Relationship between Poststroke Depression and Ischemic Lesion Location†

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Background: *Depression is more frequently detected in stroke patient compared to other medical illness with equal disability. The relationship between poststroke depression and ischemic stroke lesion location is controversial.*
Objective: *To determine the relationship between early onset poststroke depression and ischemic stroke lesion location.*
Material and Method: *A cross-sectional analysis was conducted. In-patients diagnosed with first acute ischemic stroke were enrolled. CT scan and MRI of the brain were performed to confirm the diagnosis of ischemic stroke as well as ischemic stroke subtypes and to determine the ischemic stroke lesion locations. Hamilton Depression Rating Scale was used to assess early onset poststroke depression within two weeks after the onset of stroke. Statistical analysis was conducted to determine the relationship between early onset poststroke depression and ischemic stroke lesion location as well as early poststroke depression and other potential factors.*
Results: *Thirty-nine patients were enrolled. The mean age (± SD) is 59.7 (± 12.3) years. Male: female ratio was 2:1. Early onset post stroke depression was found in 11 patients (28.2%). Mild depressive, less than major depressive, and major depressive level were found in five patients (12.8%), five patients (12.8%), and one patient (2.6%) respectively. Factors that statistically significantly related to early onset poststroke depression are left sided stroke lesion, female gender, and absence of hypertension.*
Conclusion: *Left sided stroke lesion, female gender, and absence of hypertension are factors contributing to early onset poststroke depression.*

Keywords: *Poststroke depression, Ischemic lesion location*

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Depression is the most common neuropsychiatric complication of stroke[1]. This condition is more frequently found in stroke patients compared to other medical illness with equal disability[2]. The prevalence of poststroke depression varies and depends on the diagnostic criteria for depression, the onset of depression after stroke and setting of the studied patients[3-7]. Poststroke depression negatively affects functional and cognitive recovery as well as mortality[8-10]. Although causes of poststroke depression remain unknown, factors including lesion location, stroke severity and disability, cognitive function and psychosocial factors have been postulated to be related to this condition[11-19]. There were conflicting evidences regarding the relationship between poststroke depression and lesion location[13,15-17,20-23]. However, there was a trend suggesting the relationship between poststroke depression and left sided brain lesion especially for the early onset of poststroke depression (less than three months from stroke onset)[17].The objective of study was to determine the relationship between early onset of poststroke depression and lesion location among Thai ischemic stroke patients.

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Material and Method

Study design

This is a cross-sectional analytic study.

Study population

The present study recruited consecutive patients with first clinical ischemic stroke documented by CT scan or MRI of the brain and admitted to King Chulalongkorn Memorial Hospital between January 2009 and January 2010. The eligible criteria included age more than 18-years-old and the capability to undergo the verbal interview. The exclusion criteria included the onset of stroke more than two weeks, intracerebral hemorrhage, bilateral stroke lesions or lesion at brainstem/ cerebellum, aphasia or moderate to severe cognitive impairment (Thai Mental State Examination (TMSE) score less than 18), and history of depression/ personality disorder or family history of depression.

Outcome measurement

The location of acute ischemic lesion was documented by a radiologist and a neurologist using CT scan or MRI of the brain. The interview questionnaires and tests which had been performed within two weeks after the onset of stroke attack included: demographic data, stroke severity and disability scale (National Institutes of Health Stroke Scale; NIHSS, Barthel index; BI and Modified Rankin Scale; mRS), cognitive function test (Thai Mental State Examination; TMSE)[24], psychosocial questionnaires (social support scale, stressful life event scale) and Hamilton Depression Rating Scale (Thai version). The Hamilton Depression Rating Scale was evaluated by a psychiatrist. All of the tests had been validated in a Thai population[24,25]. Patients with Hamilton Depression Rating Scale (HDRS) more than seven are considered to have depression. DSM-IIIR rating scale was used to classify the depressive level[26].

Statistical analysis

SPSS version 16 (SPSS Inc., Chicago, IL, USA) was used for statistical analysis. Baseline data was demonstrated in means and standard deviations for continuous data and percentage for categorical data. For comparison between the groups, categorical data *i.e.* depression versus non-depression were analyzed using Chi-square test and Fisher's Exact Test (sample size less than five). Multivariate analysis was used to detect the association between poststroke depression and location of the lesion as well as association between poststroke depression and other factors. P-value < 0.05 was considered to be statistically significant.

Ethical consideration

The Institutional Review Board of the Faculty of Medicine, Chulalongkorn University, Bangkok, Thailand, had approved the present study, which was to be carried out in compliance with the International guidelines for human research protection as Declaration of Helsinki, The Belmont Report, CIOMS Guideline, and International Conference on Harmonization in Good Clinical Practice (ICH-GCP).

Results

Thirty-nine patients were enrolled in the present study. The mean age ($\pm$ SD) was 59.7 $\pm$ 12.3 years. Male: female ratio was 2:1. CT scan of the brain was performed in all 39 patients and additional MRI of the brain was done in five patients (12.8%). Ischemic lesion location in the left and right hemispheric was found in 21 patients (53.8%) and 18 patients (46.2%), respectively. The mean score ($\pm$ SD) of NIHSS, BI, and mRS were 4.4 ($\pm$ 2.7), 68.6 ($\pm$ 24.4), and 3.1 ($\pm$ 1.4) respectively. Early onset poststroke depression was found in 11 patients (28.2%). Mild depressive, less than major depressive, and major depressive level were detected in five patients (12.8%), five patients (12.8%), and one patient (2.6%) respectively. The mean time of depression assessment after stroke onset was 3.4 days.

Comparison of the baseline data between depression and non-depression groups was demonstrated in Table 1 and only hypertension was statistically significantly different between the groups. Multivariate analysis showed the association of depression and left sided lesion, female gender, as well as absence of hypertension (Table 2).

Detailed lesion locations are shown in Table 3. There were no significant differences in the frequency of lesion locations between patients with and without depression. Nevertheless, ischemic lesions at subcortical white matter, caudate nucleus, lentiform nucleus, and anterior limb of internal capsule seemed to be more frequently detected in patients with depression than patients without depression.

Discussion

The prevalence of poststroke depression in the present study was 28.2%. The prevalence was not different from previous studies, which reported the prevalence around 11 to 46%[3-7,27]. Left hemispheric

Table 1. Characteristics of the depression group and the non-depression group

Characteristics	Depression (n = 11), n (%)	No depression (n = 28), n (%)	p-value
Age			0.243
< 60 years	7 (63.6)	12 (42.9)	
≥ 60 years	4 (36.4)	16 (57.1)	
Gender			0.131
Female	6 (54.5)	7 (25.0)	
Male	5 (45.5)	21 (75.0)	
Marital status			1.000
Non-married	3 (27.3)	7 (25.0)	
Married	8 (72.7)	21 (75.0)	
Family size			1.000
Single	10 (90.9)	24 (85.7)	
Others	1 (9.1)	4 (14.3)	
Education			0.400
Below university graduate	10 (90.9)	21 (75.0)	
University graduate or higher education	1 (9.1)	7 (25.0)	
Hometown			0.228
Bangkok	10 (90.9)	19 (67.9)	
Outside Bangkok	1 (9.1)	9 (32.1)	
Employment			0.713
Employed	8 (72.7)	17 (60.7)	
Unemployed	3 (27.3)	11 (39.3)	
Diabetes melitus			1.000
No	8 (72.7)	20 (71.4)	
Yes	3 (27.3)	8 (28.6)	
Hypertension			0.010
No	8 (72.7)	7 (25.0)	
Yes	3 (27.3)	21 (75.0)	
Dyslipidemia			0.276
No	9 (81.8)	17 (60.7)	
Yes	2 (18.2)	11 (39.3)	
Current tobacco use			0.663
Yes	3 (27.3)	5 (17.9)	
No	8 (72.7)	23 (82.1)	
Current alcohol abuse			0.719
No	8 (72.7)	18 (64.3)	
Yes	3 (27.3)	10 (35.7)	
Social support scale			0.655
Moderate and high level	10 (90.9)	23 (82.1)	
Low level	1 (9.1)	5 (17.9)	
Stressful life event scale			0.693
Moderate and high level	9 (81.8)	20 (71.4)	
Low level	2 (18.2)	8 (28.6)	
NIHSS			0.649
< 7	10 (90.9)	22 (78.6)	
≥ 7	1 (9.1)	6 (21.4)	
BI			0.510
< 50	6 (54.5)	12 (42.9)	
≥ 50	5 (45.5)	16 (57.1)	
mRS			1.000
≥ 4	7 (63.6)	17 (60.7)	
< 4	4 (36.4)	11 (39.3)	

NIHSS = National Institutes of Health Stroke Scale; BI = Barthel Index; mRS = Modified Rankin Scale; TMSE = Thai Mental State Examination

Table 1. (Cont.)

Characteristics	Depression (n = 11), n (%)	No depression (n = 28), n (%)	p-value
TMSE			0.562
< 24	2 (18.2)	2 (7.1)	
≥ 24	9 (81.8)	26 (92.9)	
Stroke lesion location			0.138
Left side	8 (72.7)	13 (46.4)	
Right side	3 (27.3)	15 (53.6)	

NIHSS = National Institutes of Health Stroke Scale; BI = Barthel Index; mRS = Modified Rankin Scale; TMSE = Thai Mental State Examination

Table 2. Multivariate analysis for the association between poststroke depression and risk factors

Characteristics	Depression (n = 11), n (%)	No depression (n = 28), n (%)	Adjusted odds ratio	p-value
Gender			12.798	0.038
Female	6 (54.5)	7 (25.0)		
Male	5 (45.5)	21 (75.0)		
Hypertension			73.709	0.005
No	8 (72.7)	7 (25.0)		
Yes	3 (27.3)	21 (75.0)		
Stroke lesion location			16.160	0.034
Left	8 (72.7)	13 (46.4)		
Right	3 (27.3)	15 (53.6)		

Table 3. Details of lesion locations in depression group and non-depression group

Lesion locations	Depression (n = 11), n (%)	No depression (n = 28), n (%)	p-value
Frontal cortex	0 (0.0)	1 (3.6)	1.000
Parietal cortex	0 (0.0)	2 (7.1)	1.000
Temporal cortex	0 (0.0)	3 (10.7)	0.545
Occipital cortex	0 (0.0)	1 (3.6)	1.000
Insular cortex	0 (0.0)	2 (7.1)	1.000
Subcortical white matter	8 (72.7)	13 (46.4)	0.171
Caudate nucleus	2 (18.2)	1 (3.6)	0.187
Lentiform nucleus	7 (63.6)	11 (39.3)	0.285
Internal capsule			
Anterior limb	2 (18.2)	0 (0.0)	0.074
Genu	1 (9.1)	1 (3.6)	0.490
Posterior limb	4 (36.4)	10 (35.7)	1.000
Thalamus	5 (17.9)	1 (9.1)	0.655

lesion was associated with early onset poststroke depression**.** This correlation was paralleled with many other studies[12,19,28]. Nevertheless, the previous study in a Thai population showed no relationship between poststroke depression and the left hemispheric lesion[27]. The reason might be explained by more duration of depression assessment from stroke onset (mean duration was 14.88 months compared to 3.4 days in the present study) that is defined as late onset poststroke depression. Late onset poststroke depression was mostly not associated with left hemispheric lesion[17,19]. In animal models, disruption

of monoaminergic neurotransmitter pathways was observed after the creation of ischemic lesions in either hemisphere[29-31]. However, neurophysiological compensation with the increasing of serotonin-receptor binding in the cerebral cortex was higher with right sided compared with left sided lesions[32]. These findings have been proposed to explain the association of left hemispheric lesion and early onset poststroke depression in humans**.** Furthermore, the present study demonstrated a trend of early onset poststroke depression in ischemic lesions at subcortical white matter, caudate nucleus, lentiform nucleus and anterior limb of internal capsule, which are also in the circuit of monoaminergic neurotransmitter.

Regarding the patients' gender, female tends to be associated with poststroke depression in the present study, which was comparable with previous studies[33-36]. The effect of gender on poststroke depression may be multifactorial included the sex differences in hormones, social role and coping mechanism to psychological stress[37,38].

Several studies found no definite association between comorbid diseases and poststroke depression[39-43]. The association between absence of hypertension and poststroke depression in the present study was interesting. Patients with a previous history of hypertension may accept the presence of hypertension and its possible role in the pathogenesis of stroke as well as its associated disability. These patients may have better psychological coping mechanism after the occurrence of stroke comparing to the patients without a history of hypertension who did not expect any stroke disability.

The relationship between the stroke disability and poststroke depression was not found in the present study. It was noticeable that the patients in the present study had quite low disability (mean score of NIHSS, BI, and mRS were 4.4, 68.6, and 3.1 respectively), moderate to high social support and no previous history or family history of depression. Therefore, the poststroke depression in the present study was unlikely caused by stroke disability and these psychosocial factors.

Some methodological limitations should be acknowledged. Patients with aphasia, moderate cognitive impairment, brain stem, and cerebellar lesions were excluded. Therefore, the results of the present study may not be applicable for all ischemic stroke patients. Moreover, CT scan, which is less sensitive than MRI to detect cerebral ischemic lesion, was used in almost all studied patients. Thus, the evaluation of the brain lesion site may not be completely adequate. Another limitation is the rather small size of the studied population.

Conclusion

Left sided stroke lesion, female gender, and absence of hypertension are factors contributing to early onset poststroke depression. The relationship between left sided lesion and early onset poststroke depression may support the role of neurobiology in depression. Moreover, closed surveillance for early poststroke depression and early management of depression in these patients is advocated.

Potential conflicts of interest

None.

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ความสัมพันธ์ระหว่างภาวะซึมเศร้าหลังการเกิดโรคหลอดเลือดสมองและตำแหน่งรอยโรคสมองขาดเลือด

มณฑล ว่องวันดี, สุขเจริญ ตั้งวงษ์ไชย, กัมมันต์ พันธุมจินดา

ภูมิหลัง: *ความชุกของภาวะซึมเศร้าในผู้ป่วยโรคหลอดเลือดสมองมากกว่าผู้ป่วยที่มีภาวะความเจ็บป่วยทางกายอื่นที่มีระดับความพิการเท่ากัน ข้อสรุปเกี่ยวกับความสัมพันธ์ระหว่างตำแหน่งของสมองขาดเลือดกับการเกิดภาวะซึมเศร้าหลังการเกิดโรคหลอดเลือดสมองยังเป็นที่ถกเถียงกันอยู่*

วัตถุประสงค์: *เพื่อหาความสัมพันธ์ระหว่างตำแหน่งของสมองขาดเลือดกับการเกิดภาวะซึมเศร้าในระยะต้นหลังการเกิดโรคหลอดเลือดสมอง*

วัสดุและวิธีการ: *การศึกษานี้เป็นการศึกษาเชิงวิเคราะห์ ณ จุดเวลาใดเวลาหนึ่ง โดยศึกษาในผู้ป่วยโรคหลอดเลือดสมองตีบระยะฉับพลันที่เกิดขึ้นครั้งแรกและได้รับการรักษาในโรงพยาบาล ผู้นิพนธ์ทำการประเมินภาวะซึมเศร้าหลังการเกิดโรคหลอดเลือดสมองโดยใช้ Hamilton Depression Rating Scale (Thai Version) ภายใน 2 สัปดาห์หลังเกิดโรคหลอดเลือดสมองตีบระยะฉับพลัน ร่วมกับเก็บข้อมูลด้านประชากรศาสตร์ จิตสังคม ระดับพุทธิปัญญา ตำแหน่งสมองขาดเลือด ความพิการหลังการเกิดโรคหลอดเลือดสมองโดยใช้แบบสอบถามและแบบทดสอบ หลังจากนั้น หาความสัมพันธ์ระหว่างภาวะซึมเศร้าหลังการเกิดโรคหลอดเลือดสมองและปัจจัยต่าง ๆ ดังกล่าวข้างต้นโดยวิธีการ ทางสถิติ*

ผลการศึกษา: *มีผู้ป่วยทั้งหมด 39 ราย อายุเฉลี่ย (± ส่วนเบี่ยงเบนมาตรฐาน) เท่ากับ 59.7 ± 12.3 ปี สัดส่วนเพศชายต่อเพศหญิงเท่ากับ 2 ต่อ 1 มีผู้ป่วยเกิดภาวะซึมเศร้าหลังการเกิดโรคหลอดเลือดสมองทั้งหมด 11 ราย หรือ ร้อยละ 28.2 แบ่งเป็นระดับความรุนแรง mild, less than major และ major เท่ากับ 5 คน หรือ ร้อยละ 12.8, 5 คน หรือ ร้อยละ 12.8 และ 1 คน หรือ ร้อยละ 2.6 ตามลำดับ ตำแหน่งของสมองขาดเลือดซีกซ้าย เพศหญิงและการไม่มีโรคความดันโลหิตสูง มีความสัมพันธ์กับการเกิดภาวะซึมเศร้าในระยะต้นหลังการเกิดโรคหลอดเลือดสมองอย่างมีนัยสำคัญทางสถิติ*

สรุป: *ตำแหน่งรอยโรคสมองขาดเลือดซีกซ้าย เพศหญิงและการไม่มีโรคความดันโลหิตสูงมีความสัมพันธ์กับการเกิดภาวะซึมเศร้าในระยะต้นหลังการเกิดโรคหลอดเลือดสมอง*