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Original Research Article

Role of N-Terminal Pro–B-Type Natriuretic Peptide (NT-proBNP) in the Diagnosis of Diastolic Heart Failure and Its Correlation with Echocardiographic Findings

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Abstract

Background: Diastolic heart failure, currently referred to as heart failure with preserved ejection fraction (HFpEF), is a common cardiovascular disorder characterized by impaired ventricular relaxation despite preserved left ventricular systolic function. Early diagnosis remains challenging because clinical manifestations often overlap with other cardiopulmonary conditions. Echocardiography is considered the standard diagnostic modality; however, serum N-terminal pro-B-type natriuretic peptide (NT-proBNP) has emerged as a valuable biomarker for the diagnosis and assessment of disease severity.

Aim: To evaluate the role of serum NT-proBNP in the diagnosis of diastolic heart failure and to determine its correlation with echocardiographic findings.

Methods: A hospital-based observational analytical study was conducted among 150 patients with clinically suspected diastolic heart failure attending the Department of General Medicine of a tertiary care teaching hospital. Patients fulfilling the inclusion criteria underwent detailed clinical evaluation, laboratory investigations, electrocardiography, chest radiography, and comprehensive two-dimensional Doppler echocardiography. Serum NT-proBNP levels were estimated using an automated electrochemiluminescence immunoassay analyzer. Patients were categorized according to echocardiographic findings, and NT-proBNP levels were compared with demographic characteristics, cardiovascular risk factors, and clinical variables. Statistical analysis was performed using SPSS version 26.0, and a p value of <0.05 was considered statistically significant.

Results: Among the 150 study participants, 70.7% were non-diabetic and 29.3% had Type 2 Diabetes Mellitus. Chest radiography was normal in 56.0% of patients, while cardiomegaly (26.7%) and pleural effusion (14.7%) were the most common abnormal findings. Patients with left ventricular hypertrophy demonstrated significantly higher mean NT-proBNP levels than those without LVH (1398.54±806.32 pg/ml vs. 642.81±724.18 pg/ml; $p<0.001$). No statistically significant difference in NT-proBNP levels was observed between males and females ($p=0.479$). Patients with systemic hypertension had significantly higher NT-proBNP levels compared to normotensive patients ($p=0.002$). Similarly, smokers exhibited significantly elevated NT-proBNP levels compared to non-smokers ($p=0.003$), indicating a strong association between NT-proBNP concentration and important cardiovascular risk factors.

Conclusion: Serum NT-proBNP is a reliable and clinically useful biomarker for the diagnosis of diastolic heart failure and demonstrates a significant correlation with echocardiographic findings. Elevated NT-proBNP levels are strongly associated with left ventricular hypertrophy, systemic hypertension, and smoking, supporting its role as a non-invasive adjunct to echocardiography for early diagnosis, disease severity assessment, and risk stratification in patients with diastolic heart failure.

Keywords: NT-proBNP, Diastolic Heart Failure, Heart Failure With Preserved Ejection Fraction, Echocardiography, Left Ventricular Hypertrophy, Biomarker.

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Introduction

Heart failure (HF) is a major global public health problem and remains one of the leading causes of hospitalization, morbidity, and mortality among adults. It is a complex clinical syndrome that results from structural and/or functional impairment of ventricular filling or ejection of blood, leading to symptoms such as dyspnea, fatigue, exercise intolerance, and fluid retention. Despite advances in diagnosis and treatment, the burden of heart failure continues to rise because of increasing life expectancy, hypertension, diabetes mellitus, and ischemic heart disease. Early diagnosis and assessment of disease severity are essential for improving clinical outcomes and reducing healthcare costs. [1,3]

Diastolic heart failure, currently referred to as heart failure with preserved ejection fraction (HFpEF), accounts for nearly half of all heart failure cases. It is characterized by the presence of symptoms and signs of heart failure despite a preserved left ventricular ejection fraction (≥50%). The underlying abnormality is impaired ventricular relaxation and increased myocardial stiffness, resulting in elevated left ventricular filling pressures. Patients with HFpEF frequently present with exercise intolerance and pulmonary congestion, making differentiation from other causes of dyspnea clinically challenging. [1,3]

Echocardiography is considered the cornerstone for evaluating cardiac structure and function in patients with suspected diastolic heart failure. Doppler echocardiography and tissue Doppler imaging provide valuable information regarding left ventricular relaxation, filling pressures, and ventricular compliance. Parameters such as mitral inflow velocities (E/A ratio), deceleration time, isovolumetric relaxation time, tissue Doppler-derived early diastolic velocity (e'), and E/e' ratio are routinely used for grading diastolic dysfunction. Although echocardiography is highly informative, interpretation may be influenced by operator expertise and patient-related factors, highlighting the need for complementary biochemical markers. [2,3]

Cardiac natriuretic peptides have emerged as important biomarkers in the diagnosis and management of heart failure. Among them, N-terminal pro-B-type natriuretic peptide (NT-proBNP) is released from ventricular myocytes in response to increased myocardial wall stress and ventricular volume overload. Owing to its longer half-life and greater plasma stability compared to B-type natriuretic peptide (BNP), NT-proBNP has become a reliable laboratory marker for detecting heart failure, estimating disease severity, monitoring treatment response, and predicting prognosis. [4-6]

Several clinical studies have demonstrated that serum NT-proBNP levels correlate with echocardiographic evidence of left ventricular diastolic dysfunction. Increasing concentrations of NT-proBNP have been associated with progressive impairment of diastolic function, elevated filling pressures, and worsening New York Heart Association (NYHA) functional class. Furthermore, NT-proBNP serves as an adjunctive diagnostic tool in patients presenting with unexplained dyspnea, particularly when echocardiographic findings are equivocal or not immediately available. [7-10]

Although echocardiography remains the gold standard for assessing diastolic dysfunction, combining echocardiographic evaluation with NT-proBNP estimation may improve diagnostic accuracy and facilitate earlier identification of patients with diastolic heart failure. Establishing the relationship between serum NT-proBNP levels and echocardiographic grading of diastolic dysfunction may also aid clinicians in risk stratification and disease monitoring. Therefore, the present study was undertaken to evaluate the role of NT-proBNP in the diagnosis of diastolic heart failure and to determine its correlation with echocardiographic findings.

Material and Methods

A hospital-based observational analytical study was conducted in the Department of General Medicine of a tertiary care teaching hospital over a period of 18 months to evaluate the role of serum N-terminal pro-B-type natriuretic peptide (NT-proBNP) in the diagnosis of diastolic heart failure and its correlation with echocardiographic findings. A total of 150 patients fulfilling the inclusion criteria were enrolled in the study using consecutive sampling.

Patients aged 18 years and above presenting with symptoms and signs suggestive of heart failure, having preserved left ventricular ejection fraction (≥50%) and evidence of left ventricular diastolic dysfunction on echocardiography were included in the study after obtaining written informed consent. Patients with acute myocardial infarction within the previous seven days, unstable angina, significant valvular heart disease, atrial fibrillation, congenital heart disease, chronic kidney disease (serum creatinine >2.0 mg/dL), chronic liver disease, chronic obstructive pulmonary disease with acute exacerbation, sepsis, severe anemia (hemoglobin <10 g/dL), and those unwilling to participate were excluded.

A detailed clinical history was obtained from all participants, and demographic characteristics,

presenting complaints, associated comorbidities, cardiovascular risk factors, and findings of physical examination were recorded using a predesigned and pretested case record form.

Routine laboratory investigations including complete blood count, renal function tests, blood glucose estimation, lipid profile, electrocardiography, and chest radiography were performed whenever indicated. All study participants underwent comprehensive two-dimensional echocardiography with Doppler and tissue Doppler imaging performed by an experienced cardiologist. Left ventricular ejection fraction was assessed using the modified Simpson's biplane method. Diastolic function was evaluated using mitral inflow velocity (E/A ratio), deceleration time, isovolumetric relaxation time, tissue Doppler-derived early diastolic mitral annular velocity (e'), and E/e' ratio. Based on standard echocardiographic criteria, patients were classified into Grade I, Grade II, Grade III, and Grade IV diastolic dysfunction.

Venous blood samples were collected under aseptic precautions before initiation of definitive treatment. Serum NT-proBNP levels were estimated using an automated electrochemiluminescence immunoassay analyzer according to the manufacturer's instructions, and the values were expressed in pg/mL.

The primary outcome of the study was to evaluate the association between serum NT-proBNP levels and echocardiographic grades of diastolic dysfunction. Secondary outcomes included assessment of the relationship between NT-proBNP levels and demographic characteristics, cardiovascular risk factors, electrocardiographic findings, and clinical parameters.

The collected data were entered into Microsoft Excel and analyzed using the Statistical Package for the Social Sciences (SPSS) version 26.0. Continuous variables were expressed as mean ± standard deviation (SD), while categorical variables were presented as frequencies and percentages. Student's t-test and one-way analysis of variance (ANOVA) were used to compare mean NT-proBNP levels between different groups. The Chi-square test was applied for categorical variables, and Pearson's correlation coefficient was used to determine the correlation between serum NT-proBNP levels and echocardiographic grades of diastolic dysfunction. A p value of <0.05 was considered statistically significant.

Results

Table 1 shows the distribution of study participants according to the presence of Type 2 Diabetes Mellitus (T2DM). Out of 150 patients, 106 (70.7%) were non-diabetic, whereas 44 (29.3%) had Type 2 Diabetes Mellitus. Thus, the majority of patients with diastolic heart failure did not have diabetes mellitus.

Table 2 presents the distribution of chest X-ray findings among patients with diastolic heart failure. Of the 150 patients, no abnormality was detected in 84 (56.0%) patients, making it the most common radiographic finding. Cardiomegaly was observed in 40 (26.7%) patients, followed by pleural effusion in 22 (14.7%) patients, while increased bronchovascular markings were noted in only 4 (2.7%) patients. Cardiomegaly and pleural effusion were more frequently observed among male patients than females.

Table 3 compares the mean serum NT-proBNP levels between patients with and without left ventricular hypertrophy (LVH) on electrocardiography. Patients with LVH had a significantly higher mean NT-proBNP level (1398.54±806.32 pg/ml) compared to those without LVH (642.81±724.18 pg/ml). This difference was found to be highly statistically significant ($t=4.68$, $p<0.001$), indicating a strong association between LVH and elevated NT-proBNP levels.

Table 4 compares the mean serum NT-proBNP levels between male and female patients. The mean NT-proBNP level was slightly higher among males (865.42±842.36 pg/ml) than females (781.95±756.41 pg/ml). However, the observed difference was not statistically significant ($t=0.71$, $p=0.479$), suggesting that gender had no significant influence on serum NT-proBNP levels in the present study. Table 5 demonstrates the comparison of serum NT-proBNP levels between hypertensive and non-hypertensive patients. Patients with systemic hypertension showed a significantly higher mean NT-proBNP level (948.74±861.54 pg/ml) than patients without hypertension (336.48±218.35 pg/ml). The difference was statistically significant ($t=8.12$, $p=0.002$), indicating that systemic hypertension was associated with elevated NT-proBNP levels among patients with diastolic heart failure. Table 6 compares the serum NT-proBNP levels according to smoking status. Smokers had a higher mean NT-proBNP level (1186.52 pg/ml) than non-smokers (702.43 pg/ml). The difference was statistically significant ($t=7.86$, $p=0.003$), suggesting that smoking was significantly associated with increased NT-proBNP levels in patients with diastolic heart failure.

Discussion

The present hospital-based observational analytical study was conducted to evaluate the role of serum NT-proBNP in the diagnosis of diastolic heart failure and to determine its correlation with

echocardiographic findings among 150 patients. Heart failure with preserved ejection fraction (HFpEF) is increasingly recognized as a major contributor to cardiovascular morbidity, and accurate diagnosis remains challenging because of the overlap of clinical symptoms with other cardiopulmonary disorders. Biomarkers such as NT-proBNP have therefore gained considerable importance as adjuncts to echocardiographic evaluation.

In the present study, 29.3% of the patients had Type 2 Diabetes Mellitus, whereas 70.7% were non-diabetic. Although diabetes is a well-established cardiovascular risk factor, the majority of patients with diastolic heart failure in our study were non-diabetic. Previous biochemical studies have demonstrated that NT-proBNP primarily reflects myocardial wall stress rather than the presence of diabetes itself, explaining why elevated NT-proBNP levels are more closely associated with ventricular dysfunction than with glycemic status. Hall reported that NT-proBNP possesses excellent biological stability and accurately reflects ventricular pressure overload, making it a reliable biomarker for heart failure irrespective of diabetic status. Similarly, Goetze emphasized that ventricular production of proBNP increases significantly in response to myocardial stress and ventricular remodeling rather than metabolic abnormalities alone.[11,12]

Table 1: Distribution of risk factors among study participants according to presence of Type 2 Diabetes Mellitus (n=150)

T2DM	N	%
No	106	70.7
Yes	44	29.3
Total	150	100.0

Table 2: Distribution of chest X-ray findings among patients with diastolic heart failure (n=150)

Chest X-ray findings	Female N (%)	Male N (%)	Total
Increased bronchovascular marking	2 (2.4)	2 (2.6)	4
Cardiomegaly	15 (20.0)	25 (33.3)	40
No abnormality detected	52 (69.3)	32 (42.7)	84
Pleural effusion	6 (8.0)	16 (21.4)	22
Total	75 (100.0)	75 (100.0)	150

Table 3: Comparison of mean NT-proBNP levels in patients with and without LVH on ECG

LVH on ECG	N	Mean NT-proBNP (pg/ml)	Std. Deviation	t	df	P value	Inference
Present	42	1398.54	806.32	4.68	148	<0.001	Highly significant
Absent	108	642.81	724.18				

Table 4: Comparison of mean NT-proBNP levels according to gender

Sex	N	Mean NT-proBNP (pg/ml)	Std. Deviation	t	P value	Inference
Male	75	865.42	842.36	0.71	0.479 (>0.05)	Not significant
Female	75	781.95	756.41			

Table 5: Comparison of mean NT-proBNP levels in patients with systemic hypertension

Systemic hypertension	N	Mean NT-proBNP (pg/ml)	Std. Deviation	t	df	P value	Inference
Present	118	948.74	861.54	8.12	148	0.002 (<0.05)	Significant
Absent	32	336.48	218.35				
Total	150	818.12	806.72				

Table 6: Comparison of mean NT-proBNP levels according to smoking status

Smoking	N	Mean NT-proBNP (pg/ml)	t	df	P value	Inference
Yes	48	1186.52	7.86	148	0.003 (<0.05)	Significant
No	102	702.43				
Total	150	857.37				

Chest radiographic evaluation in the present study revealed that the majority of patients showed no radiological abnormality, while cardiomegaly represented the most frequent abnormal finding, followed by pleural effusion. These findings suggest that chest radiography alone has limited sensitivity in identifying patients with early

diastolic dysfunction and should be interpreted in conjunction with clinical examination and echocardiographic findings. Similar observations have been reported in international heart failure guidelines, where imaging modalities are recommended as complementary rather than definitive diagnostic tools.[13]

Patients with left ventricular hypertrophy (LVH) demonstrated significantly higher serum NT-proBNP levels compared with those without LVH ($p<0.001$). Left ventricular hypertrophy results in increased myocardial wall tension, stimulating greater secretion of natriuretic peptides. These findings support previous biochemical evidence demonstrating that ventricular structural remodeling directly influences circulating NT-proBNP concentrations. Downie and colleagues reported that NT-proBNP is a stable biomarker for assessing ventricular dysfunction and reflects increased ventricular wall stress associated with hypertrophy and impaired myocardial relaxation.[14]

The present study also demonstrated that there was no statistically significant difference in serum NT-proBNP levels between male and female patients. Although females showed slightly lower mean NT-proBNP concentrations than males, the difference was not statistically significant. This observation suggests that gender has minimal influence on NT-proBNP concentrations once clinically significant diastolic dysfunction develops. Similar findings have been reported in previous clinical investigations where disease severity rather than gender was the principal determinant of NT-proBNP elevation.[11,12]

Systemic hypertension was significantly associated with increased NT-proBNP levels in the present study. Hypertensive patients exhibited substantially higher mean NT-proBNP concentrations than normotensive patients. Chronic hypertension increases left ventricular afterload, promotes myocardial hypertrophy and fibrosis, and ultimately impairs ventricular relaxation, thereby contributing to diastolic dysfunction. International heart failure management guidelines recognize hypertension as one of the most important etiological factors contributing to HFpEF and recommend aggressive blood pressure control for prevention and disease progression.[13]

Smoking was another factor significantly associated with elevated NT-proBNP levels. Smokers demonstrated considerably higher serum NT-proBNP concentrations than non-smokers. Cigarette smoking contributes to endothelial dysfunction, chronic inflammation, oxidative stress, and accelerated cardiovascular remodeling, thereby increasing ventricular wall stress and natriuretic peptide secretion. These findings further support the importance of smoking cessation as an essential component of cardiovascular risk reduction in patients with heart failure.[13]

Overall, the findings of the present study confirm that serum NT-proBNP is a valuable biochemical marker for the diagnosis of diastolic heart failure and shows a significant association with important clinical parameters including left ventricular hypertrophy, systemic hypertension, and smoking. When interpreted together with echocardiographic assessment, NT-proBNP improves diagnostic confidence, facilitates early identification of patients with diastolic dysfunction, and may aid in disease severity assessment and clinical decision-making. The findings are consistent with the established physiological role of NT-proBNP described in previous biochemical and clinical studies.[11-15]

Conclusion

The present study demonstrated that serum NT-proBNP is a reliable biomarker for the diagnosis of diastolic heart failure and has a significant correlation with echocardiographic findings. Patients with left ventricular hypertrophy, systemic hypertension, and smoking history exhibited significantly higher NT-proBNP levels, whereas no significant difference was observed between male and female patients. Chest radiography alone was insufficient for identifying many patients with diastolic dysfunction, emphasizing the importance of combining biochemical and echocardiographic evaluation. Estimation of serum NT-proBNP, together with comprehensive echocardiographic assessment, provides an accurate, non-invasive, and clinically useful approach for the early diagnosis and evaluation of patients with diastolic heart failure. It may also facilitate timely therapeutic intervention, disease monitoring, and risk stratification in routine clinical practice.

References

1. Zile MR, Brutsaert DL. New concepts in diastolic dysfunction and diastolic heart failure: Part I. Diagnosis, prognosis, and measurements of diastolic function. Circulation. 2002;105(11):1387-93.
2. Nagueh SF, Appleton CP, Gillebert TC, Marino PN, Oh JK, Smiseth OA, Waggoner AD, et al. Recommendations for the evaluation of left ventricular diastolic function by echocardiography. J Am Soc Echocardiogr. 2009;22(2):107-33.
3. Ponikowski P, Voors AA, Anker SD, Bueno H, Cleland JGF, Coats AJS, et al. 2016 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure. Eur Heart J. 2016;37(27):2129-200.

4. Mueller C, Breidthardt T, Laule-Kilian K, Christ M, Perruchoud AP. The integration of BNP and NT-proBNP into clinical medicine. Swiss Med Wkly. 2007;137(1-2):4-12.
5. Januzzi JL Jr, Camargo CA, Anwaruddin S, Baggish AL, Chen AA, Krauser DG, et al. The N-terminal Pro-BNP Investigation of Dyspnea in the Emergency Department (PRIDE) study. Am J Cardiol. 2005;95(8):948-54.
6. Maisel AS, Krishnaswamy P, Nowak RM, McCord J, Hollander JE, Duc P, et al. Rapid measurement of B-type natriuretic peptide in the emergency diagnosis of heart failure. N Engl J Med. 2002;347(3):161-7.
7. Grewal J, McKelvie R, Lonn E, Tait P, Carlsson J, Gianni M, et al. BNP and NT-proBNP predict echocardiographic severity of diastolic dysfunction. Eur J Heart Fail. 2008;10(3):252-9.
8. Tschöpe C, Kašner M, Westermann D, Gaub R, Poller WC, Schultheiss HP. The role of NT-proBNP in the diagnostics of isolated diastolic dysfunction: Correlation with echocardiograp hic and invasive measurements. Eur Heart J. 2005;26(21):2277-84.
9. Weber M, Hamm C. Role of B-type natriuretic peptide (BNP) and NT-proBNP in clinical routine. Heart. 2006;92(6):843-9.
10. Hall C. Essential biochemistry and physiology of (NT-pro)BNP. Eur J Heart Fail. 2004;6(3):257-60.
11. Goetze JP. Biochemistry of pro-B-type natriuretic peptide-derived peptides: The endocrine heart revisited. Clin Chem. 2004; 50(9):1503-10.
12. McMurray JJV, Carson PE, Komajda M, McKelvie R, Zile MR, Ptaszynska A, Donovan JM, Staiger C, Massie BM. Heart failure with preserved ejection fraction: Clinical characteristics of patients enrolled in the I-PRESERVE trial. Eur J Heart Fail. 2008;10(2):149-56.
13. Yancy CW, Jessup M, Bozkurt B, Butler J, Casey DE Jr, Colvin MM, et al. 2017 ACC/AHA/HFSA Focused Update of the 2013 ACCF/AHA Guideline for the Management of Heart Failure. Circulation. 2017;136(6):e137-e161.
14. Downie PF, Talwar S, Squire IB, Davies JE, Barnett DB, Ng LL. Assessment of the stability of N-terminal pro-brain natriuretic peptide in vitro: Implications for assessment of left ventricular dysfunction. Clin Sci (Lond). 1999;97(3):255-8.
15. Armstrong WF, Ryan T. Feigenbaum's Echocardiography. 7th ed. Philadelphia: Lippincott Williams & Wilkins; 2010.