Original Article

DOI: 10.7860/JCDR/2026/84219.23662

Prognostic Value of Admission NT-proBNP for Stroke Severity and 90-Day Functional Outcome in Acute Ischaemic Stroke: A Hospital-based Cohort Study

Internal Medicine Section

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ABSTRACT

Introduction: Acute Ischaemic Stroke (AIS) is a leading cause of morbidity and mortality worldwide. Accurate prognostic markers are essential for early risk stratification. N-Terminal Pro-Brain Natriuretic Peptide (NT-proBNP), a cardiac biomarker, has been proposed as a predictor of stroke severity and outcome, but its clinical utility remains incompletely defined.

Aim: To assess whether admission NT-proBNP is associated with stroke severity and 90-day functional outcome in AIS and to explore its relationship with Trial of Org 10172 in Acute Stroke Treatment (TOAST) subtypes.

Materials and Methods: This hospital-based prospective cohort study conducted in the Department of General Medicine, Mahatma Gandhi Medical College and Research Institute (MGMCRI), Puducherry, India, over a two-year period (June 2023 - July 2025) and included 90 consecutive patients with AIS. Baseline demographics, risk factors, National Institutes of Health Stroke Scale (NIHSS) scores and TOAST classifications were recorded. Serum NT-proBNP levels were measured at admission. Functional outcome was assessed using modified Rankin Scale (mRS). Statistical analysis done using Statistical package for Social Sciences (SPSS) (v_24.0), where inferential statistics were applied based on the type of the variable and their normality. Diagnostic utility assessed by Receiver Operating Characteristic (ROC) analysis.

Results: The mean age of the patients was 61.3±12.5 years with 59/90 (65.6%) males. The mean NT-proBNP was 173.6±220.1pg/mL. Elevated NT-proBNP (>125 pg/mL) was observed in 32/90 (35.6%) of patients. NT-proBNP levels correlated significantly with NIHSS (ρ=0.43, p<0.001) and mRS (ρ=0.21, p=0.048), but not with TOAST subtypes. Logistic regression identified NT-proBNP as an independent predictor of unfavourable outcome {Odds Ratio (OR)=1.00, 95% Confidence Interval (CI) 1.00–1.01, p=0.049}. ROC analysis showed NIHSS had superior predictive accuracy (AUC-0.78, cut-off-6, sensitivity-0.73, specificity=0.71) compared with NT-proBNP {Area Under Curve (AUC-0.69, cut-off=113 pg/mL, sensitivity-0.68, specificity=0.71}.

Conclusion: NT-proBNP is significantly associated with stroke severity and functional outcomes in AIS, though NIHSS remains the stronger predictor. NT-proBNP, with its high NPV, serves as a valuable adjunct biomarker to enhance prognostication. Further large-scale studies are warranted to validate its integration into routine stroke assessment.

Keywords: Biomarker, Modified Rankin scale, N-Terminal pro-brain natriuretic peptide

INTRODUCTION

The AIS remains a major global health burden ranking as the second leading cause of death and disability worldwide [1]. Despite progress in therapeutic interventions, it continues to be associated with substantial morbidity and mortality, particularly in low- and middle-income countries [1,2]. Stroke risk increases exponentially from the age of 30 years where 95% of strokes occur at age 45 years or older, while they can happen at any age, even during childhood [1,3]. The Trial of Org 10172 in Acute Stroke Treatment (TOAST) classification, which divides ischaemic strokes into five classes according to their aetiology was published in 1993 by Adams HP et al., [4].

The AIS occurs due to sudden interruption of cerebral blood flow, resulting in neuronal death, neurological deficits and long-term disability [5,6]. World Health Organisation (WHO) criteria as an acute focal neurological deficit lasting >24 hours [5]. The severity of AIS is influenced by multiple mechanisms including thromboembolic, haemodynamic, atherothrombotic and microangiopathic processes as it causes damage to the Blood-Brain-Barrier (BBB). Neuroinflammation following AIS further exacerbates neuronal injury and worsens clinical outcomes [6,7].

The potential significance of biomarkers in stroke causation has piqued researchers' curiosity. The plasma NT-proBNP is a neurohormone primarily synthesised and secreted by ventricular cardiomyocytes in response to myocardial wall stress caused by pressure or volume overload and routinely utilised measure in the evaluation of Heart Failure (HF) [8,9]. It regulates cardiovascular and renal homeostasis through natriuresis, vasodilation and modulation of the renin-angiotensin-aldosterone system. NT-proBNP also exerts anti-fibrotic and lipolytic effects, thereby playing a protective role against cardiovascular stress [8,10,11]. Notably, NT-proBNP is not limited to cardiac origin, it is also released by the hypothalamus and astrocytes, particularly under conditions of cerebral ischemia [9,11,12].

It is believed that NT-proBNP plays a significant role in stroke, particularly in haemodynamic control during the acute phase of ischaemic stroke [13]. Emerging evidence suggests that NT-proBNP levels are significantly elevated in patients with AIS, independent of underlying cardiac dysfunction. Several studies have demonstrated that high NT-proBNP concentrations correlate with stroke severity, larger infarct volumes, cardioembolic aetiology and worse functional outcomes. Shibazaki K et al., reported that plasma NT-proBNP levels were significantly higher in cardiometabolic stroke than in other AIS subtypes and were strongly associated with baseline National Institutes of Health Stroke Scale (NIHSS) scores [14]. Similarly, Chen X et al., demonstrated that NT-proBNP levels, when

combined with NIHSS, provided a powerful prognostic indicator for clinical outcomes [15]. These findings highlight the potential of NT-proBNP as a biomarker in risk stratification, prognostication and possible in guiding therapeutic decision making in AIS.

The novelty of the present study lies in evaluating NT-proBNP as an early biomarker to predict both the severity and clinical outcome of AIS, especially in resource-limited settings here rapid and reliable prognostic indicators are essential. While existing tools such as the NIHSS and TOAST classification aid in clinical assessment, they are time-consuming and subject to interobserver variability [16,17]. Identifying a biomarker that is objective, reproducible and widely accessible can complement clinical scoring systems. Hence, the present study was designed with the aim of assessing the role of NT-proBNP in AIS and its clinical outcomes, with specific objectives to determine its correlation with stroke severity (NIHSS), classify AIS subtypes as per TOAST and evaluate neurological outcomes using the mRS.

MATERIALS AND METHODS

A hospital-based prospective cohort study was conducted in the Department of General Medicine, Mahatma Gandhi Medical College and Research Institute (MGMCRI), Puducherry, India, over a two-year period (June 2023-July 2025). Patients admitted through the Outpatient Department of Medicine (OPD), neurology Outpatient Clinic (OPC) casualty and the medical Intensive Care Unit (ICU) were recruited after confirmation of AIS by neuroimaging {Computed Tomography (CT) or Magnetic Resonance Imaging (MRI) of brain}. Ethical clearance was obtained from the Institutional Human Ethics Committee, MGMCRI (MGMCRI/Res/01/2023/126/IHEC/110). Written informed consent was obtained from all participants or their legal guardians prior to inclusion. Data confidentiality was ensured, and access to study records was restricted to the investigators.

Sample size calculation: The required sample size was calculated as 90. It was calculated based on the mean level of NT-proBNP among AIS patient at 72 hours was 273.78±451.91 by Sayan S and Kotan D, with α at 0.05 and an estimate error of 10% yielded the sample size of 79 [18]. The formula used for calculation:

$$n \geq \left(\frac{Z1\text{-}\alpha}{\frac{2}{d}} \times \sigma\right)^2 = \frac{1.96^2 \times 451.91^2}{100^2} = \frac{784,541.72}{10000} = 78.45$$

(where Z1-α/2 – 1.96 at 95% Confidence Interval (CI); σ (estimated standard deviation) - 451.91; d (estimated error) - 10%). Adjusting for 10% non response rate final N=78.45/0.90=87.17 rounded to highest figure of 90. A convenience sampling method was used to recruit participants until the sample size was achieved.

Inclusion and Exclusion criteria: All adult patients (>18 years) with radiologically proven acute infarct consistent with a vascular distribution, who presented within 24 hours of symptom onset [5], were eligible for inclusion. Exclusion criteria included acute HF, Transient Ischaemic Attack (TIA), cardioembolic stroke, atrial fibrillation, recurrent stoke, haemorrhagic stroke, Atrial Fibrillation (AF), Chronic Kidney Failure (CKD), seizures and pregnant women.

Study Procedure

Study procedure is presented in [Table/Fig-1]. A detailed clinical history and physical examination were performed, including vital signs, anthropometric measurements and neurological assessment. Routine blood investigation including Complete Blood Count (CBC), Renal Function Test (RFT) and Liver Function Tests (LFT), electrolytes, Fasting Lipid Profile (FLP), Electrocardiogram (ECG) were carried out. Plasma NT-proBNP levels were measured within 24 hours of admission and for analysis it is classified as elevated (>125 pg/mL) and normal (<125 pg/mL), a commonly used clinical reference thresholds for NT-proBNP [19]. Blood samples were collected in Ethylenediaminetetraacetic Acid (EDTA) tubes, centrifuges and plasma stored at -80°C. NT-proBNP concentrations were determined using a chemiluminescent enzyme immunoassay employing a two-step sandwich method with monoclonal antibodies specific to human NT-proBNP.

Patient diagnosed or having clinical signs of AIS presenting to the Department of General Medicine and Neurology
↓
Patients underwent a detailed history, confirmed ischaemic stroke with neuroimaging, a thorough clinical examination was done
↓
After the informed consent of the patient for participation in the study
↓
Patients undergone the following lab investigations - routines (CBC, RFT, electrolytes, LFT, ECG), FLP, BNP, after obtaining consent
↓
NIHSS score was calculated NIHSS score grading: 1 to 8 is mild, 9 to 15 is moderate and ≥ 16 is severe. An increase of >2% points in the NIHSS score was defined as progression.
↓
TOAST criteria assessed to know etiological subtype of the stroke TOAST criteria large artery atherosclerosis Small vessel occlusion Cardio embolism Stroke of determined ethology
↓
Compared the outcome of NT-proBNP values with NIHSS score
↓
Calculated modified Rankin Scale (mRS) and assessed neurological outcome

[Table/Fig-1]: Study procedure.

Neurological severity was assessed using the NIHSS at admission [20,21] and classified as per the scale. Stroke aetiology was classified using TOAST criteria [4] as large-artery atherosclerosis, small-vessel occlusion, cardioembolism, other determined, or undetermined cause. Functional outcome was evaluated at 90-days using the mRS [22,23].

STATISTICAL ANALYSIS

Statistical analyses as conducted using SPSS (ver_24.0). Continuous variables were expressed as mean and SD or median (interquartile range) based on the distribution of the data, while categorical variables were presented as frequencies and percentages. Chi-square test applied for categorical variables and independent t-test or Mann-Whitney U-test was used for the continuous variables based on the normality. A two-tailed p<0.05 was considered statistically significant.

RESULTS

A total of 90 patients with AIS were enrolled and their demographic and co-morbid status is presented in [Table/Fig-2]. The mean age of the study participants was 61.3±12.5 years. The distribution of the study participants based on NIHSS score, TOAST category, mRS scoring and NT-proBNP is presented in [Table/Fig-3]. The mean NT-proBNP among the study participants was 173.6±220.1 pg/mL with

Variables	Results n (%)
Age (in years)	
<40	4 (4.4)
41-60	44 (48.9)
61-80	34 (37.8)
>81	8 (8.9)
Gender	
Female	31 (34.4)
Male	59 (65.6)
Systemic hypertension (yes)	67 (74.4)
Diabetes (yes)	53 (58.9)

[Table/Fig-2]: Distribution of demographic and clinical variables of the study participants (N=90).

Variables	Results n (%)
NIHSS score category	
Minor stroke (0-4)	45 (50.0)
Moderate stroke (5-15)	41 (45.6)
Moderate-to-severe stroke (16-20)	3 (3.3)
Severe stroke (21-42)	1 (1.1)
TOAST category	
Large artery atherosclerosis	47 (52.2)
Small vessel occlusion	43 (47.8)
mRS functional outcome	
Favourable (0-2)	68 (75.6)
Unfavourable (>2)	22 (24.4)
NT-proBNP (pg/mL)	
Elevated (>125 pg/mL)	32 (35.6)
Normal (<125 pg/mL)	58 (64.4)

[Table/Fig-3]: Distribution of study participants based on the outcomes.
NIHSS: National institute of health stroke severity; TOAST: Trial of Org 10172 in acute stroke treatment; mRS: modified Rankin Scale; NT-proBNP: brain natriuretic peptide

a median of 83 pg/mL {Interquartile Range (IQR): 43.5 - 217.8 pg/mL} and its association with stroke severity and subtypes showed that a significant association between elevated NT-proBNP with moderate-to-severe and severe stroke were statistically significant (p=0.001) and with mRS (p=0.016) [Table/Fig-4].

Variables	NT-proBNP (pg/mL)		p-value*
	Normal (n=58) (<125 pg/mL) n (%)	Elevated (n=32) (>125 pg/mL) n (%)	
NIHSS score category			
Minor stroke (0-4)	35 (77.8)	10 (22.2)	0.001
Moderate stroke (5-15)	22 (53.7)	19 (46.3)	
Moderate-to-severe stroke (16-20)	1 (33.3)	2 (66.7)	
Severe stroke (21-42)	0 (0.0)	1 (100.0)	
TOAST category			
Large artery atherosclerosis	28 (59.4)	19 (48.3)	0.430
Small vessel occlusion	30 (40.6)	13 (51.7)	
mRS functional outcome			
Favourable	49 (59.4)	19 (84.5)	0.016
Unfavourable	9 (40.6)	13 (15.5)	

[Table/Fig-4]: Association of NT-proBNP based on NIHSS score and TOAST category among the study participants.
*Pearson's Chi-square test; p-value <0.05 statistically significant and indicated in boldface. NIHSS: National institute of health stroke severity; TOAST: Trial of Org 10172 in acute stroke treatment; mRS: modified Rankin Scale; NT-proBNP: brain natriuretic peptide

Outcome analysis revealed that demographic and other factors were not significantly associated with functional outcome (mRS) except, stroke severity by NIHSS (p=0.013) and elevated NT-proBNP (p=0.017) were significantly associated with unfavourable outcomes [Table/Fig-5]. The TOAST subtypes did not significantly influence the outcomes shown in [Table/Fig-6].

Variables	mRS functional outcome		p-value*
	Favourable (0-2) (n=68) n (%)	Unfavourable (>2) (n=22) n (%)	
Age (in years)			
<40	2 (50.0)	2 (50.0)	0.18
41–60	35 (79.5)	9 (20.5)	
61–80	27 (79.4)	7 (20.6)	
>81	4 (50.0)	4 (50.0)	
Gender			
Female	24 (77.4)	7 (22.6)	0.97
Male	44 (74.6)	15 (25.4)	
Systemic hypertension	52 (77.6)	15 (22.4)	0.57
Diabetes	41 (77.4)	12 (22.6)	0.32
NIHSS score			
Minor stroke (0-4)	40 (88.9)	5 (11.1)	0.013
Moderate stroke (5-15)	26 (63.4)	15 (36.6)	
Moderate-to-severe stroke (16-20)	1 (33.3)	2 (66.7)	
Severe stroke (21-42)	1 (100.0)	0 (0)	
TOAST category			
Large artery atherosclerosis	35 (74.5)	12 (25.5)	0.80
Small vessel occlusion	33 (76.7)	10 (23.3)	
NT-proBNP (pg/mL)			
Elevated (>125pg/mL)	19 (27.9)	13 (59.1)	0.017
Normal (<125pg/mL)	49 (72.1)	9 (40.9)	

[Table/Fig-5]: Association of demographic and other factors based on the mRS scale.
*Chi-square test. p-value <0.05 was statistically significant and indicated in boldface. NIHSS: National institute of health stroke severity; TOAST: Trial of Org 10172 in acute stroke treatment; mRS: modified Rankin Scale; NT-proBNP: brain natriuretic peptide.

Variables	TOAST category		p-value*
	Large vessel Mean±SD	Small vessel Mean±SD	
NIHSS	5.67±4.39	5.12±3.07	0.54
NT-proBNP (pg/mL)	185.22±221.72	160.88±220.37	0.63
mRS	1.74±1.59	1.81±1.43	0.83
Hospital stays (in days)	4.85±2.97	4.37±2.31	0.41

[Table/Fig-6]: Association of TOAST category with the outcomes among the study participants.
*Mann-Whitney U-testt. p-value <0.05 was statistically significant and indicated in boldface. NIHSS: National institute of health stroke severity; TOAST: Trial of Org 10172 in acute stroke treatment; mRS: modified Rankin Scale; NT-proBNP: brain natriuretic peptide.

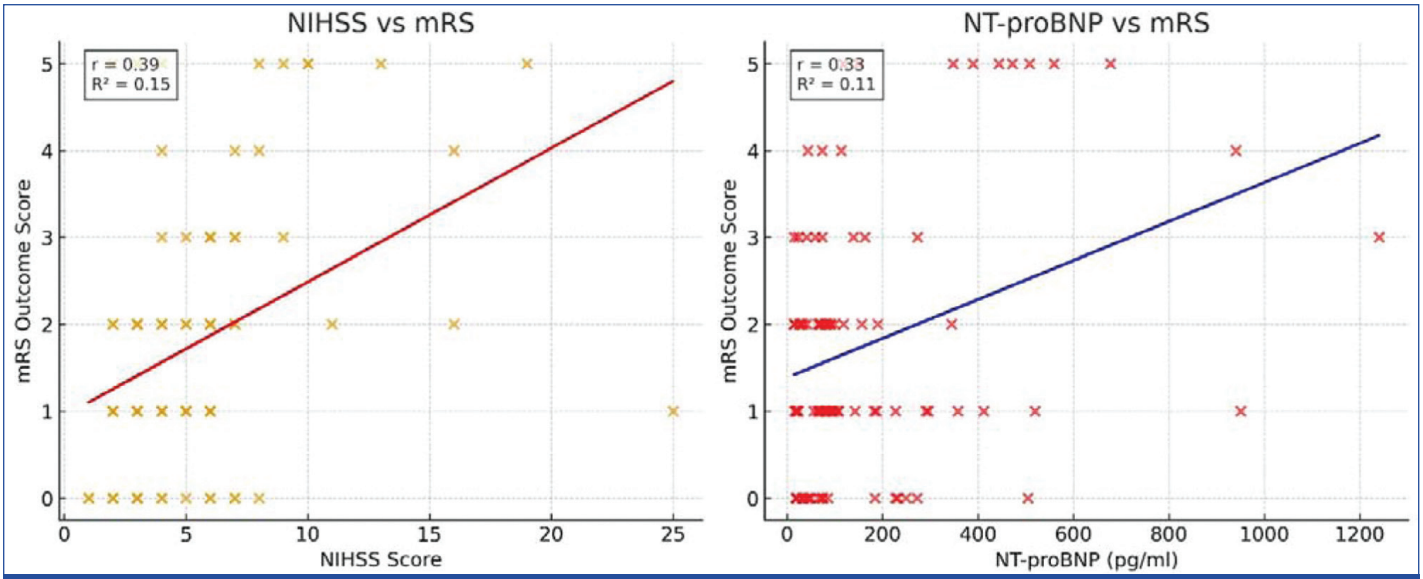
Spearman's correlation demonstrated a moderate positive correlation between mRS and NIHSS (ρ=0.43; p<0.001) and a weak positive correlation with NT-proBNP (ρ=0.21; p=0.048), while NIHSS and NT-proBNP were moderately correlated (ρ=0.41; p<0.001) indicating that increase in NIHSS score severity will ultimately increases NT-proBNP [Table/Fig-7,8]. Univariate analysis revealed that NIHSS, NT-proBNP and hospital stay period were significant predictors of unfavourable outcome, but after adjustment, NT-proBNP remained an independent predictor (OR=1.00; 95% CI: 1.00 - 1.01; p=0.049) for mRS scoring [Table/Fig-9]. Diagnostic ability of NIHSS and NT-proBNP for mRS outcome were presented in [Table/Fig-10,11], indicating their utility in excluding poor outcomes due to high negative predictive value (0.89 vs 0.87, respectively).

Variables	Spearman ρ	p-value*
mRS vs Age	-0.13	0.21
mRS vs NIHSS	0.43	<0.001
mRS vs NT-proBNP	0.21	0.048
NIHSS vs NT-proBNP	0.41	<0.001

[Table/Fig-7]: Correlation of mRS with other variables.
*Spearman's correlation; p-value (two-tailed) <0.05 statistically significance and indicated in boldface. NIHSS: National institute of health stroke severity; NT-proBNP: brain natriuretic peptide

DISCUSSION

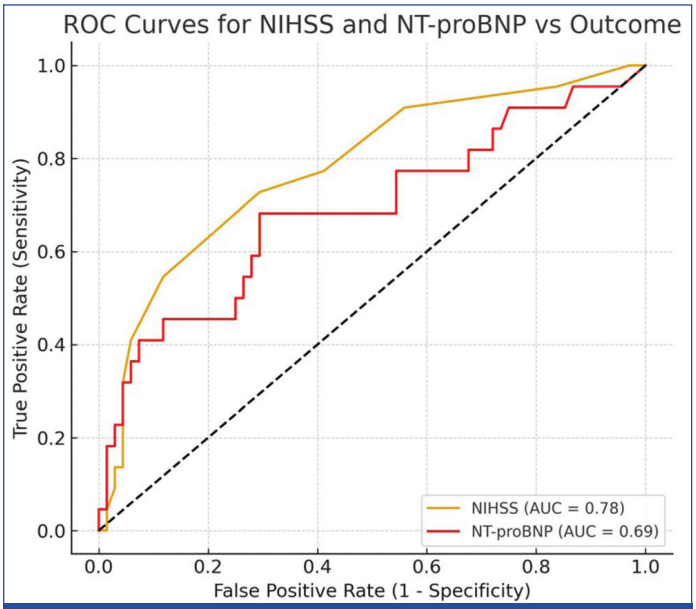
The present study investigated the role of NT-proBNP in AIS and its association with stroke severity, aetiological subtypes and functional outcome. The authors major findings were that elevated NT-proBNP levels were significantly associated with stoke severity (NIHSS) and unfavourable outcomes (mRS>2), NT-proBNP demonstrated moderate diagnostic accuracy with high NPV and no significant



[Table/Fig-8]: Scatterplot of mRS with NIHSS and NT-proBNP.

Variables	Unadjusted OR (95% CI)	Unadjusted p-value	Adjusted OR (95% CI)	Adjusted p-value
Age (years)	1.00 (0.96 - 1.04)	0.865	0.98 (0.93 - 1.03)	0.371
Gender (Male vs Female)	1.17 (0.42 - 3.26)	0.766	1.99 (0.50 - 7.84)	0.326
Hypertension (Yes vs No)	0.62 (0.21 - 1.79)	0.376	0.33 (0.09 - 1.22)	0.097
Diabetes (Yes vs No)	0.85 (0.31 - 2.29)	0.741	0.55 (0.16 - 1.90)	0.343
NIHSS score	1.23 (1.06 - 1.42)	0.007	0.71 (0.41 - 1.23)	0.221
NT-proBNP (pg/mL)	1.00 (1.00 - 1.01)	0.005	1.00 (1.00 - 1.01)	0.049
Hospital stays (days)	1.24 (1.08 - 1.41)	0.002	1.57 (0.97 - 2.54)	0.065
TOAST subtype: Small Vessel Occlusion	0.88 (0.34 - 2.32)	0.802	0.64 (0.19 - 2.15)	0.473

[Table/Fig-9]: Logistic regression for the mRS with other variables.
OR (Odds Ratio): Represents the odds of unfavourable outcome (mRS >2) compared to favourable outcome (mRS 0–2); CI: Confidence interval; Unadjusted OR: Results from univariate logistic regression models for each variable; Adjusted OR: Results from multivariate logistic regression including all predictors (Age, Gender, Hypertension, Diabetes, NIHSS, NT-proBNP, Hospital stay, TOAST subtypes); p-value <0.05 was considered statistically significant and indicated in boldface; Reference categories: Female gender, absence of hypertension, absence of diabetes, normal BNP (<125 pg/mL) and baseline TOAST subtype are reference groups. NIHSS: National institute of health stroke severity; TOAST: Trial of Org 10172 in acute stroke treatment; mRS: modified Rankin Scale; NT-proBNP: brain natriuretic peptide.



[Table/Fig-10]: ROC curve for predicting NIHSS and NT-proBNP by mRS.

Variables	NIHSS	NT-proBNP
Cut-off	6	113
Sensitivity	0.73	0.68
Specificity	0.71	0.71
Positive predictive value	0.44	0.43
Negative predictive value	0.89	0.87
Accuracy	0.71	0.7
Youden's Index	0.43	0.39
Area under curve	0.78	0.69
p-value	<0.001	<0.001

[Table/Fig-11]: Diagnostic ability of NIHSS and NT-proBNP by mRS.
p-value <0.05 was considered statistically significant and indicated in boldface; NIHSS: National institute of health stroke severity; NT-proBNP: brain natriuretic peptide

association was observed with TOAST subtypes. These results underscore the potential of NT-proBNP as a prognostic biomarker in AIS, complementing clinical severity scales.

NT-proBNP is secreted by ventricular myocytes in response to wall stress and haemodynamic overload [8,9]. Beyond its established role in cardiac dysfunction, elevated NT-proBNP has been reported in patients with AIS, particularly those with cardioembolic stroke mechanism [14,24-26]. In the present cohort, although TOAST subtypes were not associated with NT-proBNP, the significant correlation with stroke severity (p=0.001) is consistent with prior studies. Di Angelantonio E et al., (2007) reported higher BNP levels in patients with more severe neurological deficits, suggesting that BNP reflects systemic stress and neuro-cardiac interaction in AIS [26]. Similarly, Shibazaki K et al., (2009) demonstrated the BNP was independently correlated with baseline NIHSS, supporting its role as a severity biomarker [24]. The correlation between NT-proBNP and NIHSS (ρ=0.41; p<0.001) observed in the present study which was moderate association yet statistically significant, which was in concordance with the study done by Manoj A et al., reported stronger correlations (ρ=0.84; p<0.001) [13]. This suggests that while NT-proBNP is useful, it may be more effective in conjunction with clinical severity scales for better severity stratification, rather than as a standalone predictor.

In the present study patients with elevated NT-proBNP were more likely to have unfavourable outcomes (p=0.016). In logistic regression, NT-proBNP remained as an independent predictor even after adjustment for confounders (OR-1.00; 95% CI 1.00-1.01; p=0.049). This aligns with the Suita study by Arafa A et al., who reported that BNP levels above 100pg/mL predicted poor functional outcome at 90 days [27]. Rodríguez-Yáñez M et al., also showed that BNP was significantly associated with mortality an disability in AIS [28]. The ROC

analysis showed that NT-proBNP (AUC 0.69), had modest predictive power but an excellent NPV (0.87), suggesting that low NT-proBNP reliably excludes poor outcomes. This is consistent with Katan M et al., who demonstrated the clinical utility of NT-proBNP in prognostic stratification, even in non cardioembolic stroke subtypes [29].

Unlike previous studies that demonstrated an association between BNP and cardioembolic strokes [24,27], while in the present study did not find a significant association between NT-proBNP and TOAST classification (p=0.430). This could be explained by the relatively small sample size and near-equal distribution between large artery atherosclerosis (52.2%) and small vessel occlusion (47.8%). It is also possible that NT-proBNP reflects systemic stress rather than aetiology-specific mechanisms, particularly in patients with multiple vascular risk factors, which has been suggested by Rodríguez-Yáñez M et al., [28].

The diagnostic accuracy of NIHSS (AUC 0.78) was superior to NT-proBNP, confirming its role as the strongest clinical predictor of functional outcome [4,15]. However, the addition of NT-proBNP provided incremental prognostic information, especially in excluding poor outcomes. A similar conclusion was reached by Shibazaki K et al., who found that BNP combined with NIHSS improved outcome prediction compared with NIHSS alone [24]. Also, study by Wouters A et al., where the NIHSS alone achieved AUCs ranging between 0.75 and 0.82 for predicting 90-day outcome [30]. Similarly, NT-proBNP in the present study falls withing the modest predictive range described in other cohorts (AUC 0.65-0.75), with cut-offs around 100-150 pg/mL in general AIS populations [28,29]. Notably, although NT-proBNP was less discriminative than NIHSS, its high NPV highlights its clinical usefulness in ruling out poor outcomes. Prior studies have also emphasised that NT-proBNP adds incremental prognostic information when combined with NIHSS, improving discrimination compared with either marker along [15,24]. While in the present study, NIHSS lost significantly due to small size and collinearity, echoing the need for larger cohorts to confirm these additive effects. The present study supports the fact that NIHSS remains the gold standard for clinical severity assessment, NT-proBNP serves as a valuable adjunct biomarker.

From a clinical standpoint, NT-proBNP measurement may aid in risk stratification, particularly in patients with moderate stroke severity where prognosis may be uncertain. Elevated NT-proBNP could identify patients requiring closer monitoring, aggressive secondary prevention and structured rehabilitation strategies. Its high NPV also makes it clinically useful to reassure clinicians regarding favourable prognosis in low-level elevations.

The strengths of the present study include prospective patient inclusion, systematic evaluation of stroke severity and outcome using validated scales and integration of NT-proBNP into multivariate analyses.

Limitation(s)

Limitations include the modest sample size and absence of detailed echocardiographic parameters to confirm underlying cardiac dysfunction. Larger, multicentric studies with longer follow-up are warranted to confirm and generalise the present findings.

CONCLUSION(S)

In the present study, elevated NT-proBNP levels were significantly associated with greater stroke severity and unfavourable functional outcomes, while no relationship was observed with TOAST subtypes. NIHSS remained the stronger predictor of outcome, but NT-proBNP provided additional prognostic value, particularly through its high NPV, making it a useful adjunct in risk stratification. Together, these findings support the integration of NT-proBNP with established clinical severity scales to enhance early prognostication in AIS. Larger multicentric studies with extended follow-up and incorporation of cardiac parameters are warranted to validate these results and to clarify the incremental value of NT-proBNP in routine clinical practice.

Acknowledgement

The authors would like to thank all the medical interns and nurses for their generous assistance and support. They also extend their gratitude to the laboratory technicians who supported the study.

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PLAGIARISM CHECKING METHODS: [Jain H et al.]
- Plagiarism X-checker: Sep 29, 2025
- Manual Googling: Feb 27, 2026
- iThenticate Software: Mar 02, 2026 (5%)

ETYMOLOGY: Author Origin

EMENDATIONS: 6

AUTHOR DECLARATION:
- Financial or Other Competing Interests: None
- Was Ethics Committee Approval obtained for this study? Yes
- Was informed consent obtained from the subjects involved in the study? Yes
- For any images presented appropriate consent has been obtained from the subjects. NA

Date of Submission: **Sep 11, 2025**
Date of Peer Review: **Dec 03, 2025**
Date of Acceptance: **Mar 05, 2026**
Date of Publishing: **Jun 01, 2026**