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Research Article

Ultrasound-Based Clinical Profiles for Predicting the Risk of Intradialytic Hypotension in Critically Ill Patients on Intermittent Dialysis

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ABSTRACT

Background: Intradialytic hypotension (IDH) is a common and serious complication of intermittent hemodialysis (IHD) in critically ill patients with acute kidney injury (AKI). Accurate pre-dialysis risk stratification remains a challenge, particularly in the ICU. This study aims to determine whether ultrasound-based cardiopulmonary profiles could predict IDH in this high-risk population.

Methods: This prospective cohort study included 100 critically ill adults undergoing IHD for AKI. All patients underwent pre-dialysis echocardiography and lung/inferior vena cava (IVC) ultrasound to assess stroke volume, cardiac output, B-lines, and IVC collapsibility index (IVC-CI). Patients were divided into two groups based on the presence or absence of IDH.

Results: IDH occurred in 35% of patients. Significant predictors of IDH included lower systolic blood pressure (124.86 ±16.02 vs. 139.92 ±22.8 mmHg, P < 0.001), higher IVC-CI [51% (13–58) vs. 27.38% (13–60), P < 0.001], sepsis (88.6% vs. 70.8%, P = 0.044), and elevated potassium (5.17 ±1.34 vs. 4.62 ±0.87 mmol/L, P = 0.015). Multivariate analysis identified IVC-CI (OR = 1.097, P < 0.001) and SBP (OR = 0.942, P = 0.001) as independent predictors. IVC-CI >49.5% predicted IDH with 68.6% sensitivity and 87.7% specificity (AUC = 0.757, 95% CI: 0.652–0.862).

Conclusion: Ultrasound-derived IVC-CI is a valuable, noninvasive tool for predicting IDH in critically ill patients receiving IHD. Incorporating sonographic profiles into routine pre-dialysis evaluation may enhance risk stratification and improve dialysis safety.

Introduction

Hemodynamic stability is a pivotal determinant of dialysis efficacy and patient outcomes in the management of acute kidney injury (AKI), particularly among critically ill individuals requiring renal replacement therapy (RRT). Intermittent hemodialysis (IHD) remains a widely used modality in intensive care units (ICUs), yet it is frequently associated with hemodynamic instability [1-2]. This complication, known as intradialytic hypotension (IDH), significantly limits the tolerability and effectiveness of IHD in this vulnerable population. According to the Kidney Disease Outcomes Quality Initiative (KDOQI), IDH is defined as a ≥20 mmHg drop in systolic blood pressure or a >10 mmHg drop in mean arterial pressure accompanied by related clinical symptoms [3]. The pathophysiology of

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hemodynamic instability during RRT is multifactorial and complex. It often involves a combination of decreased cardiac output, distributive shock, and impaired autonomic compensatory responses, such as sympathetic activation and vascular tone regulation. These physiologic derangements are especially pronounced in critically ill patients, making them more susceptible to sudden drops in blood pressure during dialysis sessions. Moreover, IDH has been associated with adverse clinical outcomes, including increased mortality, prolonged hospital stays, and reduced renal recovery [2].

Both patient-related factors (e.g., cardiac dysfunction, hypovolemia, autonomic dysregulation) and procedure-related elements (e.g., ultrafiltration rate, dialysate composition and temperature, changes in plasma osmolality) contribute to the development of IDH. The accurate assessment of fluid status is crucial for optimizing dialysis prescriptions and minimizing the risk of hypotensive events [4]. Traditional clinical indicators, however, may be insufficient or misleading in the ICU setting. In this context, bedside ultrasound techniques—particularly transthoracic lung ultrasound and inferior vena cava (IVC) diameter measurement—have emerged as valuable, noninvasive tools for evaluating pulmonary congestion and intravascular volume status. Lung ultrasound, through the quantification of B-lines, provides a reliable estimate of extravascular lung water, while IVC indices offer insight into central venous pressure and preload conditions [5]. The primary aim of this study is to determine whether distinct pre-dialytic cardiopulmonary profiles, as assessed by echocardiography and ultrasound, can predict the occurrence of IDH in critically ill patients undergoing IHD. The secondary aim is to identify clinical and sonographic risk factors associated with hemodynamic instability in this population, which may contribute to prolonged hospitalization and increased in-hospital mortality.

Methods

Study design and setting

This prospective cohort study was conducted at the Intensive Care Units of Misr University for Science and Technology over a 13-month period, from May 2021 to June 2022. A total of 100 critically ill adult patients undergoing IHD for AKI were enrolled. The study aimed to evaluate the predictive role of ultrasound-based cardiopulmonary profiles in identifying patients at risk of developing IDH.

This study was conducted in accordance with the ethical standards outlined in the Declaration of Helsinki. Ethical approval was obtained from the Ethical Committee of the Faculty of Medicine, Cairo University (Code: MD-118-2021). All participants were informed about the study objectives and procedures, and written informed consent was obtained prior to enrollment. Confidentiality and anonymity were ensured throughout the study.

Eligibility Criteria

Eligible patients were adults aged over 18 years with AKI stage 3, as defined by the Kidney Disease: Improving Global Outcomes (KDIGO) guidelines [6], and requiring IHD during their ICU stay. Patients were excluded if they had significant left ventricular systolic dysfunction (ejection fraction ≤40%), severe valvular heart disease, COPD, increased intra-abdominal pressure, inadequate transthoracic ultrasound windows, or declined participation.

Study procedures

All included patients underwent comprehensive baseline evaluation and daily monitoring during their ICU admission. Clinical data collection included detailed medical history focusing on age, sex, comorbidities (e.g., hypertension, diabetes, ischemic heart disease, and chronic kidney disease), and vital signs such as blood pressure, heart rate, respiratory rate, and urine output. Standard laboratory investigations (CBC, serum creatinine, urea, sodium, potassium, and arterial blood gases) were performed upon ICU admission and monitored throughout the study. Electrocardiograms (ECG) were recorded on admission and repeated every 24 hours to detect any rhythm abnormalities or ischemic changes. All patients underwent echocardiographic and ultrasonographic assessment prior to dialysis.

Echocardiographic Assessment

Transthoracic echocardiography (TTE) was performed to assess hemodynamic parameters. Left ventricular outflow tract (LVOT) diameter was measured in the parasternal long axis view at mid-systole near the aortic annulus [7]. The LVOT velocity time integral (VTI) was obtained from the apical five-chamber view using pulsed-wave Doppler. Stroke volume was calculated using the formula: Stroke Volume (SV) = LVOT area × VTI, where LVOT area = $\pi \times (\text{LVOT diameter}/2)^2$.

Cardiac output (CO) and cardiac index (CI) were subsequently derived.

IVC Ultrasound

IVC diameter and collapsibility index were measured using a 2–5 MHz convex probe placed in the subxiphoid position with the liver as an acoustic window. M-mode was used to record maximal IVC diameter during expiration and minimal diameter during inspiration. The IVC-CI was calculated as (IVCmax − IVCmin) / IVCmax [8].

Lung Ultrasound (LUS)

LUS was performed using a low-frequency curvilinear probe (3–6 MHz) in accordance with the BLUE protocol. Three key examination points per hemithorax (upper anterior, lower anterior, and PLAPS) were assessed in supine and/or semi-recumbent positions [8]. B-lines were defined as vertical, hyperechoic reverberation artifacts originating from the pleural line, extending to the bottom of the screen, and moving synchronously with respiration [9]. The presence of ≥3 B-lines in a single intercostal space was considered indicative of pulmonary congestion.

Dialysis procedure and group classification

All patients underwent IHD as per institutional protocols. Dialysis was initiated in hemodynamically stable patients (MAP ≥65 mmHg) who were not on vasopressors or on low-dose norepinephrine (≤0.3 μg/kg/min) for at least 6 hours before initiation. Dialysis indications included volume overload, severe metabolic acidosis, hyperkalemia, and uremic complications.

Based on their intradialytic hemodynamic response, patients were categorized into two groups: Group A: patients who developed hypotension leading to interruption of dialysis, and Group B: patients who tolerated dialysis without hypotensive episodes

Outcomes

The primary outcome of this study was the occurrence of IDH, defined as a symptomatic drop in SBP ≥20 mmHg or MAP ≥10 mmHg leading to interruption of the dialysis session. Secondary outcomes included identification of pre-dialytic ultrasound-based cardiopulmonary profiles (e.g., IVC-CI, B-line score, LVOT VTI) associated with increased risk of IDH, as well as the impact of IDH on dialysis completion, ICU length of stay, and in-hospital mortality.

Statistical methods

Data were coded and analyzed using IBM SPSS Statistics version 28 (IBM Corp., Armonk, NY, USA). Quantitative variables were summarized as mean ± standard deviation, while categorical variables were presented as frequencies and percentages. Group comparisons for normally distributed quantitative variables were conducted using the independent samples t-test, whereas the Mann–Whitney U test was applied for non-normally distributed data. Categorical variables were compared using the chi-square (χ^2) test; however, when expected cell counts were below 5, Fisher's exact test was used. To identify independent predictors of IDH, a multivariate logistic regression analysis was performed, and results were presented as OR with 95% CI. A ROC curve analysis was also conducted to evaluate the predictive performance of significant continuous variables, including the IVC-CI, and to calculate the AUC. A P value < 0.05 was considered statistically significant.

Results

Patients who developed IDH had a significantly lower prevalence of ischemic heart disease compared to those without IDH (28.6% vs. 50.8%, P = 0.032). In contrast, sepsis was significantly more common among patients with IDH than those without (88.6% vs. 70.8%, P = 0.044). There were no significant differences between the groups regarding age (P = 0.102), sex (P = 0.897), hypertension (P = 0.765), diabetes mellitus (P = 0.695), or chronic kidney disease (P = 0.334) (Table 1).

Patients with IDH exhibited significantly lower systolic blood pressure compared to those without IDH (124.86 ±16.02 vs. 139.92 ±22.8 mmHg, P < 0.001) and significantly lower mean arterial pressure (88.83 ±12.93 vs. 97.63 ±16.38 mmHg, P = 0.007). Additionally, pre-hemodialysis low-dose vasopressor use was significantly more common in the IDH group (42.9%) than in the non-IDH group (0%) (P < 0.001). Other variables, including diastolic blood pressure (P = 0.058), pulse (P = 0.241), respiratory rate (P = 0.199), temperature (P = 0.073), urine output (P = 0.115), and mechanical ventilation prior to dialysis (P = 0.105), showed no statistically significant differences between the groups (Table 2). Patients who developed IDH had significantly higher total leukocyte counts compared to those without IDH [14.4 (5.5–32.7) vs. 13 (4.5–38) $\times 10^3/\mu L$, P = 0.044], as well as significantly lower pH levels (7.28 ±0.09 vs. 7.32 ±0.07, P = 0.038), indicating greater acidosis. Additionally, serum potassium levels were significantly higher in the IDH group (5.17 ±1.34 vs. 4.62 ±0.87 mmol/L, P = 0.015). Other laboratory parameters, including hemoglobin (P = 0.78), platelet count (P = 0.30), PCO_2 (P = 0.634), PO_2 (P = 0.871), bicarbonate (P = 0.145), urea (P = 0.313), creatinine (P = 0.414), and sodium (P = 0.516), showed no statistically significant differences between groups (Table 3). Patients who developed IDH had a significantly higher inferior vena cava collapsibility index compared to those without IDH [51 (13–58)% vs. 27.38 (13–60)%, P < 0.001], indicating lower intravascular volume and greater fluid responsiveness. Other ultrasound and echocardiographic parameters, including stroke volume (P = 0.533), cardiac index (P = 0.948), cardiac output (P = 0.837), and presence of B-lines (P = 0.916), did not show significant differences between the groups (Table 4). The IDH group experienced significantly more complications than the non-IDH group. All patients with IDH required vasopressors during or after dialysis (100% vs. 0%, P < 0.001), and dialysis interruption occurred exclusively in this group (100% vs. 0%, P < 0.001). Mechanical ventilation during ICU stay was significantly more frequent among IDH patients (31.4% vs. 4.6%, P <

0.001). Furthermore, in-hospital mortality was markedly higher in the IDH group compared to the non-IDH group (20% vs. 1.5%, P = 0.002) (Table 5).

Multivariate logistic regression analysis revealed that IHD was independently associated with a lower risk of developing IDH (OR = 0.252, 95% CI: 0.082–0.771, P = 0.016), while lower SBP (OR = 0.942, 95% CI: 0.91–0.976, P = 0.001) and higher IVC-CI (OR = 1.097, 95% CI: 1.048–1.149, P < 0.001) were significant independent predictors of increased IDH risk (Table 6).

Table 1- General characteristics of the studied groups

		Total	IDH		P value
			Yes (n = 35)	No (n = 65)	
Age (years)		65.5 ±7.25	67.11 ±8.3	64.63 ±6.51	0.102
Sex	Females	38 (38)	13 (37.1)	25 (38.5)	0.897
	Males	62 (62)	22 (62.9)	40 (61.5)	
HTN		87 (87)	30 (85.7)	57 (87.7)	0.765
DM		71 (71)	24 (68.6)	47 (72.3)	0.695
CKD		88 (88)	29 (82.9)	59 (90.8)	0.334
IHD		43 (43)	10 (28.6)	33 (50.8)	0.032
Sepsis		77 (77)	31 (88.6)	46 (70.8)	0.044*

Data are presented as mean ± SD, n (%), HTN: hypertenstion, IDH: Intradialytic hypotension, n: number, DM: Diabetes mellitus, CKD: Chronic kidney disease, IHD: Ischemic heart disease, *: Significant P value.

Table 2- Hemodynamics and vitals between the studied groups

	Total	IDH		P value
		Yes (n = 35)	No (n = 65)	
Systole (mmHg)	134.65 ±21.83	124.86 ±16.02	139.92 ±22.8	< 0.001*
Diastole (mmHg)	74.55 ±14.32	70.86 ±12.51	76.54 ±14.92	0.058
MAP (mmHg)	94.55 ±15.77	88.83 ±12.93	97.63 ±16.38	0.007*
Pulse (bpm)	90.99 ±13.24	93.11 ±12.41	89.85 ±13.62	0.241
RR (breaths per minute)	18.34 ±3.92	19.03 ±4.15	17.97 ±3.76	0.199
Temp (°C)	37.4 ±0.51	37.53 ±0.57	37.34 ±0.46	0.073
UOP (mL/hour)				
0	74 (74)	22 (62.9)	52 (80)	0.115
50	24 (24)	12 (34.3)	12 (18.5)	
100	2 (2)	1 (2.9)	1 (1.5)	
Pre-HD VP (Low Dose)	15 (15)	15 (42.9)	0 (0)	< 0.001*
MV predialysis	12 (12)	7 (20)	5 (7.7)	0.105

Data are presented as mean ± SD, n (%), IDH: Intradialytic hypotension, SD: Standard deviation, MAP: Mean arterial pressure, RR: Respiratory rate, Temp: Temperature, UOP: Urine output, Pre-HD VP: Pre-hemodialysis low-dose vasopressor use, MV: Mechanical ventilation, *: Significant P value.

Table 3- Laboratory findings between the studied groups

	Total	IDH		P value
		Yes (n = 35)	No (n = 65)	
HBG (gm/dl)	9.76 ±1.33	9.71 ±1.45	9.79 ±1.27	0.78
TLC (×10³/µL)	13.45 (4.5 - 38)	14.4 (5.5 - 32.7)	13 (4.5 - 38)	0.044*
PLT (×10³/µL)	263 (18 - 739)	259 (18 - 739)	267 (78 - 739)	0.30
PH	7.3 ±0.08	7.28 ±0.09	7.32 ±0.07	0.038*
PCO_2 (mmHg)	33.06 ±6.8	32.62 ±7.25	33.3 ±6.6	0.634
PO_2 (mmHg)	42 (27 - 157)	43 (28 - 96)	41.8 (27 - 157)	0.871
HCO_3 (mmol/L)	16.26 ±3.95	15.47 ±4.15	16.68 ±3.8	0.145
Urea (mg/dL)	195.5 (45 - 438)	210 (45 - 420)	194 (45 - 438)	0.313
Creat (mg/dL)	6.6 (2.8 - 23)	6.7 (2.8 - 18)	6.5 (2.8 - 23)	0.414
Na (mmol/L)	132.89 ±6.46	132.31 ±8.01	133.2 ±5.5	0.516
K (mmol/L)	4.81 ±1.09	5.17 ±1.34	4.62 ±0.87	0.015*

Data are presented as mean ± SD, Median (range), IDH: Intradialytic hypotension, HBG: Hemoglobin, gm/dl: grams per deciliter, TLC: Total leukocyte count, PLT: Platelet count, PH: Potential of hydrogen (acid-base balance), PCO_2: Partial pressure of carbon dioxide, PO_2: Partial pressure of oxygen, HCO_3: Bicarbonate, Creat: Creatinine, Na: Sodium, K: Potassium, *: Significant P value.

Table 4- Ultrasound and echo findings between the studied groups

	Total	IDH		P value
		Yes (n = 35)	No (n = 65)	
IVC-CI (%)	36 (13 - 60)	51 (13 - 58)	27.38 (13 - 60)	< 0.001*

SV (mL)	50.99 (20.35 - 95.35)	49.7 (20.35 - 93.75)	52.23 (28.65 - 95.35)	0.533
CI (L/min/m²)	2.62 (1.05 - 5.62)	2.54 (1.05 - 4.9)	2.62 (1.26 - 5.62)	0.948
COP (L/min)	4.67 (1.9 - 10)	4.66 (1.9 - 10)	4.91 (1.98 - 9.06)	0.837
B-lines	55 (55)	19 (54.3)	36 (55.4)	0.916

IDH: Intradialytic hypotension, IVC-CI: Inferior vena cava collapsibility index, SV: Stroke volume, CI: Cardiac index, COP: Cardiac output, *: Significant P value.

Table 5- Complications between the studied groups

	Total	IDH		P value
		Yes (n = 35)	No (n = 65)	
Vasopressors need	35 (35)	35 (100)	0 (0)	< 0.001*
MV during ICU stay	14 (14)	11 (31.4)	3 (4.6)	< 0.001*
Dialysis interruption	35 (35)	35 (100)	0 (0)	< 0.001*
Mortality	8 (8)	7 (20)	1 (1.5)	0.002*

Date are presented as n (%), IDH: Intradialytic hypotension, MV: Mechanical ventilation, *: Significant P value.

Table 6- Multivariant logistic regression analysis to detect independent predictors of IDH

	OR (95% CI)	P value
IHD	0.252 (0.082 - 0.771)	0.016*
Sepsis	1.441 (0.32 - 6.496)	0.635
Systole (mmHg)	0.942 (0.91 - 0.976)	0.001*
PH	0.004 (0 - 2.687)	0.096
IVC-CI (%)	1.097 (1.048 - 1.149)	< 0.001*

IDH: Intradialytic hypotension, OR: Odds ratio, CI: Confidence interval, IHD: Ischemic heart disease, PH: Potential of hydrogen, IVC-CI: Inferior vena cava collapsibility index, *: Significant P value.

ROC curve analysis was performed for the IVC-CI to predict IDH. It revealed a significant AUC of 0.757 with a 95% confidence interval ranging from 0.652 to 0.862, suggesting a good ability to predict IDH. The best cutoff value was >49.5%, at which the sensitivity was 68.6% and the specificity was 87.7% (Figure 1).

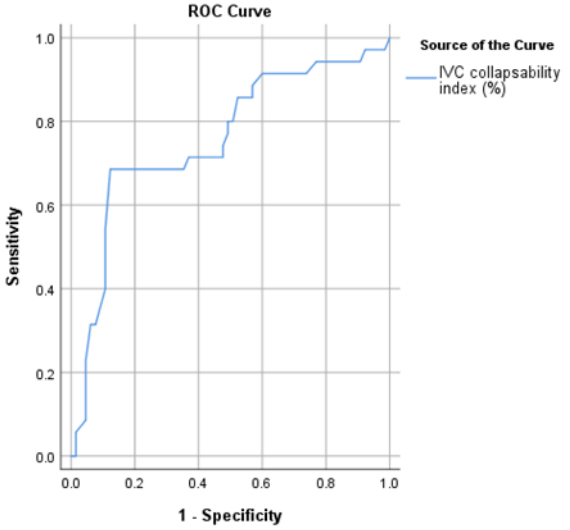

Figure 1- ROC curve for prediction of intradialytic hypotension using IVC-CI

Discussion

In this prospective cohort study of critically ill patients undergoing IHD, we investigated clinical and ultrasound-based predictors of IDH. Our findings revealed that certain demographic and clinical characteristics, as well as specific pre-dialytic parameters, were associated with increased risk of IDH. Most patients in our study were male, with a mean age of 65.5 years. This distribution was consistent between the IDH and non-IDH groups, and no significant differences were observed in terms of age or sex. Similarly, hypertension, diabetes mellitus, and chronic kidney disease were common comorbidities across the cohort, but only ischemic heart disease showed a significant inverse association with IDH. These findings align with those reported by Allinovi et al. [10] and da Hora et al. [11], who also documented predominantly male cohorts with similar age profiles and comorbidity patterns.

Vital signs prior to dialysis were strongly associated with the development of IDH. Specifically, SBP and MAP were significantly lower in patients who experienced hypotensive episodes. The mean systolic blood pressure in the IDH group was 124.86 mmHg versus 139.92 mmHg in the non-IDH group, while MAP was 88.83 mmHg versus 97.63 mmHg (P = 0.007). These findings are in agreement with da Hora et al. [11] and Allinovi et al. [10], who also identified lower pre-dialytic BP as a reliable predictor of IDH. On the other hand, diastolic blood pressure, heart rate, respiratory rate, and temperature did not significantly differ between the groups, consistent with findings from Feng et al. [12], where baseline hemodynamic parameters such as SBP and MAP showed variable association with IDH.

With regard to perfusion markers, urine output was not significantly different between the IDH and non-IDH groups in our study. Most patients (74%) were anuric. This aligns with the findings of Allinovi et al. [10], who

reported no significant difference in oligo-anuria status between groups. However, our results differ from Kim et al. [13], who identified low urine output as a significant predictor of IDH. This discrepancy may reflect differences in fluid management practices or patient selection criteria.

The use of vasopressors prior to dialysis was significantly more common among patients who developed IDH (42.9%) compared to none in the non-IDH group. This finding emphasizes the vulnerability of hemodynamically unstable patients to dialysis-induced circulatory shifts. Similar observations were reported by da Hora et al. [11] and Kim et al. [13], who both found a strong association between vasopressor use and IDH. These results underscore the need for cautious fluid removal strategies and continuous hemodynamic monitoring in this subgroup.

MV was also a significant risk factor for IDH, with 20% of ventilated patients in our cohort experiencing hypotensive episodes. This finding is in line with previous studies by da Hora et al. [11] and Kim et al. [13], both of which highlighted impaired cardiovascular reflexes and increased susceptibility to volume shifts in mechanically ventilated patients. These patients often lack autonomic compensatory mechanisms, making them prone to sudden drops in blood pressure during dialysis.

Laboratory parameters associated with IDH included elevated total leukocyte count, metabolic acidosis (low pH), and hyperkalemia. Each of these variables was significantly different between groups, pointing to a more inflamed and metabolically unstable profile in patients who developed IDH. These findings agree with the study by Kim et al. [13], who identified similar laboratory risk factors. In contrast, hemoglobin, serum creatinine, sodium, and bicarbonate did not differ significantly, which is consistent with prior work by Aoyama et al. [14] and da Hora et al. [11].

Regarding lung ultrasound, the presence of B-lines—commonly associated with pulmonary congestion—did not show any significant association with IDH in our study ($P = 0.916$). This matches the results of Aoyama et al. [14], who reported no meaningful differences in B-line count between IDH and non-IDH groups. However, our findings differ from those of Allinovi et al. [10], who found a greater frequency of IDH among patients with fewer B-lines. These contrasting results may reflect differences in the timing of ultrasound assessment or operator variability in LUS interpretation.

Among echocardiographic parameters, only the IVC-CI was significantly associated with IDH. Patients with an IVC-CI ≥40% were more likely to experience hypotension during dialysis, supporting the role of IVC-CI as a non-invasive predictor of volume responsiveness. Conversely, stroke volume, cardiac index, and cardiac output did not differ significantly between groups, consistent with the findings of Feng et al. [12] and da Hora et al. [11]. These results reinforce the utility of IVC-based volume assessment over complex echocardiographic measurements in this setting.

As expected, IDH was associated with several adverse outcomes in our cohort. All patients with IDH required vasopressors and experienced dialysis session interruption. Moreover, mechanical ventilation was more common among these patients, and the mortality rate was significantly higher in the IDH group (20% vs. 1.5%). These observations align with the findings of Kim et al. [13], who reported higher mortality among IDH patients, and da Hora et al. [11], who attributed increased 28-day mortality to intradialytic circulatory instability.

Finally, our ROC curve analysis confirmed the predictive utility of key parameters. Systolic blood pressure had an AUC of 0.696, while MAP had an AUC of 0.666. The IVC-CI demonstrated an AUC of 0.789, with a cutoff point >49.5%, yielding 68.6% sensitivity and 87.7% specificity. These values are consistent with Kora et al. [15], who reported an AUC of 0.79 for IVC-CI and recommended it as the most accurate non-invasive predictor of IDH.

This study has several limitations that should be considered when interpreting the results. First, it was conducted at a single center with a relatively small sample size, which may limit the generalizability of the findings to broader ICU populations. Second, the observational nature of the study precludes establishing causal relationships between the identified predictors and intradialytic hypotension. Third, some variables—such as fluid balance, vasopressor dose titrations, and echocardiographic measurements—may have been influenced by inter-operator variability despite standardized protocols. Lastly, while we utilized bedside ultrasound to assess cardiopulmonary profiles, we did not account for dynamic changes in these parameters during dialysis, which may have provided additional insights into hemodynamic tolerance.

Conclusion

Ultrasound-derived IVC-CI is a valuable, noninvasive tool for predicting IDH in critically ill patients receiving IHD. Incorporating sonographic profiles into routine pre-dialysis evaluation may enhance risk stratification and improve dialysis safety.

References

[1] Villa G, Husain-Syed F, Saitta T, Degl'Innocenti D, Barbani F, Resta M, et al. Hemodynamic Instability during Acute Kidney Injury and Acute Renal Replacement Therapy: Pathophysiology and Clinical Implications. Blood Purif. 2021;50(6):729-39.

[2] Douvris A, Zeid K, Hiremath S, Bagshaw SM, Wald R, Beaubien-Souligny W, et al. Mechanisms for hemodynamic instability related to renal replacement therapy: a narrative review. Intensive Care Med. 2019;45(10):1333-46.
[3] Kanbay M, Ertuglu LA, Afsar B, Ozdogan E, Siriopol D, Covic A, et al. An update review of intradialytic hypotension: concept, risk factors, clinical implications and management. Clin Kidney J. 2020;13(6):981-93.
[4] Hamrahian SM, Vilayet S, Herberth J, Fülöp T. Prevention of Intradialytic Hypotension in Hemodialysis Patients: Current Challenges and Future Prospects. Int J Nephrol Renovasc Dis. 2023;16:173-81.
[5] Kearney D, Reisinger N, Lohani S. Integrative Volume Status Assessment. Pocus j. 2022;7(Kidney):65-77.
[6] Levin A, Stevens PE, Bilous RW, Coresh J, De Francisco AL, De Jong PE, et al. Kidney Disease: Improving Global Outcomes (KDIGO) CKD Work Group. KDIGO 2012 clinical practice guideline for the evaluation and management of chronic kidney disease. Kidney international supplements. 2013;3(1):1-150.
[7] Schuster AH, Nanda NC. Doppler echocardiographic measurement of cardiac output: comparison with a non-golden standard. Am J Cardiol. 1984;53(1):257-9.
[8] Vaish H, Kumar V, Anand R, Chhapola V, Kanwal SK. The Correlation Between Inferior Vena Cava Diameter Measured by Ultrasonography and Central Venous Pressure. Indian J Pediatr. 2017;84(10):757-62.
[9] Lichtenstein D, Mezière G, Biderman P, Gepner A. The comet-tail artifact: an ultrasound sign ruling out pneumothorax. Intensive Care Med. 1999;25(4):383-8.
[10] Allinovi M, Palazzini G, Lugli G, Gianassi I, Dallari L, Laudicina S, et al. Pre-Dialysis B-Line Quantification at Lung Ultrasound Is a Useful Method for Evaluating the Dry Weight and Predicting the Risk of Intradialytic Hypotension. Diagnostics (Basel). 2022;12(12).
[11] da Hora Passos R, Caldas J, Ramos JGR, dos Santos Galvão de Melo EB, Ribeiro MPD, Alves MFC, et al. Ultrasound-based clinical profiles for predicting the risk of intradialytic hypotension in critically ill patients on intermittent dialysis: a prospective observational study. Critical Care. 2019;23(1):389.
[12] Feng Y, Zou Y, Zheng Y, Levin NW, Wang L. The value of non-invasive measurement of cardiac output and total peripheral resistance to categorize significant changes of intradialytic blood pressure: a prospective study. BMC Nephrol. 2018;19(1):310.
[13] Kim SG, Yun D, Lee J, Kim YC, Kim DK, Oh KH, et al. Impact of intradialytic hypotension on mortality following the transition from continuous renal replacement therapy to intermittent hemodialysis. Acute Crit Care. 2023;38(1):86-94.
[14] Aoyama Y, Kajimoto K, Hori M, Otsubo S, Nitta K. Predicting factors of intradialytic hypotension in the last half of hemodialysis therapy. Renal Replacement Therapy. 2021;7(1):18.
[15] Kora M, Tawfeek A, El-Zorkany K, Abd El-Mohsen A. The relationship between hypoalbuminemia and intradialytic hypotention in hemodialysis patients. Menoufia Med J. 2020;33(1):110.