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ORIGINAL RESEARCH

Admission Hyperglycaemia as a Predictor of Stroke Severity and Mortality: Evidence from a Prospective Emergency Department Cohort

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ABSTRACT:

Background: Admission hyperglycaemia is commonly observed in acute stroke and has been linked to poorer neurological outcomes. Its role as a prognostic marker in emergency settings warrants evaluation.

Objective: To assess the association between admission random blood sugar (RBS) levels and stroke severity, and to determine its relationship with in-hospital outcomes.

Methods: This prospective observational study included 115 adult patients with radiologically confirmed stroke presenting to a tertiary care emergency department. Admission RBS levels were recorded, and stroke severity was categorized using the National Institutes of Health Stroke Scale (NIHSS). Statistical analysis was performed with $p < 0.05$ considered significant.

Results: Mean admission RBS levels increased significantly with stroke severity, rising from 175.6 mg/dL in minor stroke to 216.5 mg/dL in severe stroke ($p < 0.01$). Overall, in-hospital mortality was 7%.

Conclusion: Admission hyperglycaemia correlates positively with stroke severity and may serve as a simple, rapid prognostic marker in acute stroke care.

Keywords: Admission hyperglycaemia; Acute stroke; Random blood sugar; NIHSS; Stroke severity; In-hospital mortality; Emergency medicine; Prognostic marker; Diabetes mellitus; Cerebrovascular accident

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INTRODUCTION

Hyperglycaemia is frequently observed in patients presenting with acute stroke and has been consistently associated with worse neurological and functional outcomes.[1] Elevated blood glucose levels at admission are seen not only in patients with pre-existing diabetes mellitus but also in non-diabetic individuals due to stress-induced metabolic responses. Increasing evidence suggests that acute hyperglycaemia may independently contribute to neuronal injury rather than merely reflecting physiological stress.[2]

Experimental and clinical studies have demonstrated that elevated glucose levels exacerbate ischemic brain injury through multiple mechanisms, including enhanced anaerobic glycolysis leading to lactic acidosis, oxidative stress, endothelial dysfunction, and disruption of the blood–brain barrier.[3] Hyperglycaemia has also been linked to larger infarct volumes, increased risk of haemorrhagic transformation, and poorer functional recovery following ischemic stroke.[1,4]

Large observational studies and meta-analyses have reported that admission hyperglycaemia is associated with significantly increased mortality and dependency at follow-up, irrespective of prior diabetic status.[1,5] Furthermore, chronic poor glycaemic control, as reflected by elevated HbA1c levels, has been correlated with greater stroke severity at presentation and worse short-term outcomes.[6-10] These findings highlight the potential role of admission glucose as a simple and accessible prognostic biomarker in acute stroke care.

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In countries such as India, where diabetes prevalence is rapidly increasing and glycaemic control often remains suboptimal, understanding the relationship between admission random blood sugar (RBS) levels and quantified stroke severity is of considerable clinical importance. Early identification of hyperglycaemia may facilitate risk stratification and targeted metabolic optimization in emergency settings.

This prospective observational study was therefore conducted to evaluate the association between admission hyperglycaemia and stroke severity, as measured by the National Institutes of Health Stroke Scale (NIHSS), and to assess its relationship with in-hospital outcomes.

MATERIALS AND METHODS

Study Design and Participants

This prospective observational study was conducted in the Department of Emergency Medicine at Maharashtra Post Graduate Institute of Medical Education and Research, MUHS, Nashik, over a two-year period. Institutional Ethics Committee approval was obtained prior to commencement of the study (Ref. No. MPGIMER Nashik/IEC/Outward No/33/2024), and written informed consent was secured from all enrolled participants. The approval remained valid throughout the study duration in accordance with institutional guidelines.

The primary objective of this study was to evaluate the association between admission random blood sugar (RBS) levels and stroke severity, as well as short-term in-hospital outcomes among patients presenting with acute cerebrovascular accidents.

A total of 115 eligible patients were included based on prior sample size estimation (expected incidence 5%, 95% confidence interval, 4% margin of error). Patients were recruited consecutively upon confirmation of stroke diagnosis.

Inclusion and Exclusion Criteria

Inclusion Criteria

- Male and female patients aged >18-years
- Patients presenting with acute neurological deficits suggestive of stroke
- Radiologically confirmed diagnosis of cerebrovascular accident

Exclusion Criteria

- Patients younger than 18-years
- Stroke secondary to trauma or road traffic accidents
- Patients intubated prior to arrival
- Patients with significant systemic comorbidities likely to independently influence glycaemic status or stroke outcomes
- Cases with incomplete clinical or laboratory data

Data Collection and Measurements

All patients presenting with focal neurological deficits meeting Cincinnati Stroke Scale criteria were assessed in the emergency department. Initial management followed standard airway, breathing, and circulation (ABC) stabilization protocols. A non-contrast CT scan of the brain was performed in all cases to confirm the diagnosis and classify stroke as ischemic or haemorrhagic.

Admission random blood sugar (RBS) levels were measured at the time of emergency presentation using standardized laboratory methods. Stroke severity was quantified using the National Institutes of Health Stroke Scale (NIHSS) and categorized as:

- Minor (0–4)
- Moderate (5–15)
- Moderate to severe (16–20)
- Severe (>21)

Demographic parameters including age, sex, and residence were recorded. Duration of hospitalization and in-hospital outcomes (survival or mortality) were documented for all participants.

Statistical Analysis

Data were recorded in a structured proforma and analyzed using SPSS version 26.0. Categorical variables were summarized as frequencies and percentages. Associations between glycaemic levels and stroke severity categories were assessed using the Chi-square test where appropriate. Continuous variables, including RBS levels, were expressed as mean ± standard deviation. Comparisons across severity groups were performed using one-way ANOVA for normally distributed data and Kruskal–Wallis test for non-normally distributed data. A $p\text{-value} < 0.05$ was considered statistically significant. Graphical representations were generated using Microsoft Excel 2021.

RESULTS

The study of 115 cerebrovascular accident patients revealed that stroke predominantly affected the elderly, with over half aged above 60-years. Urban residents formed the majority of cases. Hypertension emerged as the leading co-morbidity, surpassing diabetes (Table 1). Ischemic stroke was the dominant subtype, accounting for nearly three-fourths of cases, whereas hemorrhagic stroke was less frequent. Men and women were almost equally represented (Table 2). These findings highlight the strong association of advancing age and hypertension with stroke occurrence and reflect epidemiological trends consistent with established vascular risk profiles.

Table 1: Distribution of study groups as per co-morbidity.

Co-morbidity	N	%
Diabetes	50	43.5%
Hypertension	65	56.5%
Total	115	100.0%

Table 2: Distribution of study groups as per gender.

Gender	N	%
Female	57	49.6%
Male	58	50.4%
Total	115	100.0%

Table 3: Distribution of study groups as per severity of stroke (NIHSS Scale).

Severity of Stroke	N	%
Minor	17	14.8%
Moderate	51	44.3%
Moderate to Severe	39	33.9%
Severe	8	7.0%
Total	115	100.0%

Stroke severity was classified based on NIHSS scores (Table 3). Moderate stroke was the most common (44.3%), followed by moderate to severe (33.9%), minor (14.8%), and severe stroke (7.0%), indicating that most patients had moderate neurological deficits.

The survival rate was high being 93.0%, while mortality was recorded in 7.0%, suggesting favourable in-hospital outcomes with standard treatment (Table 4).

Table 4: Distribution of study groups as per Outcome.

Outcome	N	%
Survived	107	93.0%
Deceased	8	7.0%
Total	115	100.0%

Comparison of stroke type with underlying co-morbidities demonstrated that hypertensive patients were significantly more likely to develop haemorrhagic stroke, whereas ischemic stroke was more common in diabetics. This association was statistically significant, reinforcing hypertension as a major risk factor for intracranial bleeding. However, when stroke severity was examined, both diabetic and hypertensive patients showed a similar pattern, with moderate stroke being the most frequent presentation. The absence of a significant difference in severity suggests that although co-morbidities influence stroke subtype, they may not substantially alter the clinical severity at presentation.

Table 5: Mean hospital stay in diabetics and hypertensives.

Variables	Group	N	Mean	SD	*p- value*
Hospital Stay in days	Diabetic	50	6.08	2.69	0.34
	Hypertensive	65	6.62	3.24	

As shown in Table 5, Hypertensive patients had a slightly longer stay (mean 6.62 days) compared to diabetic patients (mean 6.08 days), but the difference was not statistically significant ($p = 0.34$).

When stroke outcomes were compared between diabetic and hypertensive patients (Table 6), a slightly higher mortality was observed in the diabetic group. Nevertheless, the majority of patients in both groups survived hospitalization. Statistical analysis revealed no significant association between underlying co-morbidity and outcome. This suggests that despite differences in stroke subtype distribution, short-term in-hospital prognosis remained broadly similar across both groups. The findings indicate that factors beyond baseline co-morbidity—such as stroke severity, timely intervention, and supportive care—may play a more decisive role in determining survival.

Table 6: Association of outcome of stroke with co-morbidity.

Outcome	Group		Total
	Diabetes	Hypertensive	
Survived	45	62	107
	90.0%	95.4%	93.0%
Deceased	5	3	8
	10.0%	4.6%	7.0%
Total	50	65	115
	100.0%	100.0%	100.0%
p- value – 0.29			

Table 7: Association of mean RBS levels with stroke severity.

Variables	Group	N	Mean	SD	*p- value*
RBS at admission (mg%)	Minor	17	175.60	81.37	<0.01
	Moderate	51	189.54	77.35	
	Mod to Severe	39	198.12	69.28	
	Severe	8	216.50	83.62	

This table and figure (Table 7, Figure 1) correlates random blood sugar (RBS) levels with stroke severity. Mean RBS values increased with stroke severity, from 175.6 mg/DL in minor stroke to 216.5 mg/DL in severe stroke. The association was statistically significant ($p < 0.01$), suggesting hyperglycaemia as a marker of worse stroke presentation.

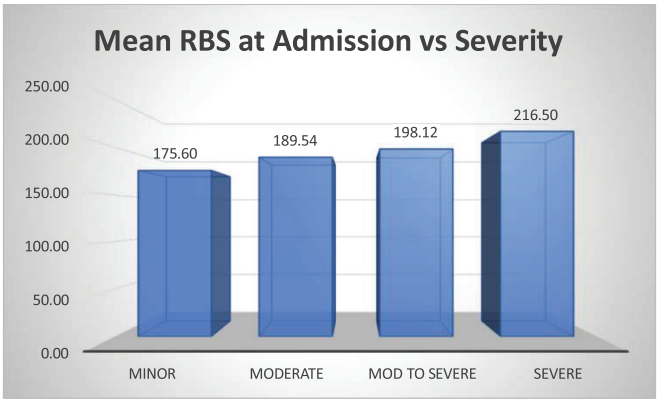

Figure 1: Mean RBS at admission and stroke severity

DISCUSSION

The present prospective observational study demonstrates a statistically significant association between admission hyperglycaemia and increasing stroke severity, as quantified by NIHSS scores. Mean random blood sugar (RBS) levels rose progressively from minor to severe stroke categories ($p < 0.01$), supporting the hypothesis that acute hyperglycaemia is not merely an epiphenomenon of physiological stress but may contribute directly to worsened neurological injury.

Our findings (Table 8) are consistent with the landmark systematic review by Capes *et al.*, which reported that stress hyperglycaemia is associated with a threefold increase in mortality in non-diabetic stroke patients and a significant increase in poor functional outcomes among diabetics.[1] Similarly, Bruno et al. demonstrated that elevated admission glucose independently predicted unfavorable outcomes and larger infarct volumes in acute ischemic stroke.[4] The graded rise in RBS observed in our cohort across NIHSS categories parallels the dynamic relationship described in the ECASS-II trial, where persistent hyperglycaemia was a strong predictor of poor recovery.[2] Pathophysiologically, hyperglycaemia exacerbates ischemic neuronal injury through increased anaerobic glycolysis, lactic acidosis, oxidative stress, and blood–brain barrier disruption.[3] These mechanisms may explain why patients in our severe stroke category demonstrated the highest mean admission glucose levels. Ntaios *et al.* further described a J-shaped association between serum glucose and stroke outcomes, indicating that both hyperglycaemia and hypoglycaemia are associated with adverse prognosis.[5] Our results specifically reinforce the detrimental effect of elevated glucose levels in the acute setting.

Although, mortality in our cohort was relatively low (7%), a trend toward higher mortality in diabetics was observed, though not statistically significant. This may reflect timely emergency stabilization, standardized stroke protocols, and supportive care. Nonetheless, the association between hyperglycaemia and stroke severity suggests that early glucose assessment in the emergency department can serve as a simple, rapid, and cost-effective prognostic marker. In the Indian context, where diabetes prevalence is increasing and glycaemic control often remains suboptimal, these findings have particular relevance. Routine monitoring and judicious correction of hyperglycaemia in acute stroke may improve risk stratification and guide early therapeutic decisions. Future multicentric studies with long-term follow-up are warranted to determine whether aggressive glucose control translates into improved neurological and functional outcomes.

Our findings that admission hyperglycemia correlates with increased stroke severity and mortality align with the broader understanding that acute metabolic dysregulation significantly amplifies end-organ vulnerability in emergency settings. The interplay between hyperglycemia and vascular instability has been increasingly emphasized in recent literature. Artificial intelligence–based early detection models in diabetic-hypertensive emergencies have demonstrated that metabolic fluctuations often precede overt hemodynamic deterioration, suggesting that hyperglycemia may serve as an early surrogate marker of systemic stress and impending organ dysfunction.[11] Such models reinforce the need for early identification of high-risk patients in the emergency department.

Furthermore, evidence from hypertensive crisis cohorts highlights the predominance of neurological involvement and intracerebral hemorrhage in patients presenting with severe blood pressure elevation.[12] When hyperglycemia coexists with acute hypertension, endothelial dysfunction, oxidative stress, and impaired cerebral autoregulation may synergistically worsen cerebral edema and infarct expansion. This pathophysiological overlap may partly explain the higher NIHSS scores and poorer outcomes observed in hyperglycemic stroke patients in our study.

The cardiovascular parallels are equally compelling. Studies evaluating myocardial infarction in emergency settings have reported significant associations between metabolic comorbidities and adverse electrical instability, including ventricular fibrillation.[13] These findings underscore how acute metabolic stress states—similar to admission hyperglycemia in stroke—may reflect systemic vulnerability rather than isolated organ pathology. Additionally, emergency triage research using the Emergency Severity Index (ESI) has demonstrated that metabolic and hemodynamic derangements influence acuity categorization and outcomes,[14] reinforcing the prognostic weight of abnormal admission parameters.

The long-term consequences of uncontrolled diabetes are dramatically illustrated in reports of advanced diabetic complications such as auto-amputation secondary to chronic metabolic derangement.[15] While, our study focuses on acute stroke outcomes, these extreme manifestations emphasize the systemic microvascular and macrovascular damage perpetuated by sustained hyperglycemia, which likely predisposes to both ischemic and hemorrhagic cerebrovascular events.

From a vascular protection standpoint, high-intensity statins have demonstrated endothelial stabilizing, anti-inflammatory, and plaque-stabilizing effects in acute coronary syndromes.[16,17] Given that hyperglycemia promotes inflammatory cytokine release and oxidative stress, early aggressive vascular risk modification—including lipid stabilization—may have parallel implications in hyperglycemic stroke populations.

Interestingly, infectious comorbidities in acute cardiovascular presentations have also been documented at higher prevalence in emergency cohorts,[18] suggesting that systemic inflammatory burden may coexist with metabolic instability. This further supports the

Table 8: Summary of key findings

- The majority of patients (53%) were aged >60 years, followed by 44.3% aged 41–60 years. Only 2.6% were aged ≤40 years.
- The mean age of the study population was 61.8 years.
- Gender distribution was nearly equal: 50.4% males and 49.6% females.
- Most patients (71.3%) were from urban areas; 28.7% were rural residents.
- Mortality was higher in diabetics (10%) than hypertensives (4.6%), but this difference was not statistically significant ($p = 0.29$).
- Mean random blood sugar (RBS) levels correlated significantly with stroke severity: increasing from 175.6 mg/Dl in minor stroke to 216.5 mg/Dl in severe stroke ($p < 0.01$).

concept that admission hyperglycemia may represent a broader marker of physiological stress rather than merely a glycemic abnormality.

Beyond acute stress hyperglycaemia, emerging metabolic biomarker research further strengthens the biological plausibility of our findings. Elevated serum uric acid levels have been shown to correlate significantly with visceral adiposity indices including BMI, waist-to-hip ratio, and sagittal abdominal diameter in prediabetic and drug-naïve diabetic individuals.[19,20] Uric acid is increasingly recognized not merely as a metabolic byproduct but as a mediator of oxidative stress, endothelial dysfunction, and low-grade inflammation—pathways that are central to both cerebrovascular injury and atherosclerotic progression. Hyperuricaemia promotes mitochondrial oxidative stress, impairs nitric oxide bioavailability, and enhances pro-inflammatory cytokine release, all of which may exacerbate ischemic neuronal damage in the setting of acute stroke. Thus, admission hyperglycaemia may represent part of a broader metabolic-inflammatory milieu characterized by insulin resistance and oxidative imbalance.

Similarly, osteocalcin—a bone-derived endocrine hormone—has recently been identified as a regulator of glucose metabolism and insulin sensitivity. Circulating osteocalcin levels are significantly reduced in patients with type 2 diabetes and demonstrate strong inverse correlations with fasting blood glucose, HbA1c, and HOMA-IR indices.[21] Reduced osteocalcin levels reflect impaired â-cell function and heightened insulin resistance, thereby contributing to sustained hyperglycaemia. In the context of acute stroke, such chronic metabolic dysregulation may predispose to more severe neurological presentation. The inverse relationship between osteocalcin and glycaemic markers observed in metabolic studies provides additional mechanistic support for the graded rise in admission RBS across increasing NIHSS categories in our cohort.

Collectively, these biomarker data suggest that admission hyperglycaemia is not an isolated stress response but rather a manifestation of deeper metabolic-vascular derangement involving oxidative stress, inflammatory activation, endothelial dysfunction, and impaired insulin signaling. This integrative metabolic framework may help explain why higher glucose levels at presentation are consistently associated with greater stroke severity and poorer outcomes.

CONCLUSION

This prospective study demonstrates a significant positive association between admission hyperglycaemia and stroke severity, with higher random blood sugar levels correlating with increasing NIHSS scores. Although overall, in-hospital mortality was low, elevated glucose levels were consistently linked to more severe neurological presentation. Admission blood glucose measurement, being rapid and readily available in the emergency setting, may serve as a simple and practical prognostic marker for early risk stratification in acute stroke care.

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