ORIGINAL ARTICLE

Inferior Vena Cava Collapsibility Index in Correlation with Central Venous Pressure and Cardiac Output in Assessment of Fluid Status in Pre-Renal Acute Kidney Injury in Critically Ill Patients

Mohamed A. Mohamed [a,*], Mohamed A. El Mahallawy [a], Mohamed A. Mohamed [b], Mahmoud H. Hemida [a]

[a] Department of Internal Medicine, Faculty of Medicine for Boys, Al-Azhar University, Cairo, Egypt
[b] Department of Radiology, Faculty of Medicine for Boys, Al-Azhar University, Cairo, Egypt

Abstract

Background: ***Patients with acute renal diseases, especially those with complicated comorbidities needing hospitalization or intensive care, require volume management based on an accurate evaluation of relative intravascular volume.***

Aim of the work: ***To measure the value of inferior vena cava(IVC) measurements measured by point-of-care ultrasound(POCUS), central venous pressure(CVP) measured via central venous catheter and cardiac output(CO) in assessment of intravascular volume status of patients, evaluating the correlation between IVC-collapsibility index(IVC-CI) in correlation to CVP and CO in assessment of volume status of critically ill patients, complementary predictors of the clinical response.***

Methods: ***Fifty male and female patients (all over the age of 18) participated in this prospective cross-sectional observational study. After obtaining consent from patients or their first-degree relatives in the case of unconscious patients, an intrathoracic central venous catheter was placed and inserted to terminate in the superior vena cava. Fluid responders(n=30) and non-fluid responders(n=20) were the two groups of patients.***

Results: ***For the group of non-fluid responders, a positive correlation(P<0.05) was found between CI and CO. For the non-fluid responder group, there was a negative correlation between CI and urine output (UOP) both immediately and after 1 hour, as well as IVC min and max(P<0.05). Within the group of fluid responders, a positive connection was found between CVP and CO, UOP at both the immediate and 1-hour intervals, and IVC minimum and maximum (P<0.05).***

Conclusions: ***In prerenal acute kidney injury in critically ill patients. CI can significantly predict mortality and non-fluid responses, while CVP can significantly predict non-fluid responses.***

Keywords: IVC; CI; CVP; CO; AKI

1. Introduction

Acute kidney injury, or AKI, is characterized by a sudden reduction in kidney function and can be detected by either a drop in urine output (≤0.5 mL/kg/h) within 7 days or an increased serum creatinine level (>0.3 mg/dL in Cr within 48 hours, a >1.5-fold increase with respect to the baseline).[1]

A prevalent disease that was linked to increased morbidity and death was acute renal failure (ARF). While ARF is reported in 3.2% to 9.6% of hospitalizations, the in-hospital death rate was about 20% and in intensive care units, it might exceed 50%. About 2 million individuals die every year from ARF, according to estimates.[2]

In the intensive care unit(ICU), AKI was a prevalent diagnosis, accounting for 13% to 78% of hospitalizations. When managing fluids, it is crucial to accurately determine their status. Hypovolemia and different forms of shock are among the many processes that contribute to its pathogenesis. Additionally, there is evidence that fluid overload in intensive care unit patients is a risk factor for AKI and 28-day mortality in this population.[3]

Accepted 15 June 2025.
Available online 31 July 2025

* Corresponding author at: Internal Medicine, Faculty of Medicine for Boys, Al-Azhar University, Cairo, Egypt.
E-mail address: ebrahimraof78@gmail.com (M. A. Mohamed).

https://doi.org/10.21608/aimj.2025.446648

When extracellular fluid volume decreased, it was known as hypovolemia. It happens when there is a lack of fluid intake relative to the amount of fluid lost by the body. Hypovolemia was effectively treated with intravascular isotonic fluid replenishment.4

The body's compensatory vasoconstrictor reaction to volume loss had no effect on the diameter of the inferior vena cava (IVC). In addition to being noninvasive, simple, and cheap, IVC-CI also allows for the acquisition of the index value with nothing in the way of training.5

Fluid resuscitation in severely sick patients was best guided by echocardiography. By observing the left ventricle, aortic outflow, inferior vena cava, and right ventricle. The prediction and measurement of fluid responsiveness, as well as the assessment of response to intravenous fluid resuscitation, are based on both static measurements and dynamic factors related to heart-lung interactions.6

The supervising physician would use a particular kind of bedside ultrasonographic evaluation known as a point-of-care ultrasound(POCUS). Clinicians have increasingly turned to POCUS in recent years, particularly in critical care and emergency settings.7

In order to assess the intravascular volume status of critically ill patients, this study aimed to measure the value of intravascular volume measurements taken by percutaneous endovascular ultrasound (POCUS), central venous pressure (CVP) measured by central venous catheter, and carbon monoxide (CO). It also evaluated the correlation between intravascular volume status and central venous pressure (CI), as well as CO and CVP, as complementary predictors of clinical response.

2. Patients and methods

Fifty male and female patients(all over the age of 18) participated in this prospective cross-sectional observational study by the use of an intrathoracic central venous catheter, after gaining consent from the patients or their first-degree relatives in the instance of a comatose patient, which was terminated in the superior vena cava. The Ethical Committee at Al-Azhar University Hospitals in Cairo, Egypt, gave its blessing before the research could begin.

Exclusion criteria:

Patients with end-stage renal disease on chronic dialysis, renal or post-renal acute kidney injury (AKI), moderate to severe tricuspid regurgitation, chronic obstructive pulmonary disease (COPD), patients for whom lying flat is not an option, and severely obese patients were not included.

Patients were divided into two groups:fluid responder group(n=30) and non-fluid responder group(n=20).

On the day of admission to the intensive care unit, all patients underwent a thorough history taking, physical examination, and a battery of laboratory tests, including complete blood counts (CBCs), potassium, sodium, calcium, phosphorus, creatinine, daily blood urea nitrogen (BUNs), and arterial blood gases (ABGs). Radiological tests included chest X-rays, abdominal and pelvic ultrasounds to determine the extent of nephropathy and rule out post-renal obstruction, and an acute physiology and chronic health evaluation II (APACHE II) score to predict mortality.

The KDIGO criteria were followed by all patients who developed AKI. This is defined as a rapid decline in kidney function, which can be detected by either a decrease in urine output (≤0.5 mL/kg/h) within 7 days or an elevated serum creatinine level >0.3 mg/dL within 48 hours, which is more than 1.5 times higher than the baseline. The patients underwent an abdominal ultrasound to measure the diameters of the internal jugular veins and CI, followed by a CVP recording while the patient was in a supine position. Baseline echocardiography was used to assess the patient's cardiac output. 2–After 15 minutes, 500 milliliters of normal saline solution were injected intravenously. The identical evaluations were subsequently repeated both immediately and one hour after the delivery of fluids. Patients were categorized as either responders (shown by a 10% rise in CO following volume) or non-responders (shown by an increase of less than 10%, no change, or even a reduction). Two centimeters below the hepatic vein-IVC junction, or around three or four centimeters from the point where the IVC meets the right atrium, was the measurement taken for the IVC diameter. Using the leading-edge approach, the maximum intraventricular diameter (IVC dmax) was determined as the maximum anterior-posterior dimension at the conclusion of expiration, which is the distance from the inner edge to the inner edge of the vessel wall. Furthermore, end-inspiration was used to estimate the minimal IVC diameter (IVCdmin). The percentage equal to [IVC dmax-IVC dmin]/IVC d max multiplied by 100% is the IVC collapsibility index. A new distensibility index (DI)—defined as DI(IVC max-IVC min)/IVC min—measures how the cycle is inverted when mechanical ventilation is used.

The aortic diameter(AoD) was measured at the annulus of the aortic valve. A formula was used to compute the aortic area(AA): AA=πÏ(AoD2/4). We computed the velocity-time integral(VTI) for aortic blood flow based on pulsed Doppler measurements

taken at the aortic annulus in the apical five-chamber view. The stroke volume(SV) and cardiac output(CO) were calculated using the following formulas: SV=VTI×AA and CO=SV×heart rate.

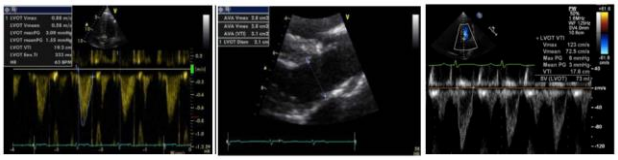

Figure 1. LVOT VTI is determined by taking a 5-chamber apical view, tracing along the edge of the velocity using a pulsed-wave Doppler at the opening of the aortic valve, and measuring the area under the curve.

By measuring the VTI and LVOT diameters at the same location, SV can be computed.

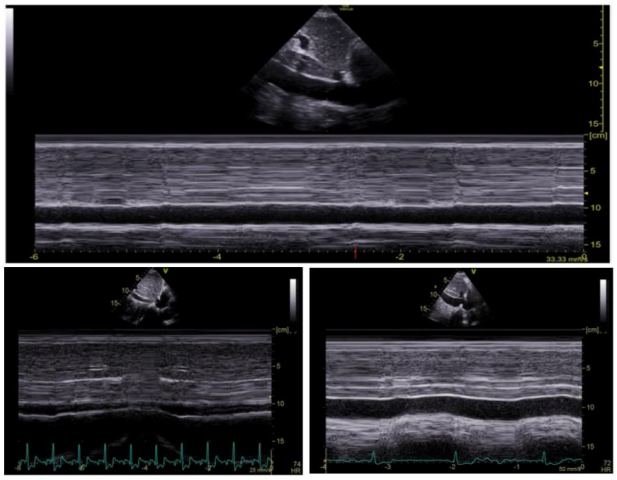

Figure 2. Evaluation of fluid responsiveness using the inferior vena cava's subcostal view.

When the RAP is large, this variance is eliminated. Lack of variance in IVC respiration indicates Fluid Unresponsiveness. FR is accurately predicted by large differences in IVC respiratory variation.

Statistical analysis

IBM's SPSS v26 (Chicago, IL, USA) analyzed data. Unpaired Student's t-test compared the groups' mean and SD quantitative variables. Sometimes Chi-square or Fisher's exact was used to analyze quality variables, such as frequency and percentage. Data was correlated using Pearson product-moment correlation. A ROC curve was used to evaluate diagnostic sensitivity, specificity, PPV, and NPV. Two-tailed P-value <0.05 indicates significance.

3. Results

Table 1. Demographic data, risk factors and UOP of the studied groups.

		FLUID RESPONDER GROUP (N=30)	NON FLUID RESPONDER GROUP (N=20)	P
AGE(YEARS)		51.43±15.95	49.2±16.84	0.637
SEX	Male	17(56.67%)	8(40%)	0.248
	Female	13(43.33%)	12(60%)	
WEIGHT(KG)		74.53±8.7	76.4±7.94	0.446
HEIGHT(M)		1.66±0.08	1.66±0.06	0.981
BMI(KG/M^2)		27.14±3.03	27.86±3.49	0.448
RISK FACTORS	Heart failure	1(3.33%)	2(10%)	0.556
	Stroke	7(23.33%)	6(30%)	0.599
	Dehydration	5(16.67%)	0(0%)	0.074
	Hepatic failure	4(13.33%)	5(25%)	0.454
	HTN	10(33.33%)	8(40%)	0.630
	DM	11(36.67%)	9(45%)	0.556
	Sepsis	11(36.67%)	8(40%)	0.812
UOP	Baseline	3.64±0.52	3.44±0.41	0.154
	Immediately	6.22±1.37	5.48±0.98	0.041*
	After 1h	7.72±1.2	6.81±1.43	0.018*

The data are shown as frequency (%) or mean±SD. *:markedly distinct as P-value<0.05, body mass index(BMI) HTN:high blood pressure, Diabetes mellitus(DM) and urinary output(UOP).

Both groups were similar in terms of age, sex, weight, height, body mass index (BMI), heart failure, stroke, dehydration, hepatic failure, hypertension, diabetes, sepsis, and ulcerative colitis (UC) Table 1.

At baseline, there was no significant difference between the two groups in terms of SBP, DBP, MAP, and HR. However, levels were considerably higher in the fluid responder group both immediately after and after 1 hour compared to the non-fluid responder group (P<0.05) Figure 3.

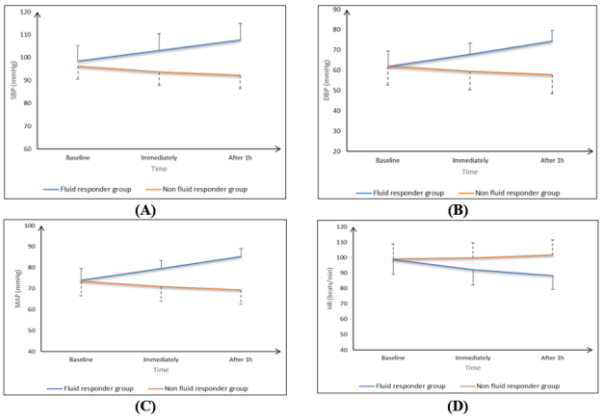

Figure 3. (A)Systolic blood pressure, (B)diastolic blood pressure, (C)mean arterial blood pressure and (D)heart rate of the studied patients.

Table 2. laboratory parameters of the studied groups.

		FLUID RESPONDER GROUP (N=30)	NON FLUID RESPONDER GROUP (N=20)	P
HB(G/DL)		11.3±1.01	11.2±1.05	0.733
WBCS(X10 ^{9}/L)		8.08±1.2	7.62±1.47	0.226
ALT(U/L)		51.47±12.57	50.45±10.61	0.767
AST(U/L)		69.63±13.58	71.3±14.25	0.679
TOTAL BILIRUBIN(MG/DL)		0.89±0.46	1.14±0.55	0.084
CREATININE	Baseline	1.67±0.16	1.77±0.18	0.054
	Immediately	1.41±0.17	1.73±0.18	<0.001*

Hemoglobin is represented as Hb, and white blood cells are represented as WBCs. Data are shown as mean±SD.*:substantially different as P-value≤0.05. Aspartate aminotransferase(AST) and alanine transaminase(ALT).

There was no discernible difference in Hb, WBCs, ALT, AST, or total bilirubin levels between the two groups. At baseline, there was no significant difference in creatinine between the two groups. However, the fluid responder group's creatinine was significantly lower than that of the non-fluid responder group right away(P<0.001) Table 2.

Table 3. Systemic examination, APACHE II, mechanical ventilation and mortality of the studied groups.

		FLUID RESPONDER GROUP (N=30)	NON FLUID RESPONDER GROUP (N=20)	P
COLLAPSIBILITY INDEX	Baseline	0.75±0.04	0.51±0.07	<0.001*
	Immediately	0.73±0.04	0.49±0.07	<0.001*
	After 1h	0.7±0.04	0.47±0.07	<0.001*
CVP(CM H_2O)	Baseline	5.09±0.91	3.61±0.49	<0.001*
	Immediately	6.06±0.39	3.71±0.33	<0.001*
	After 1h	7.33±0.64	3.88±0.67	<0.001*
IVC MIN(CM)	Baseline	8.9±2.93	11.4±2.01	0.002*
	Immediately	11.1±3.01	12.75±2	0.036*
	After 1h	13.93±3.76	14.8±1.91	0.347
IVC MAX(CM)	Baseline	14.43±3.32	16.3±2.13	0.031*
	Immediately	16.83±3.17	19.15±1.9	0.005*
	After 1h	19.13±2.8	20.75±1.83	0.027*
SV(ML)	Baseline	39.37±15.35	41.4±19.38	0.681
	Immediately	49.77±15.4	39.65±19.17	0.045*
	After 1h	51.5±15.68	38.8±19.34	0.014*
CO(L/MIN)	Baseline	4.48±1.3	4.39±1.65	0.827
	Immediately	4.97±1.32	4.04±1.65	0.031*
	After 1h	5.22±1.27	3.59±1.64	<0.001*

Data are presented as mean±SD.*:significantly different as P-value≤0.05. CVP:Central venous pressure, IVC:Inferior vena cava, SV:Stroke volume, CO:Cardiac output, APACHE:Acute physiology and chronic health evaluation.

The fluid responder group had a considerably greater Collapsibility Index(CVP) at baseline, immediately after, and one hour later than the non-fluid responder group(P<0.001). IVC min was considerably lower in the fluid responder group at baseline and shortly after compared to the non-fluid responder group(P<0.05). After an hour, there was no significant difference between the two groups. IVC max was considerably lower in the fluid responder group at baseline, right away, and one hour later than in the non-fluid responder group(P<0.05). At baseline, there was no significant difference in SV, CO, or mechanical ventilation between the two groups. However, after one hour, the fluid responder group had considerably greater levels of these parameters compared to the non-fluid responder group(P<0.05). The fluid responder group had significantly lower mortality and APACHE II scores than the non-fluid responder group(P<0.05) Table 3.

Table 4. Correlation between CI and CVP, CO, UOP at immediately and after 1h and IVC min and max of the studied groups, mechanical and non-mechanical ventilation groups.

			CI
FLUID RESPONDER GROUP	CVP	r	-0.400
		P	0.028*
	CO	r	-0.946
		P	<0.001*
	UOP immediately	r	-0.394
		P	0.03*
	UOP after 1h	r	-0.512
		P	0.003*
NON FLUID RESPONDER GROUP	IVC min	r	-0.692
		P	< 0.001*
	IVC max	r	-0.761
		P	< 0.001*
	CVP	r	-0.002
		P	0.993
	CO	r	0.453
		P	0.045*
	UOP immediately	r	-0.5009
		P	0.024*
	UOP after 1h	r	-0.704
		P	0.005*
	IVC min	r	-0.897
		P	< 0.001*
	IVC max	r	-0.469
		P	0.036*
MECHANICAL VENTILATION GROUP(N=15)	CVP	r	0.146
		P	0.603
NON MECHANICAL VENTILATION GROUP(N=35)	CVP	r	0.176
		P	0.3102

r:Pearson coefficient, *significant p value, CVP:Central venous pressure, IVC:Inferior vena cava, SV:Stroke volume, CO: Cardiac output, CI:Collabsability index, UOP:Urine output.

In the group of non-fluid responders, there was no link seen between CI and CVP. In the fluid responder group, there was a negative connection(P<0.05) between CI and CVP, CO, UOP both immediately and after one hour, as well as IVC min and max. Between the mechanical ventilation group and the non-mechanical ventilation group, there was no link found between CI and CVP. In the non-fluid responder group, CI and CO showed a positive connection(P<0.05). In the non-fluid responder group, there was a negative connection(P<0.05) between CI and UOP both immediately and after one hour, as well as IVC min and max Table 4.

Table 5. Correlation between CVP and CO, UOP at immediately and after 1h and IVC min and max of the studied groups.

			CVP
FLUID RESPONDER GROUP	CO	r	0.495
		P	0.005*
	UOP immediately	r	0.5687
		P	0.001*
	UOP after 1h	r	0.492
		P	0.005*
	IVC min	r	0.766
		P	<0.001*
	IVC max	r	0.616
		P	<0.001*
NON FLUID RESPONDER GROUP	CO	r	-0.456
		P	0.043*
	UOP immediately	r	0.622
		P	0.003*
	UOP after 1h	r	0.579
		P	0.007*
	IVC min	r	0.741
		P	<0.001*
	IVC max	r	0.656
		P	0.001*

r:Pearson coefficient, *significant p value, CVP:Central venous pressure, IVC:Inferior vena cava, SV: Stroke volume, CO:Cardiac output, UOP:Urine output.

In the non-fluid responder group, CVP and CO had a negative connection(P<0.05). In the fluid responder group, there was a significant positive connection(P<0.05) between CVP and CO, UOP immediately and one hour later, and IVC min and max. In the non-fluid responder group, there was a positive connection(P<0.05) between CVP and UOP at the time of the response, one hour later, and IVC min and max Table 5.

Using cut-off values of≤0.59 and 3.8, CI and CVP

may both significantly predict non-fluid responders(P<0.001 and AUC=0.990 and 0.878), with 90% and 75% sensitivity, 93.33% and 676.67% specificity, 90% and 68.2% PPV, and 93.3% and 82.1% NPV. When the cut-off is less than 0.59, CI may accurately predict death(P=0.01 and AUC=0.754) with 83.33% sensitivity, 65.91% specificity, 2.44% PPV, and 0.754% NPV Figure 4.

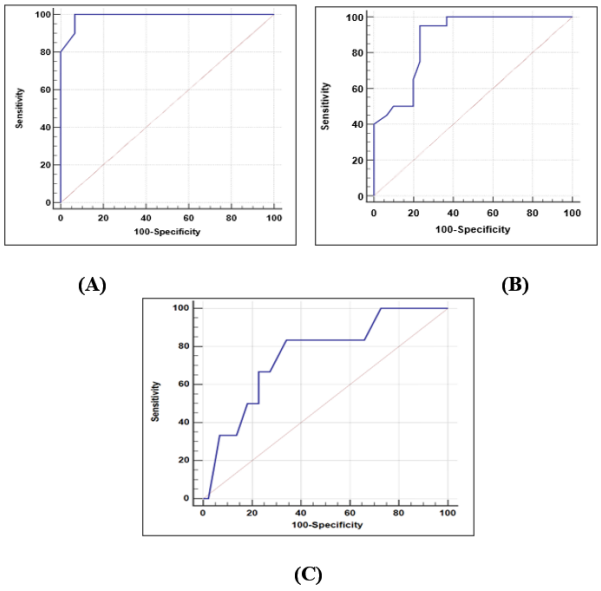

Figure 4. ROC curve of (A)CI, (B)CVP in prediction of Non fluid responder, (C)CI in prediction of mortality

4. Discussion

According to our findings, the urine output UOP in the fluid responder group was considerably higher than that of the non-fluid responder group both immediately and after one hour, with no significant difference between the two groups at baseline.

Jambeih et al.[8] demonstrated that group 1's UOP was noticeably higher than group 2's.

In the current trial, creatinine was much lower in the fluid responder group right away than in the non-fluid responder group, and it was not significantly different between the two groups at baseline.

Jambeih et al.,[8] demonstrated that creatinine clearance(78[+or-] 93% against 8[+or-] 64%, p=0.002) and creatinine[85% versus 31%, p=0.0002.

Al Arnous et al.,[9] demonstrated that with time, the mean IVC collapsibility index dropped statistically considerably.

The fluid responder group in the current study had considerably lower mortality and APACHE II scores than the non-fluid responder group. There was no discernible difference in mechanical ventilation between the two groups. Respiratory fluctuations in patients exhibiting spontaneous ventilation are quite unpredictable. Significant correlations have been observed between hypovolemia and low CVP in critically sick patients who are spontaneously breathing and IVC-CI ≥50%.[10]

In the Fluid responder group in the current experiment, there was a negative connection between CI and CVP. In the group of non-fluid responders, there was no relationship found between CI and CVP.

According to our research, the fluid responder group's CI and CO had a negative association. In the group of non-fluid responders, CI and CO showed a positive connection. The heart pumps more blood via the circulatory system when CO levels rise. This rise may be the result of the injected saline solution's volume expanding, which raises the preload and, in turn, the stroke volume. The blood flow via the SVC rises as CO levels rise.

According to our findings, the Fluid responder group's CVP and CO showed a positive association. In the group of non-fluid responders, there was a negative correlation between CVP and CO.

Between the mechanical ventilation group and the non-mechanical ventilation group in the current study, there was no link found between CI and CVP.

Dodhy,[11] resulted in lower regression coefficients for IVC maximal diameter (r=0.779) and collapsibility index (-0.725) in patients given mechanical ventilation compared to those who breathed normally (r=0.850) and 0.899, respectively.

Our data shows an inverse relationship between the fluid responder group's CI and UOP (substantially). A negative correlation was found between CI and UOP (after) in both the fluid responder and non-fluid responder groups.

Our results indicate that CVP was positively associated with UOP(at the time) in both the fluid responder and non-fluid responder groups. The fluid responder group's CVP and UOP were positively correlated with those of the non-fluid responder group after one hour.

Al Arnous et al.,[9] demonstrated that the IVC collapsibility index was negatively correlated with both CVP and UOP.

For both sets of data, we found a positive relationship between CVP and IVC min. In both groups, CVP was positively correlated with IVC max.

Al Arnous et al.,[9] determined that the IVC collapsibility index was negatively correlated with all CVPs.

According to Dodhy,[11] there was a strong relationship between IVC and CVP measures.

In this study, a negative connection was found between CI and both the minimum and maximum intraventricular volume(IVC) in both

groups.

Our results show that CI can accurately predict non-fluid responders(P<0.001 and AUC=0.990) at a cutoff of 0.59 with a sensitivity of 90%, specificity of 93.33%, PPV of 90%, and NPV of 93.3%. At a cutoff of 0.59, CI was able to predict death with a substantial 83.33% sensitivity, 65.91% specificity, 2.44% PPV, and 0.754% NPV(P=0.01 and AUC=0.754).

Al Arnous et al.,[9] stated that the optimal IVC cutoff for low CVP diagnosis was 28.5% or higher, with a sensitivity of 100%, specificity of 94.7%, PPV of 94.4%, and NPV of 100%(P<0.001 and AUC=0.998)

Shalaby et al.,[12] found that at a cut off of ≤1.73 and >33.42, with sensitivity of 71.40 and 79.80, specificity of 75.60 and 96.60, positive predictive value(PPV) of 90.30 and 97.76, and negative predictive value(NPV) of 46.73 and 66.36, respectively, IVC max. and IVC CI can strongly predict(AUC=0.786 and 0.915, and P<0.001, respectively).

At a cutoff of≤3.8, CVP strongly predicts non-flu responders(P<0.001 and AUC=0.878) with a sensitivity of 75%, specificity of 676.67%, PPV of 68.2%, and NPV of 82.1% in the current study.

Muller et al.,[13] reported that a cut-off of 40% had the best ROC curve for predicting volume responsiveness measured by an increase in echocardiographic CO of at least 15%.

The study's sample size was limited, which was one of its limitations. The research was place in just one location. The outcomes could have been different if the participants hadn't been chosen at random.

4. Conclusion

In prerenal acute kidney injury in critically ill patients. CI can significantly predict non fluid responder and mortality, while CVP can significantly predict non fluid responder.

Disclosure

The authors have no financial interest to declare in relation to the content of this article.

Authorship

All authors have a substantial contribution to the article

Funding

No Funds : Yes

Conflicts of interest

There are no conflicts of interest.

References

1. Abdullah, AA.; Ganda, IJ. The role of urine angiotensin-II(ANG-II) in predicting the occurrence of acute kidney injury(AKI) induced by sepsis in children.Bali Med J.2024;13:715-21.
2. Anga, K.; Makembi, A.; Amisi, É., et al. Morbidity and mortality of acute renal failure in COVID-19 patients in intensive care according to waves/variant: case of the grand hôpital de l'est francilien site de meaux.Open J Intern Med.2024;14:1-14.
3. Wang, N.; Jiang, L.; Zhu, B., et al. Fluid balance and mortality in critically ill patients with acute kidney injury: a multicenter prospective epidemiological study.Crit Care.2015;19:371.
4. Imbachi, LDI.; Ortega, MAM.; Martínez, NVP., et al. Hypovolemic shock Hypovolemic Shock: How Does Lactic Acid Affect the Heart?International Journal of Medical Science and Clinical Research Studies.2024;4:294-6.
5. Cosgun, Z.; Dagistan, E.; Cosgun, M., et al. Can inferior vena cava diameter and collapsibility index be a predictor in detecting preoperative intravascular volume change in pediatric patients?Journal of Cardiovascular Emergencies.2021;7:47-51.
6. Soliman-Aboumarie, H.; Pastore, MC.; Galiatsou, E., et al. Echocardiography in the intensive care unit: An essential tool for diagnosis, monitoring and guiding clinical decision-making.Physiol Int.2021;189:89-96.
7. Rath, K.; Bonomo, J.; Ballman, K. Point-of-care ultrasonography for advanced practice providers: A training initiative.JNP.2022;189:22-45.
8. Jambeih, R.; Keddissi, JI.; Youness, HA. IVC measurements in critically ill patients with acute renal failure.Crit Care Res Pract.2017;2017:35-9.
9. Arnous, A.; Omar, MA.; Dorgham, E., et al. Inferior vena cava collapsibility index versus central venous pressure for assessment of intravascular volume status in injured traumatized patients.EJHM.2022;87:1752-7.
10. Nagdev, AD.; Merchant, RC.; Tirado-Gonzalez, A., et al. Emergency department bedside ultrasonographic measurement of the caval index for noninvasive determination of low central venous pressure.Ann Emerg Med.2010;55:290-5.
11. Dodhy, AA. Inferior vena cava collapsibility index and central venous pressure for fluid assessment in the critically ill patient.J Coll Physicians Surg Pak.2021;31:1273-7.
12. Shalaby, MIM.; Roshdy, HM.; Elmahdy, WM., et al. Correlation between central venous pressure and the diameter of inferior vena cava by using ultrasonography for the assessment of the fluid status in intensive care unit patients.EJHM.2018;72:5375-84.
13. Muller, L.; Bobbia, X.; Toumi, M., et al. Respiratory variations of inferior vena cava diameter to predict fluid responsiveness in spontaneously breathing patients with acute circulatory failure: need for a cautious use.Crit Care.2012;16:188-200.