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STUDY OF THE ASSOCIATION BETWEEN LESION LOCATION AND POST-STROKE DEPRESSION

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ABSTRACT

Background: Post-stroke depression (PSD) is a common stroke sequel. It is an independent predictor of poor prognosis for stroke, and The relationship between post-stroke depression and lesion location is controversial. Aim: The aim of this study is to evaluate the relationship between lesion location and post-stroke depression. Materials and Methods: This is a Observational prospective cohort study involved 50 patients with proven diagnosis of stroke admitted at the Department of Neurology, Tishreen University Hospital, Lattakia, during the period between September 2021- August 2022 .Patients were divided into two groups: the first group included those with PSD (18 patients). The second group included patients without PSD (32 patients). Results and discussion: Out of 50 patients, 28 were male and 22 were female, with mean age of the patients was 61.48 ± 10.6. Frequency of PSD after 2 months of stroke was 36%. 10% of patients had severe depression. Anxious depression was more frequent and was found in 38.9%. There was no significant differences between the two groups regarding of age, sex, hypertension, living alone, or smoking. There were significant differences between two groups regarding of the lesion location in the frontal lobe (P-value: 0.001, Odds Ratio: 8.8),right-sided Lesion (P-value: 0.02, Odds Ratio: 3.8), and ischemic stroke (P-value: 0.01, Odds Ratio: 3.1). PSD was associated with increased disability after 2 months. Characteristics of the stroke were not associated with the severity or type of PSD, or presence of suicidal ideation. Conclusion: The current study shows that lesion in the frontal lobe, right hemisphere, and with ischemic type predict PSD, which helps to early detection and treatment as well as prevention of PSD.

KEYWORDS: Stroke, lesion location, post-stroke depression.

INTRODUCTION

Stroke is defined as a Sudden onset of a neurologic deficit from a vascular mechanism: 85% are ischemic and 15% are primary hemorrhages. Stroke is a leading cause of neurologic disability in adults; There are 700,000 strokes annually in the United States,[1] and 200,000 stroke related deaths.[2] Depression is a mood disorder that can simultaneously affects one's emotions, energy, and motivation.[3] Post-stroke depression (PSD) is the most common emotional disturbance, and the most important long-term psychosocial consequence following stroke.[4] PSD affects between 30-50% of stroke patients.[5] PSD increase within 3 months from stroke, despite an improvement in disability.[6] so, it can be observed in those who are perceived to be functionally independent in their activities of daily living.[7] The risk of PSD remain high, even 1–3 years after stroke,[8] and a fifth of stroke survivors are still depressed at 5 years following it.[9] PSD is associated with increased disability, and poor functional and cognitive outcomes in stroke survivors.[10,11] and it has a negative impact on rehabilitation process.[12-14] 7–10% of patients have suicidal ideation following stroke.[15,16] In addition, patients with PSD have more 12-month post-stroke healthcare use than nondepressed patients.[17] There were conflicting evidences regarding the relationship between PSD and lesion location.[18] The aim of study is to evaluate the relationship between post-stroke depression and lesion location among Syrian stroke patients.

Study population

The present study recruited consecutive patients with first clinical stroke and admitted to the Department of

Neurology, Tishreen University Hospital during the period between September 2021- August 2022. The eligible criteria included age more than 18-years-old and isolated lesion evidenced by CT or MRI of the brain. The exclusion criteria included aphasia or cognitive impairment, history of depression or other mental disorders, severe and disabling chronic diseases, other neurological diseases, endocrine diseases, alcoholic or addicted patients.

Outcome measurement

The location of acute lesion was documented by a neurologist using CT scan or MRI of the brain.

The interview questionnaires and tests which had been performed during first 24 hours after stroke and about 2 months after stroke included: demographic data, medical and mental history, stroke severity and disability scale (National Institutes of Health Stroke Scale; NIHSS), and Beck's Depression Inventory (Arabic version)[19] which is among the first scales specifically meant to measure the severity of depression and is commonly used as a scale in clinical research.[28] A BDI score over 23 was associated with a severe depression. Depression diagnosis was set according to DSM-V by a psychiatrist.

Statistical analysis

SPSS version 20 (IBM Corporation) 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 posts-stroke depression and location side and type of the lesion. P-value < 0.05 was considered to be statistically significant.

RESULTS

Fifty patients were enrolled in the present study. The mean age (+ SD) was 61.48±10.6 years. 56% were male, 50% were smoker, 52% had hypertension and 2(4%) individuals were home alone.

Lesions in thalamus(18%), internal capsule(14%) and temporal lope(12%), were more frequency. 27% of lesions were left sided . 78% were ischemic. 35.9% of infarctions were lacunar. the mean score of NIHSS at admission and after two months was 5.28 and 3.14 respectively.

36% of the patients had PSD, and severe depression was detected in 10%. The Dominant depressive symptom in depressed patients was weight loss, feeling sad and Psychomotor agitation respectively (table1). Anxious depression was more frequently and was detected in 39.9% of patients. Suicidal ideation was found in 55% of depressed patients (table9). No statistically significant association was found between PSD and age, gender, presence of hypertension, smoking or living alone (table 2). Frontal lesions (table3), right-sided lesions and ischemic type of stroke (table4) had statistically significant association with PSD.

No statistically significant association was found between PSD and infarction volume or stroke severity and disability at admission (table5). However, PSD was associated with increased disability after 2 months. (table5) Characteristics of the stroke were not associated with severity or type of PSD, or presence of suicidal ideation (table6,7,8,9). However, severe depression was more frequent in ischemic type of stroke (table6) and in the frontal, partial, temporal lobes internal capsule and brainstem lesions (table7).

Right-sided lesions were more frequent in severe depression and in anxious pattern of depression, whereas left sided were more frequent in melancholic pattern(table8).

Suicidal ideation was more frequent in the right frontal and partial lobes lesions, left internal capsule lesions, right-sided lesions and anxious pattern of depression (table9).

Table 1: Dominant depressive symptom in the depression group and the non-depression group.

Dominant depressive symptom	Depression	No depression
weight loss	4(22.2%)	3(9.4%)
Psychomotor agitation	3(16.7%)	8(25%)
feeling sad	3(16.7%)	3(9.4%)
social isolation	2(11.1%)	1(3.1%)
insomnia	1(5.6%)	4(12.5%)
health worries	1(5.6%)	2(6.3%)
Concentration difficulty	1(5.6%)	1(3.1%)
hypersomnia	1(5.6%)	1(3.1%)
loss of energy	1(5.6%)	1(3.1%)

Table 2: Characteristics of the depression group and the non-depression group.

Characteristics		Depression	No depression	P-value
Gender	Male	8(44.4%)	20(62.5%)	0.2
	Female	10(55.6%)	12(37.5%)	
Age		61.11±11.4	61.68±10.4	0.8
Smoking		9(50%)	16(50%)	1
Living alone		0(0%)	2(6.3%)	0.2
Hypertension		12(66.7%)	14(43.8%)	0.1

Table 3: Association between lesion location and PSD.

Lesion location	Depression	No depression	P-value	O.R
Frontal lobe	4(22.2%)	1(3.1%)	0.001	8.8
Thalamus	3(16.7%)	6(18.8%)	0.3	-
Internal capsule	3(16.7%)	4(12.5%)	0.5	-
Parietal lobe	2(11.1%)	3(9.4%)	0.2	-
Temporal lobe	2(11.1%)	4(12.5%)	0.1	-
Lentiform nucleus	2(11.1%)	3(9.4%)	0.2	-
Brainstem	2(11.1%)	3(9.4%)	0.2	-
Occipital lobe	0(0%)	3(9.4%)	0.09	-
Caudate nucleus	0(0%)	1(3.1%)	0.1	-
Cerebellum	0(0%)	2(6.3%)	0.8	-
Subcortical white matter	0(0%)	2(6.3%)	0.8	-

Table 4: Association between Lesion Characteristics and PSD.

Lesion Characteristics		Depression	No depression	P-value	O.R
side	Right	12(66.7%)	11(34.4%)	0.02	3.8
	Left	6(33.3%)	21(65.6%)		
type	ischemic	16(88.9%)	23(71.9%)	0.01	3.1
	hemorrhagic)	0(0%)	9(28.1%)		
	venous	2(11.1%)	0(0%)		
volume	lacunar	6(37.5%)	8(34.8%)	0.8	-
	Non lacunar	10(62.5%)	15(65.2%)		

Table 5: Association between stroke severity and PSD

NIHSS	Depression	No depression	P-value
at admission	5.83±3.1	4.96±3.3	0.3
after 2 months	4.88±2.9	2.15±2.7	0.002
change	- 0.952±0.2	- 2.811±0.6	0.0001

Table 6: Association between Lesion Characteristics and severety of PSD.

Lesion Characteristics		*Not severe*	*severe*	P-value
side	Right	20(44.4%)	3(60%)	0.07
	Left	25(55.6%)	2(40%)	
type	ischemic	34(75.6%)	5(100%)	0.05
	hemorrhagic)	9(20%)	0(0%)	
	venous	2(4.4%)	0(0%)	
volume	lacunar	13(38.2%)	1(20%)	0.4
	Non lacunar	21(61.8%)	4(80%)	

Table7: Association between lesion location and severety of PSD

Lesion location	*Not severe*	*severe*	P-value
Frontal lobe	4(8.9%)	1(20%)	0.06
Parietal lobe	4(8.9%)	1(20%)	0.06
Temporal lobe	5(11.1%)	1(20%)	0.5
Internal capsule	6(13.3%)	1(20%)	0.7
Brainstem	4(8.9%)	1(20%)	0.09
Occipital lobe	3(6.7%)	0(0%)	0.1
Thalamus	9(20%)	0(0%)	0.8
Lentiform nucleus	5(11.1%)	0(0%)	0.5
Caudate nucleus	1(2.2%)	0(0%)	0.1
Cerebellum	2(4.4%)	0(0%)	0.2
Subcortical white matter	2(4.4%)	0(0%)	0.2

Table 8: Association between Lesion Characteristics and PSD'spattern.

Stroke characteristics		melancholic	anxious	Atypical	No pattern	P-value
location	Frontal	1(33.3%)	3(42.9%)	0(0%)	0(0%)	0.2
	Non frontal	2(66.7%)	4(57.1%)	3(100%)	5(100%)	
side	Right	1(33.3%)	5(71.4%)	2(66.7%)	4(80%)	0.5
	Left	2(66.7%)	2(28.6%)	1(33.3%)	1(20%)	
NIHSS	at admission	4.33±3.5	5.71±4.2	5.33±1.1	7.20±1.9	0.6
	after 2 months	3.66±3.7	4.71±3.7	5±1.7	5.8±2.2	0.8

Table 9: Association between Lesion and depression Characteristics and Suicidal ideation.

characteristics		Suicidal ideation (10)	No Suicidal ideation (8)	N (18)	P-value
Lesion location	Right Frontal lobe	2(%20)	2(%25)	4	0.3
	Right Parietal lobe	2(%20)	0	2	
	Left Internal capsule	2(%20)	0	2	
Lesion side	Right	7(%70)	5(%40)	12	0.6
	Left	4(%40)	2(%25)	6	
Depression's pattern	anxious	4(%40)	3(%37)	7	0.5
	melancholic	2(%20)	1(%5)	3	
	Atypical	2(%20)	1(%12)	3	
Severe depression		4(%40)	1(%12)	5	0.5

DISCUSSION

The prevalence of post-stroke depression in the present study was 36%. The prevalence was not different from previous studies[5]. Frontal lesion was an independence risk factor of PSD which agree with previous studies.[20] Reason might be explained by the essential role of prefrontal cortex in the emotional behavior and concentration through neural circuits which connect it with brainstem, diencephalon, limbic system and basal ganglia.[21-23] for example: frontal-subcortical circuits (FSC) and limbic-cortical-striatal-pallidal-thalamic circuits (LCSPTC). Another explanation is the contributions of the monoamine system. According to the theory, lesions in the brain cause unprecedented interruptions of the biogenic amine-containing axons, which ascend from the brainstem as it extends to the cerebral cortex.[24] and the disturbances caused by the lesions found in the frontal lobes prevent monoaminergic bundles from ascending in a normal and healthy pattern. One explanation for the correlation between PSD and right hemisphere is that lesions in our study were far of left frontal lobe because of the exclusion of aphasic patients who often have lesions in left frontal lobe. Nevertheless, our result was paralleled with many other studies.[25-27] No statistically significant association was found between PSD and stroke severity and disability at admission. This excludes the psychosocial hypothesis of PSD. The correlation between PSD and poor functional outcomes _represented by the change in NIHSS scores_ after stroke explain PSD's impact on rehabilitation process. The reason of absence of association between characteristics of the stroke and severity or type of PSD and suicidal ideation might be the small size of our sample which an important limitation of this study.

CONCLUSION

PSD has a significant impact on stroke outcome. Moreover, lesion in the frontal lobe, right hemisphere, and with ischemic type predict PSD, which helps to early detection and treatment as well as prevention of PSD.

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