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Correlation Study of Inferior Venacava Collapsibility Index And Perfusion Index as Predictors of Fluid Responsiveness in Critically ill Patients

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ABSTRACT

Introduction: Fluid responsiveness assessment is crucial in managing critically ill patients. This study evaluates the correlation and efficacy of Inferior Vena Cava Collapsibility Index (IVCCI) and Perfusion Index (PI) in predicting fluid responsiveness.

Objectives: The primary objective was to evaluate the correlation between the IVCCI and PI as predictors fluid responsiveness in critically ill patients. Secondary objectives involved studying the individual accuracy of the IVCCI and PI in predicting of fluid responsiveness and exploring whether their combined use improves overall accuracy in predicting fluid responsiveness in this patient population.

Methodology: This prospective study enrolled 80 critically ill patients requiring hemodynamic monitoring and fluid resuscitation. IVCCI was measured using bedside ultrasonography, and PI was derived from pulseoximetry. Hemodynamic parameters were recorded before and after a standardized fluid bolus. Receiver Operating Characteristic (ROC) curves were generated to assess the predictive performance of IVCCI and PI.

Results: Among 80 enrolled patients, responders (N=35) demonstrated significantly lower baseline cardiac output (4.5 ± 0.23 vs 4.76 ± 0.22, $p<0.01$) and higher IVCCI (35.13 ± 13.41 vs 28.53 ± 10.17, $p=0.015$) compared to non-responders. After a standardized fluid bolus, responders exhibited higher cardiac output (5.31 ± 0.24 vs 4.9 ± 0.27, $p<0.01$) and lower IVCCI (22.01 ± 15.84 vs 28.88 ± 11.71, $p=0.029$). IVCCI and PI before fluid bolus showed a very weak positive correlation ($r = 0.080$, $p = 0.478$). Moreover, IVCCI before fluid bolus had better sensitivity (0.745) and specificity (0.727) than PI before fluid bolus (sensitivity: 0.574, specificity: 0.485), with AUC values of 0.693 and 0.455, respectively.

Conclusion: IVCCI demonstrates efficacy in predicting fluid responsiveness, with a very weak positive correlation to PI. Notably, IVCCI before fluid bolus outperforms PI before fluid bolus in sensitivity and specificity, highlighting its superior predictive accuracy in critically ill patients.

INTRODUCTION

In the dynamic field of critical care medicine, the effective management of fluid status is paramount to ensuring optimal tissue perfusion and organ function in patients. Fluid resuscitation is a cornerstone in the care of critically ill patients, especially those with circulatory insufficiency.[1,2] However, the challenge lies in identifying patients who will truly benefit from fluid administration, as indiscriminate fluid therapy may lead to adverse outcomes, such as tissue edema and impaired oxygenation.[3]

In recent years, ultrasound has emerged as a valuable tool for guiding fluid resuscitation.[4] This shift has been prompted by the limitations of traditional static hemodynamic indices, including central venous pressure or pulmonary artery occlusion pressure, which have shown limited value in predicting fluid responsiveness.[5,6] Contrarily, studies focusing on dynamic indices, such as stroke volume (SV) variation or pulse pressure variation, which analyse preload dependence, have been validated as useful predictors of fluid responsiveness.[7,8] However, the practical application of these dynamic indices has been hindered by their reliance on invasive arterial access, limiting their utility in routine clinical practice. This limitation underscores the need for non-invasive alternatives that offer real-time insights into fluid responsiveness. Amid these emerging parameters, the Inferior Vena Cava collapsibility index (IVCCI) and Perfusion Index (PI) have garnered attention for their potential utility.

The IVCCI, measured by ultrasound, dynamically reflects changes in the diameter of the inferior vena cava during the respiratory cycle. Real-time information about intravascular volume status is provided, and studies suggest that a significant decrease in IVC collapsibility may indicate fluid responsiveness, signifying a preload reserve effectively mobilized with fluid administration.[9-11]

Complementing the IVCCI, the Perfusion Index, derived from pulseoximetry, offers an assessment of peripheral perfusion. It is a non-invasive measure based on the pulsatile nature of arterial blood flow, reflecting changes in peripheral vascular tone. An increasing Perfusion Index has been associated with fluid responsiveness, indicating improved tissue perfusion and oxygenation following fluid administration.[12,13]

This prospective observational study is designed with the primary objective of evaluating the correlation between the IVCCI and PI as predictors fluid responsiveness in critically ill patients. Our secondary objectives include assessing the individual accuracy of the IVC collapsibility index and Perfusion Index in predicting fluid responsiveness and exploring whether their combined use enhances the overall accuracy of predicting fluid responsiveness in this patient population. Through a comprehensive analysis of these indices, we seek to contribute valuable insights into their utility for guiding fluid resuscitation strategies in the dynamic landscape of critical care medicine.

METHODOLOGY

This prospective observational study aimed to investigate the predictive value of the inferior venacava collapsibility index (IVCCI) and perfusion index (PI) for fluid responsiveness in critically ill patients admitted to the intensive care unit (ICU). Ethical approval was obtained from the institutional review board, and the study adhered to ethical guidelines. The study included adult patients aged 18 years and above who were admitted to the ICU and required hemodynamic monitoring and fluid resuscitation. Exclusion criteria encompassed pregnant individuals, patients with known heart or lung disease, those with a history of chronic kidney disease or end-stage renal disease, severe liver disease or liver failure, allergies to ultrasound gel and factors such as the inability to postpone fluid administration, signs of active pulmonary edema, or posing a clinical risk from additional intravenous fluids during study procedures.,

After informed and written consent for participation, data were systematically collected using a structured proforma, encompassing demographic details, medical history, results from physical examination, laboratory and radiographic findings, and parameters from hemodynamic monitoring. Bedside ultrasonography was employed to measure cardiac output (CO) and IVCCI before fluid bolus (IVCCI_Before) and after (IVCCI_After)a 500 ml crystalloid solution administered over 15 minutes using the M-turbo ultrasound system (Fujifilm Sonosite Inc., Bothell, WA, USA). Trained physician, well-versed in standardized techniques, conducted the measurements.

Patients meeting the inclusion criteria were positioned supinely, and the phased-array probe was adeptly maneuvered to visualize the IVC in long-axis view. IVCCI was calculated during a standardized respiratory cycle using M-mode focusing on maximum (IVC max) and minimum (IVC min) diameter. IVCCI was calculated as (IVC max – IVC min) / IVC max.

The echocardiographic examination involved capturing a parasternal two-dimensional (2D) view, specifically measuring the aortic diameter (AoD) at the aortic valve annulus. Subsequently, the aortic area (AA) was determined using the formula $AA = \pi \times (AoD^2 / 4)$. Pulsed wave Doppler was applied to assess aortic blood flow at the aortic annulus within the apical five-chamber view, allowing the calculation of the velocity-time integral (VTI) for aortic blood flow. The measurement of stroke volume (SV) and cardiac output (CO) was given by: $SV = VTI \times AA$ and $CO = SV \times \text{heart rate}$. Evaluation of the aortic area was specifically conducted at baseline due to the presumption of its constancy throughout the test. Fluid responsiveness was defined as an increase in cardiac output of at least 10% in response to the fluid administration, as measured by transthoracic echocardiography. Perfusion index values were obtained before (PI_Before) and after (PI_After) the standard fluid bolus using Mindray UMEC-12 monitors. The pulseoximeter probe was carefully placed on the patient's fingertip, with efforts made to reduce potential external factors that might affect signal accuracy.

Statistical analysis using SPSS v21 included descriptive statistics, correlation analysis, and regression analysis to explore relationships between IVCCI, PI, and fluid responsiveness, with a significance level set at 0.05. The study adhered to the principles of the Declaration of Helsinki and other relevant ethical guidelines, ensuring informed consent and strict confidentiality.

Figure 1: Echocardiographic Methodology: A) Long-axis view for IVC, calculating IVCCI during a standardized respiratory cycle using M-mode (IVC max and min). B) Parasternal 2D view, measuring aortic diameter (AoD) at the aortic valve annulus. C) Pulsed Doppler in the apical five-chamber view to assess aortic blood flow and calculate the velocity-time integral (VTI).

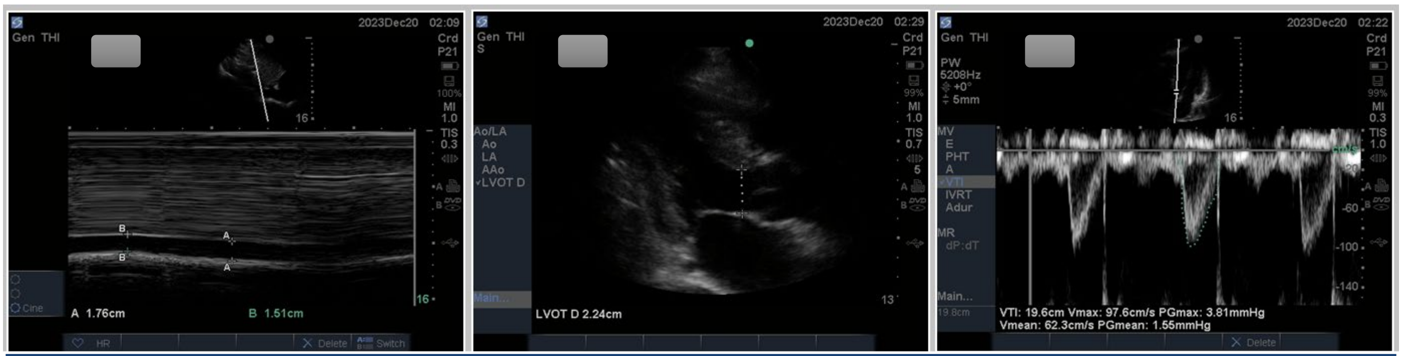

Fig 1: Echocardiographic Methodology: A) Long-axis view for IVC, calculating IVCCI during a standardized respiratory cycle using M-mode (IVC max and min). B) Parasternal 2D view, measuring aortic diameter (AoD) at the aortic valve annulus. C) Pulsed Doppler in the apical five-chamber view to assess aortic blood flow and calculate the velocity-time integral (VTI).

RESULTS

In this study, a total of 103 participants were screened for eligibility of which 80 participants were enrolled and further analysed (Figure 2). Study of Cardiac output (>10% increase) before and after the fluid bolus differentiated Responders (N=35) and Non-Responders (N=45).

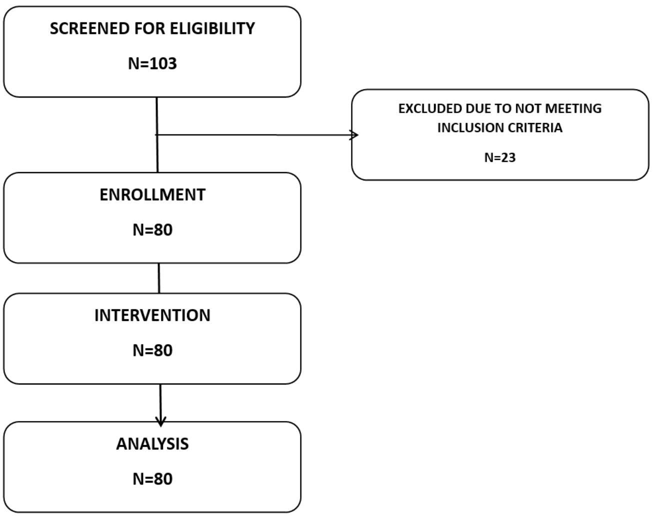

Fig 2: CONSORT FLOW DIAGRAM

Table 1 shows the demographic analysis revealing no significant differences in age, gender, or comorbidities ($p > 0.05$). Hemodynamic parameters pre- and post-fluid bolus are shown in Table 2 and 3. Before the fluid bolus, both Responders as well as Non-Responders had comparable HR, MAP and PI. But, Responders had significantly lower cardiac output (4.5 ± 0.23 vs 4.76 ± 0.22) and higher IVCCI (35.13 ± 13.41 vs 28.53 ± 10.17). Similarly, after the fluid bolus, HR, MAP and PI were comparable. However, cardiac output was significantly higher in Responders (5.31 ± 0.24 vs 4.9 ± 0.27) and IVCCI was significantly lower (22.01 ± 15.84 vs 28.88 ± 11.71). These findings underscore the role of hemodynamic monitoring in predicting fluid responsiveness in critically ill patients.

Table 1: Patient Demographics

Patient Demographics		Responder (N=35)	Non-Responder (N=45)	P-value
Age in Years (Mean ± SD)		46.22 ± 18.09	46.31 ± 18.06	0.984
Gender (n)	Male	22	33	0.316
	Female	13	12	
DM (n)	Yes	7	14	0.263
	No	28	31	
HTN (n)	Yes	9	10	0.716
	No	26	35	
COPD (n)	Yes	6	9	0.745
	No	29	36	
IHD (n)	Yes	5	3	0.260
	No	30	42	

Table 2: Hemodynamic parameters before fluid bolus in Responders and Non-Responders (HR: Heart Rate, MAP: Mean Arterial Pressure, VTI: Velocity Time Integral, IVC: Inferior Vena Cava, IVCCI: Inferior Vena Cava Collapsibility Index)

Before Fluid Bolus	Responder (Mean ±SD)	Non Responder (Mean ±SD)	P-value
HR (beats/min)	93.62 ± 9.87	91.11 ± 12.19	0.324
MAP(mmHg)	68.22 ± 10.1	67.35 ± 10.04	0.702
VTI(cm)	16.05 ± 2.24	17.1 ± 4.03	0.157
Cardiac Output(L/min)	4.5 ± 0.23	4.76 ± 0.22	0.000
Stroke Volume(ml)	48.57 ± 5.47	53.21 ± 7.48	0.003
IVC minimum(cm)	1.53 ± 0.23	1.78 ± 0.25	0.000
IVC maximum(cm)	2.39 ± 0.21	2.49 ± 0.2	0.027
IVCCI(%)	35.13 ± 13.41	28.53 ± 10.17	0.015
Perfusion Index(%)	2.40 ± 1.09	2.60 ± 1.08	0.434

Table 3: Hemodynamicparameters after fluid bolus in Responders and Non-Responders (HR: Heart Rate, MAP: Mean Arterial Pressure, VTI: Velocity Time Integral, IVC: Inferior Vena Cava, IVCCI: Inferior Vena Cava Collapsibility Index)

After Fluid Bolus	Responder (Mean ±SD)	Non-Responder (Mean ±SD)	P-value
HR (beats/min)	91.77 ± 6.13	93.97 ± 9.18	0.225
MAP(mmHg)	70.54 ± 8.32	69.02 ± 8.85	0.436
VTI(cm)	19.17 ± 2.55	17.07 ± 4.13	0.010
Cardiac Output(L/min)	5.31 ± 0.24	4.90 ± 0.27	0.000
Stroke Volume(ml)	58.19 ± 5.14	52.62 ± 5.91	0.000
IVC minimum(cm)	1.79 ± 0.25	1.81 ± 0.28	0.746
IVC maximum(cm)	2.33 ± 0.22	2.56 ± 0.22	0.000
IVCCI(%)	22.01 ± 15.84	28.88 ± 11.71	0.029
Perfusion Index(%)	2.60 ± 1.08	2.75 ± 0.93	0.506

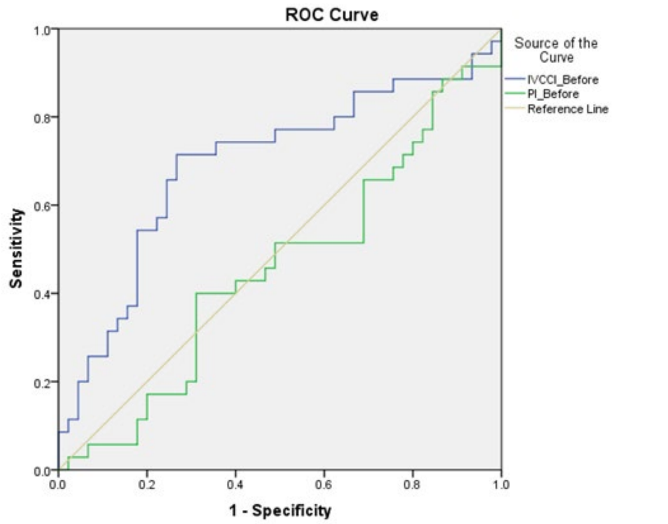

Fig 3: Receiver operating characteristic (ROC) curves for IVCCI_Before and PI_Before in predicting fluid responsiveness

Table 4: Fluid Responsiveness Predictors: IVCCI_Before and PI_Before Performance Metrics

	Cut-off	Sensitivity	Specificity	AUC	p-value	Lower Bound	Upper Bound
IVCCI_Before	28.52	0.745	0.727	0.693	0.003	0.57	0.815
PI_Before	2.43	0.574	0.485	0.455	0.494	0.327	0.584

The calculated cut-offs for IVCCI_Before (28.52) and PI_Before (2.43) demonstrated distinctive performance metrics (Table 4).IVCCI_Before exhibited moderate sensitivity (0.745) and specificity (0.727), suggesting a balanced capacity to identify true positives and negatives. In contrast, PI_Before, with lower sensitivity (0.574) and specificity (0.485), displayed a less discriminative ability. The area under the curve (AUC) further underscored IVCCI_Before's superiority (AUC = 0.693, p = 0.003) in contrast to PI_Before (AUC = 0.455, p = 0.494) (Figure 3), emphasizing the former's better overall predictive accuracy in discerning fluid responsiveness in critically ill patients.

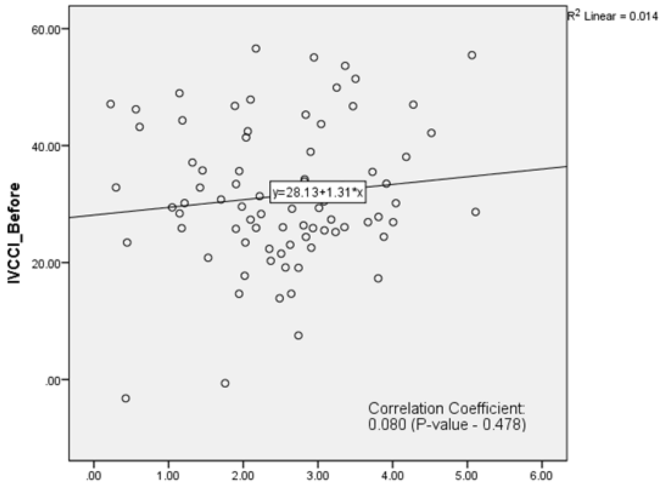

Fig 4: Scatter plot depicting the relationship between IVCCI_Before and PI_Before. A fitted line illustrates the trend, and the correlation coefficient (r = 0.080, p = 0.478) is displayed on the chart, indicating a very weak positive correlation

Figure 4 summarizes the correlation analysis between IVCCI_Before and PI_Before, indicating a very weak positive correlation (r = 0.080, p = 0.478) that is not statistically significant. The logistic regression analysis was carried out to predict fluid responsiveness in the studied population. The variable IVCCI_Before emerged as a significant predictor, with a coefficient (B) of-0.050 and a p-value of 0.019. The model achieved an overall correct classification rate of 65.0%, demonstrating particular efficacy in identifying Nonresponders with an accuracy of 82.2%. The chi-square test indicated a significant enhancement in model fit with the inclusion of IVCCI_Before (χ^2 = 6.196, df = 1, p = 0.013). In contrast, PI_Before did not contribute significantly to the model (p = 0.857).

DISCUSSION

In our study of critically ill patients requiring fluid resuscitation or hemodynamic monitoring, we assessed the predictive potential of Inferior Vena Cava Collapsibility Index (IVCCI) and Perfusion Index (PI) for fluid responsiveness. The results of our study suggest IVCCI before fluid bolus has better predictive accuracy for fluid responsiveness as compared to PI.

The calculated cut-off values for IVCCI_Before (28.52) and PI_Before (2.43) provided valuable insights into their predictive performance. IVCCI_Before demonstrated a balanced sensitivity (0.745) and specificity (0.727), highlighting its capacity to correctly identify both Responders and Non-Responders. In contrast, PI_Before displayed lower sensitivity (0.574) and specificity (0.485), indicating a less discriminative ability in predicting fluid responsiveness.The area under the curve (AUC) analysis further supported the superior predictive accuracy of

IVCCI_Before compared to PI_Before. The AUC for IVCCI_Before (0.693, p = 0.003) indicated a moderate level of accuracy, suggesting its potential as a standalone predictor. Conversely, the AUC for PI_Before (0.455, p = 0.494) fell below the threshold for reliable prediction.

IVCCI as a predictor

Comparing our study with existing research underscores the diversity in methodologies and patient populations aimed at identifying fluid responsiveness predictors. In an ICU setting, Airapetian et al highlighted spontaneous ventilation and a 0.9% saline challenge, reporting a sensitivity of 31%, specificity of 97%, and an AUROC of 0.62 (95% CI: 0.49-0.74) for SV>10%.[14] In contrast, our study, focusing on IVCCI_Before, exhibited a higher sensitivity (74.5%) and comparable specificity (72.7%). Similarly using crystalloid administration during spontaneous ventilation, Lanspa et al reported an AUROC of 0.83 (95% CI: 0.58-1) for CI>15%.[15] Distinctions may arise from variations in the fluid challenge method, patient characteristics, and the definition of fluid responsiveness.

Studies done in Emergency department in spontaneously breathing had contrasting findings. Corl et al[16] utilized Passive Leg Raising (PLR) test, resulting in a sensitivity of 46%, specificity of 84%, and an AUROC of 0.46 (95% CI: 0.21-0.71), whereas de Valk et al[17] gave a saline challenge and reported a higher sensitivity (83%), lower specificity (67%), and an AUROC of 0.741. Discrepancies in findings could be influenced by patient characteristics, the use of PLR, and differences in definition of fluid responsiveness. While Corl et al[16] defined it as > 10% increase in Cardiac output, de Valk et al[17] defined it as increase in Systolic blood pressure by >10mmHg.Employing Hydroxyl Ethyl Starch administration during spontaneous ventilation, Muller et al. reported a sensitivity of 70%, specificity of 84%, and an AUROC of 0.77 (95% CI: 0.6-0.88) for SV>15%.[10]

Differences may be influenced by variations in patient populations, fluid challenge types, and criteria for defining fluid responsiveness. These variations highlight the intricate nature of predicting fluid responsiveness, emphasizing the necessity to consider patient-specific factors and study-related variables in interpreting and applying these results clinically.

Perfusion index as a predictor

Perfusion index (PI) from pulseoximetry holds potential as a non-invasive predictor of fluid responsiveness in critical care, correlating with changes in cardiac output following fluid administration. Hui Lian et al[18] in their study in patients with septic shock on mechanical ventilation, correlated the values of changes in PI before and after fluid bolus of 250 to 750 ml over 30 minutes to changes in cardiac index measured. They found AUC (0.776, p<0.001) between PI value vs Cardiac Index.[18] These findings are in contrast to our study where PI and Cardiac Output had no significant correlation.

In septic shock patients on norepinephrine infusion, Hasanin et al investigated the potential of pulseoximetry derived peripheral perfusion index (PPI) as a non-invasive predictor of fluid responsiveness. Following a fluid challenge test, ΔPPI, calculated as (PPI after 200 mL - baseline PPI)/baseline PPI, showed moderate predictive ability (AUC = 0.82, sensitivity 76%, specificity 80%, PPV 92%, NPV 54%, cutoff ≥ 5%). Importantly, a significant correlation between ΔPPI and ΔVTI induced by the fluid challenge was observed.[12]

Lu et al's investigation into pleth variability index (PVI) in septic shock patients, utilizing the passive leg raising (PLR) test, demonstrated a strong predictive role. Responders, defined by a ≥10% increase in cardiac index, exhibited higher pre-PLR stroke volume variation (SVV) and PVI, with robust ROC analysis AUC values of 0.948 for SVV and 0.957 for PVI.[19] In contrast, our study, focusing on PI changes concerning cardiac output before and after fluid bolus, did not reveal a significant correlation. These results are in contrast with our study. The observed differences in fluid responsiveness in septic shock can be attributed to subtle variations in patient characteristics, study methodologies, and research designs. The discrepancies highlight the necessity for further research to clarify the specific conditions under which PI or related indices may offer reliable predictions of fluid responsiveness.

Correlation and Logistic Regression Analysis:

The correlation analysis between IVCCI_Before and PI_Before revealed a very weak positive correlation (0.080, p = 0.478), which was not statistically significant. This suggests that these two indices capture distinct aspects of fluid responsiveness, further highlighting the need for a multifaceted approach in assessing hemodynamic status.

The logistic regression analysis identified IVCCI_Before as a significant predictor, with a coefficient (B) of-0.050 and a p-value of 0.019. The model achieved an overall correct classification rate of 65.0%, demonstrating particular efficacy in identifying Non-Responders with an accuracy of 82.2%. The chi-square test indicated a significant enhancement in model fit with the inclusion of IVCCI_Before (χ^2 = 6.196, df = 1, p = 0.013). In contrast, PI_Before did not contribute significantly to the model (p = 0.857). Further refinement and the addition of complementary variables may hold the potential to improve the accuracy and comprehensiveness of the predictive model.

LIMITATIONS OF STUDY

Our study provides valuable insights into using IVCCI and Perfusion Index for fluid responsiveness in critically ill patients. However, the small prospective sample size limits generalizability. Variations in patient characteristics and clinical protocols could influence results, and the observational design prevents definitive causation. Focusing on specific parameters might overlook other relevant factors, and our study primarily explored short-term outcomes, leaving long-term implications unexplored. Future research with larger cohorts, consideration of additional variables, and exploration of long-term outcomes is essential to enhance the robustness and relevance of our findings.

CONCLUSION

In conclusion, our study aimed to assess the efficacy of IVCCI and PI as predictors of fluid responsiveness in critically ill patients. The results indicate that IVCCI demonstrates efficacy in predicting fluid responsiveness, with a very weak positive correlation to PI. Notably, IVCCI before fluid bolus outperforms PI before fluid bolus in sensitivity and specificity, highlighting its superior predictive accuracy in critically ill patients.

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CONFLICT OF INTEREST None

FINANCIAL DISCLOSURE None

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