Study on risk factors for late seizure in post-ischemic stroke patients in Vietnam

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Summary

Introduction: Late post-stroke seizure (LS) occurs ≥ 7 days after the stroke onset. LS usually occurs within the first 2 years, but the risk of occurrence within 10 years is up to 71.5%. Strategies to prevent and minimize the negative impacts caused by LS are still ineffective. Therefore, building a model of factors that can accurately predict the risk of LS is an urgent priority. *Objective*: Describe clinical and laboratory and imaging findings characteristics and prognosis of seizures in patients after supratentorial stroke and analyze risk factors for LS in this patient group. *Subject and method*: The study design is a prospective cohort study with longitudinal follow-up. The study subjects included 739 patients diagnosed with cerebral infarction, treated at the Department of Neurology, the Department of Stroke - 108 Military Central Hospital and the Stroke Center - Phu Tho Provincial General Hospital. Patients were diagnosed with LS based on clinical symptoms and electroencephalograms. Related factors include: general characteristics (age, gender, BMI...), stroke characteristics (Glassgow, NIHSS, lesion location, lesion area...), blood tests (complete blood count, serum biochemistry...). These factors were analyzed by multivariable logistic regression model to determine independent prognostic factors for late seizure. *Results*: Univariate analysis showed that factors affecting the risk of LS included: age, history of arrhythmia, previous stroke, early post-stroke seizure (ES), cortical lesions, temporal lobe lesions, embolism from the heart (TOAST), white blood cells, urea, creatinine, Cholesterol and LDL-C. Logistic regression model showed that history of ES was the only independent predictor of LS (OR: 5.906; 95%CI: 1.374-25.373; p<0.001). *Conclusion*: Early post-stroke seizure was an independent predictor and increased the risk of post-stroke epilepsy.

Keywords: Ischemic stroke, Post stroke epilepsy, Predictive factors.

I. BACKGROUND

Stroke is a major health problem worldwide and is also a major cause of disability. Post-stroke seizure (PSS) in the elderly occurs in about 30 - 50% of patients and PSS increase the risk of death, reduces quality of life, and reduces the ability to recover function after a stroke. Prolonged treatment with antiepileptic drugs also increases the risk of recurrent stroke. PSS is divided into two types: early post-stroke seizure (ES) and late seiure (LS). Late seizure begins 7 days after a stroke and the risk of seizure recurrence after 10 years for LS is 71.5% [1].

The cause of LS is due to complex changes in the structure and physiology of neurons including: (1) astrocytic proliferation, (2) damage to the blood-brain barrier, (3) genetics and genes; (4) changes in the structure of the brain network; (5) changes in regional hemodynamics in the brain [2].

Evidence shows that the risk factors for PSS are still inconsistent and hardly unpredictable. Studies have shown that many factors are associated with a

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high incidence of PSS such as hemorrhagic stroke, cortical involvement, extensive lesion, stroke severity, decreased consciousness and vascular risk factors [1]. In Vietnam, there has been no study describing the clinical and laboratory and imaging findings characteristics and assessing the risk factors for post-stroke epilepsy. Therefore, this study was conducted with the following objectives: To describe the clinical and laboratory and imaging findings characteristics of LS and to investigate the risk factors for LS.

2. SUBJECTS AND METHODS

2.1. Research subject

Inclusion criteria:

All patients were diagnosed with cerebral infarction based on clinical features and imaging test according to WHO standards.

No personal history of seizures or epilepsy prior to diagnosis of cerebral infarction

Patient ≥ 18 years old.

Agree to participate in the study.

Exclusion criteria:

Patients with neurological disorders other than cerebral infarction that may explain the seizures include: brain tumors, trauma, nervous system infections, brain and nervous system surgery, and vasculitis.

Patients lost information before the end of the study.

2.2. Research process and research standards

2.2.1. Research design

This is a cross-sectional, prospective, longitudinal study. We collected patients diagnosed with cerebral infarction according to WHO criteria, treated at the Department of Neurology and the Department of Stroke, 108 Military Central Hospital, and Stroke Center-Phu Tho General Hospital. Patients were monitored for symptoms of LS through clinical symptoms and EEG when suspected. Information was collected including general characteristics, clinical, laboratory and imaging findings symptoms and the relationship of these characteristics and the incidence of LS.

2.2.2. Research process

Patients were diagnosed with cerebral infarction using the WHO definition of cerebral infarction criteria [3]. Clinical and laboratory and imaging findings examinations were conducted and information was collected directly, and a research form was filled out. Patients were followed up from the time of diagnosis of stroke to the end of the study. The form of follow-up included examination and recording of patient information at follow-up visits at the medical facility. In cases where patients did not return for regular check-ups, the researcher would call every 6 months to collect information about the seizures. If the patient had a suspected clinical seizure, a routine EEG was recorded at the Department of Neurology, Stroke Center - 108 Military Central Hospital and Stroke Center - Phu Tho General Hospital.

2.2.3. Standards used in the study:

General characteristics: age, gender, BMI, medical history and lifestyle.

Smoking history: smoking more than 20 packs * years, patients who have stopped smoking for < 3 months are still considered to have a history of smoking.

History of epilepsy: Early epilepsy is defined as a seizure diagnosed according to the 2014 ILAE criteria [4], occurring within 7 days after the diagnosis of stroke.

Characteristics of stroke:

Location of lesion (subcortical/cortical involvement): diagnosis is based on CT scan or MRI of the brain. Subcortical lesions are defined as lesions limited to subcortical structures such as the internal capsule, thalamus, and basal ganglia. Other lesions, including those localized in the cerebral cortex alone or mixed cortical-subcortical lesions, are considered cortical-involvement lesions.

The subgroup classification of cerebral infarction was based on the criteria of the Trial of

Org 10172 in Acute Stroke Treatment (TOAST) [5]. The subgroups included in the evaluation included: 1) large-artery atherosclerosis, 2) cardioembolism, 3) small-vessel occlusion, 4) stroke of other determined etiology, and 5) stroke of undetermined etiology. The subgroups of unspecified and unknown causes were not included in the evaluation.

Features of blood test

Blood tests for hematology and biochemistry include: red blood cells, white blood cells, platelets, glucose, creatinine, cholesterol, triglycerides, LDL-C, HDL-C, sodium, potassium. These tests are all taken at the time of diagnosis of cerebral infarction.

Epilepsy is defined as a seizure diagnosed according to the ILAE 2014 criteria, satisfying 1 of the following 3 criteria: (1) At least two unprovoked (or reflex) seizures occurring > 24 h apart; (2) one unprovoked (or reflex) seizure and a probability of further seizures similar to the general recurrence risk (at least 60%) after two unprovoked seizures, occurring over the next 10 years; (3) diagnosis of an epilepsy syndrome [4], appearing after the 7th day from the diagnosis of cerebral infarction.

2.2.4. Data processing and analysis

Data were processed by SPSS 26.0 software.

Categorical variables are described as proportions (%). Quantitative variables are described as means ± standard deviations . In univariate analysis, the Chi-square test was used to assess the association between categorical variables and late seizure status (yes/no); the independent t-test was used to assess the association between quantitative variables (normally distributed) and LS status, or the Mann-Whitney U test (if the variable was not normally distributed). We used logistic regression to assess the influence between associated factors and LS status. Results were presented as odds ratios (OR) and 95% confidence intervals (95%CI). A p value <0.05 was considered statistically significant. All tests were 2-tailed.

2.2.5. Research ethics

The research topic is purely for scientific research purposes, serving the diagnosis and treatment of diseases, and for no other purpose. The participating patients are clearly explained and advised by the researcher about the purpose and process of the research.

Patient information is kept completely confidential and used only for scientific research purposes. The study has been approved by the scientific research council and the medical council of 108 Central Military Hospital.

III. RESULT

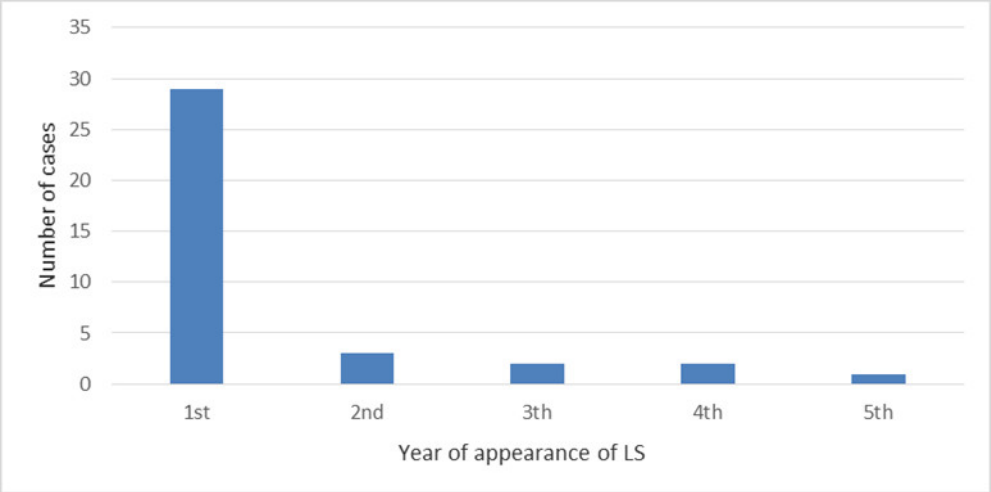

Chart 1. Incidence of Late Seizure over time

Comment: The time of appearance of LS is mainly the first year after the onset of stroke (29/37 cases).

Table 1. General characteristics and LS

Characteristic		LS (n = 53)	No LS (n = 686)	Total	p
Age (n = 739)		64.5 ± 10.9	59.0 ± 9.8	59.3 ±9.9	t=-3.341 p=0.001
BMI (n = 468)		23.1 ± 2.7	22.7 ± 2.6	22.7 ± 2.5	t= -0.796 p= 0.427
Gender (n=739)	Male (n/%)	27 (5.4)	474 (94.6)	501 (67.8)	χ^2 = 0.195 p= 0.659
	Female (n/%)	11 (4.6)	227 (95.4)	238 (32.2)	
History	Hypertension	27 (71.1)	449 (64.1)	476 (64.4)	χ^2=0.771 p= 0.380
	Diabetes	5 (13.2)	121 (17.3)	126 (17.1)	χ^2=0.429 p= 0.512
	Metabolic disorders	1 (2.6)	33 (4.7)	34 (4.6)	χ^2=0.354 p= 0.552
	Smoke	4 (10.5)	69 (9.8)	73 (9.9)	$\chi^{2=}$0.019 p= 0.891
	Valvular heart disease	2 (5.3)	16 (2.3)	18 (2.4)	χ^2=1.348 p= 0.235*
	Arrhythmia	8 (21.1)	30 (4.3)	38 (5.1)	χ^2=20.791 P<0.001
	Previous stroke	31 (81.6)	136 (19.4)	167 (22.6)	X^2=79.672 p<0.001
	Early post-stroke seizure*	30 (78.9)	23 (3.3)	53 (7.2)	X^2= 309.993 p<0.001
SeLeCT Score		4.2 ± 1.1	3.5 ± 1.2	3.5 ± 1.2	t=-3.884 P<0.001

*: Fisher's exact Test

Comment:

The mean age of the study group was 59.3. The LS group had a higher mean age than the non-LS group (p=0.001).

The male/female ratio of the study group was ~2/1. The mean BMI of the study group was 22.7. There was no difference in the incidence of LS between the subgroups based on the above two factors.

64.4% of patients had hypertension. The next most common comorbidities were previous stroke (22.6%) and diabetes (17.1%). 7.2% of patients had a history of stroke. Among the factors related to history and comorbidities, arrhythmia, previous stroke, and history of stroke were associated with stroke (p<0.001).

Table 1Characteristics of stroke and cerebral infarction

Characteristic		LS (n = 53)	No LS (n = 686)	Total	p
Glasgow ($\overline{X}$ ±SD) (n = 739)		14.1 ±1.6	14.4 ± 1.4	14.3 ± 1.5	t=0.948 p=0.343
NIHSS (mean rank)* (n = 739)		319.8	372.7		Z= -1.490 p=0.136
Side of lesion (n = 739)	Left	18 (5.3)	319 (94.7)	337 (45.6)	χ^2=0.093 p=0.882
	Right	18 (7.6)	349 (92.4)	367 (49.7)	
	Mixed	2 (5.7)	33 (94.3)	35 (4.7)	
Location** (n = 739)	Subcortical	20 (3.6)	535 (96.4)	555 (75.1)	χ^2=10.817 p=0.002
	Cortical involvement	18 (9.8)	166 (90.2)	184 (24.9)	
Lobar involvement (n = 739)	Frontal	9 (23.7)	91 (13.0)	100 (13.5)	χ^2=3.529 p=0.083
	Parietal	30 (78.9)	582 (83.0)	612 (82.8)	χ^2=0.421 p=0.516
	Temporal	16 (42.1)	186 (26.5)	202 (27.3)	χ^2=4,400 p=0.036
	Occipital**	5 (13.2)	45 (6.4)	50 (6.8)	χ^2=2.642 p=0.214
ASPECT (n=739)		8.9 ± 1.5	8.7 ± 1.4	8.7 ± 1.2	t=-0.990 p=0.323
TOAST (n = 712) **	Large-artery atherosclerosis	9 (4.8)	180 (95.2)	189(26.5)	χ^2=30.704 p<0.001
	Small-vessel occlusion	19 (3.9)	474 (96.1)	493 69.2)	
	Cardioembolism	8 (26.7)	22 (73.3)	30 (4.2)	

**Compare Mean rank with Mann Whitney U Test*

*** Fisher's exact Test*

Comment:

Glasgow coma index and NIHSS were included in the analysis of stroke severity. There was no correlation between these indices and LS.

Similarly, the lesion area based on cerebral hemisphere location also had no significant correlation with LS (p=0.882). In terms of the extent of stroke lesion, cortical involvement lesions had a higher incidence of LS than subcortical lesions (9.8% vs. 3.6%, p=0.002).

Regarding the location of lesion based on lobe, temporal lobe lesions were associated with LS, with 42.1% of the LS group having lesions in this lobe, compared with 26.5% in the non-LS group (p=0.036). Lesions in other lobes were not associated with LS.

ASPECT scores did not differ between the group with and without LS. Meanwhile, in terms of stroke causes, cardioembolic stroke subtype had a statistically significant higher incidence of stroke than other causes (p<0.001).

Table 3. Multivariate regression model of factors associated with LS

Characteristic		Coefficient B	Exp (B)/ OR	p	95%CI
Age		-0.009	0.991	0.772	
Arrhythmia		1,439	4.216	0.329	
Previous stroke		0.520	1.682	0.479	
Early post-stroke seizure		1.776	5.906	<0.001	1,374-25,373
Temporal lobe injury		-0.733	0.481	0.325	
Cortical involvement		1,185	3,271	0.067	
TOAST	Large-artery atherosclerosis	1			
	Small-vessel occlusion	1,014	2,757	0.521	
	Cardioembolism	0.765	2,149	0.613	
White blood cells		0.048	1,050	0.466	
Urea		0.265	1,304	0.121	
Creatinine		-0.018	0.982	0251	
Cholesterol		-0.296	0.743	0.161	

Comment: The only independent prognostic factor was history of ES. Patients with a history of ES had a 5.9-fold (95%CI: 1.374-25.373;p<0.001) increased risk of LS compared to the group without a history of ES.

IV. DISCUSSION

Through a study based on data from 739 infarction stroke patients with a longitudinal follow-up period of 5 years, we recorded 38 patients with LS, accounting for 5.1% of the total number of patients. The incidence of LS fluctuates quite a lot across studies. Author Chen reported a LS incidence of 2.6% in a study population of 4126 patients, with a follow-up period of 5 years [6]. The meta-analysis of author Zou recorded a higher incidence of 5% [7]. From the studies, it can be seen that the incidence of LS in our study is quite similar to the results from other studies. Unlike ES, LS occurs after stroke from the 7th day to many years after the time of stroke, so statistics on the incidence of LS lasting for years will be more accurate. Studies show that the highest incidence of LS after stroke is usually in the first 1-2 years [8, 9]. The majority of patients with LS were at 1 year after stroke (29 cases, accounting for 76.3%). The SeLECT score developed by Graham et al. is considered the most commonly used prognostic score for LS after stroke, with sensitivity and specificity of approximately 60% and 100%, respectively [10]. When evaluating the SeLECT score on this group of patients, we found that the average SeLECT score of the group with LS was significantly higher (4.2 vs. 3.4; p<0.001). With the SeLECT cut-off point = 3.5; The sensitivity and specificity of this score were 71.1 and 43.1%, respectively, when evaluated in our study patient group.

Univariate analysis showed that the group with LS had a higher mean age than the group without LS. However, the multivariate model did not show a significant effect of age on the risk of epilepsy (Table 4). This result is similar to many other studies. Univariate analysis in the study by Galovic showed that age did not have a significant effect on the risk of epilepsy (HR: 0.9; p=0.16) [11]. Bladin's multicenter

prospective study also did not show a difference in age between the 2 groups with/without post-stroke epilepsy [12]. Studies by Chen and Lahti also showed a similar trend [6, 8]. However, in Phan's meta-analysis, young age and female gender were 2 factors that increased the risk of post-stroke epilepsy. The authors suggested that younger subjects have larger brain volumes, respond more to epileptic stimuli, and often have larger brain regions and large blood vessel lesions than older patients, thereby increasing the incidence of late-onset epilepsy [13]. Graham's prospective study showed strong evidence that older age is a protective factor for late-onset epilepsy, with a 10-year hazard ratio (10-year HR) of 0.21 (95%CI: 0.06-0.66, p=0.004) when comparing patients > 85 years old with patients <65 years old, which represents a 78% reduction in the incidence of late-onset epilepsy in the older group [9].

Our study did not record any association between gender and LS. Most studies have shown that gender does not have a significant impact on the risk of LS [9, 11, 12, 14, 15]. Similar trends were also recorded in the pooled analyses of SFZou (2015) and zhang (2014) [7, 16]. On the other hand, Joshep Phan's analysis indicated that it may be because women are more likely to have complete anterior cerebral artery occlusion than men, thereby increasing the risk of LS in women compared to men [13]. However, in general, studies agree that the incidence of LS is similar in both sexes.

Regarding medical history and comorbidities, univariate analysis noted that arrhythmia, previous stroke, and history of early epilepsy were comorbid factors associated with late-onset epilepsy. The influence of medical history and comorbidities on late-onset epilepsy remains controversial. Hypertension is closely related to small vessel disease and increases the risk of temporal lobe epilepsy, which has been demonstrated in a mouse model [13], but studies have not yet reached a consensus on the influence of this factor [6, 15]. In addition, some studies have shown that hypertension is a protective factor for LS, with a lower frequency of LS in the hypertensive group [8, 17, 18]. Some studies have shown that a history of stroke is a factor that increases the risk of late-onset epilepsy [13, 19]. However, other studies have not shown a significant difference between these factors [12, 17, 18]. A history of atrial fibrillation was noted to affect the risk of stroke after stroke in the study of LP.Kammersgaard [14], however, other studies have not shown a clear association between this factor [6, 12]. One factor that is believed to have a strong influence on the risk of stroke is the presence of stroke in history. Our study showed that ES is the only independent predictor of the risk of stroke. Patients with a previous ES have a 5.9-fold higher risk of stroke (1.374-25.373; p<0.001). A study by author Galovic in 2018 aimed at building a prognosis model for LS (SeLECT) in infarct stroke patients showed that ES is one of the strong prognostic factors for the appearance of LS. Patients with ES will increase the risk of LS by 4.8 times within the next 10 years (p<0.0001) [11].

The severity of stroke is a factor that is often included in the assessment of risk factors that may affect the occurrence of LS. However, in this study, when using both Glasgow and NIHSS scores, we did not record any association with the risk of LS. To date, the impact of stroke severity on stroke remains controversial in many studies. Galovic et al. noted that patients with an NIHSS score ≥ 11 had a 2.7-fold higher risk of stroke within 10 years (p=0.008) than patients with an NIHSS score ≤ 3 [11]. However, other studies have shown the opposite result. Kammersgaard et al. showed that the Scandinavian Stroke Scale (SSS) score was similar between the two groups of patients (35.9 vs. 36.3, p=0.9) [14]; Meanwhile, Graham et al. also noted a similar trend when evaluating the incidence of stroke between Glasgow scores (p=0.156) [9]. The pooled analysis of Zhang et al. did not provide a pooled value for this factor because of the high level of heterogeneity between the two groups of patients [16]. It is possible that the influence of stroke severity on the risk of stroke is not much, and the subjectivity in assessing stroke severity partly affects the inconsistency of this factor.

One factor that many studies have found to have the strongest influence on the risk of stroke is cortical involvement. In this study, univariate analysis showed that patients with cortical involvement had a higher incidence of stroke than those with subcortical lesion (9.8% vs. 3.5%, p<0.002). However, the multivariate model did not show a statistically significant difference at p=0.05. This result is similar to many studies in the world [11, 12, 20, 21]. Bladin's prospective study showed that the location of the lesion in the cerebral cortex independently affected the increased risk of post-stroke post-stroke epilepsy. The multivariate model of this study showed that the location of the lesion in the cerebral cortex increased the risk of post-stroke epilepsy in both the infarction group (HR: 2.09; p=0.01) and the cerebral hemorrhage group (HR: 3.16; p=0.008) [12]. In addition, Galovic et al. also showed similar results when reporting the risk of LS due to the factor of cerebral cortex lesion (adjusted for age and sex) was 4.2 times higher than that of the group without cerebral cortex lesion (p=0.0003) [11]. The meta-analysis of author Wang also showed the influence of this factor on both ES and LS risk, in which, cerebral cortex damage increased 11.84 times (3.77-19.83, p=0.001) the risk of LS in patients after stroke [20]. However, the multivariate model in LP Kammersgaard's study showed that cerebral cortex damage was not an independent factor affecting the risk of post-stroke epilepsy (OR: 1.2; 95%CI: 0.5-2.7; p=0.67) [14]. The mechanism of epileptogenesis related to cortical involvement has been studied by many researchers. Supported hypotheses include: (1) the development of new nerve cell axons to replace cells that die due to injury, but these axons develop abnormal electrical connections and increase physiological discharge stimulation; (2) γ - aminobutyric acid (GABA)ergic neurons , responsible for inhibiting excessive electrical impulses, undergo degeneration with manifestations such as dendritic retraction, reduced fiber length, and changes in inhibitory synaptic structure on pyramidal cells; (3) loss of trophic support from interneurons [22].

Temporal lobe location also increased the incidence of LS in our analysis (42.1% vs. 26.5%, p=0.036). Studies on post-traumatic epilepsy show that temporal lobe lesions have the highest incidence of epilepsy, followed by frontal lobe [23, 24]. Gina Sizemore et al suggested that the common point in the epileptic physiology of epilepsy and brain injury is the electrophysiological disturbance of ion channel damage when brain cells die [25]. However, the impact of lesion location on the risk of epilepsy is not much, except for some studies showing that epilepsy due to complete anterior cerebral artery lesions increases the risk of epilepsy more than partial lesions [9, 13]. Multivariate analysis results have not recorded an independent impact of temporal lobe lesion location on the increased risk of epilepsy.

Regarding the cause of stroke according to the TOAST classification, we noted that the incidence of LS in the group of patients with cardioembolic stroke subtype was higher than that in other groups of causes. However, this relationship was not statistically significant when analyzed in a multivariate model. Galovic's multivariate analysis also did not record an independent effect of the stroke cause on the risk of LS (p=0.65) [11]. Similarly, the multicenter study of C.Ferreira- Atueta et al did not record a significant effect of the infarction stroke cause on the risk of LS (p=0.104) [21].

V. CONCLUSION

The average follow-up time of the group with late seizures was 1.91± 1.16, and of both groups was 3.61 ± 0.74. There were 3.86% of patients with late seizures after stroke, mainly starting in the first year after stroke with 10/41 (24.4%). The average age was 63.8 ± 10.9 years, and 73.2% of patients were male. There were 40/41 (97.6%) patients with late seizures with motor onset, of which 56.1% had generalized seizures, 17/45 (41.5%) had focal seizures with 23 consciousness, and 6/41 had seizures with impaired consciousness. 73.7% of patients with focal seizures had no decreased consciousness and 26.3% had decreased consciousness. The characteristics of the lesions on CT-MRI include: bilateral lesions, frontal lobes, cerebral infarction and anterior cerebral artery region, increasing the risk of LS. Video EEG detected abnormalities in 10/41 (24.4%) cases, mainly sharp

waves 8/41 (19.5%), commonly in the parietal and temporal lobes 9/41 (22%). Longitudinal follow-up and assessment of mRS scores at hospital discharge 6 to 1 year after discharge, and at the end of the study showed that. LS did not increase the risk of death or reduce the level of recovery.

Associated factors include old age, previous stroke, stroke severity (low Glasgow Coma Scale), atrial fibrillation, abnormalities on echocardiography, mixed bilateral lesions on CT/MRI, frontal lobe, cerebral cortex, anterior cerebral artery region, cerebral infarction type and ES are statistically significant risk factors for LS.

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