Relationship between Poststroke Depression and Ischemic Lesion Location†

Monton Wongwandee MD*, Sookjaroen Tangwongchai MD**, Kammant Phanthumchinda MD***

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* Division of Neurology, Department of Internal Medicine, Faculty of Medicine, Srinakharinwirot University, Nakhon Nayok, Thailand
** Department of Psychiatry, Faculty of Medicine, Chulalongkorn University, Bangkok, Thailand
*** Division of Neurology, Department of Internal Medicine, Faculty of Medicine, Chulalongkorn University, Bangkok, Thailand

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 neuro-psychiatric 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.
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-III-R 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 (± SD) was 59.7 ± 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 (± SD) of NIHSS, BI, and mRS were 4.4 (± 2.7), 68.6 (± 24.4), and 3.1 (± 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>
<thead>
<tr>
<th>Characteristics</th>
<th>Depression (n = 11), n (%)</th>
<th>No depression (n = 28), n (%)</th>
<th>p-value</th>
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<td>12 (42.9)</td>
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<tr>
<td>≥ 60 years</td>
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<td>16 (57.1)</td>
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<tr>
<td><strong>Gender</strong></td>
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<td></td>
<td>0.131</td>
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<td>Yes</td>
<td>2 (18.2)</td>
<td>11 (39.3)</td>
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<td><strong>Current tobacco use</strong></td>
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<td>5 (17.9)</td>
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<td>8 (72.7)</td>
<td>23 (82.1)</td>
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<td><strong>Current alcohol abuse</strong></td>
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<td>18 (64.3)</td>
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<td>10 (35.7)</td>
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<td>10 (90.9)</td>
<td>23 (82.1)</td>
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<tr>
<td>Low level</td>
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<td>5 (17.9)</td>
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<tr>
<td><strong>Stressful life event scale</strong></td>
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<td>0.693</td>
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<tr>
<td>Moderate and high level</td>
<td>9 (81.8)</td>
<td>20 (71.4)</td>
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<td>Low level</td>
<td>2 (18.2)</td>
<td>8 (28.6)</td>
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<td><strong>NIHSS</strong></td>
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</tr>
<tr>
<td>&lt; 7</td>
<td>10 (90.9)</td>
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<td>≥ 7</td>
<td>1 (9.1)</td>
<td>6 (21.4)</td>
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<td><strong>BI</strong></td>
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<td>≥ 50</td>
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<td><strong>mRS</strong></td>
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<tr>
<td>≥ 4</td>
<td>7 (63.6)</td>
<td>17 (60.7)</td>
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</tr>
<tr>
<td>&lt; 4</td>
<td>4 (36.4)</td>
<td>11 (39.3)</td>
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</tbody>
</table>

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

Table 1. (Cont.)

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Depression (n = 11), n (%)</th>
<th>No depression (n = 28), n (%)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>TMSE</td>
<td></td>
<td></td>
<td>0.562</td>
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<tr>
<td>&lt; 24</td>
<td>2 (18.2)</td>
<td>2 (7.1)</td>
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</tr>
<tr>
<td>≥ 24</td>
<td>9 (81.8)</td>
<td>26 (92.9)</td>
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</tr>
<tr>
<td>Stroke lesion location</td>
<td></td>
<td></td>
<td>0.138</td>
</tr>
<tr>
<td>Left side</td>
<td>8 (72.7)</td>
<td>13 (46.4)</td>
<td></td>
</tr>
<tr>
<td>Right side</td>
<td>3 (27.3)</td>
<td>15 (53.6)</td>
<td></td>
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</table>

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

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

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

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

lesion was associated with early onset poststroke depression. This correlation was paralleled with many other studies\(^{12,19,20}\). 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. However, neurophysiological compensation with the increasing of serotonin-receptor binding in the cerebral cortex was higher with right sided compared with left sided lesions. 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. The effect of gender on poststroke depression may be multifactorial included the sex differences in hormones, social role and coping mechanism to psychological stress.

Several studies found no definite association between comorbid diseases and poststroke depression. 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.

References
ความสัมพันธ์ระหว่างภาวะซึมเศร้าหลังการเกิดโรคหลอดเลือดสมองและตำแหน่งของโรคสมองขาดเลือด

มณฑล ว่องวันดี, สุขเจริญ ตั้งวงษ์ไชย, กัมมันต์ พันธุมจินดา

ภูมิหลัง: ความชุกของภาวะซึมเศร้าในผู้ป่วยโรคหลอดเลือดสมองมากกว่าผู้ที่มีภาวะความเจ็บป่วยทางกายอื่นที่มีระดับความพิการทั้งคู่ ซึ่งสูญเสียประโยชน์ทางด้านแนวคิดของสมองขาดเลือดกับการเกิดภาวะซึมเศร้า หลังการเกิดโรคหลอดเลือดสมองยังเป็นที่ถกเถียงกันอยู่

วัตถุประสงค์: เพื่อหาความสัมพันธ์ระหว่างตำแหน่งของสมองขาดเลือดกับการเกิดภาวะซึมเศร้าในระยะต้นหลังการเกิดโรคหลอดเลือดสมอง

วัสดุและวิธีการ: การศึกษานี้เป็นการศึกษาเชิงวิเคราะห์ ณ จุดเวลาใดเวลาหนึ่ง โดยศึกษาในผู้ป่วยโรคหลอดเลือดสมองด้วยระยะเวลาหลังจากเกิดเหตุการณ์แล้ว และได้รับการรักษาในโรงพยาบาล ผู้วิจัยทำการประเมินภาวะซึมเศร้าหลังการเกิดโรคหลอดเลือดสมองโดยใช้ Hamilton Depression Rating Scale (Thai Version) ภายใน 2 สัปดาห์หลังเกิดโรคหลอดเลือดสมองด้วยระยะเวลาหลังจากเกิดเหตุการณ์แล้ว รวมทั้งสิ้น 7 สัปดาห์ ระดับทั้งหมดบาทระดับ ด้านแนวคิดของสมองขาดเลือด ความพิการหลังการเกิดโรคหลอดเลือดสมองโดยใช้แบบสอบถามและแบบทดสอบหลังจากนั้น หาความสัมพันธ์ระหว่างภาวะซึมเศร้าหลังการเกิดโรคหลอดเลือดสมองและปัจจัยต่าง ๆ ด้วยวิธีการทางสถิติ

ผลการศึกษา: มีผู้ป่วยทั้งหมด 39 ราย อยู่ใน几句 ที่บ้าน (ร้อยละ 79.5) ที่บ้าน 59.7 ± 12.3 ปี สัดส่วนเพศชายต่อเพศหญิงเท่ากับ 2.3 มีภาวะมาเกิดภาวะซึมเศร้าหลังการเกิดโรคหลอดเลือดสมอง 11 ราย หรือ ร้อยละ 28.2 แบ่งเป็นระดับความรุนแรง mild, less than major และ major รวมทั้งหมด 5 คน หรือ ร้อยละ 12.8, 5 คน หรือ ร้อยละ 12.8 และ 1 คน หรือ ร้อยละ 2.6 ตามลำดับ ตำแหน่งของสมองขาดเลือดซ้ายเพศหญิงและт I มีความสัมพันธ์กับการเกิดภาวะซึมเศร้าในระยะต้นหลังการเกิดโรคหลอดเลือดสมองอย่างมีนัยสำคัญทางสถิติ

สรุป: ตำแหน่งของสมองขาดเลือดซ้ายเพศหญิงและการไม่มีโรคความดันโลหิตสูงมีความสัมพันธ์กับการเกิดภาวะซึมเศร้าในระยะต้นหลังการเกิดโรคหลอดเลือดสมอง