Associations between Brain Imaging Characteristics and Cognition in Post-Stroke Patients

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Background: *Cerebrovascular disease is the main risk factor for dementia. Post-stroke dementia is a major cause of disability in adults and seniors. Ischemic lesions in certain areas of the brain can lead to cognitive and neuropsychiatric symptom change.*

Objective: *To investigate association among brain imaging characteristics, vascular risk factors, and cognitive function in post stroke patients; to examine for risk factors of post-stroke dementia; and to evaluate interrater agreement between CT and MRI, with specific regard to white matter lesions.*

Material and Method: *This observational study in 100 stroke patients aged more than 15 years was conducted at Siriraj Hospital in Bangkok, Thailand. Brain imaging (CT or MRI) was performed in all patients. Cognitive and neuropsychiatric status was evaluated at 2-4 weeks after discharge and at the 6-12 month follow-up visit. Dementia was defined according to DSM IV criteria. Risk factors for and odd ratios of post-stroke dementia were analyzed.*

Results: *Dementia was diagnosed in 15 of 85 patients (17.6%). Vascular dementia was the most commonly observed type of dementia. Anterior circulation stroke (p = 0.033, OR: 4.5), lacunar infarction (p = 0.022, OR: 5.46), severe central atrophy (p = 0.042,OR: 14.67), and anterior white matter lucencies (p = 0.028, OR: 4.27) were all significantly different between the dementia and non-dementia groups. Risk factors associated with post-stroke dementia were educational level less than 6 years (p = 0.012, OR: 15.2), history of previous stroke (p = 0.048, OR: 3.88), and diabetes mellitus (p = 0.049, OR: 6.9). Interrater agreement for white matter lesion visual rating between CT and MRI brain was 0.637. No significant association between neuropsychiatric symptoms and brain lesion was found.*

Conclusion: *Prevalence of post-stroke dementia in this study was 17.6%. Combination of multiple clinical risk factors (DM, history of previous stroke, and low educational level) and brain lesion (anterior circulation, central atrophy, and anterior WML) can contribute to development of post-stroke dementia. Among brain imaging findings, severe central atrophy had the strongest association with dementia (OR: 14.7). Among evaluated risk factors, educational level less than 6 years was the strongest predictor of post-stroke dementia (OR: 15.2). CT scan of the brain was reliable, compared to MRI, for detecting white matter lesion (kappa level = 0.637, 75.5% agreement).*

Keywords: *post-stroke dementia, Thailand, white matter lesions*

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Stroke is a major cause of physical disability and dependency in adults and seniors. Stroke also increases risk of cognitive impairment[1]. Vascular risk factors are risk factors for both Alzheimer's disease (AD) and vascular dementia (VaD)[2].

Etiologies of post-stroke dementia include vascular dementia, neurodegenerative dementia (AD), and mixed dementia[3]. Prevalence of post-stroke dementia at 6 months to 1 year varies from 6%-30%[4-8]. The mechanisms of post-stroke dementia are not completely known or understood. Certain vascular risk factors may increase dementia risk. Cerebral ischemic lesions can lead to vascular cognitive impairment (VCI) or post-stroke dementia (PSD)[10]. White matter lesions and strategic infarction have been described as risk factors for both VCI and PSD[11]. Moreover, neuropsychiatric symptoms, such as depression and apathy, are commonly found in post-stroke individuals. Post-stroke depression has significant effect on quality of life and stroke recovery.

The primary objective of this study was to investigate for correlations among brain imaging characteristics, risk factors, and cognitive function in post-stroke patients. Secondary objectives were to examine for risk factors for and prevalence of post-

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stroke dementia and to explore neuropsychiatric symptoms in post-stroke individuals.

Material and Method

This observational cross-sectional study was conducted at Siriraj Hospital-Thailand's largest university-based tertiary referral center. One hundred stroke patients who were admitted to Siriraj Stroke Unit during the 2006 to 2007 study period were enrolled in this study. Written informed consent was obtained from all study participants prior to inclusion. This study was approved by Siriraj Institutional Review Board (SIRB), Faculty of Medicine Siriraj Hospital, Mahidol University, Bangkok, Thailand.

Inclusion criteria (both of the following)

1) Patients diagnosed with acute ischemic stroke aged older than 15 years.

2) The ability to communicate in Thai language.

Exclusion criteria (any one or more of the following)

1) Diagnosed with dementia before stroke.

2) Expired or was lost to follow-up after discharge.

3) Unable to perform neuropsychological test.

4) Imaging study was not performed or was lost.

5) Patients with aphasia.

Either computerized tomography (CT) or magnetic resonance imaging (MRI) was performed in all patients. Cognitive and neuropsychiatric status was evaluated at 2-4 weeks after discharge and at the 6-12 month follow-up.

All patients were evaluated by a board certified neurologist. Cognitive functions were assessed by Thai Mental State Examination (TMSE)[12] and category verbal fluency test. Neuropsychiatric Inventory (NPI)[13] was used to evaluate 12 neuropsychiatric domains after stroke.

Dementia was diagnosed by Diagnostic and Statistical Manual of Mental Disorders, 4th edition (DSM-IV) criteria[14], which requires impairment of memory and impairment of at least one other cognitive domain (executive function, apraxia, agnosia, aphasia) and documented impaired activity of daily living. Cognitive domains were assessed by neuropsychological battery.

Patients with post-stroke dementia were subsequently subdivided into one of the following groups: VaD (National Institute of Neurological Disorders and Stroke criteria)[15]; possible AD (Alzheimer's Disease and Related Disorders Association criteria)[16]; cerebrovascular disease (CVD); or, mixed AD and VaD.

Comprehensive history taking and neuropsychological examination was conducted. Patients with pre-stroke dementia were excluded from the study.

Clinical subtypes of stroke were classified using Oxfordshire Community Stroke Project classification (OCSP)[17]. Based on the extent of symptoms, stroke episodes were classified, as follows: total anterior cerebral infarction (TACI), partial anterior cerebral infarction (PACI), lacunar infarction (LACI), or posterior cerebral infarction (POCI). Educational level was divided into 3 categories: low (0-6 years), middle (6-12 years), and high (>12 years).

Computerized tomography (CT) and magnetic resonance imaging (MRI) images of the brain were reviewed by the same neuroradiologist, who was blinded to patient clinical information. University of Edinburg's CT/MRI scan reading form 18 was used to collect imaging data.

Sites of infarction included anterior, posterior or subcortical areas of the brain. Extent of deep white matter lesions (WMLs) was graded from 0-2, as follows: 0 = no lesion; 1 = lucency to region adjoining ventricles; and 2 = lucency covering the entire region from lateral ventricle to cortex. WMLs were also classified as either anterior or posterior white matter lucency. Brain atrophy was classified as either central or cortical reduction in brain tissue, and was graded from 0-2, as follows: 0 = no atrophy; 1 = mild to moderate atrophy; and, 2 = severe atrophy. Patients were then divided into either the dementia or non-dementia groups. Characteristics of brain lesion and risk factors associated with cognitive impairment were then compared between the post-stroke dementia (PSD) and non post-stroke dementia (non-PSD) groups.

Statistical analysis

All statistical analyses were performed using SPSS statistics version 18 (SPSS, Inc., Chicago, IL, USA). Fisher's exact test and Pearson's chi-square test were used to analyze qualitative data. Student T-test and linear-by-linear association model were used to analyze quantitative and ordinal data, respectively. Stepwise logistic regression was performed to predict the outcome of a categorical dependent variable. Kappa analysis was used to calculate interrater agreement between CT and MRI for evaluation of white matter lesions. Kappa values of less than 0.4 indicate poor

agreement, 0.41 to 0.6 indicate moderate agreement, 0.61 to 0.8 indicate good agreement, and values more than 0.81 indicate excellent agreement[19]. The *p*-values less than 0.05 were regarded as being statistically significant.

Results

Of the 100 stroke patients enrolled in this study, one patient was excluded because of prior diagnosis with dementia, nine patients were lost to follow-up, and five patients were excluded due to loss of brain imaging. The remaining 85 patients were included in the analysis.

Post-stroke dementia (PSD) was diagnosed in 15 patients (15/85; 17.6%) and vascular dementia was diagnosed in 9 of those patients (9/15; 60%). One of those 9 patients had cerebellar cognitive affective syndrome with dementia, 5 patients had AD with CVD (5/15; 33.3%), and 1 patient had mixed AD & VaD (1/15; 6.7%).

Neuropsychiatric symptoms found in post-stroke patients included irritability (22/85; 25.9%), depression (17/85; 20%), anxiety (16/85; 18.8%), sleep and night time behavioral disorder (9/85; 10.6%), apathy (7/85; 8.2%), agitation (3/85; 3.5%), aberrant motor behavior (2/85; 2.4%), and 1 each for disinhibition, delusion, and hallucination (1/85; 1.2% for each).

According to Oxfordshire clinical classification of stroke, 19 patients (22.3%) had TACI/PACI, 13 patients (15.2%) had POCI, 43 patients (50.5%) had LACI, and 10 patients (11.7%) had unspecified clinical classification. However and from brain imaging studies, only 10 patients (11.7%) had total or partial anterior circulation stroke, 28 patients (32.9%) had posterior circulation stroke, and 46 patients (54.1%) had lacunar stroke. Accordingly, the prediction rate of the Oxfordshire clinical classification of stroke was 56% (42 of 75) (95% CI: 44.7, 66.7).

In our study, we found no significant difference in age between groups (69.4 vs. 58.4, $p = 0.248$). Low educational level (<6 years) was found more often in the dementia group than in the non-dementia group (78.6% vs. 30.9%, $p = 0.001$). History of previous stroke and diabetes mellitus were also significant predictors of dementia betweem the PSD and non-PSD groups (33.3% vs. 11.4%, $p = 0.048$ and 60% vs. 32.9%, $p = 0.049$, both respectively). Family history of dementia, smoking, alcohol use, hypertension and dyslipidemia were not significant predictors of dementia (Table 1).

The brain lesions that significantly predicted post-stroke dementia between groups were anterior circulation (33.3% vs. 10%, $p = 0.033$, respectively) and lacunar infarction (86.7% vs. 54.3%, $p = 0.022$, respectively). Posterior circulation was not a good predictor of dementia (53.3% vs. 32.9%, $p = 0.116$, respectively).

The Degree of cortical atrophy was not different between the PSD and non-PSD groups. Severe central atrophy was significantly different between groups ($p = 0.042$). Anterior white matter lucency was significantly associated with PSD ($p = 0.028$), while posterior white matter lucency was not ($p = 0.745$) (Table 2).

Fifty-three patients had both CT and MRI performed. Interrater reliability of WMLs between brain CT and MRI scans showed kappa level of 0.637, which indicated good agreement. The percentage of agreement was about 75.5% (40 of 53).

We selected significant variables to calculate the odds ratio of risk for developing PSD. Low educational level (less than 6 years) had OR of 15.2 (95% CI: 1.8,127, $p = 0.012$), but the other two education threshold had no significant statistical correlation with dementia.

Stepwise logistic regression revealed odds ratio for diabetes mellitus of 6.9 (95% CI: 1.7, 29.1). The brain lesions that correlated with post-stroke dementia were anterior circulation (OR: 10.4; 95% CI: 1.9, 54.7) and lacunar infarction (OR: 10.7; 95% CI: 1.7, 65.8).

Severe central atrophy (not mild to moderate atrophy) and anterior white matter lucency were both predictors of post-stroke dementia (OR: 14.6; 95% CI: 1.4, 156.9 and OR: 4.3; 95%CI: 1.1, 17.3, respectively).

No correlation was identified between neuropsychiatric symptoms and characteristics of brain imaging. Neuropsychiatric symptoms were not predictors of post-stroke dementia in this study.

Discussion

This is the first study in Thailand to investigate association between brain imaging characteristics and risk of developing post-stroke dementia. Prevalence of post-stroke dementia in our study was 17.6%, which was within the estimated range of 6-32%reported in a prior study[3]. VaD was the most commonly observed type of PSD in this study.

Risk factors found to be associated with

Table 1. Clinical risk factors in patients with and without post-stroke dementia

	Dementia	Non-dementia	*p*-value
Age, mean	69.4+9.3	58.5+13.2	0.248
Educational level			
<6yrs	11 (78.6%)	21 (30.9%)	0.001
6-12yrs	2 (14.3%)	18 (26.5%)	
>12yrs	1 (7.1%)	29 (42.6%)	
Family history of dementia	1 (6.7%)	1 (1.4%)	0.324
Smoking	3 (20%)	25 (35.7%)	0.194
Alcohol use	5 (33.3%)	38 (54.3%)	0.165
Previous stroke	5 (33.3%)	8 (11.4%)	0.048
Diabetes mellitus	9 (60%)	23 (32.9%)	0.049
Hypertension	10 (66.7%)	48 (68.6%)	0.554
Dyslipidemia	12 (80%)	55 (78.6%)	0.605

Data presented as n (%); *p-value*<0.05 indicates statistical significance

Table 2. Characteristics of brain lesion associated with post-stroke dementia

Characteristics	Dementia	Non-dementia	*p*-value
Anterior circulation	5 (33.3%)	7 (10%)	0.033
Posterior circulation	8 (53.3%)	23 (32.9%)	0.116
Lacunar infarction	13 (86.7%)	38 (54.3%)	0.022
Cortical atrophy			
- None	4 (26.7%)	34 (48.6%)	0.285
- Mild to moderate	9 (60%)	30 (42.9%)	
- Severe	2 (13.3%)	6 (8.6%)	
Central atrophy			
- None	1 (6.7%)	22 (31.4%)	0.042
- Mild to moderate	10 (66.7%)	42 (60%)	
- Severe	4 (26.7%)	6 (8.6%)	
Anterior white matter lucencies			
- None	3 (20%)	37 (52.9%)	0.028
- Lucency to region adjoining ventricles	9 (60%)	26 (37.1%)	
- Lucency covering from lateral ventricle to cortex	3 (20%)	7 (10%)	
Posterior white matter lucencies			
- None	9 (60%)	46 (65.7%)	0.745
- Lucency to region adjoining ventricles	5 (33.3%)	17 (24.3%)	
- Lucency covering from lateral ventricle to cortex	1 (6.7%)	7 (10%)	

Data presented as n (%); *p*-value <0.05 indicates statistical significance

post-stroke dementia were low educational level (<6years), history of previous stroke and diabetes mellitus. Among those, educational level less than 6 years demonstrated the strongest association with post-stroke dementia (OR: 15.2). Low level of education was found in prior study to be a risk factor for dementia. Low cognitive reserve and unhealthy lifestyle in people with low education could be an explanation. These factors could contribute to the development of cognitive impairment[9,20-22].

Regarding vascular risk factors, history of previous stroke and diabetes mellitus were significantly associated with dementia (OR: 3.88 and OR: 3.07,

Table 3. Odds ratio of post-stroke dementia

	Odds ratio (range)	*p*-value
Educational level		
- <6 yrs	15.2 (1.82-127)	0.012
- 6-12 yrs	3.2 (0.27-38.1)	0.353
History of stroke	3.9 (1.1-14.3)	0.048
Diabetes mellitus	3.1 (0.9-9.6)	0.049
Imaging finding		
Anterior circulation	4.5 (1.2-16.9)	0.033
Lacunar infarction	5.5 (1.1-26.3)	0.022
Central atrophy		
- Mild to moderate	5.2 (0.6-43.6)	0.631
- Severe	14.7 (1.4-157)	0.048
Anterior white matter lucencies		
- Lucency to region adjoining ventricles	4.3 (1.1-17.3)	0.042
- Lucency covering entire region from lateral ventricle to cortex	5.3 (0.9-31.7)	0.069

p-value <0.05 indicates statistical significance

Table 4. Adjusted odds ratio calculated by stepwise logistic regression

	Crude odds ratio (range)	Adjusted odds ratio (range)	*p*-value
Diabetes mellitus	3.1 (0.9-9.6)	6.9 (1.7-29.1)	0.04
Anterior circulation stroke	4.5 (1.2-16.9)	10.4 (1.9-54.7)	0.03
Lacunar infarction	5.5 (1.1-26.1)	10.7 (1.7-65.8)	0.02

p-value<0.05 indicates statistical significance

respectively). In previous studies, smoking and hypertension were also found to be significantly related to dementia, but we could not demonstrate this association in our study[9].

The sites and types of infarction associated with post-stroke dementia were anterior circulation stroke and lacunar infarction (OR: 10.4 and OR: 10.7, respectively). This resulting cognitive impairment may be the result of damage to the frontal cortical/subcortical circuits. No association was observed between posterior circulation stroke and post-stroke dementia, which is consistent with prior studies[4,22].

WMLs and their severity are considered to be a risk factor for dementia. WMLs cause ischemic tissue damage, which includes infarction, gliosis, and/or loss of myelin. These WMLs disrupt the frontal-subcortical circuit, which results in dementia[23]. In this study, we found correlations between WML and PSD (OR: 4.27 for grade 1 and OR: 5.29 for grade 2). CT scan of the brain was less reliable than MRI for detecting white matter lesion (kappa level = 0.637, 75.5% agreement). Small WMLs can be missed on CT scans of the brain.

Severe central atrophy had the strongest association with PSD in our study (OR: 14.67). Cortical atrophy and mild to moderate central atrophy were not found to be predictors of post-stroke dementia. There could be some confounding factors, because WMLs and lacunar infarction are frequently found in central atrophy and they might share a similar pathogenesis.

Neuropsychiatric symptoms, such as apathy and depression were not significantly associated with clinical risk factors or characteristics of brain lesion in our study. This may be due to the small sample size.

This study has some mentionable limitations.

First, the sample size was not sufficiently large enough to achieve statistical significance in some outcomes. Second, this study was conducted in a single center and may not be representative of overall Thai population. Third, some data were lost, which may have affected statistical significance in this study. Last and finally, the cognitive assessment performed was rudimentary. Accordingly, future study in PSD is recommended that includes larger population size, longer follow-up time, and detailed neuropsychological evaluations.

In conclusion, prevalence of post-stroke dementia in this study was 17.6%. Combination of multiple clinical risk factors (DM, history of previous stroke, and low educational level) and brain lesion (anterior circulation, central atrophy, and anterior WML) can contribute to development of post-stroke dementia. Among brain imaging findings, severe central atrophy had the strongest association with dementia (OR: 14.7). Among evaluated risk factors, educational level less than 6 years was the strongest predictor of post-stroke dementia (OR: 15.2). CT scan of the brain was reliable for detecting white matter lesion (kappa lvel = 0.637, 75.5% agreement).

What is already known to this topic?

Prevalence of post-stroke dementia in Caucasian population has been studied and reported.

What this study adds?

1. Prevalence of post-stroke dementia in Thai population.

2. Correlation between MRI and CT scan relative to white matter lesion.

3. Incidence of dementia etiology in patients with history of stroke.

4. Differences between stroke etiology clinical characteristics (Oxfordshire Community Stroke Project classification) and anatomical pathology.

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This was an unfunded study.

Potential conflict of interest

None.

References

1. Murray CJ, Lopez AD. Global mortality, disability, and the contribution of risk factors: Global Burden of Disease Study. Lancet 1997; 349: 1436-42.
2. Snowdon DA, Greiner LH, Mortimer JA, Riley KP, Greiner PA, Markesbery WR. Brain infarction and the clinical expression of Alzheimer disease. The Nun Study. JAMA 1997; 277: 813-7.
3. Leys D, Henon H, Mackowiak-Cordoliani MA, Pasquier F. Poststroke dementia. Lancet Neurol 2005; 4: 752-9.
4. Pohjasvaara T, Erkinjuntti T, Ylikoski R, Hietanen M, Vataja R, Kaste M. Clinical determinants of poststroke dementia. Stroke 1998; 29: 75-81.
5. Inzitari D, Di Carlo A, Pracucci G, Lamassa M, Vanni P, Romanelli M, et al. Incidence and determinants of poststroke dementia as defined by an informant interview method in a hospital-based stroke registry. Stroke 1998; 29: 2087-93.
6. Censori B, Manara O, Agostinis C, Camerlingo M, Casto L, Galavotti B, et al. Dementia after first stroke. Stroke 1996; 27: 1205-10.
7. Tang WK, Chan SS, Chiu HF, Ungvari GS, Wong KS, Kwok TC, et al. Frequency and determinants of poststroke dementia in Chinese. Stroke 2004; 35: 930-5.
8. Tatemichi TK, Foulkes MA, Mohr JP, Hewitt JR, Hier DB, Price TR, et al. Dementia in stroke survivors in the Stroke Data Bank cohort. Prevalence, incidence, risk factors, and computed tomographic findings. Stroke 1990; 21: 858-66.
9. Tatemichi TK, Desmond DW, Mayeux R, Paik M, Stern Y, Sano M, et al. Dementia after stroke: baseline frequency, risks, and clinical features in a hospitalized cohort. Neurology 1992; 42: 1185-93.
10. Erkinjuntti T, Hachinski VC. Rethinking vascular dementia. Cerebrovasc Dis 1993; 3: 3-23.
11. Pohjasvaara T, Mantyla R, Salonen O, Aronen HJ, Ylikoski R, Hietanen M, et al. How complex interactions of ischemic brain infarcts, white matter lesions, and atrophy relate to poststroke dementia. Arch Neurol 2000; 57: 1295-300.
12. Train the Brain Forum, Thailand. Thai Mini Mental State Examination (TMSE). Siriraj Hosp Gaz 1993; 45: 359-74.
13. Cummings JL, Mega M, Gray K, Rosenberg-Thompson S, Carusi DA, Gornbein J. The Neuropsychiatric Inventory: comprehensive assessment of psychopathology in dementia. Neurology 1994; 44: 2308-14.
14. American Psychiatric Association. Diagnostic and

statistical manual of mental disorders. 4th ed. Arlington, VA: American Psychiatric Association; 1994.
15. Chui HC, Victoroff JI, Margolin D, Jagust W, Shankle R, Katzman R. Criteria for the diagnosis of ischemic vascular dementia proposed by the State of California Alzheimer's Disease Diagnostic and Treatment Centers. Neurology 1992; 42: 473-80.
16. McKhann G, Drachman D, Folstein M, Katzman R, Price D, Stadlan EM. Clinical diagnosis of Alzheimer's disease: report of the NINCDS-ADRDA Work Group under the auspices of Department of Health and Human Services Task Force on Alzheimer's Disease. Neurology 1984; 34: 939-44.
17. Bamford J, Sandercock P, Dennis M, Burn J, Warlow C. Classification and natural history of clinically identifiable subtypes of cerebral infarction. Lancet 1991; 337: 1521-6.
18. Brain Research Imaging Centre. Image analysis tools [Internet]. 2017 [cited 2017 Mar 15]. Available from: http://www.sbirc.ed.ac.uk/research/imageanalysis.html
19. Altman DG. Some common problems in medical research. In: Altman DG, editor. Practical statistics for medical research. London: Chapman & Hall; 1991: 396-439.
20. Stern Y, Andrews H, Pittman J, Sano M, Tatemichi T, Lantigua R, et al. Diagnosis of dementia in a heterogeneous population. Development of a neuropsychological paradigm-based diagnosis of dementia and quantified correction for the effects of education. Arch Neurol 1992; 49: 453-60.
21. Gorelick PB, Brody J, Cohen D, Freels S, Levy P, Dollear W, et al. Risk factors for dementia associated with multiple cerebral infarcts. A case-control analysis in predominantly African-American hospital-based patients. Arch Neurol 1993; 50: 714-20.
22. Pendlebury ST, Rothwell PM. Prevalence, incidence, and factors associated with pre-stroke and post-stroke dementia: a systematic review and meta-analysis. Lancet Neurol 2009; 8: 1006-18.
23. Filley CM. The behavioral neurology of cerebral white matter. Neurology 1998; 50: 1535-40.

การศึกษาความสัมพันธ์ระหว่างลักษณะภาพทางรังสีวินิจฉัยกับภาวะสมองเสื่อมภายหลังการเกิดโรคหลอดเลือดสมอง

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ภูมิหลัง: *โรคหลอดเลือดสมองเป็นปัญหาที่พบบ่อย และเป็นปัจจัยเสี่ยงที่สำคัญของภาวะสมองเสื่อมภายหลังการเกิดโรคหลอดเลือดสมอง (Post stroke dementia) ซึ่งภาวะดังกล่าวเป็นสาเหตุให้เกิดความพิการและความยากลำบากในการดูแลผู้ป่วย โดยที่พบว่าลักษณะและตำแหน่งรอยโรคในสมองบางบริเวณจะมีความสัมพันธ์กับความรู้คิด และความผิดปกติทางจิตวิทยาของผู้ป่วยโรคหลอดเลือดสมองได้*

วัตถุประสงค์:

1) เพื่อศึกษาความสัมพันธ์ระหว่างลักษณะภาพทางรังสีวินิจฉัยกับภาวะสมองเสื่อมภายหลังการเกิดโรคหลอดเลือดสมอง

2) เพื่อศึกษาปัจจัยเสี่ยงของการเกิดภาวะสมองเสื่อมภายหลังการเกิดโรคหลอดเลือดสมอง

3) เพื่อศึกษาหาค่าความเชื่อมั่น (interrater agreement) ระหว่างการใช้เอกซเรย์คอมพิวเตอร์ (CT scan) กับเอกซเรย์คลื่นแม่เหล็กไฟฟ้า (MRI) ในการดูรอยโรคใน white matter

วัสดุและวิธีการ: *การศึกษานี้ใช้สถิติเชิงพรรณนาแบบตัดขวาง (Cross-sectional descriptive study) ทำการศึกษาในผู้ป่วยโรคหลอดเลือดสมองจำนวน 100 คน อายุตั้งแต่ 15 ปีขึ้นไป ที่ได้รับการรักษาที่หอผู้ป่วยโรคหลอดเลือดสมอง โรงพยาบาลศิริราช โดยผู้ป่วยทุกรายจะได้รับการตรวจเอกซเรย์คอมพิวเตอร์สมอง (CT Brain) หรือเอกซเรย์คลื่นแม่เหล็กไฟฟ้าสมอง (MRI brain) หลังจากนั้นผู้ป่วยจะได้รับการประเมินความรู้คิดและภาวะผิดปกติทางจิตวิทยา 2 สัปดาห์ และ 6 เดือน หลังออกจากโรงพยาบาล โดยที่จะใช้เกณฑ์การวินิจฉัย DSM IV ในการวินิจฉัยภาวะสมองเสื่อม*

ผลการศึกษา: *จากการศึกษาพบผู้ป่วยสมองเสื่อมภายหลังโรคหลอดเลือดสมองทั้งหมด 15 ราย จากจำนวนทั้งหมด 85 ราย (17.6%) โดยเมื่อพิจารณาลักษณะทางรังสีวิทยาที่สัมพันธ์กับภาวะสมองเสื่อมพบว่าสัมพันธ์กับหลอดเลือดสมองที่ตีบในหลอดลือดสมองส่วนหน้า ($p = 0.033$, OR = 4.5) หลอดเลือดสมองเส้นเล็ก ($p = 0.022$, OR = 5.5) การฝ่อของเนื้อสมอง (severe central atrophy) ($p = 0.042$,OR = 14.7) รอยโรคใน white matter ส่วนหน้า ($p = 0.028$, OR = 4.3) ส่วนปัจจัยเสี่ยงของภาวะสมองเสื่อมพบว่าสัมพันธ์กับการศึกษาที่น้อยกว่า 6 ปี ($p = 0.012$, OR = 15.2) ประวัติเคยเป็นโรคหลอดเลือดสมองมาก่อน ($p = 0.048$, OR = 3.9) และโรคเบาหวาน ($p = 0.049$, OR = 6.9) ค่าความเชื่อมั่นระหว่างเอกซเรย์คอมพิวเตอร์ (CT scan) กับเอกซเรย์คลื่นแม่เหล็กไฟฟ้า (MRI) เท่ากับ 0.637 และไม่พบความสัมพันธ์ระหว่างลักษณะที่จำเพาะทางรังสีกับภาวะผิดปกติทางจิตวิทยา*

สรุป: *พบความชุกของภาวะสมองเสื่อมภายหลังโรคหลอดเลือดสมอง 17.6% โดยลักษณะรอยโรคในสมองที่พบว่าสัมพันธ์กับภาวะสมองเสื่อมมากที่สุดคือ severe central atrophy (OR = 14.7) ส่วนปัจจัยเสี่ยงที่สัมพันธ์กับภาวะสมองเสื่อมมากที่สุดคือระดับการศึกษาที่น้อยกว่า 6 ปี โดยที่เอกซเรย์คอมพิวเตอร์ (CT scan) มีค่าความเชื่อมั่นในการตรวจรอยโรคใน white matter ดีพอกับเอกซเรย์คลื่นแม่เหล็กไฟฟ้า (MRI) (kappa level = 0.637, 75.5% agreement)*