










Original article

Inferior vena cava ultrasonography could predict acute kidney injury in critically ill patients

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Abstract

Context: Acute kidney injury (AKI) is a complication frequently encountered in critically ill patients and is associated with poorer outcomes. Bedside ultrasonographic assessment (POCUS) has proven to be a valuable tool for noninvasively assessing hemodynamic status. However, few studies evaluate the usefulness of inferior vena cava (IVC) measurements of acute kidney injury management among critically ill patients.

Objective: This study aimed to address this gap in knowledge by evaluating the effectiveness of inferior vena cava measurements and their relationship with acute kidney injury management in critically ill patients.

Keywords: Acute kidney injury, Critically ill patient, Intensive care unit, Inferior vena cava, Ultrasonography.

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Methodology: Patients admitted to the intensive care unit (ICU) were selected and underwent daily bedside ultrasonographic assessment to monitor the inferior vena cava. The collapsibility index (IC VCI [%] = $(D_{\max} - D_{\min}) / D_{\max}$) was calculated, and a cutoff point of <20 % was established to define the absence of hypovolemia (no volume response). B-mode measurements of the transverse axis of the long (Ld), short (Sd) diameter, and inferior vena cava area at the maximum and minimum time were also performed, and calculations were made. As a result, the development of acute kidney injury and its severity were determined according to the KDIGO classification.

Results: The increase in daily cumulative balance was associated with acute kidney injury (620 +1,116 mL vs 115 +816 mL, 16; $P=0.001$), with no differences in the daily amount of liquids administered for both groups (1,841 +1,071 vs 1,602 +1,602; $P=0.1444$). During daily follow-up, in the acute kidney injury group an increase in both maximum (D_{\max}) and minimum (D_{\min}) inferior vena cava diameters was associated with greater acute kidney injury severity according to KDIGO criteria (D_{\max} Coef. 0.187 - D_{\min} Coef. 0.160, $P>|Z|$: 0.008 - 0.006, CI >95 % [0.049 / 0.324] - [0.046 / 0.273]). This association was particularly notable in patients admitted post cardiovascular surgery ($n=14$, Coef. 0.761, $P>|Z|$: 0.005, CI >95 % [0.229 / 1.292]).

Conclusions: Serial longitudinal and transverse axis bedside ultrasonographic assessment measurements of the inferior vena cava can help predict the severity of acute kidney injury in critically ill patients, especially in postoperative cardiovascular surgery patients.

La ultrasonografía de la vena cava inferior podría predecir lesión renal aguda en pacientes críticamente enfermos

Resumen

Contexto: la lesión renal aguda (LRA) es una complicación que se encuentra con frecuencia en pacientes críticamente enfermos y se asocia con peores resultados. La evaluación ultrasonográfica a pie de cama (POCUS) ha demostrado ser una herramienta valiosa para evaluar el estado hemodinámico de forma no invasiva. Existen pocos estudios que evalúen la utilidad de las mediciones de la vena cava inferior (VCI) en relación con la lesión renal aguda en el paciente crítico.

Objetivo: para abordar esta brecha de conocimiento, este estudio tuvo como objetivo evaluar la efectividad de las mediciones de la vena cava inferior y su relación con la lesión renal aguda en pacientes críticos.

Metodología: se seleccionaron pacientes ingresados en la unidad de cuidados intensivos (UCI) y se les realizó monitorización con evaluación ultrasonográfica a pie de cama diaria de la vena cava inferior. Se calculó el índice de colapsabilidad (IC VCI [%] = $(D_{\max} - D_{\min}) / D_{\max}$) y se estableció un punto de corte <20 % para definir la ausencia de hipovolemia (sin respuesta de volumen). También se realizaron mediciones en modo B del eje transversal del diámetro largo (Ld), corto (Sd) y área de la vena cava inferior en el tiempo máximo y mínimo, y se realizaron cálculos. Como resultado, se determinó el desarrollo de lesión renal aguda y su gravedad según la clasificación KDIGO.

Resultados: el aumento del balance acumulado diario se asoció con la lesión renal aguda (620 +1,116 mL vs 115 +816 mL, 16; $p=0.001$), sin diferencias en la cantidad diaria de líquidos administrados para ambos grupos (1,841 +1,071 vs 1,602 +1,602; $p=0.1444$). Durante el seguimiento diario, en el grupo con lesión renal aguda se pudo establecer que el aumento de D_{\max} y D_{\min} se asoció con mayor gravedad según KDIGO (D_{\max} Coef. 0.187 - D_{\min} Coef. 0.160, $p>|Z|$: 0.008 - 0.006, IC >95 % [0.049 / 0.324] - [0.046 / 0.273]), especialmente en pacientes ingresados post cirugía cardiovascular ($n=14$, Coef. 0.761,



$p > |Z|$: 0.005, IC >95 % [0.229 / 1.292]).

Conclusiones: las mediciones seriadas de evaluación ultrasonográfica a pie de cama de los ejes longitudinal y transversal de la vena cava inferior pueden ayudar a predecir la gravedad de la lesión renal aguda en pacientes críticos, especialmente en pacientes posoperatorios de cirugía cardiovascular.

Palabras clave: lesión renal aguda, paciente crítico, unidad de cuidados intensivos, vena cava inferior, ecografía.

Introduction

Acute kidney injury (AKI) is a complication frequently found in critically ill patients. Its incidence ranges from 30 % to 60 %, increasing patient morbidity and their need for renal support therapy (10-15 %), with attributable mortality between 20 % and 50 % [1,2].

A review of the possible pathophysiological mechanisms reveals a complex relationship between vascular, tubular, and inflammatory factors. Among prerenal and renal causes, volume overload, interstitial edema, inflammation, microcirculation alterations, hypoperfusion in the renal medulla, acute tubular necrosis, nephrotoxins, and intratubular obstruction have been widely documented [3–11]. However, knowledge of pathophysiology remains incomplete, especially on the underlying molecular processes that condition tubular epithelial cell injury and repair mechanisms or maladaptation related to intraparenchymal fibrosis and progression to chronic kidney disease.

Different studies performed in animal models that underwent induction of sepsis and AKI with subsequent renal biopsies showed that acute tubular necrosis was relatively uncommon. However, when present, it reflected an alteration of macrocirculatory (e.g., increased renal plasma flow) and microcirculatory variables (e.g., decrease in intraglomerular pressure due to nitric oxide-induced efferent arteriolar vasodilatation). This points to a possible functional origin of the renal lesion [12,13].

Additionally, both deficit and excess intravascular volume and positive fluid balances have been associated with worse renal outcomes. These conditions may worsen the overall prognosis of AKI and its progression to renal failure [14–16]. Determining volume status and volume overload in clinical practice represents a clinical challenge. The relationship between elevated central venous pressure (CVP) values and their association with the development of AKI in patients with cardiovascular disease has been described [17]. However, routine measurements of CVP are not recommended due to multiple variables that can affect their adequate interpretation [18].

Point of Care Ultrasound (POCUS) has emerged as a helpful tool for evaluating critically ill patients, allowing non-invasive hemodynamic monitoring, assessment of patients' clinical condition, and guidance in therapeutic decisions such as administration of intravenous fluids [19, 20]. Competences to perform ultrasonographic evaluations by emergency and critical care physicians have been established in different guidelines and consensus [21–26]. In addition, the acquisition of specific skills by trainees in emergency centers and medical residents has demonstrated an excellent inter-observer correlation [27, 28].

A study published in 2008 by the Canadian research group of Beaubien-Souligny *et al.* examined ultrasonographic measurements of the inferior vena cava (IVC), as well as hepatic and renal Doppler assessments in patients undergoing cardiac surgery before and after the procedure. The study found that markers of venous congestion are independently associated with AKI after cardiac surgery [29]. This association was also described in patients with cardiorenal syndrome [30].

Subsequently, the same research group proposed the venous excess ultrasonographic scoring system (VExUS), in which the IVC is assessed first. If it measures >2 cm, Doppler measurements of the hepatic veins, portal, and renal venous circulation are performed. This score demonstrated a strong association between IVC dilatation and alterations in the Doppler flow pattern with the development of AKI [31]. However, this score was examined in cardiovascular surgery and cardiorenal syndrome patients, and further validation in other critical patient scenarios is required [32].

Materials and methods

Study design and patients

The Bioethics Committee of the Universidad Tecnológica de Pereira evaluated and approved this study under registration code 63 – 230821.

In accordance with the World Medical Association's Declaration of Helsinki informed consent was obtained from all patients. Conscious patients provided written consent, while consent for unconscious patients was obtained from their family members.

This longitudinal study was conducted in an intensive care unit (ICU) in Pereira (Colombia). Patients aged >18 years admitted to ICU for different causes were selected. The exclusion criteria included history of chronic kidney disease, known creatinine levels within the last three months before admission with glomerular filtration rate (GFR) <60 mL/min/1.73m²

determined by the Modification of Diet in Renal Disease (MDRD) equation, creatinine at admission greater than the reference value for age and sex (in case of unknown previous creatinine), pregnancy, renal transplantation, prior renal support therapy, documented obstructive etiology of AKI and/or patients receiving palliative care.

A protocol for measurement collection was defined (see Appendix). The principal investigator, who had previously performed a procedural curve of 50 validated measurements assessed by a graduate radiologist with over five years of experience, performed the imaging and measurements in accordance with the standards established for critical care personnel [24–26]. A Mindray TE7 ultrasound machine was used, and images were obtained with a P4-2s sector-phased transducer. Measurements were collected upon ICU admission and then daily for up to five days during admission, or less if the patient was transferred or deceased. Additionally, relevant clinical variables and AKI criteria based on the KDIGO classification were monitored, with daily updates to the classification.

Ultrasonographic measurements

The patient was placed supine with the head elevated between 0° and 30°. Ventilatory mode data and programming parameters were recorded if the patient was under ventilatory support. With the sector transducer in abdominal visualization mode, M-mode images of the longitudinal axis of the IVC were obtained through the subxiphoid window. Images were recorded between 3 - 4 cm from the cavoatrial junction and 1 cm below the hepatic veins. If obtaining an adequate window was challenging due to factors such as post-cardiovascular surgery, dressings, or drains, a transhepatic window was used, typically between the anterior and mid-axillary lines. The transducer was then rotated on its axis 90° to the left of the patient to visualize the transverse axis of the intrahepatic IVC and record B-mode measurements in the transverse axis of the IVC through the subxiphoid or transhepatic window, as appropriate.

Measurements on the longitudinal axis of the IVC taken in M mode

Images that allowed evaluation of at least three respiratory cycles were obtained. Subsequently, calipers were used to measure, selecting the image that best represented the IVC's maximum (Dmax) and minimum (Dmin) diameters. The collapsibility index (IVC CI) was calculated using the formula: $IVC\ CI\ (\%) = (D_{max} - D_{min}) / D_{max}$.

Due to the critical condition of patients and the impossibility of performing the sniffing test in those with spontaneous breathing, as well as the marked variability of programming of mode and ventilatory parameters expected in patients in need of ventilatory support, it was

decided to systematically use the IVC CI for all patients, to define responsiveness to volume and volume status. As cut-off points for IVC CI, values $<20\%$ are defined as a lack of fluid responsiveness (euvolemia or hypervolemia) and $>50\%$ to define the prediction of responsiveness to fluid administration (hypovolemia), according to recommendations of the American Society of Echocardiography [33, 34].

Measurements made in the transverse axis of the IVC taken in B mode

Images were obtained in cine mode, which allowed the evaluation of at least three respiratory cycles. Calipers were then used to measure the short and long diameters (Sd and Ld) at the maximum and minimum phases of the IVC, selecting the image that best represented these values. Tracings of the diameter measurements were taken between them perpendicularly, taking the measurement made first on the long diameter as a reference line. The S/L ratio was calculated at the maximum moment. The calculation of the variation of the long diameter, which is defined in this study as the delta of Ld (ΔLd), was carried out using the formula: $\Delta Ld (\%) = (Ld \text{ max} - Ld \text{ min}) / Ld \text{ min}$. The delta of Sd (ΔSd) was calculated with the formula: $\Delta Sd (\%) = (Sd \text{ max} - Sd \text{ min}) / Sd \text{ min}$. Measurements of the maximum and minimum area (Max Area and Min Area) of the previously selected images were also obtained. The variation of the IVC area ($\Delta Area$) was calculated using the formula: $\Delta Area (\%) = (Area \text{ max} - min) / Area \text{ min}$ (Figure 1).

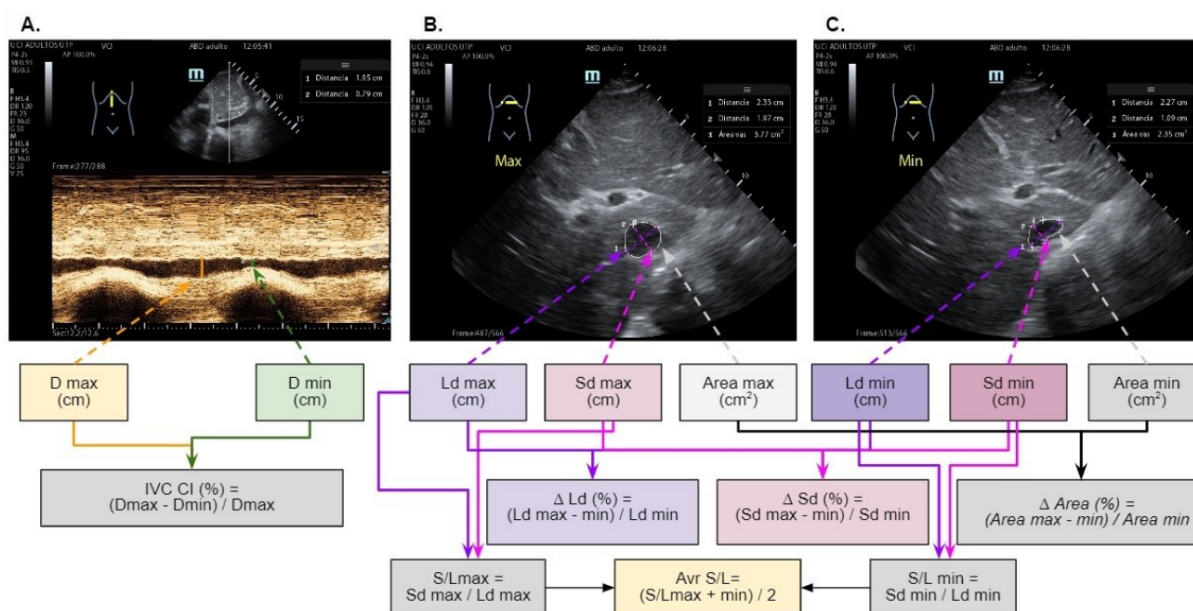


Figure 1. IVC measurements and derived calculations

Source: Author's elaboration.

Statistical analysis

Univariate analysis was performed, and central tendency and dispersion measures were established for quantitative variables. The Shapiro-Wilks test confirmed that the quantitative random variables followed an approximately normal distribution. Proportions were calculated for nominal and ordinal variables. In the bivariate analysis, acute kidney injury (AKI) was established as the outcome variable. Fisher's exact test and the approximate Chi-square test were used as statistics to establish whether or not there was independence. For hypothesis testing, a contrast evaluation was performed through parametric and non-parametric statistics according to the distribution of the variables of interest. Finally, a model that adjusts the correlation of repeated measures over time was made. Variables were adjusted using the Generalized Estimation Equation, and the calculations described were performed using the R statistical package.

Results

Fifty-eight patients were analyzed, and 51.7 % (n=30) presented acute kidney injury (AKI). Mortality was 23.3 % (n=7, p: 0.05294). Characteristics of patients admitted to the ICU are shown in Table 1.

Table 1. Clinical characteristics of the patients

Characteristics	No AKI	AKI	P
	n = 28 (48,3 %)	n=30 (51,7 %)	
Sex			
Male, n (%)	18 (64.3 %)	16 (53.3 %)	0.397
Female, n (%)	10 (35.7 %)	14 (46.7 %)	
Age			
Average years (min-max)	57 (19 - 88)	64 (23 - 83)	0.21
Anthropometric data			
BSA, m ² (min - max)	1.51 (1.02 – 2.13)	1.52 (0.91 – 1.94)	0.976
BMI, k/m ² (min-max)	25 (19 - 32)	25 (20 - 33)	0.281
Cause of admission			

Cardiovascular postoperative, n (%)	6 (21.4 %)	8 (26.7 %)	1
Cardiovascular, n (%)	5 (17.9 %)	6 (20.0 %)	0.762
Sepsis, n (%)	8 (28.6 %)	5 (16.7 %)	0.351
Septic shock, n (%)	2 (7.1 %)	4 (13.3 %)	0.671
Neurocritical, n (%)	5 (17.9 %)	6 (20.0 %)	1
Respiratory failure, n (%)	3 (10.7 %)	7 (23.3 %)	0.3
Other post-surgical, n (%)	2 (7.1 %)	4 (13.3 %)	0.671
Hypovolemic shock, n (%)	2 (7.1 %)	1 (3.3 %)	1
Comorbidities			
Hypertension, n (%)	13 (48.4 %)	13 (43.3 %)	1
Diabetes, n (%)	9 (32.1 %)	6 (20.0 %)	0.373
AMI/CD, n (%)	6 (21.4 %)	7 (23.3 %)	1
Heart failure, n (%)	3 (10.7 %)	7 (23.3 %)	0.3
COPD, n (%)	4 (14.3 %)	6 (20.0 %)	0.731
Dyslipidemia, n (%)	2 (7.1 %)	6 (20.0 %)	0.255
Severity index			
SOFA score at admission (min-max)	4 (2 - 9)	5 (1 - 10)	0.041
SOFA score at 3 rd day (min-max)	2 (0 - 7)	5 (1 - 13)	0.013

SOFA Delta (min-max)	-2 (-6 - 2)	0 (-4 - 5)	0.099
Vasopressor			
1 Vasopressor, n (%)	14 (50.0 %)	23 (76.7 %)	0.055
2 Vasopressors, n (%)	3 (10.7 %)	5 (16.7 %)	
Low (<0.1 mcg/kg/min), n (%)	9 (32.1 %)	11 (36.7 %)	0.107
Medium (0.1-0.5 mcg/kg/min), n (%)	4 (14.3 %)	10 (33.3 %)	
High (>0.5mcg/kg/min), n (%)	1 (3.6 %)	2 (6.7 %)	
Mechanical ventilation			
IMV, n (%)	10 (35.7 %)	22 (73.3 %)	0.031
Days of IMV (mín - máx)	3.2 (1 - 6)	3 (1 - 6)	0.459
VExUS Score			
VExUS 0, n (%)	11 (42.3 %)	9 (31.0 %)	0.249
VExUS 1, n (%)	8 (30.8 %)	15 (51.7 %)	
VExUS 2, n (%)	6 (23.1 %)	3 (10.3 %)	
VExUS 3, n (%)	1 (3.8 %)	2 (6.9 %)	
Status at ICU discharge			
Alive, n (%)	27 (96.4 %)	23 (76.7 %)	0.052
Dead, n (%)	1 (3.6 %)	7 (23.3 %)	

Source: Author's elaboration.

Among those who developed AKI, the onset occurred within the first four days of admission, with two out of three cases presenting within the first 24 hours (Day 1: 63.3 %, n=19; Day 2: 20.0 %, n=6; Day 3: 13.3 %, n=4; Day 4: 3.3 %, n=1) (Figure 2A). The presence and severity of AKI were established according to the KDIGO scale, which shows a score from 1 to 3 (75). About 36.7 % of all patients (n=11) were classified KDIGO 1, 50.0 % (n=15) KDIGO 2, and 13.3 % (n=4) KDIGO 3 (Figure 2B). The BUN/creatinine ratio was significantly elevated according to AKI severity, 19.8, 32.9, and 26.8 for KDIGO 1, 2 and 3 (Figure 2C).

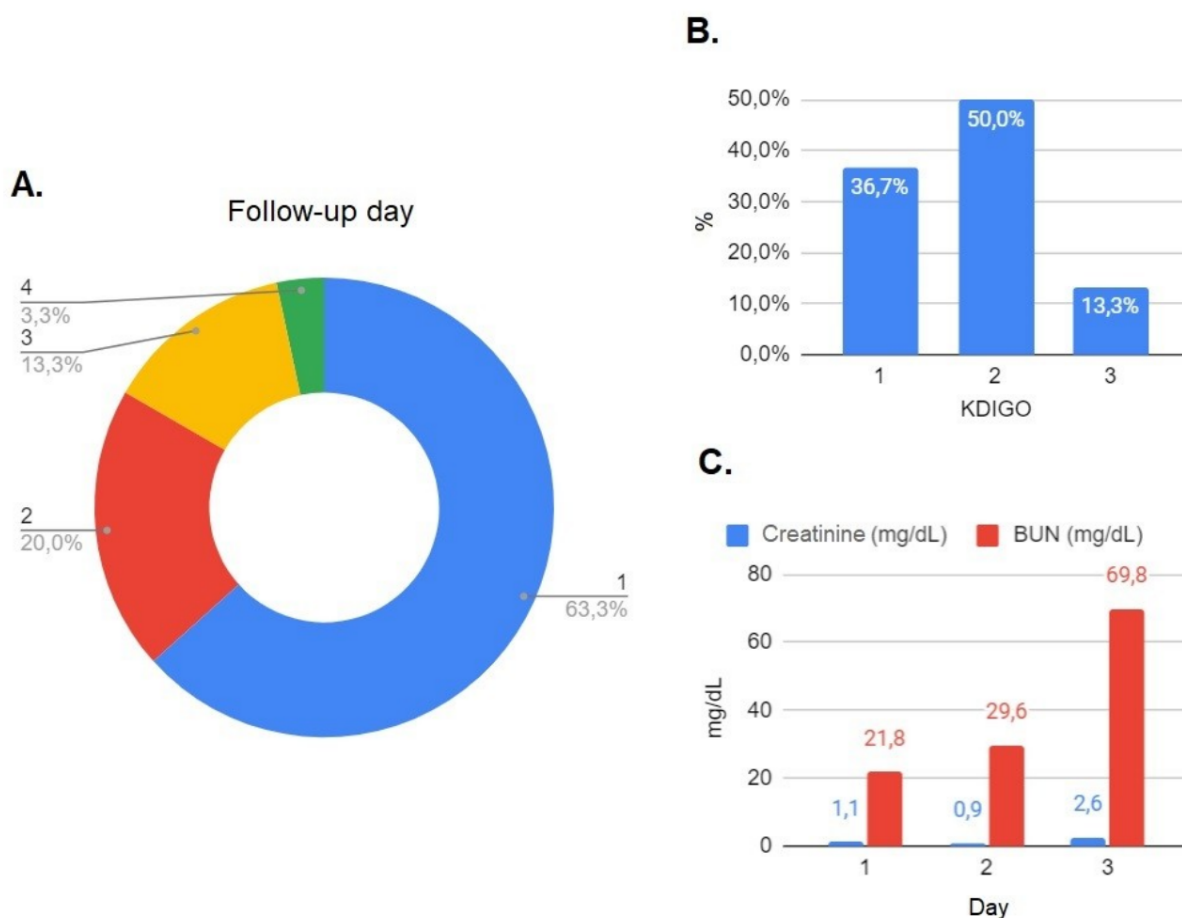


Figure 2. Follow-up and kidney function

Source: Author's elaboration.

Regarding criteria defined by KDIGO, distributions for urine output (UO) presented a difference in medians of 0.74 and 1.30 ml/kg/h between the group with and without AKI. Despite not being included in the definition criteria, BUN had medians of 20.80 and 15.25 between both groups. Both variables had statistically significant differences after being contrasted through the Wilcoxon test ($P < 0.0001$). Creatinine values did not show differences in the median (0.80 in both groups), although they did show significant differences in their maximum values (3.40 vs. 1.1 mg/dL) (Table 2).

The cumulative daily fluid balance was higher in patients with AKI (620.3 +1116.9 mL vs. 115.29 +816 mL; $P = 0.001$), as was the overall cumulative fluid balance (1114.5 +2222.25 mL vs. 275.17 +1273.37 mL; $P = 0.0002$), while fluids administered per day showed no significant differences (1841.6 +1071.3 vs. 1602.0 +1602.0; $P = 0.1444$) (Table 3).

Table 2. Defining criteria for AKI assessed using the Wilcoxon test

Variable	No AKI (remarks = 69)					AKI (remarks = 97)					P
	Average	sd	min	mdn	max	Average	sd	min	mdn	max	
UO (mL/k/h)	1.39	0.65	0.57	1.3	3.31	0.92	0.67	0.07	0.74	3.4	<0.0001
Creatinine (mg/dL)	0.76	0.2	0.3	0.8	1.1	0.98	0.53	0.4	0.8	3.4	0.0152
BUN (mg/dL)	17.39	10.58	3.2	15.25	78.2	25.4	17.66	5.9	20.8	98.4	<0.0001

Table 3. Fluid Balance

A											
Variable	No AKI (remarks = 69)					AKI (remarks = 97)					P
	Average	sd	min	mdn	max	Average	sd	min	mdn	max	
Fluids administered (mL)	1602	1205	14	1645	4222	1841	1071	22	1835	4702	0.1444
Daily fluid balance (mL)	115	816	-2764	129	2145	620	1164	-4852	529	3035	0.001
Cumulative balance (mL)	275	1273	-2503	0	8196	1114	2222	-7466	308	8225	0.0002

B						
Cumulative fluid balance (P= 0,0002)	Total (n = 58)		No AKI (n = 28)		AKI (n = 30)	
	Average	min - max	Average	min - max	Average	min - max
Fluid balance Day 1, mL (min-max)	470	-8162	118	-4330	799	-8162
Fluid balance Day 2, mL (min-max)	872	-9664	-144	-6527	1615	-9664
Fluid balance Day 3, mL (min-max)	1.536	-11059	728	-7278	1961	-11059
Fluid balance Day 4, mL (min-max)	1.876	-10996	1720	-7302	1939	-9998
Fluid balance Day 5, mL (mín - máx)	2.49	-15691	2091	-9227	2775	-15691

Source: Author's elaboration.

In a follow-up with patients with invasive mechanical ventilation the level of PEEP administered was significantly higher in those who presented AKI (P= 0.0044) (Table 4). However, no significant differences were found between the tidal volume administered (Vt: P=0.201) or the ratio of arterial oxygen pressure and inspiratory O2 flow (PaFi: P=0.1137).

Table 4. Correlation between daily IVC measurements and KDIGO classification

Measurements	Patients (n)	Remarks	Coef.	Std. err	Z	P> Z	IC >95 %	Chi2
D max.	58	223	0.187	0.0702	2.66	0.008	0.0493 / 0.3247	7.08
(M-mode)								
D min.	58	223	0.16	0.0577	2.77	0.006	0.0467 / 0.2732	7.67
(M-mode)								
Δ Ld	58	223	-0.4664	0.1673	-2.79	0.005	5.735740072	7.77
(B-mode)								

IVC measurements

A total of 223 observations were made in the 58 selected patients, yielding 1,784 measurements and 1,631 derived calculations. Additionally, the IVC collapsibility index (IVC CI) was performed for all patients for data analysis. Prediction of volume responsiveness was defined according to the recommendations of the American Society of Echocardiography, being described as non-respondents (euvoolemia or hypervolemia) with cut-off values <20 % and respondents (hypovolemia) to values >50 % [33,34].

In the bivariate analysis, IVC measurements and their derived calculations showed no significant differences between the two groups. However, when analysis models were applied to evaluate the prediction of volume responsiveness, it was found that on admission (day 0), 63.33 % (n=19) of patients in the AKI group had a non-respondent profile versus 39.28 % (n=9) in the group without AKI. This was evaluated using the Kruskal-Wallis test, yielding a value of 3.40 (P=0.065). From day 1 onward, fluid responsiveness was lost in both groups, without significant differences in IVC measurements (Figure 3).

When grouping measurements according to the cause of admission and comparing them with the daily fluid intake and balance, the AKI group consistently found a profile of no responsiveness to volume. In contrast, a more heterogeneous response was observed in the group without AKI, although no significant differences were found over time.

Multivariate analysis showed that, in IVC long-axis measurements (M-mode), increases in Dmax (Figure 4) and Dmin diameters were associated with higher severity on the KDIGO scale (Coef.: Dmax 0.1870 / Dmin 0.1600, P>|Z|: 0.008 / 0.006, CI >95 % [0.049 / 0.324] / [0.046 / 0.273]).

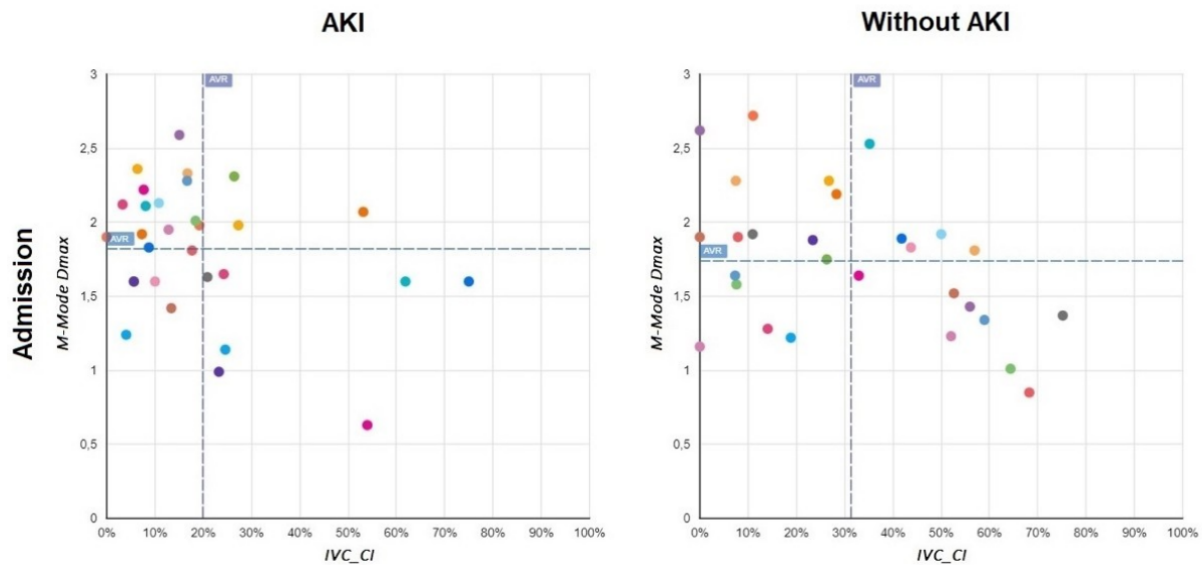


Figure 3. Admission measurements of IVC and Dmax in patients who developed AKI versus those who did not

Source: Author's elaboration.

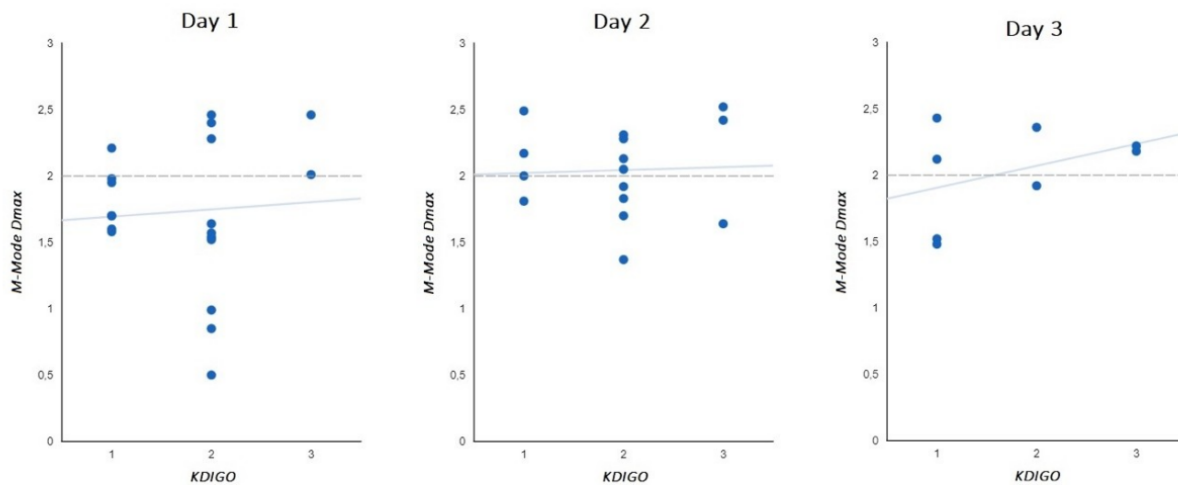


Figure 4. Daily behavior of IVC Dmax measurements according to KDIGO

Source: Author's elaboration.

On the other hand, in IVC transverse axis measurements (B-mode), multivariate analysis showed that the increase in long-diameter delta (ΔLd) is associated with a lower KDIGO score (Coef. -0.4664, $P > |Z|$: 0.005, CI >95 % [-0.794 / -0.138]) (Table 4).

In the analysis by subgroups, patients who were admitted postoperatively for cardiovascular surgery and who presented increased Dmax (longitudinal view IVC M-mode) were

associated with greater severity as determined by the KDIGO scale ($n=14$, Coef. 0.7612, $P>||Z|$: 0.005, CI >95 % [0.2295 / 1.2928]), as were patients with hypovolemic shock ($n=2$, Coef. 1.1987, $P>||Z|$: <0.001, CI >95 % [0.7087 / 1.6888]).

In patients who were discharged from the ICU alive, lower Dmax values were associated with greater severity as determined by the KDIGO scale ($n=50$, Coef. -0.5621, $P>||Z|$: <0.001, CI >95 % [-0.6726 / -0.4516]).

Discussion

Point of care ultrasound (POCUS) has emerged as a beneficial tool for evaluating critically ill patients and is handy for non-invasive hemodynamic monitoring [19, 20]. In the FENICE study, it was determined that primary triggers for fluid administration were hypotension (58.7 %), oliguria (18 %), and vasopressor weaning (7.1 %). Unfortunately, ultrasonography prediction of fluid administration responsiveness was only performed in 2 % of the cases [35].

POCUS measurements of IVC have been used to predict responsiveness to fluid administration [36, 37], to estimate volemic status, and to determine noninvasively mean right atrial pressures [33, 34]. Currently, their usefulness remains debated, as various clinical conditions can influence these measurements (38), and different meta-analyses have questioned their reliability in predicting responsiveness to fluid administration [39–41].

In this study, AKI occurred in 57.1 % ($n=30$) of patients, with an associated mortality of 23.3 %, data similar to those found in global epidemiological studies [1, 2]. It is worth highlighting that 2 out of 3 patients who developed AKI did so within the first 24 hours (Day 1: 63.3 %, $n=19$; Day 2: 20.0 %, $n=6$; Day 3: 13.3 %, $n=4$; Day 4: 3.3 %, $n=1$), highlighting its early onset. The severity of AKI can be established by KDIGO classification. This study showed that urine output (UO) was the variable that most influenced the KDIGO 1 and 2 classification, while creatinine levels were determinant for the KDIGO 3 classification. These data may be related to the Clinical Practice Guideline for Acute Kidney Injury, which describes the value of decreased urine output as a much earlier marker of AKI than elevated serum creatinine values [42]. Importantly, in our study, 86.7 % ($n = 26$) of patients with AKI were classified as KDIGO 1 and 2.

Blood urea nitrogen (BUN), although not part of the KDIGO classification, showed a strong association with AKI ($P<0.001$). Furthermore, the elevation of the BUN/creatinine ratio was directly related to the severity of AKI: 19.8, 32.9, and 26.8 for KDIGO 1, 2, and 3, respectively. In clinical practice, oliguria has been related to hypovolemic states and usually induces an

automatic clinical decision to increase the administration of intravenous fluids. However, a decrease in urinary output as an isolated clinical finding is not necessarily the result of intravascular volume depletion and may even be present in patients with hypervolemia, as shown in patients with cardiorenal syndrome.

When fluid administration and balance were evaluated, a significant association was found between the increase in cumulative balance and the development of AKI (620 +1,116 mL vs. 115 +816 mL; $P=0.001$), as well as the increase in overall cumulative balance (1,114 +2,222 mL vs. 275 +1,273 mL; $P=0.0002$). This association has already been described in reviews published by Bellomo and Ronco's research group [43,44].

About ultrasonographic measurements of the IVC and derived calculations, it was determined to systematically use the collapse index IVC CI for all patients, regardless of whether they were on ventilatory support, due to the impossibility of performing sniff tests in many patients with spontaneous breathing and the variability of the inspiratory drive observed in patients undergoing IMV. This decision was made based on studies conducted in similar populations with the same limitations [45,46]. The cut-off points for IVC CI were <20 % for no volume response (euvolemia or hypervolemia) and >50 % to define the prediction of volume response (hypovolemia) [33,34,47].

On admission (day 0), 63.3 % of patients in the AKI group ($n=19$) had an IVC CI <20 % vs. 39.2 % in the non-AKI group ($n=9$). At the other extreme, an IVC CI >50 % was seen in 13.3 % of the AKI group ($n=4$) vs. 28.5 % of the non-AKI group ($n=8$). In the bivariate analysis of the total observations, no significant results were obtained in the IVC measurements (Dmax, Dmin, Area max, Area min, Ld and Sd max and min) nor their derived values (IVC CI, Δ Area, Δ Sd, Δ Ld, the S/L ratio, and their average). When daily follow-up analysis was performed, the IVC CI at admission (day 0) showed a borderline association with the development of AKI (Kruskal-Wallis test = 3.40, $P=0.065$). These values suggest a profile of non-response to volume administration (euvolemia or hypervolemia) in patients who developed AKI. After the first 24 hours, both groups lost volume responsiveness similarly. However, the behavior of the measurements shows a tendency to be more heterogeneous in the group of patients without AKI.

Although an IVC CI <20 % at the time of admission did not reach statistical significance ($P=0,065$), it is necessary to consider the importance of objectively evaluating the volume status using tools such as IVC ultrasonography, thus establishing strategies for using or limiting fluid administration [48,49].

Kanji and coworkers had already described this fact in 2014 [50] in a study involving 220 critically ill patients in shock of undetermined etiology. They evaluated a strategy of restrictive fluid administration guided by ultrasonographic tracing (n=110) versus conventional management (n=110). The restrictive strategy was shown to be associated with improved survival (66 % vs. 56 %, $P=0.04$), reduced stage 3 acute kidney injury (20 % vs. 39 %), and more days of life without the need for renal replacement therapy (28 [9.7-28] vs. 25 [5-28], $P=0.04$). Even more relevant results considering that the reduction in intravenous fluid administration was only significant in the first 24 hours of follow-up (49 [33-74] vs. 66 [42-100] ml/kg, $P=0.01$).

Different analysis models were performed in the multivariate analysis, finding that when evaluating IVC measurements concerning the KDIGO scale, the increase in Dmax ($P=0.008$) and Dmin ($P=0.006$) was associated with increased severity of AKI. In contrast, the rise in ΔLd ($P=0.005$) was associated with decreased severity.

Increased Dmax (>2 cm), together with a decrease in IVC CI (volume unresponsiveness profile), has been classically associated with higher central venous pressure values (CVP 15, range 10-20 mmHg), which has demonstrated worse renal outcomes [33-35]. The ΔLd indicates the degree of vessel distensibility. According to the findings, we can deduce that the greater the distensibility of the IVC in the transverse axis, the less severe the AKI. IVC assessments in the transverse axis have already been considered in other studies that have evaluated the distensibility area and the short and long axis diameters ratio, showing reliable measurements for volume response prediction and estimation of CVP [36,51,52].

In the analysis models by cause of admission, it was possible to establish that in patients admitted postoperatively for cardiovascular surgery, the increase in Dmax was associated with greater severity on the KDIGO scale (n=14, observations: 55, $P>|Z|$ 0.005, CI $>95\%$ [0.2295 / 1.2928]). These observations may be related to the findings reported in the study by Aslaner et al., where they describe that the increase in Dmax is the most reliable POCUS parameter for predicting AKI in patients with compromised ventricular function, with a cut-off point >1.7 cm (Sensitivity: 100 %, 95 % CI: 83.2-100; Specificity: 70.2 %, 95 % CI: 61.6-77.7) [53].

In contrast, IVC measurements in surviving patients showed a negative correlation between the increase in Dmax concerning the severity of KDIGO classification (n=50, observations: 195, $P>|Z|$ <0.001 , CI $>95\%$ which we theorize, could be explained by previous clinical or physiological situations that may be related to lower tolerance to increased intravascular volume from patients who develop AKI.

Conclusions

The serial POCUS assessment of the IVC performed in the M-mode view of the vessel's longitudinal axis and the B-mode of the transverse axis provides us with data that can bring us closer to the reality of the volemic status of critically ill patients. Furthermore, the finding of the association between the severity of AKI and the increase in Dmax, especially in post-cardiovascular surgery patients, added to the profile of non-response to volume at admission and the increase in cumulative balance recorded at daily follow-up, speaks to the harmful effect of the rise in mean proper atrial pressures on the development and severity of AKI.

Further studies are considered important to validate these findings and determine the behavior of IVC measurements in different scenarios of critically ill patients.

Ethical considerations

This study was conducted in accordance with ethical guidelines and institutional regulations. Informed consent was obtained from all participants or their legally authorized representatives if the patient was unable to provide consent. Patient confidentiality was strictly maintained, and all data were anonymized to ensure privacy. The study protocol was approved by the institutional ethics committee.

Conflict of interest

The authors declare no potential conflicts of interest.

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Authors contributions

Euclides José Cruz Echeverry: Conceptualization, Investigation, Project administration, Resources, Software, Writing – original draft; Mateo Aguirre Flórez: Writing – review & editing, English translation; Andrés Eduardo Bernal: Conceptualization, Investigation, Methodology, Supervision, Validation, Writing – original draft; Fernando José Villabona García: Methodology, Supervision, Validation; Carlos Eduardo Giraldo Ospina: Validation; José William Martínez: Formal analysis, Methodology, Supervision, Validation.

Consent form statement

Prior to data acquisition, an informed consent was obtained. If the patient was intubated or unable to provide consent, a legally authorized representative or family member signed the consent on their behalf.

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