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Lenticulostriate Artery Involvement is Predictive of Poor Outcomes in Superficial Middle Cerebral Artery Territory Infarction

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Purpose: Patients with superficial middle cerebral artery (MCA) territory infarction may have concomitant lenticulostriate artery (LSA) territory infarction. We investigated the mechanisms thereof and the outcomes of patients with superficial MCA territory infarction according to the presence or absence of LSA involvement.

Materials and Methods: Consecutive patients with first-ever infarction in the unilateral superficial MCA territory were included in this study. They were divided into the superficial MCA only (SM) group and the superficial MCA plus LSA (SM+L) group.

Results: Of the 398 patients, 84 patients (21.1%) had LSA involvement (SM+L group). The SM+L group more frequently had significant stenosis of the proximal MCA or carotid artery and high-risk cardioembolic sources. Stroke severity and outcomes were remarkably different between the groups. The SM+L group showed more severe neurologic deficits (National Institute of Health Stroke Scale score 10.8±7.1 vs. 4.0±5.0, p<0.001) and larger infarct in the superficial MCA territory (40.8±62.6 cm^3 vs. 10.8±21.8 cm^3, p<0.001) than the SM group. A poor functional outcome (mRS >2) at 3 months was more common in the SM+L group (64.3% vs. 15.9%, p<0.001). During a mean follow-up of 26 months, 67 patients died. All-cause (hazard ratio, 2.246) and stroke (hazard ratio, 9.193) mortalities were higher in the SM+L group than the SM group. In multivariate analyses, LSA involvement was an independent predictor of poor functional outcomes and stroke mortality.

Conclusion: LSA territory involvement is predictive of poor long-term outcomes in patients with superficial MCA territory infarction.

Key Words: Lenticulostriate artery, middle cerebral artery, cerebral infarction, prognosis, mortality

INTRODUCTION

The middle cerebral artery (MCA) is the most commonly affected artery in ischemic stroke.[1-3] The MCA territory can be divided into the superficial MCA territory and lenticulostriate artery (LSA) territory.[4] Patients with superficial MCA territory infarction often develop devastating or fatal stroke.[5,6]

Patients with superficial MCA territory infarction may show concomitant involvement of the LSA territory. The LSA commonly originates from the proximal segment (M1) of the MCA trunk or the superior division of the MCA.[7] Thus, the presence of concomitant LSA territory involvement in patients with superficial MCA territory infarction provides information on the relevant artery of infarction, useful for the determination of stroke mechanisms.[8,9] In addition, co-existing LSA involvement in patients with superficial MCA territory infarction may suggest 1) occlusion of the MCA by larger thrombi, which may result in larger MCA territorial infarctions; 2) more frequent involvement of the adjacent corticospinal tract, which can result in more severe or frequent motor weakness; and 3) a higher risk of brain herniation, because of deeply located sizable infarctions. All these factors may affect patient outcomes. Furthermore, stroke mechanisms may differ in patients with superficial

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MCA territory infarction, depending on the presence or absence of co-existing LSA territory infarction.

In the present study, we compared the mechanisms thereof, severities of stroke, and long-term outcomes in patients with the superficial MCA territory infarction in relation to the presence or absence of LSA involvement.

MATERIRALS AND METHODS

Study population and group

This was a retrospective observational study using a prospectively registered cohort data.[10] The present study included patients admitted between January 2010 and December 2012 with first-ever infarction involving the unilateral MCA superficial territory within 7 days of symptom onset. Patients who had co-exiting infarctions in other territories were excluded. The patients were evaluated using standardized protocols, including brain computed tomography (CT), magnetic resonance imaging with diffusion-weighted imaging (DWI), and cerebral angiographic studies, such as MR angiography, CT angiography, and digital subtraction angiography. Cardiac evaluation included transesophageal echocardiography (TEE), transthoracic echocardiography (TTE), heart CT scan, and Holter or continuous electrocardiography monitoring during patient stay in the stroke unit. The patients were managed using standardized protocols of our stroke center based on given guidelines.[11,12]

We divided the patients with superficial MCA territory infarction into two groups according to the presence of concomitant LSA territory infarction: the superficial MCA only (SM) group and the superficial MCA plus LSA (SM+L) group. The Institutional Review Board of Severance Hospital, Yonsei University Health System approved this study and waived the need for informed consent, owing to the retrospective and observational nature of the study.

Definition of risk factors

Hypertension was defined as blood pressure recordings of systolic ≥140 mm Hg or diastolic ≥90 mm Hg on repeated measurements during hospitalization or being on antihypertensive medication. Diabetes mellitus was diagnosed based on a fasting plasma glucose level ≥7 mmol/L, a glycated hemoglobin value ≥6.5%, or when patients had been treated with oral hypoglycemic agents or insulin. Hypercholesterolemia was defined as a fasting serum total cholesterol level ≥6.2 mmol/L, low-density lipoprotein-cholesterol >4.1 mmol/L, or being treated with lipid lowering agents after a diagnosis of hypercholesterolemia. Patients were regarded as current smokers if they smoked within 1 year prior to admission.

Mechanism of stroke

The mechanism of stroke was determined based on the Trial of Org 10172 in the Acute Stroke Treatment (TOAST) classification.[13] This study included patients with cardioembolism, large artery atherosclerosis, or stroke of undetermined etiology due to two or more identified causes or due to negative evaluation. Patients with other determined etiologies were excluded from the study. Significant stenosis of the MCA or internal carotid artery (ICA) was defined as stenosis >50% or occlusion of the artery relevant to the infarction. We identified high-risk potential cardiac source of embolism (PCSE) based on the TOAST classification. The stroke subtype and the degree of arterial stenosis were discussed during weekly stroke conferences and determined in consensus among stroke specialists. These data were registered in the prospective registry.

Infarction volume

The volume of infarction was measured on DWI in a semi-automatic manner using Xelis software (Infinitt, Seoul, Korea). An investigator (EHK) who was blinded to the clinical information measured the infarction volumes for the superficial MCA territory and those for the LSA territory. The infarction volume of the superficial MCA territory was compared between the groups.

Stroke severity and long-term outcomes

Stroke severity was determined on the basis of National Institute of Health Stroke Scale (NIHSS) at admission. Significant brain swelling was defined as brain edema that required osmotherapy or hemicraniectomy. Poor functional outcome was defined as a modified Rankin Scale (mRS) score >2 at 3 months after stroke onset. Long-term mortality and causes of death were identified by using death certificates from the Korean National Statistical Office, by reviewing medical records, and by conducting face-to-face or telephone interviews with a research nurse or stroke specialist. Mortality data from the Korean National Statistical Office are considered reliable, since they are collected on the basis of a unique 13-digit identification code assigned to subjects at birth and on the death certificate.[14] The cause of death was coded according to the International Classification of Disease (ICD), 10th revision. Stroke mortality was defined by the ICD code as I60–I69. Patients who were alive on August 31, 2014 were censored.

Statistical analyses

All statistical analyses were performed using SPSS software for Windows (version 20.0; SPSS Inc., Chicago, IL, USA). The Pearson χ^2 test was used to compare the frequency of categorical variables. The independent t-test was used to compare continuous variables. The variables achieving *p*-values less than 0.05 in the univariate analyses were entered for multivariate analyses using the logistic regression model to determine the factors associated with poor functional outcome. With regard to long-term mortality, Kaplan-Meier analysis was used to estimate survival, and the log-rank test was used to compare rate estimates. We tested the proportional-hazards assumption by including

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an interaction term between variables and natural log-transformed follow-up time. Also, we checked log-minus-log survival plots. All variables met a proportional-hazards assumption. Cox proportional hazard regression analysis was performed to calculate crude and adjusted hazard ratios (HR) with 95% confidence intervals (CI). All statistical analyses were two-tailed, and $p<0.05$ was considered statistically significant.

RESULTS

Demographic and clinical characteristics of the patients

During the study period, 1754 consecutive patients were registered to the prospective cohort. At first, 885 patients were excluded because they did not have any infarction in the superficial MCA territory. After excluding 180 patients with co-existing infarctions in other territories or bilateral superficial MCA territories, 689 patients had unilateral infarction involving the superficial MCA territory. Of the 689 patients, we excluded 291 patients who received thrombolytic treatment, had a history of previous cerebral infarction, had other determined etiology based on the TOAST classification, or did not undergo DWI or cerebral angiography. Finally, 398 patients were included in this study (Supplementary Fig. 1, only online).

Of the 398 patients with superficial MCA territory infarction, the mean age was 66.9±11.7 years and 273 (68.6%) were men. Of the 398 patients, 369 (92.7%) underwent at least one cardiac evaluation (heart CT in 278, TEE in 245, and TTE in 197), and 295 patients (74.1%) were evaluated using continuous electrocardiography monitoring for at least 24 hours. Eighty-four patients (21.1%) had concomitant lesions in the LSA territory (SM+L group). The SM+L group showed higher levels of white blood cells than the SM group. The other demographic characteristics were not different between the groups (Table 1).

Table 1. Comparison of Baseline Characteristics

	SM (n=314)	SM+L (n=84)	*p* value
Age, yr	66.5±11.4	68.6±13.0	0.148
Sex, men	219 (69.7)	54 (64.3)	0.338
Hypertension	222 (70.7)	60 (71.4)	0.896
Diabetes	136 (43.3)	34 (40.5)	0.641
Hypercholesterolemia	45 (14.3)	8 (9.5)	0.249
Current smoking	82 (26.1)	17 (20.2)	0.268
Coronary heart disease	74 (23.6)	19 (22.6)	0.855
Previous medication			
Antiplatelet	116 (36.9)	25 (29.8)	0.222
Anticoagulant	23 (7.3)	3 (3.6)	0.216
Statin	68 (21.7)	17 (20.2)	0.778
Antihypertensive	138 (43.9)	43 (51.2)	0.236
Diabetes medication	25 (8.0)	4 (4.8)	0.316
Initial NIHSS	4.02±4.97	10.83±7.11	<0.001
Significant brain swelling	10 (3.2)	13 (15.5)	<0.001
Laboratory results			
Hemoglobin, g/dL	13.9±2.1	14.2±1.8	0.157
Hematocrit, %	40.7±5.8	41.9±4.9	0.091
White blood cells, K/mm^3	8.0±2.8	9.1±3.2	0.005
Platelet, K/mm^3	245.5±84.2	247.9±73.6	0.818
Prothrombin time, INR	1.0±0.3	1.0±0.2	0.848
ESR, mm/H	25.8±23.5	25.9±21.7	0.976
hsCRP, mg/dL	11.3±29.5	22.5±50.0	0.057
Total cholesterol, mmol/L	4.7±1.1	4.6±1.1	0.453
LDL cholesterol, mmol/L	2.7±0.9	2.8±0.9	0.181
HDL cholesterol, mmol/L	1.1±0.3	1.1±0.3	0.928
Fasting sugar, mmol/L	6.6±2.4	6.6±2.1	0.907
Glycated hemoglobin, %	6.6±1.4	6.6±1.3	0.685

SM, superficial middle cerebral artery only; SM+L, superficial middle cerebral artery plus lenticulostriate artery; NIHSS, National Institutes of Health Stroke Scale; INR, international normalized ratio; ESR, erythrocyte sedimentation rate; hsCRP, high sensitivity C-reactive protein; LDL, low-density lipoprotein; HDL, high-density lipoprotein.
Data are number (%), mean±standard deviation.

Mechanism of infarction

Stroke subtypes were different between the groups. Large artery atherosclerosis and two or more causes were more common in the SM+L group. Negative evaluation was more frequent in the SM group (20.1%) than in the SM+L group (4.8%). Compared with the SM group, the SM+L group more commonly had significant stenosis of the MCA or ICA relevant to an infarction and high-risk PCSE (Table 2).

Table 2. Subtype and Stroke Mechanisms

	SM (n=314)	SM+L (n=84)	*p* value
Stroke subtype			0.003
Large artery atherosclerosis*	76 (24.2)	31 (36.9)	
Cardioembolism	109 (34.7)	27 (32.1)	
Two or more causes*	66 (21.0)	22 (26.2)	
Negative evaluation	63 (20.1)	4 (4.8)	
Stroke mechanism			
Significant MCA stenosis	43 (13.7)	33 (39.3)	<0.001
Significant ICA stenosis	45 (14.3)	28 (33.3)	<0.001
High-risk PCSE	84 (26.8)	35 (41.7)	0.008

SM, superficial MCA only; SM+L, superficial MCA plus lenticulostriate artery; MCA, middle cerebral artery; ICA, internal carotid artery, PCSE, potential cardiac source of embolism.
Values are numbers (%).
*p<0.005; comparison with negative evaluation using Bonferroni post-hoc analysis.

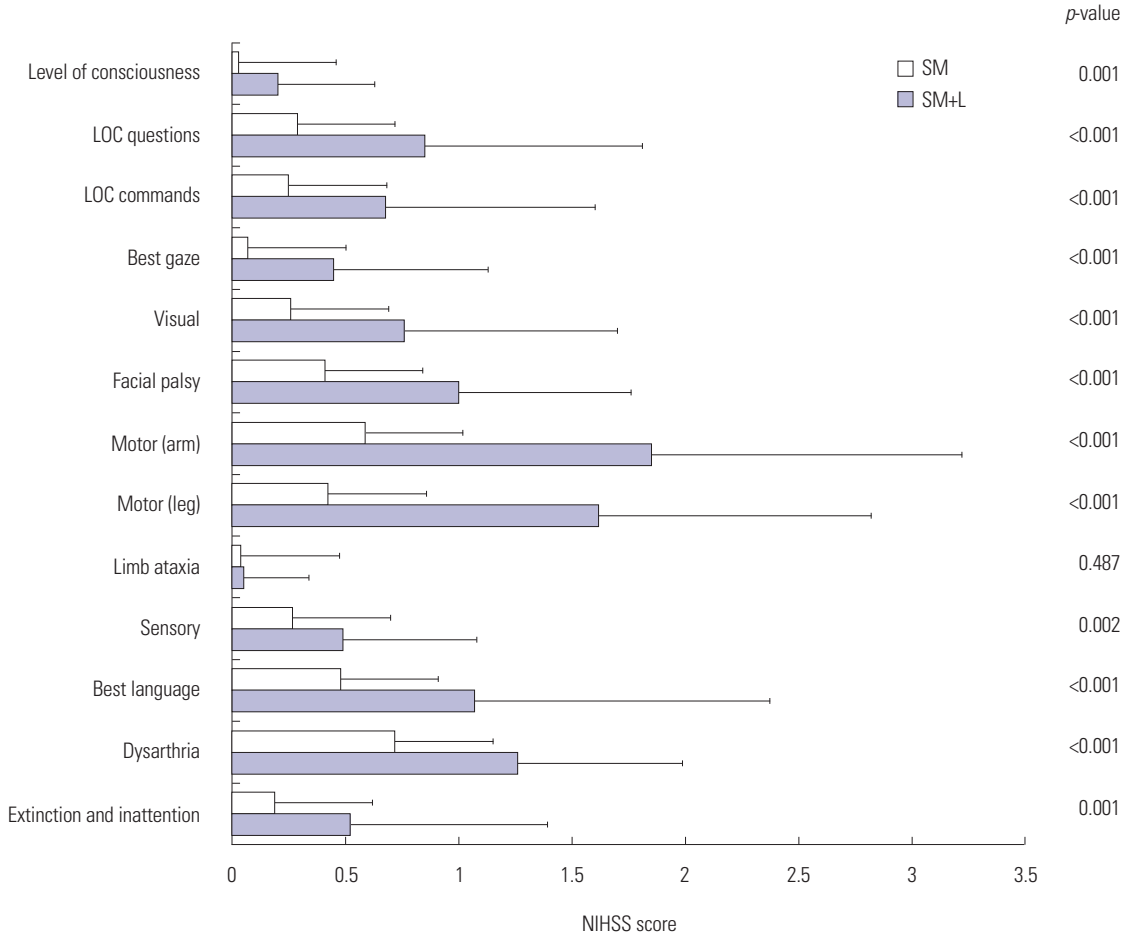

Fig. 1. Comparison of mean NIHSS scores for each item between the groups. Scores for all NIHSS items except that for limb ataxia are significantly higher in the SM+L group. NIHSS, National Institute of Health Stroke Scale; SM, superficial middle cerebral artery only; SM+L, superficial middle cerebral artery plus lenticulostriate artery; LOC, loss of consciousness.

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Initial stroke severity and infarct volumes

Initial neurologic deficits were more severe in the SM+L group than in the SM group (mean NIHSS, 10.83±7.11 vs. 4.02±4.97, p<0.001) (Table 1). We compared the scores of each NIHSS item between the groups to determine whether the higher NIHSS score in the SM+L group was due to symptoms related with LSA lesions, such as weakness, dysarthria, and sensory changes. In doing so, scores for all NIHSS items, except those for ataxia, were significantly higher in the SM+L group (Fig. 1). The infarct volume of the superficial MCA territory was larger in the SM+L group than in the SM group (40.8±62.6 cm^3 vs. 10.8±21.8 cm^3, p<0.001) (Supplementary Fig. 2, only online). Significant brain swelling was also more frequent in the SM+L group (15.5% vs. 3.2%, p<0.001) (Table 1).

Long-term outcomes

Poor functional outcome (mRS>2) at 3 months was more common in the SM+L group than in the SM group (64.3% vs. 15.9%, p<0.001) (Fig. 2). Univariate analysis showed that the patients with poor functional outcome were less commonly men, were

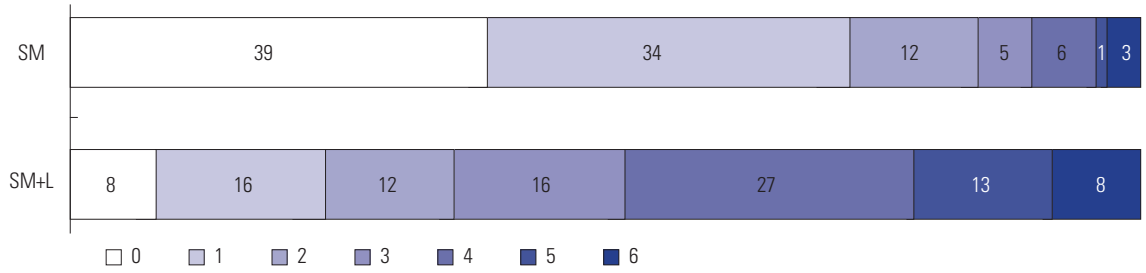

Fig. 2. Modified Rankin Scale at 3 months of symptom onset. Numbers are percentages (%). SM, superficial middle cerebral artery only; SM+L, superficial middle cerebral artery plus lenticulostriate artery.

Table 3. Univariate Analysis of Factors Associated with a Poor Outcome at 3 Months

	Patient with favorable outcome (n=294)	Patient with poor outcome (n=104)	*p* value
Age, yr	65.1±11.6	72.0±10.6	<0.001
Sex, men	210 (71.4)	63 (60.6)	0.040
LSA involvement	30 (10.2)	54 (51.9)	<0.001
Hypertension	204 (69.4)	78 (75.0)	0.279
Diabetes	124 (42.3)	46 (44.2)	0.716
Hypercholesterolemia	41 (13.9)	12 (11.5)	0.535
Current smoking	83 (28.2)	16 (15.4)	0.009
Coronary heart disease	71 (24.1)	22 (21.2)	0.535
Initial NIHSS	3.11±3.73	12.10±6.76	<0.001
Significant brain swelling	3 (1.0)	20 (19.2)	<0.001
Significant MCA stenosis	37 (12.6)	39 (37.5)	<0.001
Significant ICA stenosis	40 (13.6)	33 (31.7)	<0.001
High-risk PCSE	71 (24.1)	48 (46.2)	<0.001
Laboratory results			
Hemoglobin, g/dL	13.9±2.0	13.8±2.2	0.315
White blood cells, K/mm^3	7.8±2.6	9.3±3.4	<0.001
Platelet, K/mm^3	245.2±79.1	248.2±90.2	0.752
Prothrombin time, INR	1.0±0.3	1.0±0.2	0.827
ESR, mm/H	23.4±21.5	32.2±26.0	0.002
hsCRP, mg/dL	8.5±22.5	28.4±54.8	0.001
Total cholesterol, mmol/L	4.7±1.1	4.5±1.1	0.045
LDL cholesterol, mmol/L	2.7±0.8	2.8±1.0	0.575
HDL cholesterol, mmol/L	1.1±0.3	1.1±0.3	0.174
Fasting sugar, mmol/L	6.4±2.4	6.9±2.4	0.121
Glycated hemoglobin, %	6.6±1.4	6.6±1.3	0.971

LSA, lenticulostriate artery; NIHSS, National Institutes of Health Stroke Scale; MCA, middle cerebra artery; ICA, internal carotid artery; PCSE, potential cardiac source of embolism; INR, international normalized ratio; ESR, erythrocyte sedimentation rate; hsCRP, high sensitivity C-reactive protein; LDL, low-density lipoprotein; HDL, high-density lipoprotein.
Data are number (%), mean±standard deviation.

more commonly older age, and had more commonly LSA involvement, current smoking history, higher initial NIHSS score, significant brain swelling, significant MCA stenosis, significant ICA stenosis, high-risk PCSE, and higher white blood cell count, ESR, hsCRP and total cholesterol levels (Table 3). Multivariate logistic regression analysis showed that LSA involvement was an independent predictor of poor functional outcome (adjusted odds ratio 3.686, 95% CI 1.693–8.027, p= 0.001), along with age, initial NIHSS, significant stenosis of the MCA or ICA relevant to an infarction, and the infarct volume of the superficial MCA territory (Table 4).

During a follow-up of mean 26±15 months, 67 patients (16.8%) died. Eighteen patients (4.5%) died of stroke. The SM+L group showed significantly higher all-cause mortality (HR, 2.246; 95% CI, 1.339–3.768) (Fig. 3A) and stroke mortality (HR, 9.193; 95% CI, 3.442–24.556) (Fig. 3B) than the SM group. Multivariate Cox regression analysis, after adjusting factors that were significant on univariate Cox regression analysis (Supplementary Table 1, only online), showed that LSA involvement was independently associated with long-term stroke mortality along with age, higher initial NIHSS, significant brain swelling, and significant stenosis of the MCA trunk (Table 5). Meanwhile, however, multivariate Cox regression analysis (Supplementary Table 2, only online), revealed that LSA involvement was not independently associated with all-cause mortality.

DISCUSSION

In the present study, we investigated whether initial stroke severity, stroke mechanism, and long-term outcomes differed among patients with superficial MCA territory infarction according to the presence or absence of co-existing LSA territory infarction. We found that initial neurologic deficits were more severe in patients with SM+L group than in those with SM alone. LSA involvement can cause weakness, sensory changes, or dysarthria,[15,16] and these symptoms might have contributed to the severe neurologic deficits in the SM+L group. However, when baseline NIHSS scores for each item were compared between the groups, all of the items except for ataxia showed higher

Table 4. Multivariate Analysis of Factors Associated with a Poor Outcome at 3 Months

	Adjusted odds ratio	95% CI	*p* value
Age, per increment of 10	2.048	1.422–2.952	<0.001
Sex, men	0.700	0.335–1.463	0.343
Initial NIHSS score	1.203	1.129–1.292	<0.001
Superficial MCA territorial infarction volume, per increment of 1 cm^3	1.016	1.002–1.032	0.031
Significant brain swelling	4.903	0.743–32.362	0.099
Significant MCA stenosis	2.868	1.209–6.806	0.017
Significant ICA stenosis	3.707	1.617–8.498	0.002
High-risk PCSE	1.514	0.702–3.264	0.290
White blood cells, per increment of 1000	1.105	0.981–1.244	0.101
Involvement of LSA territory	3.686	1.693–8.027	0.001

NIHSS, National Institute of Health Stroke Scale; MCA, middle cerebral artery; ICA, internal carotid artery; PCSE, potential cardiac source of embolism; LSA, lenticulostriate artery; CI, confidence interval.

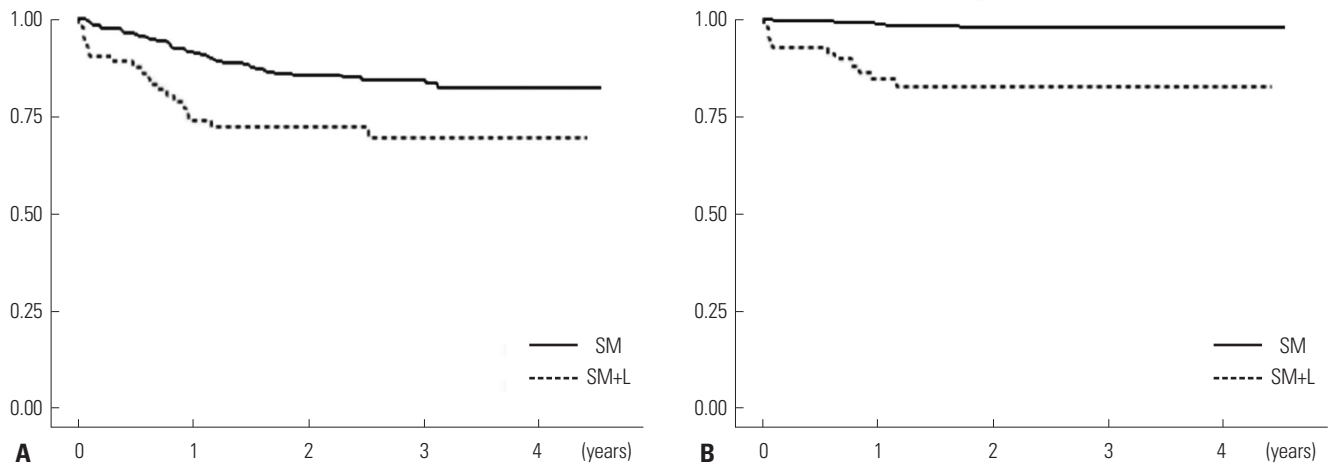

Fig. 3. Kaplan-Meier curve for all-cause mortality (A) and stroke mortality (B). SM, superficial middle cerebral artery only; SM+L, superficial middle cerebral artery plus lenticulostriate artery.

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Table 5. Cox Regression Model of Stroke Mortality in Multivariate Analysis

	Model 1		Model 2	
	HR (95% CI)	*p* value	HR (95% CI)	*p* value
Age, yr, per increment of 10	2.114 (1.300–3.537)	0.003	1.983 (1.193–3.297)	0.008
Sex, men	3.231 (0.946–11.042)	0.061	2.009 (0.632–6.383)	0.237
Involvement of LSA territory	3.652 (1.071–12.454)	0.038	4.230 (1.394–12.838)	0.011
Erythrocyte sedimentation rate	1.008 (0.998–1.028)	0.431		
Initial NIHSS score	1.117 (1.020–1.222)	0.016		
Superficial MCA territorial infarction volume, per increment of 1 cm^3	1.000 (0.990–1.011)	0.958		
Significant brain swelling			3.302 (1.094–9.961)	0.034
Significant MCA stenosis			3.176 (1.069–9.441)	0.038
Significant ICA stenosis			3.108 (0.992–9.742)	0.052

LSA, lenticulostriate artery; NIHSS, National Institute of Health Stroke Scale; MCA, middle cerebral artery; ICA, internal carotid artery.
Model 1 includes age, sex, erythrocyte sedimentation rate, initial NIHSS, superficial territorial infarction volume and involvement of LSA and model 2 includes age, sex, significant brain swelling, significant MCA stenosis, significant ICA stenosis and involvement of LSA.

scores in the SM+L group. These results suggest that the severe neurologic deficits in SM+L patients may be attributed to the wider hemispheric dysfunction, not simply the consequence of the additional LSA territory infarction. Consistent with this hypothesis, the infarction volume of the MCA superficial territory excluding that in the LSA territory was much greater in SM+L patients than in SM patients.

The initially severe stroke in the SM+L group could be, in part, ascribed to differences in the stroke mechanisms between SM and SM+L. In the present study, the SM+L group more frequently had significant stenosis or occlusion in the MCA or ICA and high-risk PCSE than the SM group. The larger infarction volume in the SM+L group might be associated with those etiologies. The possible mechanisms of superficial MCA territory infarction are embolism from the heart, artery-to-artery embolism from the proximal arterial trees, such as the aorta, carotid artery and MCA, and in-situ thrombosis of the MCA,[17,18] To produce the concomitant LSA lesion, thrombi from the heart or proximal arteries should be large enough to occlude the MCA trunk or multiple to involve both the superficial MCA and LSA territories.[19,20] In patients with significant stenosis of the MCA, thromboemboli from the heart or proximal arteries might have been more easily trapped at the MCA trunk to completely occlude the MCA.[21] In addition, cardioembolism is known to produce larger infarction and more severe stroke.[22,23] Finally, the local MCA trunk atheroma could also be the source of the emboli to both the LSA and the superficial MCA branches.

In this study, the presence of LSA territory infarction was an independent predictor of poor long-term outcomes and increased risk of mortality, even after adjustment for the initial NIHSS score and infarction volume. Functional outcome measured by mRS, largely depends on the degree of motor weakness.[24] Motor tracts, which pass as compact bundles through the internal capsule,[25] could be more frequently damaged in the SM+L group, and this might have caused more severe motor weakness and impeded functional recovery. In patients with large superficial territory infarction, additionally sizable infarctions in the subcortex due to LSA involvement might increase the risk of compressing deep brain structures and brain herniation. In a previous study, patients who had both large artery atherosclerosis and cardioembolism showed very high long-term mortality, while those without showed very low long-term mortality.[26] Consistent with the previous findings, the SM+L group more frequently had two or more causes (large artery atherosclerosis and cardioembolism), while the SM group more frequently had undetermined etiology due to negative evaluation.

This study has limitations. First, this study is not free from selection bias because this was a retrospective study, although the cohort data were prospectively and consecutively collected. Second, patients who received reperfusion therapy were excluded from this study. Consequently, some patients with hyperacute MCA infarction were not included in this analysis. Third, the mortality rate of this study is relatively low, compared to the previous studies.[27,28] We excluded patients with co-existing infarctions in other territories or those with recanalization therapy. These patients might have initially more severe stroke that may be associated with stroke mortality. In addition, cause of death was determined based on medical records or death certificates in some patients. Thus, stroke mortality might not be accurately reflected in some patients. Finally, although the infarction volume in the superficial MCA territory can be influenced by the presence and degree of collateral circulations,[20,29] they could not be determined because the angiographic studies were not standardized.

In conclusion, among patients with superficial MCA territory infarction, the presence of co-existing LSA territory infarction was associated with initially severe stroke and larger infarct volume in the MCA superficial territory. LSA involvement was almost the most powerful predictor of poor functional outcome and stroke mortality. Therefore, greater concern should be given when treating patients with MCA superficial territory infarction and a co-existing LSA lesion.

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