

Relationship between the ischemic stroke subtypes and risk factors included clinical outcome from Prasat Neurological Institute stroke registry

**Krida Na Songkhla, MD.,
Tasanee Tantirittisak, MD.,
Suchat Hanchaiphiboolkul, MD.,
Yodkhwan Wattanasen, MD.**
Prasat Neurological Institute

Corresponding author:

Krida Na Songkhla, MD.
Email: krida009@yahoo.com

Background and Purpose – The etiologies of ischemic stroke affect prognosis, outcomes and treatments of patients. The objective of this study is to look for the differences in risk factors, stroke severities, complications, treatments and prognosis in each stroke subtype.

Methods – We prospectively studied 140 consecutive patients with acute ischemic stroke [37.9% women, median age of 61.5 (53–72) years] from June, 2013 to August, 2013. All patients were categorized by TOAST (Trial of Org 10172 in Acute Stroke Treatment) stroke subtypes, based on the MRI Imaging; Small vessel Atherosclerosis (SAO), Large vessel Atherosclerosis (LAA), Cardiac Embolism (CE), Other Determined etiology (OD) and other Undetermined etiology (UND).

Results – This study found that the age, severity, complications, treatment strategies, costs and outcomes were significantly different in each subtype of stroke ($p < 0.001$). SAO (45%) was most often found in our study. Patients with CE were older (68.5 years old) than those with other subtypes and patients with OD were the youngest (mean age of 39). Prevalence of obesity (33.3%), complications and also higher cost were found more common in patients with CE than those of SAO and LAA. The treatments with intravenous thrombolytic (11.1%) and anticoagulant (83.3%) were prescribed more common in patients with CE. The good clinical outcome (mRS 0–2) was more often found in SAO subtype than in LAA and CE subtype.

Conclusions – Risk factor profiles, treatment, clinical outcome and prognosis of each stroke subtype are different. Treatments and prevention should be optimized for each subtype.

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Key words: Stroke, Stroke subtypes, risk factors

Introduction

Stroke registry is a valuable tool for obtaining information on epidemiology, clinical course, and diagnostic evaluation of stroke patients and can help to assess the efficacy of their treatments and functional outcomes. Prasat Neurological Institute has created stroke registry since June 2013 to collect the database of all patients with acute/ subacute ischemic stroke and transient ischemic attack (TIA) who were treated at Prasat Neurological Institute (PNI).

The important etiologies of ischemic stroke are large-artery atherosclerosis (macroangiopathy), cardioembolism, and cerebral small-vessel disease (microangiopathy). Other causes of ischemic stroke are cervical artery dissection, cerebral vasculitis, coagulopathies, hematologic disorders, and others. The differences in causes may affect the differences in severity, treatments and outcomes of the patients. However, the relationships of stroke risk factors, clinical course and stroke subtypes are varies in many Asian studies.⁽¹⁻³⁾ This study aims to determine the relationships of risk factors, severity, acute treatment strategies, complications and clinical outcomes in each stroke subtype.

Subjects and Methods

One hundred and forty patients with acute stroke who had stroke symptoms within 7 days were prospectively studied from 1st June, 2013 to 31th August, 2013. All of the patients received MRI brain examination within 72 hours after admission. Carotid duplex sonography, an ECG and/or ECG monitoring, basic blood tests, and additional laboratory investigations were done as required. Each patient was classified into one of five major ischemic stroke subtypes by using TOAST (Trial of Org 10172 in Acute Stroke Treatment) criteria based on the MRI imaging into Small vessel Atherosclerosis (SAO), Large vessel Atherosclerosis (LAA), Cardiac Embolism (CE), Other Determined etiology (OD), other Undetermined etiology (UND) by the agreement of a radiologist and a neurologist. Severity of stroke was evaluated by the National Institutes of Health Stroke

Scale (NIHSS). The clinical outcome was evaluated by the modified Rankin Scale (mRS) and Barthel Index Scale (BI) at three months follow up.

Data Evaluation and Statistics

Continuous variables are presented as median and interquartile. Categorical variables are presented as percentages. Comparisons of categorical variables between all subtypes were analyzed with Fisher's Exact Test. Kruskal-Wallis Test was used to determine the association of these factors with the ischemic stroke subtypes.

Results

A total of 140 ischemic stroke patients who were admitted during June,2013 to August, 2013 were included in this study. Of these patients, 53 were women and 87 were men, and had median age of 61.5 years (interquartile range 53-72). [Table 1] Small vessel atherosclerosis (SAO) was the most common cause of stroke (n=63, 45%), followed by LAA (n=48, 34.3%), CE (n=18, 12.9%), OD 6 (4.3%), and UND 5 (3.6%) respectively. Age distribution differed significantly between etiologic subtypes (p<0.001). CE, LAA, SAO had higher median age (68.5, 62, 60 years respectively) however UND and OD had lower median age (49, 39 years). [Table 2]

Baseline characteristics of the patients classified by stroke subtypes were presented in Table 2. Patients with LAA and OD had more severe neurological deficit (median NIHSS of 7) than those with SAO (median NIHSS of 4) and UND (median NIHSS of 3). Within the first week, brain edema and secondary parenchymal hemorrhage was more commonly found in CE (22.2%). Pneumonia and upper gastro-intestinal bleeding were more prevalent in OD (16.7%). [Table 3] The median cost of treatment was highest in CE (42,830 baht) and lowest in SAO (25,210 baht). The good outcome at 90 days follow up (mRS 0 to 2) was most frequently found in patients with UND (100%) and SAO (95.2%), whereas a poor outcome (mRS 4 to 5) was prevalent in patients with OD (50%), followed by those with CE (29.4%) and LAA (16.7%).

Intravenous thrombolysis was given in 5% of the patients. CE was the most common stroke subtype that received intravenous rt-PA (11.1%) followed by LAA (8.3%) and SAO (1.6%). Low molecular weight heparin (LMWH) was most frequently administered in patients

with CE (83.3%) and OD (50%) but rarely prescribed in SAO (1.6%). Oral anticoagulant for long-term secondary prevention was administered in patients with CE (94.4%). Antiplatelet was subscribed in all patients with LAA, SAO, OD, and UND for secondary prevention. [Table 4]

Table 1 Baseline characteristics of patients in our study

	Male (n=87)	Female (n=53)	Total
Age, years, median (interquartile range)	60 (53-72)	63 (51-72)	140
Previous stroke, (%)	27.6	22.6	36
Previous TIA, (%)	8	0	7
Hypertension, (%)	64.4	67.9	92
Diabetes, (%)	34.5	30.2	46
Dyslipidemia, (%)	57.5	77.4	91
Current smoking, (%)	64.4	26.4	70
Daily alcohol consumed, (%)	52.9	17	55
Snoring, (%)	56.3	28.3	64
Coronary heart disease, (%)	6.9	5.7	9
Cardiac arrhythmia, (%)	12.6	11.3	17
Internal carotid stenosis, (%)	6.9	3.8	8
BMI, (kg/m ²)			
≤ 22.9	40.2	34	53
23-24.9	22.9	35.8	45
≥25	29.9	30.2	42
TOAST classification*			
LAA (%)	70.8	29.2	48
CE (%)	66.7	33.3	18
SAO (%)	55.6	44.4	63
OD (%)	50	50	6
UND (%)	60	40	5

(*Small vessel Atherosclerosis (SAO), Large vessel Atherosclerosis (LAA), Cardiac Embolism (CE), Other Determined etiology (OD) and other Undetermined etiology (UND))

Table 2 Relationship between risk factors, severity at baseline and stroke subtypes

Etiology of ischemic stroke*							
Parameter	LAA n =48	CE n=18	SAO n =63	OD n =6	UND n=5	Total n =140	P
Total group (%)	34.3	12.9	45	4.3	3.6	100	---
Female (%)	29.2	33.3	44.4	50	40	37.9	0.498
Male (%)	70.8	66.7	55.6	50	60	62.1	
Age, y (interquartiles Range)	62 (54.25-74.0)	68.5(51.25-71.5)	60 (55-72)	49 (48-66)	39 (31-66)	61.5(53-72)	<0.001
Previous stroke (%)	22.9	27.8	27	33.3	20	25.7	0.948
Previous TIA (%)	6.2	0	3.2	33.3	0	5	0.086
Hypertension (%)	79.2	66.7	57.1	66.7	40	65.7	0.089
Diabetes (%)	39.6	33.3	30.2	33.3	0	32.9	0.510
Dyslipidemia (%)	62.5	61.1	68.3	50.0	80	65	0.808
Current smoking (%)	60.4	33.3	44.4	66.7	60	50	0.220
Daily alcohol consumed (%)	35.4	50	38.1	33.3	60	39.3	0.689
Snoring (%)	41.7	55.6	46.0	16.7	80	45.7	0.272
Coronary heart disease (%)	8.2	11.1	4.8	0	0	6.4	0.748
Cardiac arrhythmia (%)	0	88.9	1.6	0	0	12.1	< 0.001
Internal carotid stenosis (%)	14.6	0	1.6	0	0	5.7	0.059
BMI(kg/m ²)							
≤ 22.9	27.1	44.4)	39.7	66.7	60	37.9	0.419
23-24.9	41.7	22.2	28.6	16.7	40	32.1	
≥25	31.2	33.3	31.7	16.7	0	30	
NIHSS on admission (median; interquartiles range)	7 (5-10)	6.5 (5-11)	4 (2-4)	7 (2.8, 18.5)	3 (2-3)	5(3-7.8)	< 0.001
mRS on admission (median; interquartiles range)	4 (3-4)	4 (3-4)	2.6 (2-3)	4 (2-4)	3 (1.5-3)	3(2.3-4)	< 0.001
BI on admission (median; interquartiles range)	62.5 (45-80)	67.5 (48.8-76.3)	85 (70-90)	52.5 (36.3-92.5)	90 (90-90)	75(60-90)	< 0.001

(*Small vessel Atherosclerosis (SAO), Large vessel Atherosclerosis (LAA), Cardiac Embolism (CE), Other Determined etiology (OD) and other Undetermined etiology (UND))

Table 3 Length of hospital stay, complications, cost and outcomes

Parameter	LAA n=48	CE n=18	SAO n=63	OD n=6	UND n=5	Total n=140	P	
Length of stay (median; interquartiles range) d	5 (4-7)	8 (5-12)	4 (3-6)	8.5 (4.7-15.5)	5 (4.5-5)	5(4-7)	0.001	
Complication within 7 d, n (%)								
Brain edema, n (%)	0	22.2	0	0	0	2.9	0.001	
Hemorrhagic transformation, n (%)	2.1	22.2	0	0	0	3.6	0.004	
GI bleeding, n (%)	8.3	5.6	0	16.7	0	4.3	0.051	
Pneumonia, n (%)	6.2	11.1	0	16.7	0	4.3	0.038	
UTI, n (%)	6.2	0	3.2	0	20	4.3	0.303	
MI, n (%)	0	5.6	0	0	0	0.7	0.207	
Cost, baht (interquartiles Range)	31,661 (25,666- 40,046)	42,831 (31,714- 60,715)	25,210 (21,623- 30,165)	34,927 (26,963- 48,163)	27,192 (26,285- 29,834)	29,338 (23,486- 37,938)	< 0.001	
Follow up 90 d								
mRS	0-2, n (%)	66.7	47.1	95.2	33.3	100	77	< 0.001
	3, n (%)	16.7	23.5	4.8	16.7	0	11.5	
	4-5, n (%)	16.7	29.4	0	50	0	11.5	
BI	<25, n (%)	2.1	0	0	0	0	1	0.001
	25-95, n (%)	68.8	94.1	47.6	66.7	20	60.4	
	>95, n (%)	29.2	5.9	52.4	33.3	80	38.8	

(*Small vessel Atherosclerosis (SAO), Large vessel Atherosclerosis (LAA), Cardiac Embolism (CE), Other Determined etiology (OD) and other Undetermined etiology (UND))

Table 4 Association between treatments and ischemic stroke subtypes

Etiology of ischemic stroke							
	LAA n =48	CE n =18	SAO n =63	OD n =6	UND n =5	Total n =140	P value
Previous treatments							
antiplatelet, %	100	60	95	100	100	92.1	0.14
warfarin, %	0	11.1	1.6	0	0	2.1	
Acute treatments							
tPA, %	8.3	11.1	1.6	0	0	5	<0.001
LMWH, %	0	83.3	1.6	50	0	13.6	
antiplatelet, %	91.7	5.6	96.8	50	100	81.4	
Secondary prevention							
antiplatelet, %	100	0	100	100	100	87.3	<0.001
warfarin, %	0	94.4	0	0	0	12.1	

(*Small vessel Atherosclerosis (SAO), Large vessel Atherosclerosis (LAA), Cardiac Embolism (CE), Other Determined etiology (OD) and other Undetermined etiology (UND))

Discussion

By TOAST classification (Trial of Org 10172 in Acute Stroke Treatment)⁽⁴⁾, this prospective study found that 45% of ischemic stroke was SAO subtype which is the dominant subtype of ischemic stroke in Thailand^(5, 6) whereas in Western countries, large thromboembolic stroke is more prevalent.^(1, 2, 7-14) Age and atrial fibrillation were significant differences among stroke subtypes ($p < 0.001$). The patients with CE subtype had atrial fibrillation and were older which was quite similar as compared to previous studies.^(8, 15) In cases of DWI/MRI image who showed multiple restriction of diffusion spots at bilateral hemisphere, however there were no cardioembolic risk factors or other cause, despite the extensive evaluations such as routine EKG monitoring, transthoracic echocardiography (TTE) with/without saline bubble test, transesophageal echocardiography (TEE), were usually classified into UND subtype. The prevalence of UND subtype in our study was 3.6%, similar to Siriraj series 4.1%⁽⁶⁾. 3 / 5 of UND patients

were young and no definite causes of stroke were found despite complete investigations. The other 2 UND patients had emboli at bilateral hemisphere on DWI/MRI and no causes had been found. However, continuous cardiac telemetry monitoring⁽¹⁶⁾ should benefit to detect paroxysmal atrial fibrillation (PAF) in cryptogenic stroke. Hypertension is a major risk factor for all stroke subtype.^(2, 7, 12, 15, 17) some previous studies reported that patients with SAO subtype tend to higher prevalence of hypertension.^(7, 12, 15) However, the present study found that the prevalence of hypertension is not different among stroke subtypes which similar to some previous studies^(11, 18). A systematic review⁽¹⁹⁾ concluded that the excess of hypertension in lacunar stroke compared with nonlacunar stroke was mainly due to bias because many of the studies included hypertension in the definition of ischemic stroke subtypes. Diabetes is another stroke risk factor which can cause elevated coagulation factors, hyperinsulinemia and depletion of microvascular neuroprotection.^(20, 21) Dyslipidemia is one of the most

important risk factors for vascular disease,⁽²⁰⁾ more common in cardiovascular disease (CAD) than cerebrovascular disease (CVD). There are strong associations of large vessel atherosclerosis and low HDL cholesterol levels. Higher HDL cholesterol levels decreased risk of ischemic stroke and the lowest levels of total cholesterol associated with increased risk of hemorrhagic stroke.⁽²²⁻²⁴⁾ However, our study was not found any difference of the prevalence of diabetes and dyslipidemia among stroke subtypes. Smoking is a strong independent risk factor for ischemic stroke.⁽²⁵⁻²⁷⁾ Our study showed, smoking was less prevalent in patients with CE, similar to previous study.^(9, 12) The relationship between alcohol consumption and ischemic stroke is uncertain.⁽²⁸⁾ Risk of stroke may increase in the hour after alcohol ingestion.⁽²⁹⁾ Similar to a study,⁽¹⁹⁾ we found that alcohol consumption was not associated to stroke subtypes. Although snoring is a risk factor for stroke through hemodynamic and hematologic changes,⁽³⁰⁾ present study was not found the relationship of snoring and stroke subtypes. Carotid stenosis (50-75% stenosis by flow criteria) was found 14.6% for LAA subtype. 1.6% of patients with SAO had asymptomatic carotid stenosis. The Body Mass Index (BMI) is a measure for human body shape based on an individual's mass and height which has the international variation. For Thai people, BMI ≤ 22.9 , 23-24.9, ≥ 25 kg/m² is used to define normal, overweight and obesity, respectively. Some studies reported the association between increased BMI and risk of stroke.^(31, 32) However, BMI was not associated to stroke subtypes in present study.

Severity, complications, length of stay (LOS), cost and outcome were significant different ($p < 0.05$) among stroke subtypes in our study. Several studies showed that patients with CE subtype had more severe stroke than others⁽³³⁻³⁵⁾. However, present study revealed median NIHSS on admission of LAA, CE and OD subtype were similar (7, 6.5, 7 respectively) however, more than those of SAO and UND subtype (4, 3 respectively). The prevalence of OD subtype in our study was 4.3% (6 patients; internal carotid artery dissection in 3 patients, vertebral artery dissection in 2 patients, Takayasu in a

patient). All of OD stroke subtype patients in our study were extracranial large arteries disease which gave more severities. The present study found that brain edema and hemorrhagic transformation were significant different among stroke subtypes and more frequent in CE subtype. The pathological mechanisms are due to large infarction and recanalization after occlusion of large vessels.^(36, 37) Severity of stroke was a risk factor for medical complications.^(38, 39) Similarly, the present study showed that pneumonia was found more common in patients with CE and OD subtype (11.1, 16.7% respectively) which was significant difference among stroke subtypes ($p = 0.038$). Cost of treatment and LOS were also significant difference among stroke subtypes and depended on the severity of stroke, least in SAO subtype which was similar to previous study.⁽⁴⁰⁾ To access primary outcome at 90 days we used standard neurological score as BI, mRS to evaluate independence ($mRS \leq 2$, $BI > 95$), and dependence ($mRS \geq 3$, $BI \leq 95$).^(15, 42) Similarly, the present result showed significant different outcome among stroke subtype, patients with SAO and UND subtype which had less severe stroke on admission had more proportion of independence.^(15, 40)

Acute treatments and secondary prevention were significant difference among stroke subtypes ($p < 0.001$). For acute treatment, thrombolytic treatment was frequently used in CE, LAA subtype (11.1, 8.3 % respectively). Anticoagulant was prescribed in the highest rate of in those with CE which represented good quality treatment strategies.

In conclusion, our result suggested that age, severity, treatment, clinical outcome of each ischemic stroke subtypes are not the same. Treatment as well as prevention should be optimized for each subtype. However, further studies with more sample size may give more information.

References

1. Moussouttas M, Aguilar L, Fuentes K, et al. Cerebrovascular disease among patients from the Indian subcontinent. *Neurology* 2006;67:894–896.
2. Gunarathne A, Patel JV, Gammon B, et al. Ischemic stroke in South Asians: a review of the epidemiology, pathophysiology, and ethnicity-related clinical features. *Stroke* 2009;40:e415–423.
3. Turin TC, Kita Y, Rumana N, et al. Ischemic stroke subtypes in a Japanese population: Takashima Stroke Registry, 1988–2004. *Stroke* 2010;41:1871–1876.
4. Adams HP, Jr., Bendixen BH, Kappelle LJ, et al. Classification of subtype of acute ischemic stroke. Definitions for use in a multicenter clinical trial. TOAST. Trial of Org 10172 in Acute Stroke Treatment. *Stroke* 1993;24:35–41.
5. Hanchaiphiboolkul S. Risk factors for early infection after an acute cerebral infarction. *J Med Assoc Thai* 2005;88:150–155.
6. Chinwatanakul S, Boonyapisit K, Pornsriniyom D, Proyoonwiwat N, Senanarong V, Chaisevikul R, et al. Siriraj Acute Stroke Unit: 10 years experience. *J Med Assoc Thai* 2012;95 Suppl 2:S235–244.
7. Kolominsky-Rabas PL, Weber M, Gefeller O, et al. Epidemiology of ischemic stroke subtypes according to TOAST criteria: incidence, recurrence, and long-term survival in ischemic stroke subtypes: a population-based study. *Stroke* 2001;32:2735–2740.
8. Grau AJ, Weimar C, Buggle F, et al. Risk factors, outcome, and treatment in subtypes of ischemic stroke: the German stroke data bank. *Stroke* 2001;32:2559–2566.
9. Murat Sumer M, Erturk O. Ischemic stroke subtypes: risk factors, functional outcome and recurrence. *Neurol Sci* 2002;22:449–454.
10. White H, Boden-Albala B, Wang C, et al. Ischemic stroke subtype incidence among whites, blacks, and Hispanics: the Northern Manhattan Study. *Circulation* 2005;111:1327–1331.
11. Ohira T, Shahar E, Chambless LE, et al. Risk factors for ischemic stroke subtypes: the Atherosclerosis Risk in Communities study. *Stroke* 2006;37:2493–2498.
12. Bejot Y, Caillier M, Ben Salem D, et al. Ischaemic stroke subtypes and associated risk factors: a French population based study. *J Neurol Neurosurg Psychiatry* 2008;79:1344–1348.
13. Michel P, Odier C, Rutgers M, et al. The Acute Stroke Registry and Analysis of Lausanne (ASTRAL): design and baseline analysis of an ischemic stroke registry including acute multimodal imaging. *Stroke* 2010;41:2491–2498.
14. Ihle-Hansen H, Thommessen B, Wyller TB, et al. Risk factors for and incidence of subtypes of ischemic stroke. *Functional neurology*. 2012;27:35–40.
15. Lavados PM, Sacks C, Prina L, et al. Incidence, case-fatality rate, and prognosis of ischaemic stroke subtypes in a predominantly Hispanic-Mestizo population in Iquique, Chile (PISCIS project): a community-based incidence study. *Lancet Neurol* 2007;6:140–148.
16. Seet RC, Friedman PA, Rabinstein AA. Prolonged rhythm monitoring for the detection of occult paroxysmal atrial fibrillation in ischemic stroke of unknown cause. *Circulation* 2011;124:477–486.
17. Lisabeth LD, Brown DL, Hughes R, et al. Acute stroke symptoms: comparing women and men. *Stroke* 2009;40:2031–2036.
18. Tanizaki Y, Kiyohara Y, Kato I, et al. Incidence and risk factors for subtypes of cerebral infarction in a general population: the Hisayama study. *Stroke* 2000;31:2616–2622.
19. Jackson C, Sudlow C. Are lacunar strokes really different? A systematic review of differences in risk factor profiles between lacunar and nonlacunar infarcts. *Stroke* 2005;36:891–901.
20. Air EL, Kissela BM. Diabetes, the metabolic syndrome, and ischemic stroke: epidemiology and possible mechanisms. *Diabetes care* 2007;30:3131–3140.
21. Navaratna D, Guo SZ, Hayakawa K, et al. Decreased cerebrovascular brain-derived neurotrophic factor-mediated neuroprotection in the diabetic brain. *Diabetes* 2011;60:1789–1796.
22. Bowman TS, Sesso HD, Ma J, et al. Cholesterol and the risk of ischemic stroke. *Stroke* 2003;34:2930–2934.

23. Tirschwell DL, Smith NL, Heckbert SR, et al. Association of cholesterol with stroke risk varies in stroke subtypes and patient subgroups. *Neurology* 2004;63:1868-1875.
24. Laloux P, Galanti L, Jamart J. Lipids in ischemic stroke subtypes. *Acta Neurol Belg* 2004;104:13-19.
25. Oksala NK, Heikkinen M, Mikkelsen J, et al. Smoking and the platelet fibrinogen receptor glycoprotein IIb/IIIa P1A1/A2 polymorphism interact in the risk of lacunar stroke and midterm survival. *Stroke* 2007;38:50-55.
26. Peters SA, Huxley RR, Woodward M. Smoking as a risk factor for stroke in women compared with men: a systematic review and meta-analysis of 81 cohorts, including 3,980,359 individuals and 42,401 strokes. *Stroke* 2013;44:2821-2828.
27. Kim J, Gall SL, Dewey HM, et al. Baseline smoking status and the long-term risk of death or nonfatal vascular event in people with stroke: a 10-year survival analysis. *Stroke* 2012;43:3173-3178.
28. Djousse L, Ellison RC, Beiser A, et al. Alcohol consumption and risk of ischemic stroke: The Framingham Study. *Stroke* 2002;33:907-912.
29. Mostofsky E, Burger MR, Schlaug G, et al. Alcohol and acute ischemic stroke onset: the stroke onset study. *Stroke* 2010;41:1845-1849.
30. Mohsenin V. Sleep-related breathing disorders and risk of stroke. *Stroke* 2001;32:1271-1278.
31. Jood K, Jern C, Wilhelmsen L, et al. Body mass index in mid-life is associated with a first stroke in men: a prospective population study over 28 years. *Stroke* 2004;35:2764-2769.
32. Park JW, Lee SY, Kim SY, et al. BMI and stroke risk in Korean women. *Obesity* 2008;16:396-401.
33. Sandercock P, Bamford J, Dennis M, et al. Atrial fibrillation and stroke: prevalence in different types of stroke and influence on early and long term prognosis (Oxfordshire community stroke project). *BMJ* 1992;305:1460-1465.
34. Jorgensen HS, Nakayama H, Reith J, et al. Acute stroke with atrial fibrillation. The Copenhagen Stroke Study. *Stroke* 1996;27:1765-1769.
35. Lin HJ, Wolf PA, Kelly-Hayes M, et al. Stroke severity in atrial fibrillation. The Framingham Study. *Stroke* 1996;27:1760-1764.
36. Hornig CR, Bauer T, Simon C, et al. Hemorrhagic transformation in cardioembolic cerebral infarction. *Stroke* 1993;24:465-468.
37. Molina CA, Montaner J, Abilleira S, et al. Timing of spontaneous recanalization and risk of hemorrhagic transformation in acute cardioembolic stroke. *Stroke* 2001;32:1079-1084.
38. Dromerick A, Reding M. Medical and neurological complications during inpatient stroke rehabilitation. *Stroke* 1994;25:358-361.
39. Roth EJ, Lovell L, Harvey RL, et al. Incidence of and risk factors for medical complications during stroke rehabilitation. *Stroke* 2001;32:523-529.
40. Yoneda Y, Uehara T, Yamasaki H, et al. Hospital-based study of the care and cost of acute ischemic stroke in Japan. *Stroke* 2003;34:718-724.

บทคัดย่อ

โรคหลอดเลือดสมองตีบแต่ละชนิดมีความแตกต่างกัน การศึกษานี้มีจุดประสงค์เพื่อหาความสัมพันธ์ของโรคหลอดเลือดสมองแต่ละชนิด กับปัจจัยเสี่ยง ความรุนแรง การรักษาในระยะเฉียบพลัน ภาวะแทรกซ้อน และผลการรักษา **วิธีการศึกษา** เป็นการศึกษาชนิดไปข้างหน้า จากทะเบียนผู้ป่วยโรคหลอดเลือดสมองของสถาบันประสาทวิทยาโดยแยกประเภทตาม TOAST stroke subtypes: small vessel atherosclerosis (SAO), large vessel atherosclerosis (LAA), cardiac embolism (CE), other determined etiology (OD), other undetermined etiology (UND) จากนั้นนำข้อมูลมาหาความสัมพันธ์ทางสถิติโดยใช้ Fisher's Exact Test หรือ Kruskal-Wallis Test **ผลการศึกษา** ผู้ป่วยทั้งหมด 140 คน ผู้ป่วยร้อยละ 45, 34.3, 12.9, 4.3, และ 3.6 ได้รับการจำแนกประเภทเป็น SAO, LAA, CE, OD, UND ตามลำดับ และพบว่าอายุ ความรุนแรงของโรค การรักษา ภาวะแทรกซ้อน และผลการรักษาสัมพันธ์กับชนิดของโรคหลอดเลือดสมองตีบอย่างมีนัยสำคัญทางสถิติ ($p < 0.001$) โรคหลอดเลือดสมองชนิด CE มีค่าเฉลี่ยของอายุ median มากที่สุด (68.5 ปี) และมีภาวะแทรกซ้อนจากสมองบวม กับการเกิดเลือดออกในบริเวณที่มีสมองตายได้บ่อย (ร้อยละ 22) **สรุป** ปัจจัยด้านอายุ ความรุนแรงของโรค วิธีการรักษา และผลการรักษาทางคลินิก มีความแตกต่างกันตามชนิดของโรคหลอดเลือดสมองตีบ ดังนั้นการรักษาและการป้องกันควรพิจารณาตามแต่ละชนิดของโรคหลอดเลือดสมองตีบ