Original Article ISSN (O):2395-2822; ISSN (P):2395-2814

Neuroanatomical Correlates of Post Stroke Depression

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Received: July 2019
Accepted: July 2019

ABSTRACT

Background: Depression after stroke is common entity known to psychiatrist from many decades. multiple theories have been given for post stroke depression ranging from functional disability, social support, emotional reaction to stroke, location of lesion etc. Considering lesion location as causative factor for post stroke depression multiple research have been done in last two decades with inconsistent results. The findings of past two decades of research indicates frontal lobe or lesion in left hemisphere as causative factor for post stroke depression with out reaching any definitive conclusion. Through this paper we have tried to find out the relationship between lesion location and post stroke depression. We have also tried to find out the relationship between size of lesion and severity of depression. **Methods:** cohort study design, tools used were CT scan, HAM-D, SPSS-22 software. **Results:** Infarct in left parital lobe resulted in significant depression as compared to infarct in right parital lobe. Over all depression post stroke was significantly more than non depressed in all areas. **Conclusion:** Lesions in left parietal lobe are significantly associated with occurrence of clinical depression. As the Size of lesion increases the risk of post stroke depression increases. Size of lesion has no positive or negative correlation with severity of depression

Keywords: Stroke, Depression.

INTRODUCTION

Depression is common in patients with brain diseases including stroke, multiple sclerosis, traumatic brain injury and Parkinson's disease.[1]
Prevalence of depression is higher in stroke patients, as compared to the estimated 10% prevalence of depression found in the general population,[2] Various studies have determined the prevalence of PSD to range from 11% to 63%.[3,4]
Depression following stroke has been associated with poor functional and rehabilitation outcomes,[5,6] increased mortality and reduced quality of life.[7,8]
It has been found out that the Stroke survivors are at greatly elevated risk for clinically significant depressive symptoms even years after stroke which is independent of functional disability or previous depressive symptoms.[9]
Though in stroke patients depression could be secondary to reaction to illness but the syndromal nature of depression and its imperfect correlation with measures of disability points towards possible anatomical cause1 one of which is location of stroke lesion.

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Stroke which is defined as a sudden loss of blood supply to the brain leads to permanent tissue damage, is caused by thrombotic, embolic or hemorrhagic event.[10] With 80% of cases are due to ischemic attacks, transient ischemic attacks, intracerebral and subarachnoidal hemorrhages being less frequent.[11]
Considering the high mortality rate due to stroke, in recent times it has been dropping due to a number of advances in the diagnosis, treatment, and management of acute stroke but the absolute number of patients of stroke are probably increasing due to the elderly population's extended life expectancy.
Common sequel of stroke are Neuropsychiatric consequences such as depression, apathy, anxiety, and cognitive impairment.[12]
The term post-stroke depression (PSD) is used in the literature to refer to the assessment of mood in stroke patients using either diagnostic criteria or rating scales.
This discrepancy in estimates of PSD prevalence is likely due to the high degree of methodological variability in such studies. These differing criteria include the study population and its inherent demographics, the research setting, the timing of depression assessment after stroke, the use of different rating scales and diagnostic criteria for depression, and the specific characteristics of the stroke one of which is anatomical location of the lesion.

Robinson et al in1981 published a series of seminal papers on the preferential location of stroke lesions in depressed patients using Hamilton depression rating scale (HAM-D) to demonstrate that the severity of depressive symptoms inversely correlated with the distance of the most anterior border of the lesion to the frontal pole in left hemisphere stroke and focal traumatic brain injury patients.[13]

In another study Robinson et al also demonstrated that the correlation between HAM-D measured depressive symptoms and a more anterior left hemisphere lesion location persisted over time and was significant at 3 and 6month follow-up.[14]

House et al in 1990 found no difference in left or right hemisphere stroke lesions with respect to Beck Depression Inventory (BDI) scores, there was no difference amongst the four lesion locations: left anterior, left posterior, right anterior, and right posterior.[15]

Singh et al in 2000 found that depressive symptoms were most significantly predicted by inferior frontal lesions.[16]

Kim and Choi-Kwon also in 2000 demonstrated that the prevalence of PSD was higher in the frontal-temporal region than parieto-occipital regions.[17]

Starkstein showed that patients with MCA strokes were more likely to be depressed than patients with brainstem or cerebellar strokes according to diagnosis derived from the PSE.[18] In another of their work publication examining stroke lesions in the basal ganglia and thalamus, which are both subcortical structures, demonstrated that patients with left basal ganglia lesions had a higher frequency and greater severity of depression than those with either right basal ganglia lesions or any sided thalamic lesions.[19]

From the above studies we can conclude that lesions in both cortical and subcortical regions of brain can lead to syndromal depression.

Size of lesion and severity of depression

In a study by Vataja et al in 2001 it was observed that depressed patients had significantly larger lesions in both the right and left caudate and pallidum.[20]

Furthermore, Nys et al in 2005 found a significant difference in lesion volume between patients with moderate to severe depressive symptoms, mild depressive symptom, and no depressive symptoms; patients with more severe symptoms had significantly larger lesions than patients with mild or no symptoms.[21]

The findings of Terroni et al. support these results with their observation that lesion volumes in the amygdala, ventral anterior cingulated cortex, subgenual cortex, hippocampal subiculum, and dorsal anterior cingulated cortex are significantly larger in patients with first major depressive episode post-stroke than non depressed patients.[22]

Tang et al. in 2010 and in 2013 failed to find a significant difference in lesion volume between depressed and non-depressed stroke patients.[23,24]

MATERIALS AND METHODS

The approval of our institutional ethics committee was obtained for this prospective study. Informed consent was obtained from all patients.

80 ischemic stroke patients with visual CT-based evidence of an acute brain infarct were recruited into this study. During the follow up ten patients dropped out of the study and were excluded from study. Therefore finally we had 70 patients as our study subjects. We chose cohort study design to conduct this study.

It examined the neuroanatomical correlates of PSD. Fresh CT scan of skull was performed. Ischemic stroke patients were recruited with visual CT-based evidence of an acute brain infarct. All the CT scans were evaluated by practicing radiologist. The exclusion criteria is patients with Intracranial hemorrhage, Severe aphasia that would preclude neuropsychiatric testing, Medical illness likely to cause psychiatric signs and symptoms, Cognitive impairment due to any reason, Past history of psychiatric illness, Alcohol dependence syndrome, Transient ischemic attack, Past history of stroke, Severe impairment in activities of daily living and Any disability accentuated by stroke.

Size of the infarct was measured.

Patients were assessed in terms of mood and cognition. Demographic information and medical history were obtained through hospital case sheets and patient interviews. Mood status was assessed using ICD-10 criteria F-32 for depression. Cognition status was assessed through MMSE test. Patients of stroke were treated and subsequently followed up in the same hospital. Patients were followed between three to six months of stroke for evaluation of symptoms of depression by using HAM-D. HAM-D has been used in many similar prior studies.

1. Purposive sampling method. CT images : MX 16 (model), 16 slice. ICD10 (diagnostic criteria for research) for diagnosing depression coded as (F 32)80.
2. Hamilton Depression Rating Scale [HAM-D]20 was used For evaluating depression. The version used has 24 items. Only items from 1 – 17 were used for scoring depression.
3. Items are scored from 0 to 2 or from 0 to 4 with total score ranging from 0 to 50. Score 7 or less = normal, 8 – 13 = mild depression, 14 – 18 = moderate depression, >19 = severe depression. HAM-D has good to excellent reliability and validity. we used Mini Mental State Examination[MMSE]81 Immediately after stroke to assess cognitive impairment. It is a 30 point cognitive test. It can be administered in less than 10 minutes, Reliability and validity is good to excellent, It is a useful test for

screening dementia. Any score below 25 was considered as dementia and were excluded from the study. And we used Modified BG Presad's classification for Socio Economic status (april 2016).

Statistical analysis:

1. Mean and standard deviation (S.D.) were obtained for quantitative data. Proportion and percentages were obtained for qualitative data.
2. Unpaired t-test was used to obtain the difference between mean of two groups.
3. Fisher's Exact test and Chi Square test were used to check the association between two parameters.
4. Analysis was done using MS-Excel and SPSS-22 software.

RESULTS

Table 1: The relationship between hemisphere lesion and occurrence of PSD

Hemisphere	No Depression	Depression	Total
Left	7	32	39
	17.9%	82.1%	100.0%
Right	3	11	14
	21.4%	78.6%	100.0%
Bilateral	5	12	17
	29.4%	70.6%	100.0%
Total	15	55	70
	21.4%	78.6%	100.0%
Pearson Chi-Square 0.924		**P value 0.630**	

This table presents number and percentage of depressed and non-depressed patients in left hemisphere lesion, right hemisphere lesion and bilateral lesion. The imbalance in the laterality of strokes (left: 39, right: 14) is evident in these data. 39 patients had left sided infarcts, while 14 patients had infarcts in the right hemisphere. 17 patients had infarct in bilateral hemisphere. 82.1% patients with left hemisphere infarct had syndromal depression, 78.6% patients with right hemisphere infarct had syndromal depression and 70.6% patients with bilateral infarct had depression.

There is no association between part of hemisphere and presence of depression (p value 0.630).

Using HAM-D score threshold of >7, 78.57% of patients screened positive for syndromal depression, which is more than prior studies.

Table 2: The relationship between lesion location and occurrence of PSD

Hemisphere	No Depression	Depression	Total	z value	p value
L[Frontal]	0	0	0	0	0
L[Multiple]	1	14	15	1.572	0.116
L[Occipital]	0	4	4	-	-
L[Sub cortical]	1	10	11	1.086	0.276
L[Temporal]	1	0	1	-	-
L[Parietal]	4	4	8	2.093	0.037
R[Frontal]	0	1	1	-	-
R[Multiple]	0	3	3	-	-
R[Occipital]	1	3	4	0.179	0.857
R[Parietal]	1	1	2	0.999	0.317
R[Sub cortical]	1	3	4	0.179	0.857
R[Temporal]	0	0	0	0	0
Multiple	5	12	17	0.922	0.358
Total	15	55	70	6.7612	0.000

This table comparing different neuroanatomical locations between depressed and non depressed patients post stroke shows overall the proportion of depressed patients is significantly more than non-depressed. Significance is present in left parietal lobe [p value 0.037]. The positive correlation observed in the left parietal lobe indicates that infarction in this region will lead to more depression when compared with right parietal lesion.

Table 3: size of lesion according to hemisphere

	N	Mean volume	Std. Deviation	Minimum	Maximum	significance
L	39	21.4103	11.83820	7.00	60.00	F = 0.434, P = 0.650
R	14	22.2857	16.45707	8.00	74.00	
M	17	24.9412	12.72041	11.00	56.00	
Total	70	22.4429	12.95660	7.00	74.00	

This table Show the mean of size of lesion in left hemisphere is 21.41 mm2 SD 11.83 ; in right hemisphere is 22.28 mm2 SD 16.45, for multiple lesion is 24.94 mm2 SD 12.72. The mean size of lesion of entire study group is 22.44 mm2 SD 12.95.

Table 4: The relationship between lesion size and occurrence of PSD

Depression	N	Mean of Size of Lesion	Std. Deviation	Std. Error Mean	Significance
no depression	15	18.0667	6.00555	1.55063	t = 2.272 p =0.027
Depression	55	23.6364	14.08488	1.89920	

This table show that there is statistically significant difference in mean of size of lesion between depressed and non-depressed patients. Depressed patients have larger size than non-depressed patients.

Table 5: Correlation between HAM-D score and size of lesion in patients having post stroke depression

N = 55	p value 0.785 (not significant)
	Pearson correlation = -0.038

There is low degree negative correlation which is not significant

This table show relationship between size of lesion and HAM-D score: there is no correlation between size of lesion and severity of post stroke depression.

DISCUSSION

This paper sought to elucidate relationship between lesion location [brain infarcts] with depression. The past three decades have failed to reach a consensus regarding the neuroanatomical correlates of PSD, although anterior infarcts or those in the basal ganglia appear to engender the greatest risk.[25,26] The past decade has however witnessed more analytically sophisticated research that incorporated our modern understanding of anatomical brain structure and function.

We found out 78.57% of our patients had clinical depression. Review of 14 studies by Whyte and Mulsantin 2002 indicated that the prevalence of major depression at one to two months post-stroke ranged from 9- 37%.[27] A wider range of 17-69% prevalence of depression following stroke has been reported among stroke survivors in Hong Kong.[28,29]

Our study population was comparable to other studies considering time since stroke which is 90 days to 180 days which is similar to other studies and taken so to avoid erroneously including patients with depressive reaction to stroke.

The mean (± SD) lesion size for this patient cohort was 22.44 ± 12.96 mm2, which is well within the range established by previous studies.[20,21,23]

The novel methodological refinement of this study was the ability to quantitatively assess the degree of tissue infarction by hand tracing on CT film. Previous studies had only qualitatively rated brain regions as being impinged upon or not, which is subject to greater observer bias. These measurements were necessary to explore the secondary hypothesis, which assess correlation between sizes of brain infarct with severity of depressive symptoms.

The correlational analyses related to the primary hypothesis did not generate any results except infarct in left parietal lobe was associated with significantly more depression than right parietal lobe [p value 0.037] and overall the proportion of depressed patients was significantly more than non-depressed patients in all areas [p value 0.000].

While four of ten studies that we studied did not determine any significant associations between depressive symptoms and stroke location in terms of hemisphere, neuroanatomy, or vascular territory affected three studies found a non-lateralized association between PSD and frontal lobe or basal ganglia strokes,[20,21,28-32] one study determined depression more with fronto-temporal than with parieto-occipital region,[17] one identified a right hemisphere frontal lobe stroke association16 and another determined a left sided basal ganglia stroke association.[33]

Due to less number of cases involving frontal areas we are not able to comment over depression due to infarct in frontal lobe which is postulated in many studies till now.[18,25,26,34,35]

The lack of accord among the past 30 years of PSD neuroimaging research and the negative findings of this study related to the primary hypothesis are possibly due to the contribution of factors other than lesion location. Biological and psychosocial causes have been proposed to work in tandem to institute depressive symptoms following stroke.[27] Physical disability, in the form of either stroke severity or functional impairment, is most commonly associated with PSD.[9,16,35-37]

Hackett and Anderson also concluded, there were significant relationships between the levels of social support and the occurrence of depression or depressive symptoms.[38] Another recent meta-analysis by Ayerbeet al.found that disability after stroke, personal history of depression pre-stroke, cognitive impairment, stroke-severity, lack of social support, and anxiety were all important factors in causing depression.[39]

While subsequent studies assessing PSD have found associations with white matter changes (WMC) and brain atrophy as factors for PSD.[24,40]

Variations in the methods of selecting the study population, i.e. inclusion/exclusion criteria, assessment time points, and lack of an operational definition of PSD in order to determine appropriate diagnoses, are all factors involved in the lack of consensus on the prevalence of PSD.[36,41]

At this time, mixed findings in this area of PSD research demonstrate that more work must be done to clarify the association between depression and lesion location.[42]

The second hypothesis of this study was to compare between size of lesion and severity of post stroke depression. We compared size of lesion with HAM-D score of depressed patients and excluded non depressed patients. As such, lesions were expected to institute greater depressive symptoms as their severity increased in terms of the extent of brain infarction. This concept of interacting mood and lesion gradients has not been explored by any previous studies. We didn't find significance in relation to second hypothesis[p value 0.785], though we found that incidence of depression is significantly associated with size of lesion [p =0.027] i.e. larger the size of lesion more are the chances of clinical depression.

CONCLUSION

1. Lesions in left parietal lobe are significantly associated with occurrence of clinical depression.
2. In this study, the findings suggest that as the Size of lesion increases the risk of post stroke depression increases.
3. Size of lesion has no positive or negative correlation with severity of depression.

Limitations

1. We didn't include white matter changes and atrophy in stroke lesion.
2. Use of CT based imaging in our study as compared to MRI based imaging in many other previous studies which give more precise image.
3. Small sample size which couldn't get significant number of patient with lesion in many brain areas.
4. Functional impairment was not specifically assessed.

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How to cite this article: Khandelwal A, Kulkarni SV, Gaikwad SJ. Neuroanatomical Correlates of Post Stroke Depression. Ann. Int. Med. Den. Res. 2019; 5(5):PY01-PY06.

Source of Support: Nil, **Conflict of Interest:** None declared