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HBA1C AND ISCHEMIC STROKE IN DIABETIC PATIENTS: A CROSS-SECTIONAL STUDY FROM A TRIBAL REGION OF GUJARAT, INDIA

Internal Medicine

Dr. Hardik Parmar* — 3rd Year Post-Graduate Resident, Department of General Medicine, Zydus Medical College and Hospital, Dahod, Gujarat, India*Corresponding Author

Dr. Mohit Desai — Professor, Department of General Medicine, Zydus Medical College and Hospital, Dahod, Gujarat, India

Dr. Abhi Patel — 3rd Year Post-Graduate Resident, Department of General Medicine, Zydus Medical College and Hospital, Dahod, Gujarat, India

ABSTRACT

Background: Diabetes mellitus is an established independent risk factor for ischemic stroke, and glycated hemoglobin (HbA1c) is a widely used surrogate of chronic glycemic exposure. Evidence suggests that elevated HbA1c is associated with greater stroke severity and poorer functional recovery; however, data from tribal and resource-limited regions of India remain scarce.**Methods:** A descriptive, observational, cross-sectional hospital-based study was conducted at the Department of General Medicine, Zydus Medical College and Hospital (ZMCH), Dahod, Gujarat, from March 2024 to October 2025. Fifty adult patients (≥40 years) with diabetes mellitus admitted with confirmed acute ischemic stroke were enrolled by consecutive sampling. HbA1c was measured at admission and categorized as <7.0%, 7.0–8.9%, and ≥9.0%. Primary outcomes were stroke severity (NIHSS) at admission and functional outcome (mRS) at discharge. Secondary outcomes included in-hospital complications, length of stay (LOS), and time to presentation. Group comparisons used chi-square and one-way ANOVA; multivariable logistic regression identified independent predictors of poor functional outcome (mRS 3–6).**Results:** Male predominance (56%) and peak age at 50–69 years were observed. Hypertension (72%) and dyslipidemia (70%) were highly prevalent. Most patients (62%) had HbA1c 7.0–8.9%; 26% had HbA1c ≥9.0%. A stepwise increase in mean NIHSS was observed across HbA1c categories (3.5 vs. 8.13 vs. 13.08; F=27.13, p<0.001). Poor discharge outcome (mRS 3–6) was significantly more frequent in the ≥9.0% group (92.3% vs. 32.3% vs. 0%; χ^2=18.76, p=0.0001). Longer LOS was associated with higher HbA1c (χ^2=18.8, p=0.0009). On multivariable logistic regression, HbA1c ordinal (OR=22.48, 95% CI: 1.03–489.82; p=0.048) and NIHSS (OR=1.87, 95% CI: 1.08–3.26; p=0.027) independently predicted poor discharge outcome.**Conclusions:** Higher HbA1c is independently associated with greater neurological deficit, poor functional outcome, and a more demanding inpatient course in diabetic ischemic stroke patients in a tribal-region hospital. HbA1c is a pragmatic, routinely available biomarker that can enhance risk stratification and clinical decision-making.

KEYWORDS

Hba1c; Glycated Hemoglobin; Ischemic Stroke; Diabetes Mellitus; Nihss; Modified Rankin Scale; Tribal Gujarat; India

INTRODUCTION

Glycated hemoglobin (HbA1c) reflects average blood glucose concentration over the preceding 8–12 weeks and is a cornerstone biomarker for the diagnosis and monitoring of diabetes mellitus [1]. Diabetes mellitus is recognized as an established independent risk factor for ischemic stroke, associated with higher in-hospital mortality, greater neurological deficits, and poorer functional recovery [1,2]. The biological basis is well-established: sustained hyperglycemia promotes endothelial dysfunction, oxidative stress, accelerated atherosclerosis, and microvascular injury—collectively increasing stroke susceptibility and worsening neurological outcomes [2].

Epidemiological evidence from large cohort studies consistently demonstrates that elevated HbA1c strongly predicts stroke incidence in patients with type 2 diabetes mellitus [3]. A U-shaped relationship has also been described, whereby both very high and very low HbA1c may elevate stroke risk through distinct mechanisms: hyperglycemia-mediated vascular injury on one end and hypoglycemia-induced neuroinflammatory activation on the other [2,4]. Despite a wealth of data from Western and East Asian populations, Indian data—particularly from tribal and resource-limited settings—remain scarce.

The Dahod district of Gujarat is a predominantly tribal region characterized by a high burden of diabetes, hypertension, and limited healthcare access. Patients in this setting frequently present late, often have sub optimally controlled diabetes, and carry substantial cardiometabolic risk. Understanding the clinical implications of HbA1c in this context is therefore of direct public health and bedside relevance.

This study evaluated the association between HbA1c and stroke severity (NIHSS) and functional outcome (mRS) in adult diabetic patients with acute ischemic stroke at ZMCH, Dahod. Secondary objectives included comparing vascular risk factors, etiologic subtypes, complications, and LOS across HbA1c categories, and estimating the independent predictive value of HbA1c for poor discharge outcome.

MATERIALS AND METHODS

Study Design and Setting

This was a descriptive, observational, cross-sectional hospital-based study conducted at the Department of General Medicine, ZMCH, Dahod, Gujarat, India from March 2024 to October 2025.

Participants

Adults aged ≥40 years with diabetes mellitus (pre-existing or newly detected) admitted with confirmed acute ischemic stroke (clinical assessment plus CT/MRI brain) were enrolled by consecutive sampling (n=50).

Inclusion criteria: (i) age ≥40 years; (ii) diabetes mellitus (any type); (iii) acute ischemic stroke as the index event; (iv) written informed consent obtained.

Exclusion criteria: (i) hemorrhagic stroke or primary subarachnoid hemorrhage; (ii) stroke mimics (seizure with Todd's paresis, hypoglycemia-related focal deficit); (iii) isolated TIA without confirmed infarction; (iv) terminal illness or absent consent.

Data Collection

Data were collected using a pre-designed Case Record Form (CRF) with source verification from clinical records, laboratory reports, and neuroimaging. Variables included demographics, vascular risk factors (hypertension, dyslipidemia, smoking, coronary artery disease [CAD]), diabetes type and medications, vital signs, NIHSS at admission, and mRS at discharge. Etiologic subtype was classified by TOAST criteria. Imaging infarct territory was categorized as MCA, ACA, PCA, lacunar, or brainstem/cerebellum. Admission glucose was categorized as <140, 140–199, or ≥200 mg/dL.

Exposure Variable

HbA1c was measured at or near admission using standard laboratory immunoturbidimetric methods (NGSP-certified, DCCT-aligned) and categorized as <7.0% (well-controlled), 7.0–8.9% (sub optimally controlled), and ≥9.0% (poorly controlled).

Outcome Measures

Primary outcomes: (i) NIHSS at admission — minor (NIHSS 0–4), moderate (NIHSS 5–15), moderate-severe (NIHSS ≥16); (ii) mRS at discharge — good outcome (mRS 0–2) vs. poor outcome (mRS 3–6).

Secondary outcomes: in-hospital complications (pneumonia, urinary tract infection, deep vein thrombosis), LOS, time from symptom onset to presentation, and in-hospital stroke recurrence.

Statistical Analysis

Descriptive statistics are reported as mean±SD or median [IQR] for continuous variables and frequency (n, %) for categorical variables. Group comparisons across HbA1c categories used chi-square or Fisher's exact test (categorical) and one-way ANOVA (continuous). A two-sided $p<0.05$ was considered statistically significant. Multivariable binary logistic regression identified independent predictors of poor functional outcome (mRS 3–6), including HbA1c (ordinal), NIHSS, age, sex, hypertension, dyslipidemia, and CAD as covariates. Odds ratios (ORs) with 95% confidence intervals (CIs) and Wald p-values are reported.

RESULTS

Baseline Demographics and Vascular Risk Factors

Fifty patients meeting eligibility criteria were enrolled. Age peaked at 50–69 years (68%) with male predominance (56%). Hypertension (72%) and dyslipidemia (70%) were highly prevalent; smoking was documented in 16% and CAD in 14%. Baseline characteristics are summarized in Table 1.

Table 1. Baseline demographics and vascular risk factors (n=50)

Variable	n	%
Age group		
40–49 years	11	22.0
50–59 years	18	36.0
60–69 years	16	32.0
70–79 years	5	10.0
Sex		
Male	28	56.0
Female	22	44.0
Vascular Risk Factors		
Hypertension	36	72.0
Dyslipidemia	35	70.0
Smoking	8	16.0
Coronary artery disease (CAD)	7	14.0

3.2 HbA1c Category Distribution

Most patients had HbA1c ≥7.0%: 62% in the 7.0–8.9% range and 26% with HbA1c ≥9.0%; only 12% were well-controlled (Table 2). This distribution reflects the prevalent pattern of suboptimal glycemic control in resource-limited settings.

Table 2. HbA1c category distribution (n=50)

HbA1c Category	n	%
<7.0%	6	12.0
7.0–8.9%	31	62.0
≥9.0%	13	26.0

Stroke Severity (NIHSS) by HbA1c Category

Stroke severity showed a pronounced graded relationship with HbA1c. In the well-controlled group (<7.0%), 66.7% had minor strokes (NIHSS 0–4); in the poorly controlled group (≥9.0%), 23.1% had moderate-severe strokes (NIHSS ≥16). Mean NIHSS rose stepwise: 3.5 (SD 2.17) → 8.13 (SD 2.51) → 13.08 (SD 3.55) across the three HbA1c strata. Both the categorical NIHSS distribution (χ^2=33.04, p<0.001) and the mean NIHSS comparison (ANOVA F=27.13, p<0.001) were highly significant (Table 3).

Table 3. Stroke severity (NIHSS) by HbA1c category

HbA1c Category	Minor (NIHSS 0–4) n (%)	Moderate (NIHSS 5–15) n (%)	Mod-Severe (NIHSS ≥16) n(%)	Total	Mean NIHSS (SD)
<7.0%	4 (66.7%)	2 (33.3%)	0 (0.0%)	6	3.5 (2.17)
7.0–8.9%	1 (3.2%)	30 (96.8%)	0 (0.0%)	31	8.13 (2.51)
≥9.0%	0 (0.0%)	10 (76.9%)	3 (23.1%)	13	13.08 (3.55)
p-value	**χ^2=33.04, p<0.001**				**F=27.13, p<0.001**

Functional Outcome at Discharge (mRS) by HbA1c Category

Poor functional outcome (mRS 3–6) was absent in the well-controlled group (<7.0%), present in 32.3% of the 7.0–8.9% group, and in 92.3% of the ≥9.0% group (χ^2=18.76, p=0.0001; Table 4). This clear gradient confirms that chronic glycemic burden independently shapes early post-stroke recovery (3,5).

Table 4. Functional outcome (mRS) at discharge by HbA1c category (χ^2=18.76, p=0.0001)

HbA1c Category	Good outcome (mRS 0–2) n (%)	Poor outcome (mRS 3–6) n (%)	Total
<7.0%	6 (100.0%)	0 (0.0%)	6
7.0–8.9%	21 (67.7%)	10 (32.3%)	31
≥9.0%	1 (7.7%)	12 (92.3%)	13

Admission Hyperglycemia and Comorbid Hypertension by HbA1c Category

Admission hyperglycemia (glucose ≥200 mg/dL) was numerically more common in higher HbA1c strata (46.2% in ≥9.0% vs. 45.2% in 7.0–8.9% vs. 33.3% in <7.0%), and hypertension co-prevalence also clustered with poorer glycemic control (92.3% vs. 64.5% vs. 66.7%). Neither difference reached statistical significance (χ^2=2.19, p=0.701 and χ^2=3.61, p=0.165, respectively), but their co-clustering is clinically important as it amplifies total vascular burden (Table 5).

Table 5. Hypertension and admission glucose category by HbA1c category

HbA1c Category	Hypertension: No n (%)	Hypertension: Yes n (%)	Glucose <140 n (%)	Glucose 140–199 n (%)	Glucose ≥200 n (%)	Total
<7.0%	2 (33.3%)	4 (66.7%)	1 (16.7%)	3 (50.0%)	2 (33.3%)	6
7.0–8.9%	11 (35.5%)	20 (64.5%)	5 (16.1%)	12 (38.7%)	14 (45.2%)	31
≥9.0%	1 (7.7%)	12 (92.3%)	4 (30.8%)	3 (23.1%)	6 (46.2%)	13
p-value	**χ^2=3.61, p=0.165**		**χ^2=2.19, p=0.701**			

Infarct Territory and TOAST Etiologic Subtype by HbA1c Category

Large-artery atherosclerosis was the predominant TOAST subtype across all HbA1c strata, followed by cardioembolism and small-vessel occlusion. MCA territory infarcts predominated in all groups. Distributions did not differ significantly for etiology (χ^2=5.09, p=0.532) or infarct territory (χ^2=6.13, p=0.633). Both are presented together in Table 6.

Table 6. TOAST etiologic subtype and infarct territory by HbA1c category

Hb A1c Category	Large-artery n (%)	Cardioembolic n (%)	Small-vessel n (%)	Other/Und. n (%)	MCA n (%)	Lacunar n (%)	Other territory n (%)	Total
<7.0%	3 (50.0%)	1 (16.7%)	1 (16.7%)	1 (16.7%)	4 (66.7%)	0 (0.0%)	2 (33.3%)	6
7.0–8.9%	16 (51.6%)	8 (25.8%)	7 (22.6%)	0 (0.0%)	18 (58.1%)	4 (12.9%)	9 (29.0%)	31
≥9.0%	6 (46.2%)	2 (15.4%)	4 (30.8%)	1 (7.7%)	6 (46.2%)	3 (23.1%)	4 (30.8%)	13
p-value	**χ^2=5.09, p=0.532**				**χ^2=6.13, p=0.633**			

Secondary In-Hospital Outcomes by HbA1c Category

Table 7 presents four secondary outcomes: in-hospital complications, time to presentation, length of stay, and in-hospital recurrence. Length of stay was significantly longer in the ≥9.0% group (χ^2=18.8, p=0.0009), with 53.8% having LOS >10 days compared to 9.7% in the 7.0–8.9% group and 0% in the <7.0% group. A trend toward delayed presentation (>24 hours) was seen in the ≥9.0% group (38.5%). Complications and recurrence were numerically higher in the poorly controlled group, though neither reached statistical significance.

Table 7. Secondary in-hospital outcomes by HbA1c category

Hb1c Category	Complication No n (%)	Complication Yes n (%)	Presentation ≤4.5 h n (%)	Presentation 4.5–24 h n (%)	Presentation >24 h n (%)	LOS ≤5 days n (%)	LOS 6–10 days n (%)	LOS >10 days n (%)	Recurrence Yes n (%)

<7.0 %	4 (66.7 %)	2 (33.3 %)	1 (16. 7%)	3 (50.0 %)	2 (33.3%)	4 (66.7 %)	2 (33.3 %)	0 (0.0%)	0 (0.0 %)
7.0–8 .9%	24 (77.4 %)	7 (22.6 %)	6 (19. 4%)	18 (58.1 %)	7 (22.6 %)	6 (19.4 %)	22 (71.0 %)	3 (9.7%)	5 (16.1 %)
≥9.0 %	9 (69.2 %)	4 (30.8 %)	4 (30. 8%)	4 (30.8 %)	5 (38.5%)	2 (15.4 %)	4 (30.8 %)	7 (53.8 %)	0 (0.0 %)
p-value	**p=0.775**		**p=0.577**			**p=0.0009***			**p=0. 182**

* Statistically significant (χ^2=18.8, p=0.0009)

Multivariable Logistic Regression — Predictors of Poor Functional Outcome

In multivariable logistic regression adjusting for age, sex, hypertension, dyslipidemia, and CAD, HbA1c ordinal (OR=22.48, 95% CI: 1.03–489.82; p=0.048) and NIHSS (OR=1.87, 95% CI: 1.08–3.26; p=0.027) independently predicted poor discharge outcome (mRS 3–6) (Table 8). CAD also reached significance (OR=114.98; p=0.020). The wide CI for HbA1c reflects the modest sample size, but the persistence of the association after adjustment is clinically meaningful.

Table 8. Multivariable logistic regression for poor functional outcome (mRS 3–6)

Predictor	OR	95% CI	p-value
HbA1c (ordinal)	22.48	1.03–489.82	0.048*
NIHSS	1.87	1.08–3.26	0.027*
Age (per 10 years)	1.02	0.30–3.47	0.975
Male sex	0.62	0.05–7.11	0.702
Hypertension	0.67	0.06–7.03	0.740
Dyslipidemia	13.26	0.78–226.28	0.074
CAD	114.98	2.12–6231.59	0.020*

DISCUSSION

This hospital-based cross-sectional study demonstrates three coherent signals: (i) a graded increase in stroke severity (NIHSS) with rising HbA1c; (ii) a markedly higher proportion of poor functional outcome (mRS 3–6) at discharge with poorer glycemic control; and (iii) a significantly longer hospital stay among patients with elevated HbA1c. These findings align with contemporary cohorts and meta-analyses linking chronic glycemic exposure to worse neurological deficits and recovery in diabetic ischemic stroke [1,2,3,5,6].

The stepwise increase in mean NIHSS (3.5 → 8.13 → 13.08; F=27.13, p<0.001) is consistent with hospital-based cohorts in which both chronic glycemia and admission hyperglycemia independently predicted initial neurological severity [1,7]. Mechanistically, chronic hyperglycemia impairs endothelial function, activates inflammatory cascades, accelerates atherosclerosis, and compromises cerebrovascular autoregulation—pathways that collectively amplify ischemic injury [2,7]. The dominance of large-artery atherosclerosis as the TOAST subtype corroborates HbA1c as a proxy for cumulative vascular injury.

The finding that 92.3% of patients with HbA1c ≥9.0% had poor discharge function (mRS 3–6) is striking. Multiple cohorts report HbA1c as an independent predictor of unfavourable 3-month outcome even after adjustment for NIHSS (3,5,8). The persistence of this signal in our logistic regression model confirms its prognostic value independent of baseline severity.

The significantly longer LOS in the ≥9.0% group (χ^2=18.8, p=0.0009) has direct resource implications in a resource-constrained tribal setting, arguing for protocolized inpatient glucose management bundles—early glucose control, complication surveillance, DVT prophylaxis, oral care, dysphagia screening—and timely rehabilitation. A trend toward delayed presentation (>24 hours) among poorly controlled patients echoes reports that comorbidity and social determinants delay stroke recognition [9,10], underscoring the need to couple stroke-awareness messaging with diabetes clinic visits.

The cohort's cardiometabolic profile—72% hypertension, 70% dyslipidemia—reinforces that HbA1c is a surrogate for diffuse vascular vulnerability rather than a narrow glycemic index [11,12]. The skew toward suboptimal control (88% with HbA1c ≥7.0%) mirrors patterns in other low-resource Indian settings and likely reflects barriers to medication adherence, dietary control, and follow-up.

Limitations include: cross-sectional design (no causal inference), single-centre recruitment, modest sample size (n=50) with wide CIs in the regression model, no 30- or 90-day follow-up outcomes, and potential HbA1c misclassification in patients with anaemia, haemoglobinopathies, or renal disease. Residual confounding from inflammatory markers, renal indices, and socioeconomic factors cannot be excluded.

CONCLUSION

HbA1c is independently and significantly associated with stroke severity at admission, poor functional outcome at discharge, and a more demanding in-hospital course in adult diabetic patients with acute ischemic stroke in a tribal-region hospital in Gujarat. The graded associations across HbA1c categories support embedding this routinely available biomarker into early risk stratification and clinical decision-making. Patients with HbA1c ≥9.0% warrant protocolized inpatient glucose management, heightened complication surveillance, and early rehabilitation planning. At the systems level, coupling stroke-awareness messaging with diabetes programs and strengthening pre-hospital referral pathways in tribal communities are practical steps to reduce treatment delays. Future prospective studies should validate findings with 90-day outcomes, incorporate continuous glucose metrics, and test structured glycemic-optimization bundles from admission through post-discharge follow-up.

DECLARATIONS

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Conflicts of interest: - The authors declare no conflicts of interest.
Ethics: - The protocol was reviewed and approved by the Institutional Ethics Committee of ZMCH, Dahod (IEC Approval No. ZMCH/IEC/036(11)-2024, dated 9th April 2024). Written informed consent was obtained from all participants or their legally authorized representatives. All data were de-identified and stored securely in accordance with the Declaration of Helsinki and ICMR guidelines.

REFERENCES

1. Kuo YW, Lee JD, Lee CP, Huang YC, Lee M. Association between initial in-hospital heart rate and glycemic control in patients with acute ischemic stroke and diabetes mellitus. BMC Endocr Disord. 2023;23(69):69. Available from: https://doi.org/10.1186/s12902-023-01325-2
2. Sacco RL, Ornello R, Foschi M. Prevention and treatment of ischemic and haemorrhagic stroke in people with diabetes mellitus: A focus on glucose control and comorbidities. Diabetologia. 2024;67(4):1192–205. Available from: https://doi.org/10.1007/s00125-024-06146-z
3. Shen Y, Shi L, Nauman E, Katzmarzyk P, Price-Haywood E, Bazzano A, et al. Association between hemoglobin A1c and stroke risk in patients with type 2 diabetes. J Stroke. 2020;22(1):87–98. Available from: https://doi.org/10.5853/jos.2019.01704
4. Kim JS, Lee G, Park KI, Oh SW. Comparative effect of glucose-lowering drugs for type 2 diabetes mellitus on stroke prevention: A systematic review and network meta-analysis. Diabetes Metab J. 2024;48:312–20. Available from: https://doi.org/10.4093/dmj.2022.0421
5. Jeong J, Park JK, Koh YH, Park JM, Bae HJ, Yun SM. Association of HbA1c with functional outcome by ischemic stroke subtypes and age. Front Neurol. 2023;14:1247693. Available from: https://www.frontiersin.org/articles/10.3389/fneur.2023.1247693
6. Singh KP, Singh R, Singh T, Kaur S, Mittal T, Goyal M, et al. Neutrophil lymphocyte ratio as a predictor of stroke severity in type 2 diabetes mellitus: A single-center study. Cureus. 2024;16(1):e51841.
7. Chen G, Ren J, Huang H, Shen J, Yang C, Hu J, et al. Admission random blood glucose, fasting blood glucose, stress hyperglycemia ratio, and functional outcomes in patients with acute ischemic stroke treated with intravenous thrombolysis. Front Aging Neurosci. 2022;14:782282.
8. Roquer J, Rodríguez-Campello A, Cuadrado-Godia E, Giralt-Steinhauer E, Jiménez-Conde J, Soriano C, et al. The role of HbA1c determination in detecting unknown glucose disturbances in ischemic stroke. PLoS ONE. 2014;9(12):e109960. Available from: https://doi.org/10.1371/journal.pone.0109960
9. Cho KH, Kwon SU, Lee JS, Yu S, Cho AH. Newly diagnosed diabetes has high risk for cardiovascular outcome in ischemic stroke patients. Sci Rep. 2021;11:12929. Available from: https://doi.org/10.1038/s41598-021-92349-y
10. Yap J, Anbalakan K, Tay WT, Ting D, Cheung CY, Sabanayagam C, et al. Impact of type 2 diabetes and microvascular complications on mortality and cardiovascular outcomes in a multiethnic Asian population. BMJ Open Diabetes Res Care. 2021;9:e001413. Available from: https://drc.bmj.com/content/9/7/e001413
11. Gofir A, Silalahi RANA, Dananjoyo K, Setyopranoto I. HbA1c level and its role on ischemic stroke patients with prediabetes. Rom J Neurol. 2023;22(2):131.
12. Nomani AZ, Nabi S, Ahmed S, Iqbal M, Rajput HM, Rao S. High HbA1c is associated with higher risk of ischaemic stroke in Pakistani population without diabetes. Stroke Vasc Neurol. 2016;1:e000018.