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Original Article

Effect of Intravenous Thrombolysis and Mechanical Thrombectomy on the Incidence of Acute Symptomatic Seizure and Post-Stroke Epilepsy in Patients with Acute Large-Vessel Occlusion

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Objective: Reperfusion therapy, such as intravenous tissue-plasminogen activator (IV-tPA) and mechanical thrombectomy (MT) for acute ischemic stroke, may increase the incidence of acute symptomatic seizure (ASS) and post-stroke epilepsy (PSE). This study aimed to analyze the effect and predictors of reperfusion therapy for ASS and PSE limited to large-vessel occlusions (LVOs).

Methods: This retrospective study classified 237 subjects with LVO into four groups: (1) IV-tPA + MT+ (n = 74 cases, (2) MT only (n = 82), (3) tissue-plasminogen activator (tPA) only (n = 28), and (4) IV-tPA – MT– (n = 53). The incidences of ASS and PSE were assessed. Potential predictors, such as etiology, functional disability, neuroimaging findings, and the SeLECT score, were statistically analyzed.

Results: There were 12 (5.1%) subjects with ASS and 10 subjects (4.2%) with PSE. The IV-tPA and MT groups had significantly high reperfusion rates, with a Thrombolysis in Cerebral Infarction score ≥2c (p = 0.01) but there were no significant differences in the increases of hemorrhagic transformation, ASS, and PSE. An Alberta Stroke Program Early Computed Tomography Score <6 was a significant predictor of ASS (p = 0.01), and an infarct volume >60 ml was a significant predictor of PSE (p = 0.01).

Conclusion: Reperfusion therapy for acute LVO was not found to increase the risk of ASS and PSE. Large-sized infarctions should be treated with care in PSE.

Keywords ▶ post-stroke epilepsy, acute symptomatic seizure, large vessel occlusions, mechanical thrombectomy

Introduction

In general, post-stroke epilepsy (PSE) occurs in 2%–14% of adults, although the rate of ischemic stroke is as low as 2%–4%.[1–7] Some critics have recently argued that an increase in reperfusion therapy, such as intravenous tissue-plasminogen activator (IV-tPA) and mechanical thrombectomy (MT), will increase the risk of epilepsy because of hemodynamic changes and hemorrhagic transformation.[1–3] Large-vessel occlusions (LVOs) themselves have a risk of acute symptomatic seizure (ASS) and PSE with hemodynamic change and large-size infarction; therefore, analysis of whole cerebral infarctions, including mild cases, is supposedly inaccurate. There are few reports limited to LVO that have described specific predictive factors of ASS or PSE. This study aimed to assess the incidences and predictors of ASS and PSE associated with LVO.

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Materials and Methods

A prospective analysis of 237 LVO cases from 2018 to 2023 at our hospital was performed. We included consecutive individuals aged 18 years who had acute LVO confirmed by computed tomography (CT), magnetic resonance imaging (MRI), or angiography. Stroke etiology was categorized according to the Trials of Org 10172 in Acute Stroke Treatment (TOAST) classification.

We excluded patients with a medical history of seizure or epilepsy before the stroke, primarily hemorrhagic stroke, transient ischemic attack, potentially epileptogenic comorbidities (i.e., intracranial tumor, cerebral venous thrombosis, arteriovenous malformation, hydrocephalus, history of severe traumatic brain injury, or brain surgery), and metabolic disorder (glucose: <36 mg/dl, >450 mg/dl; Na <115 mEq/L; Ca < 5 mEq /L, urea nitrogen <100 mg/dl; creatinine >10 mg/dl). Patients with a ≥1-year follow-up period were also excluded.

Definitions

ASS was defined as a clinically observed convulsion or nonconvulsive status epilepticus (NCSE) recorded on an electroencephalogram (EEG) within the first 7 days after stroke, in agreement with the International League Against Epilepsy guidelines.[8] PSE was defined as at least one unprovoked seizure 7 days after a stroke. The occurrence of PSE was ascertained by reviewing follow-up medical records. In patients with altered consciousness or with a worsening of the focal neurological signs, a new MRI (diffusion weighted imaging [DWI], hypervascularity of MRA, hyperperfusion of arterial spin labeling, etc.) was done to rule out the progression of the new ischemic stroke, hemorrhagic transformation, or epilepsy. EEG was performed according to the indication of the attending physician. The EEG monitoring over 24 h was not performed.

Treatment protocol and patient evaluation

Bolus administration of alteplase 0.9 mg/kg with 10% of the dose and 90% given as a 1-h perfusion IV-tPA was performed. MT was performed through the femoral access whenever possible using both the aspiration catheter and stent retriever. Intravenous anesthesia with propofol was completed at the postoperative management. Regarding our medication protocol, there is no prophylactic antiseizure medication administered. The intravenous injection of diazepam and fosphenytoin is used to stop the convulsion, and general anesthesia with propofol is performed for status epilepticus. Simultaneously, the drip or oral medication of levetiracetam 1000 mg or lacosamide 100–300 mg is usually administered. The discontinuation of the oral medication for ASS is decided after discharge from the hospital.

The collected data contained the following variables: age, sex, vascular risk (hypertension, diabetes mellitus, hyperlipidemia, atrial fibrillation), the National Institutes of Health Stroke Scale (NIHSS) score, and modified Rankin Scale (mRS) at baseline, 3 months, and 1 year after. Radiological: Alberta Stroke Program Early CT score (ASPECTS); infarct volume (ml); and hemorrhagic transformation at baseline, 24 h, and 7 days after. LVOs were classified as those in the internal carotid artery, anterior cerebral artery, middle cerebral artery (M1 and M2), posterior cerebral artery, vertebral artery, and basilar artery. The territories of cerebral infarction, such as cortical involvement[9] and middle cerebral artery lesions,[6] which are considered to be at risk of PSE, were assessed. Regarding the prediction of PSE, we checked the accuracy of the SeLECT score.[6]

The Thrombolysis in Cerebral Infarction (TICI) score (the subjects who had not undergone angiography were examined by MRI or CT angiography and assessed by two or more expert neurosurgeons) and bleeding complications were confirmed. The European Cooperative Acute Stroke Study classification was used to assess hemorrhagic transformation.[9]

The primary outcomes were as follows:

(1) The incidences of ASS and PSE and their significant differences between the IV-tPA + MT+, MT-only, and IV-tPA-only groups compared with the IV-tPA – MT– group.

(2) Predictors related to the risk of developing ASS and PSE.

This study was approved by the Ethics Committee of Ken-o Hospital (No. 000023).

Statistical analyses

Fisher's exact test was used to analyze categorical data, and Student's *t*-test or the Mann–Whitney *U*-test was used for nonparametric analysis of continuous data. The Kruskal–Wallis test or Fisher's exact test was used to perform multiple comparisons of the demographic, clinical, and risk characteristics for the control, MT, and IV-tPA groups. Logistic regression analysis and odds ratios were used to determine the predictors of ASS and PSE. EZR ver.1.54 (Jichi Medical University, Saitama Medical Center, Saitama, Japan) was used to perform statistical

analyses. Values of p <0.05 were treated as indicative of statistical significance.

Results

A total of 237 LVO subjects (64% male, median age = 82 years) were classified by TOAST as follows: A: n = 81, C: n = 143, D: n = 3, O: n = 4. The complications following MT were subarachnoid hemorrhage (SAH): n = 3 cases, dissection: n = 3, and cerebral bleeding: n = 1. SAH and dissection were mild enough to have no influence on the prognosis. For large cerebral infarctions, cerebral bleeding made evaluation of the effect on prognosis difficult.

ASS developed in 12 (5.1%) subjects as tonic–clonic seizures within 4 days from stroke onset, with 9 of the 12 occurring within 24 h. EEG and new MRI excluding the scheduled MRI were not performed for all of them. A case complicated with SAH during MT developed ASS; however, the cause was unknown because of its large infarction (70 ml) with cortical involvement. Antiepileptic drugs were prescribed (levetiracetam for nine subjects, lacosamide for one subject) continuously, and none transited into PSE. The frequency of PSE was 4.2% (n = 10), with a median delay of 210 days (interquartile range: 9–365 days). NCSE and tonic–clonic seizures were observed in five subjects each, none of them was status epilepticus without any special MRI findings. Only five subjects had undergone EEG, interictal epileptiform discharge was found in four subjects, and focal flattening without an epileptiform component was found in one subject.

The characteristics of the four groups (IV-tPA + MT group: 74 subjects, MT-only group: 82 subjects, IV-tPA-only group: 28 subjects, IV-tPA-MT– group: 53 subjects) are presented in **Table 1**. There were significant differences in high NIHSS (p = 0.01), low ASPECTS (p = 0.02) at admission, atrial fibrillation (p = 0.01), and occlusion vessel (middle cerebral artery, internal carotid artery) (p = 0.01) between the IV-tPA + MT group and MT-only group. However, the affected subjects gained good recanalization, with a TICI score >2c (p = 0.01), and there were no significant differences in the infarct volumes (ml), cortical location, and hemorrhagic transformation (>3) despite the low ASPECTS (p = 0.01) after 24 h. There were also no significant differences in the incidences of ASS (p = 0.43) and PSE (p = 0.75) among the four groups.

The univariate analysis identified ASPECTS <6 at admission and 24 h after (p <0.05, p = 0.02, respectively) as significant predictors of ASS (**Table 2**). On multivariate analysis, ASPECTS <6 24 h after remained as the only independent predictor of ASS (p = 0.01) (**Table 3**).

The univariate analysis also identified a median infarct volume of 75 (63–114) ml (p = 0.02) as a significant predictor of PSE (**Table 2**). The multivariate analysis with the best cutoff point of 54 ml (**Fig. 1**) identified an infarct volume >60 ml (p = 0.01) as the only independent predictor of PSE (**Table 3**). The SeLECT score: median 4 (3.3–4) was not significantly associated with the PSE of LVO (p = 0.13).

Discussion

Recently, advances in reperfusion therapy have improved the survival rate of cerebral infarction, and PSE that subsequently develops in 2%–14% of these patients[1–6] is responsible for 30%–50% of the increased incidence rate of elderly epilepsy.[10] ASS due to ischemic cerebral disease occurs in 3%–8% of patients with cerebral infarction,[1,4,5,11] and 35% of these patients experience recurrence as PSE.[12]

However, recent studies have suggested that reperfusion therapy that causes hemodynamic changes and hemorrhagic transformation might have an increased risk of epilepsy.[1–3] However, the results of our four-group comparison did not show a relationship between reperfusion therapy and either ASS (5.1%) or PSE (4.2%). Appropriate patient selection and postoperative care could result in a good reperfusion rate and good prognosis with few hemorrhagic complications.

Our multivariate analysis identified ASPECTS <6 at 24 h after as an independent predictor of ASS (p = 0.01). Previous studies have reported that higher NIHSS,[11] pneumonia, hemodynamic change, and hemorrhagic transformation[1] were associated with ASS. The reason that we did not find that NIHSS and ASPECTS at admission were significant predictors of ASS was, presumably, because our study included cases at the initial diagnosis that were mostly mild because of rapid emergency transport or they were recovery cases following treatment.

In our study, ASS did not lead to PSE, and the SeLECT score was not associated with the prediction of PSE. Although ASS is caused by an increase in cerebral excitability due to a usually reversible disturbance of cerebral homeostasis affecting the blood–brain barrier, ion-channel function, and neurotransmitter release, PSE is mostly caused by structural changes with chronic inflammation and gliosis.[13] Given that ASS has been previously recognized in 20% of acute basilar artery occlusion cases[14]

Table 1 Baseline demographics and risk factors of acute symptomatic seizures and post-stroke epilepsy across treatment groups (n = 237)

	MT+, IV-tPA+ n = 74	MT only n = 82	IV-tPA only n = 28	MT–, IV-tPA– n = 53 (control)	*p*-Value
Age (median, IQR)	83 (77–88)	82 (74–87)	79 (70–88)	81 (75–87)	0.5
Female gender (n, %)	31 (13.1)	25 (10.5)	10 (4.2)	19 (8)	0.53
Hypertension (n, %)	60 (25.3)	64 (27)	24 (10)	44 (18.6)	0.84
Diabetes mellitus (n, %)	7 (3)	10 (4.2)	6 (2.5)	13 (5.5)	0.08
Hyperlipidemia (n, %)	11 (4.6)	17 (7.2)	6 (2.5)	7 (3)	0.59
Atrial fibrillation (n, %)	59 (24.9)	43 (18.1)	15 (6.3)	17 (7.2)	0.01
NIHSS (median, IQR)					
At admission	15 (10–19)	16 (8–20)	7 (4–18)	6 (2–20)	0.01
3 months after	4 (2–16)	8 (2–15)	3 (1–10)	6 (1–20)	0.36
1 year after	3 (1–13)	6 (1–15)	2 (0–7)	4 (0–19)	0.34
ASPECTS (median, IQR)					
At admission	8 (6–9)	8 (6–9)	9 (8–10)	8 (6–9)	0.02
24 h after	6.5 (5–8)	7 (5–8)	8 (7–9)	8 (6–9)	0.01
Occlusion vessel (n, %)					
Middle cerebral artery (M1)	37 (15.6)	32 (13.5)	11 (4.6)	10 (4.2)	0.01
Middle cerebral artery (M2)	4 (1.7)	9 (3.8)	8 (3.4)	6 (2.5)	
Internal carotid artery	23 (9.7)	29 (12.2)	1 (0.4)	17 (7.2)	0.01
Anterior cerebral artery	0 (0)	0 (0)	2 (0.8)	0 (0)	
Posterior cerebral artery	2 (0.8)	1 (0.4)	4 (1.7)	5 (2.1)	
Vertebral artery	4 (1.7)	2 (0.8)	2 (0.8)	9 (3.8)	
Basilar artery	4 (1.7)	6 (2.5)	0 (0)	6 (2.5)	
TICI score (≥2c) (n, %)	46 (19.4)	59 (24.9)	14 (5.9)	11 (4.6)	0.01
Infarct volume (ml) (median, IQR)	21 (6–74)	24 (5–68)	11 (2–42)	12 (2–46)	0.16
Cortical location (n, %)	45 (19)	50 (21.1)	13 (5.5)	27 (11.4)	0.33
Hemorrhagic transformation (>3) (n, %)	11 (4.6)	18 (7.6)	5 (2.1)	4 (1.7)	0.22
SeLECT score (median, IQR)	3 (1–4)	3 (2–5)	2 (1–3.3)	3 (1–4)	0.38
mRS (0–2) 3 months after (n, %)	30 (12.7)	29 (12.2)	13 (5.5)	21 (8.9)	0.79
mRS (0–2) 1 year after (n, %)	36 (15.4)	33 (14.1)	14 (6)	24 (10.3)	0.7
Acute symptomatic seizure (n, %)	6 (2.5)	2 (0.8)	1 (0.4)	3 (1.3)	0.43
Post-stroke epilepsy (n, %)	2 (0.8)	5 (2.1)	1 (0.4)	2 (0.8)	0.75

p-Value of < 0.05 was determined significant.
ASPECTS: Alberta Stroke Programme Early CT score; IQR: interquartile range; IV-tPA: intravenous tissue-plasminogen activator; mRS: modified Rankin Scale; MT: mechanical thrombectomy; NIHSS: National Institutes of Health Stroke Scale; TICI: Thrombolysis in Cerebral Infarction

and 13.6% of posterior circulation ischemia cases,[15] we suggest that disorder of the brainstem reticular formation and the renticulocortical synchronization mechanisms may be the cause.[16] Moreover, the territory of the posterior artery (hippocampus, thalamus, and brainstem) is reportedly a risk factor for epilepsy.[5] The SeLECT score examining ASS, large-artery atherosclerotic etiology, and middle cerebral arterial territory involvement supposedly is difficult for predicting PSE following LVO, and often includes cardiogenic etiology or the posterior circulation territory.

Conversely, administering the latest generation antiepileptic drugs for most cases of ASS might prevent progression to PSE. The fact that all of them occurred as transient tonic–clonic seizures but not status epilepticus without any special MRI epileptic findings may also be the reason. According to the European guideline, prophylactic administration for PSE is generally not recommended without sufficient evidence; however, it is allowed for a high possible risk of PSE with ASS.[4] LVO cases often develop ASS because of the sudden disturbance of cerebral homeostasis, but they would not have a high risk of PSE if the cerebral infarctions became mild. If administration of antiepileptic drugs to patients is continued only to treat ASS, physicians should consider discontinuing their administration.

Given that our predictor for PSE was an infarct volume >60 ml ($p = 0.01$), which is in addition to previously reported[17] predictors of 30 cm^3, >50 × 50 mm, and >5 slices,[12] additional care is needed for large-size infarctions. Other reported predictors include ASS, higher NIHSS, cortical involvement,[5] hemorrhagic transformation, mRS ≥2 at 3 months post-stroke,[3] epilepsy >6 h,[17] and EEG abnormality.[18] Given that some NCSEs are often difficult

Table 2 Predictors for acute symptomatic seizures and post-stroke epilepsy

	Acute symptomatic seizure (n = 12)	*p*-Value	Post-stroke epilepsy (n = 10)	*p*-Value
Age (median, IQR)	85 (77–88)	0.62	78 (77–85)	0.76
Female gender (n, %)	6 (7.1)	0.36	1 (1.2)	0.1
Hypertension (n, %)	11 (5.7)	0.47	7 (3.6)	0.41
Diabetes mellitus (n, %)	3 (8.3)	0.4	2 (5.6)	0.65
Hyperlipidemia (n, %)	2 (4.9)	1	3 (7.3)	0.38
Atrial fibrillation (n, %)	8 (6)	0.56	8 (6)	0.19
NIHSS at admission (median, IQR)				
On admission	14 (10–20)	0.59	19 (13–21)	0.14
3 months after	19 (7–21)	–	10 (8–15)	0.11
1 year after	17 (4–20)	–	10 (4–15)	0.17
ASPECTS <6				
At admission (n, %)	5 (2.1)	<0.05	4 (1.7)	0.08
24 h after (n, %)	7 (3)	0.02	5 (2.1)	0.13
Occlusion (n, %)		0.82		0.93
Middle cerebral artery (M1)	3 (3.3)		4 (4.4)	
Middle cerebral artery (M2)	2 (7.4)		2 (7.4)	
Internal carotid artery	5 (7.1)		3 (4.3)	
Anterior cerebral artery	0 (0)		0 (0)	
Posterior cerebral artery	0 (0)		0 (0)	
Vertebral artery	1 (5.9)		1 (5.9)	
Basilar artery	1 (6.2)		0 (0)	
TICI (≥2c) (n, %)	6 (4.6)	0.77	6 (4.6)	1
Infarct volume (ml) (median, IQR)	55 (19–137)	0.09	75 (63–114)	0.02
Cortical location (n, %)	7 (5.2)	1	8 (5.9)	0.19
Hemorrhagic transformation (>3) (n, %)	3 (7.9)	0.42	3 (7.9)	0.2
mRS (0–2) 3 months after (n, %)	2 (2.2)	–	3 (3.2)	0.74
mRS (0–2) 1 year after (n, %)	2 (1.9)	–	3 (2.8)	0.35
SeLECT score (median, IQR)	–		4 (3.3–4)	0.13
Acute symptomatic seizure (n, %)	–		0 (0)	1
Seizure type (n)				
Tonic–clonic seizure	12 (100)		5 (50)	
Non-convulsive status epilepticus	0 (0)		5 (50)	
Electroencephalogram (n, %)	0 (0)		5 (41.7)	
Interictal epileptiform discharge	–		4 (8.3)	
Focal flattening	–		1 (41.7)	
Anti-seizure medication (n, drug) at 1 year post-discharge	11 (LEV) 1 (LAC)		9 (LEV) 1 (LEV + PER)	

p-Value of <0.05 was determined significant.
IQR: interquartile range; LAC: lacosamide; Lev: levetiracetam; PER: peranpanel

Table 3 Multivariate logistic regression for acute symptomatic seizures (n = 12) and post-stroke epilepsy (n = 10)

	Predictors	OR	95% CI	*p*-Value
Acute symptomatic seizure	ASPECTS <6 24 h after	6.3	1.6–24.5	0.01
Post-stroke epilepsy	Infarct volume >60 ml	11.9	2.5–57.8	0.01

p-Value of < 0.05 was determined significant.
ASPECTS: Alberta Stroke Program Early CT score; CI: confidence interval; OR: odds ratio

to diagnose, early diagnosis by EEG or neuroimaging findings and early treatment is important.

Limitations

A study limitation was the small sample size, which was the result of excluding deaths because most LVO cases were severe, and many of the patients died in the short term. Advances in the setup for swift MT, such as the stroke network and guidelines, may have caused bias in the baseline parameters among the four groups, such as age and ASPECTS at admission. Our clinically diagnosed seizures by non-confirmation with EEG had a possible risk

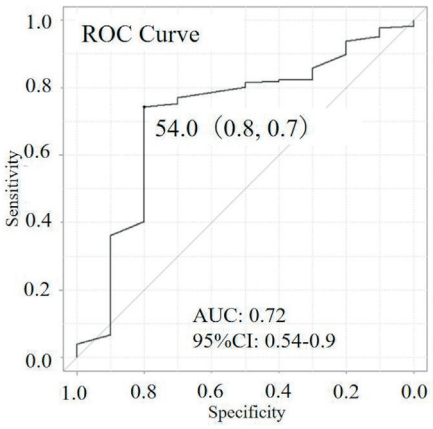

Fig. 1 Receiver operating characteristic (ROC) curve analysis of cerebral infarction volume (ml) for post-stroke epilepsy. The cutoff point was 54 ml. The area under the ROC curve (AUC) was 0.72 (95% confidence interval [CI]: 0.54–0.9).

of mistaken impaired consciousness seizure for cardiovascular disease. We also had a possible risk of missing NCSE from unconscious patients because we did not perform EEG monitoring over 24 h.

Conclusion

The study results did not show that reperfusion therapies, such as IV-tPA and MT, were associated with an increase in the incidence of ASS or PSE in patients with LVOs. Given that an improving survival rate might increase the risk of PSE, patients with large infarctions should be carefully monitored for PSE.

Disclosure Statement

The authors report no conflict of interest concerning the materials or methods used in this study or the findings reported in this paper.

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