ORIGINAL ARTICLE

Factors Related to Early Neurological Deterioration in Lacunar Stroke and Its Influence on Functional Outcome

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Abstract

Background: ***Up to 20 percent of cerebral infarcts are caused by lacunar infarction. Although patients with lacunar infarction often have a good prognosis, there is a substantial chance of early neurological degeneration(END), which can have a negative impact on their recovery.***

Aim and objectives: ***Identifying risk variables for end-stage neuropathy (END) and its impact on functional outcome in patients with acute lacunar stroke.***

Subjects and methods: ***From May 2024 through November 2024, fifty patients hospitalized to the emergency department and stroke unit at Al-Azhar University hospitals (Al-Hussien and Bab El-Shaeria) with symptoms of acute ischemic stroke were participating in this prospective study.***

Results: ***Metabolic risk factors were prominent, with HbA1c at 7.5(SD=2.8), triglycerides 165.4 mg/dL(SD=38.0), and LDL cholesterol 167.5 mg/dL(SD=36.5), underscoring the role of dyslipidemia and poor glycemic control in stroke pathophysiology. Neurological severity was moderate at baseline(NIHSS=6.4, SD=2.5) with some deterioration(mean change=1.4, SD=1.2). However, functional outcomes improved, with mRS decreasing from 2.0 at discharge to 1.0 at three months, reflecting recovery with proper management.***

Conclusion: ***Elevated HbA1c and LDL cholesterol levels were strongly associated with deterioration, emphasizing the role of metabolic control in stroke progression. The baseline NIHSS score emerged as the most significant predictor of deterioration, underscoring the importance of initial stroke severity assessment. The logistic regression and discriminant function models demonstrate high predictive accuracy, reinforcing their utility in clinical decision-making.***

Keywords: Lacunar stroke; Neurological deterioration; Risk factors

1. Introduction

Twenty percent or more of all cerebral infarctions are caused by lacunar infarction. The prognosis is usually good, although there is a high chance of early neurological degeneration (END) in individuals with lacunar infarction, which could result in a bad end. Little is known about the causes or methods for preventing end-stage neuropathy (END) in people with lacunar stroke.[1]

There is a great deal of variation and uncertainty regarding the mechanisms of lacunar infarction. Lacunar infarction is unique in that it does not have a known cause, unlike strokes caused by cardioembolism or major artery steno-occlusion. Autopsy results have established lipohyalinosis as a key hypertension-related pathogenesis of lacunar stroke. Results from autopsies have revealed the presence of microatheroma, atherosclerosis, arteriosclerosis, and arteriolosclerosis. Potentially contributing to lacunar stroke is branch atherosclerotic disease (BAD).[2]

Penetrating artery lipohyalinosis and small vessel spasm due to hypertension can explain the mechanism by which premorbid hypertension is directly connected with the onset of lacunar infarction, according to the previous study. Curiously, this process can also account for END, and there have been reports of hypertension as a predictor of END, albeit the findings are contentious.[3]

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Multiple studies have shown conflicting results on the effects of blood pressure reduction on endothelium-induced neuropathy (END). Nevertheless, the types of strokes were not categorized in these research. While one study found that induced hypertension improved motor performance after lacunar infarction, another found that END was associated with increased blood pressure.[4]

Finding out what causes end-stage neuropathy (END) and how it affects functional outcome in individuals with acute lacunar stroke was the primary goal of this research.

2. Patients and methods

From May 2024 through November 2024, fifty patients hospitalized in the emergency department and stroke unit of Al-Azhar University hospitals (Al-Hussien, Bab El-Shaeria) with symptoms of acute ischemic stroke were selected for this prospective study.

Inclusion criteria:

Age 18 years or older, with first clinically and radiologically evident lacunar infarctions.

Exclusion criteria:

Individuals who have experienced a prior ischemic stroke, TBI, intracranial bleeding, cranial interventions/surgery, neuroinflammatory illness, or traumatic brain injury.

There were two groups of patients. The case group consisted of 25 patients who had suffered a stroke and had END as well as lacunar infarctions. An acute subcortical minor ischemic lesion (<20mm) on magnetic resonance imaging (MRI) inside the region of penetrating arteries without evidence of corresponding major artery occlusion was deemed a lacunar stroke. The control group, consisting of 25 stroke patients, included those with lacunar infarctions but no end-stage cerebrovascular disease (END).

Data collection:

All patients were subjected to the following:

Patients' age, gender, and comorbidities were taken into account when compiling this demographic data. Hypertension, diabetes mellitus, cigarette smoking, alcohol consumption, coronary heart disease, and atrial fibrillation were also documented. Medications administered while hospitalized included intravenous thrombolysis, antiplatelet (dual or single antiplatelet), anticoagulant, and antihypertensive medication. A complete neurological evaluation was performed upon discharge. The results of the modified Rankin Scale (mRS) were evaluated upon admission, at 24 hours, and after three months of follow-up.

Clinical assessment:

On admission:

Random blood glucose; blood pressure measurement; time from onset to hospital admission; National Institute of Health Stroke Scale Score(NIHSS) at admission to assess stroke severity was assessed at admission, after 24 hours and after 3 months of follow-up.

Laboratory assessment:

Complete blood count, erythrocyte sedimentation rate, CRP, UA, hepatic, renal function tests, serum cholesterol, triglycerides, low-density lipoprotein, fasting, random blood glucose level, and the haemoglobin A1c(HbA1c) test.

Outcome measurement:

A persistent increase in the NIHSS score of two points or more within the first twenty-four hours of admission was deemed the main endpoint, or END. Good functional result, as defined as an mRS of 0-2 at discharge, was the secondary objective.

Ethical considerations:

The research followed all of the guidelines established by the International Declaration of Helsinki and its subsequent revisions with regard to research ethics. We got the go-ahead from the Al-Azhar Faculty of Medicine's Ethics Committee. Prior to participating in the study, participants or their guardians were asked to provide their informed consent.

Statistical analysis:

Version 20.0 of the IBM Statistical program for the Social Sciences (SPSS) software program was used for data analysis. Quantitative data was represented by percentages and numbers. The distribution was checked for normalcy using the Shapiro-Wilk test. Standard deviation, median, interquartile range (IQR), range (minimum and maximum), and mean were used to characterize quantitative data. The acquired results were considered statistically significant at the 5% level.

To compare distinct groups, a chi-square test is used for categorical data. A student's t-test is used to compare two groups when the quantitative variables are normally distributed. The Mann-Whitney test is used to compare two groups when the quantitative variables are not normally distributed. To compare more than two groups, the Kruskal-Wallis test is used for quantitative variables that do not follow a normal distribution.

3. Results

Table 1.Baseline data of the studied lacunar stroke patients.

	NUMBER	%
MALES	20	40.0
FEMALES	30	60.0
DM	20	40
HTN	19	38
AF	5	10
IHD	7	14
SMOKER	10	20
INCREASED SBP/DBP	12	24
HYPERGLYCEMIA≥200MG/DL	16	32
HGA1C≥10%	13	26
SINGLE ANTIPLATELET THERAPY	26	52
DUAL ANTIPLATELET THERAPY	24	48
ANTIHYPERTENSIVE	17	34

The baseline characteristics of the studied lacunar stroke patients reveal a slightly higher prevalence of females(60.0%) compared to males(40.0%). A significant proportion of the patients had comorbid conditions, with diabetes mellitus(DM) present in 40% and hypertension(HTN) in 38% of the cohort. Notably, atrial fibrillation(AF) was relatively uncommon, affecting only 10% of patients, while ischemic heart disease(IHD) was observed in 14%.

Lifestyle factors showed that 20% of patients were smokers. Among laboratory and clinical parameters, increased systolic/diastolic blood pressure(SBP/DBP) was recorded in 24%, while hyperglycemia(≥200mg/dl) and high HbA1c levels(≥10%) were present in 32% and 26% of patients respectively indicating poor glycemic control in a considerable portion of the group.

Lastly, dual antiplatelet therapy usage was noted in 48% of the patients, while single antiplatelet was reported in 52% of the patients reflecting an effort to manage stroke risk factors in this population. This data highlights the multifactorial nature of lacunar stroke and the prominence of modifiable risk factors such as hypertension, diabetes, and smoking in this cohort,(table 1).

Table 2. Baseline and follow-up data of the studied lacunar stroke patients.

	MEAN	SD	MEDIAN	IQR		SHAPIRO-WILK	KOLMOGOROV-SMIRNOVA
AGE	61.28	8.47	61.50	53.75	0.02	0.02*	0.10
ONSET	13.96	6.57	15.5	8.00	19.00	0.061	0.031*
HGA1C	7.49	2.79	6.35	5.08	0	<0.001*	<0.001*
TGS	165.36	38.03	172.50	128.00	0.026	0.03*	0.02*
TOTAL CHOLESTEROL	198.54	37.66	206.50	175.50	0.01	0.01*	0.04*
LDL	167.46	36.51	169.50	149.25	0.005	0.01*	0.11
NIHSS	6.42	2.52	7.00	4.00	0.022	0.02*	<0.001*
CHANGE	1.38	1.18	1.50	0.00	0	<0.001*	<0.001*
NIHSS AFTER 24-HOURS	7.80	3.36	8.00	4.75	0.041	0.04*	0.04*
MRS	2.34	1.02	2.00	1.75	0	<0.001*	<0.001*
MRS AFTER 24-HOURS	2.34	1.02	2.00	1.75	0	<0.001*	<0.001*
MRS AT DISCHARGE	2.20	0.95	2.00	1.00	0	<0.001*	<0.001*
MRS 3-MONTHS	1.16	0.87	1.00	0.00	0	<0.001*	<0.001*

The baseline and follow-up data of the studied lacunar stroke patients provide a comprehensive analysis of various clinical and biochemical parameters. The mean age of the patients was 61.28 years(SD=8.47), with a median of 61.50, indicating a predominance of older adults. The onset of symptoms showed a wide range(median=15.5, IQR=8.00–19.00), reflecting variability in disease presentation. Glycemic control appeared suboptimal, with mean HbA1c levels of 7.49(SD=2.79), and triglycerides(TGs) and total cholesterol levels averaged at 165.36mg/dL(SD=38.03) and 198.54mg/dL(SD=37.66), respectively, indicating dyslipidemia as a common finding. LDL cholesterol was notably high, with a mean of 167.46mg/dL(SD=36.51).

Neurological outcomes measured by the NIHSS revealed moderate severity at baseline(mean=6.42, SD=2.52), with significant deterioration noted over time(mean change=1.38, SD=1.18). Functional outcomes assessed using the mRS showed progressive recovery, with a median score of 2.00 at discharge improving to 1.00 after three months. These trends underscore the potential for recovery with appropriate management.

Normality testing using the Shapiro-Wilk and Kolmogorov-Smirnov tests highlighted significant deviations from normal distribution for most variables($p<0.05$ for many parameters), suggesting that non-parametric statistical methods may be more appropriate for analyzing these data. Overall, the dataset underscores the importance of addressing risk factors like dyslipidemia and glycemic control and highlights measurable improvements in functional and neurological outcomes over time,(table 2).

Table 3. Factors associated with deterioration among the studied lacunar stroke patients.

		NO DETERIORATION (N=25)		DETERIORATION (N=25)		TEST VALUE	P-VALUE
		No.	%	No.	%		
GENDER	Male	14	56	6	24	5.33	0.021*
	Female	11	44	19	76		
DM		6	24	14	56	5.33	0.021*
HTN		6	24	13	52	4.16	0.041*
AF		3	12	2	8	0.22	0.64
IHD		4	16	3	12	0.17	0.68
SMOKER		6	24	4	16	0.50	0.48
INCREASED SBP/DBP		3	12	9	36	3.95	0.047*
CBG ≥200 MG/DL		4	16	12	48	5.88	0.015*
HGA1C ≥10%		3	12	10	40	5.09	0.024*
ANTI PLATELETS	Single	11	44	15	60	1.282	0.258
	Dual	14	56	10	40		
ANTI-HTN		6	24	11	44	2.23	0.136

The chi-square test was used at a 0.05 level of significance, as indicated by the (*) symbol.

The analysis of factors associated with deterioration among the studied lacunar stroke patients reveals several significant associations. Gender played a notable role, with males less likely to experience deterioration(24% vs. 56%, p=0.021), while females demonstrated a higher prevalence of deterioration(76% vs. 44%). Diabetes mellitus(DM) was significantly more common in the deterioration group (56% vs. 24%, p=0.021), suggesting its contribution to worse outcomes.

Similarly, hypertension (HTN) was associated

with deterioration (52% vs. 24%, p=0.041), emphasizing the importance of controlling blood pressure in these patients. Although atrial fibrillation (AF) and ischemic heart disease (IHD) did not show significant associations (p>0.05), increased systolic/diastolic blood pressure (SBP/DBP) was more frequent in deteriorated patients (36% vs. 12%, p=0.047). Hyperglycemia (CBG≥200mg/dL) and elevated HbA1c levels (≥10%) were significantly associated with deterioration, with higher rates observed in the deteriorated group (48% vs. 16%, p=0.015; and 40% vs. 12%, p=0.024, respectively). These findings underscore the critical role of glycemic control in preventing adverse outcomes.

Other factors, such as smoking, dual antiplatelets, and antihypertensive medications, did not exhibit significant differences between groups (p>0.05).

Overall, these results highlight modifiable risk factors, including glycemic control and hypertension management, as critical targets for improving outcomes in lacunar stroke patients,(table 3).

Table 4. Other factors associated with deterioration among the studied patients.

	NO DETERIORATION (N=25)			DETERIORATION (N=25)			TEST VALUE	P-VALUE
	Median	IQR		Median	IQR			
AGE	63.0	52.5	69.0	61.0	54.0	71.0	-0.14	0.89
ONSET	16.0	7.0	20.0	14.0	9.0	18.5	-0.11	0.92
HGA1C	5.2	4.7	6.8	8.9	6.4	11.0	-4.02	<0.001*
TGS	166.0	126.5	189.0	181.0	132.5	200.0	-0.75	0.46
TOTAL CHOLESTEROL	189.0	150.5	223.0	217.0	192.5	233.5	-2.79	0.01*
LDL	159.0	125.5	190.0	181.0	165.0	201.0	-2.94	<0.001*
NIHSS	4.0	3.0	7.0	8.0	6.5	9.0	-4.72	<0.001*
CHANGE	0.0	0.0	1.0	2.0	2.0	3.0	-6.25	<0.001*
NIHSS AFTER 24 HOURS	5.0	3.5	7.0	11.0	8.5	12.0	-5.72	<0.001*
MRS	2.0	1.0	2.5	3.0	2.0	4.0	-2.50	0.01*
MRS AFTER 24 HOURS	2.0	1.0	2.5	3.0	2.0	4.0	-2.50	0.01*
MRS AT DISCHARGE	2.0	1.0	2.0	3.0	2.0	3.0	-2.57	0.01*
MRS 3MONTHS	1.0	0.0	1.0	2.0	1.0	2.0	-2.29	0.02*

Mann-Whitney test was used at a 0.05 level of significance, as indicated by the (*) symbol.

The analysis of additional factors associated with deterioration among the studied patients provides valuable insights into clinical and biochemical contributors. Age and onset time did not show significant differences between patients with and without deterioration(p>0.05), suggesting these variables were not major determinants in this cohort. However, glycemic control, as reflected by HbA1c levels, was markedly worse in the deterioration group(median=8.9, IQR=6.4–11.0) compared to those without deterioration(median=5.2, IQR=4.7–6.8), with a highly significant p-value(<0.001). This emphasizes the impact of poor glycemic control on adverse outcomes.

Lipid profiles also highlighted significant differences. Total cholesterol(median=217.0, IQR=192.5–233.5) and LDL cholesterol(median=181.0, IQR=165.0–201.0) were significantly higher in the deterioration group compared to their counterparts (p=0.01 and p<0.001, respectively), underlining the role of dyslipidemia in stroke progression. Neurological status, assessed by NIHSS, showed significantly higher scores at baseline(median=8.0, IQR=6.5–9.0) and after 24-hours(median=11.0, IQR=8.5–12.0) among deteriorated patients(p<0.001 for both), indicating more severe neurological impairment in this group.

Functional outcomes assessed via the mRS demonstrated poorer recovery among deteriorated patients across various time points. At discharge and three months, the mRS scores were significantly higher in the deterioration group(p=0.01 and p=0.02, respectively), reflecting prolonged disability. Additionally, the change in NIHSS scores was significantly greater among deteriorated patients(median=2.0, IQR=2.0–3.0, p<0.001), suggesting more pronounced clinical worsening,(table 4).

Table 5. Logistic regression model to identify deterioration among lacunar stroke patients.

	BETA	S.E.	WALD TEST	P-VALUE	ADJUSTED OR
AGE	0.01	0.08	0.01	0.94	1.01
MALE	1.80	1.21	2.20	0.14	6.03
HGA1C	0.57	0.27	4.65	0.03*	1.77
NIHSS	0.74	0.30	6.05	0.01*	2.10
LDL	0.03	0.02	1.86	0.17	1.03
INCREASED SBP/DBP	1.52	1.38	1.20	0.27	4.55
MRS	0.42	0.70	0.37	0.54	1.53
CONSTANT	-16.02	6.69	5.73	0.02	0.00

Beta:regression coefficient; SE:standard error, *:significant p-value, Nagelkerke R-Square=0.795

Omnibus-test=45.35 (p<0.001*), Classification table=92% were correctly classified

The ROC curve for the regression model showed an AUC of 0.963 (95% CI of 0.919-1.00)

The logistic regression analysis identified significant predictors of deterioration among lacunar stroke patients. Glycemic control, represented by HbA1c, was a key factor, with a beta coefficient of 0.57(p=0.03). The adjusted odds ratio(OR) of 1.77 indicates that for every unit increase in HbA1c, the odds of deterioration increase by 77%. Baseline NIHSS scores were also significant predictors(beta=0.74, p=0.01), with an adjusted OR of 2.10, suggesting a two-fold increase in the odds of deterioration per unit increase in NIHSS score.

While other factors like age, LDL levels, and increased SBP/DBP showed elevated odds ratios, their associations were not statistically significant(p>0.05). Notably, male gender exhibited a higher odds ratio(OR=6.03) but did not reach significance(p=0.14), possibly due to sample size constraints. The constant term was significant(p=0.02), further emphasizing the strength of the model.

The model demonstrated excellent predictive ability, with a Nagelkerke R^2 value of 0.795,

indicating that approximately 80% of the variability in deterioration was explained by the included predictors. The Omnibus test was highly significant($p<0.001$), confirming the model's robustness.

Furthermore, 92% of cases were correctly classified, showcasing its practical utility. The ROC curve analysis reinforced this, with an AUC of 0.963(95% CI:0.919–1.00), signifying excellent discriminative ability,(table 5; figure 1).

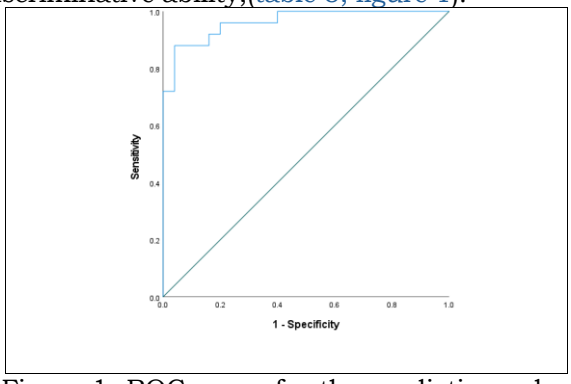

Figure 1. ROC curve for the prediction value of the regression model.

4. Discussion

This study highlights key clinical and biochemical characteristics of lacunar stroke patients and their progression over time. The mean age was 61.3-years(SD=8.5), with a median symptom onset of 15.5-hours(IQR=8.0–19.0), indicating variability in disease presentation.

Metabolic risk factors were prominent, with HbA1c at 7.5(SD=2.8), triglycerides 165.4mg/dL(SD=38.0), and LDL cholesterol 167.5mg/dL(SD=36.5), underscoring the role of dyslipidemia and poor glycemic control in stroke pathophysiology.

Neurological severity was moderate at baseline(NIHSS=6.4, SD=2.5) with some deterioration(mean change=1.4, SD=1.2). However, functional outcomes improved, with mRS decreasing from 2.0 at discharge to 1.0 at three months, reflecting recovery with proper management.

Consistent with our results, ten studies included in a systematic review applied the TOAST classification, which supports a diagnosis of lacunar infarction when hypertension and diabetes are present, while excluding carotid stenosis>50% and potential cardiac embolic sources such as atrial fibrillation.[5]

Our study found that poor glycemic control emerged as a major contributor to deterioration. Elevated HbA1c levels(≥10%) were significantly associated with adverse outcomes($p=0.024$), and logistic regression analysis confirmed its predictive value, with an adjusted odds ratio(OR) of 1.77($p=0.03$).

In alignment with our findings, a meta-regression analysis from a large systematic review of 67 studies (13,407 participants) identified diabetes and smoking as significant risk factors for early neurological deterioration. However, the definition of early neurological deterioration varied across studies, ranging from<24 hours to 3 weeks.[6]

Dyslipidemia in the present study was another critical factor reported in the present study, with significantly higher total cholesterol and LDL levels observed among deteriorated patients($p=0.01$ and $p<0.001$, respectively). Given the established role of lipid abnormalities in atherosclerosis and cerebrovascular disease, aggressive lipid-lowering interventions may be beneficial in mitigating stroke progression.

In line with our findings, a Korean study of 131 patients reported that 13% experienced early neurological deterioration after admission. Univariate analysis identified diabetes, systolic blood pressure, TG, cholesterol, LDL-C, and homocysteine as risk factors. After multivariate adjustment, high TG levels(>145mg/dL) remained an independent predictor of early neurological deterioration(OR=11.46, 95% CI=1.07–122.87, P=0.044), highlighting the role of dyslipidemia in stroke progression.[7]

Our study reported that increased systolic/diastolic blood pressure(SBP/DBP) was more prevalent in deteriorated patients(36% vs. 12%, $p=0.047$), aligning with existing evidence that BP fluctuations contribute to secondary brain injury.

In agreement with our findings, a study of 638-lacunar stroke patients identified key risk factors for early neurological deterioration. Elevated admission systolic blood pressure was significantly associated with early neurological deterioration. Specifically, systolic blood pressure of 160–179mmHg(aOR 9.395, 95% CI 4.310–20.479, $p<0.001$) and SBP≥180mmHg(aOR 16.030, 95%CI 5.991–42.891, $p<0.001$) markedly increased the risk, highlighting the critical role of blood pressure management in preventing the neurological decline in lacunar stroke.8

In concordance with our findings, a meta-regression analysis from a large systematic review of 67 studies (13,407 participants) identified hypertension as a significant risk factor for early neurological deterioration.[6]

A study of 113 patients with lacunar infarctions found that 31.9% had a poor outcome. Factors associated with worse prognosis included older age, hypertension, high body temperature, elevated systolic blood pressure, serum glucose, and IL-6 levels, along with a lower Canadian Stroke Scale score at admission. In multivariate analysis, only systolic blood pressure remained a significant predictor, aligning with our findings.[9]

NIHSS scores were significantly higher among deteriorated patients at baseline(median=8.0, p<0.001) and continued to worsen over 24 hours. Logistic regression confirmed NIHSS as an independent predictor(OR=2.10, p=0.01), reinforcing its utility in early risk stratification.

In agreement with our findings, a study of 638-lacunar stroke patients identified key risk factors for early neurological deterioration. Higher admission NIHSS scores(aOR 1.132, 95% CI 1.046–1.225, p=0.002) highlight the critical role of baseline NIHSS in predicting a neurological decline in lacunar stroke.[8]

Our study identified female gender as a potential risk factor for early neurological deterioration in lacunar stroke patients, though logistic regression did not confirm a significant association.

Consistently, a study of 638 lacunar stroke patients reported that the female sex(aOR 2.752, 95% CI 1.277–5.933, p=0.010) significantly increased the risk of early neurological deterioration.[8]

Aligning with our findings, a meta-regression analysis from a systematic review of 67 studies (13,407 participants) identified female sex as a risk factor for early neurological deterioration.[6]

A multivariable logistic regression model in 638-lacunar stroke patients(65.8% male) identified independent predictors of early neurological deterioration. Higher admission NIHSS score(aOR 1.132, p=0.002), female sex(aOR 2.752, p=0.010), and elevated admission systolic blood pressure(160–179 mmHg: aOR 9.395, p<0.001;≥180mmHg: aOR 16.030, p<0.001) were significantly associated with early deterioration. Conversely, shorter onset-to-admission time(aOR 0.995, p=0.031), SBP drop≥20mmHg within 3 days (aOR 0.037, p<0.001), and thalamic lacunar infarction (aOR 0.098, p=0.033) were protective factors.[8,10]

4. Conclusion

Elevated HbA1c and LDL cholesterol levels were strongly associated with deterioration, emphasizing the role of metabolic control in stroke progression. The baseline NIHSS score emerged as the most significant predictor of deterioration, underscoring the importance of initial stroke severity assessment. The logistic regression and discriminant function models demonstrate high predictive accuracy, reinforcing their utility in clinical decision-making.

Disclosure

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