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Role of inferior vena cava assessment in volume management in acute kidney injury patients

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Abstract

Background Intravascular volume assessment is critical to guiding volume management in patients with acute kidney injury (AKI). This study aimed to compare the impact of using inferior vena cava (IVC) diameter and collapsibility index (IVC-CI) measurements versus clinical assessment on the management of volume status and improvement of renal function in patients with AKI.

Methods This prospective comparative study included 88 patients with AKI or AKI on top of chronic kidney disease (CKD) who were randomly allocated into two groups: In group 1, volume status was managed according to IVC assessment, while in group 2, volume status was managed according to clinical assessment. In addition, group 1 patients were assessed clinically and compared with IVC measurements in the same group.

Results There was moderate agreement between the IVC and clinical methods in diagnosing 86.4% of patients ($P < 0.001$) within group 1. The percentage of patients with edema increased post-treatment in group 2. There was no statistically significant difference between the two groups regarding renal recovery and survival.

Conclusion Bedside ultrasonographic IVC assessment is a non-invasive method that can facilitate volume management in AKI patients, helping to administer fluids more wisely without unintended excess fluid administration.

Keywords Acute kidney injury, Volume assessment, Inferior vena cava diameter, Collapsibility index

Background

AKI causes a sudden decrease or loss of renal function, which results in the retention of waste products, electrolyte imbalances, and changes in volume status. The timing and type of renal support, proper volume control, and the management of nephrotoxic drugs all pose challenges in the treatment of AKI [1]. Fluid therapy has proven to be crucial in the management of AKI. Both hypovolemia and excess fluid administration have been found to negatively impact patient outcomes and to be associated with higher rates of morbidity and mortality. Numerous methods, ranging from history and clinical assessment up to invasive measurements, have been proposed to aid in the assessment of the volume status of the patient. The use of history, clinical examination, and laboratory tests in the assessment of intravascular volume status and volume responsiveness has low sensitivity and specificity [2].

Accurate assessment of intravascular volume is vital in guiding the fluid management of patients with acute or chronic kidney diseases, especially those with multiple comorbidities necessitating hospitalization or intensive care since these patients are predominantly not in a stable condition and may have a mismatch between extravascular and intravascular volume or between intravascular volume and blood pressure. This category of patients may exhibit extravascular volume overload signs such as

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pulmonary edema and swelling of the lower extremities, yet they may experience intravascular volume depletion, which results in decreased effective circulatory volume with reduced renal perfusion, low fractional excretion of sodium (FeNa), and elevated blood urea nitrogen (BUN), creatinine, and BUN/creatinine [3].

Mahrous and his colleagues stated that the IVC is a vessel with high capacity that can distend and collapse. Therefore, when the volume is depleted, the IVC will collapse and have a reduced diameter. On the contrary, IVC diameter increases, and its collapsibility decreases in the presence of hypervolemia. The ability of the IVC to collapse or distend in response to changes in volume status can be easily detected by ultrasound. Also, IVC ultrasonography results can completely rule out the possibility of overt intravascular hypo- or hypervolemia in each patient [4]. This study aimed to study the role of the assessment of IVC diameter and collapsibility index (IVC-CI) and its impact on the management of volume status and improvement of renal function in comparison to clinical assessment in AKI patients.

Methods

Study design and setting

This prospective comparative study was conducted in the period between April 2021 and April 2022 in the nephrology and dialysis unit. The study protocol was approved by the institutional review board (IRB Code: MS.20.02.1056), and written informed consent was obtained from all patients.

This study included 88 patients who presented to the department of nephrology with AKI (defined according to KDIGO guidelines [5] as a rise in serum creatinine by 0.3 mg/dl above the baseline within 48 h, an increase in serum creatinine $\geq$ 1.5 times the baseline creatinine within prior 7 days, or a urine output of 0.5 ml/kg/h within 6 h) or AKI on top of CKD. Patients were randomly allocated using the closed envelope method into two groups according to volume status assessment and method of management, where each group included 44 patients. The first group of patients was assessed and managed using IVC diameter and IVC-CI measurement, while the second group included patients who were managed according to clinical assessment of their volume status.

Patients were excluded from the study if they needed hemodialysis or continuous renal replacement therapy (CRRT) after admission; had untreated obstructive uropathy, pulmonary embolism, COPD, increased intra-abdominal pressure, advanced hepatic or cardiac disease, right-sided heart failure, pulmonary hypertension, tricuspid regurgitation, or intra-thoracic disease; were younger than 18 years old; or lacked informed consent.

In group 1, IVC measurements were recorded by a well-trained and experienced ultrasonography nephrologist at the time of admission before fluid or diuretic administration and then daily. Ultrasound was used to visualize the long axis of the IVC at the subcostal window, while patients were in the supine position. The M-mode ultrasonography using the LOGIQ F6 device, GE Healthcare, Waukesha, WI 53188, USA, was used to generate a time-motion record of the IVC diameter, approximately 2 cm caudal to its junction with the right atrium or approximately 1 cm distal to the hepatic vein inlet to the IVC. The maximum (IVC max) and minimum (IVC min) diameters of the IVC (normally 1.5 to 2.5 cm) over a single respiratory cycle were collected [3]. The IVC-CI was calculated as (IVC max − IVC min)/IVC max.

Patients with a small IVC diameter (< 1.5 cm) and an IVC-CI $\geq$ 50% were considered hypovolemic and were given fluid replacement (Fig. 1), while patients with an increased IVC diameter (> 2.5 cm) and an IVC-CI < 20% were considered hypervolemic and were given diuretics. Patients with IVC diameters of 1.5–2.5 and IVC-CI < 50% were considered euvolemic [3]. Additionally, patients in the first group were assessed clinically on admission by another nephrologist who was blinded to the results of the IVC measurements. The results of the clinical assessment were compared with those of the IVC measurements in the first group on admission to check if there was discrepancy between both methods.

The fluid management in group 2 patients was independent of the IVC measurement and depended on the clinical examination. Clinical evaluation of patients included assessment of extra- and intra-vascular volume status, including the history of fluid intake and losses, blood pressure (BP), pulse, respiratory rate, puffy or sunken eyelids, presence of congested neck veins, presence of edema and its grade, presence of lung congestion or not, assessment of skin turgor, assessment of capillary refill time (< 2 s), cold peripheries, passive leg raising test, postural hypotension, urine output, and measurement of central venous pressure if available. Patients were assessed clinically on admission and then daily. Patients who had systolic BP < 100 mmHg, pulse > 90, cold peripheries, capillary refill time > 2 s, and a positive leg raising test were considered hypovolemic and were given fluids, while patients who had edema, either pulmonary or peripheral, with congested neck veins were considered hypervolemic and were given diuretics. Patients who did not show signs of hypo- or hypervolemia were considered euvolemic (Fig. 1). Demographic data and baseline characteristics were recorded for patients in both groups. Laboratory investigations were recorded daily for patients in both groups.

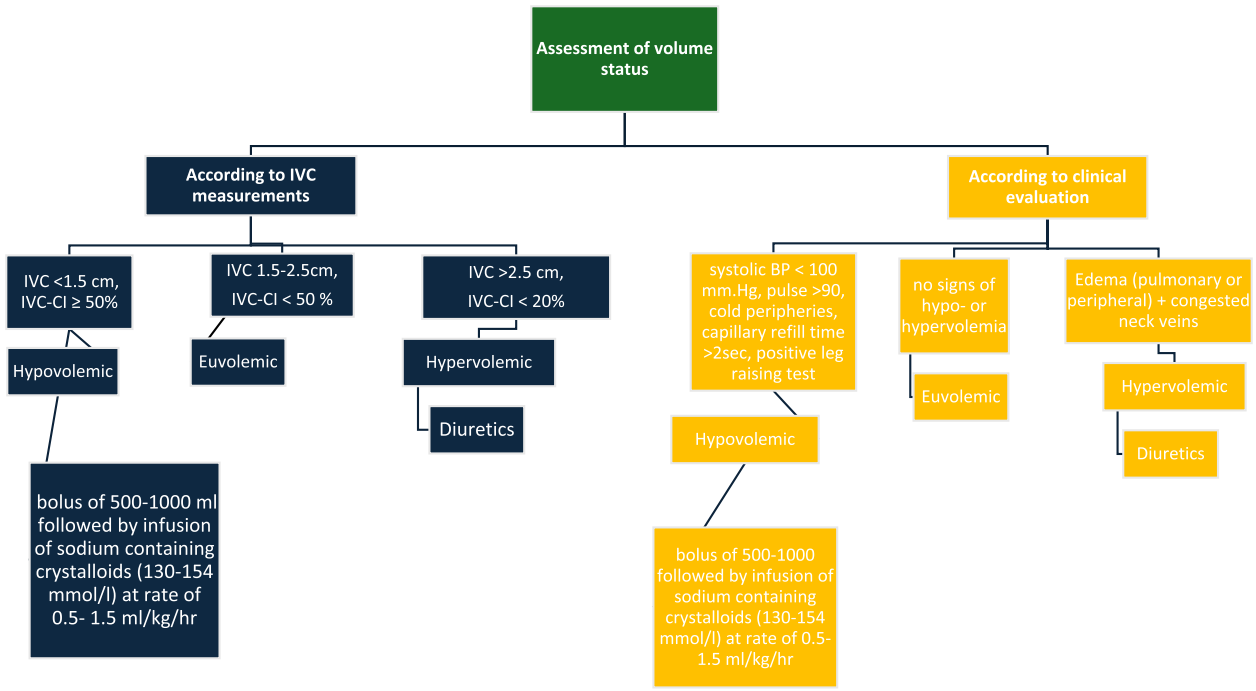

Fig. 1 Algorithm for initial assessment and management of volume status in studied cases

Volume management was individually tailored for each patient based on the assessment of the patient's fluid needs and urine output, rather than infusing a predefined amount of fluid. According to the initial assessment of volume status on admission, hypovolemic patients in both groups were given a bolus of 500–1000 ml, followed by an infusion of sodium-containing crystalloids (130–154 mmol/l) at a rate of 0.5–1.5 ml/kg/hour, considering that many of these patients were oliguric on admission. Hypervolemic patients were given diuretics in the form of furosemide. Doses of furosemide were individualized for each patient. Euvolemic patients were only given maintenance daily fluid needs. Patients were reassessed daily, and fluid intake was re-adjusted according to patients' needs.

The outcomes of the study included renal recovery and patient survival. Complete recovery of renal function was considered if serum creatinine returned to the baseline (normal or value before AKI), while partial recovery was considered if creatinine dropped more than 50% but did not reach the baseline values. Patients whose creatinine levels did not drop more than 50% or increased were considered as non-recovery.

Statistical analysis

Data were entered and analyzed using IBM SPSS (IBM Corp., 2019; IBM SPSS Statistics for Windows, Version 26.0; Armonk, NY: IBM Corp.). Qualitative data were expressed as N (%). Quantitative data were initially tested for normality using Shapiro–Wilk's test, with data being normally distributed if $p > 0.050$. The presence of significant outliers (extreme values) was tested by inspecting box plots. Quantitative data were expressed as mean ± standard deviation (SD) or median and interquartile range (IQR = 75^{th} percentile [Q3]–25^{th} percentile [Q1]).

Chi-Square or Fisher's exact test was used to test the association between two categorical variables. An Independent Sample *t*-test was used to compare normally distributed quantitative data between two groups. The Mann–Whitney *U*-test was used to compare non-normally distributed quantitative data between two groups. The analysis of covariance (ANCOVA) is an extension of the one-way ANOVA to incorporate a covariate variable (pre-treatment value).

The McNemar test was used to determine if there are differences in a dichotomous dependent variable between two related groups. The Wilcoxon signed-rank test was used to determine whether there was a median difference between paired observations. Cohen's kappa (κ) was used to measure the agreement between two methods (IVC vs. clinical) of judgment for categorical scales (volume status). Classification of Cohen's kappa (κ): The strength of agreement was considered poor, fair, moderate, good, or very good if the value of κ was < 0.20, 0.21–0.40, 0.41–0.60, 0.61–0.80, or 0.81–1.00, respectively. For any of the

tests used, results were considered statistically significant if the p value was ≤ 0.050.

Results

The baseline clinico-demographic and laboratory data of the two groups are shown in Table 1. All patients in both groups were classified as stage 3 according to KDIGO staging of AKI (creatinine 3 times the baseline or > 4 mg/dl on initiation of the study). Table 2 shows the results of Cohen's kappa test, which was used to determine if

Table 2 Agreement between two methods of judgment (IVC vs clinical) on volume status of patients in group 1

Volume status by IVC	Volume status clinically		Kappa	*p* Value
	Hypovolemic	Hypervolemic		
Hypovolemic	34	4	0.548	**< 0.001**
Euvolemic	1	1		
Hypervolemic	0	4		

Data is *N*

Table 1 Baseline clinico-demographic and laboratory data of the two groups

Characteristic	Group 1 (no. 44)	Group 2 (no. 44)	Total	*P* value
Age (years)	60 (19—79)	58 (19 – 77)	60 (19–79)	0.977$^{\$}$
Sex: Male	22 (50%)	20 (45.5%)	42 (47.7%)	0.670*
Female	22 (50%)	24 (54.5%)	46 (52.3%)	
BMI (kg/m^2)	27.4 ± 3.7	28.8 ± 3.8	27.9 ± 3.8	0.119$^{@}$
Diabetes	22 (50%)	24 (54.5%)	46 (52.3%)	0.269*
Hypertension	30 (68.1%)	33 (75%)	63 (71.5%)	0.508*
CKD	18 (40.9%)	11 (25%)	29 (32.9%)	0.178*
SBP (mmHg)	129.1 ± 23.1	128.7 ± 22.2	129 ± 22.6	0.935$^{@}$
DBP (mmHg)	76.5 ± 10.5	76.5 ± 11.1	76.5 ± 10.6	0.972$^{@}$
MAP (mmHg)	94.1 ± 13.7	93.9 ± 14.2	94 ± 13.8	0.950$^{@}$
Dyspnea	12 (27.3%)	14 (31.8%)	26 (29.5%)	0.359*
Congested neck veins	4 (9%)	5 (11.3%)	9 (10.2%)	0.284^{f}
- No of pt. with CVP line	9 (20.5%)	10 (22.7%)	19 (21.5%)	0.117*
- CVP measurement (cm)	5 (3–10)	9 (4–24)	9 (3–24)	0.113$^{\$}$
Edema	11 (25%)	11 (25%)	22 (25%)	0.152*
Respiratory rate	21.6 ± .4	23 ± 3.6	22.2 ± 3.5	0.084$^{@}$
Baseline O2 saturation (%)	96 (91–98)	96 (92–99)	96 (91–99)	0.467$^{\$}$
Baseline Urine output (ml/day)	200 (100–440)	200 (300–500)	200 (300–500)	0.726$^{\$}$
Clinically assessed volume status				
- Hypovolemic	35 (79.5%)	34 (77.3%)	69 (78.4%)	0.815*
- Hypervolemic	9 (20.5%)	10 (22.7%)	19 (21.5%)	
Volume status by IVC (group 1)				
-Hypovolemic	38 (86.4%)			
-Euvolemic	2 (4.5%)			
- Hypervolemic	4 (9.1%)			
Baseline serum creatinine mg/dl	11.8 (9.4–14)	8.9 (6–12.9)	9 (7–14)	0.432$^{\$}$
Baseline hemoglobin g/dl	9.8 (8.8–11)	9.5 (7.7–10.9)	9.6 (5.6–15)	0.294$^{\$}$
Baseline hematocrit	36 (34–40)	36 (34–40)	36 (30–45)	0.852$^{\$}$
Baseline serum albumin g/dl	3.5 (3.2–4)	3.4 (3–3.8)	3.4 (3–4.5)	0.548$^{\$}$
Baseline serum sodium mEq/L	135 (132.5–137)	134 (131–139)	135 (122–151)	0.529$^{\$}$
Baseline serum potassium mEq/L	4 (3.5–7)	3.9 (3.4–5.7)	4 (2.6–7)	0.869$
IV fluid and diuretic therapy				
- IV fluid	38 (86.4%)	34 (77.3%)	72 (82%)	0.315^{f}
-Diuretic	4 (9.1%)	10 (22.7%)	14 (16%)	0.315^{f}
-Diuretics (furosemide) doses in hypervolemic patients (mg/d)	80 (40–240)	80 (40–200)	80 (40–240)	0.215$^{\$}$

Categorical data are *N* (%) and test of significance is Chi-Square test* or Fisher's exact test^{f}. Quantitative data are median (minimum–maximum) and test of significance is Mann–Whitney *U*-test$^{\$}$ or mean ± SD and test of significance is Independent-Samples t-test$^{@}$

there was an agreement between the two methods used for the assessment of the volume status of patients in group 1 (IVC vs. clinical) on admission. There was moderate agreement between the two methods (K = 0.548, $P < 0.001$) in diagnosing 38 (86.4%) patients (34 patients were hypovolemic, whereas 4 patients were hypervolemic). However, there was disagreement between the two methods in the diagnosis of six (13.6%) cases (five patients were considered hypervolemic by the clinical method when the IVC method diagnosed four of them as hypovolemic and one as euvolemic). On the other hand, one patient was considered hypovolemic by the clinical method when the IVC method diagnosed him as euvolemic).

The comparison between IVC diameter and IVC-CI measurements before and after treatment in the IVC group (Table 3) showed a statistically significant normalization of IVC measurements after management towards the euvolemic state ($P < 0.001$). Table 4 showed that on comparing post-treatment data between the two groups, there was a statistically significantly higher post-treatment respiratory rate, serum sodium, and urine output in group 2. Also, nine patients with CVP lines in the first group were studied, and we found statistically significantly higher post-treatment CVP measurements than pre-treatment.

On comparing the changes in different clinical parameters before and after management of the volume status between the two groups, there was a significant improvement in dyspnea in group 1 patients ($P = 0.012$). The proportion of patients with dyspnea in group 2 decreased from 32% pre-treatment to 16% post-treatment ($P = 0.063$). The percentage of patients with edema increased posttreatment in group 2 patients (from 25 to 34%), with nine cases having newly formed edema (Table 5). On studying different outcomes in both groups including renal and survival outcomes, duration of follow-up of cases, and duration of hospital stay, no statistically significant difference was encountered. However, two cases (4.5%) in group 2 did not survive (Table 6).

Table 3 IVC diameter and IVC-CI measurements before and after treatment in IVC group

Statistic	Pre-treatment	Post-treatment	*p* Value
IVC-min	0.575 (0.4125–0.8)	1 (1–1.2)	**< 0.001**
IVC -max	1.25 (0.925–1.6)	1.6 (1.5–1.8)	**< 0.001**
IVC-CI	52.9% (47.5–57.1)	37 (33–40)	**< 0.001**

Test of significance is Wilcoxon signed ranks test

Discussion

To the best of our knowledge, this is the first work to study the management of hospitalized AKI patients using IVC measurement in comparison to clinical assessment of volume status and describe different outcomes in relation to both methods. In our study, there was moderate agreement between the two methods of assessment (IVC versus clinical assessment) in group 1 of patients. The

Table 4 Comparisons of post-treatment data (adjusted for pre-treatment levels) between the two groups

Parameter	Group 1				Group 2				*P* value	Partial η^2
	Unadjusted		Adjusted		Unadjusted		Adjusted			
	Mean	SD	Mean	SE	Mean	SD	Mean	SE		
SBP (mmHg)	129.71	19.514	129.6	2.8	131.61	24.508	131.7	3.6	0.657	0.002
DBP (mmHg)	77.1	9.14	77.11	1.5	79	13	79	1.9	0.435	0.008
MAP (mmHg)	94.6	11.17	94.6	1.78	96.6	15.8	96.6	2.3	0.504	0.006
CVP (cm)	10.78	3.03	10.97	0.94	12.7	2.2	12.5	0.889	0.272	0.075
RR (/min)	19.5	2.22	19.7	0.361	22.1	3.7	21.78	0.47	**0.001**	0.133
O2 SAT (%)	96.6	0.886	96.6	0.182	96.29	1.8	96.27	0.236	0.229	0.018
UOP (ml/day)	2736.5	841.6	2740.5	112.7	3177.4	960.8	3170.8	145.9	**0.022**	0.064
Albumin (g/dl)	3.5	0.497	3.36	0.045	3.077	0.541	3.308	0.059	0.503	0.006
HB (g/dl)	9.537	1.54	9.455	0.144	9.26	1.1	9.399	0.187	0.813	0.001
HCT	36.2	2.76	36.2	0.282	35.68	2.856	35.69	0.365	0.272	0.015
Na (mEq/L)	136.15	3.6	136.134	0.611	139.61	5.64	139.647	0.791	**0.001**	0.134
K (mEq/L)	4.02	0.7	4.1	0.1	4.3	0.9	4.25	0.3	0.265	0.018
Serum creatinine involving CKD (mg/dl)	3.19	2.187	3.224	0.220	2.74	1.74	2.686	0.285	0.140	0.027
Serum creatinine excluding CKD (mg/dl)	2.44	1.75	2.536	0.273	2.4	1.715	2.276	0.317	0.539	0.007

Test of significance is one-way ANCOVA. Partial η^2 is a measure of effect size. Boldface for significant *p*-values

SD standard deviation, *SE* standard error, *SBP* systolic blood pressure, *DBP* diastolic blood pressure, *MAP* mean arterial blood pressure, *CVP* central venous pressure, *RR* respiratory rate, *HR* heart rate, *O2 SAT* oxygen saturation, *UOP* urine output, *HB* hemoglobin, *PLT* platelets, *HCT* hematocrit, *WBCs* white blood cells, *Na* serum sodium

Table 5 Pre-posttreatment changes in categorical data

Group	Parameter	-ve pre/-ve post	-ve pre/ + ve post	+ ve pre/-ve post	+ ve pre/ + ve post	*p* Value
Group 1	Dyspnea	31	1	10	2	**0.012**
	Edema	32	1	6	5	0.727
	Congested neck vein	40	0	2	2	0.500
Group 2	Dyspnea	30	0	7	7	0.063
	Edema	24	9	5	6	1.000
	Congested neck vein	39	0	3	2	0.250

Data is *N*. test of significance is McNemar's test

Table 6 Outcome in the two groups

Outcome	Group 1	Group 2	Total	*p* Value
Renal outcome				
- Complete recovery	33 (75%)	31 (70.5%)	64 (72.7%)	0.755
- Partial recovery	6 (13.6%)	9 (20.4%)	15 (17%)	
- Non-recovery	5 (11.4%)	4 (9.1%)	9 (10.3%)	
Survival outcome				
Survivor	44 (100%)	42 (95.5%)	86 (97.7%)	0.137
Non-survivor (deceased)	0 (0%)	2 (4.5%)	2 (2.3%)	
Duration of follow up till recovery (in days)	5 (3–9)	6 (4–10)	5 (4–9)	0.324[$]
Duration of hospital stay (days)	6 (5–12)	7 (4–13)	7 (4–13)	0.256[$]

Data is *N* (%). The test of significance is Fisher's exact test, quantitative data are median (minimum–maximum), and test of significance is Mann–Whitney *U*-test[$]

two methods agreed on 34 patients diagnosed as hypovolemic and on 4 patients diagnosed as hypervolemic. However, four patients were diagnosed as hypervolemic by the clinical method when the IVC method diagnosed them with decreased intravascular volume; one patient was diagnosed as hypervolemic by the clinical method when the IVC method diagnosed this case as euvolemic; and one patient was diagnosed as hypovolemic by the clinical method when the IVC method diagnosed this case as euvolemic.

In our study, nine patients with CVP lines in the first group were studied, and we found statistically significantly higher post-treatment CVP measurements than pre-treatment. In addition, there were statistically significantly higher IVC measurements and lower IVC-CI levels post-treatment as compared with pre-treatment levels in the first group. This is mostly due to fluid replacement and improved volume depletion. This was also reported by Schefold and his colleagues, who found that IVC diameter and IVC-CI were highly correlated with CVP measurements, and that was statistically significant. This might be of value in avoiding unneeded excess fluid administration in such patients with multiple comorbidities [6].

This also agrees with a prospective cross-sectional study carried out by Thanakitcharu and his colleagues, which included seventy patients. This study suggested that the IVC-CI could offer help in the non-invasive evaluation of intravascular volume in critically ill patients [7]. Furthermore, Shalaby and his colleagues found that CVP and IVC maximal diameter (IVC max) were strongly associated, where low CVP < 10 cm H2O can be anticipated if IVC max ≤ 1.73 cm, with a sensitivity and specificity of 71.4% and 75.6%, respectively [8].

The results of this study also showed that there was a change in the diameter of the IVC and IVC-CI in response to either fluid or diuretic administration, which was statistically significant ($p < 0.001$). These findings agree with Elbaih and Housseini (2018), who reported that an IVC-CI greater than or equal to 50% had 100% specificity and sensitivity in predicting fluid responsiveness. They also reported that bedside ultrasonographic measurement of the IVC, together with clinical signs, may be a strong adjunct in the assessment of volume status in these patients [9]. The results stated by Bortolotti et al. (2018) also support our results, indicating that the CI and the diameter of the IVC assessed during deep inspiration are noninvasive bedside tools for predicting fluid responsiveness in patients with septic shock [10]. In addition, a study by Ismail et al. (2022) showed that CVP had a significant correlation with IVC-CI at different times of measurements following fluid replacement, and the p value was ≤ 0.001 [11].

However, Long et al. conducted a meta-analysis in 2017 that included 17 studies and found that fluctuations in IVC diameter with respiration had a low ability to predict fluid responsiveness, especially in spontaneously breathing patients. When using IVC measurement by ultrasound to make decisions in the fluid management of the patient, clinical findings should be considered. As a predictor of fluid responsiveness, IVC ultrasound has a sensitivity and specificity of 0.63 and 0.73, respectively [12]. In 2020, Orso et al. conducted a meta-analysis of IVC-CI using 20 studies. The sensitivity and specificity were 0.71 and 0.75, respectively. He also stated that fluid responsiveness cannot be accurately predicted using an ultrasonographic assessment of IVC diameter and its variations with respiration [13].

In the current study, we found that recovery of AKI (either complete or partial) occurred in most of the patients in both groups 1 and 2. The results were similar between the two groups, which indicates that the management of fluid balance in AKI patients according to the measurement of IVC was successful, similar to the usual clinical method, and had a good impact on the recovery of renal function. In the present study, we also had a larger number of CKD patients in group 1 (40.9%) than in group 2 (25%), which may have affected the percentage of patients with renal recovery in group 1.

Our results agree with those of Jambeih and his colleagues, who studied 33 patients with AKI to determine the ability of bedside ultrasonographic measurement of the IVC to predict the effect of fluid supplementation on the improvement in renal function. Their study included two groups of patients. Patients in group 1 received fluids in accordance with their US-IVC. In patients in group 2, fluids were given in discordance with their US-IVC measurement. In group 1 patients, there was a significant improvement in creatinine [85% versus 31%] and urine output (0.86×0.54 versus 0.45×0.36 ml/kg/h, $p = 0.03$). They concluded that fluid therapy administration in accordance with IVC measurement was associated with improved renal function [14]. The latter study included only 33 patients in both groups and lacked randomization.

Our findings are also in line with the results of a study carried out by Yepes-Hurtado et al. (2016), which showed that there was an improvement in renal function and a reduction in creatinine after fluid administration in their patients in response to IVC measurements [15]. In agreement with our results is also the study by Elhadidy et al. (2020), who studied 120 patients with liver cirrhosis and AKI and found that 75 patients (62.5%) with prerenal AKI responded to fluid supplementation, and AKI improved in these patients after their intravascular volume status was corrected [16].

Brochard and his colleagues found that fluid loading may be harmful in patients with hypervolemia and decreased renal perfusion caused by low cardiac output, where more fluid loading may cause pulmonary edema and decreased oxygenation. Additionally, excess fluid loading in ICU patients has been shown to be an independent risk factor for the development of AKI. However, fluid supplementation in many hypovolemic patients with prerenal AKI may be beneficial for improving renal perfusion pressure [17].

In our study, we found that the percentage of group 1 patients with dyspnea decreased from 27.3% to 6.8% and that difference was statistically significant ($p = 0.012$). In addition, O2 saturation was found to be statistically significantly higher in patients in group 1 post-treatment than pre-treatment. Also, in our study, we found a statistically significantly higher post-treatment respiratory rate and newly observed edema in group 2 vs. group 1 patients. The latter results indicate that group 2 patients received excessive, unnecessary fluid administration as compared with group 1 patients. This may be explained by a better assessment of volume status guided by IVC measurements in group 1, which led to appropriate fluid administration with the avoidance of hypervolemia and pulmonary edema. In contrast to group 2, which was managed clinically, this might have caused excess fluid administration that led to aggravation of hypervolemia in group 2 patients. To the best of our knowledge, we did not find any other studies discussing these findings in relation to IVC measurement before and after treatment.

Finally, we suggest that AKI patients with decreased IVC diameters and increased IVC-CI, together with patients with normal IVCs, can benefit from fluid supplementation. Patients with increased IVC diameters and decreased IVC-CI may be hypervolemic, and evaluation of their clinical condition is required with the avoidance of excess fluid administration. We think that simple IVC measurements can facilitate clinical decision-making in patients who present with AKI and can help to administer fluids more wisely without unintended excess fluid administration. The use of IVC measurements in the assessment of volume status non-invasively will help to avoid CVP line insertion in many cases. This will help to improve morbidity and reduce mortality.

This study had some limitations. Most of the patients included in this study were hypovolemic, with a lower percentage of hypervolemic patients. Our study included more CKD patients in the IVC group, which may have affected the outcome of this group. We did not compare the methods used for the assessment of volume status in group 1 patients to one of the gold standard methods used for this purpose, so the superiority of the IVC method over the clinical one cannot be confirmed yet.

Long-term follow-up after discharge from our unit was not available. Studying a larger number of patients and long-term post-discharge follow-up is recommended.

Conclusion

Bedside ultrasonographic IVC measurement is a non-invasive method that can facilitate volume management in AKI patients, helping to administer fluids more wisely without unintended excess fluid administration.

Abbreviations

AKI	Acute kidney injury
CKD	Chronic kidney disease
BUN	Blood urea nitrogen
FeNa	Fractional excretion of sodium
IVC	Inferior vena cava
IVC-CI	Inferior vena cava collapsibility index
IVC max	The maximum diameter of the IVC
IVC min	Minimum diameter of the IVC
KDIGO	Kidney disease: improving global outcomes
US-IVC	Ultrasonographic IVC measurement

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Availability of data and materials
The datasets generated and analyzed during the current study are not publicly available due to local university policy but are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate
The study protocol was approved by the Institutional Review Board of Mansoura University (IRB Code: MS.20.02.1056). All patients included in the study, or their caring relatives, were well informed about the study, and a written informed consent was obtained. This work has been carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) for studies involving humans.

Consent for publication
Not applicable. Manuscript does not contain data from any individual person.

Competing interests
The authors declare that no conflict of interest.

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