ORIGINAL ARTICLE

Site of Stroke : Correlation with Cognitive Deficits, Symptoms of Anxiety and Depression, and Quality of Life

NILAMADHAB KAR, S. P JOHN

ABSTRACT

The relation between site of stroke and cognitive deficits, anxiety, depression, and quality of life was done in 40 stroke patients using Blessed Mental Status Test, Hospital Anxiety and Depression Scale, Manchester short assessment of quality of life scale. Lesion localization was done by CT scan. In a relatively short period after stroke, specificity for any hemisphere or arterial territory of any side (left or right) was not evident for anxiety, depression, cognitive deficits or level of QOL. Considering the arterial territories involved, MCA infarcts were associated with greater cognitive deficits, anxiety and poor QOL. ACA infarcts had least anxiety. PCA infarcts were associated with better QOL and least cognitive deficits.

Key words: Stroke, cognitive deficits, anxiety, depression, quality of life

INTRODUCTION

It is well known that stroke frequently leads to cognitive deficits (Desmond et al, 1996) and emotional problems (Johnson et al, 1995) of variable severity. The factors associated with the variability are site of stroke and associated other physical conditions. Various cognitive deficits including dementia occur depending upon the site and the extent of damage due to stroke (Leishman, 1987).

Depression and anxiety disorders are more common after stroke than is generally recognized (House et al 1991; Statkstein et al, 1989). Post stroke depression is correlated with cognitive deficits (Kauhanen et al, 1999). Compared to many studies on depression after stroke very little attention has been given to post stroke anxiety (Johnson et al, 1995).

Dimensions of quality of life include physical, functional, psychological and social health. Stroke along with its impact on physical and mental health can affect all these domains. Depression secondary to stroke can influence quality of life (King 1996). The objective of the present study was to find our relationship of cognitive deficits, anxiety and depression and QOL with site of stroke and the arterial territory involved.

MATERIALS AND METHODS

All consecutive patients with stroke who came for consultation or admission to a medical unit of Kasturba Medical College, Manipal during September 1999 were studied. The patients who had sub-arachnoid haemorrhage, extradural haemorrhage, transient ischaemic attacks, and mass lesions were excluded. Uncooperative patients and patients with speech arrest were excluded. Detailed clinical history and demographic variables were collected. The site of the lesion and the arterial territory involved were determined from clinical findings and CT Scan. The cognitive deficits were assessed by Blessed Mental Status Test Score (Kaufman, 2001). Three items of the questionnaire were modified to make it more culture relevant. Hospital Anxiety and Depression Scale (HADS) (Zigmond and Snaith, 1983) was used to quantify anxiety and depressive symptoms. Assessment of QOL was done using Manchester short assessment of quality of life scale (MANSA), which focuses on satisfaction with life as a whole (Priebe et al, 1999). Greater score of MANSA indicates better QOL. Comparison of means of various parameters was done by using ANOVA and t test. The relation between various continuous variables was studied by correlations. Comparison of groups was done by chi-square tests.

RESULTS

The sample consisted of 40 patients (31 males and 9 females) with a mean age of 53.3 (SD 10.8). There were 20 patients each on the two sides of the hemispheres. Five (12.5%) (2L, 3R) patients had anterior cerebral artery infarct (ACA), 19 (47.59%) (8L, 11R) had middle cerebral artery infarct (MCA) and 16 (40.0%) (10 L, 6 R) posterior cerebral artery infarct (PCA). Mean duration since infarct was 18.5 weeks. All the patients were educated (median: 8 years of education).

There were no differences in the anxiety, depression (HADS). cognitive (BMS) or QOL (MANSA) scores of patients with left or right hemisphere infarct. Comparison of these variables associated with territories of individual cerebral arteries of left and rights sides we did not find any significant difference. One-way ANOVA comparing these variables suggested significant difference for anxiety (F 4.83, p 0.014), QOL (F22.3, $p<0.01$) and BMS (F 8.5, p 0.001) amongst territories of cerebral arteries. Specific findings for each arterial territorial infarct are given Table 1. Significant impairment in QOL. and cognitive functions were noticed in MCA and ACA infarct in comparison to PCA infarets. Anxiety symptoms were significantly less in ACA infarcts. There was no difference in degree of depressive symptoms associated with infarcts of different arteries. Cognitive deficits (BMS score) and QOL were negatively correlated (Pearsons correlation —0.326, $p<0.05$).

Degree of anxiety was also negatively correlated with cognitive deficits (Pearsons correlation —0.445, p0.01). The anxiety and depressive symptoms were positively

TABLE I. Arterial territory involved in the infarct and the associations

Variables	ACA	MCA	PCA
Mean Age + SD*	51.6±9.8	50.0±12.4	57.8±7.5
QOL (MANSA)#	57.2±2.49	53214.4	61.6±3.18
BMS rest score @	11.6±1.5	10.4±2.4	7.93±1.9
Anxiety (HADS) $	2.2±0.45	6.8±2.69	6.68±3.8
Depression (HADS) Ä	8.6±0.8	8.52±2.2	8.31±1.85

*MCA vs PCA : t=2.19, df=33, p<0.05;
ACA vs MCA : t=1.87, df=22, p=0.075, ACA vs PCA : t=3.18, df=19, p<0.005, MCA vs PCA : t=6.48, df=33, p<.000
@ ACA vs MCA : t=3.38, df=33, p=0.002
$ ACA vs MCA : t=3.78, df=22, p<0.001; ACA vs PCA : t=2.57, df=19, p<0.05, MCA vs PCA : t-0.14, df=33, p=0.89
Ä ACA vs MCA : t=0.07, df=22, p=0.95; ACA vs PCA : t=0.35, df=19, p=0.74 MCA vs PCA : t=0.3, df=33, p=0.76

correlated (Pearson's correlation —0.474, p<0.05. Considering the caseness based on HDRS score of 11 or more there were no differences between the sides or arterial territories.

DISCUSSION

The study compared anxiety depression, cognitive deficits and QOL following strokes of different sides and arterial territories. Before discussing the results several methodological limitations should be acknowledged. The sample size was inadequate, as there were only a few considering the individual arteries of any side. Volumetric assessment of the infarers was not done, which could have given the severity of the infarct. Similarly we have not data on the disability which may influence the QOL. Effects of concurrent physical conditions and medications were not assessed. However the sites of the infarcts were specific to the arterial level and are clinically relevant. All the lesions were specified by CT findings. The study suggested few interesting findings.

There was no difference in the degree of depression among the sites studied. There are divergent findings in the literature on post stroke depression. Anatomical correlates of post stroke depression change over time and may explain inter-study differences in the association of lesion location with post stroke depression (Shimoda and Robinson, 1999). Compared to the short-term follow up findings of Shimoda and Robinson (1999) we also found no hemisphere-related differences. It has been reported that depression was found to be significantly more common among patients with frontal infarction (Cummings 1985). However considering the arterial territories we did not find any relation between lesion location and depression.

Anxiety symptoms were comparatively less in ACA infarers. Lesions in the medial frontal and orbitofrontal areas are associated with euphoria, inappropriate jocularity, mania or hypomania (Cummings, 1985) and specific orbitomedial and cingulate lesions in psychosurgery bring out change in anxiety levels (Hay et al, 1993). These reports suggest that anxiety may be an unlikely out come of the infarct in this area.

Interpretation of QOL findings is complicated by the lack of pre-stroke control. However in a comparative post stroke study QOL levels of different sides and marterial territory involved can still suggest the role played by them. It has been reported that QOL profile of patients with lesions in the left hemisphere is slightly better compared with those of patients with lesions in the right hemisphere, with the exception of the ability to communicate (de Haan et al 1995). However in this study the cognitive deficits and the QOL of both sides were comparable. Moreover cognitive deficits and QOL were found to have negative correlation. It is known that two hemispheres differ in their recovery from the cognitive impacts of infacts (Desmond et al 1996). It may be interesting to study the process of cognitive recovery and change of QOL.

Considering individual arterial territory, lowest QOL was observed following MCA infact, which was associated with higher cognitive deficits and anxiety. Depression, which predicts post stroke depression (King et al 1996), however was comparable in all sites. This suggests that other factors may be responsible, and cognitive deficit, which correlated significantly with QOL, could be one.

In condusion, in a relatively short period after stroke, specificity for any hemisphere or arterial territory of any side (left or right) were nor evident for anxiety, depression, cognitive deficits or level of QOL. Considering the arterial territories involved MCA infacts were associated with greater cognitive deficits, anxiety and poor QOL. ACA infacts had least anxiety PCA infarcts were associated with better QOL. and least cognitive deficits. Future studies should look into effects of the period since stroke, neuro-defecits and disabilities, functional status and activities of daily living with the psychological and cognitive symptoms and QOL.

ACKNOWLEDGEMENT

This study was supported in part by Quality of Life Research and Development Foundation.

REFERENCES

Leishman,W.A. (1987) Organic Psychiatry. the Psychological Consequences of Cerebral Disorder. Oxford: Blackwell Scientific publishers

Starkstein, et al (1989) Mood changes after right-hemisphere lesions. British Journal of Psychiatry; 155. 79-85

Cummings, J.L. (1985) Behavioural Disorders associated with frontal lobe injury. In Clinical Neuropsychiatry, (Editor: Cummings] L) ppS7 67, Grune and Stratton: Orlando

de Haan, R.J. Limburg, M.Van der et al (1995) Quality of life after stroke. Impact of stroke type and lesion location. Stroke, 26, 402-408

Desmond, D.W Moroney,j.T. Sano, M. Stern, Y. (1996) Recovery of cognitive function after stroke. Stroke. 27. I 798-1803

Hay P. Sachdev, P. Cumming, S. et al (1993) Treatment of obsessive-compulsive disorder by psychosurgery. Acts Psychiatrica Scandinavica, 87(3). 197-207.

House, A. Dennis, M. Mogridge, L. et al (1991) Mood disorders in the year after first stroke. British Journal of Psychiatry, 158, 83-92 4

Johnson, G. Survill, RW Anderson et al (1995) Screening instruments for depression and anxiety following stroke: experience in the Perth community stroke study. Acts Psychiatrica Scandinavica, 91, 252-257

Kaufman, D.M. (2001) Dementia. In clinical neurology for psychiatrists. 5th edition, 123-174 Philadelphia: WB Saunders

Kauhanen, M. Korpelainen, J.T. Hiltunen, et al (1999) Post stroke depression correlates with cognitive impairment and neurological deficits. Stroke 30(9), Stroke 27, 1467-1472

King, R.B. 1996. Quality of Life after Stroke. Stroke 27, 1467-1472

Priebe, S.Huxley, ft Knight, S. Evans, S. (1999) Application and results of the Manchester short assessment of quality of life (MANSA). International Journal of Social Psychiatry, 45 (I), 7-12.

Shimoda, K. and Robinson, R. G. (1999) The relationship between post stroke depression and location in long term follow up. Biological Psychiatry, 45. 187-192

Zigmond, A.S. Snaith, R.P. (1983) The Hospital Anxiety and Depression Scale. Acta Psychiarrica Scandinavica. 67, 361-370.

*NILAMADHAB KAR, MD. DPM, DNB, Consultant Psychiatrist, Wolverhampton Primary Care NHS Trust, Corner House Resource Centre, 300, Dunstall Road, Wolverhampton, VsV6 ONZ, United Kingdom, Email: nmadhab@yahoo.com,

S. P JOHN. MD student in Medicine, Kasturba Medical College, Manipal

***Correspondence**