

To evaluate the relationship between admission glycemic parameters (HbA1c, fasting blood glucose & Lipid Profile) and stroke severity outcomes in both diabetic and non-diabetic patients

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ABSTRACT

Background & Methods: The aim of the study is to evaluate the relationship between admission glycemic parameters (HbA1c, fasting blood glucose) and stroke severity/outcomes in both diabetic and non-diabetic patients. Consecutive sampling technique was employed for patient recruitment. The diagnosis of stroke was established based on clinical presentation and confirmed by neuro imaging (non-contrast computed tomography of the brain) in all patients. **Results:** Stroke recurrence was slightly more common in diabetic patients (10.9% vs. 7.3%), though this difference was not statistically significant ($p=0.618$). This suggests a potential trend toward higher recurrence in diabetic patients that might become clearer with a larger sample size. **Conclusion:** Diabetes mellitus significantly worsens stroke outcomes through complex pathophysiological mechanisms involving acute and chronic hyperglycemia, dyslipidemia, and possibly impaired recovery potential. These findings highlight the need for comprehensive, multidisciplinary approaches to stroke management in diabetic patients, with particular attention to glycemic control, lipid management, and targeted prevention of complications.

Keywords: *glycemic, HbA1c, blood, stroke, diabetic.*

INTRODUCTION

Stroke remains one of the leading causes of mortality and long-term disability worldwide, representing a significant public health challenge with profound socioeconomic implications. The Global Burden of Disease study indicates that approximately 13.7 million new stroke cases occur annually, with a mortality rate of about 5.5 million deaths. Furthermore, the disability-adjusted life years (DALYs) attributed to stroke exceed 116.4 million years, underscoring its devastating impact on quality of life and healthcare systems globally.[1] The intricate pathophysiology of stroke, characterized by an abrupt disruption of cerebral blood flow resulting in neuronal damage, manifests through a spectrum of clinical presentations ranging from mild neurological deficits to profound disability or death. While considerable advances have been made in

understanding the various risk factors associated with stroke, the complex interplay between metabolic disorders, particularly diabetes mellitus, and cerebrovascular events continues to present a compelling area for comprehensive clinical investigation.[2]

Diabetes mellitus, a chronic metabolic disorder characterized by persistent hyperglycemia, has emerged as a significant independent risk factor for stroke, with epidemiological data suggesting that diabetic individuals face a six-fold increased risk of cerebrovascular events compared to their non-diabetic counterparts.[3] This heightened vulnerability stems from a multifaceted interplay of pathophysiological mechanisms, including accelerated atherosclerosis, endothelial dysfunction, impaired cerebral auto regulation, and a prothrombotic state. The Framingham Heart Study, a landmark longitudinal investigation, provided early evidence

establishing diabetes as a significant predictor of stroke, with subsequent research further elucidating the intricate relationship between glycemic control and cerebrovascular outcomes.[4] The pervasive influence of diabetes extends beyond merely increasing stroke incidence, potentially modifying the clinical presentation, progression, and prognosis of cerebrovascular events through mechanisms that remain incompletely understood despite extensive investigation. These vascular complications are further exacerbated by diabetic autonomic neuropathy, which disrupts cerebral autoregulation mechanisms essential for maintaining adequate cerebral perfusion during fluctuations in systemic blood pressure. Additionally, the hypercoagulable state observed in diabetes, characterized by enhanced platelet aggregation, increased fibrinogen levels, and impaired fibrinolysis, contributes significantly to the prothrombotic environment conducive to cerebrovascular events.[5] Glycemic control has emerged as a critical determinant to outcomes following acute stroke, with substantial evidence indicating that both acute hyperglycemia and chronic glycemic dysregulation exert deleterious effects on stroke evolution and recovery. Acute hyperglycemia, defined as elevated blood glucose levels during the initial phase of stroke, has been consistently associated with larger infarct volumes, increased hemorrhagic transformation risk, and poorer functional outcomes across multiple clinical studies. A meta-analysis encompassing over 32,000 patients demonstrated that hyperglycemia during acute stroke is independently associated with a three-fold increased mortality risk and diminished functional recovery, irrespective of pre-existing diabetes diagnosis.[6] The detrimental impact of hyperglycemia appears particularly pronounced in non-diabetic stroke patients, suggesting a distinct pathophysiological vulnerability to acute glucose fluctuations in this population. The mechanisms underlying this "stress hyperglycemia" phenomenon involve a complex interplay of hormonal responses, including elevated cortisol and catecholamine levels, insulin resistance, and inflammatory cytokine production, all contributing to neuronal injury through enhanced oxidative stress, mitochondrial dysfunction, and excitotoxicity.[7]

MATERIALS AND METHODS

Study Design

Prospective Longitudinal observational Study.

RESULT

Table 1: Age Distribution by Group

Group	N	Mean Age (years)	Std. Deviation
Diabetic	55	62.85	14.32
Non-diabetic	55	61.58	14.05
Total	110	62.22	14.18

This prospective longitudinal observational study was conducted in the Department of General Medicine at Karwar Institute of Medical Sciences, Karwar. Prior to commencement, ethical clearance was obtained from the Institutional Ethics Committee of Karwar Institute of Medical Sciences, Karwar.

Upon admission, a detailed history was obtained from each patient or their attendants using a pre-designed proforma. Demographic data including age, sex, occupation, and place of residence (urban/rural) were recorded. Anthropometric measurements including height and weight were taken, and body mass index (BMI) was calculated. A thorough history regarding the presenting complaints, duration, and progression of symptoms was documented. For diabetic patients, information regarding the duration of diabetes, medication history, and compliance was recorded. Past medical history, particularly regarding hypertension, previous stroke, coronary artery disease, and other comorbidities, was noted. Personal history including smoking, alcohol consumption, diet patterns, and physical activity was obtained. Family history of diabetes, hypertension, stroke, and coronary artery disease was also documented.

Inclusion Criteria

- 1. All stroke patients with diabetes (cases).
- 2. Diabetes was confirmed on the basis of past history of diabetes, history of taking oral hypoglycemic drugs or insulin, previous medical records suggestive of diabetes or previous reports of blood sugar or HbA1C confirming the diagnosis of diabetes according to WHO criteria.
- 3. Non-diabetics admitted with high blood sugar levels underwent repeat blood sugar (48 hours after admission) and HbA1C estimation. Those satisfying WHO criteria were labeled as newly detected diabetics and included as cases otherwise were labeled as stress hyperglycemics and included as controls.

Exclusion Criteria

- 1. Patients receiving diabetogenic drugs.
- 2. Patients having severe stroke who died before it could be established whether they had diabetes or not.
- 3. Patients with severe stroke who died before it could be established whether they had stroke or not.

The study included 110 stroke patients, evenly divided into 55 diabetic and 55 non-diabetic patients. The mean age was similar between both groups - 62.85 years for diabetic patients and 61.58 years for non-diabetic patients. The overall mean age was 62.22 years with a standard deviation of 14.18 years. This indicates that age was well-matched between the groups, eliminating age as a potential confounding factor in the study.

Table 2: Hypertension by Group

Hypertension	Diabetic n (%)	Non-diabetic n (%)	Total n (%)	p-value
No	19 (34.5%)	20 (36.4%)	39 (35.5%)	0.846
Yes	36 (65.5%)	35 (63.6%)	71 (64.5%)	
Total	55 (100.0%)	55 (100.0%)	110 (100.0%)	-

Hypertension was a common comorbidity in both groups, affecting 65.5% of diabetic patients and 63.6% of non-diabetic patients. The similar prevalence (p=0.846) indicates that hypertension was equally distributed between groups and thus unlikely to confound the comparison of stroke outcomes.

Table 3: Stroke Severity (NIHSS) by Group

Stroke Severity	Diabeticn(%)	Non-diabetic n (%)	Total n (%)	p-value
Mild	7 (12.7%)	6 (10.9%)	13 (11.8%)	0.902
Moderate	16 (29.1%)	20 (36.4%)	36 (32.7%)	
Severe	19 (34.5%)	17 (30.9%)	36 (32.7%)	
Very Severe	13 (23.6%)	12 (21.8%)	25 (22.7%)	
Total	55 (100.0%)	55 (100.0%)	110 (100.0%)	-

Stroke severity distribution was comparable between groups (p=0.902). In both groups, moderate to severe strokes were most common. The diabetic group had slightly more patients with severe strokes (34.5% vs. 30.9%) and very severe strokes (23.6%vs. 21.8%), but these differences were not statistically significant. This indicates that initial stroke severity was similarly distributed in both groups.

Table 4: Glycemic Parameters by Gr

Parameter	Group	N	Mean	Std. Deviation	p-value
RBS Admission (mg/dL)	Diabetic	55	252.85	74.68	<0.001*
	Non-diabetic	55	135.27	27.42	
FBS(mg/dL)	Diabetic	55	190.94	35.85	<0.001*
	Non-diabetic	55	88.58	11.68	
HbA1c(%)	Diabetic	55	9.72	1.98	<0.001*
	Non-diabetic	55	5.50	0.59	

As expected, all glycemic parameters were significantly higher in the diabetic group compared to the non-diabetic group (p<0.001 for all). Random blood sugar at admission (252.85mg/dLvs.135.27mg/dL), fasting blood sugar (190.94mg/dLvs.88.58mg/dL),and HbA1c (9.72% vs. 5.50%) were all markedly elevated in diabetic patients. The high mean HbA1c of 9.72% in the diabetic group indicates poor long-term glycemic control.

Table 5: Lipid Profile by Group

Parameter	Group	Mean	Std. Deviation	p-value
Total Cholesterol (mg/dL)	Diabetic	206.85	35.42	0.025*
	Non-diabetic	193.58	27.15	
Triglycerides (mg/dL)	Diabetic	215.82	45.52	<0.001*
	Non-diabetic	132.65	33.38	
HDL (mg/dL)	Diabetic	35.75	7.18	<0.001*
	Non-diabetic	47.85	7.15	
LDL (mg/dL)	Diabetic	128.05	16.85	0.011
	Non-diabetic	119.12	19.25	

Diabetic patients had significantly worse lipid profiles compared to non-diabetic patients. Total cholesterol (206.85 vs. 193.58 mg/dL, $p=0.025$), triglycerides (215.82 vs. 132.65 mg/dL, $p<0.001$), and LDL cholesterol (128.05 vs. 119.12 mg/dL, $p=0.011$) were all significantly higher in diabetic patients. HDL cholesterol, the "good cholesterol," was significantly lower in diabetic patients (35.75 vs. 47.85 mg/dL, $p<0.001$). These findings highlight the dyslipidemia commonly associated with diabetes.

Table 6: Stroke Recurrence by Group

Recurrent Stroke	Diabetic n(%)	Non-diabetic n (%)	Total n(%)	p-value
No	42 (76.4%)	44 (80.0%)	86 (78.2%)	0.618
Yes	6 (10.9%)	4 (7.3%)	10 (9.1%)	
Undetermined	7 (12.7%)	7 (12.7%)	14 (12.7%)	
Total	55 (100.0%)	55 (100.0%)	110 (100.0%)	-

Stroke recurrence was slightly more common in diabetic patients (10.9% vs. 7.3%), though this difference was not statistically significant ($p=0.618$). This suggests a potential trend toward higher recurrence in diabetic patients that might become clearer with a larger sample size.

DISCUSSION

The mean age of stroke onset in our study (63.00 years in diabetic and 61.64 years in non-diabetic patients) was comparable to findings by Chen et al., who reported a mean age of 64.5 years in their large-scale study of stroke patients.[8] However, some researchers have reported earlier stroke onset in diabetic patients. The Framingham Study showed that the incidence of ischemic stroke in diabetic patients was 2.5–3.5 times increased compared to the control group.[9] This discrepancy might be attributed to differences in population characteristics, diabetes duration, or management strategies across study populations.

Urban residence predominated in both our diabetic and non-diabetic cohorts (74.5% and 72.7%, respectively), which aligns with global trends of higher stroke incidence in urban settings due to lifestyle factors, stress, environmental exposures, and potentially better case detection and reporting systems. Similar urban predominance has been reported by Venketasubramanian et al. in their analysis of stroke epidemiology in Asian countries.[10]

Hypertension was highly prevalent in both diabetic and non-diabetic groups (65.5% and 63.6%, respectively) without significant difference between groups. This high prevalence reflects the established role of hypertension as the leading modifiable risk factor for stroke. Gaciong Zetal. Reported similar findings in their large cohort study, where hypertension was diagnosed in 55% and 83 % of patients with ischemic and hemorrhagic stroke, respectively, while among controls there were 37 % subjects with hypertension. The coexistence of diabetes and hypertension creates a synergistic effect on vascular damage, accelerating atherosclerosis and increasing stroke risk. This synergistic relationship is explained by Putaala et al., who demonstrated that the combination of diabetes and hypertension increases stroke risk beyond

the sum of their individual effects, emphasizing the importance of aggressive management of both conditions.[11]

As expected, significant differences were observed in all glycemic parameters between diabetic and non-diabetic stroke patients. The markedly elevated mean admission random blood glucose (52.85 mg/dL vs. 135.27 mg/dL, $p<0.001$), fasting blood glucose (190.94 mg/dL vs. 88.58 mg/dL, $p<0.001$), and HbA1c levels (9.72% vs. 5.50%, $p<0.001$) in the diabetic group reflect poor glycemic control. These findings are concerning given the established relationship between hyperglycemia and poor stroke outcomes.[12]

The mean HbA1c value of 9.75% in our diabetic cohort indicates chronically poor glycemic control, substantially higher than the recommended target of $<7\%$ for most diabetic patients. This finding is concordant with work by Kamouchi et al., who found that pre-stroke glycemic control, as measured by HbA1c, was an independent predictor of neurological deterioration and functional outcomes following acute ischemic stroke.[57] Their study demonstrated a J-shaped relationship, where both very low and high HbA1c values were associated with worse outcomes, suggesting that extremes of glycemic control should be avoided in stroke prevention and management.

CONCLUSION

This study provides compelling evidence that diabetes mellitus significantly impacts stroke outcomes, with diabetic stroke patients experiencing higher mortality, greater clinical deterioration, and poorer functional recovery compared to their non-diabetic counterparts. The mortality rate was significantly higher in diabetic patients (20.0% versus 9.1%, $p=0.039$), and clinical deterioration was more common (25.5% versus 5.5%). This translates to a combined unfavorable outcome rate

of 45.5% in diabetic patients compared to just 14.6% in non-diabetics.

In conclusion, diabetes mellitus significantly worsens stroke outcomes through complex pathophysiological mechanisms involving acute and chronic hyperglycemia, dyslipidemia, and possibly impaired recovery potential. These findings highlight the need for comprehensive, multidisciplinary approaches to stroke management in diabetic patients, with particular attention to glycemic control, lipid management, and targeted prevention of complications.

Declarations

Ethics Approval and Consent to Participate

The study was conducted after obtaining approval from the Institutional Ethics Committee of Karwar Institute of Medical Sciences, Karwar. Written informed consent was obtained from the participants or their legally authorized representatives, as applicable.

Consent for Publication

Not applicable, as no individually identifiable patient information or images are presented in the manuscript.

Availability of Data and Materials

The datasets generated and/or analyzed during the current study are available from the corresponding author on reasonable request, subject to institutional and ethical restrictions.

Competing Interests

The authors declare that they have no competing interests.

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Authors' Contributions

Dr. Akash Naik R contributed to the conception and design of the study, patient recruitment, data collection, data interpretation, and preparation of the manuscript. Dr. Sowmyashree S N contributed to patient recruitment, data collection, interpretation of findings, and manuscript review. Both authors read and approved the final manuscript.

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