observed in 83.33% of cases and 73.81% of controls. USG abdomen findings also differed significantly (χ^2 = 29.317, p <0.001): Grade 3 renal parenchymal changes were predominant in cases (57.14%), while Grade 1–2 changes were more common in controls. On 2D echocardiography, 97.62% of cases showed no regional wall motion abnormality (RWMA), whereas all controls exhibited concentric left ventricular hypertrophy (LVH): mild in 59.52% and moderate in 40.48% (χ^2 = 80.222, p <0.001). Ejection fraction distribution also differed significantly between groups (χ^2 = 12.982, p = 0.043).

Association of Serum Albumin and CRP with IDH

The combined distribution of serum albumin and CRP between the two groups is summarized in Tables 7 and 8. Among cases with IDH, 66.7% had hypoalbuminemia and 97.6% had elevated CRP. Among controls without IDH, 81.0% had normal serum albumin and 66.7% had low CRP. There was a significant association between IDH and both hypoalbuminemia (p <0.001) and elevated CRP (p <0.001).

Table 1. Mean Age Comparison Between Cases and Controls

Variable	Cases Mean ±SD	Controls Mean ±SD	p-value
Age (years)	51.29 ± 14.71	50.88 ± 11.29	0.888

SD, standard deviation. p-value derived by independent samples t-test.

Table 2. Age Distribution Between Cases and Controls

Age Category	Cases (n)	Cases (%)	Controls (n)	Controls (%)	p-value
≤30 years	4	9.52	2	4.76	0.27
31–40 years	5	11.90	6	14.29	
41–50 years	12	28.57	8	19.05	
51–60 years	11	26.19	20	47.62	
>60 years	10	23.81	6	14.29	

χ^2 = 5.17, df = 4. p-value derived by chi-square test.

Table 3. Sex Distribution Between Cases and Controls

Sex	Cases (n)	Cases (%)	Controls (n)	Controls (%)	p-value
Male	27	64.29	32	76.19	0.233
Female	15	35.71	10	23.81	0.344

χ^2 = 1.424, df = 1. p-value derived by chi-square test.

Table 4. Comorbidity Prevalence Between Cases and Controls

Comorbidity	Cases (n)	Cases (%)	Controls (n)	Controls (%)	p-value
Hypertension	23	54.76	40	95.24	<0.001*
Diabetes mellitus	35	83.33	28	66.67	0.078
Hypothyroidism	2	4.76	0	0.00	0.359

*Statistically significant ($p < 0.05$). χ^2 test used for all comparisons.

Table 5. Vital Signs and Renal Function Comparison Between Cases and Controls

Parameter	Cases Mean	Cases SD	Controls Mean	Controls SD	p-value	Sig.
Pulse (beats/min)	92.60	8.52	92.98	14.94	0.886	NS
SBP (mmHg)	142.95	18.37	128.86	16.73	<0.001	*
DBP (mmHg)	84.38	8.23	83.57	14.27	0.751	NS
Urea (mg/dL)	71.68	54.69	111.01	61.78	0.003	*
Creatinine (mg/dL)	6.63	2.82	6.22	5.23	0.652	NS
Duration of renal failure (years)	3.45	1.87	5.05	2.27	0.001	*

SBP, systolic blood pressure; DBP, diastolic blood pressure; SD, standard deviation; NS, not significant; * $p < 0.05$ (independent t-test).

Table 6. Biochemical Parameters Including Serum Albumin, CRP, and Hemoglobin in Cases and Controls

Parameter	Cases Mean	Cases SD	Controls Mean	Controls SD	p-value	Sig.
Serum albumin (g/dL)	2.73	0.65	3.62	0.68	<0.001	*
CRP (mg/dL)	8.21	6.02	2.53	6.08	<0.001	*
Total protein (g/dL)	6.45	0.72	5.92	0.78	0.002	*
ALP (U/L)	126.76	71.14	74.48	38.04	<0.001	*
Hemoglobin (g/dL)	10.03	0.96	10.85	1.00	<0.001	*

WBC count (×10³/μL)	7.76	2.34	7.35	2.39	0.426	NS
Total bilirubin (mg/dL)	0.68	0.37	0.63	0.28	0.469	NS
AST (U/L)	32.64	20.76	31.29	13.56	0.724	NS
ALT (U/L)	30.40	20.90	35.40	15.03	0.212	NS

CRP, C-reactive protein; ALP, alkaline phosphatase; WBC, white blood cell; AST, aspartate aminotransferase; ALT, alanine aminotransferase; SD, standard deviation; NS, not significant; * p <0.05 (independent t-test or Mann-Whitney U test as appropriate).

Table 7. Serum Albumin and CRP Status in Cases (IDH) vs. Controls (No IDH)

Parameter	Cases (n)	Cases (%)	Controls (n)	Controls (%)	p-value
Hypoalbuminemia (<3.5 g/dL)	28	66.7	8	19.0	<0.001*
Normal albumin (≥3.5 g/dL)	14	33.3	34	81.0	-
Elevated CRP (>1 mg/dL)	41	97.6	14	33.3	<0.001*
Normal CRP (≤1 mg/dL)	1	2.4	28	66.7	-

IDH, intradialytic hypotension; CRP, C-reactive protein. * Chi-square test. Hypoalbuminemia defined as serum albumin <3.5 g/dL (NKF-KDOQI); elevated CRP defined as >1 mg/dL.

Table 8. Imaging and Echocardiographic Findings Between Cases and Controls

Finding	Cases (n)	Cases (%)	Controls (n)	Controls (%)	p-value	Test
USG: Grade 3 parenchymal changes	24	57.14	6	14.29	<0.001*	χ^2 = 29.32
USG: Grade 2 parenchymal changes	14	33.33	12	28.57	-	-
2D Echo: Mild LVH	0	0.00	25	59.52	<0.001*	χ^2 = 80.22
2D Echo: Moderate LVH	1	2.38	17	40.48	-	-
2D Echo: No	41	97.62	0	0.00	-	-

RWMA (normal)						

USG, ultrasonography; LVH, left ventricular hypertrophy; RWMA, regional wall motion abnormality. * $p < 0.05$.

DISCUSSION

This hospital-based case-control study investigated the relationship between serum albumin, CRP, and the occurrence of IDH in ESRD patients on MHD. The principal findings were: (1) serum albumin was significantly lower in patients with IDH compared to those without IDH; (2) hypoalbuminemia was present in nearly two-thirds of the IDH group; (3) CRP was markedly elevated in nearly all patients with IDH; and (4) both biomarkers demonstrated significant statistical associations with IDH, supporting their utility as predictive markers in this clinical context.

The study groups were well-matched for age and sex. Mean age was approximately 51 years in both groups, consistent with the demographics reported in comparable studies from the region.[18,19,20] The absence of significant sex differences between cases and controls is concordant with findings from Kora et al.[21] and Al-Etreby et al.[22] Although some European cohorts have reported higher IDH rates in female patients and older age groups. [23,24] our findings align with earlier Indian data where no such sex-based differences were observed.[18]

The finding of significantly lower serum albumin in IDH patients (2.73 ± 0.65 g/dL vs. 3.62 ± 0.68 g/dL; $p < 0.001$) is consistent with the established pathophysiology. Hypoalbuminemia reduces plasma oncotic pressure, impairs intravascular volume refilling during the ultrafiltration process, and predisposes patients to premature declines in blood pressure.[13,25] Our finding of hypoalbuminemia in 66.7% of IDH cases mirrors the 70% reported by Prasad et al.[18] and the findings of Pakfetrat et al.,[20] where mean albumin was significantly lower in the dialysis-induced hypotension (DIH) group (3.9 ± 0.4 g/dL) compared to normotensive patients (4.3 ± 0.5 g/dL). Similarly, Al-Etreby et al.[22] demonstrated a significant negative correlation between serum albumin and the magnitude of intradialytic blood pressure decline. The seminal Canadian study by Foley et al.[14] established that each 1 g/dL decrease in mean serum albumin was independently associated with de novo cardiac failure, ischemic heart disease, and overall mortality in ESRD, reinforcing albumin as a critical prognostic biomarker.

The observation that CRP was elevated in 97.6% of IDH cases compared to 33.3% of controls ($p < 0.001$), and that mean CRP was 3.25-fold higher in the cases group, is particularly noteworthy. The mechanistic link between CRP elevation and IDH is thought to involve immune activation-mediated endothelial dysfunction, impaired vasoconstriction, and cytokine-driven cardiovascular instability. Pakfetrat et al.[20] found that the incidence of DIH was significantly higher in patients with elevated high-sensitivity CRP compared to those with normal levels (41.4% vs. 13.8%; $p = 0.038$). Tomita et al.[26] demonstrated that the maximal percent change in MAP during HD correlated significantly with serum CRP ($r = 0.67$; $p < 0.05$) in patients with symptomatic DIH, implicating IL-6-driven immune activation in pathogenesis. Abd El Wahab et al.[27] reported that 90% of patients who developed IDH had positive CRP, compared to only 10% in the non-IDH group ($p < 0.001$), closely mirroring our findings.

The relationship between hypoalbuminemia and CRP is complex. CRP elevation is associated with increased inflammatory cytokine activity, particularly IL-6, which downregulates hepatic albumin synthesis, creating a cycle of inflammation-mediated hypoalbuminemia.[28] In the present study, the incidence of hypoalbuminemia was higher among patients with elevated CRP compared to those with normal CRP, consistent with independent reports.[29] However, a formal correlation analysis between the two was not performed, which represents a limitation.

In our study, controls exhibited a significantly higher prevalence of hypertension (95.24%) compared to cases (54.76%; $p < 0.001$), while pre-dialysis SBP was paradoxically higher in the cases group. This apparent discrepancy may reflect the complex hemodynamic profile of IDH patients, where higher baseline SBP predialysis does not preclude intradialytic hypotension if cardiac reserve is impaired or ultrafiltration volumes are large. The presence of concentric LVH in all controls, as opposed to predominantly normal echocardiographic findings (no RWMA) in cases, may reflect a longer dialysis duration in controls (5.05 ± 2.27 vs. 3.45 ± 1.87 years), which is known to promote ventricular remodeling. Hypoalbuminemia has been identified as an independent predictor of LVH in hemodialysis patients.[14,22] The lower hemoglobin in cases compared to controls may further contribute to cardiac strain and hypotensive vulnerability.

The finding that blood urea was significantly lower in cases than controls is initially counterintuitive; however, this may reflect lower dietary protein intake in the hypoalbuminemic group, or differences in dialysis vintage, since cases had a shorter MHD duration than controls. Lower BUN associated with malnutrition in ESRD patients is a recognized phenomenon and may reflect overall catabolic burden rather than superior dialysis adequacy.

From a public health perspective, the high prevalence of IDH among CKD patients on MHD has substantial

implications. IDH increases the risk of acute myocardial injury, cerebrovascular ischemia, and loss of residual renal function, all of which augment healthcare costs and reduce patient quality of life.[10,11] Both serum albumin and CRP are inexpensive, universally available laboratory tests that can be incorporated into routine pre-dialysis assessment to stratify IDH risk. Patients identified as high-risk could benefit from targeted nutritional supplementation, optimization of dialysis prescriptions (e.g., cool dialysate, sodium profiling, reduced UFR), and anti-inflammatory interventions.

The strengths of this study include the case-control design with a well-matched comparison group, a clearly defined case definition consistent with international guidelines, use of validated laboratory methodologies, and a comprehensive battery of clinical and biochemical assessments. The study was conducted at a single tertiary center with a dedicated nephrology unit, ensuring consistency of dialysis prescription and monitoring.

LIMITATIONS

The study is limited by its single-center, cross-sectional design and use of convenient sampling, which may introduce selection bias. IDH and biomarker assessments were conducted at a single session rather than longitudinally. Multivariate logistic regression to identify independent predictors was not performed, and high-sensitivity CRP (hsCRP) was not specifically utilized. The relatively small sample size may limit the study's statistical power for subgroup analyses.

CONCLUSION

This case-control study demonstrates a significant and clinically meaningful association between hypoalbuminemia, elevated CRP, and the occurrence of intradialytic hypotension in ESRD patients on maintenance hemodialysis. Mean serum albumin was substantially lower and mean CRP significantly higher in patients who developed IDH compared to those without IDH. Hypoalbuminemia was present in two-thirds of cases and elevated CRP in nearly all. These findings support the inclusion of routine serum albumin and CRP monitoring in the clinical management pathway of hemodialysis patients. Early identification of at-risk individuals through these simple biomarkers may facilitate targeted nutritional and dialytic interventions, ultimately reducing the burden of IDH-related cardiovascular morbidity and improving outcomes in this vulnerable population. Larger prospective multicenter studies with multivariate analyses are warranted to validate these findings and to establish evidence-based thresholds for clinical decision-making.

REFERENCES

1. Hoffman M. Picture of the Kidneys. WebMD. 2021. Available from: https://www.webmd.com/kidney-stones/picture-of-the-kidneys
2. Dare AJ, Fu SH, Patra J, et al. Renal failure deaths and their risk factors in India 2001–13: nationally representative estimates from the Million Death Study. Lancet Glob Health. 2017;5(1):e89-95.
3. Xie Y, Bowe B, Mokdad AH, et al. Analysis of the Global Burden of Disease study highlights the global, regional, and national trends of chronic kidney disease epidemiology from 1990 to 2016. Kidney Int. 2018;94(3):567-81.
4. Tonelli M, Wiebe N, Culleton B, et al. Chronic kidney disease and mortality risk: a systematic review. J Am Soc Nephrol. 2006;17(7):2034-47.
5. GBD Chronic Kidney Disease Collaboration. Global, regional, and national burden of chronic kidney disease, 1990–2017: a systematic analysis for the Global Burden of Disease Study 2017. Lancet. 2020;395:709-33.
6. Raja SM, Seyoum Y. Intradialytic complications among patients on twice-weekly maintenance hemodialysis: an experience from a hemodialysis center in Eritrea. BMC Nephrology. 2020;21:163.
7. Daugirdas JT. Pathophysiology of dialysis hypotension: an update. Am J Kidney Dis. 2001;38(4 Suppl 1):S11-7.
8. McIntyre CW, Salerno FR. Diagnosis and Treatment of Intradialytic Hypotension in Maintenance Hemodialysis Patients. Clin J Am Soc Nephrol. 2018;13(3):486-9.
9. Reilly RF. Attending Rounds: A Patient with Intradialytic Hypotension. Clin J Am Soc Nephrol. 2014;9(4):798-803.
10. Stefánsson BV, Brunelli SM, Cabrera C, et al. Intradialytic hypotension and risk of cardiovascular disease. Clin J Am Soc Nephrol. 2014;9(12):2124-32.
11. Shoji T, Tsubakihara Y, Fujii M, et al. Hemodialysis-associated hypotension as an independent risk factor for two-year mortality in hemodialysis patients. Kidney Int. 2004;66(3):1212-20.
12. Yuwen P, Chen W, Lv H, et al. Albumin and surgical site infection risk in orthopaedics: a meta-analysis. BMC Surg. 2017;17(1):7.
13. Nakamoto H, Honda N, Mimura T, Suzuki H. Hypoalbuminemia is an important risk factor of hypotension during hemodialysis. Hemodial Int. 2006;10(Suppl 2):S10-5.

14. Foley RN, Parfrey PS, Harnett JD, et al. Impact of hypertension on cardiomyopathy, morbidity and mortality in end-stage renal disease. Kidney Int. 1996;49(5):1379-85.
15. Nehring SM, Goyal A, Patel BC. C Reactive Protein. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2021.
16. Hafez MZ, Abd El Aziz WAA, Abd El Wahab SMA. Relation of serum albumin level and C-reactive protein to hypotensive episodes during hemodialysis sessions. Egypt J Hosp Med. 2018;71(1):2274-9.
17. Tomita M, Malhotra D, Dheenana S, Shapiro JI, Henrich WL, Santoro TJ. A potential role for immune activation in hemodialysis hypotension. Ren Fail. 2001;23(5):637-49.
18. Prasad ML, Mazumdar P, Malhotra A, Das M. The relationship between hypoalbuminemia and intradialytic hypotension in hemodialysis patients. Int J Sci Res. 2020;9(5):35-7.
19. Kora M, Tawfeek A, El-Zorkany K, Abd El-Mohsen AH. The relationship between hypoalbuminemia and intradialytic hypotension in hemodialysis patients. Menoufia Med J. 2020;33(1):110-5.
20. Pakfetrat M, Roozbeh J, Malekmakan L, et al. Relation of serum albumin and C-reactive protein to hypotensive episodes during hemodialysis sessions. Saudi J Kidney Dis Transpl. 2010;21(4):707-11.
21. Kora M, Tawfeek A, El-Zorkany K. The relationship between hypoalbuminemia and intradialytic hypotension. Menoufia Med J. 2020;33:110-5.
22. Al-Etreby EA, Abd El-Aziz A, El-Waseef O, El-Moselhy EA. The association between hypoalbuminemia and intradialytic hypotension in hemodialysis patients. Egypt J Hosp Med. 2018;63(1):185-94.
23. Tislér A, Akócsi K, Hárshegyi I, et al. Comparison of dialysis and clinical characteristics of patients with frequent and occasional hemodialysis-associated hypotension. Kidney Blood Press Res. 2002;25(2):97-102.
24. Capuano A, Sepe V, Cianferno P, et al. Cardiovascular impairment, dialysis strategy and tolerance in elderly and young patients on maintenance hemodialysis. Nephrol Dial Transplant. 1993;5:1023-30.
25. Moon KH, Song IS, Yang WS. Hypoalbuminemia as a risk factor for progressive left ventricular hypertrophy in hemodialysis patients. Am J Nephrol. 2000;20(6):396-401.
26. Tomita M, Malhotra D, Dheenana S, et al. A potential role for immune activation in hemodialysis hypotension. Ren Fail. 2001;23(5):637-49.
27. Abd El Wahab SM, Hafez MZ, Abd El Aziz WA. Relation of serum albumin level and C-reactive protein to hypotensive episodes during hemodialysis sessions. Egypt J Hosp Med. 2018;71(1):2274-9.
28. Coutinho HM, Leenstra T, Acosta LP, et al. Pro-inflammatory cytokines and C-reactive protein are associated with undernutrition in the context of Schistosoma japonicum infection. Am J Trop Med Hyg. 2006;75(4):720-6.
29. Kaysen GA, Stevenson FT, Depner TA. Determinants of albumin concentration in hemodialysis patients. Am J Kidney Dis. 1997;29(5):658-68.
30. Menon V, Greene T, Wang X, et al. C-reactive protein and albumin as predictors of all-cause and cardiovascular mortality in chronic kidney disease. Kidney Int. 2005;68(2):766-72.