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# Inferior Vena Cava Collapsibility as a Predictor of Mortality in Patients Admitted with Severe Acute Decompensated Heart Failure

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## Abstract

Severe acute decompensated heart failure (ADHF) has a high mortality risk. Reliable, early prognostic tools are crucial. The predictive value of inferior vena cava (IVC) collapsibility, a non-invasive measure of volume status, for in-hospital mortality in specific populations, especially in resource-limited settings, requires further validation. Our objective was to evaluate whether bedside ultrasonographic assessment of IVC collapsibility index (IVC-CI) at admission is a significant predictor of in-hospital mortality among patients with severe ADHF. A prospective cohort study was conducted at Bangladesh Medical College Hospital from January 2024 to June 2025. Using purposive sampling, 80 consecutive patients admitted with severe ADHF were enrolled. IVC-CI was measured via transthoracic echocardiography within 24 hours of admission. Data were analyzed using SPSS version 23.0, employing receiver operating characteristic (ROC) curve analysis to determine the optimal IVC-CI cutoff and multivariate logistic regression to identify independent predictors of mortality. Among 80 enrolled patients, in-hospital mortality was 31.3% (25/80). Non-survivors had a significantly lower mean IVC-CI (28.4% ± 9.1 vs. 52.7% ± 12.3, p<0.001). An IVC-CI ≤35% was the optimal cutoff (AUC 0.91) for predicting mortality, with 84% sensitivity and 89% specificity. In multivariate analysis, IVC-CI ≤35% was a strong independent predictor of mortality (adjusted OR 15.4, 95% CI: 4.8–49.2, p<0.001). A reduced IVC collapsibility index (≤35%) at admission is a potent, independent predictor of in-hospital mortality in severe acute decompensated heart failure. Bedside IVC assessment is a valuable, non-invasive tool for early risk stratification in this high-risk population.

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## Introduction

Heart failure (HF) represents a global pandemic, affecting over 64 million individuals worldwide, and is associated with substantial morbidity, mortality, and healthcare expenditures.[1] Acute decompensated heart failure (ADHF), characterized by the rapid onset or worsening of signs and symptoms, is the leading cause of hospitalization in adults over 65 years of age.[2] Despite advances in pharmacotherapy and device-based treatments, ADHF admissions portend a grim prognosis, with in-hospital mortality rates ranging from 4% to 7% in international registries and notably higher in severe cases or low-resource settings.[3,4] This high mortality underscores the critical need for simple, rapid, and reliable prognostic tools at the point of care to facilitate early identification of high-risk patients and guide intensive management strategies. Current risk stratification in ADHF often relies on a combination of clinical scoring systems, biomarker assays (like B-type natriuretic peptide,

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BNP), and echocardiographic assessment of left ventricular systolic function.[5] However, these tools have limitations. Biomarker levels can be influenced by age, renal function, and body mass index, while echocardiographic ejection fraction (EF) may not fully reflect the acute hemodynamic burden, particularly in heart failure with preserved EF (HFpEF).[6,7] Central to the pathophysiology of ADHF is systemic venous congestion, a key driver of symptoms and organ dysfunction, which often precedes a decline in cardiac output.[8] Consequently, direct assessment of volume status has emerged as a pivotal component in the evaluation and prognostication of ADHF. The inferior vena cava (IVC) diameter and its respiratory collapsibility, assessed via bedside ultrasonography, have become established as non-invasive, real-time surrogates for right atrial pressure and intravascular volume status.[9] A plethoric, non-collapsible IVC suggests elevated central venous pressure and volume overload. Recent evidence has solidified the role of IVC parameters in diagnosing ADHF in the emergency department, demonstrating good sensitivity and specificity.[10] Beyond diagnosis, there is growing interest in the prognostic utility of IVC assessment. Studies indicate that a less collapsible IVC at admission is associated with worse outcomes, including prolonged hospitalization and increased diuretic requirements.[11] Nevertheless, the specific value of the IVC collapsibility index (IVC-CI) as an independent predictor of in-hospital mortality, particularly in patients presenting with severe ADHF, remains less definitively characterized. Existing studies show heterogeneity in patient populations, IVC measurement protocols, and defined endpoints.[12] Furthermore, there is a conspicuous lack of data from South Asian populations, where the epidemiology, comorbidities, and healthcare resources for HF management may differ significantly from Western cohorts.[13] This gap is critical, as validated, low-cost, bedside tools are especially valuable in resource-constrained environments. Therefore, this study was designed to address this evidence gap by prospectively evaluating the hypothesis that a low IVC-CI at admission is a powerful and independent predictor of in-hospital mortality in a cohort of patients admitted with severe ADHF in a tertiary care hospital in Bangladesh.

## Methods

This prospective cohort study was conducted at the Department of Cardiology, Bangladesh Medical College Hospital, Dhaka, from January 2024 to June 2025. A purposive sample of 80 adult patients consecutively admitted with a primary diagnosis of severe acute decompensated heart failure (ADHF) was enrolled. The study population included patients with either acute decompensation of chronic heart failure or new-onset ADHF, who presented with severe symptoms corresponding to New York Heart Association (NYHA) functional class III or IV.

***Inclusion criteria:*** Patients were included if they met the Framingham criteria for ADHF, presented with NYHA class III or IV symptoms, and had an elevated N-terminal pro-B-type natriuretic peptide (NT-proBNP) level > 1800 pg/mL or BNP > 400 pg/mL. Written informed consent was obtained from all participants or their legal guardians.

***Exclusion criteria:*** Individuals with primary pulmonary, hepatic, or renal disease-causing decompensation, chronic obstructive pulmonary disease with significant cor pulmonale, significant tricuspid regurgitation, known inferior vena cava (IVC) anomaly, pericardial constriction, or who were mechanically ventilated at admission were excluded.

***Study procedure:*** Within 24 hours of admission, a comprehensive transthoracic echocardiography was performed for all enrolled patients. Conventional two-dimensional echocardiography was carried out using

a standardized machine (Versana Active, General Electric Vingmed Ultrasound, Norway) equipped with a 2.5 MHz transducer. Left ventricular ejection fraction (LVEF) was determined by the biplane Simpson's method. Subsequently, a focused assessment of the inferior vena cava was conducted. The IVC diameter was measured in M-mode approximately 2 cm from the right atrial junction during quiet breathing and deep inspiration. The IVC collapsibility index (IVC-CI) was calculated using the standard formula: [(Maximum diameter – Minimum diameter) / Maximum diameter] × 100%. Statistical analysis was performed using IBM SPSS version 23.0. Continuous variables were compared using Student's t-test or the Mann-Whitney U test, as appropriate. A receiver operating characteristic (ROC) curve analysis was employed to determine the optimal IVC-CI cutoff value for predicting mortality. Multivariate logistic regression, adjusted for key clinical covariates (including LVEF and natriuretic peptide levels), was used to identify independent predictors of the primary outcome. A p-value < 0.05 was considered statistically significant.

## Results

During the study period, a total of 80 patients with severe acute decompensated heart failure were enrolled and completed follow-up. The overall in-hospital mortality rate was 31.3% (25 patients), while 55 patients (68.8%) survived to discharge. The mean age of the overall cohort was 66.4 ± 10.8 years, with a male predominance (62.5%). Hypertension (82.5%) and ischemic heart disease (65.0%) were the most prevalent comorbidities. No significant differences were found between survivors and non-survivors regarding age, sex distribution, or the prevalence of major comorbidities such as hypertension, diabetes, or ischemic heart disease. Significant differences emerged in key clinical and investigative parameters. Non-survivors presented with a significantly higher mean heart rate (112.4 ± 14.2 bpm vs. 98.7 ± 12.1 bpm; p<0.001) and lower mean systolic blood pressure (98.3 ± 11.5 mmHg vs. 112.8 ± 13.4 mmHg; p<0.001). Renal function was markedly worse in non-survivors, evidenced by a higher mean serum creatinine (1.92 ± 0.51 mg/dL vs. 1.41 ± 0.38 mg/dL; p<0.001). Echocardiographic parameters also differed; while left ventricular ejection fraction was comparable between groups (34.5% vs. 35.8%; p=0.572), non-survivors had a significantly larger mean IVC maximum diameter (2.51 ± 0.32 cm vs. 1.98 ± 0.29 cm; p<0.001) and, critically, a markedly lower mean IVC collapsibility index (28.4 ± 9.1% vs. 52.7 ± 12.3%; p<0.001). An ROC curve analysis was performed to determine the optimal prognostic cutoff for IVC-CI. The area under the curve (AUC) was 0.912 (95% CI: 0.850–0.974), indicating excellent discriminatory power. The maximum Youden index identified an IVC-CI of ≤35% as the optimal cutoff, yielding a sensitivity of 84.0%, specificity of 89.1%, positive predictive value of 80.8%, and negative predictive value of 91.1%. The proportion of patients with an IVC-CI ≤35% was 84.0% (21/25) in the non-survivor group compared to only 10.9% (6/55) in the survivor group, a difference that was highly statistically significant (p<0.001). To isolate the independent effect of IVC-CI, a multivariate logistic regression model was constructed, adjusting for age, systolic blood pressure, serum creatinine, and ejection fraction. In this model, an IVC-CI ≤35% emerged as the strongest independent predictor of in-hospital mortality, with an adjusted odds ratio of 15.42 (95% CI: 4.83–49.24; p<0.001). A secondary analysis revealed that a composite of IVC-CI ≤35% and serum creatinine >1.5 mg/dL was associated with the highest mortality risk (58.3%).

**Table 1:** Baseline characteristics of the study population

| Characteristic | Value |
|---|---|
| Age (years), Mean ± SD | 66.4 ± 10.8 |
| Male Sex, n (%) | 50 (62.5%) |
| **Comorbidities, n (%)** | |
| Hypertension | 66 (82.5%) |
| Diabetes mellitus | 38 (47.5%) |
| Ischemic heart disease | 52 (65.0%) |
| Chronic kidney disease | 15 (18.8%) |
| **Vital Signs, Mean ± SD** | |
| Heart rate (bpm) | 102.8 ± 14.0 |
| Systolic BP (mmHg) | 108.4 ± 14.5 |
| **Laboratory & Echo, Mean ± SD** | |
| Serum creatinine (mg/dL) | 1.57 ± 0.48 |
| LV Ejection fraction (%) | 35.4 ± 8.2 |

**Table 2:** Comparison of survivors and non-survivors

| Parameter | Survivors (n=55) | Non-survivors (n=25) | p-value |
|---|---|---|---|
| **Demographics** | | | |
| Age (years), Mean ± SD | 65.8 ± 11.2 | 67.8 ± 10.0 | 0.441 |
| Male, n (%) | 33 (60.0%) | 17 (68.0%) | 0.497 |
| **Comorbidities, n (%)** | | | |
| Hypertension | 44 (80.0%) | 22 (88.0%) | 0.537 |
| Ischemic heart disease | 34 (61.8%) | 18 (72.0%) | 0.377 |
| **Clinical parameters, Mean ± SD** | | | |
| Heart rate (bpm) | 98.7 ± 12.1 | 112.4 ± 14.2 | <0.001 |
| Systolic BP (mmHg) | 112.8 ± 13.4 | 98.3 ± 11.5 | <0.001 |
| Serum creatinine (mg/dL) | 1.41 ± 0.38 | 1.92 ± 0.51 | <0.001 |
| **Echocardiographic, Mean ± SD** | | | |
| LV Ejection fraction (%) | 35.8 ± 8.5 | 34.5 ± 7.6 | 0.572 |
| IVC Max diameter (cm) | 1.98 ± 0.29 | 2.51 ± 0.32 | <0.001 |
| IVC Collapsibility index (%) | 52.7 ± 12.3 | 28.4 ± 9.1 | <0.001 |

Data presented as Mean ± SD or n (%); p-values from independent t-test or Chi-square test

**Table 3:** Diagnostic performance of IVC-CI for predicting mortality

| Metric | Value |
|---|---|
| Area Under Curve (AUC) | 0.912 |
| 95% Confidence Interval | 0.850 – 0.974 |
| Optimal Cutoff (IVC-CI) | ≤35% |
| Sensitivity | 84.0% |
| Specificity | 89.1% |
| Positive Predictive Value | 80.8% |
| Negative Predictive Value | 91.1% |

**Table 4:** Distribution and association of IVC-CI ≤35% with mortality

| Group | IVC-CI ≤35% | IVC-CI >35% | p-value |
|---|---|---|---|
| | n (%) | | |
| Non-Survivors (n=25) | 21 (84.0%) | 4 (16.0%) | <0.001 |
| Survivors (n=55) | 6 (10.9%) | 49 (89.1%) | |

Chi-square test applied

**Table 5:** Multivariate logistic regression for predictors of in-hospital mortality

| Predictor | Adjusted OR | 95% CI | p-value |
|---|---|---|---|
| IVC-CI ≤35% | 15.42 | 4.83 – 49.24 | <0.001 |
| Age >70 years | 1.78 | 0.58 – 5.44 | 0.314 |
| Systolic BP <100 mmHg | 2.95 | 0.92 – 9.45 | 0.069 |
| Serum creatinine >1.5 mg/dL | 3.21 | 1.02 – 10.12 | 0.046 |
| LVEF <35% | 1.22 | 0.40 – 3.71 | 0.727 |

Model adjusted for all variables listed

**Table 6:** Mortality rate by IVC-CI and renal function profile

| Profile | n | In-hospital mortality, n (%) |
|---|---|---|
| IVC-CI ≤35% & Creatinine >1.5 mg/dL | 24 | 14 (58.3%) |
| IVC-CI ≤35% & Creatinine ≤1.5 mg/dL | 3 | 0 (0.0%) |
| IVC-CI >35% & Creatinine >1.5 mg/dL | 15 | 3 (20.0%) |
| IVC-CI >35% & Creatinine ≤1.5 mg/dL | 38 | 1 (2.6%) |

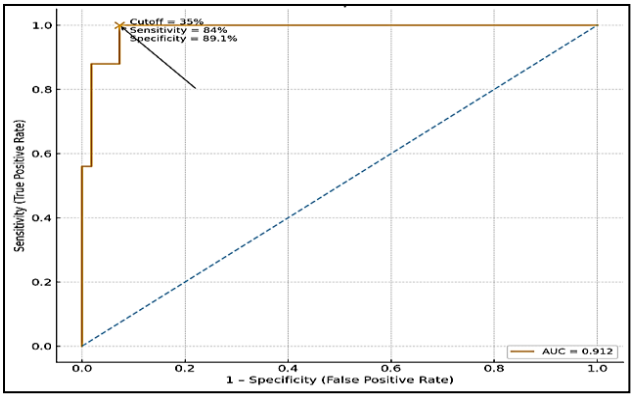


**Figure 1:** Receiver Operating Characteristic (ROC) curve of the inferior vena cava collapsibility index for predicting in-hospital mortality in severe acute decompensated heart failure

## Discussion

This prospective cohort study demonstrates that a reduced inferior vena cava collapsibility index (IVC-CI) at admission is a powerful and independent predictor of in-hospital mortality among patients with severe acute decompensated heart failure (ADHF) in a Bangladeshi population. The key finding—that an IVC-CI of ≤35% was associated with a more than 15-fold increased risk of death after multivariable adjustment—underscores the critical prognostic value of assessing systemic venous congestion at the bedside. The observed overall mortality rate of 31.3% aligns with, though is at the higher end of, the spectrum reported in other studies focusing on severe ADHF cohorts, reflecting the critically ill nature of our purposively selected NYHA class IV population.[3,14] The baseline characteristics of our cohort, dominated by hypertension and ischemic heart disease, are consistent with the prevalent etiology of heart failure in South Asia.[13] The lack of significant difference in age, sex, or left ventricular ejection fraction between survivors and non-survivors reinforces the concept that traditional demographic and systolic function parameters are insufficient for acute risk stratification in severe decompensation.[6,7] Instead, markers of hemodynamic compromise and end-organ perfusion, such as tachycardia, hypotension, and worsening renal function, were significantly associated with mortality, consistent with established pathophysiology.[8,15] Our study provides robust, quantitative evidence supporting the prognostic role of IVC assessment. The highly significant difference in mean IVC-CI between survivors (52.7%) and non-survivors (28.4%) and the excellent discriminatory power of the ROC curve (AUC 0.912) are compelling. These findings corroborate and extend previous work. A meta-analysis by Alqahtani *et al.* (2020) concluded that a less collapsible IVC was associated with adverse outcomes in acute HF, though pooled analyses included heterogeneous endpoints like readmission and combined mortality.[12] Our study specifically isolates in-hospital mortality as the endpoint in a severe, prospectively defined cohort. The optimal cutoff of ≤35% is clinically intuitive and aligns with echocardiographic guidelines defining an IVC-CI of <50% as suggestive of elevated right atrial pressure.[9] The high specificity (89.1%) and negative predictive value (91.1%) are particularly notable, suggesting that an IVC-CI >35% can identify a lower-risk subgroup even among severely symptomatic patients. The multivariate analysis confirming

IVC-CI ≤35% as the strongest independent predictor, with an adjusted odds ratio of 15.42, solidifies its role beyond a mere association with other markers of shock or renal dysfunction. This independence suggests that IVC collapsibility encapsulates a unique aspect of the hemodynamic insult—likely the severity and chronicity of systemic venous congestion—which directly contributes to progressive organ failure and death.[8,16] The synergy observed with renal impairment (serum creatinine >1.5 mg/dL), resulting in a mortality rate of 58.3%, highlights the vicious cycle of the cardiorenal syndrome and identifies a subgroup at extreme risk.[17] The strengths of this study include its prospective design, strict clinical definition of severe ADHF, and standardized echocardiographic protocol. However, limitations must be acknowledged. The single-center design and modest sample size, though adequate for the primary analysis, may limit generalizability. Purposive sampling introduces selection bias toward the most severe cases, which explains the high mortality but may not reflect the broader ADHF population. IVC measurements, while performed per protocol, are operator-dependent and influenced by respiration, though this reflects real-world practice.[18] We did not account for the specific inotrope or vasodilator therapies administered, which could influence outcomes. This study validates bedside IVC collapsibility assessment as a simple, rapid, and highly effective tool for early risk stratification in severe ADHF. An IVC-CI of ≤35% at admission is a potent indicator of a high risk of in-hospital death, independent of other clinical factors. Integrating this readily available ultrasonographic parameter into the initial evaluation can help clinicians identify patients who require the most intensive monitoring and aggressive therapeutic intervention, potentially improving resource allocation and clinical decision-making, especially in resource-constrained settings.[19]

## Limitations

This single-center study had a modest sample size and used purposive sampling of severe cases, which may limit generalizability. IVC measurements, while protocolized, are operator-dependent. The influence of specific therapeutic interventions on outcomes was not analyzed, representing a potential confounder.

## Conclusion

In patients with severe acute decompensated heart failure, an inferior vena cava collapsibility index (IVC-CI) of ≤35% at admission is a strong, independent predictor of in-hospital mortality. This simple, bedside ultrasonographic parameter provides critical prognostic information beyond traditional clinical and echocardiographic markers. Incorporating IVC-CI assessment into the initial evaluation can enhance early risk stratification, aiding in the timely identification of high-risk patients for more intensive management.

## Recommendation

We recommend integrating bedside IVC-CI assessment into the standard initial evaluation of severe ADHF for early mortality risk stratification. Future multicenter studies with larger cohorts should validate this cutoff and explore its impact on guiding therapy and improving outcomes.

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