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Research Article

A Study Predicting The Prevalence Of The Depression And The Anxiety And Their Association Among The Post Stroke Survivors.

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Running Title : Depression And Anxiety in Stroke Patients.

Abstract

Objective: This study was conducted in post stroke patients to assess the prevalence of depression and its correlation with anxiety , to give an account of patients experience between self and others and to generate a data for clinicians and other health care providers to consider these important predictors when assessing and managing depression and anxiety among stroke patients for a value-added treatment that could address both conditions in patients to enhance functional recovery and ease the burden of patient care

Methods: The patients which fulfilled the inclusion criteria were enrolled for the study. After taking an informed consent and after a detailed explanation about the questionnaire the patients were provided with the Hospital Anxiety and Depression Scale (HADS) and were asked to tick the box beside the reply that is closest to how they have been feeling in the past week. The data collected was subjected to data analysis.

Results: The mean± SD scores of depression were 11.70±2.377 and 12.65±2.779 with the p-value of 0.02 ≤0.05 which is statistically significant in order words the relationship between anxiety and depression scores is likely not due to the chance and r-value of 0.37, indicates moderate positive correlation. This means that as anxiety scores increase, depression scores tend to increase.

Conclusion: There is a moderate positive correlation between anxiety and depression scores, and this relationship is statistically significant. PSA is less related to stroke than PSD since its primary predictors comprise lifetime anxiety disorders, including obsessive-compulsive disorder (OCD) and agoraphobia without panic disorder. PSD is less predictable than PSA as a post-stroke affective disorder because it is stroke-related.

Keywords : Stroke, Depression, Anxiety , Neural Plasticity, Neural transmission.

INTRODUCTION

Depression is the most frequent psychiatric condition after stroke and is associated with negative health outcomes. Prevalence of depression is said to be 24% (95% CI 21 to 28) by clinical interview and 29% (95% CI 25 to 32) by rating scales.[1]

Routine screening for PSD is recommended[2,3] However, guidelines differ about the optimal time the stroke survivors should be screened for PSD. Patients with early-onset (within 3 months after stroke) depression have a high risk of experiencing persistent depression.[1]

Post-stroke depression (PSD) and post-stroke anxiety (PSA) are common with stroke.[4] About 30% of stroke survivors clinically have PSD symptoms at some point following stroke[5] and PSA prevalence is around 20-25%.[6] PSD leads to 3.4-7 times higher mortality rate than is seen among patients without PSD.[7]

Anxiety and depression may also co-occur in patients with stroke and in two systematic literature reviews, PSD has been positively correlated with PSA.[8] This would suggest a need for a value-added treatment that could address both conditions in patients to enhance functional recovery and ease the burden of patient care.

The assessment of PSD across sites and studies is highly variable in terms of methods, instruments, cut-off scores, definitions of depression and types of depression, settings, and time points of assessment and different screenings have been employed by a clinician for depression assessment[9] like Hospital Anxiety and Depression Scale (HADS).[10] Center for Epidemiologic Studies of Depression Scale (CESD).[11] Hamilton Depression Rating Scale (HAMD or HDRS).[12] Patient Health Questionnaire-9 (PHQ-9).[13] Beck Depression Inventory (BDI);[14] and Geriatric Depression Scale (GDS) .[15]

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PSA has been closely associated with PSD and tends to endure for longer periods when the two conditions co-occur.[16-17] Pre-stroke depression, stroke severity, early anxiety, and dementia or cognitive impairment after stroke appear to be predictive of post-stroke anxiety.[18] PSA has been typically assessed by the GAD-7.[19] the Beck Anxiety Inventory (BAI).[20] HADS.[21] and the Hamilton Anxiety Rating Scale (HAM-A).[22]

Anxiety is associated with poor self-control, immobility, and fatigue.[23] It can compromise rehabilitation outcome and negatively impact the quality of life after stroke.[24]

Schottke and Giabbiconi[25] found a significant correlation between PSD prevalence and PSA prevalence. The two disorders are primarily associated with the experiences and consequences of the stroke itself. Another study by Fang et al. [26] showed that about 30 percent of stroke survivors had depressive symptoms while 20–25 percent experience signs of anxiety. Though PSD and PSA share a vital link, the predictors and diagnosis of the conditions vary slightly. PSA is less related to stroke than PSD since its primary predictors comprise lifetime anxiety disorders, including obsessive-compulsive disorder (OCD) and agoraphobia without panic disorder. In contrast, PSD cannot be predicted by lifetime depression. Therefore, PSD is less predictable than PSA as a post-stroke affective disorder because it is stroke-related [27] However, both PSD and PSA may result in adverse neurological and functional deficits if psychological interventions are not implemented in time. The relationship between PSD and PSA can be best elaborated by looking into the rehabilitation services provided to patients suffering from the disorders, as well as shared diagnostics, comorbidity, severity, etiology, and risk factors.

This study was conducted in post stroke patients to assess the prevalence of depression and its correlation with anxiety to give an account of patients experience between self and others and to generate a data for clinicians and other health care providers to consider these important predictors when assessing and managing depression and anxiety among stroke patients for a value-added treatment that could address both conditions in patients to enhance functional recovery and ease the burden of patient care. Paolucci et al.[28] argue that when not treated or managed quickly, PSD and PSA have the potential to increase disability, which is usually caused by neurological impairment, by more than fifteen percent in stroke survivors. However, PSD leads to poor functional and cognitive recovery, which at times makes it difficult for the affected person to participate in rehabilitation.

Our study was based on descriptive correlation design. The patients which fulfilled the inclusion criteria were enrolled for the study. The patients were provided with the Hospital Anxiety and Depression Scale (HADS) and were asked to tick the box beside the reply that is closest to how they have been feeling in the past week.

SCALES

Hospital Anxiety and Depression Scale (HADS)

Which involves a series of questionnaires mentioned at **Table 1** was used to calculate the total scores of Depression (D) _____ and Anxiety (A) _____ for each patient. The HADS is a validated scale consisting of 14 questions, seven for depression and seven for anxiety. Each question has four responses with a score of 0-3 , with higher scores indicating greater frequency of symptoms.

Instructions: Read each item and circle the reply which comes closest to how you have been feeling in the past week. Don't take too long over your replies: your immediate reaction to each item will probably be more accurate than a long thought-out response.

Table 1. Hospital Anxiety and Depression Scale (HADS).

Item	Anxiety Score	Item	Depression Score
1. I feel tense or wound up			1. I feel as if I am slowed down:
Most of the time	3	Nearly all of the time	3
A lot of the time	2	Very often	2
Time to time occasionally	1	Sometimes	1
Not at all	0	Not at all	0
2. I get a sort of frightened feeling like 'butterflies in the stomach':		2. I still enjoy the things I used to enjoy:	
Not at all	0	Definitely as much	0
Occasionally	1	Occasionally Not quite so much	1
Quite often	2	Only a little	2
Very often	3	Not at all	3
3. I get a sort of frightened feeling like something awful is about to happen:		3. I have lost interest in my appearance:	
Very definitely and quite badly	3	Definitely	3

Yes, but not too badly	2	I don't take as much care as I should	2
A little, but it doesn't worry me	1	I may not take quite as much care	1
Not at all	0	I take just as much care as ever	0
4. I feel restless as if I have to be on the move:		4.I can laugh and see the funny side of things:	
Very much indeed	3	As much as I always could	0
Quite a lot	2	Not quite so much now	1
Not very much	1	Definitely not so much now	2
Not at all	0	Not at all	3
5. Worrying thoughts go through my mind:		5. I look forward with enjoyment to things:	
A great deal of the time	3	A much as I ever did	0
A lot of the time	2	Rather less than I used to	1
From time to time but not too often	1	Definitely less than I used to	2
Only occasionally	0	Hardly at all	3
6. I get sudden feelings of panic:		6. I feel cheerful:	
Very often indeed	3	Not at all	3
Quite often	2	Not often	2
Not very often	1	Sometimes	1
Not at all	0	Most of the time	0
7. I can sit at ease and feel relaxed:		7. I can enjoy a good book or radio or TV programme:	
Often	0	Definitely	0
Sometimes	1	Usually	1
Not often	2	Not often	2
Very seldom	3	Not at all	3

Study population

Inclusion Criteria

1. Both males and females
2. 2-3 months post stroke
3. Ability to follow and understand commands.
4. 18–60 years old

Exclusion Criteria

1. Inability to follow and understand commands.
2. A confirmed history of epilepsy before enrolment
3. History of previous brain injury.

Sample size

Forty patients were recruited from the outpatient settings.

Methodology

The patients which fulfilled the inclusion criteria were enrolled for the study. After taking an informed consent and after a detailed explanation about the questionnaire the patients were provided with the Hospital Anxiety and Depression Scale (HADS) and were asked to tick the box beside the reply that is closest to how they have been feeling in the past week. The data collected was subjected to data analysis.

Data Analysis

The data analysis was carried out by using SPSS Software. Pearson's Correlation coefficient was used to measure the correlation between depression and anxiety as summarized at Table 3. In this study the mean± SD scores of depression were 11.70±2.377 and 12.65±2.779 with the p-value of 0.02 ≤0.05 as summarized at Table 2 and **Figure 1** and **Figure 2** which is statistically significant in order words the relationship between anxiety and depression scores is likely not due to the chance and r-value of 0.37, indicates moderate positive correlation. This means that as anxiety scores increase, depression scores tend to increase as well, but the relationship is not particularly strong. **Figure 3** summarizes the corelation heatmap of depression and anxiety scores.

Table 2. Mean± SD Scores of Anxiety and Depression.

	Depression Scores	Anxiety Scores
Mean	11.70	12.65
Std. Deviation	2.377	2.779
Std . Error of Mean	0.3759	0.4393

Table 3. Pearson's Correlation Coefficient, P and r value.

Pearson r	
r	0.3719
95% confidence interval	0.06828 to 0.6124
R squared	0.1383
P value	
P (two tailed)	0.0181
Significant (alpha=0.05)	Yes
Number of XY Pairs	40

Figure 1. The Mean ±SD of Anxiety and Depression scores in post stroke patients.

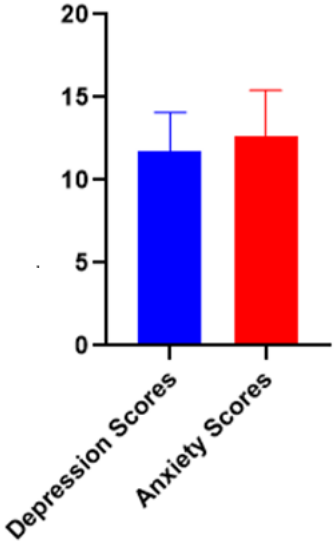


Figure 2. Corelation between Depression and Anxiety scores in post stroke patients

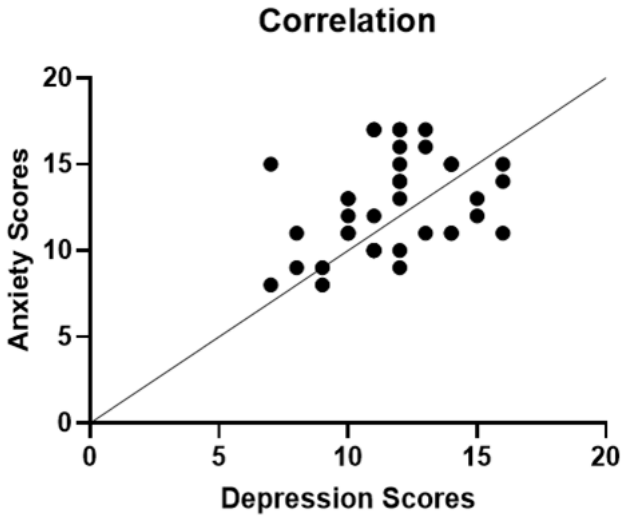
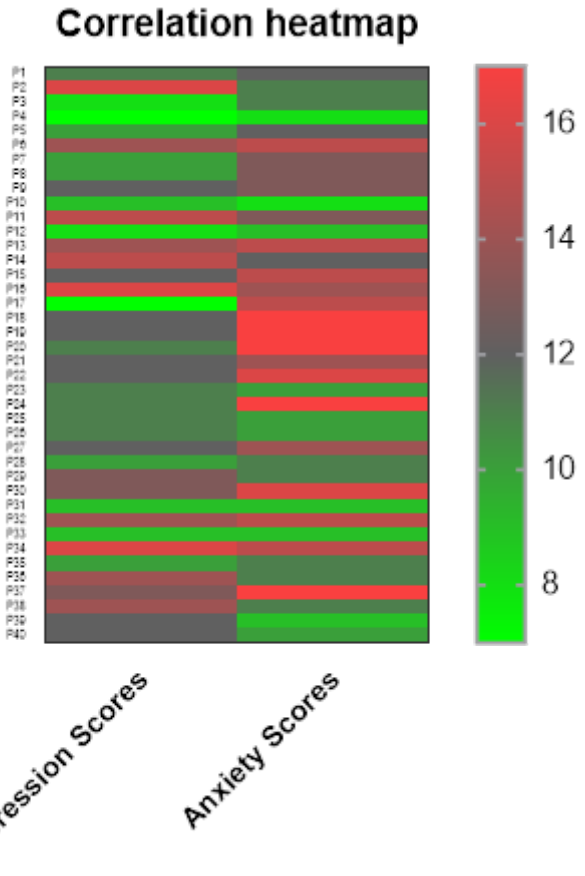


Figure 3. Corelation heat map of depression and Anxiety scores



DISCUSSION

There is a moderate positive correlation between anxiety and depression scores, as the mean± SD scores of depression were 11.70±2.377 and 12.65±2.779 with the P-value of 0.02 ≤0.05 and this relationship is statistically significant. PSA is less related to stroke than PSD since its primary predictors comprise lifetime anxiety disorders, including obsessive-compulsive disorder (OCD) and agoraphobia without panic disorder. In contrast, PSD cannot be predicted by lifetime depression. Therefore, PSD is less predictable than PSA as a post-stroke affective disorder because it is stroke-related [29] However, both PSD and PSA may result in adverse neurological and functional deficits if psychological interventions are not implemented in time.

Cramer argues that motor disorders are associated with reduced quality of life in eighty-two percent of patients. In his study he found that about sixty-five percent of affected individuals face difficulties in effectively incorporating paretic hands into daily activities. Their subjective well-being is also significantly affected because lower extremity motor status increases their disability level. In most cases, people who have experienced stroke find it difficult to perform regular and routine day-to-day activities such as walking, bathing,

sitting, or turning in bed without aid. The association of the challenges faced with PSD and motor activity is not surprising since the disorder causes physical stress that leads to motor impairments Severe hyper-locomotor deficit, which is among the risk factors of PSD, may lead to a high degree of disability for a patient if prevention and treatment strategies are not applied early enough. All these mentioned impairments can increase the anxiety levels in a patient which in turn can increase his levels of depression.

Post-stroke rehabilitation is usually conducted to help stroke survivors to achieve the best overall outcome for their conditions. Some skills impaired by a stroke that people can regain by adhering to the regimen proposed in their programs include an increase in sensation, ability to walk, improved coordination, and improved visual acuity ,Thus a proper tailored rehabilitation program may significantly improve the anxiety levels in a post stroke patient by mechanism of neural plasticity which may alter his/her depression levels and thereby limiting the dose of antidepressants but it cannot offer the support needed to reverse any brain damage caused thus necessitating the use of neuroprotectors and antidepressants for managing depression in a patient.

Based [29] on the monoamine theory, PSD's pathophysiology causes significant depletion of serotonin, norepinephrine, and dopamine neurotransmitter levels in the CNS. Gu et al.[30,31] Disruption of neurotransmission induces the release of proinflammatory cytokines, which can cause abrupt dysfunction of cortical circuits responsible for mood regulation and monoamine production. Thus according to monoamine theory tricyclic antidepressants and selective serotonin reuptake inhibitors (SSRIs), and serotonin-norepinephrine reuptake inhibitors (SNRIs) [32,33] are used to manage the PSD.

This study concludes that depression and anxiety are coexistent in a post stoke patient and when assessing and managing depression and anxiety among stroke patients the value-added treatment should address both the conditions in patients to enhance functional recovery and ease the burden of patient care.

Further studies should be conducted for corelation between anxiety and depression based on the area of the brain affected. In addition, lesion-location analysis conducted by Li et al. [34] showed that patients with acute frontal lobe infarction had increased chances of having PSA. However, unlike PSD, it is impossible to locate a single location in the brain that is directly related to PSA .

Abbreviations

PSD: Post stroke Depression.
PSA: Post stroke Anxiety.
OCD: Obsessive Compulsive Disorder.
SPSS: Statistical Package for Social Sciences.

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