The frequency of post-stroke depression in an outpatient elderly population*

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Abstract

Background: The purpose of the present study is to determine the frequency and severity of depression in post-stroke patients.

Methods: Based on a cross-sectional research design, 30 recent stroke outpatients were assessed with DSM-IV interview for depression and two self-rating depression scales, CES-D and BDI. Sex differences in depression, the relationship between depression and the location of the brain lesion and other clinical manifestations (sensory, motor and verbal) were also determined.

Results: 68% of the patients reported depression on CES-D, among them 64% suffered from moderate to severe depression according to BDI and interview-based information. Women were more depressed than men (63% and 37% respectively). Differences were found in lesion side, with patients with lesions in right side reporting higher levels of depression than patients with left side lesions. Motor disturbances were the most prevalent clinical features.

Conclusion: There seems to be an increased incidence of depression in stroke patients. The mechanism of pathophysiological processes underlying this association is poorly understood and is an important area for full investigation. Since depression is a significant risk factor for stroke it is important that psychiatric examination of post-stroke patients be conducted.

Keywords: stroke, depression, CES-D, BDI, location of the lesion

Introduction

Stroke is a sudden nonconvulsive focal neurological deficit produced by insufficiency of blood circulation to the brain [1]. In most western countries, 0.2% of the population suffer a stroke each year [2]. In addition to major impact of stroke on patients' physical health, many patients experience emotional disorders following stroke. The frequent appearance of emotional disorders following stroke has been recognized for many years. Kraeplein (1921) recognized

that cerebrovascular infraction may "engender states of depression". Goldstein (1948) found that patients with left hemisphere injury developed depression, irritability and explosive outbursts which he termed the "catastrophic reaction"[3]. Depression is a common neuropsychiatric consequence of stroke affecting approximately 40% of the patients. In addition to the psychosocial stress due to disability, loss of independence, and worsening of quality of life, neurobiological factors such as site of infarcts and brain atrophy have also been proposed to be re-

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lated to depression after stroke [4]. In recent years, post-stroke depression (PSD) has attracted worldwide interests. Studies conducted throughout the world have found a prevalence rate of major depression of 19.3% among hospitalized patients and 23.3% among outpatient samples [5].

Longitudinal studies of stroke patients have shown that about 20% of these patients will develop Major and another 20% will develop Minor depression during the first year after stroke [2]. Because PSD is not yet well recognized and has not been extensively studied in Iran and the lack of published articles on the association of these two clinical entities and also because depression is a treatable condition that may have implications for poorer outcome in relation to rehabilitation and mortality, it is therefore important to identify depression in stroke patients. In an effort to determine which factors influence the expression of post-stroke depression, we also discussed findings of PSD with particular emphasis on its prevalence, gender, distribution, location of the brain lesion and associated clinical features in this article.

Methods

A sample of 30 recent stroke outpatients (maximum three months post-stroke) who were referred to the neurology clinics of Rasoul-e-Akram and Mostafa Khomeini hospitals in Tehran for follow up from 2003 to 2004 and had agreed to participate in the investigation, were selected. 12 patients (40%) were male and 18 (60%) were female. The age range of the patients was between 30 to 79 years (mean age of 62 years). Patients with severe functional disabilities and history of clinical depression were not included.

The measures for assessing clinical depression included: the Center for Epidemiologic Studies Depression Scale (CES-D) which developed in to a large community study reporting depression symptoms. Although designed primarily for epidemiological studies, research

has indicated, however, that it is a psychometrically sound screening instrument that may be particularly useful in older adults. It consists of 20 items and scores range from 0 to 60. The sensitivity and specificity of the scale compares favorably with Beck Depression Inventory and Zung self-rating depression scale. Test-retest reliability over weeks averaged 57 and internal consistency was high (0.92). Validity was satisfactory assessed by correlation with other depression rating scales. A cut off of 16 and higher has been suggested to differentiate patients with clinical depression from normal controls [6].

Beck Depression Inventory (BDI) is one of the most widely used instruments in assessing the intensity of depression in psychiatrically diagnosed patients. It consists of 21 symptoms and attitudes that could be rated from 0 to 3 in terms of intensity. A cut off of 12/13 is taken to indicate the presence of depression. The scale had excellent external consistency and validity as scored by independent ratings (r = 0.93), it also has a high coefficient alpha (0.80) [7].

Information regarding independent variables of age, time elapsed since stroke, location of the lesion (left or right hemisphere) and the associated clinical features (sensory, motor, verbal, etc) were obtained from patients' charts.

The patients who agreed to participate in the study first screened for depression on CES-D and for those who obtained a score of 16 and higher, BDI was administered. The descriptive statistics and Pearson correlation coefficient was used for analysis of data.

Results

Table 1 illustrates the frequency of clinical depression in the sample. From a total of 30 stroke patients 22 (73.3%) met the criteria for clinical depression on CES-D and BDI with a mean intensity score of 25 on BDI. The association of two scales was high, with a correlation coefficient of 0.79.

Table 2 presents the frequency of depression

Diagnosis	Frequency	Percent
Clinical Depression	22	73.3
No Depression	8	26.7
Total	30	100

Table 1. Frequency distribution of clinical depression In stroke patients.

in terms of selected variables of gender, location of the lesion and the associated clinical features. As Table 2 shows, women were more depressed than men (68% and 32% respectively), patients with right hemisphere lesions were more depressed than left hemisphere lesions (54% compared to 46%) and motor disturbances were the most prevalent clinical features.

Conclusion

We found a high prevalence rate of depression in our post stroke patients (73%). This rate is higher than those reported for outpatient samples (23.3%)[5] and (33%) [8] and reflects the severity of depression in our sample.

Just as functional depression may arise from several mechanisms, the cause of PSD is likely to represent a mixture of etiologic factors. Socio-demographic, psychological and biological risk factors mediate the relationship between stroke and depression [3]. The association of depression with worse prognosis in stroke patients lends more support to the hypothesis that psychological rather than neurological factors

Variables	N	Percent
Male	7	32
Female	15	68
Location of the lesion		
Right temporo-parietal	6	27
Left temporo-parietal	6	27
Right basal ganglia	6	27
Left basal ganglia	3	14
Right parieto-occipital	1	5
Clinical features		
Motor disturbances	14	64
Verbal-motor disturbances	5	22
Sensory-motor disorder	2	9
Others	1	5

Table 2. Frequency of depression in selected variables.

are the main determinants of poststroke depression [9]. Along this line it seems that insufficient social support systems in Iran may have contributed in the overrepresentation of PSD in our study.

Our findings demonstrated that women suffer from depression more than men by a ratio of 2:1. This finding is consistent with a number of comprehensive epidemiological studies on stroke which have found a higher prevalence rate of depression among females [10]. Using a Multiple Logistic Regression model, Sharpe et al [11] found female sex and recurrent stroke as independent predictors of self-report depression. In Anderson et al's study [12] female gender correlated significantly with PSD. Eriksson et al [13] reported PSD in 12.4% of their male and 16.4% of their female stroke survivors.

Lavretsky et al [14] reported sex differences in brain anatomy as an important factor in the pathophysiology of geriatric depression. Female sex might be a risk factor in PSD; a point which is consistent with the results of our study. The higher percent of depression in patients with right hemisphere lesions (54%) compared to patients with left hemisphere lesions (46%) in our study was a conflicting finding.

It has been hypothesized that PSD results from left hemisphere lesions and the importance of lesion location in relation to depression has been supported in some, though not all subsequent investigations. In a recent publication based on a meta-analysis, Narushima et al [15] reported an association between poststroke depression and lesion location in patients with left hemisphere stroke but not among patients with right hemisphere stroke. According to Astrom [16] and Shimoda [17] left hemisphere lesion prominence in poststroke depression has mostly been shown in studies 3 or more months after stroke. The anatomic correlates of depression after stroke might change over time and this association might be weaker or nonexistent in longer follow-up.

Bhogal Sanjit [18] showed that much of the

heterogeneity across studies reflected differences in methodology. The direction of association between left hemisphere lesion location and PSD varied depending on whether patients were sampled as inpatients or from the community. Change in the direction of association was also observed across assessment intervals from the acute stroke to the chronic stroke phase. Differences in the measurement of depression and presentations of results also may have contributed to the heterogeneity of the findings.

Lesion location plays some part among a complex set of other determinants in contributing to depression after stroke but several key factors should be addressed before future research is undertaken. Recent systematic reviews of the many studies in this field have not supported the hypothesis of the significance of stroke lesion location and subsequent depression [19]. Motor disturbances were found to be the most prominent clinical features in PSD. While the association of depression with some movement disorders such as Parkinson disease is well established [20], little is known about causal relationship.

Stroke survivors have a greatly elevated risk for clinically significant depressive symptoms which is often undiagnosed and untreated. There is pressing need for further research to improve clinical practice in this area of stroke care and it is important that psychiatric examination of post-stroke patients is conducted.

References

- 1. Adams RA, Victor M. Principles of neurology. 3rd ed. New York: McGraw Hill; 1985. pp. 569-640.
- 2. Jorge RE, Robinson RG. Post-Stroke depression. Geriatrics & Aging 2004; 7:26-32.
- 3. Starkstein SE, Robinson RG. Post-stroke Mood Disorders. Neurology and Psychiatry: Meeting of Minds. Karger Basel 1989: 192-209.
- 4. Vataja R, Pohjasvaara T, Leppavuori A, Mantyla R, et al. Magnetic Resonance Imaging Correlates of Depression after Ischemic Stroke. Arch Gen Psychiatry 2001; 58:925-931.
 - 5. Robinson RG. Post Stroke Depression: prevalence,

- diagnosis, treatment and disease progression. Biol Psychiatry 2003; 54: 376-387.
- 6. Burns A, lawlor B, Craig S. Assessment Scales in Old Age Psychiatry. 2nd ed. London: Martin Dunitz; 2004.pp.14.
- 7. Beck AT, Steer RA, Garbing MG. Psychometric Properties of the Beck Depression Inventory: 25 Years of Evaluation. Clin Psychol Rev 1988; 8: 77-100.
- 8- Hackett Maree L, Yapa C, Parag V, Anderson Craig S. Frequency of depression after stroke. Stroke 2005; 36:1300-1305.
- 9. Bozikas Vasilis P, Gold G, Kovari E, Hermann F, Karavatos A, Giannakopoulos P, et al. Pathological correlates of poststroke depression in elderly patients. Am J Geriatr Psychiatry 2005; 13: 166-169.
- 10. Whyte Ellen M, Mulsant Benoit H, Vanderbilt J, Dodge Hiroko H, Ganguli M. Depression after stroke: A prospective epidemiological study. Journal of the American Geriatrics Society 2002; 52: 774-778.
- 11. Sharpe M, Hawton K, Seagroatt V, Bamfford J, House A, Molyneux A, et al. Depressive disorders in long-term survivors of stroke. British Journal of Psychiatry 1994; 164: 380-386.
- 12. Andersen G, Vestergaard K, Ingemannn-Nielson M, Lauritzenn L. Risk factors in post-stroke depression. Acta Psychiatr Scand 1995; 92: 193-19.
- 13. Eriksson M, Asplund K, Glader Eva-Lotta, Norving BO, Stegmayr B, Terent A, et al. Self-reported depression and use of antidepressants after stroke: a national survey. Stroke 2004; 35: 936-942.
- 14. Lavretsky H, Kurbanyan K, Ballmaier M, Munitz J, Toga A, Kumar A. Sex differences in brain structure in geriatric depression. American Journal of Geriatric Psychiatry 2004; 12: 653-657.
- 15. Narushima K, Chan KL, Kosier JT, Robinson RG. Does cognitive recovery after treatment of poststroke depression last? A two years follow-up of cognitive function associated with poststroke depression. Am J Psychiatry 2003;160:1152-1162.
- 16. Astrom M, Adolffson R, Asplund K. Major depression in stroke patients: a 3 year longitudinal study. Stroke 1993; 24: 976-982.
- 17. Shimoda K, Robinson RG. The relationship between post-stroke depression and lesion location in long-term follow-up. Biol Psychiatry 1999; 45: 287-292.
- 18. Bhogal Sanjit K, Teasell R, Speechly M. Lesion location and poststroke depression. Stroke 2004; 35: 794-802.
- 19. Carson JA, Machale S, Allen K, Lawrie SM, Dennis, House M, et al. Depression after stroke and lesion location: a systematic review. Lancet 2000; 359: 122-127.
- 20. Nilsson FM, Kissing LV, Sorensen TM, Andersen PK, Bolwig TG. Affective disorders in neurological diseases: a case register-based study. Acta Psychiatr Scand 2003; 108: 41-50.