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ORIGINAL ARTICLES

Depression Following Cerebrovascular Disease

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Summary :

A total of seventy cases of cerebrovascular disease were studied during a period of one and half month. After clinical assessment and using Hamilton rating scale for depression, 40% patients were found to have depression after stroke. Most of the patients of stroke and those of depression lies between 41 to 60 years of age. Incidence of depression after stroke were found equal in both the sexes. Most of the patients were literate representing rural and Urban Population equally. Eighty six percent of depressives had non-haemorrhagic type of stroke. Depression was found to be independent of site of lesion. Depression was commonly found in acute and after first attack of stroke. Majority of the patients had associated hypertension. Only 15% of patients having post-stroke depression were getting antidepressant therapy.

Introduction :

Depression is common in patients with cerebrovascular disease and is a well recognised barrier to recovery after cerebrovascular disease[1].

From a number of studies it is revealed that the prevalence of depression in cerebrovascular disease is more in older patients. Most of the studies have recently demonstrated that major and minor depressive disorders occur in 30-50% of stroke patients and lasts more than one year if not treated, although they do respond to tricyclic antidepressants[2].

The aetiology of post-stroke depression remains unknown. During 1940s there was a general acceptance that depression may be a psychological response of the organism to severe strees[3]. The prevalence of depression after cerebrovascular disease is not exactly known in our country as no study of depression following cerebrovascular disease has yet been carried out. The present study may give some idea about the relationship between depression and cerebrovascular disease, which would in turn increase the aware-

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ness about possible presence of depression after cerebrovascular disease. The aim of this study was to measure the relationship of depression with cerebrovascular disease considering the parameter like onset, type and site of stroke, length of illness, previous history of stroke and any other associated diseases. The patients were assessed clinically to identify the features in favour of a diagnosis of depression and later Hamilton Rating Scale for depression was applied to quantify it.

Persistent and refractory depression of mood has often been reported to follow stroke. This symptom is commonly explained as an expected psychological reaction of the patient to his disability or restrictions in activity. Goldberg and Hiller (1979) and Robinson aud Price (1982) reported that about 30% consecutive patients examined were depressed as measured by the General Health Questionnaire[1],[4]. Key (1962) reported a higher incidence of cerebrovascular disease in elderly patients with late onset depression compared with elderly patients whose first depression had occurred in early life[3]. Several studies have demonstrated the importance of lesion location in the pathogenesis of depression following cerebrovascular disease. Left hemisphere lesions, mainly in the frontal cortex or basal ganglia, have a strong association with post-stroke depression, and the closer the lesion to the frontal pole more is the severity of depression. Subcortical strophy, probably before the actual stroke may constitute an important risk factor for the development of post-stroke depression[2],[5],[6]. It was also found that immediately after stroke, depressed patients had a significantly lower functional status than non-depressed patients[2].[7]. So, it is seen that post-stroke impairements do not seem to cause depression, but once depression occurs, physical impairements may perpetuate depression and/or depression may inhibit functional recovery from stroke. Language problems may lead to a secondary psychological reaction because of the understandable importance of language function to man (Benson, 1973)[8]. It was reported that depression is more common in patients with non-fluent aphasias than in patients having fluent or global aphasias[2]. The aetiology of depression following cerebrovascular disease remains uncertain, but the inability to find out a definite 'reactive' cause for these depression and controversies related to it suggests that neurophysiological processes may play an important role.

Materials and Methods :

Seventy consecutive patients with stroke admitted in the hospital from 1st December, 1989 to 15th February, 1990 were included in the study. Of these, 53 cases were taken from Neurology units of IPGMR, Dhaka, eight cases from Cardiovascular unit of Dhaka

Medical College Hospital and nine from Sir Salimullah Medical College and Mitford Hospital, Dhaka.

Cerebrovascular disease refers to all forms of vascular disease which affect the brain: any combination of stroke, transient ischemic attack (TIA) or multi-infarct dementia (MID) may result[9,10]. Patients having depression and other psychiatric illness in the past or previous history of psychiatric treatment before cerebrovascular disease or family history of psychiatric disorders were excluded. History of the patients were taken after admission and thorough clinical and neurological examination were done. Most of the cases, diagnosis were confirmed by CT scans. Relevant sociodemographic information and information related to cerebrovascular disease like duration, previous history of cerebrovascular disease and associated diseases were noted. Types of stroke after relevant investigations were also recorded.

Depression was first diagnosed clinically, then Hamilton Rating Scale for depression has 17 items with a five point score[11]. A score 15 or more was considered to identify depression. The relevant data was represented in tabulation form and statistical analysis was done where needed.

Results :

In total seventy cases of stroke were collected in the course of one and half month. Forty eight were males and 22 were females. Their age ranged between 21 and 100 years with a mean of 56.43 years. Among the patients of stroke 28 (40%) were fonnd to be suffering from depression (20 males and 8 females) with a mean age of 56.87 years. The male female ratio here was 2.5 : 1

Both stroke and post-stroke depression was found mostly in male retired from different services and in female housewives. Though urban and rural people presented in stroke population but depression was found slightly higher in the subjects who came from rural areas. Maximum cases of both in stroke and post-stroke depression were middle income group.

Most of the patients of post-stroke depression (88.57%) were found to have non-haemorrhagic stroke. (Table-III)

In 46.43% of patients having depression, the duration of stroke varied between 16 and 30 days.

Among the depressed patients 24 were having their stroke for the first time in their life and 20 patients of depression had associated hypertension, diabetes, ischemic heart disease, epilepsy or their combinations.

Among the post-stroke depression, patients only six were receiving antidepressants (TCA). They are those who are having score in Hamilton Rating Scale for depression (two between 30 to 34, three between 25 to 29 and one between 20 to 24 score group. (Table-I,II)

Table—I

Distribution of the patients of post-stroke depression according to their score on Hamilton Rating Scale for depression.

Score	Total patients with PSD (n=28)	Left (n=16)	Right (n=11)	Both (n=1)
15-19	5	3	1	1
20-24	11	7	4	0
25-29	8	6	3	0
30-34	4	1	3	0
Mean Score	26.32	23.25	25.64	17.00

$X^2=6.315$, df=6, P<5.00

Table—II

Distribution of patients of post-stroke depression according to antidepressants they were taking.

Post-stroke depressives	Hamilton Rating Scale Score 15-19	20-24	25-29	Group 30-34	(n)	Total %
Patients with no antidepressants	6	9	4	3	22	78.57
Patients with antidepressants (TCA)	0	1	3	2	6	21.43

$x^2=30.14$, df=3, P <0.001

Table—III

Distribution of the patients of stroke and post-stroke depression according to the type of stroke

Type	Stroke	%	Post-stroke depression	%
Haemorrhagic	8	11.43	4	14.29
Nonhaemorrhagic	62	88.57	24	85.71
Total	70	100	28	100

$x^2 = 0.39$, df = 1, $P < 0.05$

Discussion :

The present study included only hospitalised stroke patients and depression was found to be 40% of these stroke patients. Those patients who scored 15 or more in Hamilton Rating Scale for depression were diagnosed to be having depressive illness. This measure reduce the tendency of overestimation of the prevalence of post-stroke depression in elderly people. Depression might have been present in many of the patients before their stroke. But in any way the figure should not go above what is found in general population. It was found that depressive neurosis' 3-8% of the 360 of his elderly patients and only 0.6% were found to suffer from depressive illness (all women)[12]. These figures are much lower than seen in patients with stroke. Moreover the possibilities of having depression before stroke was excluded by taking elaborate history.

About the presence of depression in stroke patients were consistent with those of other studies where it varies between 30-50%[2,12].

Most of the post-stroke depression patients were found to be above 40 years of age (24 out of 28) which simulates the age incidence of depression in general population. No difference was found in the mean age between patients of depression and that of stroke as a whole. This may be due to the fact that both depression and stroke are more common after 40s. Although both in stroke population and in patients with post-stroke depression there were more males than females but no significant difference ($p < 0.005$) was found between stroke and that of occurence of post-stroke depression when sex incidence was considered.

Most of the patients in both patients with stroke and post-stroke depression

were literate. This was because patients were collected from city hospitals and people from in around the city come for treatment here.

Although rural and urban population were found to represent almost equally in both the group, it is assumed that rural people who are financially better off can avail education and also avail treatment in bigger city hospitals. It should also be added that though the studied population did not represent the country population as a whole it would at least give some definite indications which might help to establish an aetiological relationship between depression with stroke. In present study relatively more patients of both stroke patients (60%) and those of post-stroke depression (67.86%) were found to come from higher middle income group. This is again the representation of the population who can avail the treatment in city hospitals.

In present study most of the patients of stroke (88.57%) and those of post-stroke depression (85.72%) had their stroke within previous two months. Depression was found in 18 of the 28 patients who had their stroke only during previous month.

In this study, 88.57% of patients of stroke and 85.71% of patients of post-stroke depression were having non haemorrhagic type of stroke which is highly significant ($p < 0.05$). These findings are consistent with approximately 85% incidence of nonhaemorrhagic stroke[9] and 82% of patients of depression following nonhaemorrhage type of stroke[11,12] Moreover, haemorrhagic strokes in most of the cases leads to fatality if proper and adequate medical assistance is not available in time.

In present study, 71.43% patients of post-stroke depression had associated with other diseases like Hypertension, diabetes IHD and Epilepsy or their combinations. Among them highest number (35.73%) were having depression Depression may be a reaction to loss of physical health and function[9].

Depression can not only be explained by a mere presence of physical disease but social disability it casues. Robinson et al (1982 and 1983) did not find any relationship between severity of the stroke and likelyhood of depression[4,5] but Ebrahim et al (1987) reported high degree of correlation between severity and development of depression[1].

Here, in this study only six out of 28 cases were getting anti-depressants. Six cases who were having very high score in Hamilton Rating Scale for depression, It has been observed that most of the patients of depression after stroke remain untreated[4]. It is also

Case-Report :

A 30 year, male, bussinesman was admitted to a private Hospital on 17-01-91 with history of sudden loss of consciousness, convulsions, with fall at early morning of same day while combing his hair. He passed stool, and had frothing from angle of the mouth, and tongue bite during few minutes of unconsciousness. He also vomited few times on gaining consciousness.

He had history of taking drugs like Canabis, Heroine, and occasionally Injectable Pethidine for the last six months or so.

Examination on admission, the patient was stuporous, irritable, and restless at times. Pulse rate was 52/mt., B. P-110/80 mm of Hg., with photophobia, neck stiffness, positive Kernig's and Babinski's sign bilaterally, optic disc was normal. He was given Intravenous Aminocaproic acid, Non steroid antiinflammatory drugs suppositories rectally twice daily and oral Diazepam-5 mg thrice daily.

Laboratory investigation showed Total white cell count-11000/cu mm. Neutrophil-78%, Lymphocyte-21%, Eosinophil-1%, sedimentation rate-35 mm in first hour. Blood urea-25 mg%, serum cholesterol-175 mg%, Blood sugar-110 mg%, Sodium-135 mg%, Potasium-6 mg%, Chloride-80 mg%. Electrocardiogram was within normal limit ; X-ray skull and chest was normal.

On 19-01-91 computerized tomography of head was done, non-contrast scan showed hyperdensity in the iv th ventricle, interhemispheric fissure and hemispheric subarachnoid spaces (Fig-1).

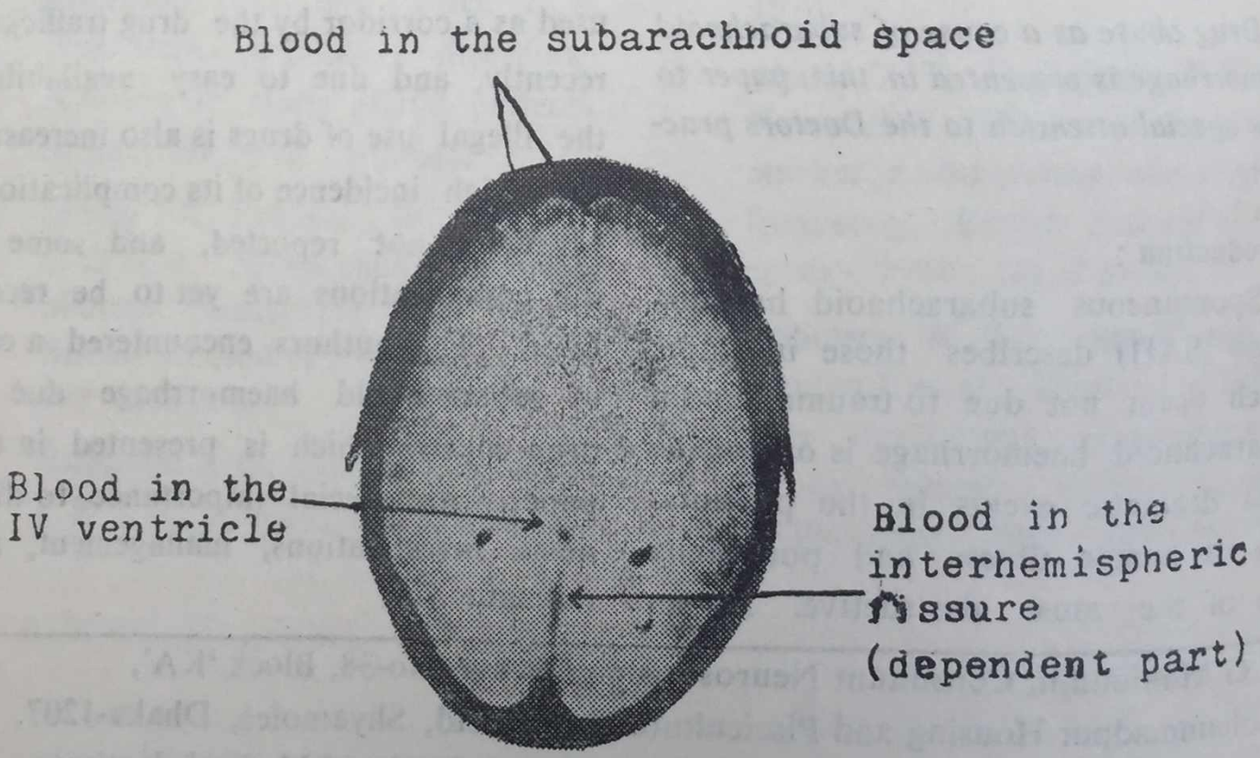

Fig 1 : *Print from positive film (Hyperdense area show Black, Isodense and Hypodense area show-White).*

Contrast CT visualized the major intracranial vasculatures (Fig-2) but no aneurysms or angiomas could be seen. His condition progressed on well and was started Injectible Dexamethasone on 20-01-91. His conscious level improved, neck stiffness disappeared, and was discharged home on 30-01-91, with an advise of follow-up in 15 day's time. He was followed up three times in four months time, and revealed no recurrence of bleeding or any other neurological deficit. He is leading perfectly a normal life.

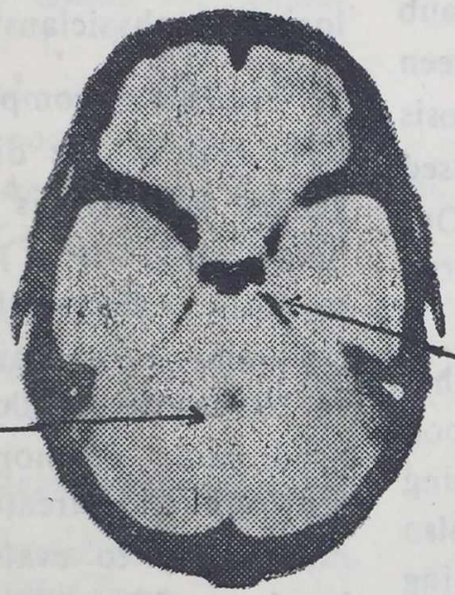

Fig 2 : *Print from positive film (Hyperdense area show-Black Isodense and Hypodense area show-White)*

Discussion :

Subarachnoid Haemorrhage (SHA) accounts for upto 14% of deaths from vascular diseases of the brain[3]. Among identifiable causes of SAH are hypertension, aneurysm, angioma, less commonly-intracranial tumour (usually malignant), and blood dyscrasias[4].

SAH can be grouped into four categories :

First there can be an identifiable underlying structural abnormality in the vasculature of the brain, i. e. aneurysma and angiomas. Secondly haemorrhage can be an expression of some other process primarily involving the brain ; such as trauma, tumour, and encephalitis. Thirdly, widespread systemic disease can secondarily cause SAH, i.e. hypertension, blood dyscrasias, and clotting defects.

Finally, in some cases no proximate cause is found[5]. Our case can be considered in the final group. Fatal SAH has been described following use of both intravenous and oral Amphetamines[6]. Other drugs like phenyl Propanolamine, Cocaine and its derivatives can also cause subarachnoid and intracerebral haemorrhage. Out of 150 patients with SAH admitted to Ben Taub Hospital from 1980 to 1987 ; seventeen patients (11% of total) had a diagnosis of SAH within 72 hours of witnessed intravenous injection of Cocaine[7]. Our patient was also known to be a drug addict.

Pathophysiologically these sympathomimetic drugs suddenly raise blood pressure and cerebral blood flow causing small arteries to rupture. It may also likely to cause rupture of preexisting aneurysms and arteriovenous malformations. Chronic use of these drugs probably cause antibody formation, leading to an immunologically mediated arteritis that can also potentiate haemorrhages and ischaemia[8]. Cerebrovascular effects of cocaine derivatives are thought to be possibly due to the inhibition of dopamine, norepinepinephrine, and serotonin reuptake at synaptic terminals[9]. Patients using cocaine may also be susceptible to SAH due to disturbances in cerebral autoregulation[10].

Classical presentation of SAH is sudden onset of severe headache, vomiting and neck stiffness; with or without transient loss of cosciousness the physicians task is to find out the likelihood of cause by proper history taking. In SAH the choice and extent of the intracranial investigations, and the consequent management of the patient are the Neurosurgeon's concern, while the later problem of prognosis is left for the Neurologists or physicians[4].

The role of computerised tomography (CT) scan in the diagnosis and management of SAH is well established[11]. Some variations in positive CT findings in SAH reflects the effects of three factors[12]. First timing-CT is more likely to be negative following a warning or prodromal haemorrhage. Second is source-refferal treatment centres are more likely to evaluate sicker patients in whom CT is more likely to be positive. Third is what we call lumbar puncture (LP) bias. Neurologists are more likely than Neurosurgeons to establish the diagnosis of SAH by L. P,[13].

We were confined only to clinical history, findings and CT scan finding in diagnosing this case of SAH due to drug abuse. It is a matter of urgument why cerebral angiography was not done? The explanation is we were able to demonstrate the cerebral vasculature with contrast infusion, and the patient followed up regularly after discharge and showed no recurrence of SAH.

We presume that our case is an unique example of SAH caused by drug abuse, and needs more cases to interprete the incidence, clinical presentation, pathophysiology, treatment, mortality, mobidity and outcome in Bangladesh.

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