The prevalence of Post Stroke Depression (PSD) & its relationship with post stroke disability and lesion localization

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Abstract

Introduction: Depression is a common neuropsychiatric sequel of stroke. PSD (Post stroke depression) can be due to psychological reaction to clinical consequences of stroke like disability or can be consequences of specific brain lesion. PSD is associated with poor long term functional and rehabilitation outcome.

Aims:

1. To determine the prevalence of PSD and its association with socio-demographical factors.
2. To study PSD and its association with post stroke disability.
3. To study the relationship between PSD & side and site of lesion.

Materials and Methods: Consecutive 52 patients with diagnosis of cerebro-vascular stroke were included in the study. The written informed consent was taken. Detailed socio-demographical variables, stroke related factors, medical and psychiatric history was obtained using a semi structured Performa. Neuro-imaging provided information on lesion location. Subjects were given HAM-D to assess depression and WHODAS 2.0 to assess disability.
Collected data analyzed using SPSS version 16.

Results: Of 52 patients, 38 were depressed (73.08%). The association between the disability and PSD was nearly significant. The association between PSD and side of lesion was not significant. The association between the site of lesion and PSD was significant. Association between bilateral cortical and sub cortical lesion with PSD was also found significant.

Conclusions: The results of our study show the high prevalence of PSD and its correlation with site (cortical and sub cortical) of lesions as well as severity of disability, which support biopsychosocial model of post stroke depression.

Keywords: Disability, Lesion Localization, Post stroke depression, Stroke.

Introduction

Stroke is defined by WHO as a rapid development of clinical signs and symptoms and focal neurological disturbances lasting for more than 24 hrs or leading to death with no apparent cause other than vascular origin.[1] Stroke is the third leading cause of death and one of the most disabling diseases, with an adverse impact on functional outcome.[2]

A common neuropsychiatric sequel of stroke is depression, with quoted rates of post stroke depression (PSD) ranging from 18% to 61%.[3-5] PSD is commonly associated with difficulty in rehabilitation, higher risk of recurrence of stroke, poor long term functional outcome and higher post stroke mortality.[6-8]

Finnstroke et al[2] found that post stroke depression is associated with poor functional recovery of patients and disability emerged as a significant independent contributor to depression. Sunnybrooke et al[3] found significant correlation between depressive symptoms and measures of activity of daily living and social handicap.

Etiologies of PSD have been explained by many theories. One states that it can be a psychological reaction to disability, like stroke sequel and other states that it can be a consequence of specific brain damage, changes in neurotransmitters and cerebral atrophy.

Although neurological theories have been widely accepted, attempts to replicate them have yielded conflicting results. The crude stroke prevalence in different parts of India range from 44.29 to 559/100,000 persons and the cumulative incidence of stroke range from 105 to152/100,000 persons per year during past two decades.[9] The global burden of disease study found the overall number of reduced DALYs (Disability-Adjusted Life Years) caused by stroke has increased worldwide from 86,010 in 1990 to 102,232 in 2010.[10] Studies found that post stroke quality of life (QoL) is reported to decrease by more than 40% compared with pre-stroke QoL.[11] It is also estimated that worldwide 25 to 74% of the stroke survivors require some assistance or are fully dependent on caregivers for activities of daily living (ADL).[12]

A decrease in stroke mortality observed in the last few years due to better health care services, the subsequent increase of survivors with residual impairments and disabilities[13] has prompted health care professionals to know more about post stroke sequels and provide better services.

In this study, we aimed to assess the above theories, the prevalence of depression and its association with lesion location (neurological) and disability (psychological consequences).

Aims and Objectives

1. To determine the prevalence of post stroke depression and its association with socio-demographical factors.
2. To study PSD and its association with post stroke disability.
3. To study the relationship between PSD & side and site of lesion.

Materials and Methods

Inclusion criteria: Patients with clinical & radiological diagnosis of cerebro-vascular stroke after 4 weeks of stroke.

Exclusion criteria

1. Acute confusional state due to general medical condition or substance use.
2. Acute Stroke with altered sensorium.
3. Aphasia.
4. History of depression at the time of or before stroke.
5. History of Dementia and significant cognitive disturbances.
6. Patients who did not give consent.

The study was approved by the institutional ethics committee of C. U. Shah medical college, Surendranagar, Gujarat. Study was carried out in same tertiary care hospital in Surendranagar district, Gujarat, India during August 2015 to January 2016. This was a cross-sectional, observational study.

Consecutive 52 patients with a diagnosis of cerebro-vascular stroke attending the department of medicine & department of physiotherapy, who satisfied the inclusion and exclusion criteria, were included in the study. The written informed consent was taken from each participant. A detailed medical and psychiatric history was obtained. Socio-demographical variables and stroke related factors were obtained using a semi structured Performa. Neuro-imaging in the form of Computed tomography & Magnetic Resonance imaging films brought by the patient were used to get information on side and site of lesion. Localization of stroke was divided into right hemispheric, left hemispheric and bilateral infarcts. Site of lesions were divided into cortical, sub-cortical, cerebellum and brainstem lesions. Disability was evaluated with WHODAS 2.0 and Depression was evaluated with HAM-D.

Scales used:

HAM-D (Hamilton Rating Scale for Depression)[14]: HAM-D has 17 items such as depressed mood, guilt, suicide, insomnia (initial, middle, delayed),work and activities, retardation, agitation, anxiety (psychic),anxiety(somatic), somatic symptoms (gastrointestinal, general, genital), insight, loss of weight. Items are scored from 0-2 or from 0-4 with total score ranging from 0-50. Scores of 7 or less is considered normal; 8-13 is considered mild; 14-18 is considered moderate; 19-22 is considered severe; 23 and above is considered very severe. It has good to excellent reliability and good validity.

WHODAS 2.0(World Health Organization Disability Assessment Schedule 2.0)[15] WHODAS 2.0 covers 6 Domains of Functioning, including:

1. Cognition – understanding & communicating
2. Mobility– moving & getting around
3. Self-care– hygiene, dressing, eating & staying alone
4. Getting along– interacting with other people
5. Life activities– domestic responsibilities, leisure, work & school
6. Participation– joining in community activities

Complex Method

The more complex method of scoring is called "item-response-theory" (IRT) based scoring. It takes into account multiple levels of difficulty for each WHODAS 2.0 item. It takes the coding for each item response as "none", "mild", "moderate", "severe" and "extreme" separately, and then uses an algorithm to determine the summary score by differentially weighting the items and the levels of severity. The SPSS algorithm is available from WHO which we have used for our results. The scoring has three steps:

Step 1 - Summing of recoded item scores within each domain.

Step 2 - Summing of all six domain scores.

Step 3 - Converting the summary score into a metric ranging from 0 to 100 (where 0 = no disability; 100 = full disability).

0-4% - No Problem

5-24% - Mild Problem

25-49% - Moderate Problem

50-95% - Severe Problem

96-100% - Complete Problem

Descriptive statistics were used to determine the socio-demographical and stroke related variables. Chi square test was used to find the significance of study parameters on categorical scale between two or more groups (p value was set at less or equal to 0.05). Collected data was analyzed using SPSS version 16.

Results

Prevalence of PSD

Patients with depression were compared to those without depression on various parameters. 52 patients were included in this study. Of the 52 patients, 38 were depressed (73.08%). Among these 14 had mild (36.84%), 10 had moderate (26.32%), 6 had severe (15.79%) and 8 had very severe (21.05%) depression on HAM-D.

Table 1: PSD & its association with socio-demographical variables and stroke related variables

Variables	Socio demographic variables	Depressed (38) (73.08%)	Non depressed (14) (26.92%)	Chi square	DF	P value
Age	20-35	02 (5.26%)	01 (7.14%)	1.66	3	0.76
	36-55	21 (55.26%)	08 (57.14%)			
	56-64	10 (26.32%)	02 (14.29%)			
	65 & >	05 (13.16%)	03 (21.43%)			
Sex	Male	26 (68.42%)	12 (85.71%)	1.55	1	0.21
	Female	12(31.58%)	02 (14.29%)			
Occupation	Unemployed	24(63.16%)	08 (57.14%)	1.6	3	0.64
	Inconsistent earning	06 (15.79%)	04 (28.57%)			
	Consistent monthly pay but not permanent	06 (15.79%)	02 (14.29%)			
	Permanent pay	02(5.26%)	00 (00%)			
SEC	I	02 (5.26%)	01 (7.14%)	3.07	4	0.54
	II	07 (18.42%)	01 (7.14%)			
	III	11 (28.95%)	03 (21.43%)			
	IV	12 (31.58%)	04 (28.57%)			
	V	06 (15.79%)	05(35.72%)			
Type of family	Joint	15(39.47%)	06(42.86%)	0.04	1	0.82
	Nuclear	23(60.53%)	08(57.14%)			
Education	Literate	21 (55.26%)	09(54.29%)	0.34	1	0.55
	Illiterate	17(44.74%)	05(35.71%)			
Marital Status	Single	02(5.26%)	00	1.83	2	0.40
	Married	34(89.48%)	12(85.71%)			
	Widow	02(5.26%)	02(14.29%)			
Area of residence	Rural	29(76.32%)	12(85.71%)	0.54	1	0.46
	Urban	09(23.68%)	02(14.29%)			
Religion	Hindu	35(92.11%)	12(85.71%)	0.48	1	0.49
	Muslim	03(7.89%)	02(14.29%)			
Duration from stroke	1-2 months	09 (23.68%)	03 (21.48%)	5.08	5	0.40
	>2-6 months	14 (36.85%)	02 (14.29%)			
	>6-12 months	07 (18.42%)	06 (42.86%)			
	>12-24 months	06 (15.79%)	02 (14.29%)			
	>2-5 years	01 (2.63%)	01 (7.14%)			
	>5-10 years	01 (2.63%)	00			
P/H/O Stroke	Present	08(21.05%)	01 (7.14%)	1.38	1	0.23
	Absent	30(78.95%)	13(92.86%)			
Number of stroke	1	30(78.95%)	13(92.86%)	1.65	3	0.64
	2	05(13.16%)	01(7.14%)			
	3	02(5.26%)	00			
	4	00	00			
	5	01(2.63%)	00			

No socio-demographical variables and stroke related variables were significantly associated with PSD.

Table 2: PSD & side of lesion

Lesion related variables	Depressed	Non depressed	Chi square	DF	P value
Right	11 (28.95%)	04 (28.58%)	2.147	3	0.542
Left	19 (50.00%)	05 (35.71%)			
Both	07 (18.42%)	05 (35.71%)			
No lesion	01 (2.63%)	00			

Among depressed patients, 19 patients (50%) had left sided lesion, 11 patients (28.95%) had right sided lesion, 7 patients (18.42%) had both sided lesion. The association between PSD and side of lesion was not significant as chi square value was 2.147 and p value: 0.54

Table 3a: PSD & site of lesion

Lesion related variables	Depressed	Non depressed	Chi square	DF	P value
Cortical	11 (28.95%)	04 (28.58%)	13.993	5	0.016
Sub cortical	06 (15.79%)	09 (64.28%)			
Cortical + sub cortical	17 (44.74%)	01 (7.14%)			
Cerebellum + brainstem	02 (5.26%)	00			
Cerebellum + brainstem + sub cortical	01 (2.63%)	00			
Normal	01 (2.63%)	00			

17 patients (44.74%) had cortical & sub cortical both site of lesion, 11 patients (28.95%) had cortical, 6 patients (15.79%) had sub cortical, 2 patient (5.26%) had cerebellar &brainstem, 1 patient (2.63%) had cerebellar, brainstem & sub cortical site of lesion. 1 patient (2.63%) had normal CT scan report. The association between the site of lesion (cortical and subcortical) and PSD was significant as chi square value: 13.993 and p value: 0.016

Table 3b: Categorical analysis of laterality of lesion and site of lesion

Laterality of lesion	Site of lesion	Depressed	Non-depressed	Chi square	DF	p value
Left	Cortical	4 (10.53%)	2 (14.29%)	3.789	3	0.29
	Sub cortical	6 (15.79%)	3 (21.43%)			
	C+SC	8 (21.05%)	0			
	CE+BS	1 (2.63%)	0			
Right	Cortical	6 (15.79%)	1 (7.14%)	3.117	2	0.21
	Sub cortical	1 (2.63%)	2 (14.28%)			
	C+SC	4 (10.53%)	1 (7.14%)			
Both	Cortical	1 (2.63%)	1 (7.14%)	10.888	4	0.03
	Sub cortical	0	4 (28.58%)			
	C+SC	5 (13.16%)	0			
	SC+CE+BS	1 (2.63%)	0			
	Normal	1 (2.63%)	0			

Association between bilateral cortical and sub cortical lesion with PSD was also found significant as chi square value: 10.888 and p value: 0.03

Table 4: PSD & Disability

Severity of disability	Depressed	Non depressed	Chi square	DF	P value
None	00	00	5.217	2	0.074
Mild	07 (18.42%)	07 (50.00%)			
Moderate	19 (50.00%)	04 (28.58%)			
Severe	12 (31.58%)	03 (21.42%)			
Extreme	00	00			

Among depressed patients, 7 patients (18.42%) had mild disability, 19 patients (50%) had moderate disability and 12 patients (31.58%) had severe disability. The association between the severity of disability and PSD was nearly significant as chi square value: 5.217 and p value: 0.074.

Discussion

We studied prevalence of Post stroke depression and its correlation with lesion localization and disability, among hospital based sample.

In our study, the prevalence of PSD was found to be 73.08%. Our study was similar to Indian studies by Srinivaso et al.[16] and Rajashekharan et al,[17] who found prevalence of major depression in hospital based sample to be 62.5% and 45.16%, in their respective studies. In another Indian study; by Srivastava et al,[18] the prevalence of PSD was found to be 35.29% in rehabilitation centres.

Study by Berg et al.,[19] reported prevalence rate of 50%. Litton J et al.,[20] reported prevalence of severe depression was 47% and mild depression was 53%. Few landmark studies (studies by Herman et al.,[3] starkstein et al.,[4] and Folstein et al.[5] reported prevalence of PSD ranging from 18-61%.

However, all these studies were done at different time periods following stroke, in different population samples (hospital based or rehabilitation based) and using different measures to score depression. Our population was hospital based so the prevalence of PSD might be higher than other community or rehabilitation based study population.

Demographical variables play an important role in development of PSD. However, most of the studies done on significance between socio-demographical variables and PSD gave contradictory findings.

In our study, no socio-demographical variables were significantly associated with PSD. Our finding were similar with study by Srinivasa Rao et al.[16] which found no significant association between PSD and socio-demographic factors like religion, education, occupation, family types, SEC, residence. Another study by Magloire Nkosi Mpembi et al.[21] also found no significant association between PSD and socio-demographic factors like age, sex, occupation and religion.

Study by Hermann et al.,[22] which reported no associations between PSD and socio-demographical variables like age and marital status. Most studies[23,24] have not found a correlation between age and PSD.

There are a few studies which are contradictory to our results, they found that PSD was associated with younger age, female gender, domicile, marital status and socio-economical class.[18,25,26]

In our study, no stroke related variables were significantly associated with PSD. This is similar to the study by Srivastava et al.,[18] which reported PSD was not related to post stroke duration. In present study association between PSD and number of stroke was not found significant, which is in contrast with study the by Paolucci et al.[27] which reported previous history of stroke having significant association with PSD.

The association between PSD and side of lesion was not significant in our study. Our finding was somewhat similar to the study by; Sato et al.,[28] and Srivastava et al.,[18] who in their respective studies , found PSD to have no correlation with lesion location. Another study by Aben et al.,[29] found no significant association between side of lesion and development of PSD.

Some studies[30-33] found that PSD was associated with right sided stroke whereas other studies[20,34-39] reported PSD to be associated with left sided stroke.

In the present study, association between the site of lesion (bilateral cortical & sub cortical) and PSD was significant similar to the study by Rajashekaran et al.[17] who also found that PSD was associated with cortical & sub cortical lesions.

A systemic review study by Carson et al.,[40] offered no support for the hypothesis that the risk of depression after stroke was affected by the location of the brain lesion. In the study by Singh et al.[41] association was found to change over time.

Considering differing subject selection (inpatients or outpatients, hospital based or rehabilitation based), the methodological variations like instrumentation (self report scales or structured interview) in the various studies, comparison is difficult among the findings of various studies and due to such limitations no significant conclusion could be reached.

Association between PSD and disability was nearly significant. This result is similar to the study by Pohjasavaara et al.,[42] who found that PSD was weakly associated with Disability. Also study by Miller et al.[10] reported that 25% to 75% of the 50 millions stroke survivors worldwide require some assistance or are fully dependent on caregivers for activities of daily living. Number of studies[43-45] report a relationship between PSD and physical functioning. While the direction of the relationship between PSD and disabiliy has not been established, it is clear that early identification and treatment of PSD is beneficial to physical recovery.

There are no published data on PSD and its relation with post stroke disability; using WHODAS 2.0.

Conclusion

The results of our study show the high prevalence of PSD and its correlation with site (bilateral cortical and sub cortical) of lesions as well as severity of disability. It is difficult to determine whether depression is due to clinical consequences of stroke or due to neurophysiological changes that may lead to depression. PSD may reflect a more complex phenomenon than merely the neuro-anatomic loci of brain damage or functional disability. Previous research has tended to focus either on the patho-physiological pathways or to important contributing psychosocial factors. Thus, the synopsis of all contributing factors into one holistic biopsychosocial model should be the main goal. This kind of study would provide us more insight into biological and psychosocial factors and their complex interactions. It will assist clinicians in the early identification of patients at higher risk for mood disturbances and those who are most likely to get benefit from treatment interventions. This, in turn, might lead to shortened hospital stays, contained healthcare costs, improved quality of life, and reduced mortality.

Limitations

The population of this study was not representative of the general population.

The exclusion and inclusion criteria were specific. It is quite possible that excluded patients (patients with acute confusional state, patients with aphasia, patients without brain imaging) would have had depression and disability. It was difficult to assess psychomotor activity in subjects with paralysis. Social support was not assessed in this study which could be a confounding factor.

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