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Evaluation of the Predictors of Hemorrhagic Transformation After Intravenous Thrombolysis in Hyperacute Ischemic Stroke

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Key words:
Acute ischemic stroke; intravenous thrombolysis; hemorrhagic transformation; NIHSS.

Abstract

Background: *Hemorrhagic transformation (HT) is a major complication after intravenous thrombolysis (IVT) in hyperacute ischemic stroke and may worsen early outcomes. The aim of the study to evaluate baseline clinical, hemodynamic, and laboratory predictors of HT among hyperacute ischemic stroke patients treated with IVT in a stroke unit of a referral neuroscience hospital.* ***Methods:*** *This observational study was conducted in the Stroke Unit of the National Institute of Neurosciences & Hospital (NINS&H), Dhaka, Bangladesh, from March 2024 to March 2025. A total of 100 patients aged 18–80 years who received IV rt-PA within 4.5 hours of symptom onset were included. HT was categorized as hemorrhagic infarction (HI1/HI2) or parenchymal hematoma (PH1/PH2) on follow-up imaging. Data were analyzed with IBM SPSS Statistics version 27. Group comparisons were performed between patients with and without HT, and univariate logistic regression was used to estimate odds ratios (ORs).* ***Results:*** *HT occurred in 12/100 (12.0%) patients (HI1 2.0%, HI2 8.0%, PH1 1.0%, PH2 1.0%). Patients with HT were older, more frequently smokers, had higher baseline and repeat NIHSS, higher admission blood pressures, and higher baseline glucose and HbA1c. In univariate models, significant predictors included age >65 years (OR 10.04), smoking (OR 11.30), lower GCS score (OR 4.72 per 1 unit decrease), higher baseline NIHSS (OR 1.87 per point), higher SBP (OR 2.02 per 10 mmHg), higher baseline glucose (OR 1.40 per mmol/L), higher HbA1c (OR 2.80 per 1%), hypertension (OR 9.17), diabetes (OR 7.26), dyslipidemia (OR 27.72), prior stroke (OR 11.54), and higher ASPECTS score (OR 4.22 per point).* ***Conclusion:*** *HT after IVT was associated with older age, smoking, greater stroke severity, elevated blood pressure, dysglycemia, and vascular comorbidities. Routinely available baseline variables may help stratify HT risk and guide monitoring intensity after thrombolysis.*

Introduction:

Hyperacute ischemic stroke represents a medical emergency characterized by abrupt loss of neurological function due to disruption of cerebral blood flow[1,2]. Intravenous thrombolytic therapy (IVT) with recombinant tissue plasminogen activator (rtPA) is a cornerstone in hyperacute ischemic stroke and improves functional outcomes

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when administered within the recommended therapeutic window[2,3]. However, hemorrhagic transformation (HT) remains a key safety concern and can worsen early clinical course and mortality[4,5]. Identifying patients at higher risk of HT could support individualized monitoring and early correction of modifiable factors such as blood pressure and glucose. Stroke severity at presentation, commonly quantified by the NIH Stroke Scale (NIHSS), is a robust predictor of outcomes after thrombolysis[6,7] and other baseline characteristics including atrial fibrillation and renal dysfunction have been associated with complications and poorer prognosis[8,9]. Because predictors can vary across populations and care settings, locally derived data from high-volume stroke units may refine pragmatic risk stratification. This study evaluates baseline demographic, hemodynamic, and laboratory predictors of HT among hyperacute ischemic stroke patients receiving IVT in a referral neuroscience hospital.

Methods:

A single-center observational study was conducted in the Stroke Unit of the National Institute of Neurosciences & Hospital (NINS&H), Dhaka, Bangladesh, from March 2024 to March 2025. Consecutive patients with hyperacute ischemic stroke who received intravenous thrombolysis (IV rt-PA) within 4.5 hours of symptom onset and were aged 18–80 years were enrolled after written informed consent from the patient or an appropriate surrogate. Hemorrhagic transformation (HT) was identified on follow-up neuroimaging and classified as hemorrhagic infarction (HI1, HI2) or parenchymal hematoma (PH1, PH2). Baseline predictors assessed included demographics (age, sex, body mass index, smoking status), neurological status and severity (Glasgow Coma Scale, baseline NIHSS and repeat NIHSS), hemodynamic parameters (systolic and diastolic blood pressure), comorbidities (hypertension, diabetes mellitus, dyslipidemia, atrial fibrillation, coronary artery disease, prior stroke), laboratory measures (baseline glucose, HbA1c, total cholesterol, LDL, HDL, triglycerides, serum creatinine), and ASPECTS score. Data were entered, cleaned, and analyzed using IBM SPSS Statistics version 27. Continuous variables were summarized as mean ± SD or median (IQR), as appropriate, and compared between HT and non-HT groups using the unpaired t-test or Mann–Whitney U test. Categorical variables were presented as frequency (%) and compared using Fisher's exact test. Univariate logistic regression was performed to estimate odds ratios (ORs) with 95% confidence intervals (CI) for predictors of HT. All tests were two-sided, and a p value <0.05 was considered statistically significant.

Results:

A total of 100 thrombolysed hyperacute ischemic stroke patients were included. Most patients were aged e+65 years (57%), and 66% were male. The mean BMI was 26.12±4.35 kg/m². Baseline neurological severity indicated moderate deficits (baseline NIHSS mean 11.96±5.29; repeat NIHSS mean 5.46±5.14), and mean GCS was 11.59±3.00. Baseline hemodynamics demonstrated mean SBP 134.29±14.60 mmHg and DBP 89.59±10.25 mmHg (Table I).

Table I. *Demographic and Clinical characteristics of the participants (n=100)*

Variables	Frequency	Percentage
Age group (years)		
35-44	6	6.0
45-54	8	8.0
55-64	29	29.0
≥65	57	57.0
Mean±SD	65.94 ±10.10	
Median (min-max)	66.50 (35-80)	
Gender		
Male	66	66.0
Female	34	34.0
BMI (kg/m^2)		
Mean±SD	26.12 ±4.35	
Median (min-max)	26.30 (18.6-36.4)	
Smoking History		
Smoker	37	37.0
Non-smoker	63	63.0
Clinical characteristics		
SBP (mmHg) Mean ± SD	134.29 ± 14.60	
Median (min-max)	140 (100-159)	
DBP (mmHg) Mean ± SD	89.59 ± 10.25	
Median (min-max)	90 (60-120)	
Pulse (bpm) Mean ± SD	80.42 ± 14.67	
Median (min-max)	81 (45-121)	
GCS score Mean ± SD	11.59±3.00	
Median (min-max)	12.0 (4.0-15.0)	
Baseline NIHSS score Mean±SD	11.96±5.2912	
Median (min-max)	(5-22)	
Repeat NIHSS score Mean±SD	5.46±5.14	
Median (min-max)	5 (0-20)	

Table II. *Baseline clinical, laboratory, and outcome characteristics of the study population (n=100)*

Variables		Mean ± SD(n)	Median (min–max)(%)
Baseline glucose (mmol/L)		8.11 ± 2.50	8.22 (3.33 – 16.0)
Baseline HbA1c (%)		6.37 ±1.18	6.10 (4.5-10.7)
Total Cholesterol (mg/dL)		179.59 ± 37.47	180.85 (100.0–249.6)
LDL (mg/dL)		102.79 ± 34.24	99.70 (46.0–178.2)
HDL (mg/dL)		42.68 ± 9.17	41.00 (20.0–65.1)
Triglycerides (mg/dL)		176.91 ± 58.56	196.00 (69.0–277.1)
Creatinine (mg/dL)		1.13 ± 0.42	1.10 (0.50 – 2.29)
ASPECTS score		8.34 ± 1.09	8.00 (7-10)
Hemorrhagic transformation		12	12.0
Types of hemorrhagic transformation	HI1	2	2.0
	HI2	8	8.0
	PH1	1	1.0
	PH2	1	1.0
In hospital mortality		2	2.0
Hospital stay		7.20±3.84	6.5 (1-15)
mRS at discharge		1.61 ± 1.71	1 (0–6)

Mean baseline glucose was 8.11±2.50 mmol/L and mean HbA1c 6.37±1.18%. Mean lipid values were TC 179.59±37.47 mg/dL, LDL 102.79±34.24 mg/dL, HDL 42.68±9.17 mg/dL, and triglycerides 176.91±58.56 mg/dL. Median ASPECTS score was 8 (range 7–10). Hemorrhagic transformation occurred in 12 patients (12%) with subtype distribution HI1 2%, HI2 8%, PH1 1%, and PH2 1% (Table II).

Patients with HT were older (median 77 vs 65 years; p=0.003) and more frequently smokers (10/12 vs 27/88; p=0.001). Sex and BMI did not differ significantly (Table III). HT was associated with differences in neurological scores and blood pressure, including lower GCS, higher baseline NIHSS, higher SBP and DBP, and higher repeat NIHSS (all p<0.05). HT was also associated with hypertension, diabetes, dyslipidemia, and prior stroke. Metabolic and lipid parameters were more adverse in the HT group, with higher baseline glucose and HbA1c, higher TC/LDL/TG, and lower HDL (Table IV).

Table III: *Baseline Demographic Characteristics of Patients Diagnosed with Hyperacute Ischemic Stroke with IV Thrombolysis*

Variables		Hemorrhagic transformation		p value
		Yes (n=12)	No (n=88)	
		f (%)	f (%)	
Age Median(IQR)		77 (70.25-79.0)	65 (60.25-72)	**^{u}0.003^{s}**
Sex	Male	6 (9.1)	60 (90.9)	^{f}0.329^{ns}
	Female	6 (17.6)	28 (82.4)	
BMI (Mean ± SD)		25.89 ±5.24	26.15 ±4.24	^{t}0.844^{ns}
Smoker		10 (27.0)	27 (73.0)	**^{f}0.001^{s}**

f=Fisher's Exact test, u= Mann-Whitney U test, s= statistically significant if p<0.05

Table IV. Clinical and biochemical predictors of Hemorrhagic Transformation in Patients Diagnosed with Hyperacute Ischemic Stroke with IV Thrombolysis

Variables	Hemorrhagic transformation		p value
	Yes (n=12) f (%)	No (n=88) f (%)	
GCS score Median (IQR)	6.0 (5.0-6.0)	12 (11-15)	<u0.001s
Baseline NIHSSMedian(IQR)	22 (19-22)	12 (6-14)	<u0.001s
SBP (mmHg)Median(IQR)	148.5 (140-150)	122 (120-147)	u0.007s
DBP (mmHg)Median(IQR)	95 (91.5-100)	89 (80-95)	u0.011s
Repeat NIHSSMedian(IQR)	17.5(16.25-19.5)	4 (1-7)	<u0.001s
Comorbidities			
HTN	11 (18.6)	1 (2.4)	f0.025s
DM	8 (29.6)	19 (70.4)	f0.003s
Dyslipidemia	11 (30.6)	25 (69.4)	<f0.001s
Atrial fibrillation	5 (20.8)	19 (79.2)	f0.153ns
CAD	2 (10.5)	17 (89.5)	f1.000ns
Prior stroke	8 (38.1)	13 (61.9)	<f0.001s
Baseline glucose (mmol/L) Median(IQR)	10.38 (8.84-11.86)	8.19 (6.05-9.26)	u0.003s
Baseline HbA1c (%) Median (IQR)	7.7 (6.52-9.27)	6.0 (5.50-6.50)	<u0.001s
TC (mg/dL) Median(IQR)	234.6 (215.47-249.60)	171.9 (145.50-196.82)	<u0.001s
LDL (mg/dL) Median(IQR)	133.4 (116.62-168.70)	99.7 (71.20-125.62)	<u0.001s
HDL (mg/dL) Mean ± SD	38.66 ± 4.35	43.23 ± 9.52	t0.009s
TG (mg/dL) Median(IQR)	224.5 (201.45-265.62)	163.6 (114.50-221.70)	u0.001s
S. Creatinine (mg/dL) Median(IQR)	1.16 (0.99-1.34)	1.06 (0.77-1.38)	u0.614ns
ASPECTS score Median(IQR)	10 (9-10)	8 (7-9)	<u0.001s

f=Fisher's Exact test
u= Mann-Whitney U test
s= statistically significant if p<0.05

HT was associated with longer hospital stay (median 9.5 vs 6 days; p=0.024) and higher in-hospital mortality (2 deaths in HT group vs none in non-HT group; p=0.013). Discharge mRS did not differ (Table V).

Table V. Hospital outcomes between patients with and without hemorrhagic transformation following IV thrombolysis

Variables	Hemorrhagic transformation		p value
	Yes (n=12)	No (n=88)	
Hospital stay (days)Median(IQR)	9.5 (8.0-11.75)	6 (4-10)	$^{u}0.024^{s}$
mRS at dischargeMedian(IQR)	1 (0-2.75)	1(0-3)	$^{u}0.878^{ns}$
In hospital mortality	2 (100)	0 (0.0)	$^{f}0.013^{s}$

u= Mann-Whitney U test

s= statistically significant if p<0.05

On univariate logistic regression, significant predictors included age >65 years, smoking, GCS score(per 1-point decrease), baseline NIHSS, SBP, baseline glucose, HbA1c, HTN, DM, dyslipidemia, prior stroke, TC, LDL, TG, and ASPECTS score (Table VI).

Table VI. *Baseline Demographic Characteristics of Patients Diagnosed with Hyperacute Ischemic Stroke with IV Thrombolysis*

Variables	Univariate Logistic Regression	
	Adjusted OR (95% CI)	p-value
Age (>65)	10.04 (1.24-81.15)	0.030
Smoker	11.29 (2.31-55.08)	0.003
GCS score	4.72 (1.93–11.51)	<0.001
(per 1-point decrease)		
Baseline NIHSS	1.86 (1.36-2.54)	<0.001
SBP	2.02 (1.16-3.52)	0.012
DBP	1.71 (0.98-3.00)	0.057
Baseline glucose	1.40 (1.09-1.80)	0.008
Baseline HbA1c	2.80 (1.67-4.71)	0.001
HTN	9.16 (1.13-74.08)	0.038
DM	7.26 (1.97-26.73)	0.003
Dyslipidemia	27.72 (3.98-226.11)	0.002
Prior stroke	11.54 (3.03-43.92)	<0.001
TC	2.18 (1.45-3.28)	<0.001
LDL	1.54 (1.20-1.99)	<0.001
HDL	0.56 (0.88-1.01)	0.101
TG	1.26 (1.08-1.49)	0.003
S.creatinine	1.14 (0.27-4.77)	0.857
ASPECTS	4.21 (1.81-9.81)	0.001

Discussion:

In our cohort of 100 hyperacute ischemic stroke patients treated with IV rt-PA, hemorrhagic transformation (HT) occurred in 12% (HI1 2%, HI2 8%, PH1 1%, PH2 1%). This frequency sits in the messy middle of what's been reported across different thrombolysis series, where rates swing depending on imaging timing, definition (any HT vs symptomatic ICH), baseline severity, and how aggressively blood pressure and glucose are controlled. For example, Viswanathan et al. reported 32% intracerebral hemorrhage/ hemorrhagic transformation at 24 hours, with 9% meeting criteria for symptomatic intracerebral hemorrhage (sICH) [10]. In contrast, Tork et al. observed lower post-rtPA bleeding rates: 6.0% asymptomatic ICH and 2.7% symptomatic ICH[2]. A smaller Egyptian thrombolysis cohort from Sohag University found HT in 2 of 21 thrombolysed patients (about 9.5%)[11]. Taken together, our 12% overall HT rate is plausible for a real-world stroke unit population, and the differences across studies likely reflect heterogeneity in case-mix (especially cardioembolic burden and stroke size), imaging criteria, and sample size.

Age and smoking emerged as prominent baseline predictors in our data. Patients with HT were older (median 77 vs 65 years, p=0.003) and were disproportionately smokers (p=0.001). In univariate logistic regression, age >65 years (OR 10.04, 95% CI 1.24–81.15, p=0.030) and smoking (OR 11.29, 95% CI 2.31–55.08, p=0.003) substantially increased the odds of HT. This aligns with the Sohag thrombolysis experience, where older age was significantly associated with HT (p=0.024)[11]. Mehta et al. similarly showed that age <65 predicted better post-thrombolysis

outcomes (p=0.02), reinforcing the broader theme that older patients carry higher complication and poorer recovery risk after IVT[12].

Stroke severity was a consistent signal across our analysis and the comparison literature. In our cohort, baseline NIHSS was markedly higher among those who developed HT (median 22 vs 12, p<0.001), and repeat NIHSS remained worse (median 17.5 vs 4, p<0.001). On univariate regression, baseline NIHSS increased HT odds (OR 1.86, 95% CI 1.36–2.54, p<0.001). External studies echo this: Sohag reported associations between HT and high admission NIHSS (trend, p=0.085) and especially NIHSS at 24 hours (p=0.001)[11]. Mehta et al. also found NIHSS <15 predicted favorable outcome and higher admission NIHSS / NIHSS >15 predicted poor outcome (both p<0.001)[12]. While some of these papers focus on outcome rather than HT specifically, severity remains tightly tied to both hemorrhagic risk and prognosis, likely via larger infarct core, more blood–brain barrier disruption, and reperfusion injury.

Hemodynamic and metabolic variables also mattered. Our HT group had higher SBP (median 148.5 vs 122, p=0.007) and DBP (median 95 vs 89, p=0.011). In regression, SBP remained significant (OR 2.02, 95% CI 1.16–3.52, p=0.012), while DBP showed a borderline association (OR 1.71, 95% CI 0.98–3.00, p=0.057). Hyperglycemia and chronic dysglycemia were strongly associated with HT: baseline glucose (median 10.38 vs 8.19, p=0.003; OR 1.40, 95% CI 1.09–1.80, p=0.008) and HbA1c (median 7.7 vs 6.0, p<0.001; OR 2.80, 95% CI 1.67–4.71, p=0.001). These findings mirror Mehta et al., where higher admission random blood sugar and higher glycated hemoglobin were linked with poor outcomes (both highly significant), and higher diastolic BP also showed association (p=0.04)[12]. Mechanistically, hyperglycemia is biologically plausible as a hemorrhagic risk amplifier through endothelial dysfunction, oxidative stress, and worsened reperfusion injury, especially in large infarcts.

Comorbid vascular risk factors and lipid variables showed strong associations with HT in our sample, although the wide confidence intervals scream "small event number." Hypertension (OR 9.16, p=0.038), diabetes (OR 7.26, p=0.003), dyslipidemia (OR 27.72, p=0.002), and prior stroke (OR 11.54, p<0.001) all increased the odds of HT. Lipid measures were also associated (TC OR 2.18, LDL OR 1.54, TG OR 1.26, all significant), while HDL was not. In the Sohag cohort, ischemic heart disease was associated with HT (p=0.048)[11]. And in the Egyptian IVT outcome study by Tork et al., AF, hypertension, and diabetes were independent predictors of poor outcome, underscoring that vascular comorbidity load remains a major determinant of post-thrombolysis trajectories (even if their primary endpoint was functional outcome rather than HT)[2].

Finally, HT was not a "harmless imaging finding" in our cohort. Patients with HT had longer hospital stays (median 9.5 vs 6 days, p=0.024) and higher in-hospital mortality (2 deaths in HT vs 0 without HT, p=0.013). This pattern matches the broader literature where hemorrhagic complications and neurological deterioration after thrombolysis cluster with worse outcomes, and symptomatic hemorrhage is particularly prognostically toxic[10].

Conclusion:

HT occurred in 12% of thrombolysed hyperacute ischemic stroke patients in our setting and was predominantly hemorrhagic infarction. Several demographic, clinical, biochemical and imaging variables were associated with HT in univariate analyses. The paradoxical association between higher ASPECTS and HT warrants cautious interpretation and may reflect selection effects within thrombolysis-eligible populations. Larger studies with richer imaging and multivariable modeling are needed to validate these predictors and refine risk stratification after IV thrombolysis.

Limitations:

This study has limitations that should be acknowledged. It is a single-center study with a modest sample size and a limited number of HT events, which restricts statistical power and limits the stability of regression estimates. Because we used univariate logistic regression, residual confounding is likely, and observed associations may not represent independent predictors. Additionally, several potentially important determinants of HT, such as onset-to-needle time, occlusion site, collateral grade, recanalization status, infarct volume, antithrombotic exposure,

and detailed imaging markers, were not incorporated. Future studies should prioritize multicenter recruitment, standardized imaging adjudication, and multivariable or penalized regression approaches suited to small event counts to develop more robust prediction models.

Data Availability:
The datasets analysed during the current study are not publicly available due to the continuation of analyses but are available from the corresponding author on reasonable request.

Conflict of Interest:
The authors declare no conflicts of interest.

Funding:
No external funding was received for this study.

Ethical consideration:
The study was approved by the Ethical Review Committee of National Institute of Neurosciences & Hospital (NINS&H), Dhaka, Bangladesh. Informed consent was obtained from each participant or caregivers of the patients.

Author Contributions:
All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; had agreed on the journal to which the article had been submitted; and agreed to be account able for all aspects of the work.

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