

# Early and Serial Assessment of N-Terminal Pro B-Type Natriuretic Peptide and Inferior Vena Cava Diameter for Mortality Prediction in Acute Decompensated Heart Failure

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## ABSTRACT

**Background:** Accurate assessment of volume status in cases with acutely decompensated heart failure (ADHF) is crucial for prognostication and management. While brain natriuretic peptide (pro-BNP) and echocardiographic inferior vena cava (IVC) diameter are commonly used surrogate markers, their combined prognostic value has not been thoroughly established.

**Methods:** This prospective cohort study included 100 adults with ADHF and reduced ejection fraction (EF <40%). Pro-BNP levels and IVC diameter were assessed on admission and after 72 hours. The primary outcome was in-hospital mortality; secondary outcomes included complications and 30-day cardiovascular mortality. Repeated measures ANOVA, ROC analysis, and correlation testing were performed to evaluate predictive value.

**Results:** In-hospital mortality occurred in 21% of cases. Pro-BNP levels were significantly higher in non-survivors both on admission (median: 11,542 pg/mL vs. 6,350 pg/mL,  $p < 0.001$ ) and after 72 hours (3,695 pg/mL vs. 3,029 pg/mL,  $p < 0.001$ ). Similarly, IVC diameter was significantly greater in the mortality group at both time points (2.85 cm vs. 2.2 cm on admission,  $p < 0.001$ ; 2.15 cm vs. 1.9 cm after 72 hours,  $p = 0.004$ ). ROC analysis revealed strong predictive power for in-hospital mortality with admission Pro-BNP >8,856 pg/mL (AUC=0.89) and IVC diameter >2.55 cm (AUC=0.81). A combined model incorporating both parameters at admission yielded the highest diagnostic accuracy (AUC=0.89; NPV=95.4%).

**Conclusion:** Pro-BNP and IVC diameter are independent yet complementary predictors of in-hospital mortality in ADHF. Combined early assessment significantly enhances risk stratification and may guide intensive monitoring and therapeutic strategies.

## Introduction

Congestive heart failure (CHF) is one of the most prominent etiologies of morbidity and death globally with poor prognosis; it leads to

substantial reduction in quality of life as well as increased health care costs [1]. The diameter of the inferior vena cava (IVC) and the extent of its respiratory collapse function as echocardiographic surrogates are indicative of right atrial pressure, while brain natriuretic peptides (BNPs) are recognized laboratory biomarkers reflecting

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cardiac wall stress. Accurate estimation of the case's volume status is a fundamental cornerstone in the management of acutely decompensated heart failure cases (ADHF). It was previously noticed that cases with higher filling pressures before discharge had a greater rate of rehospitalization [2]. Sometimes signs of cardiac filling pressure on physical examination are difficult to elicit the accurate cardiac filling pressures, and invasive hemodynamic monitoring is sometimes too aggressive to apply in all cases and carries a risk due to its invasive nature and inherent risk of complications [3].

For this reason, BNP and echocardiographic assessments are commonly employed in routine clinical practice to evaluate cardiac filling pressures and volume status. Brain natriuretic peptide, along with its precursor N-terminal pro-B natriuretic peptide (NT-Pro BNP), is released in response to myocardial wall stretch and demonstrates a strong correlation with pulmonary capillary pressure [4].

The IVC diameter measured by echocardiography has numerous practical advantages: being easily measured bedside by any conventional ultrasound equipment involving handheld devices, it is easy and rapid to perform and requires little experience along with lung ultrasound [5]. Right-sided cardiac filling pressure and intravascular volume status are primarily reflected by the diameter of the IVC [6]; furthermore, the IVC diameter can be used as a quick indicator of lung congestion, unlike laboratory biomarkers, which rely on secretion, degradation, and excretion, and unlike lung ultrasonography, which may be used to measure the quantity of extravascular lung water and its change after therapy [7]. Ultrasound measurement of the IVC can also be utilized to follow up cases during in-hospital stay and guide and monitor diuretic therapy as demonstrated in the CAVA-ADHF study in which the ultrasound examination was confined to the IVC to make the intervention as simple and as straightforward as possible [8].

Nevertheless, we found insufficient evidence available on the relationship between IVC diameter and BNP levels and the short-term outcome and death in cases with acute decompensated heart failure (ADHF).

Accordingly, this study aims to evaluate the relationship between the levels of natriuretic peptides and echocardiographic-derived indices of right atrial pressure (IVC diameter and collapsibility) on admission and after 72 hours with in-hospital mortality in cases with ADHF.

## Methods

### Design and population

A prospective cohort investigation was performed on 100 cases presented to the emergency room (ER) or outpatient clinic in our hospital with ADHF and EF below 40%, i.e., HFrEF. Ethical approval for this study

was obtained from the Local Ethical Committee of the Faculty of Medicine, Kaser El Aini, Cairo University (Approval No. 12154;2019, dated 13.07.2023). Written informed consent was obtained from all participants prior to enrollment. The study was conducted in full accordance with the ethical principles outlined in the Declaration of Helsinki of the World Medical Association for research involving human subjects.

### Eligibility criteria

Cases aged between 18 and 80 years presenting with ADHF and an LVEF below 40%—i.e., HFrEF—were included in the study. Exclusion criteria encompassed cases with severe systolic dysfunction (EF <20%), those with left ventricular assist devices, individuals receiving positive pressure ventilation or requiring inotropic support, and cases with suspected or confirmed COVID-19 infection. Additional exclusions included cases with massive ascites, active malignancies undergoing chemotherapy, those on regular dialysis, cases with acute coronary syndrome, and individuals diagnosed with mediastinal syndromes. These conditions were excluded due to their potential to significantly influence hemodynamics, IVC diameter, cardiac filling pressures, or Pro-BNP levels.

### All cases were subjected to

#### Cardiac biomarker evaluation

Cardiac biomarkers, including NT-proBNP, within the first 24 hours of hospital admission and again at 72 hours. The analysis was performed using a sandwich enzyme-linked immunosorbent assay (ELISA) technique. Specifically, a polyclonal anti-NT-proBNP antibody was pre-coated onto 96-well microplates (Roche Diagnostics, Indianapolis, IN, USA). Standards and patient samples were added, allowing any NT-proBNP present to bind to the immobilized antibody. A horseradish peroxidase (HRP)-conjugated monoclonal anti-NT-proBNP antibody was used as the detection reagent. Following substrate development with tetramethylbenzidine (TMB) and the addition of a stop solution, optical density was measured at 450 nm. The absorbance was directly proportional to the NT-proBNP concentration.

#### Echocardiographic assessment

Transthoracic echocardiographic examinations were conducted within the first 24 hours of admission and repeated at 72 hours. Examinations were performed by an experienced echocardiographer following the recommendations of the American Society of Echocardiography (ASE) [9]. Particular attention was given to IVC diameter and its respiratory variation, assessed from the subcostal view approximately 1 cm from its junction with the right atrium. IVC collapsibility was categorized as  $\geq 50\%$ ,  $< 50\%$ , or no collapse, aiding in the evaluation of volume status and right atrial pressure [10].

### Therapeutic interventions and monitoring

Guideline-directed medical therapy tailored for heart failure with reduced ejection fraction (HFrEF) was administered, including diuretics, beta-blockers (initiated once patients achieved clinical euvolemia), angiotensin-converting enzyme (ACE) inhibitors, angiotensin receptor blockers (ARBs), angiotensin receptor-neprilysin inhibitors (ARNIs), sodium-glucose co-transporter 2 (SGLT-2) inhibitors, and mineralocorticoid receptor antagonists (MRAs) as clinically indicated. Notably, none of the enrolled patients required or received regular renal replacement therapy during the study period.

### Supportive monitoring and laboratory follow-up

In-hospital monitoring, including electrocardiography, oxygen saturation, fluid balance, and daily body weight assessment, adjusted as needed based on the clinical condition. Ultrafiltration was employed temporarily in cases demonstrating significant diuretic resistance. Routine laboratory evaluations, with particular focus on serum electrolytes and renal function parameters, were performed throughout the study period to ensure appropriate therapeutic adjustments and early detection of complications.

### Outcomes

The primary result was in-hospital mortality; the secondary result included the incidence of complications and cardiovascular mortality at one-month follow-up.

### Statistical analysis

All data were systematically collected, tabulated, and analyzed using the Statistical Package for the Social Sciences (SPSS), version 24, to ensure rigorous statistical evaluation. Qualitative data were expressed as frequency and percentage; quantitative data were expressed as median (IQR), mean, and standard deviation (SD). The t-test, Mann-Whitney U test (MW), and chi-square test were used. Correlation analysis was done using Pearson's correlation or Spearman's correlation when appropriate. The 2-way repeated measures ANOVA was performed to evaluate changes in pro-BNP and IVC diameter over time (at admission and after 72 hours) in relation to mortality. ROC was used to select the best cutoff value with the best sensitivity, specificity, PPV, and NPV. A p-value < 0.05 was considered significant, and a p-value < 0.001 was considered highly significant.

### Results

The studied group had a balanced gender distribution and a high prevalence of common cardiovascular risk factors, particularly hypertension (57%) and hyperlipidemia (48%). Dyspnea was the most frequent presenting symptom (55%), and oliguria was the most

common sign of heart failure (56%). The mean age was  $53.8 \pm 22.3$  years, with a mean hospital stay of  $9.6 \pm 2.1$  days. Laboratory findings showed elevated pro-BNP levels on admission ( $8274.4 \pm 3306.8$  pg/mL), which markedly decreased after 72 hours ( $3181.6 \pm 1446.0$  pg/mL). Echocardiographic parameters revealed a reduced ejection fraction ( $34.7 \pm 3.6\%$ ), increased ventricular dimensions, and moderately elevated RVSP ( $44.9 \pm 12.2$  mmHg), consistent with advanced cardiac dysfunction (Table 1).

**Table 1- Demographic, Clinical, Laboratory, and Echocardiographic Data of the Studied Group**

Variable	Value
Gender	
Male	44 (44%)
Female	56 (56%)
Risk Factors	
Diabetes Mellitus (DM)	43 (43%)
Hyperlipidemia	48 (48%)
Hypertension (HTN)	57 (57%)
Coronary Artery Disease (CAD)	30 (30%)
Prior Heart Failure	33 (33%)
COPD	38 (38%)
Bronchial Asthma (BA)	33 (33%)
Predominant Symptoms	
Dyspnea	55 (55%)
Chest Pain	48 (48%)
Orthopnea	46 (46%)
Heart Failure Signs	
Oliguria	56 (56%)
Pulmonary Rales	44 (44%)
Jugular Vein Distention	53 (53%)
Lower Limb Edema	39 (39%)
Diminished Conscious Level (DCL)	39 (39%)
Age (years)	$53.8 \pm 22.3$
Length of Stay (days)	$9.6 \pm 2.1$
APACHE II Score	$15.9 \pm 4.7$
SOFA Score	$2.4 \pm 1.2$
Weight (kg)	$73.6 \pm 8.4$
Height (m)	$1.60 \pm 0.10$
BMI (kg/m <sup>2</sup> )	$28.5 \pm 4.2$
Temperature (°C)	$37.3 \pm 0.25$
Respiratory Rate (/min)	$27.0 \pm 1.99$
Heart Rate (bpm)	$106.4 \pm 13.5$
MAP (mmHg)	$82.0 \pm 7.4$
SpO <sub>2</sub> (%)	$87.3 \pm 6.6$
Laboratory Data on Admission	
Pro-BNP (pg/mL)	$8274.4 \pm 3306.8$
Troponin I (ng/mL)	$0.019 \pm 0.012$
CK Total (IU/L)	$385.2 \pm 149.7$
CK-MB (IU/L)	$39.58 \pm 13.2$
Pro-BNP (pg/mL) after 72 hours	$3181.6 \pm 1446.0$

Echocardiographic Data on Admission	
LVEDD (mm)	52.4 ± 10.1
LVESD (mm)	43.4 ± 8.4
LVEDV (ml)	136.4 ± 38.3
LVESV (ml)	79.1 ± 16.1
Ejection Fraction (EF %) – Simpson’s method	34.7 ± 3.6
LA (mm) – PLAX view	39.0 ± 6.4
RVSP (mmHg)	44.9 ± 12.2
TAPSE (mm)	18.9 ± 4.5
IVC Diameter (cm)	2.4 ± 0.52
IVC Diameter (cm) after 72 hours	1.9 ± 0.39

APACHE II: Acute Physiology and Chronic Health Evaluation II; BA: Bronchial Asthma; BMI: Body Mass Index; CAD: Coronary Artery Disease; CK: Creatine Kinase; MB: Myocardial Band; cm: Centimeter; COPD: Chronic Obstructive Pulmonary Disease; DCL: Diminished Conscious Level; DM: Diabetes Mellitus; EF: Ejection Fraction; HTN: Hypertension; HR: Heart Rate; IU/L: International Units per Liter; IVC: Inferior Vena Cava; LA: Left Atrium; LVEDD: Left Ventricular End-Diastolic Diameter; LVEDV: Left Ventricular End-Diastolic Volume; LVESD: Left Ventricular End-Systolic Diameter; LVESV: Left Ventricular End-Systolic Volume; MAP: Mean Arterial Pressure; PLAX: Parasternal Long Axis; Pro-BNP: N-terminal pro B-type Natriuretic Peptide; RR: Respiratory Rate; RVSP: Right Ventricular Systolic Pressure; SOFA: Sequential Organ Failure Assessment; SpO<sub>2</sub>: Peripheral Oxygen Saturation; TAPSE: Tricuspid Annular Plane Systolic Excursion.

Over half of the patients (52%) showed clinical improvement during the initial hospital course, while 48% experienced early deterioration. In-hospital all-cause mortality was 21%, with cardiovascular-related deaths accounting for 31.6%. Complications occurred in 65.8% of the studied group, indicating a high burden of adverse outcomes during hospitalization (Table 2).

Patients who died during hospitalization had significantly higher median Pro-BNP levels on both admission (11,542 vs. 6,350 pg/mL,  $p < 0.001$ ) and at discharge (3,695 vs. 3,029 pg/mL,  $p < 0.001$ ). Additionally, IVC diameter was significantly greater

among non-survivors at admission (2.85 vs. 2.2 cm,  $p < 0.001$ ) and after 72 hours (2.15 vs. 1.9 cm,  $p = 0.004$ ). No statistically significant differences were observed between survivors and non-survivors in serum creatinine, liver enzymes, cardiac enzymes, echocardiographic volumes, ejection fraction, or other hemodynamic parameters (Table 3).

There was no significant association between IVC respiratory collapse categories and in-hospital mortality ( $p = 0.939$ ), with a similar distribution of collapse grades observed among survivors and non-survivors.

ROC curve analysis demonstrated that Pro-BNP levels on admission  $>8856$  pg/mL predicted in-hospital mortality with an AUC of 0.890, 85.7% sensitivity, and 78.5% specificity ( $p < 0.001$ ), while levels  $>3244$  pg/mL at 72 hours showed an AUC of 0.75, 80.9% sensitivity, and 55.7% specificity ( $p = 0.0004$ ) (Figure 1 A). For IVC diameter, a threshold  $>2.55$  cm at admission yielded an AUC of 0.81 with 71.4% sensitivity and 75.9% specificity ( $p < 0.001$ ), and  $>1.95$  cm at 72 hours showed an AUC of 0.70 with 71.4% sensitivity and 59.5% specificity ( $p = 0.0038$ ) (Figure 1 B).

The combined use of Pro-BNP and IVC diameter on admission demonstrated strong predictive accuracy for in-hospital mortality (AUC: 0.89), which slightly decreased at 72 hours (AUC: 0.79) (Figure 2A). The full model incorporating all four variables achieved the highest performance (AUC: 0.897), indicating enhanced prognostic utility when integrating both biomarkers at both time points (Figure 2B).

We also found a strong positive correlation between the pro-BNP level on admission and echocardiography-derived IVC diameter ( $r = 0.78$ ;  $P < 0.001$ ) (Figure 3A) and a weak positive correlation between the echocardiography-derived IVC diameter and pro-BNP level after 72 hours ( $r = 0.36$ ;  $P < 0.001$ ) (Figure 3B).

**Table 2- In hospital course, complications and cardiovascular mortality at one month follow up after discharge in the studied cases**

Variable	Category	Number (%)
Initial Hospital Course	Improved	52 (52.0%)
	Initial Deterioration	48 (48.0%)
In-Hospital Mortality (All-Cause)	Yes	21 (21.0%)
	No	79 (79.0%)
Complications	Yes	52 (65.8%)
	No	27 (34.2%)
Cardiovascular Mortality	Yes	25 (31.6%)
	No	54 (68.4%)

**Table 3- Comparison between the mortality and non-mortality groups in the studied cases**

Variable	Mortality		P value
	No (N = 79)	Yes (N = 21)	
Pro-BNP on Admission (pg/mL)	6350 (5634–8700)	11542 (9290.5–13896)	$< 0.001^*$
Pro-BNP on Discharge (pg/mL)	3029 (2054–3498)	3695 (3258–5818)	$< 0.001^*$
IVC Diameter on Admission (cm)	2.2 (1.9–2.5)	2.85 (2.5–3.2)	$< 0.001^*$
IVC Diameter at 72 Hours (cm)	1.9 (1.6–2.0)	2.15 (1.85–2.3)	0.004*

Data are presented as median (IQR), \*: statistically significant P value  $< 0.05$ .

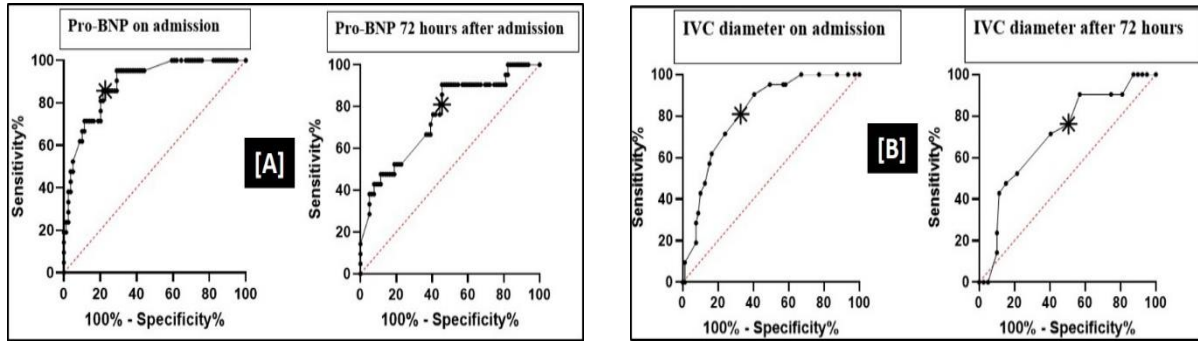


Figure 1- ROC curves of [A] Pro-BNP and [B] IVC diameter on admission and after 72 hours for predicting in-hospital mortality.

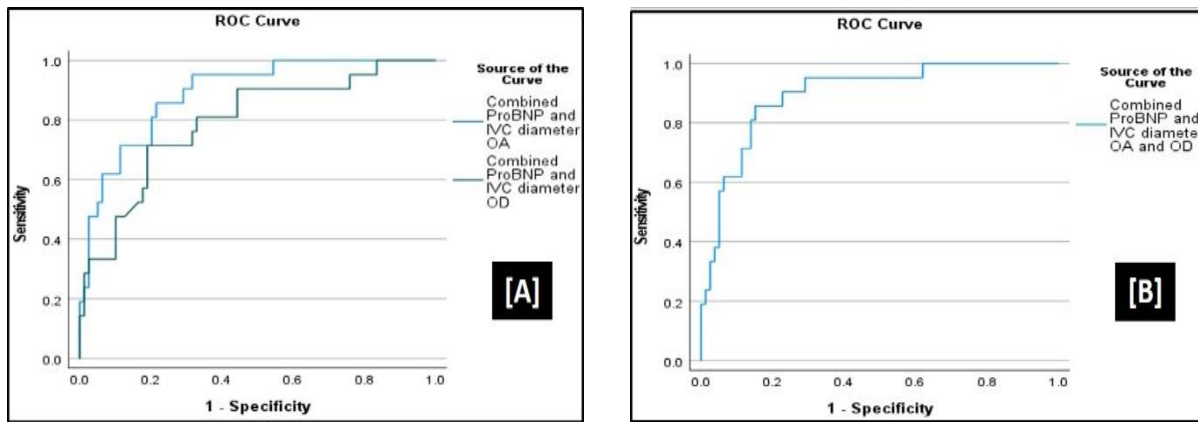


Figure 2- ROC curves showing [A] combined Pro-BNP and IVC on admission, and [B] full model combining Pro-BNP and IVC on admission and after 72 hours for mortality prediction

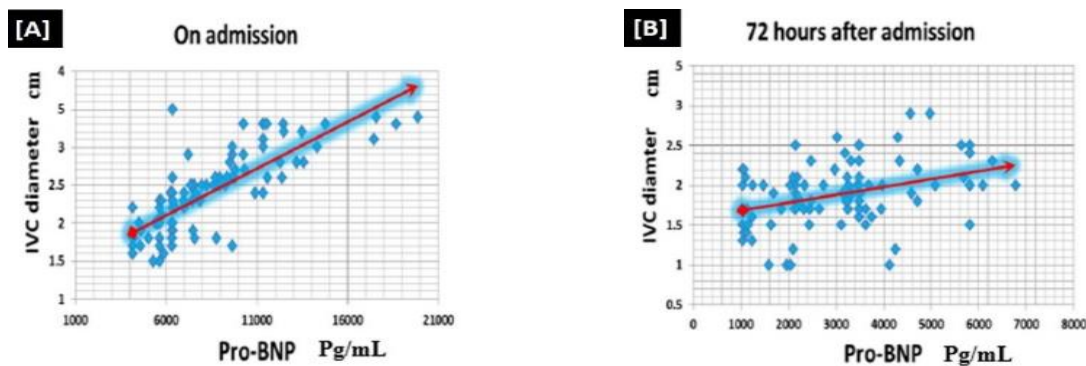


Figure 3- Correlation between Pro-BNP level and IVC diameter (cm) [A] on admission & [B] 72 hours after admission.

**Discussion**

The prevalence of HF worldwide is continuing to rise, as well as the number of hospitalizations by ADHF, leading to the classification of CHF as one of the primary causes of morbidity and mortality globally [11]. Precise case determination of volume status is a crucial key in the

treatment plan of cases with ADHF owing to the fact that cases with raised filling pressures had higher rehospitalization rates.

BNP and N-terminal pro. BNP are excreted secondary to increased heart muscle cell wall stress and strain, and they can be used for the diagnosis and monitoring of cases with HF. Despite the fact that natriuretic peptide

levels increase in relation to illness severity, it is extremely important to clarify that this does not happen solely in HF. Furthermore, their levels are influenced by other parameters, such as body mass and renal function [12].

Ultrasound measurement of the IVC diameter serves as an alternative surrogate for the case's intravascular volume status and can be used to accurately reflect the right atrial pressure. Furthermore, variations in IVC diameter correspond closely with changes in pulmonary capillary wedge pressure [13].

So, we thought to study the relationship of both parameters (IVC diameter and pro BNP) with mortality in cases with ADHF.

A recent comprehensive study on the frequency, incidence, and survival of HF cases found a broad mortality rate range from 4% to 45%. This wide variation in the mortality rate between the studies can be explained by the different HF etiologies, phenotypes, disease stages on cases' admission, and cases' associated comorbidities in the various trials [14]; this may make it difficult to find a common, single parameter as a sensitive risk marker for the cases' prognosis.

In our study, we did not find a significant difference in the demographic data between the mortality and survivor groups. We were surprised to find that diabetes was more prevalent in the survivors group than the mortality group (P value of 0.046). Most prior research consistently reveals poorer outcomes in persons with diabetes [15]. This inconsistency can be explained by the fact that our study's P value was marginally significant (0.046), and the number of cases was limited, with no long-term follow-up.

Our cohort showed a significant difference in BNP levels on admission and 72 hours after admission (P value < 0.001) compared to the non-mortality group. A pro-BNP level > 8856 pg/ml on admission and > 3244 pg/ml 72 hours later might predict hospital mortality (AUC = 0.89, P value < 0.001) and (AUC = 0.75, P value = 0.0004), respectively. These findings suggest that ADHF cases with greater pro-BNP levels on admission had a higher risk of early death. As a result, close monitoring and extensive secondary prevention for these cases may change the prediction of unfavorable outcomes and aid in the identification of cases that might benefit from fast, aggressive and effective therapy.

Our findings are consistent with the results of the research published by Januzzi et al. on 702 cases with acute HF to determine appropriate pro-BNP cut-points for predicting short-term aggressive, mortality and found that among those who died in follow-up, the median pro-BNP concentration at presentation was 3277 pg/mL (interquartile range, 1086-9868 pg/mL); this was substantially higher than the pro-BNP concentration in those who survived, which was 299 pg/mL (interquartile range, 71-1807; P<.001) [16, 17].

Similarly, Bhatia et al. [18] conducted a study on cases with ADHF requiring emergent in-hospital admission and found a significantly higher median Pro-BNP value on admission in cases with fatal outcomes [3670 (IQR-2745 to 3980)] than in those who survived beyond five days of hospitalization [1340 (IQR- 987 to 1670)]. Both studies verify the predictive function of pro-BNP level on admission in short-term outcome and mortality.

On the contrary, Khanam et al. [19] did not find a significant difference in median BNP upon admission between his study groups (dead: 872.1 (571.3 to 1587.9) pg/ml; alive: 808.4 (497.9 to 1325.3) pg/ml, p = 0.165). However, follow-up BNP levels were considerably higher in the mortality group, 617.7 (319.1 to 1260) pg/ml, compared to the survival group, 282.5 (136.2 to 487.3) pg/ml (p < 0.001). They also noticed that the median percentage change in the BNP levels was substantially different between the two groups (alive: -66.1 (-81.3 to -37.4)%; dead: -31.1 (-57 to 36.2)%, p < 0.001).

Our cohort's recorded echocardiographic data revealed a significant difference in IVC diameter on admission and 72 hours after hospitalization between both groups (P values <0.001 and 0.004), respectively. Furthermore, we found that an IVC diameter on admission > 2.55 cm and an IVC diameter after 72 hours > 1.95 cm can be used as a cutoff value to predict hospital mortality (AUC = 0.81, P value < 0.001 and AUC = 0.7, P value = 0.0038), respectively. As a result, our findings contribute to the expanding body of evidence that dilated IVC may indicate worse outcomes in people with ADHF. This can be linked to higher filling cardiac pressures and pulmonary arterial hypertension that may exacerbate pre-existing right ventricular failure and increase tricuspid regurgitation, resulting in systemic venous congestion.

Our findings support the data of Jobs et al. [8] on 1101 ADHF cases, who found a substantial increase in all-cause mortality in cases with dilated IVC (p < 0.01). Furthermore, Lee et al. [20] investigated whether a dilated IVC in cases with a history of advanced systolic HF and a 30% EF was a prognostic predictor, concluding that an IVC diameter greater than 2.1 cm predicted cardiovascular mortality or hospitalization in these cases. Cubo-Romano et al. [21] found that a slightly lower cutoff value of 1.9 cm of IVC diameter on admission was an accurate predictor of mortality with 100% sensitivity and 38% specificity.

Surprisingly, Haag et al. [22] did not find a connection between IVC maximal diameter and all-cause mortality or readmission. This could be explained by their study's small sample size (34 of 130 (26.2%) cases died during follow-up) and the fact that the prognostic value of congestion markers may change, and most likely increase, during the course from admission to discharge of an ADHF hospitalization to the subsequent chronic stable phase. Furthermore, our findings contradicted

those of Josa-Laorden et al. [23], who showed that there were no significant differences in total mortality between IVC dilatation ( $p = .156$ ) and collapse ( $p = 0.480$ ). In our study, combining both pro-BNP and IVC at admission yielded an AUC of 0.890, reinforcing the additive predictive value of assessing both markers together. The combination measured after 72 hours demonstrated slightly lower performance (AUC = 0.79), yet remained statistically significant. A composite model incorporating four variables (Pro PNB and IVC at admission and after 72 hours) yielded the highest AUC (0.897), indicating that an integrated evaluation improves prediction accuracy. Notably, the highest negative predictive value (NPV) was 95.7% in this full combined model, suggesting that cases with consistently low values across all markers are highly unlikely to experience in-hospital mortality.

To our knowledge, no previous study has specifically examined the prognostic value of combining pro-BNP and IVC diameter in HF cases. However, Liu et al. [24] investigated the effect of the IVC collapse index (CI) on the prognosis of 98 cases with ADHF. They found that the AUCs for NT-proBNP and IVC-CI in predicting short-term prognosis were 0.806 and 0.847, respectively. He also reported a relationship between NT-proBNP, IVC-CI, and volume indices.

We detected a high positive correlation ( $r=0.78$ ) between the Pro BNP level on admission and the echocardiographically determined IVC diameter ( $P$  value  $< 0.001$ ). After 72 hours, we detected a slight positive connection ( $r=0.36$ ) between the echocardiographically determined IVC diameter and Pro BNP level ( $P$  value  $< 0.001$ ). In research expressly designed to answer this question, Hebl et al. [25] discovered only a positive, albeit minor, connection between IVC diameter and BNP ( $r = 0.24$ , 95% CI: 0.01-0.44;  $P = 0.03$ ) and NT-Pro BNP ( $r = 0.27$ , 95% CI: 0.05-0.47;  $P = 0.01$ ). Our study only included cases with HF<sub>rEF</sub> ( $EF < 40\%$ ) and excluded cases with HF and mid-range ejection fraction (HF<sub>mEF</sub>) or HF and preserved ejection fraction (HF<sub>pEF</sub>), which may explain the difference in results.

Our study has certain limitations: being a single-center study with a relatively limited number of cases. Only in-hospital and short-term events were conducted.

## Conclusion

This study demonstrates that both pro-BNP and IVC diameter are reliable and complementary predictors of in-hospital mortality. The early assessment (at admission) of these markers provides stronger prognostic value than measurements taken after 72 hours. Importantly, the combination of both parameters significantly improves predictive accuracy, and a composite model incorporating all four variables yields the highest diagnostic performance (AUC=0.897).

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