ORIGINAL ARTICLE OPEN ACCESS

Utility of NT-proBNP in the Diagnosis of Heart Failure Phenotypes in a Sub-Saharan African Population

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ARTICLE HISTORY

Received 02/06/2025
Accepted 03/12/2025
Published 01/08/2026

ABSTRACT

Background:

N-terminal pro-B-type Natriuretic Peptide (NT-proBNP) is a cornerstone in the diagnostic algorithm for heart failure (HF). However, its behaviour across different HF phenotypes, particularly in sub-Saharan African (SSA) populations where patient characteristics and HF aetiologies differ, is not well described. This study evaluated the utility of NT-proBNP in diagnosing HF phenotypes in a Nigerian cohort using the 2016 European Society of Cardiology (ESC) criteria.

Methods:

In a prospective observational study, 130 adults with HF were classified into HF with reduced ejection fraction (HFrEF, LVEF<40%), mid-range EF (HFmrEF, LVEF 40-49%), and preserved EF (HFpEF, LVEF≥50%) based on comprehensive assessment including echocardiography and clinical evaluation. Plasma NT-proBNP was measured using a high-sensitivity ELISA. The Kruskal-Wallis test was used to compare NT-proBNP levels across phenotypes. Diagnostic performance was assessed by the proportion of patients meeting the ESC NT-proBNP threshold (>125 pg/mL for ambulatory and >300 pg/mL for hospitalized patients).

Results:

The median NT-proBNP level for the entire cohort was 3800.0 pg/mL (IQR: 2900.0-4500.0). All 130 patients (100%) had NT-proBNP levels above the ESC diagnostic thresholds. There was no significant difference in median NT-proBNP levels between the three phenotypes (HFrEF: 3900.0 pg/mL [IQR: 3075-4600], HFmrEF: 3700.0 pg/mL [IQR: 2275-3700], HFpEF: 3850.0 pg/mL [IQR: 2700-4325]; p=0.461). The biomarker was equally elevated in both inpatient and outpatient settings.

Conclusion:

NT-proBNP is a highly sensitive biomarker for confirming the diagnosis of HF in this SSA population, with 100% of patients exceeding the recommended ESC cut-offs. Contrary to findings from other regions, NT-proBNP levels were uniformly and markedly elevated across all HF phenotypes, showing no discriminatory value between HFrEF, HFmrEF, and HFpEF. This suggests that while NT-proBNP is indispensable for ruling out HF in this setting, its level cannot be used to infer the underlying phenotype.

N-terminal pro-B-type Natriuretic Peptide | Heart Failure | Diagnosis | Phenotypes | Sub-Saharan Africa | Echocardiography | Biomarker

Introduction

Heart failure (HF) is a global pandemic with a particularly devastating impact in sub-Saharan Africa (SSA), where it affects a younger population and is driven by a unique mix of aetiologies, including hypertensive heart disease, cardiomyopathies, and rheumatic heart disease. [1, 2] Accurate diagnosis is the critical first step in effective management. However, the clinical diagnosis of HF can be challenging, as its signs and symptoms are non-specific and can be mimicked by other conditions such as obesity, chronic lung disease, and renal failure.[3]

The incorporation of natriuretic peptides (NPs), B-type natriuretic peptide (BNP) and its N-terminal pro-hormone (NT-proBNP), into diagnostic guidelines has been a major advance in the management of HF.[4] These peptides are released primarily from the cardiac ventricles in response to volume expansion and pressure overload, making them sensitive biomarkers of myocardial wall stress.[5] The 2016 guidelines of the European Society of Cardiology (ESC) mandate elevated levels of NPs as a necessary criterion for the diagnosis of HF, especially for the challenging entity of heart failure with preserved ejection fraction (HFpEF).[6] The recommended diagnostic thresholds are NT-proBNP >125 pg/mL for ambulatory patients and >300 pg/mL for those who are hospitalized.[6]

While the diagnostic utility of NPs is well-established in high-income countries, their performance characteristics can be influenced by age, renal function, body mass index (BMI), and the aetiology of HF.[7,8] In SSA, where the HF profile is distinct, data on NT-proBNP are scarce. Patients present at a younger age, and comorbidities like obesity and renal disease may have differing prevalences and impacts.[9] Furthermore, studies from other regions typically show a gradient of NP levels, with the highest concentrations in HF with reduced ejection fraction (HFrEF) and lower, though still elevated, levels in HFpEF.[10,11]This is pathophysiologically plausible because HFrEF, characterized by dilated ventricles, generates greater wall stress than the thick-walled, small-cavity ventricles often seen in HFpEF.[12]

It remains unclear whether this NP gradient between phenotypes holds true in SSA populations. A previous study from Nigeria reported significantly elevated BNP levels in HF patients compared to controls but did not perform a detailed cross-phenotype analysis.[13] Understanding the behaviour of NT-proBNP across the spectrum of HF in SSA is crucial for validating current international guidelines in this context and for informing local diagnostic pathways.

Therefore, this study aimed to evaluate the utility of NT-proBNP in the diagnosis of HF phenotypes in a prospective cohort of Nigerian patients, specifically assessing its sensitivity in confirming HF diagnosis according to ESC criteria and its ability to discriminate between HFrEF, HFmrEF, and HFpEF.

Methods

Study Design and Population

This was a prospective observational study conducted at the Aminu Kano Teaching Hospital (AKTH), Kano, Nigeria, between October 2019 and May 2020. The study enrolled 130 consecutive adult patients (age ≥18 years) with a clinical diagnosis of HF. Patients were recruited from the emergency department, cardiology clinic, and general medical wards. Exclusion criteria included inability to provide informed consent, age <18 years, and poor echocardiographic windows.

The study was approved by the AKTH Health Research Ethics Committee, and all participants provided written informed consent.

Diagnostic Criteria and Patient Classification

The diagnosis and classification of HF were strictly based on the 2016 ESC guidelines.[6]

- **HF Diagnosis:** Required the presence of typical symptoms (e.g., breathlessness, orthopnoea, fatigue) and/or signs (e.g., elevated jugular venous pressure, pulmonary crackles, peripheral oedema) caused by a structural or functional cardiac abnormality.
- **Phenotype Classification:** Patients were classified into three groups based on left ventricular ejection fraction (LVEF) measured by echocardiography:
- HFrEF: LVEF <40%
- HFmrEF: LVEF 40-49%
- HFpEF: LVEF ≥50%
- **Additional Criteria for HFpEF and HFmrEF:** For a diagnosis of HFpEF or HFmrEF, in addition to symptoms/signs and the respective LVEF, an elevated NT-proBNP level was required alongside objective evidence of other cardiac structural or functional alterations. This evidence included at least one of the following:
- Left atrial volume index (LAVI) >34 mL/m²
- Left ventricular mass index (LVMI) ≥115 g/m² for males or ≥95 g/m² for females
- E/e' ratio ≥13
- Average e' (septal and lateral) <9 cm/s

Clinical and Echocardiographic Assessment

All patients underwent a detailed clinical evaluation, including history, physical examination, and assessment of NYHA functional class. Echocardiography was performed by a trained investigator under the supervision of a consultant cardiologist using a Sonoscape SSI 8000 ultrasound system, according to contemporary guidelines.[14] Standard 2D, M-mode, and Doppler measurements were obtained. LVEF was calculated using the Teichholz method. Diastolic function was assessed using pulsed-wave Doppler of the mitral inflow and tissue Doppler imaging of the mitral annulus.

Blood Sample Collection and NT-proBNP Measurement

Venous blood samples were collected from each participant after an 8-12 hour overnight fast at the time of recruitment. Samples were drawn into gel separator tubes under aseptic conditions. The samples were allowed to clot and then centrifuged at 2000-3000 rpm for 20 minutes to separate the serum. The serum was aliquoted and stored at -20°C until analysis.

Serum NT-proBNP levels were quantitatively measured using a high-sensitivity competitive Enzyme-Linked Immunosorbent Assay (ELISA) kit (E1239Hu, Bioassay Technology Laboratory). The assay procedure followed the manufacturer's instructions. In brief, the assay uses a monoclonal antibody coated onto a microtitre plate. NT-proBNP in the sample competes with an NT-proBNP-horseradish peroxidase (HRP) conjugate for binding sites. After incubation and washing, a substrate solution is added, and the colour developed is inversely proportional to the concentration of NT-proBNP in the sample. The reaction was stopped, and the absorbance was read at 450 nm using a BIORAD microplate reader (model PR 3100 TSC). A calibration curve was constructed from calibrators of known concentration, and the NT-proBNP concentration of the samples was deduced from this curve. All samples were run in duplicate.

The intra-assay coefficient of variation (CV) was <8%, and the inter-assay CV was <10%. The assay has a detection limit of 4.12 ng/L and is linear up to 1900 ng/L.

Statistical Analysis

Data were analysed using SPSS version 21 (IBM Corp., Armonk, NY). Continuous variables were tested for normality using the Shapiro-Wilk test. Normally distributed data were presented as mean ± standard deviation (SD), and non-normally distributed data (including NT-proBNP) were presented as median with interquartile range (IQR). Categorical variables were expressed as frequencies and percentages. The Kruskal-Wallis test was used to compare NT-proBNP levels across the three HF phenotypes (HFrEF, HFmrEF, HFpEF). The Mann-Whitney U test was used to compare NT-proBNP

levels between inpatients and outpatients. A two-sided p-value of <0.05 was considered statistically significant. The diagnostic sensitivity of NT-proBNP was calculated as the proportion of patients with NT-proBNP levels above the ESC-recommended cut-offs.

Results

Baseline Characteristics of the Study Cohort

A total of 130 patients was included in the final analysis. The baseline demographic, clinical, and echocardiographic characteristics of the cohort, stratified by HF phenotype, are presented in Table 1. The mean age of the participants was 49.8 ± 18.1 years, with 76 (58.5%) being female. Patients with HFpEF were older than those with HFrEF (mean age 54.5 vs. 46.8 years, p=0.070 for trend, with 41.2% of HFpEF patients ≥65 years vs. 19.2% of HFrEF, p=0.032).

The most common aetiology of HF in the entire cohort was hypertensive heart disease (50.0%), followed by peripartum cardiomyopathy (19.2%) and ischaemic heart disease (11.5%). As expected, echocardiographic parameters differed significantly between groups. LV end-diastolic dimension was largest in HFrEF and smallest in HFpEF (67.4 mm vs. 51.3 mm, p<0.001). Conversely, parameters indicative of diastolic dysfunction, such as E/A ratio and deceleration time, showed a pattern consistent with more advanced diastolic impairment in HFrEF (higher E/A, shorter DT) but confirmed the presence of diastolic dysfunction in all groups.

Table 1: Baseline Characteristics of the Study Population Stratified by Heart Failure Phenotype

Characteristic	All Patients (N=130)	HFrEF (n=78)	HFmrEF (n=18)	HFpEF (n=34)	P-value
Demographics					
Age (years), mean ± SD	49.8 ± 18.1	46.8 ± 17.0	53.7 ± 16.2	54.5 ± 19.5	0.070
Female, n (%)	76 (58.5)	48 (61.5)	9 (50.0)	19 (55.9)	0.629
Clinical					
NYHA Class III/IV, n (%)	117 (90.0)	75 (96.2)	17 (94.4)	25 (73.5)	0.001
Systolic BP (mmHg), mean ± SD	117 ± 27	113 ± 25	129 ± 27	120 ± 29	0.070
Body Mass Index (kg/m²), mean ± SD	25.1 ± 7.2	24.8 ± 7.4	24.1 ± 4.8	26.2 ± 7.9	0.535
Key Aetiologies, n (%)					
Hypertensive Heart Disease	65 (50.0)	32 (41.0)	13 (72.2)	20 (58.8)	-
Peripartum Cardiomyopathy	25 (19.2)	24 (30.8)	1 (5.6)	0 (0.0)	-
Ischaemic Heart Disease	15 (11.5)	10 (12.8)	3 (16.7)	2 (5.9)	-
Rheumatic Heart Disease	10(7.7)	2(2.6)	1(5.6)	7(20.6)	
DCM	10(7.7)	10(12.8)	0	0	
Cor Pulmonale	2(1.5)	0	0	2(5.9)	
Pericardial disease	2(1.5)	0	0	2(5.9)	
RCM	1(0.8)	0	0	1(2.9)	
Echocardiography					
LVEF (%), mean ± SD	38.3 ± 16.9	26.5 ± 6.4	44.2 ± 2.9	62.2 ± 8.6	<0.001
LVEDD (mm), mean ± SD	62.2 ± 12.2	67.4 ± 10.4	60.0 ± 5.6	51.3 ± 11.2	<0.001
E/A ratio, median (IQR)	2.1 (0.63-2.60)	2.4 (1.7-3.0)	1.28 (0.60-2.14)	0.74 (0.47-2.20)	<0.001
E/e' ratio, median (IQR)	11 (8-15)	11 (9-15)	13 (8.8-17.3)	10.5 (7.8-17.8)	0.694
Deceleration Time (ms), mean ± SD	141.1 ± 52.2	117.2 ± 41.0	167.7 ± 46.6	181.7 ± 46.6	<0.001
Comorbidities, n (%)					
Hypertension	77 (59.2)	41 (52.6)	14 (77.8)	22 (64.7)	0.110
Diabetes Mellitus	20 (15.4)	6 (7.7)	6 (33.3)	8 (23.5)	0.008
Atrial Fibrillation/Flutter	17 (13.1)	7 (9.0)	2 (11.1)	8 (23.5)	0.120
Chronic Kidney Disease*	35 (26.9)	13 (16.7)	10 (55.6)	12 (35.3)	0.002
Anaemia†	65 (50.0)	43 (55.1)	6 (33.3)	16 (47.1)	0.230

* Defined as eGFR <60 ml/min/1.73m²; †Defined as PCV <39% (men) and <36% (women). NYHA: New York Heart Association; BP: Blood Pressure; LVEF: Left Ventricular Ejection Fraction; LVEDD: Left Ventricular End-Diastolic Dimension; IQR: Interquartile Range.

NT-proBNP Levels and Diagnostic Sensitivity

The median NT-proBNP level for the entire cohort was 3800.0 pg/mL (IQR: 2900.0 - 4500.0 pg/mL). A critical finding was that all 130 patients (100%) had NT-proBNP levels that exceeded the 2016 ESC diagnostic thresholds for HF (i.e., >125 pg/mL for ambulatory and >300 pg/mL for hospitalized patients). This 100% sensitivity held true for both hospitalized and ambulatory patients. The median NT-proBNP among the 64 inpatients was 3800.0 pg/mL (IQR: 2800-4600) and among the 66 outpatients was 3950.0 pg/mL (IQR: 2900-4475), with no significant difference between these groups (p=0.822).

NT-proBNP Levels Across Heart Failure Phenotypes

The distribution of NT-proBNP levels across the three HF phenotypes is shown in table 2. There was no statistically significant difference in median NT-proBNP levels between patients with HFrEF, HFmrEF, and HFpEF (p=0.461).

- HFrEF: 3900.0 pg/mL (IQR: 3075-4600)
- HFmrEF: 3700.0 pg/mL (IQR: 2275-3700)
- HFpEF: 3850.0 pg/mL (IQR: 2700-4325)

The overlap in NT-proBNP values between the groups was substantial, as illustrated by the wide and overlapping IQRs.

Table 2: NT-proBNP Levels by Heart Failure Phenotype and Patient Status

Group	n	Median NT-proBNP (pg/mL)	IQR	P-value
Overall	130	3800.0	2900.0 - 4500.0	-
By Phenotype				0.461
HFrEF	78	3900.0	3075 - 4600	
HFmrEF	18	3700.0	2275 - 3700	
HFpEF	34	3850.0	2700 - 4325	
By Patient Status				0.822
Inpatient	64	3800.0	2800 - 4600	
Outpatient	66	3950.0	2900 - 4475	

IQR: Interquartile Range.

Discussion

This study provides an analysis of NT-proBNP utility in a prospective, well-characterized SSA HF cohort diagnosed using contemporary ESC criteria. The two principal findings are: first, NT-proBNP demonstrated 100% sensitivity for confirming the diagnosis of HF, with every single patient exceeding the recommended ESC cut-off values. Second, and more notably, there was no significant gradient in NT-proBNP levels across the HF phenotypic spectrum, with HFpEF patients displaying similarly markedly elevated levels as those with HFrEF. These findings partially aligns with and extends observations from a previous Nigerian study by Karaye et al., which reported elevated BNP in HF patients but did not perform a formal statistical comparison across EF-based phenotypes.[13]

The exceptional sensitivity of NT-proBNP in this cohort is striking. International guidelines, while affirming the high diagnostic accuracy of NPs, do not claim 100% sensitivity.[6,15] Studies like the PRIDE study found that using an NT-proBNP cut-off of 450 pg/mL for patients aged <50 years and 900 pg/mL for those ≥50 years had a sensitivity of 90% for diagnosing acute HF.[16] However, the cut off used in our patients is lower following the recommended ESC guidelines.[6]The universal elevation in our patients suggests a population presenting with a high degree of myocardial stress and clinical severity. This is corroborated by the fact that 90% of our patients were in NYHA functional class III or IV, indicating advanced symptomatic status. The median NT-proBNP level of 3800 pg/mL in our study is substantially higher than levels reported in Western HF registries and clinical trials for both HFrEF and HFpEF.[17,18]It also appears higher than median levels described in some broader African studies like the INTER-CHF study.[9] This likely reflects late presentation and diagnosis in our setting, where patients often endure symptoms for prolonged periods before accessing tertiary care. The lack of a significant difference between inpatients and outpatients further suggests that even those managed ambulatorily in this context have severe, decompensated disease.

The absence of a significant difference in NT-proBNP levels between HFrEF, HFmrEF, and HFpEF is a key finding that contrasts with the established literature. Numerous studies have consistently shown that NP levels are higher in HFrEF than in HFpEF.[10,11,19] The proposed mechanism is that the dilated, thin-walled ventricle in HFrEF experiences greater wall stress according to the Law of Laplace, providing a stronger stimulus for NP secretion than the thick-walled, small-cavity ventricle typical of HFpEF.[12] Our results challenge the universal applicability of this pathophysiological model in this SSA population.

Several factors may explain this discrepancy. First, the overwhelming burden of comorbidities in our cohort, particularly hypertension, may be a major driver of NP release irrespective of LVEF. Chronic pressure overload leads to left ventricular hypertrophy and fibrosis, which are potent stimuli for NP synthesis.[20] In our study, hypertension was highly prevalent across all phenotypes (52.6% in HFrEF to 77.8% in HFmrEF). The sustained high-pressure load in a hypertrophied heart, even with preserved EF, could generate sufficient wall stress to produce NT-proBNP levels comparable to those seen in systolic failure.

Second, the aetiological profile is different. The high prevalence of peripartum cardiomyopathy (PPCM) in our HFrEF group (30.8%) is distinctive. PPCM is an acute or subacute form of HF, and the intense inflammatory and cytotoxic myocardial injury involved may lead to a different pattern of biomarker release compared to chronic ischaemic or idiopathic dilated cardiomyopathy [21]. Furthermore, the significant burden of rheumatic heart disease (RHD) in the HFpEF group (20.6%), where pressure or volume overload from regurgitant or stenotic lesions directly increases atrial and ventricular strain, potently stimulating NP secretion. [22]

Third, the role of renal function must be considered. Renal impairment reduces the clearance of NT-proBNP, leading to its accumulation [7]. In our cohort, chronic kidney disease (eGFR <60 ml/min/1.73m²) was present in 26.9% of patients and was most prevalent in the HFmrEF group (55.6%). This could have contributed to the elevated levels across the board and potentially minimized inter-phenotype differences.

Fourth, the relative lack of obesity in our cohort may be a factor. Obesity is known to suppress NP levels, a phenomenon often attributed to increased NP clearance receptors in adipose tissue or reduced synthesis.[8,23] The mean BMI in our cohort was 25.1 kg/m², with only 18.5% being obese (BMI ≥30 kg/m²). This is lower than the prevalence of obesity in many Western HFpEF cohorts, where it often exceeds 50%.[24] The absence of this suppressing effect in our population may allow the "true" myocardial wall stress to be more fully reflected in the circulating NT-proBNP levels, unmasking the severe pathophysiological burden even in HFpEF.

Implications for Clinical Practice and Laboratory Medicine

From a diagnostic perspective, our findings robustly validate the use of NT-proBNP as a rule-*out* test in this population. A normal NT-proBNP level in a patient with suspected HF would make the diagnosis highly unlikely. However, the lack of discriminatory power between phenotypes means that clinicians cannot use the absolute NT-proBNP value to infer whether a patient has reduced or preserved EF. This places greater emphasis on the necessity of echocardiography for phenotyping all HF patients in SSA, as treatment strategies differ significantly. For the clinical laboratory, this study confirms the reliability and high clinical sensitivity of the NT-proBNP ELISA assay in a real-world SSA setting.

Study Limitations

This study has limitations. It is a single-centre study, which may affect generalizability, though it provides a detailed view from a major referral centre. We did not have a control group of asymptomatic individuals to define a true reference range or calculate specificity. The study was not powered to examine the complex multivariate relationships between NT-proBNP, phenotypes, and comorbidities like renal function in depth

Conclusion

In this SSA HF cohort, NT-proBNP proved to be an exceptionally sensitive biomarker for confirming the diagnosis of heart failure, with 100% of patients exceeding the ESC-recommended diagnostic thresholds. This underscores its critical value in the diagnostic algorithm. However, unlike in many other populations, NT-proBNP levels were uniformly and markedly elevated across all HF phenotypes, showing no significant gradient from HFrEF to HFpEF. This suggests that the pathophysiology driving NP release in this population may be different, potentially related to a high burden of hypertensive heart disease, unique aetiologies, and late presentation. Consequently, while NT-proBNP is indispensable for diagnosing HF, it cannot be used to distinguish between phenotypic subtypes in this context, mandating universal access to echocardiography for appropriate management.

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HOW TO CITE

Haruna Abubakar Alhaji, Ayoola Yekeen Ayodele, Hussain Muhammad Sulaiman, Babalawan Muhammad, Kabir Abdussalam, Mohammed Fatima Isa. Utility of NT-proBNP in the Diagnosis of Heart Failure Phenotypes in a Sub-Saharan African Population. *Delta Oghara Medical Journal*. 2026;2(1).

https://domj.org/index.php/start/article/view/utility-nt-probnp-diagnosis-heart-failure-phenotypes-sub-saharan-african-population

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