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Acute Changes in Random Blood Glucose Levels Following Atorvastatin Therapy in Ischemic Stroke Patients

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Abstract

Background: *Stroke involves a rapid onset of neurological deficits that last for 24 hours or more or lead to death. While Atorvastatin is highly effective for secondary prevention in ischemic stroke patients, it may cause a diabetogenic side effect that can raise blood glucose levels.* ***Objective:*** *This study aims to assess the immediate changes in Random Blood Glucose (RBG) levels after starting Atorvastatin therapy in hospitalized ischemic stroke patients with a history of diabetes mellitus and hypertension. Methods: This was a retrospective, descriptive cohort study using purposive sampling. The sample included adult inpatients diagnosed with ischemic stroke, known diabetes mellitus, and hypertension who were prescribed Atorvastatin at Bhayangkara Hospital from January to June 2024. Patients with incomplete medical records were excluded. RBG levels were monitored over a short-term period of 3 days. Data were analyzed using descriptive statistics and a paired t-test to compare glucose levels before and after therapy.* ***Results:*** *Of the 28 eligible patients, most were male (16; 57.1%) and aged 55–64 years (15; 53.6%). The most common profile included ischemic stroke, diabetes mellitus, hypertension, and dyslipidemia (8 patients; 28.6%). The paired t-test showed no statistically significant difference between baseline and 3-day post-treatment RBG levels ($p = 0.993$; $p > 0.05$).* ***Conclusion:*** *In this small cohort, short-term (3 days) Atorvastatin therapy did not significantly increase RBG levels in ischemic stroke patients with diabetes mellitus. However, due to the small sample size (n=28) and short observation period, these results cannot be generalized, as long-term effects of statins on blood glucose usually take longer to manifest.*

Keywords: *Atorvastatin; Blood Glucose; Diabetes Mellitus; Ischemic Stroke; Patient Safety*

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INTRODUCTION

Ischemic stroke triggers an acute neuroinflammatory and metabolic crisis, necessitating immediate secondary prevention strategies. Current guidelines from the American Heart Association and American Stroke Association (AHA/ASA) mandate early, high-intensity statin therapy—primarily Atorvastatin (40–80 mg)—owing to its potent low-density lipoprotein cholesterol (LDL-C) lowering capabilities and pleiotropic effects, including plaque stabilization and endothelial repair [1, 2]. Despite these established cardiovascular benefits, intensive statin therapy is increasingly recognized for its potential to cause diabetes. Statins impair glucose homeostasis through multi-pathway mechanisms: they downregulate glucose transporter 4 (GLUT4) expression in peripheral tissues, leading to insulin resistance, and inhibit pancreatic beta-cell function by disrupting voltage-gated calcium channels and decreasing intracellular ATP production [3].

Patients are particularly vulnerable to Stress-Induced Hyperglycemia (SIH), an abrupt, brief metabolic spike caused by cortisol and adrenaline counter-regulatory mechanisms after brain ischemia, during the first 72 hours of an ischemic stroke [4]. Clinically, it is crucial to maintain stable blood glucose throughout this initial window. Uncontrolled glycemic fluctuations or severe hyperglycemia can worsen overall clinical outcomes, exacerbate tissue damage, and directly increase the size of infarcts [5]. Although atorvastatin is an essential component of acute neuroprotection, little is known about its immediate, concurrent effects on blood glucose dynamics during this high-stress period, creating a crucial blind spot for thorough patient safety management.

While the long-term diabetogenic effects of statins over months or years are well-documented in landmark clinical trials, literature regarding the acute, short-term impact of statin initiation during the hyperacute phase of stroke remains conflicting. In an inpatient setting, the clinical picture is highly confounded by stress-induced hyperglycemia, an acute metabolic surge driven by cortisol and epinephrine counter-regulatory mechanisms following cerebral ischemia. How the immediate introduction of high-intensity Atorvastatin interacts with this acute stress-hyperglycemia phase, especially in patients with preexisting metabolic vulnerabilities like diabetes mellitus, is not fully clarified in current clinical literature.

Furthermore, post-stroke glycaemic management in regional clinical settings often operates under strict empirical constraints. While continuous glucose monitoring or long-term HbA1c tracking represents the gold standard for assessing true glycaemic variability, in resource-constrained or secondary care settings, real-world clinical practice frequently relies on serial Random Blood Glucose (RBG) monitoring during the initial 3-day acute stabilization window.

Therefore, this study aims to describe the clinical profile and assess blood glucose fluctuations during the first three days of Atorvastatin therapy in ischemic stroke patients. By explicitly focusing on acute-phase stability and immediate glycemic changes during the critical 72-hour post-stroke period, this study provides localized clinical insights into the safety of metabolic management during acute stroke management at Bhayangkara Hospital, Kediri.

MATERIALS AND METHODS

Materials and Tools

This study employed a retrospective, pragmatic research design. Data were collected from medical records from January to June 2024, while data gathering occurred from January to June 2025. A purposive sampling technique based on specific eligibility criteria was used. The inclusion criteria were carefully defined to capture immediate metabolic responses under real-world clinical constraints. This study included adult patients aged 15 years and older admitted with an acute episode of focal central nervous system dysfunction. Eligible participants had a confirmed diagnosis of acute ischemic stroke, a documented history of type 2 diabetes mellitus, and a concurrent prescription for high-intensity Atorvastatin therapy (40–80 mg) started within 24 hours of hospital admission. Additionally, included patients must have undergone serial Random Blood Glucose (RBG) monitoring during hospitalization, with complete medical and laboratory records containing at least 2 RBG exams within the first 72 hours after admission, specifically from the date of admission up to 3 days later during statin therapy. Since concurrent glucose-lowering therapies can influence RBG levels, blood glucose management was strictly controlled to avoid confounding factors. Patients on insulin or Oral Antidiabetic Drugs (OADs) were required to maintain a stable, unchanged dosage throughout the 3-day observation period (Day 1 to Day 3). To prevent fluctuations in RBG levels caused by changing antidiabetic regimens, any cases involving titration, major dosage adjustments, or medication switching during these 72 hours were systematically excluded. The primary tools and data sources included inpatient electronic medical records from Bhayangkara Hospital, Kediri. A total of 28 patients met these specific eligibility criteria during the baseline period from January to June 2024.

Method

This study retrospectively analysed short-term alterations in blood glucose levels during the hyperacute phase of stroke among hospitalized patients with comorbid diabetes mellitus at Bhayangkara Hospital in Kediri. To assess immediate glycaemic fluctuations while accounting for the clinical reality of regional care settings, patients' laboratory data were reviewed at two distinct time points during the critical 72-hour stabilization window: baseline (upon initial hospital admission, reflecting the combined baseline metabolic state and potential stress-induced hyperglycemia) and on day three (approximately 72 hours post-admission) under ongoing Atorvastatin therapy. This short-term observation window was intentionally chosen to evaluate immediate glycaemic stability before chronic confounding factors or extended out-of-hospital medication adjustments occurred.

Ethical Considerations

This study protocol was officially approved by the Ethics Committee of the Bhakti Wiyata Kediri Institute of Health Sciences under the research permit letter number 084/FFPB.4.5/II/2025. Bhayangkara Hospital in Kediri City formally granted institutional administrative permission and access to the medical records database under the decree number Sket/275/V/LIT.2.1/2025.

Data Analysis

Before hypothesis testing, the distribution of the numerical data was evaluated using the Shapiro-Wilk normality test, which is mathematically optimized for sample sizes below 50 (n=28). A p-value greater than 0.05 indicated a normally distributed dataset, justifying parametric testing. To evaluate the acute impact of early Atorvastatin initiation on immediate glycemic stability, a bivariate analysis was performed. A paired t-test was used to compare baseline and day-three RBG levels within the same individuals. This paired design allows each patient to serve as their own control, partially mitigating unmeasured baseline confounders. All statistical analyses were conducted with a significance threshold set at an alpha value (alpha) of 0.05. A p-value less than 0.05 was required to reject the null hypothesis and indicate a statistically significant acute glycemic shift.

RESULTS AND DISCUSSION

Profile Data

Gender

The demographic profile of the 28 monitored patients is presented in Tables 1 and 2, which show the gender and age distributions. Our cohort demonstrated a higher prevalence of ischemic stroke among male patients (n = 16; 57.1%) than among female patients (n = 12; 42.9%) (Table 1).

Table 1. Percentage of Patients by Gender

Gender	Patient Count	Percentage (%)
Male	16	57
Female	12	43
Number	28	100

The male predominance observed in this study (57.1%) aligns with well-established epidemiological patterns in cerebrovascular disease. Global data from the World Health Organization (WHO) and regional metrics from the Indonesian Health Survey (SKI) consistently demonstrate that males carry a disproportionately higher incidence of ischemic stroke during early and middle-to-late adulthood. [9]. A combination of biological and behavioral components drives this trend. Behaviorally, males exhibit a higher prevalence of modifiable, high-impact cardiovascular risk factors, most notably chronic tobacco smoke exposure, which directly induces endothelial injury. Biologically, the earlier onset of atherosclerotic plaque accumulation in males is linked to the absence of endogenous estrogen's vasoprotective effects. Estrogen plays a critical role in females by modulating lipid metabolism, enhancing nitric oxide production, and maintaining arterial elasticity. Consequently, the lack of this hormonal shield accelerates vascular remodeling and premature stenosis in males of similar age [10].

Age

According to the study, the majority of the study population was concentrated within the 55–64 age range, accounting for 15 patients (53.6%). The remaining distribution across other age cohorts was relatively sparse, with the Age 74 group representing the second-largest at 10.7% (n = 3) (Table 2).

The high concentration of stroke occurrences within the 55–64 age group (53.6%) underscores age as a powerful, non-modifiable driver of cerebrovascular events. This clinical distribution closely mirrors national findings from the Indonesian Health Survey (SKI). It aligns with the biological paradigm established by the American Heart Association (AHA), which notes that the probability of suffering an ischemic stroke doubles every decade after age 55 [9].

Table 2. Distribution of Patients Based on Age

Age Range (Years)	Patient Count	Percentage (%)
35-44	2	7
45-54	7	25
55-64	15	54
≥ 74	3	11
65-74	1	4
Total Number	**28**	**100**

The underlying mechanisms are rooted in progressive macrovascular and microvascular degeneration. As individuals enter their sixth decade of life, sustained hemodynamic stress triggers a structural shift characterized by endothelial cell senescence, collagen cross-linking, and a marked reduction in cerebral arterial elasticity. When this age-related stiffening intersects with a lifetime of lipid accumulation within the tunica intima, plaque formation accelerates. This progressive vascular stenosis reduces baseline cerebral blood flow and severely impairs cerebral autoregulation, making the aging brain highly vulnerable to acute ischemic occlusion when systemic hemodynamics fluctuate [10].

Disease History

Based on the patient's medical history (Table 3), the sample in this study included 28 patients. The most common medical histories were diabetes mellitus and hypertension (14 patients, 50%). The most striking finding in our clinical profile is that 100% of the patient cohort had a history of diabetes mellitus, with 50% presenting with the specific lethal combination of diabetes mellitus and hypertension. The synergistic coexistence of these two pathologies creates a profound metabolic and mechanical burden that rapidly destroys vascular integrity.

Chronic hyperglycemia promotes the irreversible formation of Advanced Glycation End-products (AGEs), which cross-link matrix proteins and stiffen vessel walls. Simultaneously, excess intracellular glucose stimulates overproduction of reactive oxygen species (ROS), causing severe oxidative stress that disrupts endothelial nitric oxide synthase (eNOS) function. When this metabolic dysfunction is superimposed on the chronic mechanical shearing forces of hypertension, endothelial damage is compounded. Hypertension causes adaptive but pathological hypertrophy of the arterial smooth muscle layer, narrowing the vessel lumen.

Table 3. Clinical Profiles of Comorbidity History in Ischemic Stroke Patients

Comorbidity Combination	n	(%)
Diabetes Mellitus and Hypertension	14	50.0
Diabetes Mellitus only	3	10.7
Heart Disease, Hypertension, and Diabetes Mellitus	3	10.7
Hypertension, Diabetes Mellitus, and Recurrent Stroke	3	10.7
Asthma, Hypertension, and Diabetes Mellitus	1	3.6
Hypertension, Diabetes Mellitus, and Vertigo	1	3.6
Heart Disease, Diabetes Mellitus, and Recurrent Stroke	1	3.6
Total	28	100.0

Together, diabetes-induced plaque instability and hypertension-induced hemodynamic stress render cerebral blood vessels highly susceptible to thrombosis. This comprehensive vascular vulnerability accounts for the high baseline rates of elevated Random Blood Glucose (RBG) observed in our study and explains why this multi-morbid population requires immediate guideline-directed therapies, such as Atorvastatin 20 mg, to protect the remaining cerebral tissue during acute ischemic stroke.

Accompanying Diagnosis

Beyond the primary diagnosis of acute ischemic stroke, all 28 patients had complex, compounding cardiovascular and metabolic comorbidities. The distribution of these comorbidities is detailed in Table 4. The most prevalent clinical triad was the coexistence of Hypertension, Diabetes Mellitus, and Dyslipidemia, observed in 8 patients (28.6%). Across all overlapping subcategories, combining patients diagnosed with either dyslipidemia or hypercholesterolemia yielded a total of 16 patients (57.1%) with baseline lipid abnormalities alongside their metabolic disorders.

Table 4. Profile of Secondary Diagnoses in Ischemic Stroke Patients

Secondary Diagnosis Profile	**n**	**(%)**
Hypertension, Diabetes Mellitus, and Dyslipidemia	8	28.6
Hypertension, Diabetes Mellitus, and Hypercholesterolemia	3	10.7
Diabetes Mellitus and Dyslipidemia	2	7.1
Hypertension, Diabetes Mellitus, Vertigo, and Dyslipidemia	1	3.6
Hypertension, Diabetes Mellitus, Vertigo, and Hypercholesterolemia	1	3.6
Coronary Artery Disease, Dyslipidemia, and Diabetes Mellitus	1	3.6
Others (Single morbidity or other combinations)	12	42.8
Total	**28**	**100.0**

The high prevalence of the metabolic triad (hypertension, diabetes mellitus, and dyslipidemia) in our acute stroke cohort (28.6% in the primary sub-cluster and over 57% with lipid disorders overall) reflects extreme macrovascular vulnerability. The coexistence of these three chronic pathologies does not merely add to cerebrovascular risk; it compounds it through a shared, aggressive, destructive pathway.

Diabetes mellitus alters lipid metabolism, driving an overproduction of small, dense low-density lipoprotein (sdLDL) particles. These smaller, denser LDL particles easily penetrate the compromised endothelial lining, where they undergo rapid oxidation and trigger an inflammatory influx of macrophages. Simultaneously, the elevated mechanical shearing forces generated by sustained hypertension damage the vascular endothelium, disrupting its natural ability to produce vasodilatory nitric oxide[14].

When dyslipidemia provides the lipid substrate, and hypertension delivers the mechanical wall stress, the progression toward advanced atherosclerosis accelerates. This process results in the formation of highly unstable, lipid-rich plaques within the cerebral arteries. Under acute circulatory pressure, these vulnerable plaques are highly susceptible to rupture and subsequent thrombosis, which explains the high baseline rate of acute ischemic stroke in this multi-morbid population [15].

Diabetes Mellitus Therapy Obtained

To manage baseline type 2 diabetes mellitus during the acute stroke hospitalization phase, patients received a wide range of antidiabetic regimens, categorized into monotherapy and polytherapy (Table 5). Monotherapy was prescribed to 15 patients (53.6%), primarily using oral Metformin 500 mg (n = 5; 17.9%) and various individual insulin formulations (Actrapid, Humalog-R, Apidra, Sansulin Log-G; n = 9; 32.1%). In contrast, 13 patients (46.4%) received polytherapy, which included complex combinations of oral hypoglycemic agents (OHAs), insulin regimens, and alpha-glucosidase inhibitors (Acarbose).

The wide variation in antidiabetic management observed in this study, ranging from single-drug oral therapies like Metformin to highly intensive multi-drug insulin regimens, introduces an important methodological caveat when interpreting glycemic stability. While our bivariate analysis showed no statistically significant change in mean RBG levels after starting Atorvastatin 20 mg (p=0.993), this apparent stability should be interpreted cautiously because it does not account for concurrent antidiabetic interventions.

Patients in this group were actively taking different pharmacodynamic agents that could influence daily blood glucose levels. For instance, 17.9% of the group was on Metformin 500 mg monotherapy, which lowers hepatic gluconeogenesis and improves peripheral insulin sensitivity without risking acute hypoglycemia [16]. However, a large part of the group was treated with intensive insulin therapy (such as short-acting Actrapid, Humalog-R, and Apidra) or secretagogues like Glimepiride and Glibenclamide [17]. These treatments actively reduce blood glucose levels and can easily conceal mild blood sugar increases caused by acute atorvastatin administration [18].

Table 5. Antidiabetic Therapy Regimens by Route of Administration

Category	Therapeutic Regimen	n	(%)
Monotherapy		15	53.57
Oral	Metformin 500 mg	5	17.86
	Gliclazide 80 mg	1	3.57
Injection	Regular Insulin	6	21.43
	Insulin Glulisine	2	7.14
	Insulin Glargine	1	3.57
Polytherapy		13	46.43
Oral only	Metformin 500 mg + Glimepiride 2 mg	3	10.71
	Metformin 500 mg + Sitagliptin 100 mg	1	3.57
	Pioglitazone 30 mg + Glibenclamide 5 mg + Acarbose 50 mg	1	3.57
Oral + Injection	Metformin 500 mg + Regular Insulin + Acarbose (50/100 mg)	2	7.14
	Metformin 500 mg + Regular Insulin	1	3.57
	Metformin 500 mg + Regular Insulin + Glibenclamide 5 mg + Acarbose 50 mg	1	3.57
	Metformin 500 mg + Regular Insulin + Glimepiride 2 mg	1	3.57
	Glimepiride 2 mg + Regular Insulin	1	3.57
	Pioglitazone 30 mg + Insulin Glargine	1	3.57
	Pioglitazone 30 mg + Sitagliptin 100 mg + Insulin Glargine	1	3.57
Total		28	100.00

The lack of a standardized glucose management protocol and the small sample size, which prevented multivariate regression, mean that active changes in antidiabetic therapy are an important unmeasured confounder. As a result, it is unclear whether the increase in the 'High' RGB category from 57% to 71% is directly caused by statins' acute diabetogenic effects or if it reflects a compensated state due to personalized treatment adjustments. This limitation underscores the need for larger, carefully controlled prospective trials to clearly distinguish the metabolic effects of statins from those of other glucose-lowering treatments.

Dyslipidemia Therapy Received

Atorvastatin 20 mg was the most prevalent regimen, prescribed to 25 patients (89.3%). Atorvastatin Intensity is initiated during the acute phase of ischemic stroke. High-intensity statin therapy (Atorvastatin 40 mg) was used in only 2 patients (7.1%), while low-intensity therapy (Atorvastatin 10 mg) was used in 1 patient (3.6%). In this cohort, the overwhelming clinical preference for Atorvastatin 20 mg (89.3%) reflects adherence to guideline-directed medical therapy for secondary stroke prevention. The current international consensus, including the American Heart Association (AHA) and American Stroke Association (ASA) guidelines, recommends immediate initiation of lipid-lowering therapy following an ischemic cerebrovascular event to mitigate recurrent risk [19,20].

While landmark secondary prevention trials like Stroke Prevention by Aggressive Reduction (SPARCL) used intensive, high-dose regimens (Atorvastatin 80 mg), clinical practice often transitions to moderate-intensity statins—such as Atorvastatin 20 mg—in Asian populations or patients presenting with complex multimorbidities (hypertension and diabetes) to balance efficacy with metabolic tolerance [21]. Although the broader literature frequently underscores the cardiovascular and cerebrovascular risk reduction achieved through statin-induced plaque stabilization and endothelial repair, the current study did not measure long-term cardiovascular outcomes, recurrent stroke rates, or survival metrics**.** The observed benefit is strictly confined to surrogate adherence to established clinical guidelines during the hyper-acute phase [22].

Blood Glucose Level Profile

Random Blood Glucose (RBG) levels were evaluated at baseline (Pre-test, Day 1) and following the acute intervention (Post-test, Day 3). Descriptively, a nominal upward shift was observed in the high RBG category (200 mg/dL), increasing from 16 patients (57.1%) at baseline to 20 patients (71.4%) post-test. Conversely, the normal RBG subgroup decreased from 14.3% to 7.1% (Figure 1).

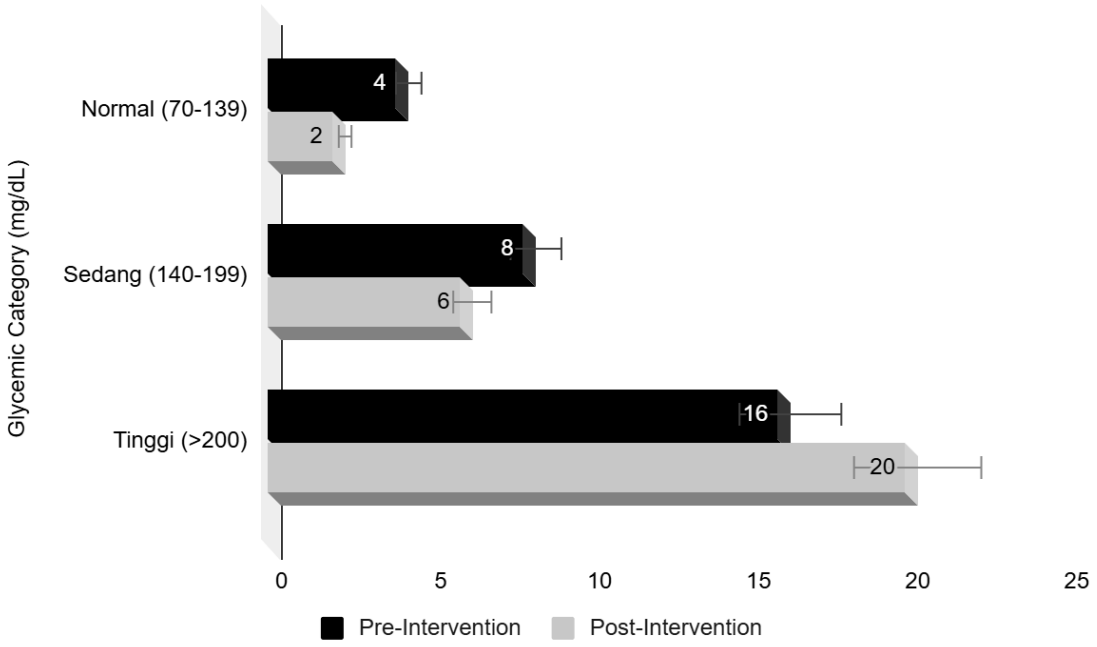

Figure 1. Comparison of RBG Category Distributions Before and After the Intervention

This study assessed the immediate impact of Atorvastatin 20 mg on RBG levels over 3 days. Descriptively, our results indicated a slight rise in the percentage of patients classified as High RBG (>200 mg/dL), increasing from 57% at baseline to 71% after the intervention (Table 6). However, an inferential analysis using a paired-samples t-test showed that this observed increase was not statistically significant ($p = 0.993$).

This statistical invariance is a critical finding. Recent clinical updates emphasize that statistical non-significance must be interpreted as a true reflection of stability rather than a structural failure of the drug [23]. It indicates that despite the raw percentage shifts, acute, short-term exposure to Atorvastatin does not trigger volatile glycemic fluctuations or acute metabolic decompensation [24]. Crucially, this stability was maintained even within our highly vulnerable study population, where 50% of the subjects had pre-existing type 2 diabetes mellitus (T2DM) complicated by comorbidities such as hypertension and acute ischemic stroke [18].

The lack of statistical significance in this study underscores a disconnect between acute clinical observations and long-term molecular frameworks. Current literature still reports the "diabetogenic effect" of statins, citing mechanisms such as downregulation of glucose transporter 4 (GLUT4) in peripheral tissues, disruption of the insulin signaling pathway, and inhibition of L-type calcium channels in pancreatic beta-cells. However, recent longitudinal cohort studies have confirmed that these molecular disruptions are usually cumulative and often take months to years of continuous exposure to cause meaningful glycemic impairment.

Given our study's short duration of only 3 days, it is unlikely that the nominal 14.3% increase in the High RBG bracket is attributable to statin-induced diabetogenesis. Instead, the observed statistical consistency ($p = 0.993$) confirms that starting Atorvastatin does not cause immediate, acute harm to glucose homeostasis, reassuring clinicians of its metabolic safety during the hyperacute phase of stroke management.

The molecular mechanisms behind statin-induced disruptions in blood sugar levels—often marked by reduced glucose transporter 4 (GLUT4) expression in fat and muscle tissue, impairment of the insulin signaling pathway inside cells, and blocking L-type calcium channels in pancreatic beta-cells—have raised concerns about the metabolic safety of these drugs [14]. In our descriptive data, the percentage of patients with high RBG (200 mg/dL) increased by 14.3% after the intervention.

However, aside from basic descriptive percentages, the paired-samples t-test revealed no significant difference ($p = 0.993$). This suggests that short-term exposure to Atorvastatin 20 mg does not lead to drastic blood sugar fluctuations or immediate metabolic problems, even in a high-risk group where half have pre-existing type 2 diabetes.

The lack of statistical significance in this study should not be seen as an anomaly needing justification, but rather as a reflection of the time-dependent nature of statin-induced metabolic changes. Hypotheses that link statin therapy to significant clinical hyperglycemia are based on chronic, cumulative exposure. Long-term clinical observations show that measurable changes in HbA1c or fasting plasma glucose usually require a latent period of more than 3 to 6 months of continuous drug therapy before they become evident. Consequently, our 3-day data reflect a narrow window of hyper-acute glycaemic stability, which cannot—and should not—be used to predict long-term diabetic risks.

Bivariate Analysis

To assess whether the descriptive shifts in RBG categories reflected statistically significant glycemic variation, a paired-samples t-test was conducted on continuous blood glucose data from Day 1 to Day 3. The bivariate analysis yielded a two-tailed p-value of 0.993, a mean difference of -2.14 mg/dL, and a t-value of -0.009. Because $p > 0.05$, the null hypothesis cannot be rejected. There is no statistically significant difference between baseline and post-statin intervention RBG levels within this acute 3-day observational window.

Due to the constraints of the baseline sample size (N = 28), advanced statistical modeling—including subgroup stratification (e.g., separating patients with and without a prior history of diabetes) and multivariate regression analysis to control for potential confounders (such as age, concurrent antihypertensive/antidiabetic regimens, or stroke severity) could not be reliably executed.

CONCLUSION AND SUGGESTION

Most ischemic stroke patients at Bhayangkara Hospital in Kediri are older men with a high burden of cardiovascular risk factors, largely driven by multimorbidity, particularly the combination of hypertension and diabetes mellitus. Inferential analysis shows that initiating Atorvastatin 20 mg early does not significantly change blood glucose levels during the first 72 hours after ischemic stroke. This is supported by glycemic correlation analysis, which finds no significant difference between baseline random blood glucose and day 3 levels after treatment ($p=0.993$). These findings suggest that administering 20 mg of Atorvastatin in the acute stroke phase does not cause clinically significant short-term blood glucose fluctuations and can be safely given immediately upon admission without affecting short-term glycemic control. These results provide reassurance that early neuroprotective lipid-lowering therapy does not compromise immediate metabolic stability. However, these results are preliminary, as the potential diabetogenic effects of statins are not evident in this short-term observation. Additionally, because cardiovascular outcomes were not assessed, no conclusions can be drawn about improvements in long-term cardiovascular risk. Larger, prospective studies with longer follow-up are necessary to confirm these initial metabolic findings.

Hospitals should continue prescribing Atorvastatin as the standard neuroprotective therapy for stroke patients. Patients with diabetes should undergo long-term glycemic monitoring, with HbA1c testing every 3 months. Future research should follow patients for 3–6 months to assess the risk of latent statin-induced diabetes.

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CONFLICT OF INTEREST

The authors declare that they have no conflicts of interest.

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