

COMPARATIVE ASSESSMENT OF NT-PROBNP IN AMI PATIENTS WITH AND WITHOUT LV DYSFUNCTION: A STUDY FROM KANACHUR INSTITUTE OF MEDICAL SCIENCES

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ABSTRACT

Background: NT-proBNP is an extremely reliable biomarker of myocardial stress, often cited in the evaluation of left ventricular (LV) dysfunction, but the comparative significance in those with a preserved versus impaired LV function in AMI patients is a relevant clinical question. **Aim:** To compare NT-proBNP levels in AMI patients with respect to LV function and to determine if it is a reliable early marker of myocardial dysfunction. **Materials and Methods:** This observational cross-sectional research study included 100 AMI patients from the Kanachur Institute of Medical Sciences, who all underwent clinical evaluation, routine investigations and echocardiography. Patients were subdivided into AMI without LV dysfunction (LVEF $\geq 50\%$) and AMI with LV dysfunction (LVEF $< 50\%$). NT-proBNP was assessed within 24 hours of admission and compared between groups, and correlated against LVEF. **Result:** NT-proBNP was significantly greater in AMI patients with LV dysfunction compared to AMI patients with preserved LV function ($p < 0.001$). A strong inverse correlation was determined between NT-proBNP and LVEF as the peptide was greater with increasing ventricular dysfunction. **Discussion:** The results suggest that NT-proBNP is a reliable biomarker that segregates AMI patients with respect to LV function and suggests myocardial strain early in presentations, suggesting a role in risk stratification and decision-making. **Conclusion:** NT-proBNP is a valuable marker for identifying LV dysfunction in AMI and may enhance early assessment and management, particularly in resource-limited cardiac care settings.

INTRODUCTION

Acute myocardial infarction (AMI) continues to be one of the leading causes of cardiovascular-related morbidity and mortality around the world, and represents a significant burden in low- and middle-income countries where burdens of ischemic heart disease have increased as a result of epidemiological transitions.^[1] Rapidly identifying high-risk AMI patients is an essential focus of contemporary care to improve survival outcomes, since timely recognition of areas of myocardial damage and corresponding treatment are directly associated with prognosis. Of the many prognostic factors available, left ventricular (LV) dysfunction is one of the most important determinants of adverse events.^[2] Following an AMI, reduced LV ejection fraction (LVEF) is strongly associated with complications such as acute heart failure, malignant arrhythmia, cardiogenic shock, and all cause long-term mortality risk post-AMI.^[3] Thus,

a timely and accurate assessment of LV function is an important aspect of clinical decision making, resource allocation, and risk stratification in the acute cardiac care setting.^[4]

In recent years, biochemical markers have assumed an even larger and complementary role to imaging-based assessments of cardiac function. Among the biochemical markers, N-terminal pro-B-type natriuretic peptide (NT-proBNP) has demonstrated consistency as a reliable biomarker of myocardial wall stress, ischemia, and ventricular dysfunction. NT-proBNP is the inactive fragment resulting from the cleavage of proBNP, which is released from ventricular myocytes with volume expansion and pressure overload.^[5,6] NT-proBNP has a longer plasma half-life than BNP, and therefore can remain elevated for a longer time after myocardial injury, making it a reliable early biomarker for diagnosis and prognosis in acute myocardial infarction (AMI).^[7]

Many clinical trials and studies have confirmed the prognostic value of NT-proBNP in predicting heart failure, mortality, future ischemic events, and complications in the post-infarction patient. Elevated NT-proBNP levels correlate closely with the severity of ventricular impairment and provide insight into myocardial strain in the first few hours after presentation of AMI.^[8,9] Additionally, NT-proBNP can supplement or even, in some circumstances, stand in for the initial echocardiographic assessment, especially in the setting of delayed or unavailable imaging-only considerations in some less resourced healthcare systems.^[10] While the clinical utility of NT-proBNP in chronic heart failure is well established, the differentiation of AMI patients with preserved vs impaired LV function using NT-proBNP is an area of growing interest.^[11]

Although NT-proBNP has proven helpful in many biochemical contexts, populations have demonstrated differences in NT-proBNP cutoff values, distribution, and relation to echocardiographic parameters. There is a paucity of literature, however, that directly examines NT-proBNP from South Indian tertiary care centers serving primarily rural and semi-urban populations, who may have different cardiovascular risk factors, access to care, and disease presentation. Therefore, a local evaluation of NT-proBNP in patients with AMI and varying demographic and clinical characteristics may provide relevant knowledge and inform decision pathways in this context.^[12]

In response to these deliberations, this study aims to investigate NT-proBNP in AMI patients with and without LV dysfunction to assess NT-proBNP as an early marker of myocardial dysfunction. Its diagnostic potential in this regional clinical context may also help improve early risk stratification, treatment decisions, and acute and post-acute cardiac care outcomes.^[13]

MATERIALS AND METHODS

Study Design and Setting

This comparative cross-sectional study was conducted at the Department of General Medicine and Cardiology of Kanachur Institute of Medical Sciences, Mangalore for a period of 18 months, involving patients diagnosed with acute myocardial infarction.

Sample Size and Sampling Procedure

Through the method of consecutive sampling, 100 patients with AMI were recruited in the study. After screening patients based on inclusion and exclusion criteria, the patients were finally included.

Inclusion Criteria

- Age of 18 years and older.
- Diagnosis of acute myocardial infarction based on clinical presentation, ECG changes, and elevated cardiac enzyme tests.
- NT-proBNP could be measured within 24 hours of admission.

- Transthoracic echocardiography was performed within 48 hours of admission.

Exclusion Criteria

- Heart failure or cardiomyopathy diagnosed prior to enrollment.
- Chronic kidney disease classed as eGFR < 60 mL/min/1.73m².
- Severe hepatic impairment.
- Chronic lung disease leading to secondary pulmonary hypertension.
- Recent major surgery or trauma.
- Patients receiving long-term medications that modify natriuretic peptides.

Study Participant Grouping

Patients were categorized into two subgroups by echocardiography determined LV ejection fraction (LVEF):

- Group A: AMI without LV dysfunction and LVEF ≥ 50%
- Group B: AMI with LV dysfunction and LVEF < 50%

Clinical Evaluation and Investigations

All patients underwent a detailed clinical evaluation including history, physical examination, and cardiovascular assessment. Routine investigations included:

- Complete blood count
- Renal and liver function tests
- Blood glucose
- Lipid profile
- Cardiac myocyte injury markers (Troponin I/T, CK-MB)
- A standard 12-lead ECG and chest X-ray were performed on each patient.

Echocardiography

Transthoracic echocardiographic assessment was performed on all patients using Philips Affiniti or similar imaging equipment. Information obtained from echocardiography included:

- LV ejection fraction
- Regional wall motion abnormalities
- LV dimensions
- Other structural abnormalities

LVEF was calculated using Simpson's biplane method.

NT-proBNP Measurement

Venous blood samples were taken within the first 24 hours of presenting to the hospital. NT-proBNP levels were measured using an electrochemiluminescence immunoassay (ECLIA), with values recorded in pg/mL.

Outcome Measures

Primary outcome:

NT-proBNP levels difference between groups.

Secondary outcomes:

- Correlation of NT-proBNP levels and LVEF.
- Association of NT-proBNP levels and clinical severity measures.

Statistical Analysis

Data were analyzed with the use of SPSS, version 26. Continuous variables were reported as mean \pm standard deviation and categorical as percentages. Independent t-test was used to examine differences in NT-proBNP levels between the groups. Pearson correlation coefficient was utilized to assess the relationship between NT-proBNP levels and LVEF. A p-value of <0.05 was considered statistically significant.

RESULTS

In the comparative study of 100 subjects presenting with AMI, NT-proBNP levels proved to be extremely helpful in distinguishing those subjects with left ventricular (LV) dysfunction from those with no LV dysfunction. Subjects in Group B (AMI with LV dysfunction) had significantly higher NT-proBNP levels (5420 ± 1640 pg/mL) than Group A (AMI without LV dysfunction) (1280 ± 540 pg/mL) ($p < 0.001$). The difference in NT-proBNP levels correlated with clinical severity, as Group B had

more dyspnea, hypotension, pulmonary congestion, and arrhythmias. There was a greater range of ages in the LV dysfunction category; however, the analysis of the demographic factors of age/gender were statistically insignificant, adding more evidence that elevated NT-proBNP was associated with myocardial impairment and not simply from inherent differences throughout the population sample.

The echocardiographic evaluation continued to support these findings, as patients with LV dysfunction showed greater regional wall motion abnormalities, LV dilatation and diastolic dysfunction. The strong inverse correlation ($r = -0.71$, $p < 0.001$) between NT-proBNP with LVEF suggests that higher levels of NT-proBNP (and therefore reduced ventricular contractility) were consistent. Overall, these findings demonstrate that NT-proBNP is a sensitive and specific marker for early detection of LV dysfunction in patients with AMI with strong associations with structural and functional impairment associated with the cardiac tissue function. Thus, its clinical utility and relevance for early risk stratification appear supported.

Table 1: Baseline Demographic Characteristics of Study Population (N = 100)

No.	Statement	Extremely Disagree (%)	Disagree (%)	Neutral (%)	Agree (%)	Completely Agree (%)
1	I prefer practicing in a skill lab before performing procedures on patients	0	2.7%	10.8%	32.4%	54.1%
2	I believe that mentors should be friendly and helpful during teaching sessions	2.7%	2.7%	5.4%	27%	62.2%
3	Engaging in skill lab practice boosts motivation	2.7%	0	2.7%	37.8%	56.8%
4	Students require guidance and attention from mentors during skill lab sessions	0	2.7%	13.5%	24.3%	59.5%
5	It is beneficial as procedures are demonstrated to make them easier for students	2.7%	0	8.1%	18.9%	70.3%
6	I've developed a professional approach through skill lab experiences	2.7%	0	5.4%	29.7%	62.2%
7	Active participation in skill labs improves performances during patient care	2.7%	0	8.1%	24.3%	64.9%
8	Skill laboratory training enhances confidence	2.7%	0	5.4%	29.7%	62.2%
9	Skill lab training is essential	2.7%	0	8.1%	24.3%	64.9%
10	Preparing mentally for skill lab learning is important	2.7%	0	13.5%	21.6%	62.2%
11	Starting skill lab training early in syllabus is crucial	10.8%	2.7%	13.5%	27%	45.9%
12	Skill lab training proves to be useful in integrating cognitive domain with psychomotor domain	2.7%	0	10.8%	24.3%	62.2%
13	Feedback from teachers about skill performance is valuable	2.7%	0	10.8%	21.6%	64.9%
14	Practical skills are better learned through hands-on training	2.7%	2.7%	10.8%	13.5%	70.3%
15	I anticipate being able to perform clinical skills under supervision by the end of the course	0	0	8.1%	37.8%	54.1%
16	I aim to apply clinical skills independently on patients	2.7%	0	2.7%	35.1%	59.5%
17	Skill lab practice instils a sense of security during the learning process	5.4%	0	5.4%	32.4%	56.8%
18	Practical skill training leads to better learning outcomes	2.7%	0	5.4%	29.7%	62.2%

Table 1 provides a summary of the baseline demographic characteristics of the AMI patients who participated in the study. There were no differences in age and sex distribution between the two groups (Group A - preserved LV function; Group B - LV dysfunction) meaning the baseline characteristics of

the two groups were similar and appropriate for a comparison. [Table 1]

Table 2: Clinical Presentation in AMI Patients

Clinical Parameter	Group A (mean ± SD)	Group B (mean ± SD)
Dyspnea (NYHA II–III)	12 (23.1%)	29 (60.4%)
Hypotension	6 (11.5%)	16 (33.3%)
Pulmonary Congestion	8 (15.4%)	23 (47.9%)
Arrhythmias	5 (9.6%)	13 (27.1%)

Table 2 shows the distribution of the main clinical features in both samples. Group B, which had LV dysfunction, reported more dyspnea, hypotension, pulmonary congestion, and arrhythmias than Group

A. These findings indicate a more severe clinical state in patients with heart failure and reduced ejection fraction. [Table 2]

Table 3: Comparison of NT-proBNP Levels Between Groups

Parameter	Group A (n=52)	Group B (n=48)	p-value
NT-proBNP (pg/mL) Mean ± SD	1,280 ± 540	5,420 ± 1,640	< 0.001

The NT-proBNP levels were significantly higher in Group B than in Group A, a very significant difference ($p < 0.001$). This supports the use of NT-

proBNP to identify AMI patients with LV dysfunction or preserved ventricular function. [Table 3]

Table 4: NT-proBNP Levels Across Age Categories

Age Group (years)	Group A (mean ± SD)	Group B (mean ± SD)
< 50	1,050 ± 420	4,800 ± 1,500
50–65	1,310 ± 560	5,510 ± 1,700
> 65	1,520 ± 590	5,780 ± 1,650

Table 4 shows that NT-proBNP values progressively increase with age in both groups. It shows that although NT-proBNP is trending upward with increasing age, Group B consistently displayed

significantly higher NT-proBNP values than Group A in all the age groups, which further supports NT-proBNP as a marker of LV dysfunction. [Table 4]

Table 5: Gender-wise Distribution of NT-proBNP

Gender	Group A (mean ± SD)	Group B (mean ± SD)
Male	1,260 ± 520	5,390 ± 1,620
Female	1,300 ± 560	5,460 ± 1,670

Table 5 compares NT-proBNP levels between male and female patients. No statistically significant differences were seen between genders in either

group, and so gender appeared to have no effect on NT-proBNP levels in this study population. [Table 5]

Table 6: Correlation Between NT-proBNP and Left Ventricular Ejection Fraction (LVEF)

Variable	Correlation Coefficient (r)	p-value
NT-proBNP vs LVEF	-0.71	< 0.001

A robust, statistically significant inverse relationship ($r = -0.71$) was reported between NT-proBNP and LVEF, such that elevated NT-proBNP values corresponded to progressively impaired contractility

of the ventricle, which further supports NT-proBNP as a sensitive biomarker of myocardial dysfunction. [Table 6]

Table 7: Echocardiographic Findings in Study Groups

Echocardiographic Parameter	Group A N = 52	Group B N = 48
Mean LVEF (%)	≥ 50%	< 50%
Extent of Regional Wall Motion Abnormalities (RWMA)	Mild	Moderate–Severe
LV Dilatation	6 (11.5%)	22 (45.8%)
Diastolic Dysfunction	10 (19.2%)	31 (64.6%)

Table 7 makes a comparison of the primary echocardiographic characteristics of the two groups; however, patients with LV dysfunction had not only greater regional wall motion abnormalities, but also

had a higher frequency of LV dilation and higher grade diastolic dysfunction than patients in Group A, indicating greater degree of structural and functional impairment of the heart. [Table 7]

Table 8: Severity of Clinical and Echocardiographic Parameters by NT-proBNP Quartiles

NT-proBNP Quartile	Dyspnea	LV Dilatation	RWMA Severity
Q1 (<1500 pg/mL)	8%	4%	Mild
Q2 (1500–3000)	18%	12%	Mild–Moderate
Q3 (3000–6000)	52%	38%	Moderate
Q4 (>6000)	71%	62%	Moderate–Severe

Table 8 shows the trend of increasing clinical severity with increasing quartiles of NT-proBNP. Higher quartiles of NT-proBNP had higher incidence of dyspnea, LV dilatation, and more severe RWMA.

Collectively, this graded pattern underscores the utility of NT-proBNP as a marker of myocardial dysfunction severity. [Table 8]

Table 9: Logistic Regression Predicting LV Dysfunction

Predictor	Odds Ratio (OR)	95% CI	p-value
NT-proBNP (>3000 pg/mL)	4.8	2.1–10.9	< 0.001
Age (>60 years)	1.4	0.8–2.5	0.21
Male gender	0.9	0.4–1.9	0.82

Table 9 shows the logistic regression models predicting factors associated with LV dysfunction. In the study, only NT-proBNP levels > 3000 pg/mL were an independent predictor of LV dysfunction. Age > 60 years and male gender were not found to be statistically significant predictors. This reinforces the strong predictor capabilities of NT-proBNP in identifying LV impairment in AMI patients. [Table 9]

dysfunction. Table 9 demonstrates that NT-proBNP levels >3000 pg/mL were a strong independent predictor of LV dysfunction with age >60 years and male gender not being significant predictors. Collectively, these data show that NT-proBNP levels reliably identify and quantify myocardial dysfunction in AMI patients.

DISCUSSION

According to Table 1, the groups were comparable at baseline in regard to age and gender. Table 2 displays the clinical presentation reflecting significantly increased dyspnea, hypotension, pulmonary congestion and arrhythmias in Group B indicating greater severity of hemodynamic derangement. Table 3 shows the NT-proBNP levels in patients with LV dysfunction and shows that it has diagnostic capability to differentiate the groups. Table 4 shows that NT-proBNP increased with age, and remained elevated in Group B in every age range. Table 5 shows there is no significant difference in NT-proBNP values by gender, indicating that NT-proBNP behaves similarly for males and females. Table 6 indicates that there is a significant inverse correlation ($r = -0.71$) between NT-proBNP and LVEF, showing that NT-proBNP is a reliable marker of deteriorating systolic function. Table 7 reiterates the strong relationship between NT-proBNP and LVEF and also shows that Group B had greater echocardiographic evidence of abnormality including more extensive regional wall motion abnormality, increased LV dilatation, and more pronounced diastolic dysfunction, compared to Group A. Table 8 presents the relationship between NT-proBNP level (stratified by quartile) and other clinical severity indicators, revealing that increasing NT-proBNP was associated with greater severity of dyspnea, LV dilatation, and RWMA. This shows a clear dose–response relationship between increasing NT-proBNP and increasing degree of clinical

In this research, it was found that the concentrations of NT-proBNP performed very well in distinguishing AMI patients with left ventricular (LV) dysfunction from those with LV preserved function, thus confirming the biomarker’s sensitivity to diagnose myocardial impairment. The mean NT-proBNP for the LV dysfunction group (Group B) was significantly higher (5420 ± 1640 pg/mL) than the preserved-LVEF (1280 ± 540 pg/mL) with p-value < 0.001. This observation is very similar to previous studies where elevated natriuretic peptides following myocardial infarction were associated with increased myocardial infarct size, reduced systolic function and worse outcomes. For instance, Maeder et al systematically demonstrated that early measured NT-proBNP following AMI inversely correlated with ejection fraction measured by cardiac MRI and predicted long-term functional recovery.^[14]

In our cohort, clinical observation further validated this biochemical distinction; patients with LV dysfunction more consistently presented with dyspnea, hypotension, pulmonary congestion, and arrhythmias. This is consistent with prior studies in high-risk MI cohorts. For instance, a substudy of the PARADISE-MI trial, Khambhati et al., indicated that higher NT-proBNP levels measured in the first week were significantly associated with incident heart failure, cardiovascular event-related death, and other events independent of troponin and clinical

covariates.^[8] These findings have further supported findings to characterize complexity that.

Similar to our results of strong inverse correlation of NT-proBNP and LVEF ($r = -0.71$), these observations have consistently been reported previously. For instance, Arbel et al. examined STEMI patients undergoing primary PCI, and found similar relationships between early NT-proBNP assessment, baseline LV dyssynchrony, and subsequent loss of LVEF over time.^[9] Additionally, the prognostic value of NT-proBNP is again reaffirmed in the long term; Vidic and colleagues indicated that NT-proBNP at 6 months following AMI was a significant predictor of long-term cardiac events, including heart failure and recurrent ischemia.^[15] Collectively, these findings support dual role of NT-proBNP as a biomarker of current ventricular dysfunction yet allowing for future adverse remodeling.

However, we want to highlight some nuances of NT-proBNP dynamics worth discussing. In the present study, NT-proBNP levels were already significantly elevated early in the AMI course and correlated with echocardiographic abnormality. However, the time of measure may affect how this biomarker is interpreted; for example, previous work by Ueland et al. (2011) has demonstrated that NT-proBNP obtained at 3 months post AMI had greater associations with long term ejection fraction and infarct size (assessed with MRI) than NT-proBNP values obtained within days of the event.^[16] In this regard, early NT-proBNP provides diagnostic value. Although, the timing of measurement and comparison to acute values may alter interpretation. An additional significant factor to consider is the role of demographic and comorbid factors on NT-proBNP levels. Our age-stratified data showed significantly raised peptide levels with increasing age (Table 4) but this did not account completely for the difference in NT-proBNP between groups across all age bands. This suggests that whilst NT-proBNP levels may change with age, the observed gradient of the biomarker is not simply related to participants' ages. This concurs with Wang et al. (2023), who assessed a large population of AMI patients and found that levels of NT-proBNP did vary according to other factors such as age, renal function and BMI but that the peptide remained a strong independent predictor of mortality.^[17] Additionally, we did not find a gender effect in our cohort (Table 5) and this too aligns with previous older observational data in post-MI outpatients where no clear gender-dependent difference in NT-proBNP's association with LVEF was noted.^[18]

The echocardiographic findings lend structural validation to the biochemical findings. Patients in group B had more extensive regional wall motion disturbances, a higher frequency of LV dilation, and greater diastolic dysfunction. The structural-functional relationship is clinically meaningful: although NT-proBNP may well serve as a marker of current systolic dysfunction, it may also reflect

diastolic stress and/or remodeling. Therefore, the incorporation of natriuretic peptides into risk models may provide a better understanding of risk stratification beyond that afforded by ejection fraction values. Older mechanistic studies have demonstrated that higher NT-proBNP at baseline will predict worsening systolic and diastolic function later on.^[11]

It is worthwhile to discuss some limitations of our study. First, while we only assessed NT-proBNP at one time point (within 24 hours of admission), repeating measurements may have richer information about the dynamics of the biomarker as well as remodelling. Second, while the $n = 100$ sample size is adequate, larger multicentre studies would yield greater power and external validity. Third, we did not fully account for renal function or other comorbidities (e.g. BMI) that can potentially affect NT-proBNP levels. Finally, while not specifically designed for this, echocardiography is widely available, and gold standard imaging such as cardiac MRI would provide a more precise characterization of infarct size and remodelling, but was not practical in this setting.

The clinical implications of our findings have major significance. The timing of NT-proBNP measurement in patients with AMI could afford clinicians the possibility of quickly identifying patients with significant LV impairment who would undergo aggressive monitoring, early heart failure treatments, or clinical oversight that is tailored to each patient. Furthermore, in resource-limited environments, where advanced imaging/referrals may not be feasible or timely, NT-proBNP could function as a practical biomarker surrogate for risk management. Additionally, threshold (ex., $>3,000$ pg/mL using our logistic regression) could be established as part of local protocols as a means to manage clinical decisions (although this would require validation at larger cohort). Overall, our study reinforces the utility of NT-proBNP as sensitive clinical relevant biomarker to detect LV dysfunction early in patients with AMI. NT-proBNP levels demonstrated a strong and direct correlation to echocardiographic imaging of structural-functional impairment and is consistent with the prior literature on prognostic value or biomarkers in AMI. Ongoing research should focus on employing serial measures, optimal cut-off values, and further integrating NT-proBNP into clinical algorithms to improve outcomes in patients with AMI.

CONCLUSION

This study demonstrates that NT-proBNP as a strong, reliable biomarker of left ventricular dysfunction in a population of patients with acute myocardial infarction. In patients with left ventricular dysfunction in this study, NT-proBNP was substantially and statistically higher in relation to clinical severity and echocardiographic abnormality,

demonstrating its importance for both diagnosis and prognosis.

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