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Comparative Study Between Piracetam and Vinpocetine on Recovery of Motor and Cognitive Functions after Ischemic Stroke

MT ISLAM[1], AKMH MOSHARROF[2], M RAHMAN[2], UK SHAHA

Abstract

Purpose of the study: *Piracetam and Vinpocetine, the nootropic agents with neuroprotective properties, have been reported in many studies to increase cerebral blood flow in patients with ischemic stroke and to improve clinical outcome. We performed prospective, randomized, double-blind study to test whether piracetam or vinpocetine improves motor and cognitive functions recovery after ischemic stroke and which one is better clinically.*

Methods: *Fifty three ischemic stroke patients were randomly allocated in two groups: 27 patients received 800mg piracetam thrice daily, 26 patients received 10mg vinpocetine thrice daily. After 4 weeks of treatment the patients were assessed by Modified Rankin Scale and Mini Mental Status Examination to see the motor and cognitive functions recovery respectively.*

Results: *In the studied population, outcome was similar in both of the treatment group. Piracetam improved both the motor and cognitive function recovery (P<0.05). Vinpocetine improve motor and cognitive functions (P<0.05) as well. However, piracetam and vinpocetine did not differ (P>0.05) in their functions after 4 weeks of treatment.*

Conclusion: *Both Piracetam and Vinpocetine appeared to improve cognitive and motor functions after ischemic stroke. They showed equally effective in their action.*

Key Words: Stroke, Cerebral ischemia, Piracetam, Vinpocetine, Motor function, Cognitive function.

Introduction

Ischemic stroke is one of the most common causes of death throughout the world and it is a major cause of disability[1]. Attempt has been made for an effective means of assisting and accelerating functional rehabilitation following stroke[2]. Although the pathogenesis of ischemic stroke is uncertain, it is well recognized that any approach which increases the availability of oxygen to the cerebral tissue, can help protect against ischemic-induced damage[3].

It has been suggested that piracetam and vinpocetine can increase oxygen availability and oxygenation in brain tissue[4]. The pharmacological effect of piracetam is an increase of cerebral microcirculation and the modulation of neurotransmission[5]. This provides more oxygen to cerebral tissues;

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improve cerebral protection in ischemia, cognition-enhancing and anticonvulsant activity[6]. Pilot studies in patients with acute ischemic stroke of moderate to severe degree have suggested that Piracetam exhibits a significant improvement of motor dysfunction, consciousness and aphasia[7]. Under hypoxic condition, piracetam increases glycolysis via the pentose phosphate cycle and thereby NADPH is increased in the brain[8]. Piracetam increases the release and turnover of dopamine[9, 10], enhance release of acetylcholine in the hippocampus and corpus striatum[11, 12], exert agonistic effect on serotonin (5-hydroxytriptamine; 5-HT) receptor and antagonize on glutamate receptor[13].

Vinpocetine exerts a brain neuroprotective effect by a combined action on cerebral circulation, and oxygen utilization without changes in systemic circulation. It exhibit cerebral protection in conditions of hypoxia/ ischaemia, cognition-enhancing and anticonvulsant activity, and improvement of rheological properties of the blood[14]. Vinpocetine appears to play a role of sodium channel inhibition in neuroprotection[15]. Positron emission tomography (PET) studies have proved that vinpocetine is able to redistribute regional cerebral blood flow and enhance glucose supply of brain tissue in ischemic post-stroke patients. Randomized placebo-control studies reported improvement of cognitive functions after vinpocetine administration in chronic cerebrovascular disorders .[16]

The rationale for the use of Piracetam and Vinpocetine in acute ischemic stroke is based on its combination of neuroprotective, haemorrhoelogical and anti-thrombotic properties.[2]

Neuroprotection in the ischemic stroke have tried in large numbers of patients to assess their efficacy and safety elaborately and we will probably have to wait until the next century before the treatments other than anticoagulant and anti-thrombolytic treatment.[5]

Therefore, a detailed study is still needed for the evaluation of suitable drugs which are given in the treatment of ischemic stroke to improve motor and cognitive function. In this study we aimed to conduct comparative study between Piracetam and Vinpocetine on motor and cognitive function recovery after Ischemic Stroke.

Materials and methods

Selection of patients

The study was conducted on 53 patients of age 30 to 80 years who had an ischemic cerebrovascular accident with in last 24 hours confirmed by CT scan of brain. Patients were required to give informed consent and to be available for a follow-up period of one month. Patients who sustained haemorrhagic stroke and history of previous stroke were excluded from the study. The study was conducted in the Department of Pharmacology and Therapeutics, Sylhet M. A. G. Osmani Medical College in collaboration with Neuromedicine Department of Sylhet M A G Osmani Medical College and Hospital between January 2005 and December 2005.

Study design

The study was prospective, randomized, double blind investigation. Prior evaluations of the patients were done by CT-scan, Modified Rankin Scale (MRS), Mini Mental

Status Examination (MMSE), heart rate and blood pressure recording. Patients were randomly assigned to receive either piracetam (800mg) or vinpocetine (10mg), 3 tablets daily (one in the morning, one in the mid-day and one at night) for four weeks. Both piracetam and vinpocetine tablets were provided by patient's attendant or nurse under supervision of the co-guide. Compliance was assessed by calculating the number of tablets which should have been taken and comparing with the number tablets actually taken by the patient over the period of the study.

The patients' motor function was assessed by Modified Rankin Scale, which ranged from 0 to 6 points, the higher the Rankin score sever motor deficit. Cognitive function was determined according to Mini Mental Status Examination, which ranged from 0 to 30 points, the higher the MMSE, the better the cognitive function. MRS and MMSE were assessed before treatment, with in 7 days of treatment and after 4 weeks of treatment. Two groups were compared according to differences noted from pre treatment until final visit at the end of 4 weeks. Patients were asked whether they had experienced any problem during intake of drug.

Data were analyzed and compiled in Microsoft excel by one- way ANOVA. Statistical tests of significance between different sets of data were done by Microcal Origin-7.0 (statistical software).

Results

A total number of 53 patients were included and 49 completed the study. Of the four patients who did not complete the study, two were from the group-I (piracetam group) and two from group-II (vinpocetine group). From group-I (piracetam group) two patients refused to continue with treatment and two patients from group-II (vinpocetine group) were lost to follow-up with out any reasons.

The age of the ischemic stroke patients studied ranged from 32 to 85 years. The frequencies of ischemic stroke in different age group varies with age (Table-I). Majority of the patients (50.94%) were from 60-69 years of age group.

Table-I

Age distribution of the study population. (NP%53)

Age(years)	No. of patients	Percentage (%)
30-39	2	3.7
40-49	4	5.66
50-59	13	24.69
60-69	27	50.94
70-79	5	11.24
80 and above	2	3.77

In this study out of 53 cases of ischemic stroke patients 37 (69.81%) were male and 16 (30.186%) were female (Figure-1).

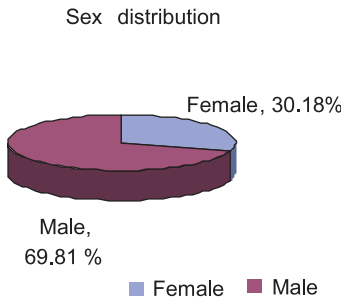

Fig.-1: *sex distribution of ischemic stroke patients.*

Out of 53 cases, 30 (56.66%) cases came from urban and 23 (43.39%) cases from rural area. The ratios of rural and urban were about 1.3:1 (Figure-2).

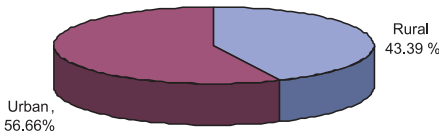

Fig.-2: *Distribution of ischemic stroke patients according to their residence.*

In this study of 53 cases, 12 (22.69%) cases were strenuous worker, e.g. Cultivation, day laborers, labor in the tea garden etc. and 41 (77.35%) cases were sedentary worker, e.g. official, businessman, housewives, retired serviceman, religious leader (Figure- 3).

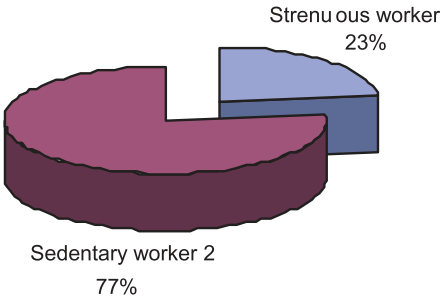

Fig.-3: *Distribution of ischemic stroke patients according to their profession.*

The most common risk factor for stroke found in this study was smoking. Out of 53 patients 33 (62.45%) were smokers. Second commonest risk factor was hypertension (54.71%). All hypertensive patients were smoker except four. Diabetes was the third commonest risk. Multiple risk factors such as smoking, hypertension, and diabetes were found together in 18 cases. Evidence of heart disease was found in 5 cases. Other risk factor found were hypercholesterolemia, obesity, alcohol intake (Table- II).

Table-II

Risk factors of the study population. (NP%53)

Risk factors	No. of patients	Percentage (%)
Smoking	33	62.45%
Hypertension	29 (Smoking+ hypertension)	54.71%
Diabetes	10	18.86%
Heart disease	5	9.43%
Hypercholesterolemia	4 (hypercholesterolemia+obesity)	7.54%
Alcohol intake	1	1.88%

Motor function recovery using Modified Rankin Scale

According to Modified Rankin Scale decreasing of score in course of time indicates the motor function recovery. That means decline of score from “06” to “0” indicates improved recovery.

a. Group-I (Piracetam treated group):

Before administration of piracetam, Modified Rankin scale (Mean±SD) was recorded as 3.88±0.78. Seven days and thirty days after inception of treatment motor function scale decreased to 3.70±0.79 and to 3.02±0.84 respectively in this treatment groups (Table-III). One way ANOVA shows significance motor function recovery in this treatment group-

[F (2, 72) =7.89; PP%0.0007]. The effect of piracetam treatment is graphically shown in Figure-4.

Table-III

Effects of piracetam and vinpocetine administered orally at a dose of 800mg and 10 mg respectively three times daily on motor function recovery estimated by Modified Rankin Scale as was measured 7 and 30 days after initiation of treatment.

	Pre treatment	After 7 days . of treatment	After 30 days of treatment	P
Group-I (Piracetam treated group) N=25	3.88±0.78	3.70±0.79	3.02±0.84*	P=0.0007
Group-II (Vinpocetine treated group) N=24	3.85±0.81	3.66±0.86	3.12±0.79[a]%	P=0.009

Values are in Mean ±SD

*Significance of difference compared to pretreatment level at P<0.05

[a]%Difference between piracetam and vinpocetine at P>0.05

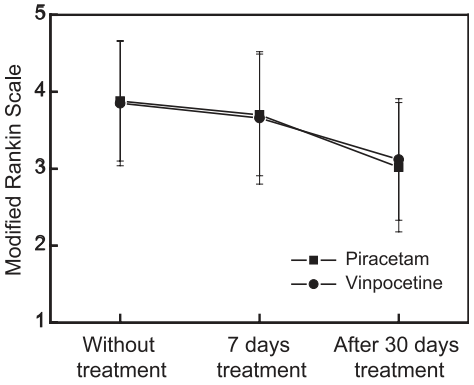

Fig.-4: *Effects of piracetam and vinpocetine on motor functions recovery after 7 days and 30 days of treatment measured by Modified Rankin Scale. (Mean± SD)*

*Significance of difference in motor function recovery in Piracetam and Vinpocetine treated group compared to pretreatment level at P<0.05

b. Group-II (Vinpocetine treated group): Before commencement of vinpocetine treatment Modified Rankin scale (Mean±SD) was recorded 3.85±0.81. After seven days and thirty days of inception of treatment motor function recovery scale decreased to 3.66±0.86 and to 3.12±0.79 respectively in this treatment group (Table-III). One way ANOVA shows significance motor function recovery in this treatment group [F (2, 69) =5.03; P=.009]. [Figure-4]

c. Comparison of motor function recovery in Piracetam and Vinpocetine treatment group:

After seven days of treatment neither piracetam nor vinpocetine showed any improvement in motor function recovery. After thirty days Modified Rankin Scale in piracetam and vinpocetine treated group was documented (Mean±SD) as 3.02±0.84 and 3.12±0.79 respectively (Table-III). One way ANOVA shows no significance difference between piracetam and vinpocetine in motor function recovery in the treatment groups- [F (1, 47) =0.199; P>0.05, NS]. The effect of both piracetam and vinpocetine treatment were shown in Figure-4.

Cognitive function recovery using Mini Mental status Examination:

In Mini Mental status scale, increase of score with time indicates the improvement of cognitive function. That means score zero (0) tends towards thirty (30) indicates better outcome.

a. Group-I (Piracetam treated group):

Before administration of piracetam Mini Mental status scale (Mean ±SD) was recorded as 14.04±6.97. After seven days and thirty days of inception of treatment cognitive function scale increased to 15.48±5.98 and to 21.08±2.87 respectively in this treatment groups (Table-IV).

Table-IV

Effects of piracetam and vinpocetine administered orally at a dose of 800mg and 10 mg respectively three times daily on cognitive function recovery estimated by Mini Mental Status as was measured 7 and 30 days after initiation of treatment.

	Pre treatment	After 7 days of treatment	After 30 days of treatment.	P
Group-I (Piracetam treated group) N=25	14.04±6.97	15.48±5.98	21.08±2.87*	P=0.0006
Group-II (Vinpocetine treated group) N=24	14.29±7.70	16.54±6.14	20.79±2.66[a]%	P=0.001

Values are in Mean ± SD

*Significance of difference compared to pretreatment level at P<0.05

[a]%Difference between piracetam and vinpocetine at P>0.05

One way ANOVA shows significance improvement of cognitive function in this treatment group- [F (2, 72) =11.18; P=0.0006]. The effect of piracetam treatment is graphically shown in Figure-5

b. Group-II (Vinpocetine treated group):

Before commencement of vinpocetine treatment, Mini Mental status scale (Mean ±SD) was recorded 14.29±7.70. Seven days and thirty days after inception of treatment scale increased to 16.54±6.14 and to 20.79±2.66 respectively in this treatment group (Table-IV). One way ANOVA shows significance improvement of cognitive function in this treatment group- [F (2, 69) =7.52; P=0.001]. The effect of piracetam treatment is graphically shown in figure-5

c. Comparison of cognitive function recovery in Piracetam and Vinpocetine treatment group:

After seven days of treatment neither piracetam nor vinpocetine showed any improvement in cognitive function recovery. After thirty days Mini Mental Status Scale in piracetam and vinpocetine treated group was documented (Mean±SD) as 21.08±2.87 and 20.79±2.66 respectively (Table-IV). One way ANOVA shows no significance difference between piracetam and vinpocetine in cognitive function recovery in the treatment groups [F (1, 47) =0.13; P=0.7175, NS]. The effect of both piracetam and vinpocetine treatment were shown in Figure-5.

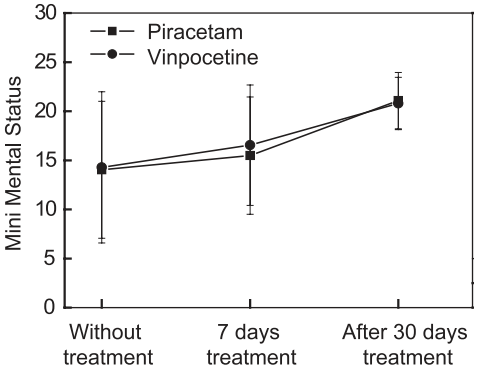

Fig.-5: *Effects of piracetam and vinpocetine on cognitive function recovery after 7 days and 30 days of treatment measured by Mini Mental Status Examination. (Mean±SD)*

*Significance of difference in cognitive function recovery in Piracetam and Vinpocetine treated group compared to pretreatment level at P<0.05

In the Group-I (piracetam treated group) there were no significant adverse reactions such as allergic reaction, GIT upset, in

given dose regimen and well tolerated.

In the Group-II (vinpocetine treated group) the drug had showed no side effects in given dose regimen and well tolerated.

Discussion

Stroke is the third leading cause of death and probably the most important cause of long term disability in most western nations [17] also it is progressing in our country.[18] Most patients who survive the acute phase of an ischemic stroke regain some of the lost function.[19] The improvement of sensori-motor function is accompanied by increased blood flow and / or metabolism in impaired region surrounding focal infarcts and in contralateral regions of the undamaged hemisphere [20, 21] which might correlate with the functional remapping of the cortex demonstrated in experimental studies.[22] Recovery after stroke is accelerated and facilitated by rehabilitation therapy, which might be supported by various drugs.[23] Whereas the effect of physiotherapy for the improvement of sensorimotor deficits is unchallenged, the efficacy of the speech therapy is still controversial with several randomized controlled trials yielding no difference in outcome between treated and non treated groups.[24]

Therefore, many trials were undertaken to enhance recovery of motor and cognitive functions after ischemic stroke with the use of many pharmacological agents [25] and have focused mainly those agents which improve cerebral reperfusion or on neuroprotection.[26]

Piracetam is regarded as the first and most important member of a class of drugs known as nootropic agents, [27] these are drugs which, under certain conditions, improve a whole series of mental functions, particularly higher cortical function such as learning ability (especially language), memory, ability to think, consciousness, etc.[28]

The principal finding is that motor function recovery measured by Modified Rankin Scale (MRS) for patients treated with piracetam and vinpocetine were significantly improved ($P<0.05$) over the course of the study. Although the improvement in Modified Rankin Scale (MRS) followed a similar pattern between piracetam and vinpocetine treated patients, the recovery was not significant difference ($P>0.05$) between piracetam and vinpocetine group (Fig-5).

In the present study piracetam exhibited statistically significant improvement ($P<0.05$) of cognitive function estimated by Mini Mental Status score in post stroke patients who received tab piracetam orally as a part of four weeks of stroke treatment (Fig-6). This is possibly due to improvement of microcirculation in cerebral vessels and also its neuroprotective effects.[28]

Neuroprotective drug vinpocetine, synthetic derivatives of apovincamine, was reported to protect against exitotoxic cell death in cell culture of rat cerebral cortex [29] and in striatal slice of rat brain.[30] Vinpocetine has also demonstrated calcium antagonist activity in vitro models of cerebral ischemia.[31] Vinpocetine showed neuroprotective and anticonvulsive effect by blocking voltage-gated Na-channel in rat cortical neurons.[32]

In our study it has been observed that vinpocetine exhibited statistically significant ($P<0.05$) improvement with time on cognitive

function (measured by Mini Mental Status Score) who received the drug as a part of four weeks of treatment (Fig-6).

Compared with pre-treatment, both groups of patients, those treated with piracetam and vinpocetine showed a significant improvement ($P<0.05$) in Mini Mental Status (MMS) rating. This implies that the more rapid and relatively complete rehabilitation of neurological function is due to the effect of piracetam and vinpocetine.[33] However, the difference between piracetam and vinpocetine on recovery of cognitive function was not significant ($P>0.05$).

A possible limitation of the study was that patient numbers were small and duration of the study was short. Also certain of the cases investigated, although fulfilling the criteria for inclusion, were considered relatively mild although the investigator tried to balance this later in the trial by recruiting more seriously debilitated patients. It is important that further study is needed with different doses of Piracetam and Vinpocetine singly or in combination on large number of patients to conform or rebut our findings and systemic reviews spanning the whole range of management of stroke are required to delineate future research.

Conclusion: Although patient number was not large and the debilitation of the patient was not very serious, the present study suggested that piracetam and vinpocetine can accelerate functional recovery after ischemic stroke to some extent and is well tolerated. The present study may enhance the recent clinical developments, comparing vascular targets to the common neuroprotective strategies and is just a step to create acceptability among the Physicians to use Piracetam or Vinpocetine in ischemic stroke treatment other than antithrombotic, anticoagulant or neuroprotective agents.

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Stroke and Diabetes Mellitus - A study of 100 patients

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Abstract:

Introduction: One hundred consecutive stroke patients admitted in different Medical units of Dhaka Medical College Hospital (DMCH), Dhaka, Bangladesh were studied. The purpose of this study was to see the relationship of diabetes mellitus with of stroke.

Materials and methods:A patient male or female of any age presented with signs and symptoms of stroke were included in this study. Any patient later diagnosed by investigation other than stroke was excluded from the study. The cases were diagnosed on the basis of clinical presentation, thorough physical examination and supportive laboratory investigations. CT scan of brain and echocardiography was done in some selected cases. All the necessary informations were collected in a standardized data sheet. All the data were analyzed manually.

Results:There were 74(74%) male and 26(26%) female patients with ratio being 2.8:1. Maximum age (56%) was in between 6th and 7th decade.Most (71%) of the patients were literate. Majority (53%) of the patients were of poor class group. Eighteen percent (18%) patients were diabetic, of them 44.4% of the patients were diabetic for 5-10 years. About 28% were diagnosed as diabetics for the first time in hospital. About 61% patients had partial or almost complete recovery when discharged, 28% patients had no improvement and 11% expired in hospital.

Conclusion: Diabetes is an independent risk factor for stroke both directly and indirectly. Indirectly it initiates vascular thrombosis, atherosclerosis, ischaemia and dyslipidaemia.

Introduction

A stroke or cerebrovascular disease is a rapidly developing episode of focal and at times global loss of cerebral function with symptoms lasting for more than 24 hours, with no apparent cause other than that of vascular origin[1]. Now-a-days stroke has a dominant place in the structure of neurological morbidity and mortality[2]. Multiple studies have shown that people with diabetes are at greater risk for stroke compared to people without diabetes. According to World Health Organization, stroke is the third commonest cause of mortality in the developed countries in the world, immediately following ischaemic heart disease and malignant diseases[2].

There is no adequate data on the incidence of stroke in Bangladesh. In one study it

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was found that stroke was the second commonest cause of emergency admission in medicine units in Dhaka Medical College Hospital and constituted about 10-12% of total admission in these units[3].

Various cross-sectional and prospective epidemiological studies have identified many risk factors for stroke and the most important of these is hypertension. The relation of risk factors other than hypertension to stroke was less emphasized. Cerebrovascular diseases are important causes of morbidity and mortality in vast majority of diabetic population specially in noninsulin dependant diabetes mellitus (NIDDM)[4]. In Bangladesh about 1-2% of population are diabetic[5]. The appearance of the disease is influenced by many etiological factors, which are risk factors. Diabetes Mellitus, hypertension and heart disease are associated risk factors for developing stroke[6]. Diabetes mellitus (DM) is one of those risk factors which can cause significant morbidity in stroke patients[2]. So this study was carried out to examine the relationship between DM and stroke along with extent of drug compliance in patient with stroke that was diabetic.

Materials and methods

This study was carried out among one hundred stroke patients admitted in different units of DMCH from August 2002 to December 2003. Patients were identified as diabetic, who has had definite history of diabetes and confirmed by laboratory test, had end organ involvement of diabetes (like diabetic retinopathy, nephropathy, and significant proteinuria). Cases of diabetes were diagnosed or reconfirmed by standard laboratory tests for detection of diabetes (WHO criteria)[7]. The cases were selected irrespective of age, sex and duration of illness. The patients were included from all socioeconomic classes. In each case, particulars of the patient and a detailed history about diabetes, hypertension, obesity and family history of stroke was noted. In case of unconscious patient, history was obtained from close relatives, particularly spouse or children. The diagnosis of stroke was made on the basis of thorough physical examination with special emphasis on nervous system and supportive laboratory investigation like complete blood count; blood sugar (RBS, FBS & blood sugar 2 hours after breakfast) blood urea, serum creatinine, serum electrolytes, serum lipid profile, ECG, and chest x-ray P/A view were done in all cases. Special investigations like CT scan of brain and echocardiography was done in some selected cases. Patients were treated with appropriate diet and hypoglycemic agents according to individual need. All the necessary information was collected in a standardized data sheet. Subsequent prognosis and course in hospital was followed and the prognosis at the time of discharge was noted. All the data were analyzed manually and presented in a tabulated form.

Results:

Out of 100 patients with stroke, there were 74 males and 26 females, the ratio being 2.8:1. The age and sex distribution of the patients is shown in Fig. 1.

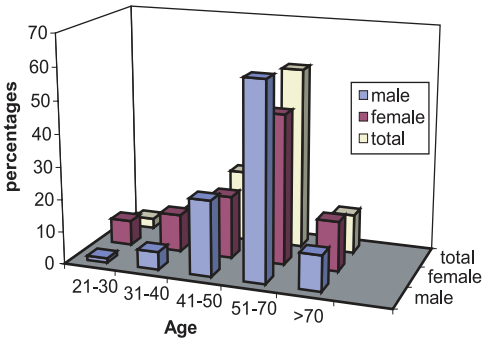

Fig-1: *Age and sex distribution of study patients (n=100).*

The highest number of patients (46%) were in the age group of 51-70 years. Among the patients, there were 3 patients in the age group of 21-30 years. The age of the oldest patient was 92 years.

Table-I

Educational status of study patients (n=100)

Literacy	Number of patients	Percentage
Illiterate	29	29.00
Literate	71	71.00
a) Gone to school	56	
b) Graduate or above	15	

The educational status is shown in table I. It shows that 29 patients had no formal schooling while 15 patients had graduation or above and 56 patients had some schooling. These patients had gone to school, some of them read up to twelve class.

Table-II

Distribution of patients in different income group and social classes (n=100).

Level of income(Taka / month)	Economic class	Number of patients	Percentage
<3000	Very poor	9	9.00
3000-5000	Poor	53	53.00
5001-15000	Middle	25	35.00
>15000	Rich	13	13.00

Among the patients nine were very poor. Majority (53%) of the patients belong to the poor income group, 25% had income between taka 5,001 to15,000 per month and only 13% had more than Tk. 15000 per month.

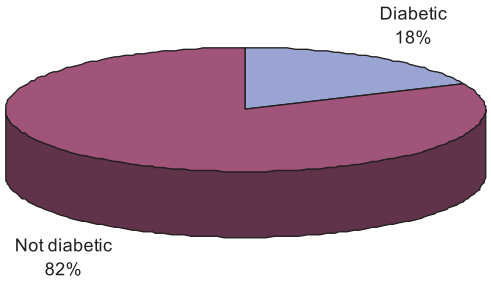

Fig-2: *Association with diabetes mellitus (n=100).*

Fig.-2 shows that 18% of stroke patients are diabetic. Eighty two percent patients are non-diabetic.

Fig.-3 shows that majority (44.4%) of patients were diabetic for 5-10 years. Only in 5 patients (27.70%) diabetes was detected for the first time after stroke.

Fig.-4: Outcome of study subjects (n=18).

Of the 18 patients, 11 (61.11%) patients had partial or almost complete recovery

when discharged, 5 (27.77%) patients had no improvement and 2 (11.11%) expired in hospital.

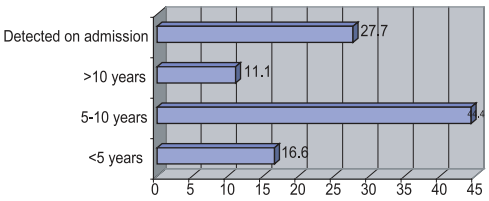

Fig-3: *Duration of diabetes (n=18).*

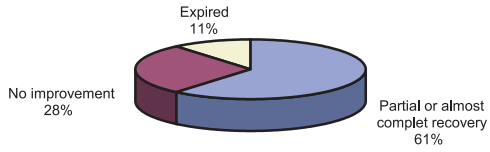

Fig-4: *Outcome of study subjects (n=18).*

Discussion

Stroke is largely a disease of elderly. The incidence of stroke usually rises sharply with age. In this study peak age incidence was between 6th and 7th decade (Fig I) which coincides with the results of various studies in home and abroad[8-12]. No case was found at the age of 20 years or below and only 3 percent of the sample population were below 30 years. This study also shows that the incidence of stroke rises with the increase of age and sharply falls in percentage after the age of 70 years. In some previous studies, it has been shown that between the sexes, men suffer with stroke more than females and it affects males 1.5 times more often than females[13, 15]. In this study 74% were males and 26% were females (Fig I), the ratio beings 2.8:1 which coincides with other study[16-17]. Mannan & Alamgir, have shown slight difference (M: F=4:1) with higher male preponderance[8]. In Spain stroke is the commonest cause of death in women and the second commonest in men[2]. In Taiwan women also experiences a higher mortality rate. In Nigeria, almost 1 in 4 men and 1 in 5 women aged 45 years can expect to have a stroke if they live to their 85th year[2]. The preponderance of males in this study may be due to cultural attitude of our society that the females are generally not brought to the hospital or as the disease is of old age and our females live shorter than their male counterpart; this was reflected indirectly in this study.

In the present study, illiterate group (Table-I) comprises the majority (71%) of the study population, 15% patients were graduates or above and 56% patients have some schooling, though they were not graduated. Twenty-nine patients had no formal schooling. This coincides sharply with the studies done previously[16, 17]. This may reflect the fact that awareness about stroke and stroke related treatment is present in the literate mass more than the illiterate reflecting the results of our study.

In this study the larger percentage (53%) of people are of poor class group and 13% are of higher class group (Table-II). Roy PK et al have shown that maximum number of patients (48%) belonged to the poor class group[16]. This reflects the fact that maximum number of stroke affected people in our country are poor. This coincides with a study done previously in our country but does not coincide with the study done by Chapman where the author has shown increased incidence of stoke in higher class group[9].

In this study 18% of stroke patients are diabetic (Fig.-2). Diabetes mellitus is one of the important risk factor for ischaemic

stroke. Diabetes mellitus enhances the atherosclerotic process and as a result cerebral thrombosis increases in diabetic patients. Diabetes promotes cerebral atherosclerosis and may increase hypertension, hyperlipidaemia and coronary heart disease, which are risk factors for stroke[18-21]. Because of metabolic changes, diabetic persons have an increased risk of thrombosis and increased blood viscocity[20, 22].

Thus, although diabetes is a primary risk factor, it's effects are clearly modulated by the presence of other risk factors[23, 24]. Wolf suggested DM is an important risk factor. Their data showed that diabetes enhances the incidence of atherosclerotic brain infarction (ABI) in women of 50 years age. However men are also affected in between 50-70 years age[23]. In one study it has been shown that nonfasting hyperglycemia is a significant risk for stroke mortality. But this association could be found only for women[25].

In this study duration of diabetes is also important. Most of the stroke patients have diabetes more than 5-10 years duration (44.5%) (Fig.-3). Rahim in his study of 165 cases of diabetic patients, all of them developed stroke in less than 10 years duration[4] . In another study, stroke was the underlying cause of death in 7% of diabetic patients[26]. Results of 16 years follow up in the Framingham study showed that diabetic subjects developed more cerebrovascular events than nondiabetics[27]. In a study by National Health and Nutrition Examination survey, diabetes was found to be a risk factor for hemorrhagic stroke in men and women and for thromboembolic stroke in women[28]. Hyperglycemia produces increased anaerobic metabolism, raised lactic acid production in ischaemic brain tissue, and cellular acidosis[25]. Marquardson commented that impaired glucose tolerance played an additive role for the atherosclerotic brain infarction[29].

This study revealed 61% had partial or complete recovery and 11% expired in hospital (Fig.-4). Mortality and morbidity enhanced by 1.5-2.0% when diabetes mellitus is present [28].There is a link between diabetes mellitus, hypertension and hyperlipidaemia, it is difficult to assess which has the most effect on stroke. However, when all three conditions are present, the relative risk of suffering from a stroke is greater[15].

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CASE REPORT

Neurocysticercosis – A Case Report

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Abstract

A young Christian Bangladeshi lady presented with headache, vomiting, convulsions and papilloedema. Her magnetic resonance imaging (MRI) showed numerous pin-head sized enhancing cystic lesions all over the brain, strongly suggestive of Neurocysticercosis. With albendazole, steroid and anticonvulsant, she responded dramatically with resolution of symptoms, signs and marked reduction of lesions in MRI. But her headache and vomiting recurred as soon as albendazole and steroid were stopped. She was given adequate dose of praziquantel with continued maintenance dose of albendazole, steroid and anticonvulsant. But she continues to have active, enhancing lesions in MRI and recurrence of symptoms without steroid.

Neurocysticercosis is infrequently encountered in Bangladesh. At least, some cases do not respond adequately to traditional anthelmintic therapy and may require long term maintenance steroid treatment to remain symptom free.

Case Report:

A seventeen year old, Christian, Bangladeshi, right handed lady presented in April 2005 with a 10 day history of headache, vomiting and convulsion. Her headache started gradually, was persistent and diffuse in nature and was gradually getting worse. It was worst in the early morning and was associated with effortless vomiting. Two days prior to her presentation she had three episodes of collapse with brief loss of consciousness associated with generalized tonic-clonic convulsions. In one episode she bit her tongue and in one became incontinent to urine. Her parents gave history of her remaining confused and 'groggy' for 15-30 minutes after each episode, when she made an apparent full recovery. She does not give any history of fever, double vision, difficulty in speech, swallowing or walking. There was no prior history of significant headaches, head injury, unconsciousness or convulsion, nor was there any family history of epilepsy. Her family and she consume pork on occasions. There was no history of intake of alcohol or any illicit drug.

On examination, she looked ill with normal general examination parameters. She was fully conscious and oriented with normal speech. All her cranial nerves were intact except that fundoscopy revealed bilateral gross papilloedema with peripapillary splinter haemorrhages. There was no other abnormality in neurological examination and her other systemic examination was normal.

She was hospitalised immediately. Her routine blood, stool and chest X-ray revealed no abnormality. An urgent MRI was performed which showed numerous pinhead sized, ring enhancing cystic

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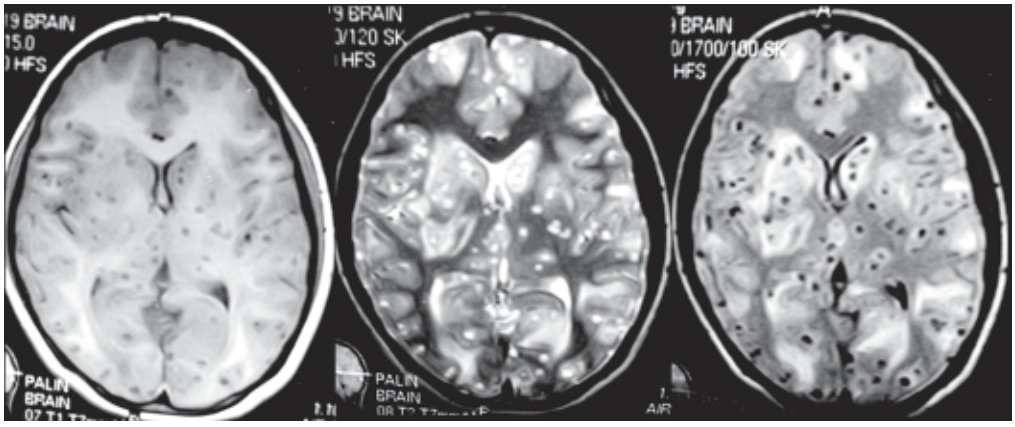
lesions with perilesional oedema scattered all over the brain and was strongly suggestive of Neurocysticercosis (see figure). She was started on intravenous (IV) dexamethasone 5 mg 6 hourly, oral albendazole 5 mg/kg three times daily and carbamazepine (CBZ) 200 mg twice daily. Within 24 hours she improved dramatically with resolution of all symptoms and signs. Her dexamethasone was replaced by oral prednisolone 60 mg/day after 3 days. Albendazole was continued for 4 weeks and then stopped, when oral prednisolone was gradually tapered off. She was discharged home with an advise to continue carbamazepine and a planned repeat MRI brain after 3 months.

Unfortunately she was again hospitalised after one month with recurrence of headache and vomiting but there was no convulsion and papilloedema on examination this time. Repeat MRI brain was performed, which revealed relatively less but similar and active enhancing lesions of cysticercosis. She was again started on oral albendazole and prednisolone with dramatic response once again. But as soon as her steroid was attempted to be discontinued, her symptoms tended to come back. She was given a course of praziquantel in a dose of 50 mg/kg/day for 2 weeks and her albendazole and steroid were continued. After 2 weeks her prednisolone was gradually reduced to a maintenance dose of 20 mg every alternate day. She was again discharged home with an advise to continue low dose alternate day oral steroid, albendazole and CBZ.

When she was reviewed 6 weeks later, she was asymptomatic with no signs at all. But her repeat MRI showed active, enhancing lesions of cysticercosis, though markedly reduced in number than initial scans. Since then she had 4 repeat MRI brain, all of which showed apparently active lesions of variable numbers. She had 4 attempts to gradually stopping prednisolone and each time she had relapse of symptoms. At the moment she remains well and stable with albendazole 5 mg/kg twice daily, carbamazepine 200 mg twice a day and oral prednisolone 20 mg every alternate day.



T1 T2 Flair

Discussion:

Human neurocysticercosis (NCC) is caused by the infection of the central nervous system with the larval forms of the pork tapeworm *Taenia solium*. Humans are the only definitive hosts for *T. solium* and pigs are the usual intermediate hosts. The adult tapeworm resides in the upper jejunum, produces eggs, which are excreted into faeces. The eggs are infectious for both humans and pigs. Infections that cause human cysticercosis follow the ingestion of T. solium eggs, usually from faecally contaminated food. The larvae hatches, penetrate the gut wall, disseminate haematogenously and encyst on many organs like brain causing NCC[1].

Neurocysticercosis is uncommon in Bangladesh. Because Bangladesh being a Muslim country and having religious barrier against pig culturing and pork eating, remains non-endemic for cysticercosis. But NCC is common worldwide and is considered endemic in many countries of the Latin America, Africa, South and Southeast Asia including India and Eastern Europe[1-7]. Epidemiological studies using computed tomography (CT) in endemic settings have revealed asymptomatic cases in 10-20% of the general populations[8-10]. In the USA and most of Europe, NCC is increasingly recognised as a cause of seizures[11], primarily in immigrants and individuals who have travelled to endemic areas. It is non-endemic in most Muslim countries[12], though has been reported in Indonesia, Saudi Arabia and Pakistan[13-15]. In Bangladesh, NCC is presumed to be confined mainly within the Christian and Hindu community, as in our case.

This particular case of NCC, presented with seizures and features of raised intracranial pressure (ICP). In fact these are the two most common documented presentations of NCC[1,16], seizures being the commonest (50-80%)[17-19]. In endemic setting, NCC is the most common cause of acquired epilepsy, and in nearly 30% of patients with seizures, the cause is NCC[8-10]. Almost half of the reported cases have generalised seizures and the other half have partial seizures[19,20-22]. Our case presented with generalised seizures.

This particular case of NCC was diagnosed on the basis of clinical ground and neuroimaging (MRI) findings. Biopsy was not agreed upon by the patient and family and serological tests for NCC was not available. As a matter of fact neuroimaging is the mainstay of diagnosis and typical findings in MRI is considered as the most sensitive and specific diagnostic tool[13,15,19,23] and may even be pathognomonic[24].

There is much debate about treatment of human NCC with cysticidal drugs[25]. In general cysticidal therapy is used where there are multiple parenchymal lesions and there is apparent benefit in patients with enhancing intraparenchymal lesions[12,13,26-29]. Our case responded to cysticidal drugs with resolution of some of the lesions in imaging with clinical improvement but it was not possible to achieve complete remission with continued use of albendazole and a course of praziquantel. Similar inadequate or no response at all has been found out in some controlled trials[1]. In our case corticosteroids were used, initially with I.V. dexamethasone and then with oral prednisolone. Both dexamethasone and

prednisolone are recommended to decrease cerebral oedema and inflammatory reaction that may be present or may ensue after start of cysticidal drugs[1,13,19]. Our case required long term low dose alternate day oral prednisolone to prevent relapse of symptoms, which does not seem to be well studied. Whereas, seizures caused by NCC requires single first line antiepileptic drug (AED) e.g. carbamazepine or phenytoin in conventional doses[13,29-32]. Our patient remained complete seizure free after initiation of carbamazepine.

In conclusion, we can say that neurocysticercosis is uncommon in Bangladesh. There was no doubt about the diagnosis. Seizures were well controlled with antiepileptic drug but complete resolution of active lesions could not be achieved by combination cysticidal drugs and long term oral steroid was necessary to prevent relapse of symptoms.

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REVIEW ARTICLE

Obesity and Overweight: Small Lifestyle Changes Can have the Biggest Impact

MD. MATIUR RAHMAN[1], FAISAL AHMED[2], MD. AZAM[3], BADRUL ALAM MONDOL[4], SHAKHAWAT HOSSAIN[5] QUAZI DEEN MOHAMMAD[6]

Introduction:

Obesity is often defined simply as a condition of abnormal or excessive fat accumulation in adipose tissue, to the extent that health may be impaired[1]. The distribution of fat induced by weight gain affects the risk associated with obesity, and the kind of disease that result. Indeed, excess abdominal fat is as great a risk factor for disease as is excess body fat per se. It is useful therefore, to be able to distinguish between those at increased risk as a result of "abdominal fat distribution", or "android obesity"; from those with the less serious "gynoid" fat distribution, in which fat is more evenly and peripherally distributed around the body. The prevalence of overweight and obesity is increasing worldwide at an alarming rate in both developed and developing countries. The report of the **WHO MONICA** project shows that in all but one male population and in the majority of female populations, during the period 1983-1986, between 50% and 75% of adults aged 35-46 years were either overweight or obese[2]. In excess of 50% of adult population and nearly one third of children in Mexico have overweight and obesity[3]. Over 97 million adults are overweight or obese in USA[4].

Classification of overweight and obesity according to BMI (Body Mass **Index)**

BMI is a simple index of weight-for-height that is commonly used to classify underweight, overweight and obesity in adult. It is defined as the weight in kilograms divided by the square of the height in meters (kg/m^2).

The classification of overweight and obesity, according to BMI, is shown in table-I.

Table-I

Classification of adults according to BMI

Classification	BMI	Risk of comorbidities
Underweight	<18.50	Low (but risk of other clinical problems increased)
Normal Range	18.50-24.99	Average
Overweight	>25.00	
Preobese	25.00-29.99	Increased
Obese class I	30.00-34.99	Moderate
Obese class II	35.00-39.99	Severe
Obese class III	≥ 40.00	Very severe

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These BMI values are age-independent and the same of both sexes. However, BMI may correspond to the same degree of fatness in different populations due, in part, to differences in body proportions. The table shows a simplistic relationship between BMI and the risk of comorbidity, which can be affected by a range of factors, including the nature of the diet, ethnic group and activity level. The risks associated with increasing BMI are continuous and graded and begin at a BMI below 25. Both BMI and a measure of fat distribution (waist circumference or waist hip ratio [WHR]) are important in calculating the risk of obesity comorbidities. Evidence suggests that the risks of being "overweight" decreases with increasing age. On the basis of mortality, the ideal body mass index (BMI) is higher in older than young adults, with an optimum BMI for people older than 65 in the young adult "overweight" range of 27-30 kg/m^2. In a systematic review, Heiat et al concluded that the relation between BMI and mortality in people older than 65 is a flat bottomed, U-shaped curve, with mortality rising only at BMI > 31 kg/m^2 and perhaps not at any BMI in people older than seventy five[5]. Most studies that examine the risk of adverse health associated with obesity in Asian countries have been based on data from Europe or the United States. However, the increased health risks associated with obesity occur in people with lower BMI in the Asia-Pacific region when the standard criteria are used. It has been recognized that the current WHO criteria to classify overweight and obesity in adult Europeans using the BMI or waist circumference may not be appropriate in Asian or Pacific Island populations. The steering committee of the Asia-Pacific perspective for redefining obesity, co-sponsored by the WHO Regional Officer for the Western Pacific (WPRU), the International Association for the Study of Obesity and the International Obesity Task Force has recommended different ranges for the Asia-Pacific region based on risk factors and morbidities (Table II). In Asians, the cut-offs for overweight (23.0 kg/m^2) and obesity (25.0 kg/m^2) are lower than the WHO criteria[6].

Table II

Proposed Classification of Weight by BMI in Adult Asians[6]

Classification	BMI	Risk *of* Co-Morbidities
Underweight	< 18.5	Low
Normal Range	18.5 -22.9	Average
Overweight > 25.0	23.0- 24.9	Increased
Pre-Obese/At Risk	25.0-29.9	Moderate
Obese I	30.0-34.99	Severe
Obese II	35	Very severe

Body Fat Distribution and Its Importance

Waist circumference and waist hip ratio

A high WHR (WHR>I.0 in men and >0.85 in women) indicates abdominal fat accumulation[7]. However, recent evidence suggests that waist circumference alone - measured at the midpoint between the lower border of the rib cage and the iliac crest - may provide a more practical correlate of abdominal fat distribution and associated ill health[8]. Waist circumference is a convenient and simple measurement that is unrelated to height[33], correlates

closely with BMI and WHR[9] and is an approximate index of intra-abdominal fat mass and total body fat[10]. Furthermore, changes in waist circumference reflect changes in risk factors for cardiovascular disease (CVD) and other forms of chronic disease[11].

Aetiology of Obesity

Obesity is a multi-cause syndrome, there are individual and population differences in energy balance (ethnical, diet behavior, longer life expectancies). Genetic factors may influence the setting of obesity, but the calorie intake, physical activity, and lifestyle are critical determinants[12].

In a few cases of obesity, specific casual factors can be identified and treated (Table-2). However, for the most part, the etiology of obesity arises from a complex interplay of behavioral and genetic factors.

Behavioral factors

Table-III

Specific Causes of Weight Gain

Endocrine factors
Hypothyroidism
Cushing's syndrome
Hypothalamic tumors or injury
Insulinoma
Drug treatment
Tricyclic antidepressants
Sulphonylurea drugs
Oral contraceptive pill (some agent)
Sodium valproate

Genetic

Prader-Willi syndrome (childhood obesity with abnormal appearance and CNS function).

There has been a substantial increase in the prevalence of obesity in developed countries over the past 20 years. However, studies of reported food intake in the UK show that individuals are consuming no more in the way of energy than they did 20 years ago. The major factor leading to obesity in the population therefore seems to be an overall decrease in activity levels[13].

Other important behavioral factors predisposing to obesity are, high-fat diets, snacking and the loss of formalized meal patterns, consumption of energy-dense foods, alcohol consumption.

Genetic factors

A few rare single gene disorders have been identified which lead to a symptom complex including obesity. These include mutations of the melanocortin-4 receptor (MC4R) that accounts for approximately 5% of severe early onset obesity, the Prader-Willi syndrome and mutations in the leptin gene[14]. **Leptin** is an adipose derived hormone, which acts on the hypothalamus and was originally thought to be a powerful homeostatic factor for maintaining body weight.

Chronic Diseases Associated with Obesity

Although obesity should be considered as a disease in its own right, it is also one of the *key* risk factors for other non-communicable diseases (NCUs), such as type 2 diabetes and CHD, together with smoking, high blood pressure and hypercholesterolemia. The adverse health consequences *of* obesity are influenced to

a greater or lesser extent by body weight, the location of body fat, the magnitude of weight gain during adulthood, and a sedentary lifestyle[15].

Cardiovascular disease

Cardiovascular disease (CVD) encompasses coronary heart disease (CHD), stroke and peripheral vascular disease. CHD and stroke account for a large proportion *of* deaths in men and women in most industrialized countries, and their incidence is increasing in developing countries. Obesity alone is the cause *of 11% of* cases of cardiac failure in men and 14% of cases in women in the United States[16].

Arterial hypertension is the most frequent cardiovascular disease in obese persons, progressing with time to left ventricular hypertrophy, often associated with dilatation, diastolic disorders, heart's rhythm disturbance, and generalized atherosclerosis[17].

The association between hypertension and obesity is well documented. Both systolic and diastolic blood pressure increase with BMI, and the obese are at higher risk *of* developing hypertension than lean individuals.

Obesity and non-alcoholic steatohepatitis: Obesity is the most important risk factor associated with non-alcoholic steatohepatitis. Non-alcoholic steatohepatitis has had a great impact due to the fact that previously, many cases of cryptogenic cirrhosis actually were attributed to this disease[18].

Cancer

A number of studies found a positive association between overweight and the incidence of cancer, particularly of hormone-dependent and gastrointestinal cancer (Table-IV).

Table-IV

Cancers with a higher reported incidence in obese persons

Hormone-dependent	Gastrointestinal/ hepatic/renal
Endometrial	Colorectal
Ovarian	Gall bladder
Breast	Pancreatic
Cervical	Hepatic
Prostate	Renal

Endocrine and metabolic disturbances associated with obesity

Endocrine abnormalities.

A positive association between obesity and the risk of developing Type 2 DM has been repeatedly observed in both cross-sectional[19] and prospective studies[20].

Recent research has shown that adipocytes (fat cells) are more than just fat depots. They also function as endocrine cells, producing many locally and distantly acting hormones, and as target cells for a great many hormones. Altered hormonal patterns have been observed in obese patients; especially in those with intra-abdominal fat accumulation[21]. Common hormonal abnormalities associated with intra-abdominal fat accumulation are listed in table-V.

Table-V

Common hormonal abnormalities associated with intra-abdominal fat accumulation.

Insulin resistance and increased insulin secretion
• Increased free testosterone and free androstenedione levels associated with decreased sex hormone binding globulin (SHBG) in women
• Decreased progesterone levels in women
• Decreased testosterone levels in men
• Increased cortisol production
• Decreased growth hormone levels

Metabolic Disturbance

Dyslipidaemia: Obese individuals are frequently characterized by a dyslipidaemic state in which plasma triglycerides are raised, HDL cholesterol concentrations are reduced and low density lipoprotein apo-B (LDL-apoB) levels are raised. This metabolic profile is most often seen in obese patients with a high accumulation of intra-abdominal fat and has contently been related to an increased risk of CHD[22].

Excessive intra-abdominal fat accumulation is also associated with a greater proportion of small, dense low-density lipoprotein (LDL) particles.

Role of Obesity in Causation of the Metabolic Syndrome

Most of the cases of the metabolic syndrome occur in persons who are overweight or obese. An excess of body fat is a reflection of overnutrition; the latter leads to accumulation of excess lipid in many tissues, notably the adipose tissue, muscle, liver and beta-cells of the pancreas. Excess lipid accumulation in this tissue appears to induce many of the biochemical changes that underlie the metabolic syndrome. An excess of fat in adipose tissue is accompanied by increased release of several products into the circulation, notably, non-esterified fatty acids (NEFA), PAI-1 and proinflammatory cytokines. Release of all these factors appear to be greater in individuals in whom excess fat is located predominantly in the trunk and peritoneal cavity. Increased release of NEFA from adipose tissue leads to accumulation of excess triglyceride in muscle and liver. Fat accumulation in muscle produces insulin resistance, whereas excess fat in the liver promotes atherogenic dyslipidaemia. Increased release of PAI-1 and inflammatory cytokines from adipose tissue seemingly promotes a prothrombotic state and proinflammatory state, respectively. Obesity also raises the blood pressure by multiple mechanisms.

How is obesity treated?

Fortunately, a weight loss of 5 to 10 percent can do much to improve health by lowering blood pressure and cholesterol levels. In addition, recent research has shown that a [5-10]. Percent weight loss can prevent type 2 diabetes in people at high risk for the disease. Method of treatment depends on level of obesity, overall health condition, and motivation to loose weight. Treatment may

include a combination of diet, exercise, behavior modification, and sometimes weight-loss drugs. In some cases of severe obesity, gastrointestinal surgery may be recommended. Remember, weight control is a life-long treatment[23]. Leptin is an attractive candidate for the treatment of obesity, whereas leptin has been successfully used in the treatment of leptin-deficient obese patients, trials in hyperleptinemic obese patients, have yielded variable results. Inhibition of protein tyrosine phosphatase 1B (PTP1B) has emerged as a highly validated, attractive target for treatment of not only diabetes but also obesity[24].

Surgery

Today, multiple surgical procedures for the treatment of obesity are available. As with most procedures, there are benefits and risks associated with open and laporoscopic gastric bypass surgery, as well as with laporoscopic adjustable gastric handing and partial biliopancreatic bypass with a duodenal switch. The risks and complications associated with bariatric surgery may be serious and in some cases life threatening. However, surgery for obesity has shown remarkable results in helping patients to achieve significant long-term weight control. In addition, it is associated with improvement and often resolution of co-morbid conditions, including type 2 diabetes mellitus, systemic hypertension, obesity hypoventilation, sleep apnea, venous stasis disease, pseudotumor cerebri, polycystic ovary syndrome, complications of pregnancy and delivery, gastroesophageal reflux disease, stress urinary incontinence, degenerative joint disease, and non-alcoholic steatohepatitis. Laporoscopic gastric bypass is rapidly becoming the procedure of choice for treatment of morbid obesity. Results demonstrate that the surgery is technically safe. Outcomes are similar to open gastric bypass, but with markedly lower incidences of wound-related and cardiopulmonary complications[25].

Conclusion :

The epidemic of obesity worldwide has spread at such an alarming rate over the last decade that most adults are now overweight or obese. The association of obesity with mortality and a broad range of significant medical comorbidities portends staggering healthcare, social, and economic costs. Treatment should be directed at the fundamental imbalance between energy intake and expenditure in the context of an environment that increasingly favors excess weight. Therefore, treatment plans need to address the multiple factors that contribute to obesity, including high-calorie diets, sedentary lifestyles, and weight--sustaining behaviors. Primary care physicians would do well to focus on helping willing patients make small changes motivated more by health promotion and fitness than by weight loss.

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Is Stress Related to Stroke?

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Summary

Stroke is the major cause of death and disability all over the world. Risk factors are certain characteristics that give an increased likelihood of a stroke occurring. Some are specific risk factors like increased age, hypertension, IHD, atrial fibrillation, diabetes mellitus, peripheral vascular disease, raised haematocrit, high cholesterol, low cholesterol, high plasma fibrinogen, smoking, alcohol, obesity, TIA, oral pill and collagen vascular disease. Some factors are under ongoing study to be proved as risk factor for stroke. Among these stress and infection are important. As a part of an attempt to address the above issue the present study was designed to investigate the stress in 472 cases of recent stroke patients. The patients were selected from Neurology Department of Sir Salimullah Medical College, Bangabandhu Sheikh Mujib Medical University, Dhaka Medical College and BIRDEM, Dhaka. The study was carried out from July 2003 to June 2005. Stress was assessed in each case. Out of 236 cases 135 were male and 101 were female. The patient's age range was 45 to 76 years and the mean age was 59.6 years with SD ± 13.2 years. CT showed that 84% had infarction, 10% had intracerebral haemorrhage and 6% had SAH. Stress was found in 163 cases and 109 controls. Statistical analysis showed that odds ratio was 2.6 with 95% CI 1.78 - 8.09 and x^2 value was 25.3 with $p<0.001$ which is very much significant. Stress was found in 149 cases of infarction and 109 controls with odds ratio 3.54 with 95% CI 2.34 - 5.34 and x^2 value 37.37 with $p<0.001$ which is very much significant. But in cases of intracerebral haemorrhage and SAH showed that stress was not a significant risk factor for them.

Introduction

The National Survey of Stroke in USA used the following definition. Stroke is a clinical syndrome consisting of a constellation of neurological findings, sudden or rapid in onset which persist for more than 24 hours and whose vascular origin are limited to :

a. Thrombotic or embolic occlusion of a cerebral artery resulting in infarction or,

b. Spontaneous rupture of a vessel resulting in intracerebral or subarachnoid haemorrhage.

Identification of potential stroke victims for preventive medical or surgical management would seem essential if the impact of the common and devastating illness is to be substantially reduced. It is imperative that the high-risk individual be detected early and management instituted before crippling brain damage occurs[2]. We all know this is a age old disease but there is little medical interest and nothing about strokes to be

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learned from Assyrian, Babylonian, Jewish or Persian writings around this time, but Hippocrates, Father of Medicine who first used the word 'apoplexy' and gave a reasonable description of stroke. Many famous people like Louis Pasteur, Winston Churchill have had strokes. But standing on the turn of the century, we can hardly claim for the cure of this disease. Cure is not the answer. Prevention is possible to some extent by detection and reduction of the risk factors of the disease. Certain risk factors, like hypertension, diabetes mellitus, ischaemic heart disease, obesity, less physical activity, smoking, etc. are claimed to be associated with stroke [3]. Recently stress and infection is added to the above list of risk factors for stroke. In Bangladesh there is no epidemiological data on cerebrovascular disease. The risk factors related to this dreadful disease are yet to be available[4].

Considering all these factors, effort has been made in the present study to detect risk factors of this disease. A hospital-based study of patients is prone to selection bias as those with more severe or unusual types of stroke are more likely to be admitted to hospitals. With this shortcoming, this study was designed to see the role of stress as a risk factor for stroke and that may help to take appropriate measures, so that some people may be rescued from this catastrophe.

It is the objective of the present study to evaluate the relationships of stress to stroke. As the prevention of stroke is very difficult, identifications of factors contributing to stroke is very important. It is not claimed that stresses are the only factors implicated in strokes but they prove to be contributory.

Materials and Method

This was a prospective study. The study was carried out in Neurology Department of Sir Salimullah Medical College, Bangabandhu Sheikh Mujib Medical University, Dhaka Medical College and BIRDEM, Dhaka and the study was carried out from July 2003 to June 2005. A total number of 472 consecutive, diagnosed patients were selected for the study and a same number of age and sex matched persons who have one or more than one risk factors for stroke were taken as control. Informed consents were taken from each patient and control subjects before his/her entry into the study. Detailed medical history and thorough physical examination were carried out.

Routine blood examination including platelet count, WBC count, RBC count, Haemoglobin, blood sugar, urine RME, lipid profile, serum electrolytes, renal function, ECG, Echocardiogram, Carotid doppler if needed, LFT, VDRL, bleeding time & clotting time if needed were done. CT scan of head and X-ray chest PA view were done in all cases. EEG and CSF study were done to exclude some disease. The above investigations were done from the pathology, biochemistry and Radiology & Imaging department of respective institution.

All relevant information from history, clinical findings and investigation results were recorded in predesigned questionnaire & data sheet.

In this study we like to define stress as a particular relationship between the person and the environment that is appraised by the person as taxing or exceeding his or her resources and danger his or her welbeing.

In general, the patients themselves were too ill, often showing markedly depressed levels of conciousness, for interview. Usually, the stroke patients who can talk or two or more close relatives were interviewed, of whom at least one was living with the patient. Sometimes it was necessary to gain further details from friends or work-mates. The interviews were taken according to a structured questionnaire about the life events and difficulties experienced in the last 12 weeks before stroke. By convention the life "events" are episodic in nature: acute illnesses, accidents, court appearances, deaths of family members, etc, while "difficulties" have persisted for more than four weeks, for example, chronic ill health, protracted marrital friction. Both events and difficulties were classified according to their content, for example, as health events, marrital difficulties. We took 12 weeks because it is very difficult to remember all the stressful events and difficulties long before that period.

We used the events of Social Readjustment Rating Scale (SRRS) as the stressors. These were death of the spouses, divorce, marrital separation, jail term, death of close family member, personal injury or illness, marriage, fired at work, marrital reconciliation, retirement, change in health of family member; pregnancy, sex difficulties, gain of new family member, business readjustment, change in financial state, death of close friend, change to different line of work, change in number of arguments with spouse, mortgage over Tk. 10,000, foreclosure of mortgage or loan, change in responsibilities at work, son or daughter leaving home, trouble with in-laws, outstanding personal achievement, wife begins or stops work, begin or end school, change in living conditions, revision of personal habits, trouble with boss, change in work hours or conditions, change in residence, change in schools, change in recreation, change in church activities, change in social activities, mortgage or loan less than Tk. 10,000, change in sleeping habits, change in number of family get-togethers, change in eating habits, vacation, Eid Festivals, minor violations of the law.

But we did not scored them. Because through this study we want to find out whether the stress has any relation to stroke or not. If we get any positive relation i.e. if stress is a risk factor for stroke then we will score them in the next study to find out the relationship of severity of stress to stroke. Events and difficulties involving the physical health of the subject were excluded from the study.

Appropriate statstical analyses like mean, standard deviation, standard error and Chi square test were done. Odds ratios and confidence intervals were calculated according to the method suggested by Morris & Gardner[5].

Results

Four hundred seventy two patients were evaluated. After clinical assessment, all patients were investigated routinely. Then all the patients were screened for the presence of stress. Same number of healthy age & sex-matched volunteers were selected as control subjects. In this study, the mean age of the patients was 59.6 years with SD ± 13.2 years and the age range was between 45 to 76 years. Sex distribution of patients showed that male female ratio was 3:2. Incidence among male was higher than that of female. But there

was some difference of incidence of stroke before 65 years and after 65 years. Up 65 years, males incidence was 36% higher and after 65 years male incidence was 25% higher. On average male incidence is 33% higher than that of female. CT scan was done only in stroke patients but not in controls. Three hundred ninty six (83.89%) had infarction, 24 (10.18%) had intracerebral haemorrhage and 14 (5.93%) had SAH. The potential risk factors for stroke were investigated.

Stress was found in 326 patients and 218 controls. Statistical analysis showed that Odds ratio was 2.6 with 95% CI 1.78 to 8.09 and x^2 was 25.3 which corresponds to p <0001. It was found among 298 infarcted patients and 218 controls. Statistical analysis showed that Odds ratio was 3.54 with 95% CI 2.34 to 5.34 and x^2 was 37.73 which corresponds to p <0001. Again stress was found among 18 patients with cerebral haemorrhage and 109 controls. Statistical analysis showed that Odds ratio was 0.69 with 95% CI 0.29 - 1.66 and x^2 was 0.33 which corresponds to p> 0.5. Stress was found among 5 patients with subarachnoid haemorrhage and 109 controls. Statistical analysis showed that Odds ratio was 0.64 with 95% CI 0.21 - 2.00 and x^2 was 0.23 which corresponds to$p > 0.5$.

Table - I shows the age distribution of the cases and controls. Maximum {312(66%)} are in the age range of 55 to 69. The incidence increases by double in each decade.

Table - I

Age distribution (n = 472)

Age in Years	Cases		Controls	
	No. (%)	Mean ± SD	No (%)	Mean ± SD
45-49	28 (5.93)	59.6 ± 13.2	32(6.77)	59.1 ± 12.96
50-54	38(8.05)		36(7.62)	
55-59	70(14.83)		74(15.67)	
60-64	86(18.22)		84(17.79)	
65-69	156(33.05)		158(33.47)	
≥ 70	92(19.91)		88(18.64)	

Table-II

Sex distribution by age (n=472)

Age	Male No. (%)	Female No.(%)
45-49	16(3.38)	12(2.54)
50-54	22(4.66)	16(3.38)
55-59	40(8.47)	30(6.35)
60-64	50(10.59)	36(7.62)
65-69	88(18.64)	68(14.40)
≥70	54(11.44)	40(8.47)
Total	270(57.2)	202(42.8)

Table - II shows the sex distribution by age. Male female ratio is 3:2. Male involvement is 33% higher but it varies with age. Before 65 years, male to female ratio is 1.36:1,i.e. male is 36% higher and above 65 years male to female ratio is 1.25 : 1, i.e. male is 25% higher.

Table-III

CT brain findings of patients (n=472)

Type of lesion	Number	%
Infarction	396	83.89
Hemorrhage	48	10.18
SAH	28	5.93
Total	472	100

Table-III shows the findings of CT scan of brain of the cases. Infarction constituted 396 (83.89%), cerebral haemorrhage 48 (10.18%) and SAH 28 (5.93).

Table-IV

Stress as a risk factor for stroke.

	Stress	
	+ve	- ve
Stroke	326	146
No stroke	218	252

OR= 2.6 95% CI= 1.78-8.09
X^2=25.3 p<.001

Table-IV shows the distribution of stress among cases (stroke group) and controls (no stroke group). OR and X^2 test prove that stress is a significant risk factor for stroke.

Table-V

Stress as a risk for infarction

	Stress	
	+ve	-ve
Infarction	298	98
No infarction	218	254

OR = 3.54 95% CI = 2.34-5.34
X^2 = 37.73 p<0.001

Table-V shows the distribution of stress among infarcted patients and controlls. OR and X^2 test prove that stress is a risk factor for infarction.

Table-VI

Stress as a risk factor for cerebral haemorrhage

	Stress	
	+ve	- ve
Hemorrhage	18	30
No hemorrhage	218	254

OR= 0.69 95% CI= 0.29 – 1.66
X^2=0.33 p<.0.5
with Yates correction

Table-VI shows the distribution of stress among cerebral haemorrhage group and control group. OR and X^2 test after yates correction proves that stress is not a risk factor for intracerebral haemorrhage.

Table- VII

Stress as a risk factor for subarachnoid haemorrhage (SAH)

	Stress	
	+ve	- ve
SAH	10	18
No SAH	218	254

OR= 0.64 95% CI= 0.21 – 2.00
X^2= .23 p<.0.5
with Yates correction not significant

Table –VIII

Stroke Types

Infarction	Hemorrhage	SAH	All strokes
OR & 95% CI	OR & 95% CI	OR & 95% CI	OR & 95% CI
3.54(2.34-5.34)	0.69(0.29-1.6)	0.64(0.21-2)	2.6(1.78-8.09)

Table-VII shows the distribution of stress among patients with SAH and controls. OR and X^2 with Yates correction prove that stress is not a risk factor for SAH.

Table-VIII shows the OR & CI of different types of strokes. It indicates that stress is a significant risk factor for infarct but not for intra cerebral haemorrhage/haemorrhage or SAH.

Discussion

This study was carried out to determine whether stress is a risk factor for stroke. The study subjects were taken from the department of Neurology (indoor and outdoor) of Sir Salimullah Medical College, Bangabandhu Sheikh Mujib Medical University, Dhaka Medical College and BIRDEM, Dhaka. During the study period, i.e. from July 2003 to June 2005, 472 patients, who had stroke, were evaluated. Also 472 age and sex matched healthy volunteers were taken as control.

Clinical examinations and laboratory investigations were done in all cases. Majority of the subjects were of age between 55 to 69 years. Bell studied 51 patients with cerebrovascular disease and the age range of the patients were 17 to 84 years, with majority of them between 50 to 69 years[6]. Rolf Adler studied 32 patients with stroke and twenty four of them were in the age range of 50 to 69 years[7]. SMZ Chowdhury studied 100 CVD patients. He found that the age range was 18 to 84. Most of his cases were between 50 to 70 years[8]. SM Arif also found that peak incidence was between 5th to 7th decade[9]. Our finding coincides with the results of other studies[10-14].

In this study the mean age$\pm$SD was 59.6$\pm$13.2 years. Control subjects were matched by age and sex. So, they also has the above age range and mean. Similar past studies had comparable age statistics of patients[6,15]. For example, Adams studied a sample with average age of 58$\pm$12 years and that of Bell was 59$\pm$11.2 years.

In this study it was seen that the frequency of stroke increases with increase of age. In the 5th decade, the incidence was 6% in the 6th decade it was 22% and in the 7th decade, the incidence was 51% i.e. the incidence increases by more than double in each decade. This finding corroborates with that of other studies where they showed that the incidence of stroke is strikingly related to age with a more than doubling of incidence rates in each successive decade for persons over 55 years of age [16-17].

In the present study, the male to female ratio was 3:2. Male involvement was 33% higher than that in female. But it varies with age. Before 65 years, male to female ratio was 1.36 : 1 i.e. male frequency was 36% higher and above 65 years male to female ratio was 1.25 : 1 i.e, male frequency was

25% higher. This finding correlates well with that of Wolf and Kurtzke, where they showed that the frequency of stroke was about 30% higher in men than women[16,18]. They also showed that the sex difference was slightly greater below age 65 years. In the previous study by Cull showed that men suffer with stroke more than women and it affects males 1.5 times more often than males[19]. Finding of this study also correlates with that of Chowdhury and Bhuiyan studied a group of patients with CVD where he found male & female ratio was 1:1 which is not similar to this study[20,21]. Alamgir showed different finding where M : F was 4 : I in contrast to this study where M : F was 1.5 : 1[10] . This difference is due to previous attitude of our society that the females are not brought to the hospital. This attitude is gradually being changed by the last 25 years; this was indirectly reflected in another Bangladeshi study[22].

CT head findings of the studied patients showed that 83.89% had infarction, 10.18% had intracerebral haemorrhage and 5.93% had sub-arachnoid haemorrhage. This finding is almost similar to Martin M Brown (1992) where he found cerebral infarction 85%, intracerebral haemorrhage 10% and subarachnoid haemorrhage 5% [23]. The present findings are quite keeping with other foreign studies and that of our country study[24].

Earlier studies were done in the '50s & '60s. Ecker in 1954 reported a series of 20 cases of stroke possibly associated with stress [25]. Additional studies have shown an association between stress and stroke, but evidence for such a link remains limited[26-30]. Carasso suggested an association between the type A personality and more severe stroke in patients with no prior history of cardiac disease[27]. Spence recently reported that individuals with altered cardiovascular measures in the context of a mentally stressful laboratory designed task were also at increased risk for progression of carotid atherosclerosis[31] .

A specific relationship has been reported between acute stressful events and stroke[32-36]. For example, during the 1991 Persian Gulf crisis, there was an increase in overall mortality from cardiac disease and stroke in Israel[36]. An association may exist for CVD and chronic life stress. A case control study of the Oxfordshire Community Stroke project noted that severe stressful life events in the year prior to stroke were more common in stroke patients than in control [37] . Severe external life stresses may increase stroke risk even many years after the source of stress is eliminated[38].

Two large population studies also suggest that perceptions and adaptations to external life stresses may be associated with increased stroke risk. One of those studies involved middle aged men in Goteborg, Sweden. An association of increased stroke risk with a perception of long-standing severe psychological stress was found[39]. In this study, there was clear correlation between stress and infarction but no clear association between long standing stress and either subarachnoid haemorrhage or intracerebral haemorrhage.

A second study, by Schneck described a potential relationship between increased stroke risk and difficulties coping with stress at work [40]. This retrospective case-control study also suggested that the risk was cumulative so that persons with greater

perceptions of chronic work stress had a larger stroke risk. Interestingly, the study by Schneck as well as other reports suggest that persons with a good social network or access to support structures have a decreased stroke risk or a better outcome, which possibly reflects a better adaptive response to stressful life events [41,42].

Recently Schneck performed a pilot feasibility study for an investigation of stress in African American patients with ischaemic stroke and controls (unpublished data). The data suggested that stroke cases may have had more stressful experiences is the year preceding stroke onset[43]

Harmsem showed that severe psychological stress indicated a significant risk for cerebral infarction, even after adjustment for other significant risk factors. This has not previously been reported[39].

Wihelmsen showed that psychological stress is a risk factor for stroke[44]. In multivariate analysis the relative risk was 1.7. In another study by Carasso R clearly indicated that both life events and personality type can influence the nature of CVD[27].

Lawrence performed a study among prisoners of war. He found that among the former prisoners of wars 9.3% reported a stroke compared to 1.2% for other World War II veterans. This result indicates a strong association between stress and stroke. The high relative risk indicates that discrete, severe stresses, even many years later, may be among the strongest risk factors for stroke[45].

Conclusion

Although the belief that stress causes stroke is common among the general public, most medical accounts of the risk factors for stroke either do not mention emotional stress or refer to it in passing as an intriguing but unsubstantiated possibility. Psychological stress is indeed a risk factor for CVD, then future studies of stress and stroke may focus on interventions such as behavioral modification of stress perceptions. As may be the case for coronary heart disease, these interventions might be effective for either primary or secondary stroke prevention. Because of paucity of information about an association between cerebrovascular disease and stressful life events, perceived stress, or abnormal stress responses, additional research is needed. Also, the physiological mechanisms through which psychological stress may cause an increased stroke risk remain to be elucidated.

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ORIGINAL ARTICLES

EEG changes in Hepatic and Renal Encephalopathy

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Abstract:

Objective: To demonstrate EEG changes in hepatic and renal encephalopathy and also to find out the diagnostic value & correlation between degree of encephalopathy.

Methods and Materials: This prospective case control study was carried out in the department of Neurology of Bangabandhu Sheikh Mujib Medical University (BSMMU) &Dhaka Medical College Hospital, Dhaka Bangladesh from January 2003 to July 2004. Sixty cases of Hepatic and renal encephalopathy (30 cases each) and 30 control subjects of same age and sex were included in the study.

Results: EEG showed abnormality in all 30 cases(100%) of hepatic and 27(90%) cases of renal encephalopathy respectively. Highest percentage of patients had focal slow waves followed by generalized slow waves, sharp and slow waves, occasional generalized slow waves, spike and waves; and triphasic waves were higher in hepatic encephalopathy (20%) compared to renal encephalopathy(3.3%), where as sharp and slow waves were higher in renal encephalopathy (20%) than hepatic encephalopathy(3.3 %).

Conclusion: EEG is a sensitive test for the diagnosis of hepatic and renal encephalopathy. Diagnostic importance of EEG for metabolic encephalopathy is much more than that of CT scan and CSF study.

Key words: EEG, encephalopathy.

Introduction:

Encephalopathy means disease of the brain usually not by any evident infection e.g there are no stigma of infection in brain & cerebrospinal fluid(CSF)study is usually normal[1]. Encephalopathy results from varied etiology are not uncommon in our country. Among them toxic encephalopathy is in top of the list. In a third world country like Bangladesh low socioeconomic condition, inadequate vaccination coverage, lack of proper health education and above all malnutrition contribute largely in the genesis of encephalopathy. Superimposed to the illiteracy & lack of awareness magnifies these problems by many folds. In advanced countries, these factors are well controlled so low profile of encephalopathy is prevailing there[1].

Encephalopathy involves the brain and interfere the functions of neurons. Neuronal activities are manifested by electrical discharge from the neurons and these electrical activities can be graphically recorded in a noninvasive way by EEG

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machine. Encephalopathy does not correlate with the degree of metabolic abnormality. Marked biochemical alteration may not produce encephalopathy, whereas mild to moderate changes in biochemical parameters may be associated with severe grades of encephalopathy. On the contrary EEG is sensitive in detecting encephalopathy even in subclinical cases. That is why this study has been designed to see the EEG changes in different grades of two commonly encountered toxic encephalopathy-hepatic and renal.

Aims and Objectives:

1. To demonstrate EEG changes and to find out diagnostic value of EEG in relation to toxic encephalopathy.
2. To demonstrate any correlation between degree of encephalopathy and EEG changes.
3. To demonstrate importance of EEG over the other diagnostic procedures like CT scan and CSF study.

Materials and Methods:

This was a prospective case control study carried out in BSMMU and Dhaka medical College Hospital, Dhaka, Bangladesh from January 2003 to June 2004.Both the Hospitals have independent neurology unit and have laboratory facilities for electrophysiological study.Thirty cases of hepatic and renal encephalopathy each and 30 control subjects matched with age and sex were included in the study. To avoid any age related physiological variation in EEG tracing patients below the age of 20 and above 60 years were excluded from the study. Any patient whose clinical features or biochemical reports were suggestive of encephalopathy due to other causes (hypoglycemia, hyperglycemia, electrolytes imbalance, endocrine disorder etc.)and cases of psychiatric illness, encephalitis or any other chronic disease were also excluded from selection.

A questionnaire incorporating history, physical examination, investigation etc. were prepared and properly filled up by the author himself. In all cases and control subjects EEG were done following standard procedure by expert EEG technician using 21 channel EEG machine. All the tracings of both study and control groups were reported by the renowned neurologist who have got profound knowledge about EEG tracing. Finally all the results were correlated with the clinical diagnosis and grading of the encephalopathy.

Besides EEG tracing other relevant investigations were also done like blood sugar, blood urea, serum creatinine, serum electrolytes and liver function test, either to establish the diagnosis of encephalopathy or to exclude differential diagnosis. They also helped to correlate the severity of encephalopathy and the findings of EEG tracing.

Results of the study subjects were compared with that of the controls and ultimate data were noted in the result sheet. Association between two variables were tested by x^2(chi square) after Yates correction. Pearson correlation coefficients were also performed in necessary cases. Proportion test was done to find out whether the diagnostic value of EEG was superior to CSF study and CT scan.

Observation and results:

Table I shows the age distribution of the study patients and control subjects. The mean age of the cases was 36.8±10.5 years and the control was 36.5±9.2 years. Analysis found no statistically significant mean age difference between the groups (p>0.05).

Table II shows that the mean age of the hepatic encephalopathic patients were a little bit higher (37.5 ±11.0) than renal encephalopathy patients (36.2 ±1.1). However analysis found no statistically significant mean age difference between hepatic and renal encephalopathic patients (p>0.05).

Table I

Age distribution of patients and control subjects

Age in years	Cases (n=60)		Controls (n=30)		Total (n=90)		P value
	No	%	No	%	No	%	
21-30	20	33.3	8	26.7	28	31.1	
31-40	21	35.0	14	46.7	35	38.9	
41-50	10	16.7	5	16.7	15	16.7	
51-60	9	15.0	3	10.0	12	13.3	
Total	60	100.0	30	100.0	90	100.0	
Mean ± SD(Years)	36.8± 10.5		36.5 ± 9.2		36.7 ± 10.0		0.894

Table II

Age distribution of cases

Age in years	Cases				P value
	Hepatic		Renal		
	Number	%	Number	%	
21-30	10	33.3	10	33.3	
31-40	9	30.0	12	40.0	
41-50	6	20.0	4	13.3	
51-60	5	16.7	4	13.3	
Total	30	100.0	30	100.0	
Mean ± SD (years)		37.5 ± 11.0		36.2 ± 1.1	0.635

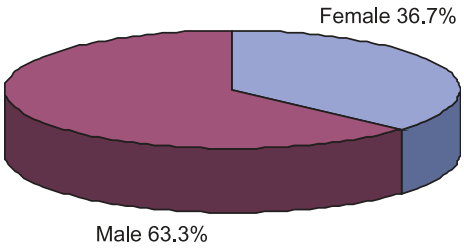

Fig.-1 :

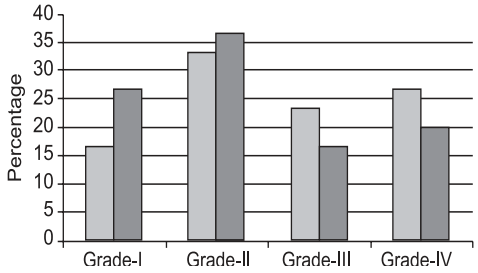

Fig.-2

Out of 90 study subjects 63.3%were male and 36.7% were female. Males were more commonly represented (1.73:1). Both in case and control equal proportion of males and female patients were selected.

Figure II showed that in both hepatic and renal encephalopathy highest percentage of cases were in grade II-33.3% and 36.7% respectively. Analysis found no statistically significant difference of grading between hepatic and renal encephalopathy (p>0.05).

Table III

Distribution of study subjects according to their clinical presentation

Symptoms	Study subjects				Total(n=60)		P value
	Hepatic(n=30)		Renal (n=30)				
	No	%	No	%	No	%	
Disorientation	23	76.7	21	70.0	44	73.3	0.559
Slurred speech	22	73.3	20	66.7	42	70.0	0.573
Drowsiness	22	73.3	14	46.7	36	60.0	0.035
Incoherent talk	14	46.7	12	40.0	26	43.3	0.602
Coma	10	33.3	4	13.3	14	23.3	0.067
Stupor	7	23.3	4	13.3	11	18.3	0.316
Myoclonic jerk	2	6.7	8	26.7	10	16.7	0.037
Seizure	1	3.3	6	20.0	7	11.7	0.107
Muscle twitching	1	3.3	5	16.7	6	10.0	0.197

Table III showed that highest percentage of patients presented with disorientation (73.3%) followed by slurred speech (70%) and drowsiness(60.0%).Analysis of clinical presentation revealed that no statistically significant difference was found between hepatic and renal encephalopathy($p>0.05$) except drowsiness and myoclonic jerks ($p<0.05$). Drowsiness was significantly high in hepatic encephalopathy (73.3%), whereas myoclonic jerks were significantly high in renal encephalopathy (26.7%) than hepatic encephalopathy.

EEG wave pattern showed (Table IV) statistically no significant difference between hepatic and renal encephalopathy ($p>0.05$).However triphasic waves were higher in hepatic encephalopathy (20%) compared to renal encephalopathy (3.3%) whereas sharp and slow waves were higher in renal encephalopathy than hepatic encephalopathy.

In grade IV encephalopathy 100% of the patients showed EEG changes both in hepatic and renal encephalopathy and the wave patterns were generalized slow waves. In grade III encephalopathy wave patterns were focal, occasionally generalized slow waves and triphasic slow waves. In grade II encephalopathy focal slow waves were equally prevalent in both hepatic and renal encephalo-pathies. In grade I encephalo-pathy 10% cases showed normal tracing in renal encephalopathy where all the tracing of hepatic encephalopathy were abnormal. Sharp and slow waves were common in renal encephalopathy (20%) than in hepatic encephalopathy (3.3%).

Table IV

Distribution of study subjects according to their wave patterns

Type of waves	Study subjects				P value
	Hepatic (n±30)		Renal (n=30)		
	No	%	No	%	
Focal slow waves	10	33.3	8	26.7	0.573
Generalized slow waves	7	23.3	6	20.0	0.754
Sharp and slow waves	1	3.3	6	20.0	0.107
Occasionally generalized slow waves	5	16.7	4	13.3	1.000
Spike waves	1	3.3	2	6.6	1.000
Triphasic waves	6	20.0	1	3.3	0.107
Low voltage tracing	0	0.0	0	0.0	-
Periodic slow waves	0	0.0	0	0.0	-
Normal waves	0	0.0	3	10.0	0.236

Table-V

Comparison of diagnostic values of EEG, CSF& CT scan study of the brain in hepatic and renal encephalopathies.

Encephal Opathy	EEG	Normal	Abnormal	% of Abnormality	CSF study	Normal	Abnormal	% of Abnormality	CT scan of brain	Normal	Abnormality	% of Abnor
Hepatic	Done in 30 cases	0	30	100 %	Done in 12 cases	12	0	0%	one in 11 cases	11	0	0%
Renal	Done in 30 cases	3	27	90%	Done in 9 cases	9	0	0%	Done In 13	13	0	0%

In hepatic encephalopathy EEG were abnormal in all 30 cases (100%). CSF study and CT scan of Brain were done in 12 &11 cases respectively and were found normal (Table-V). In renal encephalopathy EEG were abnormal in 27 cases (90%). CSF study and CT scan of brain were done 9 & 13 cases respectively and were also found normal(Table V)

Discussion:

Present study was designed to evaluate the EEG changes in two commonly encountered toxic encephalopathy-hepatic and renal. In this study EEG was abnormal in 95% of the encephalopathy patients. Five percent patients however showed normal tracing, this is inconsistent with the study of Fish BJ et al[2]. Former study done by Mohammad QD[3] observed 80% abnormality in his series and 20% cases were recorded as normal. This high rate of normal tracing may be due to the facts that he used 8 and 16 channel EEG machine where the electrodes were placed at a larger gap keeping more area of the brain uncovered as compared to 21 channels EEG machine. Five percent normal recording may be explained by the fact that deeper neuronal discharge could not be recorded by superficially placed scalp electrodes[2].

EEG wave patterns in this series were focal slow waves(Hepatic-33.3%, renal-26.7%), Generalized slow waves (Hepatic-23.3%, renal- 20.0%), occasionally generalized slow waves (Hepatic-16.7%, renal -13.3%), Triphasic waves (Hepatic-20.0%, renal-3.3%), Sharp and slow waves (Hepatic-3.3%, renal-20.0%), Spike waves (Hepatic-

3.3%, renal- 6.6%). These types of wave patterns were also seen by Mohammad QD[3]. In this series triphasic waves were more prevalent in hepatic encephalopathy than in renal encephalopathy. These findings were consistent with the findings of Fisher GG[4]. On the other hand, sharp and slow waves were more common in renal encephalopathy (20.0%) than in hepatic encephalopathy (3.3%). Similar abnormalities were noted by Fisher GG[4].

EEG have got definite relationship with the degree of encephalopathy[5]. In grade I encephalopathy focal slow waves (FSW), and sharp and slow waves (SSW) were noted. Occasionally generalized slow waves (OGSW) and focal slow waves (FSW) and triphasic waves were seen in Grade II and Grade III encephalopathy. Similarly Fish BJ et al[2] found preponderance of triphasic waves in Grade II and Grade III encephalopathy. In Grade IV wave patterns observed were generalized slow waves.

Similar changes in waves of different grades of encephalopathy found in different studies[6,7].

EEG may be the most useful of the commonly used laboratory diagnostic test in the evaluation of the encephalopathy patients[6]. In this study EEG were abnormal in 100% cases of hepatic encephalopathy and in 90% of renal encephalopathy patients, whereas no abnormality was detected by CT scan and CSF study in 24 cases(Hepatic 11, renal 13) and 21 cases (Hepatic 12, renal 9) respectively. This finding is consistent with the finding of Mohammad QD who found no abnormality in CT scan and CSF studies in his study[3]. So it was observed that EEG had greater diagnostic value than both CT scan (P<0) and CSF study (P<001) for the dignosis of metabolic encephalopathies. Similar result was also shown by Lockwood AH et al[8]. Ustanday Y et al[9] also fail to show any change in CT scan in encephalopathies but however he found signal intensity changes by MRI in his study.

In both study and control groups male and female ratio was 1.73:1 and 2:1 respectively which means that the disease has got more predilections for males. In the study of Mohammad QD et al[3] male and female ratio was 1.7:1; other studies in advanced countries showed much closer ratio. This higher male incidence in our country may be because of males are admitted in the hospital more commonly than females. Females are comparatively less cared and usually they donot get admitted into hospital because of socioeconomic and religious ground.

In the present study common clinical features that were noted are- disorientation (73.3%), slurred speech (70.0%), drowsiness (60.0%), and incoherent talk (43.3%). Next common clinical features were coma (23.3%), stupor (18.3%) and myoclonic jerks (16.7%). Least common were muscle twitching (10.0%) and seizure (11.7%). Mohammad QD et al[3] in his study also found similar clinical presentations.

Conclusion:

EEG is a sensitive test for the diagnosis of encephalopathy. Although EEG changes are mostly nonspecific but in toxic encephalopathy specific waves may be observed. Abnormal wave patterns are directly related with the degree of encephalopathy. Diagnostic importance of EEG in the context of encephalopathy is

much more than that of CT scan and CSF study. So it is recommended to take the advantage of this noninvasive, less costly procedure in the diagnostic evaluation of two commonly encountered toxic encephalopathy-hepatic and renal.

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Effect of Irregular Use of Antihypertensive Therapy on Stroke

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Abstract

Stroke is the third commonest cause of death in the world. Out of them 85% suffer from ischemic stroke and 15% percent suffer from hemorrhagic stroke. It is found that in both types of stroke hypertension is the most important risk factor and regular use of antihypertensive drugs is very useful to prevent stroke. This prospective study had been carried out among 500 admitted and outdoor stroke patients in Dhaka National Medical College and Hospital (DNMCH), Dhaka, Bangladesh during the period of September 2002 to March 2005. Preset proforma was used showing patient's age, sex, presence or absence of hypertension, regular or irregular use of antihypertensive drugs and finally the patients were categorized by findings of CT scan of brain. A total of 300 (60%) patients were male; and 200 (40%) were female; 365 (73%) of them were hypertensive but out of these hypertensive patients 245 (67.12%) were on regular antihypertensive drugs and 120 (27%) were on irregular drug. Among the patients on regular drugs 210 (85.7%) had ischemic stroke and 35 (14.2%) had hemorrhagic stroke. It is to be mentioned here again that the number of patients having irregular drug is 120; out of which 70 (58.3%) had ischemic stroke and 50 (41.6%) had hemorrhagic stroke. The objective of the study was to estimate the proportions of stroke patients using regular or irregular antihypertensive drugs and thereby giving emphasis to the use of regular antihypertensive drugs by the patients. It may be concluded that irregular antihypertensive users are more affected by hemorrhagic stroke than regular antihypertensive users.

Introduction

Stroke is one of the leading cause of death and disability in the world, costing billions of dollar annually as direct medical expenditure as well as indirect loss in productivity. Eighty five percent patients suffer from ischemic stroke and fifteen percent patients suffer from hemorrhagic stroke. Ischemic stroke leads to death of brain tissue. So substantial damage may result in death or permanent disability. On the other hand hemorrhagic stroke leads to displacement of brain tissue by haematoma and if vital centers are compressed it may lead to death. Age is the strongest determinant of stroke[1]. Stroke in people aged 75-84 years is 25 times more common than in people aged 45-54 years.

Hypertension is strongly associated with risk of stroke[1-3]. Although most informations relate diastolic blood pressure, but the risk associated with systolic blood pressure is similar and

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possibly stronger and even isolated systolic hypertension is associated with increased risk[4,5,6,7]. Diastolic blood pressure and stroke is log liner, with no evidence of threshold below which the risk becomes stable. Stroke incidence is 1.25 times greater in men. But the percentage difference in stroke risk associated with a given difference in blood pressure is similar in male and female at all levels of blood pressure and doubles with each 7.5 mm of Hg rise in diastolic blood pressure in Western population and 5mm of Hg in Japanese and Chinese populations[8, 9]. The incidence of stroke with lower blood pressure is associated with vascular disease and treated hypertension. Mild hypertension is of greatest risk due to its prevalence greater than any other risk factor. The association between increased blood pressure and stroke is less in the elderly than in the middle age[10]. It is not clear whether hypertension is a risk factor for very elderly, where stroke may be associated with low pressure which may be due to pre-existing cardiovascular or other diseases[11]. Hypertension probably increases risk of stroke by increasing extent and severity of atheroma and the prevalence of small vessel disease in the perforating arteries within the brain[11-16].

The effect of risk factors on stroke is usually additive and multiplicative. So the presence of several risk factors put the individual at higher risk.

The aims of the study was to find out the proportion of male and female affected by stroke in the society. The percentage of hypertensive and nonhypertensive patients among stroke cases were identified and also to find out whether there is any role of regular and irregular antihypertensive therapy on the pattern of stroke.

Materials and Methods

This prospective study was conducted at DNMCH on 500 stroke patients attending at out-patient & in-patient department of Medicine. A structured questionnaire was used for data collection. Preset questionnaire was used for each patient composed of age, sex, presence or absence of hypertension, regular or irregular use of antihypertensive drugs and CT scan findings of brain.

Analysis was done by chi-squared test. Where $x^2 = 33.79$ at $P = .001$ level, the result was highly significant.

Results

Among the 500 patients, 300 (60%) were male and 200 (40%) were female, seventy three percent of them were hypertensive and 27% normotensive. Among the hypertensive patients 67.2 % were on regular antihypertensive therapy and 32.8% were irregular. The patients on regular drug suffered from 85.7% ischemic and 14.3% hemorrhagic stroke and the other patients on irregular drug had 58.4% ischemic and 41.6% hemorrhagic stroke. Nonhypertensive persons suffered from 77.7% ischemic stroke and 22.3% hemorrhagic stroke. Male patients suffered from ischemic stroke in 90% cases and hemorrhagic stroke in 10% cases. Most of the Females i.e. 83.33 % suffered from ischemic and 16.66 % hemorrhagic stroke.

Table-I

Sociodemographic characteristics of the patients (n = 500)

Age in years	Number	Percent (%)
<50	80	16
50 – 90	420	84
Sex Male	300	60
Female	200	40

In table I among 500 patients majority (420, 84%) were between 50 – 95 years & only 80 (16%) were below 50 years of age; males were 300 in number (60%) & 200 (40%) were females.

Table-II

Distribution of Hypertension among the patients (n = 500)

Hypertension	Frequency	Percentage
Yes	365	73
No	135	27
Total	500	100

In table-II hypertension was detected among 365 (75%) patients & rest 135 (27%) were normotensive.

Table-III

Pattern of use of antihypertensive drugs (n = 365)

Use of antihyper tensive drugs	Frequency	Percentage
Regular	245	67
Irregular	120	33
Total	365	100

In table III all hypertensive patients used antihypertensive drugs but out of them 67% (245) used regularly and 33% (120) used irregularly.

In table IV among 365 hypertensive patients majority 280 (76.7%) & only 85 (23.3%) had ischemic & hemorrhagic strokes respectively. Among 245 regular user of antihypertensive drugs 210 (85.7%) had ischemic stroke. Among the irregular user of antihypertensive drugs (120), 70 (58.3%) & 50 (41.7%) were suffered from ischemic & hemorrhagic stroke respectively.

Table-IV

Pattern of stroke among the hypertensive patients (n = 365)

Pattern of use of antihypertensive drugs	Ischemic stroke	Hemorrhagic stroke	Total
Regular	210 (85.7%)	35 (14.3%)	245 (100%)
Irregular	70 (58.3%)	50 (41.7%)	120 (100%)
Total	280 (76.7%)	85 (23.3%)	365 (100%)

Discussion

The vast majority of stroke patients have well recognized risk factors of vascular disease, mostly known before the onset of stroke [4]. This suggests it should be possible to make a considerable impact on stroke incidence by reducing the prevalence of causal risk factors in the population and by screening and case finding for high risk individuals to whom preventive treatment may be offered.

The cooperative studies of Veterans Administration and more recent report by Collins and associates after 14 randomized trial of antihypertensive drugs demonstrated that long term control of hypertension decreases the incidence of both athero-thrombotic infarction and intracerebral hemorrhage[15].

Treatment of hypertension reduces stroke risk[9,15]. Blood pressure treatment resulting in fall of systolic blood pressure by 10 to 12 mm of Hg and diastolic BP by 5 to 6 mm of Hg is associated with 38% reduction of stroke[16]. Treatment of isolated systolic hypertension in the elderly is also effective in reducing stroke risk and showed 36% reduction in both fatal and nonfatal stroke over 5 years in aged 60 years and above. It also reduces progression of carotid stenosis.

The overall mortality for hemorrhagic stroke is 25% to 60%. Due to improved detection and treatment of hypertension the incidence of intracerebral Hemorrhage has been reduced since 1980. Broderick et al in 1993 studied 188 patients with primary intracerebral Hemorrhage and determined the cause as hypertension in 72% cases[17].

In summary of seventeen treatment trials of hypertension throughout the world involving nearly fifty thousand patients, there was 38% reduction in all strokes and 40% reduction in fatal strokes favouring systematic treatment of hypertension. This effect was true in both white and blacks and in all ages[18].

In a study of 331 consecutive cases risk of intracerebral hemorrhage among hypertensive patients included discontinuation of antihypertensive therapy.

Age is the strongest risk factor for both hemorrhagic and ischamic stroke, which can not be modified. In this study 73% patients were hypertensive and 27% normotensive. About 67% of the hypertensive patients were on regular antihypertensive therapy and 33% were irregular. The patients on regular drug suffered from 85.7% ischemic and 14.3% hemorrhagic stroke and the other patients on irregular drugs had 58.4% ischemic and 41.6% hemorrhagic stroke. In this study the persons having regular antihypertensives had less number of hemorrhagic stroke (14.3%) and the result was 10.83 at P = .001 level which is greater than table value hence the result is highly significant (P < .001). On the other hand irregular antihypertensive users developed 41.7 % hemorrhagic stroke.

In a study of 331 consecutive hospital cases of primary ICH verified by computed tomography or autopsy, occurring during the period of 1990 through 1992, and 331 age and sex-matched community- based control subjects in a city wide study involving 13 hospitals. Hypertension approximately doubled the risk of ICH (adjusted odds ratio [OR] = 2.45; 95% confidence interval [CI]: 1.61 to 3.73). The OR associated with hypertension was significantly greater among those who had ceased taking medications, supervised and unsupervised (OR = 1.95 ; 95% CI: 2.25 to 11.02), compared with those who had not (OR = 1.95; 95% CI : 1.20 to 3.16), were under the age of 55 years (OR = 7.68; 95% CI: 2.65 to 22.5). However, those dying from ICH displayed a greater risk of ICH due to hypertension than survivors, with the ratio of the two ORs being 5.47 (95% CI: 1.23 to 24.44). These findings provide evidence for a greater increase in risk of ICH due to hypertension among younger persons and

those discontinuing antihypertensive therapy. This is the first direct evidence for a link between irregular uses of antihypertensive medication and risk of stroke; targeting these individuals for more intensive monitoring and education of the importance of risk factor modification may help to reduce the impact of this form of stroke[19].

Conclusion

Most patients have recognized vascular risk factors of stroke like hypertension, and taking regular antihypertensive drugs may have a positive impact on reduction of incidence of stroke. In this study it was tried to find out the importance of taking antihypertensive drugs regularly. The results may be biased as there are so many risk factors of stroke like diabetes, hyperlipidemia, smoking, structural and functional heart disease, vascular malformation and other hypercoagulable states which were not searched for. So, it may not be conclusive but positive impact of taking regular antihypertensive drug may be emphasized. It is well established that female patients are less susceptible to develop coronary disease before 50 years of age. This study also indicated the same i.e. 62.5% male and 37.5 % female were affected though the number of patients were only 16% of the total number, this information may have an additive value. This study also determined the male female ratio which is 1.5 times more in case of male. So, to have a conclusion, larger study involving more number of patients with maximum risk factor evaluation is necessary.

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Stroke Pattern in a Private Hospital and its Association with two Modifiable Risk Factors– Hypertension and Diabetes Mellitus

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Abstract

A retrospective descriptive study was carried out in a private hospital at New Eskaton Road, Dhaka, Bangladesh to find out the stroke pattern and its association with two modifiable risk factors, diabetes mellitus and hypertension. Data were collected from the discharge file of those admitted patients who were discharged during the period from July 2003 to December 2003. A total of 657 patients were included in the study. Results showed that male (63.6%) predominates over female (36.6%). And 69.9% of patients had hypertension and 25.9% of patients had diabetes mellitus. Most of patients were hemiplegic (78.4%) of which 42.5% had right sided hemiplegia and 21.6% had left sided hemiplegia. Infarction type of stroke (67.9 %) predominates over hemorrhagic types (32.1%). In conclusion the study has shown that stroke was more common in male and infarction was commoner in both sexes. Diabetes mellitus and hypertension were common accompaniment of stroke as modifiable risk factors. Hemiplegic strokes were commoner and predominantly affects dominant hemisphere.

Introduction

Stroke is a rapidly developing episode of focal and at times global loss of cerebral function with symptoms lasting more than 24 hours or the patient dies within 24 hours with no apparent cause other than those of vascular origin [1]. It is the third commonest cause of death in developed world after ischemic heart disease and cancer [2,3]. This is the important cause of hospital admission and long term disability in most industrialized population[4]. Diabetes mellitus was found to be the strongest risk factor for death from stroke in a study in Finnis population[5]. Smoking and systolic blood pressure was found to be independent risk factor among both sexes in the same study[5]. Numerous studies were carried out in the world to see the stroke pattern and its association with many modifiable risk factors. In Bangladesh such study is not adequate. The aim of this study was to see the stroke pattern and its association with two very common and established modifiable risk factors hypertension and diabetes mellitus, in Bangladesh perspective. The study was carried out in a neurology oriented private

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hospital in Dhaka where a considerable number of patients get admitted with stroke.

Materials and methods:

This retrospective study was carried out in a neurology oriented private hospital in Dhaka, Bangladesh. Data were collected from the discharged file of those admitted patients who were discharged during the period from July 2003 to December 2003. All the patients who were clinically diagnosed as stroke and were proved by CT scan, were included in the study. Discharged files of 657 patients were analyzed. Those who died during hospital stay were not included in the study. Non hemiplegics and hemiplegic patients were isolated to see the side predilection for stroke. Non hemiplegics includes either bilateral hemiplegia or monoplegia or simple facial palsy of upper motor neuron type or no gross neurological deficit. Right sided and left sided hemiplegics were also separated to see the dominant hemisphere involvement. None of the hemiplegics were recorded to be left handed that is right hemispheric dominance. History of hypertension and diabetes mellitus were recorded for analysis. As the data were collected from the discharged file of the patients, whether the patients were known diabetic or hypertensive or diagnosed as hypertensive or diabetic on admission could not be isolated. All the patients had essential hypertension but the type of hypertension whether isolated systolic or diastolic hypertension predominates the stroke population could not be separated. Diabetic patients were found to suffer from type 2 diabetes mellitus. Average duration of illness from hypertension or diabetes could not be determined because of inadequate information recorded in the file. Patients with all age groups of both sexes were included. Other modifiable and non-modifiable risk factors such as history of smoking, oral contraceptive pills, elevated lipid profile, family history, obesity, cardiac disease, atherosclerosis and many other minor risk factors were not analyzed as the records and investigations regarding these factors were inadequate and incomplete in many of the discharged files.

Statistical Methods: All the data were collected and compiled properly from the entire stroke patients. The data were analyzed statistically by using SPSS package for windows. Results were expressed in percentage and mean ± standard deviation.

Results:

In this series study population was 657. Out of these 63.6% were male and 36.4 % were female. Male female ratio was 1.74:1 (Fig. No-1). Mean age was 60.90±13 years. Most of the patients were between 61 to 70 years (Fig No-2). A good number of young adult with age ranging between 30 and 50 years (23.28 %) were suffering from stroke. Senile population with age ranging between 71 and 100 years constitutes third highest among stroke sufferers (20.29%). Hypertension was found to be the major modifiable risk factor and diabetes mellitus was the second commonest risk factor found. Most of the patients were hypertensive (69.9%) and 25.9% of patients were diabetic (Table No-I). Maximum (51.4%) patients had isolated hypertension with no accompanying diabetes mellitus and 18.41% patients had both hypertension and diabetes mellitus. Two major modifiable risk factor hypertension and diabetes mellitus were absent in only 5.32 % of patients.

We have isolated hemiplegics and non hemiplegics analyzed them to see the frequencies of involvement of dominant hemisphere. Most of the stroke patients were hemiplegic (78.4%). Non-hemiplegic (21.6%) group included either bilateral hemiplegia or monoplegia or isolated facial palsy of upper motor neuron type or no gross neurological deficit but had CT scan proved haemorrhagic or ischaemic lesion in the brain. Out of 657 patients majority had a predilection for right side (42.5%, Table No-II). In the right sided hemiplegic group 63.44 % were male and in the left sided hemiplegic group 62 71% were male. Side predilection for hemiplegia was almost equal in both sexes.

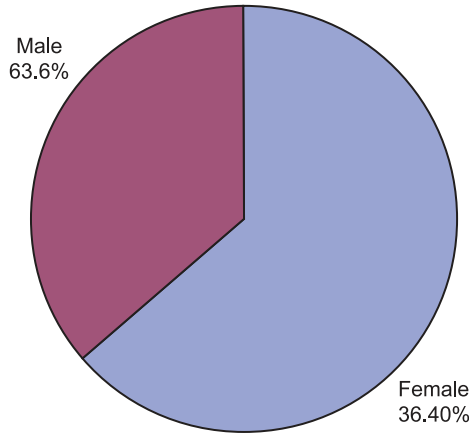

Fig.-1: *Distribution of sex of the study subjects (M=63.6%, Female=36.40%).*

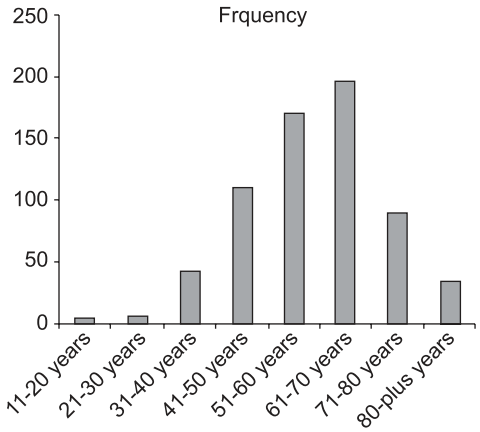

Fig.-2: *Distribution of age of the study subjects.*

Most of the patients in our study population as proved by CT scan were found to suffer from ischaemic infarction (67.9 %, Table No-3)). Rest of the patients who were suffering from haemorrhagic lesion included intracerebral haemorrhage and subarachnoid haemorrhage. Haemorrhagic groups were not isolated to classify different types of haemorrhage and area or blood vessel involved. Similarly site of involvement of brain in infarction type were not recorded in the discharged file.

Table-I

Distribution of Hypertensive and Diabetic subjects included in the study (n=657)

Disease	Frequency	Percent
Hypertensive	459	69.9
Normotensive	198	30.1
Diabetic	170	25.9
Non-diabetic	487	74.1

n = Number of subjects

Table-II

Distribution of sides of the subjects affected(n=657).

Side affected	Frequency	Percent
Right hemiplegia	279	42.5
Left hemiplegia	236	35.9
Monoplegia and no Neurological deficit	142	21.6
Total	657	100.0

n= Number of subjects

Table-III

Distribution of Haemorrhagic and Infarctive strokes of the study subjects (n - 657).

Type	Frequency	Percent
Haemorrhagic	211	32.1
Infarctive	446	67.9
Total	657	100.0

Discussion:

A few aetiological factors of stroke patients in the present study shows pictures that can be correlated with many international and national study. In our study 63.6 % were male and 36.4 % were female patients. In a study, it was found that women accounted 48% of the study population in hospital based stroke registry over 10 years period[6]. Hayee et al analyzed data of 1272 admitted stroke patients and found that 65.17 % were male [7] . Another study by Alam et al showed that out of 1020 stroke patients 89.32% were male[8]. Arif et al studied 100 admitted stroke patients and found that 74% of the patients were male[9]. The incidence of stroke is 19 % higher among men than women of all races[10]. So it is evident that males are more frequently affected by stroke than female. In this study population age range was from 15 years to 100 years. Mean age was 60.90±13 years. Most of the patients were above 50 years age (76.56 %), 50.38% were above 60 years. Youngers were not exempted from stroke, 23.43 % were below 40 years age. Hayee et al showed that out of mean age of 63.29 years, 9.98 % had age range of 15 to 45 years[7]. Alam et al. showed that 52.65% of their patients were between the ages of 40 and 60 years, 16.19 % were diabetic and 81.98% were hypertensive[8]. Roth EJ found that stroke is primarily a disease of older individual, but 28 % strokes occur in persons younger than 65 years[10].

In our study two modifiable risk factor, hypertension and diabetes mellitus were significantly high. 69.9 % of the patients were hypertensive and 25.9 % were diabetic. Department of preventive medicine, University of South California in one study found that hypertension, diabetes, heart failure and smoking were significant stroke risk factor in both men and women [11].Bangou-Bredent at al in his study revealed that 76% of the patients suffered from hypertension. Hypertension, obesity, elevated serum lipid and diabetes mellitus were independently and significantly associated with ischemic stroke[12]. Study done by Arif et al. showed that 67.5% of his patients were hypertensive[9]. Greshan et al in their Framingham study showed that among stroke survivors 67 % have chronic hypertension[13]. Diabetes mellitus isolated or in conjunction with hypertension were found significantly associated with stroke. In this study 25.9 % of the patients were diabetic. Tuomilehoto et al made a risk factor survey with over 16500 patients and found that diabetic subjects have a very high risk of death from stroke particularly women and duration of diabetes was an important factor contributing to the risk of stroke[5]. Diabetes mellitus increases the relative risk of ischemic stroke to 3 to 6 times that of general population. This risk can be partly attributed to the higher prevalence of hypertension and heart disease among diabetics, even after controlling for these factors, diabetes independently double the risk[14,15,16]. Framingham study also showed that the prevalence of diabetes among survivors was 20%. As CT scan

were done in all patients included in the study, we found that 67.9 % had CT scan proved infarction and 32.1% had hemorrhagic lesion that included intra-cerebral and subarachnoid hemmorrhage. In many studies on pathogenesis of stroke, it has been shown that more than two third of cases are due to infarction. In general 80 to 85 percent of patients suffer from ischemic infarction and 15 to 20 % suffers from hemorrhage[2]. Minor variation in our study may be due to the fact that many minor stroke patients with no or negligible neurological deficit do not get admitted into the hospital.

In the present study, we found that 78.38% of patients had either right sided or left sided hemiplegia, 22.6% had either monoplegia or simple facial palsy or no gross neurological deficit. Bilateral hemiplegia were not found among stroke survivors. Of hemiplegic patients 42.5% had right sided hemiplegia 35.9% had left sided hemiplegia. There was no significant variation among male and female regarding side predilection. So it is evident from this study that in stroke, dominant hemispheres are predominantly affected and there is no gender specificity for side predilection. This study was done in a neurology oriented private hospital in Dhaka. Many important data regarding modifiable and non- modifiable risk factors, such as smoking, family history, lipid profile, contraceptive use etc. could not be analyzed because of inadequate and incomplete information found in discharged file. The study showed that stroke pattern in our society is almost similar to that of other community in the world. More studies are needed to establish the pattern of stroke and their risk factor association in our community.

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