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ORIGINAL ARTICLES

Blood Monocyte Count In Hypertensive Ischemic Stroke Patients

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Abstract:

Background: *Traditional clinical risk factors for stroke do not predict all individual at risk of developing the events and only two-third of all strokes can be attributed to know casual risk factors. So it is necessary to explore the non-traditional risk markers, as predictors for future events. Hence the study was designed to find out the association of blood monocyte count in hypertensive ischemic stroke patients.* ***Methodology:*** *This case control study was carried out in the Department of Neurology, Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka . Total 100 cases were studied, 50 of them hypertensive ischemic stroke patients regarded as case, and another 50 were hypertensive haemorrhagic stroke patients regarded as control. The socio-demographic and clinical data were collected by semi-structured questionnaire and the blood was collected from both cases and controls and were analized by auto analyzer for total count and differential count, at the Department. of Clinical Pathology, BSMMU. Statistical analysis were done by SPSS version 16.* ***Results:*** *Total 100 cases were studied in this study. All of them were hypertensive patients and other risk factors like diabetes mellitus, dyslipidemia, cigarrette smoking were also present. The mean(±SD) systolic blood pressure was 144.6±12.37 mmHg & diastolic blood pressure was 84.5±.57 mmHg in case group. In control group the mean(±SD) systolic blood pressure was 139.2±8.41 mmHg & the mean(±SD) diastolic blood pressure was 82.6±6.64 mmHg. The difference of mean percentage value of monocyte count was statistically significant between the case and the control group in unpaired t-test, but others were not significant. The mean (±SD) monocyte was 5.78±1.2 % in case group. In control group mean (±SD) monocyte was 3.46±1.2%.* ***Conclusion:*** *Raised blood monocyte count is significantly and independently associated with ischemic stroke of hypertensive patients.*

Key words: *Hypertension, ischemic stroke, monocyte*

Introduction:

Among the neurological diseases of adult life, stroke is ranked first in frequency and importance. It is the third most common cause of adult death in the developed world, only next to the ischemic heart disease and the cancer; and after the age of 40 years, it is the most common cause of physical disability[1].

It is a common medical emergency with an annual incidence ranges from 180 to 300 per 100,000 population[2]. In Bangladesh about 40% to 50% of beds are occupied by stroke patients in a neurology ward[3].

Many risk factors are associated with stroke. Among them arterial hypertension is responsible for increasing the relative risk of stroke, an estimated three- to four fold[4] and effective control of hypertension has the potential to reduce the risk of primary stroke by nearly 40%[5]. In a hospital based prospective study in stroke clinic of Bangabandhu Sheikh Mujib Medical University (BSMMU) had shown that 60% patients were known to have hypertension before stroke, 15% were of them detected after stroke, where as 25% were found to be non-hypertensive[6].

Hypertension is responsible for both ischemic and hemorrhagic stroke. In large vessel disease atherosclerosis and small vessel, microatherosclerosis is the pathologic lesion by hypertension , and in hemorrhagic stroke, lipohyalinolysis[7,11].

The initiation and progression of atherosclerosis is an inflammatory process[7]. This process involving the infiltration of circulating monocytes into the vessel wall, with subsequent differentiation and involvement of other cells leading to the development of an atherosclerotic plaque.

Along with infiltration of monocyte, many inflammatory markers like cytokines, chemokines and adhesion molecules are released or expressed by macrophages, smooth muscle cells, and the endothelium. These can be contributed as risk markers of atherosclerosis.

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But in one comparative study it was demonstrated that monocyte count, but not CRP or IL-6, is an independent risk marker for sub clinical carotid atherosclerosis[8].

The traditional clinical risk factors for cardiovascular disease do not predict all individual at risk of developing cardiovascular events[9] and only two-third of all strokes can be attributed to know casual risk factors[10]. So it is necessary to explore the non-traditional risk markers, as predictors for future events. The blood monocyte count is one of that important risk marker.

Objectives

- To find out the association of raised monocyte count with hypertensive ischemic stroke patients.
- To compare the measurements of monocyte count between hypertensive ischemic stroke patients and hypertensive hemorrhagic stroke patients.

Materials and Methods:

It was a case control study, carried out in the Department of Neurology, BSMMU, Dhaka from January 2008 to December 2009 for a duration of two years. Stroke patients (both ischemic and hemorrhagic), who were attended the stroke clinic in the out-patient department and got admitted in the hospital were included. A total of 100 subjects were included, 50 cases and 50 controls. Hypertensive stroke patients were selected by purposive sampling method considering the similarity regarding age and sex in both case and control group. Patients with abnormal cardiac conditions giving rise to cardio-embolic stroke and with history of infection or fever weeks before stroke onset were excluded from the case group. On the other hand, patients with large hemorrhages of unclear origin; with intraventricular hemorrhage or extension; with sub arachnoid hemorrhage were excluded from the control group. Data were collected by semi-structured questionnaire by the investigator. Data of control were taken after collection of cases so that proper matching of age and sex could be done. The blood count was done by auto-analyzer and rechecked by a consultant.

Statistical Analysis

All data was recorded systematically in preformed data collection form and quantitative data was expressed as mean and standard deviation and qualitative data as frequency distribution and percentage. Specificity and sensitivity of the monocyte count for diagnosing ischemic stroke were analysed by Receiver-operator characteristic (ROC) curve. Statistical analysis was performed by using SPSS for windows version 16.0. Ninety five percent confidence limit was taken. Probability value <0.05 was considered as level of significance.

Results and Observations

The results were given in the tabulated form:

The mean(±SD) age was 54.1±8.0 years in case and 54.0±8.2 in control group. Maximum number was found in the age group of 51-60 in case and 41-50 years in control group (Table-I).

Among the100 study subjects, 68.0% were male and rest 32.0% were female patients in case group. In control group 74.0% were male and rest 26.0% were female patients (Table-II).

The mean (±SD) systolic blood pressure (mm/Hg) was 144.6±12.37 mmHg in case group. In control group the mean (±SD) systolic blood pressure was 139.2±8.41 mmHg. The mean (±SD) diastolic blood pressure (mm/Hg) was 84.5±.57 mmHg in case group. In control group the mean (±SD) diastolic blood pressure was 82.6±6.64 mmHg. The mean systolic blood pressure difference was statistically significant ($p<0.05$) between case and control group but diastolic blood pressure was almost similar between two groups in unpaired t-test (Table-III).

Table-I

Distribution of the study patients' age by groups

Age in years	Case (n=50)		Control (n=50)		P value
	n	%	n	%	
41-50	14	28.0	18	36.0	
51-60	19	38.0	17	34.0	
61-70	16	32.0	15	30.0	
>71	1	2.0	0	0.0	
Mean ±SD	54.1±8.0		54.0±8.2		0.508^{NS}

NS= not significant

P value reached from unpaired't' test

Table-II

Distribution of the study patients' sex by group

Sex	Case (n=50)		Control (n=50)		P
	n	%	n	%	value
Male	34	68.0	37	74.0	0.508 NS
Female	16	32.0	13	26.0	

NS= not significant
P value reached from chi square test

Table-III

Mean distribution of Arterial Blood Pressure (mm of Hg) of the study patients

Blood pressure (mm/Hg)	Case (n=50) Mean±SD	Control (n=50) Mean±SD	Pvalue
Systolic blood pressure	144.6±12.37	139.2±8.41	0.012S
Range (min-max)	(130-170)	(125-160)	
Diastolic blood pressure	84.5±6.57	82.6±6.64	0.153NS
Range (min-max)	(70-100)	(70-95)	

S= significant, NS=not significant
P value reached from unpaired 't' test

The mean (±SD) duration of hypertension was 8.3±4.2 years in case group. In control group the mean (±SD) duration of hypertension was 8.2±4.5 years. The mean duration of hypertension difference was not statistically significant (p>0.05) between two groups in unpaired t-test (Table-IV).

Regular taking of anti-hypertensive drugs was found 14(28.0%) patients in case and 22 (44.0%) in control group (Table-V). No significant (p>0.05) difference was found between case and control group in chi square test.

The mean (±SD) total count was 9.47±2.4 $X10^9$/L in case group. In control group the mean (±SD) total count was 8.24±2.5 $X10^9$/L. The mean (±SD) neutrophil was 6.06±2.48 $X10^9$/L in case group. In control group the mean (±SD) neutrophil count was 4.77±1.4 $X10^9$/L. The mean (±SD) monocyte count was 0.54±0.1 $X10^9$/L with their ranged from 0.27 to 0.80 $X10^9$/L in case group. In control group the mean (±SD) monocyte count was 0.39±0.4 $X10^9$/L with their ranged from 0.02 to 0.8 $X10^9$/L (Table-VI). The mean total count, neutrophil and monocyte difference were statistically significant (p<0.05) between case group and control group in unpaired t-test, but others were not significant (p>0.05).

The mean (±SD) monocyte was 5.78±1.2 % with their ranged from 0.7 to 7.6% in case group. In control group the mean (±SD) monocyte was 3.46±1.2 % with their ranged from 0.2 to 5.3%. The mean monocyte difference was statistically significant (p<0.05) between case and control group in unpaired t-test, but others were not significant (p>0.05) (Table-VII).

The area under the receiver-operator characteristic (ROC) curves (Fig.-1) for the Ischemic stroke predictors was

Table-IV

Distribution of the Mean duration of hypertension of the study patients (years)

	Case (n=50) Mean±SD	Control (n=50) Mean±SD	Pvalue
Duration of hypertension	8.3±4.2	8.2±4.5	0.958NS
Range (min-max)	(3-18)	(1-20)	

NS= not significant
P value reached from unpaired 't' test

Table V

Distribution of the study patients' status of talking anti-hypertensive drugs by groups

Status of taking anti-hypertensive drugs	Case (n=50)		Control (n=50)		Pvalue
	n	%	n	%	
Regular	14	28.0	22	44.0	0.095^{NS}
Irregular	36	72.0	28	56.0	

NS= not significant
P value reached from chi square test

Table VI

Distribution of the study patients by Total count, Neutrophil count, Lymphocyte count, Monocyte count and Eosinophil count in absolute value ($X10^9/L$).

	Case (n=50) Mean±SD	Control (n=50) Mean±SD	P value
Total count (TC)	9.47±2.4	8.24±2.5	0.013^{S}
Range (min-max)	(6.5-15.9)	(6.9-15)	
Neutrophil	6.06±2.48	4.77±1.4	0.002^{S}
Range (min-max)	(2.61-9.8)	(2.35-8.92)	
Lymphocyte	2.71±1.3	2.71±0.7	0.988^{NS}
Range (min-max)	(0.86-6.78)	(1.83-4.4)	
Monocyte	0.54±0.1	0.39±0.4	0.019^{S}
Range (min-max)	(0.27-0.80)	(0.02-0.80)	
Eosinophil	0.27±0.2	0.49±0.7	0.051^{NS}
Range (min-max)	(0.0-0.93)	(0.0-1.9)	

S= significant, NS= not significant
P value reached from unpaired 't' test

Table-VII

Distribution of the study patients by Neutrophil count, Lymphocyte count, Monocyte count and Eosinophil count in percentage value (%).

	Case (n=50) Mean±SD	Control (n=50) Mean±SD	P value
Neutrophil	61.2±15.8	58.4±9.6	0.285^{NS}
Range (min-max)	(39.9-83.9)	(41.7-77.8)	
Lymphocyte	30.0±9.5	32.6±8.9	0.166^{NS}
Range (min-max)	(13.3-53.6)	(15.1-49.4)	
Monocyte	5.78±1.2	3.46±1.2	0.001^{S}
Range (min-max)	(0.7-7.6)	(0.2-5.3)	
Eosinophil	3.02±2.1	3.75±3.4	0.238^{NS}
Range (min-max)	(0-8.7)	(0-11)	

S= significant, NS= not significant
P value reached from unpaired 't' test

depicted in the Table VIII. Based on the receiver-operator characteristic (ROC) curves monocyte had the best area under curve compared to neutrophil. Receiver-operator characteristic (ROC) were constructed using monocyte value of the patients between two groups, which gave a monocyte cut off value of >0.523 as the value with a best combination of sensitivity and specificity for Ischemic stroke predictors. At this monocyte cut-off value of >0.523, the sensitivity and specificity of monocyte in diagnosing of Ischemic stroke predictors was found to be 82.0% and 64.0%, respectively with correctly classified 72.0% of the patients. Neutrophil cut-off value of >4.355, the sensitivity and specificity of neutrophil in diagnosing of Ischemic stroke predictors was found to be 84.0% and 34.0%, respectively with correctly classified 42.0% of the patients (Table VIII; Fig. 1)

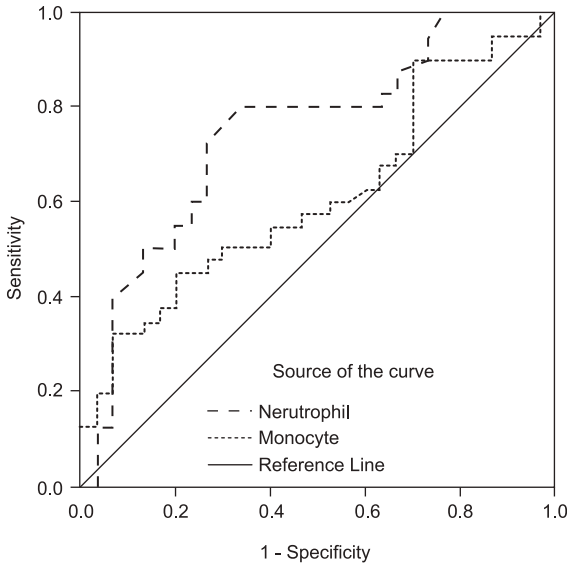

Fig.-1: *Receiver-operator characteristic curves of Monocyte and Neutrophil for detection of ischemic stroke.*

Table-VIII

Receiver-operator characteristic (ROC) curve of Monocyte and Neutrophil for prediction of Ischemic stroke

	Sensitivity	Specificity	Area under the ROC curve	95% Confidence interval(CI)	
				Lower bound	Upper bound
Monocyte	82.0	64.0	0.757	0.66	0.85
Neutrophil	84.0	34.0	0.637	0.528	0.746

Discussion:

The aim of the study was to find out the association of raised monocyte count with hypertensive ischemic stroke patient. The following results and observation were made.

As all the respondents in both group had stroke and it was likely to present one or more risk factors in a single patient. It was tiring to include only hypertensive patients, though not possible in all respondents. Out of all patients 44.0% had Smoking habit in case group and 26.0% in control group, and 76.0% of patients of case group, 68% of control group had history of tobacco and betel nut chewing. Out of all patients 22.0% of case group and 24.0% of control group had diabetes mellitus and 16.0%% of case group and 12.0% of control group had ischemic heart disease. There were no significant ($p>0.05$) difference between the two groups with these variables. The mean triglyceride difference was statistically significant ($p<0.05$) between case and control group in unpaired t-test, but the mean total cholesterol was not significant ($p>0.05$); hemorrhagic stroke patients had lower total cholesterol and triglyceride level than ischemic stroke.

All the 100 patients had arterial hypertension. Mean (±SD) duration of hypertension was 8.3±4.2 in case group and 8.2±4.5 in control group. In case group, 72.0% and in control group 56.0% were irregularly took antihypertensive drugs.

There were significant difference between the mean values of absolute total count, neutrophil count and monocyte count between case and control group. The mean (±SD) total count was 9.47±2.4 $X10^9$/L in case group. In control group mean (±SD) total count was 8.24±2.5 $X10^9$/L. The mean (±SD) neutrophil was 6.06±2.48 $X10^9$/L in case group. In control group the mean (±SD) neutrophil count was 4.77±1.4 $X10^9$/L.The mean(±SD) monocyte count was 0.54±0.1 $X10^9$/L in case group. In control group mean (±SD) monocyte count was 0.39±0.4 $X10^9$/L.

The difference of mean percentage value of monocyte count was statistically significant between the case and the control group in unpaired t-test, but others were not significant. The mean (±SD) monocyte was 5.78±1.2 % in case group. In control group mean (±SD) monocyte was 3.46±1.2%.

From previous studies[12,13], it was reported that the monocyte count raised between 3 and 7 days after the stroke onset; again Cortina et al.[14] studied with the blood, that drawn in the first 24 hour after acute stroke, also detected the higher monocyte count. So it can be said that, the monocyte count difference may be more related to the pre-stroke inflammatory status rather than as a consequence of stroke. In this study, blood was taken from the patients within one month of the stroke onset; we exclude the patients with monocytosis and the patients with history of fever or infection within the week before stroke onset were excluded. For these the higher monocyte count in the ischemic stroke patients than the hemorrhagic stroke patients that were observed in this study may be a result of pre-stroke inflammatory process.

In a large series of study of 236 patients Cortina et al.[14] have showed that, the mean (±SD) value of monocytes ($X10^9$/L) of lacunar infarction patients' were 0.698(±0.383) and that of deep intracerebral hemorrhagic patients' were 0.555(±0.336). There were significant (*p*= 0.002) difference between the two conditions with Odd ratio 0.999 (95% CI 0.998 to 0.999). This study results were consistent with these findings, though the absolute mean values were lower. Probably, these were because of normal racial variations of the values.

With Receiver-operator characteristic curve analysis it was observed that blood monocyte count was 82% sensitive and 64% specific for the diagnosis of ischemic stroke and with cut of value >0.523 it correctly classified 72% of patients.

Conclusion of the study

Raised blood monocyte count is significantly and independently associated with ischemic stroke of hypertensive patients although mean value was within normal range but significant difference was observed between case and control group. Further studies needed to evaluate the predictive role of this observation.

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Characteristics of headaches in migraine patients

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Abstract

Background: *Headaches in migraine vary from a dull ache to a severe throbbing pain among patients and even in the same patient from time to time. Knowing the characteristics of headache among the patients will help to evaluate each patient and in specific treatment. Objective: To evaluate the characteristics of headache, its associated symptoms, precipitating and relieving factors.* ***Materials and methods:*** *It was an observational, analytical study. One hundred and thirty migraine patients were diagnosed on the basis of International Headache Society (IHS) criteria and were evaluated for the duration of migraine, quality of pain, severity, frequency of attack, duration of episode, location of pain, time of onset, associated symptoms, precipitating and relieving factors.* ***Results:*** *Maximum patient (93.84%) were in the age group of 15-34 years. Female (60%) were dominant over male patient. Moderate (87.69% case), aching (60% case) and generalized (67% case) pain predominate. About 85% patient reported that they had been suffering 'for years' with migraine. Regarding duration of each attack, about 64% reported their episode persists 'for days'. Most common associated symptoms reported were nausea, photophobia, phonophobia and insomnia. Regarding precipitating factors, 58.5% reported sunlight and 33% reported television. Sleep, analgesics and antiemetics were reported as relieving factors in a large number of cases.* ***Conclusion:*** *This study concludes that migraine is a disease of younger age group with female preponderance. Headache characteristics are more or less specific and similar among the patients.*

Key wards: *Characteristics, Headache, Migraine*

Introduction:

Migraine is a familial disorder characterized by recurrent attack of headaches widely variable in intensity, frequency and duration. Attacks are commonly unilateral and are usually associated with anorexia, nausea and vomiting. In some cases they are preceded by, or associated with, neurological or mood disturbances. All the above characteristics are not necessarily present in each attack or in each patient[1].

Migraine is a remarkably common condition and distributed world wide. Its prevalence among Caucasians is in the range of 4 to 6 percent among men and 13 to 18 percent among women. The reported numbers are lower among Asians. Migraine may have its onset in childhood but usually begins in adolescence. In more than 80% of patients, the onset is before 30 years of age. Migraine usually diminishes in severity and frequency with age[2].

Approximately 60% of patients with migraine experience 2 or more attacks per months, and more than 75% of migraineurs reports severe or extremely severe pain during attacks. More than 90% of patients report on impaired ability to function during migraine attacks, and 53% report severe disability requiring bed rest[3]. Indirect costs of migraine related to decreased productivity and lost days of work is very high.

In this context, this study was undertaken to evaluate the characteristics of migraine headache for better understanding of management.

Aims and objectives

To evaluate the characteristics of migraine headache in terms of its total duration, frequency of attack, quality of pain, intensity, duration of episode, location, associated symptoms, precipitating and relieving factors. This evaluation will help to provide specific treatment.

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Materials and methods:

This study was an observational analytical study conducted at Neurology Out Patients Department (NOPD) of Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka during the period from July 2004 to March 2006. A total number of 130 patients were ultimately included from initial 200 cases of migraine patients diagnosed by the criteria of International Headache Society (IHS). Verbal and written consent were taken from all patients. Detailed history of headache regarding its total duration, severity, location, time of onset, precipitating and relieving factors were taken. Visual analogue scale (VAS) was used for rating the intensity and severity of headache.

Results:

Table-I shows age group of migraine patients. One hundred and thirty cases were recorded between the ages of 15 to 54 years. Mean age was found 25.13±0.71 years. Maximum patients (47.69% and 46.15%) were fall in age group of 15 to 34 years. Table-II shows distribution of sex. Number of male patients were 51 (40%) and female patients was 79 (60%). A female dominance were found among the patients.

Table-I

Distribution of age of patients

Age Group	Number(n=130)	Percent
15-24	62	47.69
25-34	60	46.15
35-44	06	4.61
45-54	02	1.54

Table-II

Sex distribution of patients

Sex	Number (n=130)	Percent
Male	51	40
Female	79	60

The characteristics of migraine headache was shown in table-III. A large number (84.61%) of patient had duration of headache for years or more and only 15.39% had months. Fifty one (39.23%) patient had throbbing quality, but 60% had aching pain. Most of the patients (87.69%) had moderate and 11.53% had severe to very severe pain. Each attack was persisted for days in 83 patient, for hours in 37 patients and continuous in 10 patients. Three or less attack was found in every month in 40 patients and 4 or more attack in 21 patient. Eight or less attack per year was in 20 patients. Continuous pain was found in 40 patients. Unilateral headache (46.15%) was less than generalized headache (51.53%). Regarding time of onset of headache, 44 patients reported its occurrence in morning, another 44 reported in anytime. Evening and night time was reported by 27 and 15 patients respectively.

Table-III

Distribution of characteristics of headache of patients

Characteristics of pain	Specificity	Frequency (n=130)	Percentage
Total duration	Months	20	15.38
	Years	110	84.61
Quality	Throbbing	51	39.23
	Aching	78	60
	Others	01	0.76
Severity	Mild	01	0.76
	Moderate	114	87.69
	Severe	15	11.53
Duration of episodes	Hours	37	28.46
	Days	83	63.84
	Continuous	10	7.69
Frequency of attack	Months (3 or less)	40	30.76
	Months (4 or more)	21	16.15
	Years (<8)	20	15.38
	Years (>8)	09	6.92
	Continuous	40	30.76
Location of headache	Unilateral	60	46.15
	Generalized	67	51.53
	Others	03	2.30
Time of onset	Morning	44	33.84
	Evening	27	20.76
	Night	15	11.53
	Anytime	44	33.84

The associated symptoms of migraine reported by the patient was shown in table-IV. Nausea and photophobia were most common symptoms and reported by 120 and 55 patients respectively. Vomiting, aura, phonophobia, vertigo and insomnia were less reported symptoms.

Table-IV

Distribution of associated symptoms of migraine

Symptoms	Frequency (n=130)	Percentage
Nausea	120	92.30
Vomiting	13	10.14
Aura	12	9.36
Photophobia	55	42.90
Phonophobia	21	16.38
Vertigo	10	7.80
Insomnia	12	9.36

Table-V clearly demonstrated the precipitating factors. Most frequent precipitating factor was sunlight (58.5%). Followed by television (33%) and insomnia (6.2%). Study and other factors were less frequent.

Table-V

Distribution of precipitating factors of migraine

Precipitating factor	Frequency (n=130)	Percentage
Television	43	33
Sunlight	76	58.5
Study	02	1.5
Insomnia	08	6.2
Others	01	0.8

From the table-VI, it is clear that the analgesics (83% case), sleep (80% case) and antiemetics (54.60% case) had the main role as relieving factors. 'Rest' was reported less frequently as the same.

Table-VI

Distribution of relieving factors among migraine patient

Relieving factors	Frequency (n=130)	Percentage
Rest	34	26.2
Sleep	104	80
Analgesics	108	83.10
Antiemetics	70	54.6
Anxiolytics	03	2.3

Discussion

There is nothing typical about headache in migraine. They vary from a dull ache to a severe throbbing headache. Quite frequently they occur on awakening from sleep. Migraine occurs usually at age group of 10 to 60 years. In this study, 15 to 54 years old patients were selected. Mean age was 25.13±0.71 years. Maximum patients (93.84%) were in the age group between 15 to 34 years.

In a study[4], mean value of age was found 11.7±2.7 years which was very low. But in another study[5], it was 39 years which was again high in relation to this study.

Number of male patient was 51 (40%) and female was 79 (60%) in this study. Similar findings were observed in other studies [6,7] also.

Characteristics of pain with respect to total duration, quality, severity, duration of episode, frequency of attack, location of pain and time of onset were assessed thoroughly in this study. Total duration attack was found in 84.61% of cases for years or more. Throbbing pain was found in 39.23% cases and aching quality in 60% cases. In a study[6] Kelman observed that 90.9% has throbbing pain and 86.5% has aching. More or less similar findings were reported in other studies[8,9]. In this study, most of the patients (87.69%) reported moderate pain. Severe pain was reported by only 11.53% of patients. Moderate and severe pain were found 62.4% and 26.4% in another study[10]. Duration of episodes were 'for days' in 63,84% cases and 'for hours' in 28.46%. In this series, 30.76% patient reported 3 or less attacks per month and 4 or more attacks in 16.15% per month. Unilateral location of headache was reported by 46.15% of patients which was slightly lower than the percentage of patients (51.53%) reported generalized headache. Time of onset of headache reported in this study was found at 'morning' and 'anytime' of day in 33.84% cases for each. Findings of this study is similar with that of Kelman[6]. Most commonly reported associated symptoms in this series was nausea (92.3%), photophobia (42.9%), phonophobia (16.38%), insomnia (9.36%) and vertigo (7.8%). Main et al. demonstrated the significant effect of photophobia and phonophobia in his study. Among the precipitating factors, sunlight (58.5%) and television (33%) were most frequently reported factors. In this study, most commonly reported relieving factors were analgesic (83%), sleep (80%) and antiemetic (53.84%).

Conclusion:

From the evaluation of the characteristics of migraine headache in this study, it can be concluded that most of the parameter like total duration, quality, severity, frequency of attack, duration of episodes, location of pain and time of onset were specific and more or less similar. Associated symptoms, precipitating and relieving factors were also unique.

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Association of Serum Magnesium with Migraine

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Abstract

Background: *Migraine is a primary headache disorder characterized by recurring attacks of pain and associated symptoms. The management modality is still unsatisfactory due to poor understanding of its cause and pathogenesis. Many clinical observations and investigations support the presence of both systemic and brain magnesium deficiency in migraineurs. Knowing the serum magnesium concentration may help to understand its pathogenesis and management modality.* ***Objective:*** *To estimate the serum concentration of magnesium and finds it association with migraine.* ***Methods:*** *Migraine patients were chosen as case and other non-headache patient without any chronic disease were taken as control. Total number of cases were 130 and controls were 80. Estimation of serum magnesium were done for both case and control groups.* ***Results:*** *Out of 210 respondent, 122 (58%) were female and 88 (42%) male. Mean age of male patients was 28.56±1.08 years and female patients was 28.22±0.89 years. Mean age of case and control were 25.13±0.71 and 33.31±10.57 years respectively. Mean values of serum magnesium concentration in case group was 2.09±2.29 mg/dl and in control 2.13±1.53 mg/dl. Low level was found in 9.23% of patients and 2.5% of control.* ***Conclusion:*** *Values of serum magnesium concentrations were found towards low in case group. True deficiency were present in some patients. Unfortunately, the study was statistically insignificant.*

Key words: *Migraine, Serum Magnesium, Association.*

Introduction:

Migraine is a familial disorder characterized by periodic, commonly unilateral, often pulsatile headache that begins in childhood, adolescence, or early adult life and recur with diminishing frequency during advancing years[1].

World Health Organization (WHO) declared migraine to be among the most disabling medical conditions experienced word-wide. Most commonly, the initial attack occurs during adolescence and by 40 years of age, 90% of those with the condition have been their first attack. After puberty it is more common in female. Once migraine has developed, it tends to recur with varying frequency through-out the patients life. Attacks have a tendency to get milder and occur less often in later years although this certainly is not a universal findings[2] .

For decades, migraine has been considered to be an episodic, neurovascular disorder, without long term consequences to the brain. Recently, it has been established that migraine acts as a risk factor for white matter lesions, silent infarctions and ischemic stroke[3].

So far, it has not been possible to determine, from the many clinical observations and investigations, a unifying theory as to the cause and pathogenesis of migraine. As a result the treatment is still remains non-specific and is largely unsatisfactory. There is increasing evidence to support the presence of both systemic and brain magnesium deficiency in migraineurs, particularly in the occipital lobes[4].

For many years, magnesium deficiency has been suspected to play a role in the pathogenesis of migraine. A theoretical basis for the role of magnesium deficiency in headaches was first proposed in 1985[5].

A magnesium deficit can result in an abnormality of mitochondrial oxidative phosphorylation and lead to gain in NMDA receptor function, thereby causing an instability of neuronal polarization because of a loss

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of ionic homeostasis. This would than leads to a state of neuronal hyperexitability and a lower threshold for spontaneous depolarization[6]. Neuronal excitability may be responsible for the phenomena of spreading cortical activation and depression, with subsequent changes in regional cerebral blood flow[7].

The magnesium concentration also directly influences the biological clock function. The biological marker for hyperfunction and hypofunction of biological clock is melatonin level secreted by pineal gland. In hypofunction the level decreases and leads to nervous hyperexcitability[8], which in tern responsible for migraine symptoms.

Magnesium concentration has also an effect on serotonin receptors, nitric oxide synthesis and release and other migraine related receptors and neurotransmitters[9].

So, it is important to knowing the state of serum magnesium concentration which may influence the pathogenesis and management modality.

Aims and Objectives

1) To estimate the serum concentration of magnesium.
2) To find the association between serum magnesium concentration and migraine.

Methods and Materials:

This was an observational analytical case-control study conducted at the Neurology Outpatient Department (NOPD) of Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka during the period from July 2004 to March 2006. One hundred and thirty migraine patients of age between 15 to 54 years were chosen as case, diagnosed on the basis of International Headache Society (IHS) criteria. Other 80 patients, who were headache free, having neurological or non-neurological diseases without any history of chronic illness were taken as control. Verbal and written consents were taken from both case and control group. Serum magnesium concentrations were estimated for both the groups.

Results:

All the respondents of case and control groups were within the age between 15 to 54 years and having both sexes, all occupation, all socioeconomic status and all religion. Mean age of case group was 25.13±0.71 years and control was 33.31±10.57 years. Maximum patient 47.69% and 46.15%, were in the age group of 15 to 24 years and 25 to 34 years respectively (table-I).

The mean ages of male and female respondents were 28.56±1.08 and 28.22±0.89 years respectively (table-II). The age difference of male and female respondents were not significant (P>0.05).

Regarding sex distribution, table-III shows a female dominance in both case and control groups. Statistically the level was not significant (P>0.05).

According to table-IV the mean of serum magnesium concentration (quantitative data) was 2.09±2.29 mg/dl and 2.13±1.53 mg/dl in case and control group respectively which was not significant (P>0.05).

Table-V compares the serum magnesium level (qualitative data) between groups. It was found that only 9.23% of cases and 2.5% of controls have low values. The level of serum magnesium between groups was not significant (P>0.05).

Table-I

Distribution of age of case and control groups

Age group	Number Case (n=130)	%	Mean of age	Number Control (n=80)	%	Mean of age
15-24	62	47.69	25.13±0.71	10	12.5	33.31±10.57
25-34	60	46.15		25	31.25	
35-44	06	4.61		40	50	
45-54	02	1.54		05	6.25	

Table-II

Distribution of age and sex of respondents

Sex	Number	Mean of age	SEM	P value
Male	88	28.56	±1.08	0.698
Female	122	28.22	±0.89	

Table-III

Sex distribution between case and control groups

Sex	Groups		P value
	Case (n=130) No. (%)	Control (n=80) No. (%)	
Male	51 (40%)	37 (46.25%)	0.465
Female	79 (60%)	43(53.75%)	

Table-IV

Comparison of values of serum magnesium concentration (quantitative data) between groups.

Group	N	Mean	SD	SEM	P value
Case	130	2.09	0.26	2.29	0.253
Control	80	2.13	0.14	1.53	

Table-V

Comparison of serum magnesium level (qualitative data) between groups.

Serum magnesium . conc	Groups		P value
	Case (n=130) No. (%)	Control (n=80) No. (%)	
Low	12 (9.23%)	02 (2.5%)	2.10
Normal	118 (90.77%)	78 (97.5%)	

Discussion:

The pathogenesis of migraine is so far poorly understood. Various clinical investigations suggest that low serum magnesium might have a role in the causation of migraine. A large number of studies were available which looked the concentration of serum magnesium of which some were found significant. In this study, the mean age of case group was 25.13±0.71 years. In a study[10], it was found 11.7±2.7 years which was very low. In another study[11], it was 39 years which was high.

The number of male and female respondent in case group was 51 (40%) and 79 (60%) in this study. Here the number of female patients were much higher than male. This study was found consistent with other studies[12,13] with relation to sex ratio.

In this series, values of serum magnesium concentration was compared between case and control group. Mean serum magnesium level was 2.09±2.29 mg/dl and 2.13±1.53 mg/dl in the groups respectively. The case group has tended towards low concentration. The relationship was found insignificant (P>0.05). Schoenen et al.[14] in 1991 had found similar results. But, Sarchielli et al.[15] in 1992 found that there was significant lowering of serum magnesium.

This study showed that only 9.23% of case and 2.5% of control have low (below normal level) serum magnesium concentration. The level was found insignificant (P>0.05) between the groups. Schoenen et al. observed similar findings.[15] But other studies[16,17,18] showed the deficiency of serum magnesium in migraine patients.

Conclusion and recommendation:

From this study, it can be concluded that low serum magnesium may be a determining factor in the causation of migraine, though the study was found insignificant. This type of study should be done with due importance which should include a very large sample for conclusive purpose.

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CASE REPORTS

An aneurysmal bone cyst with D1 collapse, D2 retrolisthesis and spinal cord compression from C7 to D2: A Rare Case Report

HARADHAN DEB NATH[1], SHASHANK SHARAD KALE[2], PANKAJ KUMAR SINGH[3], NISHANT GOYAL[4]

Abstract:

Aneurysmal bone cyst is a benign tumor. If complete resection cannot be done, recurrence rate is very high. We discuss the case because spinal aneurysmal bone cyst is a rare bony disease. The operation was done by posterior decompression and fusion and fixation by pedicle screw at D3, D4 level and lateral mass screw at C5, C6 level and fixed by rods.

Key words: *Aneurysmal bone cyst, retrolisthesis, collapse vertebrae, spinal cord compression.*

Abbreviation: *AIIMS (All India Institute of Medical Science), AB (Aneurysmal Bone Cyst).*

Introduction:

Aneurysmal bone cyst in the spine, involvement of the thoracic, lumbar cervical or sacral level, in order of frequency is not very common.[1] Vertebral aneurysmal bone cysts generally arise in the posterior osseous elements, including the neural arches, laminae, transverse and spinous processes; the vertebral bodies are affected less frequently and rarely does this occur in isolation without posterior osseous abnormalities. Aneurysmal bone cysts may extend from one vertebra to another, to an adjacent rib, or to the paraspinal soft tissues, simulating the appearance of infection or a malignant tumor.[2,3,4]

Case Report:

A 8-years-old-boy was admitted in the Department of Neurosurgery, AIIMS, with the complaints of neck and upper back pain for two months. Pain radiated to mid and lower back region for the same duration. The boy developed progressive weakness of both lower and upper limbs for one and half month and complete paralysis of both lower limbs for fifteen days. There was also frequency of micturation for one month. There was bowel involvement. There was no history of unconsciousness or seizures.

On local examination there was a swelling at the upper back region. Consistency was bony hard and it was tender.

On neurological examination, the higher mental functions were normal, speech was normal. Patient could not walk and all cranial nerves were normal.

There was gross loss of muscle bulk of both lower limbs. Muscle power was grade zero of all groups in both lower limbs and muscle power of upper limbs, all groups were grade three. Tendon reflexes were exaggerated . MRI and CT scan of cervical and dorsal spine showed there was D1 collapse and D2 retrolisthesis with cord compression at C7 and D1 (anterior and posterior) (Figure 1a, 1b, Figure 2).

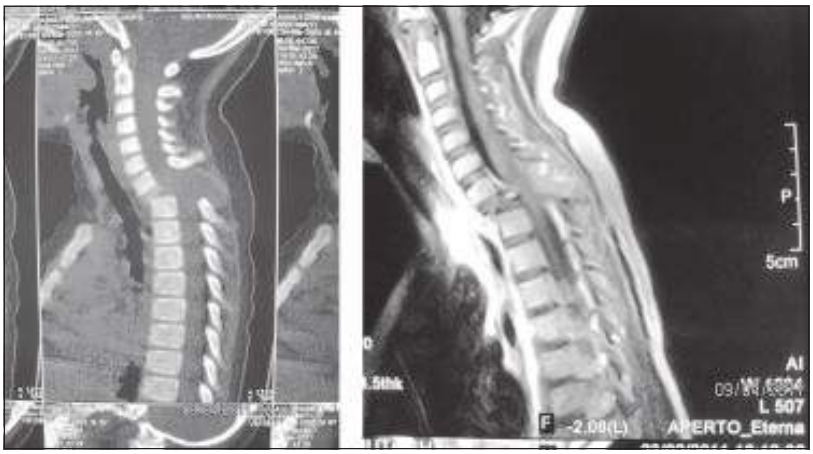

Fig.-1 a and 1 b: *Shows complete collapse of D1 and D2 retrolithiasis with cord compression from C7 to D2 level*

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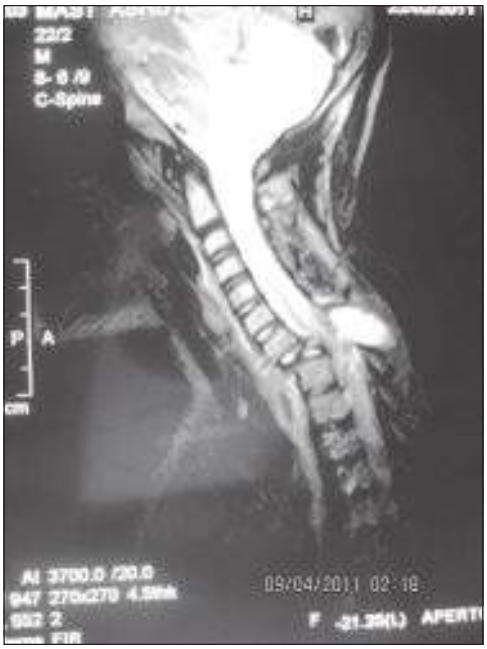

Fig.-2: *Collapse of D_1 and D_2 retrolithiasis with cord compression from C_7 to D_2 level.*

Peroperative findings were, D1 lamina and spinous process destroyed by an osteolytic mass lesion. C7 lamina and superior facet of right D2 destruction was also present. Compression on dura was present and was relieved. Tumour tissue was sent for frozen section. Frozen section showed aneurysmal bone cyst. Under image intensifier, pedicle screws were fixed bilaterally at D3, D4 and lateral mass screw were fixed bilaterally at C5, C6 (titanium, synthes). Rod (titanium, synthes) of appropriate length and appropriate shape was placed in between the above mentioned site by poly-axial screws and fixed on the D3, D4 vertebrae by screws with top nuts. Rod fixed on the C5, C6, vertebrae by screws subsequently (Figure 3a, 3b). Distraction applied and

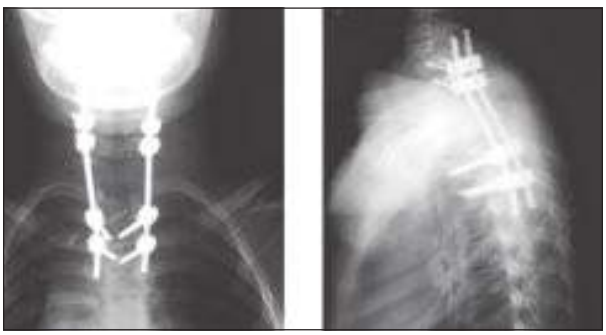

Fig.-3a and 3b: *Shows postoperative picture of upper dorsal and cervical spine fixation of cervical C5, C6, D3 and D4 with rod and screw*

screws tightened. Morselized bone graft harvested from the iliac crest and placed between C5 and D4. Paraspinal muscles loosely approximated. Sheath, fascia, subcutaneous tissue and skin closed in layers. Aseptic dressing done and patient shifted to the ICU. Histopathological examination revealed aneurysmal bone cyst. Postoperative period was uneventful. Patient's bladder symptoms improved during postoperative period. At 5 weeks follow up, the child was walking with support

Discussion:

Aneurysmal bone cyst commonly involves the metaphyses of long bones (proximal humerus, femur, tibia) and the flat bones of the pelvis. In the spine it commonly involves the neural arch.[5] The cyst is occasionally associated with other tumors of the bone, such as osteoblastoma, osteosarcoma, giant cell tumor and fibrous dysplasia[6,7] and has occasionally been reported to occur at a site of previous trauma to the bone.[8]

Rarely in advanced cases does the ABC exert pressure on the spinal cord which can lead to neurologic deficits.[9,10,11]

Clinically the patient usually presents with pain and stiffness of the spine. Palpable mass is seen in only 6% of cases. On radiographs the lesion demonstrates loss of pedicle of involved vertebrae.[12] The preferred method for evaluation of an ABC is magnetic resonance imaging which can provide much more information than a CT scan.[13]

The different diagnosis of back pain in children are other tumors of bone such as osteoblastoma, osteoid osteoma, giant cell tumor and fibrous dysplasia. Non-neoplastic causes are osteomyelitis, juvenile rheumatoid arthritis, SLE, scheuermann disease.[14]

The optimal treatment of aneurysmal bone cysts of the spine remains a subject of controversy. Treatment options for aneurysmal bone cyst have included simple curettage with or without bone grafting, complete excision, embolisation, radiation therapy or a combination of these modalities.[15,16]

If postoperative deformity develops, surgical stabilization is indicated. The clinical course of aneurysmal bone cysts is sometimes unpredictable and local recurrences have been described after various type of treatments.[17]

However, possibility of complications such as sarcomatous change, myelopathy, deformation of

vertebrae make this mode of treatment less desirable.[18]

Selective embolisation of the tumour is possible in large tumours that have high risk of bleeding and in such places where curettage would be difficult.[19] Intra-lesional injection of sclerosing agents is also an effective method for treatment of ABC. Overall cure rates of 87% have been achieved.[20]

Aneurysmal bone cyst is vascular malformation. It is the disease of adolescent. Our patient's muscle power had improved. He could walk with support. Early diagnosis and treatment reduce the patient suffering.

Conclusion:

Spinal aneurysmal bone cyst is a rare bony disorder. Cervicothoracic junction is a difficult area to operate and stabilize especially in a case of 8 years old child. Though muscle power was grade zero. Patient could walk with support after one and half months follow up.

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Mekels Cave Lipoma: A Rare Case Report

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Abstract

Mekel's cave lipoma with trigeminal neuralgia is a rare case report. Usual tumors of Mekel's cave are trigeminal schwannoma and meningoma. The Commonest cause of trigeminal neuralgia are aberrant vessel, tumor and multiple sclerosis. We report the case because there is rare association with lipoma and trigeminal neuralgia.

Key words: Lipoma, Mekel's cave, Neuralgia, Magnetic resonance imaging.

Introduction:

Incidence of lipoma, 8 in 10,000 autopsies. Usually found in or near the midsagittal plane, particularly over the corpus callosum. Lipomas in this region are frequently associated with agenesis of the corpus collasum. The tuber cinereum and quadrigeminal plate are less frequently affected.[1] Location of intracranial lipoma decreasing in order of frequency include the interhemispheric fissure, quadrigeminal cistern, suprasellar cistern, cerebellopo-pontine angle and Sylvian fissure.[2-5] The previous case report was bilateral Mekel's cave lipoma,noncontagious extention of intratentorial lipoma.[6] Another patient with intracranial lipoma involving the cerebellopontine angle, presented with diplopia and trigeminal neuralgia.[7] As lipoma is uncommon site for Mekel's cave, so we report this case and there is association with trigeminal neuralgia.

Case Report:

A 28 years old woman had a history of episodic lancinating pain at left half of face and forehead for last 5 years. Pain was increased by touching, or exertion and releaved by medication. No history of unconsciousness or difficulty in vision. No history of hearing loss. Pain was not increased by chewing of food. On examination, all general parameter of the patient were normal. Higher psychic function, speech, gait were normal. Hyperaesthesia at left V2,V3 dermatome. Other cranial nerves functions were normal. There was no limb weakness. Other motor and sensory functions were normal. MRI of brain, the T1 weighted image showed slight hyperintense lesion than cerebrospinal fluid at left Mekel's cave and T2 weighted image showed hyperintense lesion (Figure I) at the same region.

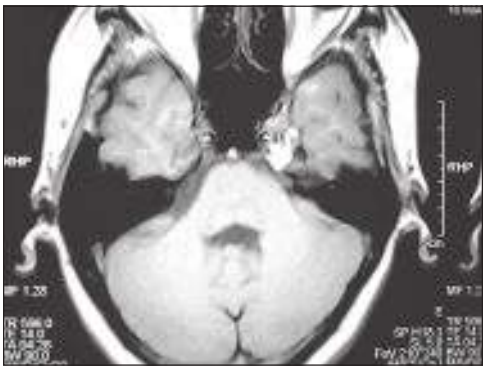

Fig.-1: *Preoperative MRI of brain shows hyperintense lesion at the left Mekel's cave*

Differential diagnosis were Mekels cave epidermoid and lipoma. An extended middle fossa approach was performed by question incision at left temporal region. Left temporal craniotomy and zygomatic ostetomy was done (Figure 2).

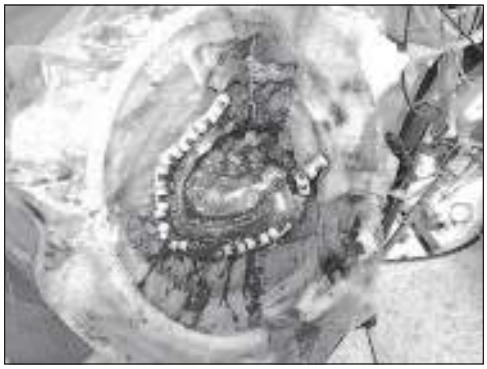

Fig.-2: *Peroperative picture of the craniotomy of the same lesion*

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Mekels cave was explored by extradural procedure. Per operative findings, there was a white yellow, glistening,avascular mass at left Mekel's cave with 5th nerve fibre splaying over it (Figure 3).

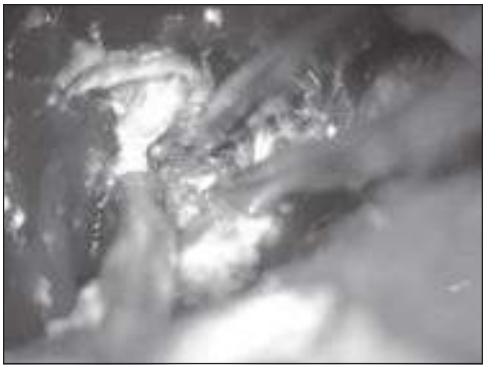

Fig.-3: *Peroperative figure shows the tumour at the Mekel's cave*

Adequate decompression was done. Histopathological report showed lipoma. Postoperative period was uneventful.

Discussion:

Intracranial lipomas are usually found incidentally on imaging or autopsy and are often associated with other developmental anomalies, such as agenesis of corpus callosum. They are located in midline structures e.g. corpus callosam, dorsal midbrain and Cerebellar vermis.[8] It may occur in isolation, but also has been described in association with a number of congenital anomalies, including, trisomy 21, Pai's syndrome, frontal encephalocele, facial anomalies. Other midline abnormalities may also be found, such as myelomeningocele and spina bifida.[9] Considering that intracranial lipomas are often associated with other congenital anomalies, such as agenesis of the corpus callosum, some authors have speculated that they are caused by neural tube derangement.[10,11] Truwit and Barkovich found that 55% of their patients with intracranial lipoms had an associated brain malformations. Although controversial and yet to be proven, embryologically, some have deposited that such tumours are due to inclusion of pluripotent mesoderm within the closing neural tube and as such do not represent hamartomas or true neoplasms but rather true congenital malformations. Generally, intracranial lipomas are benign masses that do not require surgery. Hydrocephalus is considered to be a rare consequence of such tumours and is usually due to obstruction at the cerebral aquaduct.[12] Martins et al found in their review of the literature regarding intracranial lipomas, only eight cases of intracranial lipomas had an associated extracranial component. They stressed that resection of the incranial portions of these tumours, all had catastrophic results and should not be dealt with surgically.[13] Sari et al[14] described a child with an interhemispheric lipoma that extended into the choroid plexus of the lateral ventricles and that was also associated with an extension of lipoma into the anterior fontanelle.

Rare tumour occur at intracranial region and can produce clinical symptoms and signs. Our case was an unique presentation. There were very few case reports in the world literature about the Mekel's cave lipoma.

Conclusion:

We have presented the case as it is a rare space occupying lesion at the Mekel's cave and also present as trigeminal neuralgia. So this will bring attention to world medical scientist. Tumour can occur at any time within the intracranial space and clinical features should not be ignored without definite investigation.

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Paroxysmal Tonic Spasm, a Frequent Manifestation in Patients with Multiple Sclerosis

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Abstract:

Objectives: *Paroxysmal tonic spasm, a poorly understood manifestation, is frequently seen in disseminated sclerosis among patients from Japan, Taiwan and other countries. We carried out this study to find out its incidence in patients suffering from multiple sclerosis in Bangladesh.* ***Methods:*** *The study was carried out in the Department of Neurology, Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka, during January 2006 and December 2008. Thirty five patients were included in this study. All patients were included by history, physical examination and laboratory tests with radiological investigations and following McDonald diagnostic criteria for multiple sclerosis. Clinical details and investigations of the patients were reviewed. Data were recorded in predesigned data collection sheet and analysis done using computerbased software (SPSS).* ***Results:*** *Out of 35 patients, 16 (45.71%) were male and 19 (54.29%) female and male & female ratio was 1:1.19. Most of the patients presented at second, third and fourth decades of life. painful or paroxysmal tonic spasm/seizure was a prominent feature affecting 11 (31.43%) patients. Most of them were precipitated by sensory stimulation or trunkal movement in 8 (72.73%) out of 11 paroxysmal tonic spasm patients.* ***Conclusion:*** *Paroxysmal tonic spasm is a common feature of multiple sclerosis patients in Bangladesh and clinical guidelines for therapeutic approaches are lacking because the mechanism of pain in multiple sclerosis patients has yet to be determined. For this, further large scale studies should be carried out.*

Key words: *Multiple sclerosis (MS), Proxysmal tonic spasus (PTS)*

Introduction:

Multiple sclerosis (MS) is an inflammatory demyelinating disease of the central nervous system, which usually begins at early adult life and pursues a variable course causing significant morbidity. MS patients have been known to present with several forms of paroxysmal symptoms and may be precipitated by exercise, hot baths, smoking and emotions[1], neck flexion[2] and eye movement[3]. The nature of the paroxysmal symptoms depends upon their site of origin, which is usually in the brainstem or spinal cord.

The paroxysmal symptoms are paroxysmal tonic spasm (PTS), paroxysmal ataxia and dysarthria, paroxysmal paresthesia, itch and pain, trigeminal neuralgia and paroxysmal akinesia. The probable mechanism underlying tonic spasm is ectopic impulse generation in demyelinated corticospinal axons spreading radially to adjacent, closely packed, demyelinated fibre by ephaptic ('false synaptic') transmission.

We look for PTS described as cramping and pulling pain triggered by movements or sensory stimuli affecting both upper and lower limbs, although more often the lower limbs. This type of pain is often nocturnal and associated with spasticity but is also seen in patients with mild disability.

Materials and Methods:

This prospective study was carried out in the Department of Neurology, Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka, from indoor and outdoor patients, during the period from January 2002

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to December 2008. A total of 35 patients were assessed, investigated and diagnosed having MS, who fulfilled the McDonald diagnostic criteria for MS[4]. Patients with recent vaccination, viral infection, systemic lupus erythematosus (SLE), rheumatoid arthritis (RA), sarcoidosis and other collagen vascular disease, neoplasm, cervical spondylosis, compressive myelopathy and metabolic disorders were excluded from the study. Thorough medical history was taken, physical examinations and relevant investigations, specially MRI of brain and spinal cord with contrast were done. Necessary laboratory procedure were done to ascertain exclusion criteria. Informed consents were taken from each patient before his/her inclusion in the study. A well planned data sheet was formulated for record keeping, that included examiner administered questionnaire concerning demographic data, clinical examination and investigation findings. Follow up was done every three month interval or as required in the Department of Neurology, BSMMU and subsequent data were recorded in the data collection sheet. Data were analyzed by computer using statistical software.

Results:

The age range of 35 patients of MS was 10 65 years, with mean (±SD) 30.56±13.44 years. The study included 12 (34.29%) patients ?20 years, 18 (51.43%) 21 40 years, 4 (11.43%) 41 60 years and only 1 (2.86%) >60 years. The peak age incidence of MS was found in 21 40 years age group (Table I).

Table I

Age distribution of the patients (n=35)

Age (years)	Frequency	Percentage
<20	12	34.29
21 40	18	51.43
41 60	4	11.43
>60	1	2.86

Out of 35 patients of MS, 16 (45.71%) were male and 19 (54.29%) female. The male female ratio was 1:1.19 (Table II).

Table II

Sex distribution of the patients (n=35)

Sex	Frequency	Percentage
Male	16	45.71
Female	19	54.29

Table III shows the course of the disease among the patients. 22 (62.86%) patients had relapsing remitting course, 10 (28.57%) had secondary progressive course and 3 (8.57%) had primary progressive multiple sclerosis.

Table III

Distribution of the patients according to course of the disease (n=35)

Disease course	Frequency	Percentage
Relapsing remitting MS	22	62.86
Secondary progressive MS	10	28.57
Primary progressive MS	3	8.57

The paroxysmal symptoms were summarized in Table IV. Out of 35 patients, 24 (68.57%) had paroxysmal paresthesia, 12 (34.29%) had paroxysmal ataxia and dysarthria, and 11 (31.43%) had paroxysmal tonic spasm. Symptoms like trigeminal neuralgia, and itch and pain were found in 1 (2.86%) patient each.

Table V shows the effect of sensory stimulation for precipitation of paroxysmal tonic spasm. Eight patients (72.73%) out of 11 patients suffered paroxysmal tonic spasm by movement or sensory stimulation. Three patients (27.26%) with PTS not pricipited by sensory stimulation or truncal movement.

Table IV

Distribution of the patients according to paroxysmal symptoms (n=35)

Paroxysmal symptoms	Frequency	Percentage
Paroxysmal paresthesia	24	68.57
Paroxysmal ataxia and dysarthria	12	34.29
Paroxysmal tonic spasm	11	31.43
Trigeminal neuralgia	1	2.86
Itch and pain	1	2.86

Table V

Effect of sensory stimulation for precipitation of paroxysmal tonic spasm (n=11)

Effect	Frequency	Percentage
Precipitated by sensory stimulation or trunkal movement	8	72.73
Not precipitated by sensory stimulation or trunkal movement	3	27.27

Discussion:

The present study was carried out to determine paroxysmal symptoms, specially paroxysmal tonic spasm, of MS patients. The study patients were taken from the Department of Neurology (indoor and outdoor), Bangabandhu Sheikh Mujib Medical University, Dhaka.

In this study, 16 (45.71%) were male and 19 (54.29%) female (ratio 1;1.19). There was no marked female preponderance like other countries. The male female ratio in various countries are USA 1:1.8[5], Northern Ireland 1:1.3[6], US Army 1:1.8[7], Japan 1:1.3[8], Taiwan 1:1[9], Hawaii 1:3.2[10], India 1:3[11] and Thailand 1:3[12]. The male female ratio is higher in our country than western and oriental countries. Predominant male health seeking pattern and less allocation of hospital bed for female patients may explain this situation.

The striking feature of this study was the high incidence of painful tonic seizure (PTS), specially in case with severe spinal cord involvement. The phenomenon was seen in 11 (31.43%) patients out of 35 MS patients. PTS in MS were first described by Matthews[13], and since then several cases with various types of PTS have been documented by others. Shibasaki and Kuroiwa[14] found PTS in 11 (17.19%) patients in a consecutive series of 64 patients with MS, a much higher incidence than has been found in western countries[7]. In Thailand, Jitpimolmard and Vejjajiva[12] observed no less than 3 (20%) out of 15 patients with MS. This study found that PTS was relatively frequent among MS patients in Bangladesh like other Asian countries where cases with severe spinal cord involvement are more frequent.

In our study, 8 (72.73%) patients out of 11 had precipitating factors, the majority being tactile stimulation or truncal movements.

Conclusion:

Paroxysmal tonic spasm is a frequent and common presenting feature of patients with multiple sclerosis. This study shows such presentation in the multiple sclerosis patients in Bangladesh. However of mechanismsuch paroxysmal tonic spasm was not fully understood. So, therapeutic approach for relieving much PTS was eacking. So, a large scale study with different modalities of treatment should be carried out. So that a guideline of management of such patients can be found in future.

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REVIEW ARTICLE

A Review of Intracranial Dural Arteriovenous Fistula

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Abstract:

Dural arteiovenous malformation is an abnormal connection between arteries and veins. Digital Substraction Angiography(DSA) is the gold standard for diagnosis. Endovascular treatment is a good therapeutic option. This article reviews classification, etiology, investigations, and different therapeutic modalities.

Key words: *Dural arteriovenous fistula (DAVF), Dural arteriovenous malformation. Onyx.*

Abbreviation: *CVR (cortial venous reflux), DSA (digital substraction angiography), MRA (magnetic resonance angiography), MRI (magnetic resonance imaging).*

Introduction:

Early descriptions of cranial DAVFs date from the first decades of the 20th century, typically in the form of single case reports, such as by Tönnis in 1936 [1]. The first presentation of the concept of dural fistulas originates from a publication by Fincher in 1951 [2]. Despite the development of cerebral angiography in the late 1920s [3], it took nearly 40 years before the DAVF emerged as a distinct entity. In those days, all DAVFs were regarded as benign in comparison with the pial arteriovenous malformations [4].A dural arteriovenous fistula, or dural arteriovenous malformation, is an abnormal connection (fistula) between arteries in the dural covering of the brain and spinal cord with veins or venous sinuses. Normally, arteries carry blood to tissues where they divide into smaller and smaller capillaries before returning to veins and venous sinuses which collect blood and direct it back to the heart. In these lesions there is no capillary bed and the arteries connect directly to the veins or venous sinuses. Venous sinuses are large channels of venous blood that occur around the brain and normally collect the venous blood which comes from the brain. In the case of the dural arteriovenous fistula, the arteries in question are called meningeal arteries because they normally feed the meninges, or the coverings of the brain and spinal cord.

This abnormal connection of an artery and a vein leads to high blood flow through the fistula which produces abnormally high pressures in the vein or sinus which can lead to the clinical manifestations discussed below. These fistulas vary in their size, location and the type of venous channel that they drain into. They comprise 10% of all intracranial vascular malformations.[5] Nevertheless, as a unique neuropathological entity, the subject DAVF deserves full attention. A DAVF consists of one or more true fistulas,i.e., direct AV connections without an intermediate capillary network or even a nidus. The fistula itself is confined to the leaflets of the dura mater, which unquestionably differentiates a DAVF from an arteriovenous malformation (AVM) that has a (sub)pial localization within the brain or spinal cord. In addition, considering the general acknowledgment that a DAVF is acquired [6–9], in contrast to the congenital nature of a vascular malformation [10], the term ''dural arteriovenous fistula'' is clearly preferable to ''dural arteriovenous malformation.'' The anatomical setting of the fistula within the dura mater explains the fact that both cranial and spinal DAVFs are recognized. Although their underlying pathophysiology is the same, their clinical presentation and behavior (and therefore their classification) are quite distinct.

Classification:

Two classification systems are in common use and both have been validated as predictive of risk of hemorrhage[11]. There are Berden and Cognard Classifications.

Borden classification[12]

I. Venous drainage directly into dural venous sinus or meningeal vein

II. Venous drainage into dural venous sinus with CVR

III. Venous drainage directly into subarachnoid veins (CVR only)

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Cognard classification:[13]

I. Venous drainage into dural venous sinus with antegrade flow

IIa. Venous drainage into dural venous sinus with retrograde flow

IIb. Venous drainage into dural venous sinus with antegrade flow and CVR

IIc. Venous drainage into dural venous sinus with retrograde flow and CVR

III. Venous drainage directly into subarachnoid veins (CVR only)

IV. Type III with venous ectasias of the draining subarachnoid veins

V. Venous drainage into the perimedullary plexus

Etiology:

The cause of DAVFs is still a matter of debate, although many causes have been posited in the literature. The development of DAVFs has been described following surgery (even in remote parts of the body) or head trauma and in relation to sinus thrombosis, without revealing a definite etiological pathway. Over the decades, two main hypotheses of pathogenesis have been advanced. The first postulates that DAVFs normally exist within the dura mater as ''dormant'' channels between the meningeal arterial circulation and the venous system. Histological and radiological studies have demonstrated that these communications are indeed present in the dura of normal individuals [14]. According to this theory, the channels open because of the venous hypertension associated with sinus thrombosis or sinus outflow obstruction [15]. Similarly, the existence of thin-walled venous pouches close to small meningeal arteries has been reported. Rupture of these fragile pouches might be responsible for direct AV communications within the dura [16]. The second hypothesis claims that the development of DAVFs is a direct consequence of neovascularization processes within the dura mater, attributable to the release of angiogenetic factors. These factors, e.g., vascular endothelial growth factor and basic fibroblast growth factor, can be either directly produced by the organization of a venous sinus thrombosis or indirectly induced by the increased intraluminal venous pressure through a tissue hypoxia pathway. Major support for the second hypothesis arose from the positive staining of excised dural fistulas for venous thrombosis [17] and angiogenetic factors [18].Histopathological studies prove that DAVFs are located within the meningeal wall of a venous sinus, although originally they had been described to reside within the thrombosed lumen of a dural sinus. The location within the wall clarifies the existence of the type of DAVF that drains directly into the cortical venous network, without venous drainage into the involved sinus [19]. On close examination, the fistula consists of small venules with a diameter of approximately 30 mm. These vessels have been named ''crack-like vessels,'' since they resemble cracks in the wall of the dural sinus after histological staining. Further immuno-histochemical assessment of the crack-like vessels revealed a layer of endothelial and smooth muscle cells and no internal elastic lamina, thus confirming their venous origin [20].

Clinical presentation: Symptoms and physical findings are highly variable, and depend on the location and anatomy of the lesion Overall, pulsatile tinnitus is the most common symptom and is present in about 60% of patients.[21] Bruit is present in about 50% of patients.[21]Hydrocephalus may be present and is attributable to venous hypertension in the superior sagittal sinus, interfering with CSF absorption.[22] Significant symptoms from DAVFs are generally divided into those attributable to hemorrhage or "non-hemorrhagic neurological defects,[23,24] which are usually due to intracranial venous hypertension. In a prospective study of intracranial hemorrhage due to an intracranial vascular malformation (brain AVM, cavernoma, or DAVF), a DAVF was the underlying cause in 6.4% of cases.[25] Followings are the risk factors for hemorrhage,[22,26,27]

1. Leptomeningeal venous drainage
2. Variceal or aneurysmal venous dilatations
3. Galenic drainage
4. Stenosis or occlusion of associated venous sinuses
5. Location in the anterior fossa, middle fossa, or tentorial incisura

Case fatality rate with hemorrhage is 20%[21,23]

Intracranial venous hypertension: The intracranial venous system is valveless and so elevated pressure within arterialized venous sinuses and/or intracranial veins may be transmitted throughout the intracranial venous system. Diffuse white matter changes on MRI due to venous congestion may be seen.[28,29]

Neurological symptoms attributable to elevated intracranial venous pressure includes, Progressive dementia (venous hypertensive encephalopathy, venous congestive encephalopathy, or progressive cognitive impairment)[22,30–33], Pseudotumor cerebri[34] and Parkinsonism[35]

Investigations:

Digital Subtraction Angiography

Catheter angiography is the best technique for the diagnosis and planning of an intracranial DAVF.[36,37] In addition to diagnosis, anatomical and haemodynamic assessment of the fistula needs to be performed. The aim is not only to identify the arterial feeders and site of fistula but also to identify the pattern and direction of venous drainage of the fistula and normal cerebral parenchyma. Selective injection of all potential arteries that may contribute to the region of dural pathology needs to be done. The acquisition of images should begin in the early arterial phase and continue in the late venous phase of the study. A dural arterial injection (contrast angiogram) may be required to study the venous drainage of of fistula and normal brain. In this technique first selective injection is done in ICA followed by ECA. Then by using resubstraction and changing of the mask runs, drainage of the fistula and and normal brain parenchyma may be superimposed. The angiogram should be analysed for feeders, type of venous drainage, retrograde flow , occlusion of sinuses, and circulation time.The common angiographic findings seen are feeders from pial and dural branches, AV shunting, venous ectasia, stenosis, calcifications, impaired drainage of cerebral parenchyma with delayed venous drainage and cortical venous ectasia.

Computed tomography of Brain

In the absence of CVR with congestion of the brain, benign DAVFs are nearly always occult on CT imaging. In case of an aggressive DAVF, unenhanced CT may show hypodensities, representing areas of edema or venous ischemia. Abnormally enlarged pial veins can sometimes be visualized as increased densities in comparison to the brain parenchyma. Contrast-enhanced CT shows enhancement of the refluxing cortical venous network. A promising technique is the development of 3D CT angiography, especially in emergencies. The disadvantage of 3D CT angiography is its poor characterization of hemodynamic details.

Magnetic resonance Imaging (MRI) and Magnetic resonance angiography (MRA) of Brain

MRI may not show some DAVFs and should not be used instead of catheter angiography to exclude the presence of a DAVF. A normal MRI does not exclude the presence of a DAVF. On MRI, it is difficult to detect a benign DAVF, although irregular or stenotic venous sinus might raise suspicion. MRA is more sensitive, but still has limitations in depicting the fistula. Aggressive DAVFs are better visualized by MRI, characterized by flow voids on the cortex corresponding to dilated pial vessels. The brain parenchyma can show T2 hyperintensity in the white matter secondary to the venous congestion of the brain, especially in the deep white matter [38]. The differential diagnosis for T2 hyperintensity includes sinus thrombosis (with venous infarction or venous congestion), demyelinization, and neoplasm. However, T2 hyperintensity in the parenchyma in combination with a surplus of pial vessels is highly suggestive of a vascular malformation). This T2 hyperintensity resolves after treatment .

Natural history

The overall annual risk of hemorrhage in patients with intracranial DAVFs is 1.8%, with a case fatality rate of 20% with hemorrhage.[21] However, the natural history of DAVFs depends strongly on the pattern of venous drainage. Most Borden Type I lesions (Cognard Type I or IIa) are considered to be “benign” whereas higher grade lesions are “aggressive.

Management

Management options for patients with a DAVF are

1. Conservative management
2. Endovascular techniques
3. Surgery
4. Radiosurgery
5. A combination of endovascular treatment, radiosurgery, and/or surgery

1. Conservative management

Conservative management (i.e., no endovascular or surgical procedure, with or without surveillance imaging) is reasonable in certain situations. Asymptomatic or minimally symptomatic Borden Type I lesions without evidence of cortical venous drainage may be managed expectantly. It is well established that spontaneous regression of DAVFs occurs in some cases. This is particularly true of cavernous sinus DAVFs,[39,40] in which spontaneous regression has been reported in up to 73% of cases.[41-43]

Intermittent manual compression is also effective for some patients with cavernous sinus or transverse-sigmoid lesions.[44-45]

2. Endovascular management:
There are two types of approach for endovascular management.

A.Transarterial approach

B.Transvenous approach

A.Transarterial approach:

1. Particulate embolization: particles of different sizes is used for embolization of branches of external carotid artery. It is not curative. Total obliteration is difficult to achieve with this method because some feeding arteries cannot be catheterized and because of the recruitment of blood supply from collateral arteries[46]. Figure 1-6 shows how the particle obliterate the dural arteriovenous fistula in a case of cavernoclival fistula.
2. N- butyl-2-cyanoacrylate or onyx embolization: Deposition of of glue into the collecting vein or fistula through the transarterial route may result in cure. NBCA has been the agent of choice,[47] Because of its resistance to recanalization. Onyx may be a good alternative.[48].

B. Transvenous approach: In these procedure lyps. Particles are used.

1. Venous embolization should be done only when a venous drainage pouch that is separate from veins draining normal brain tissue can be identified and accessed with a microcatheter.[47] When embolization is limited to the venous outlet immediately adjacent to the nidus, with preservation of functional drainage, Venous embolization with platinum coils can provide adequate treatment.

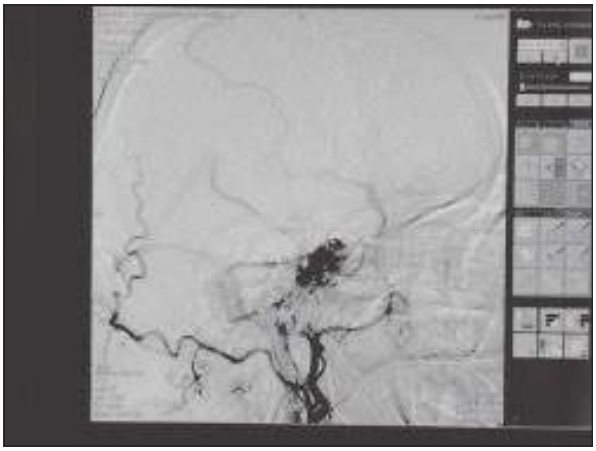

Fig.-1: *A DURAL AVF (caverno clival fistula) of a 56 years old male. ECA injection reveals feeding artey is ascending pharyngeal, middle meningeal and accessory meningeal artey.*

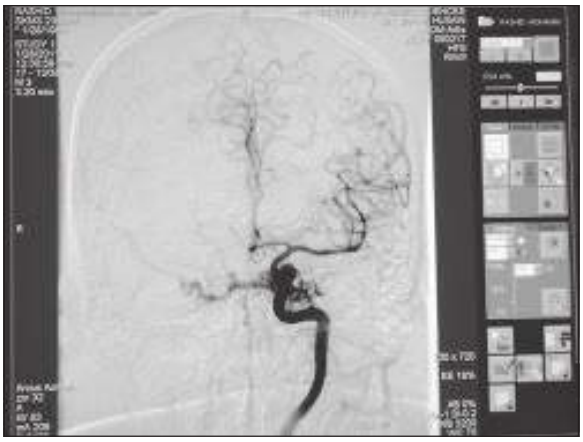

Fig.-2: *Left ICA injection reveals indirect carotico cavernous fistula.*

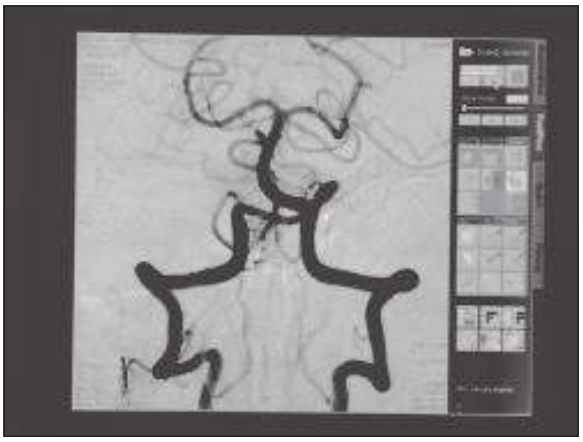

Fig.-3: *small contribution from both vertebral artey to caverno clival fistula.*

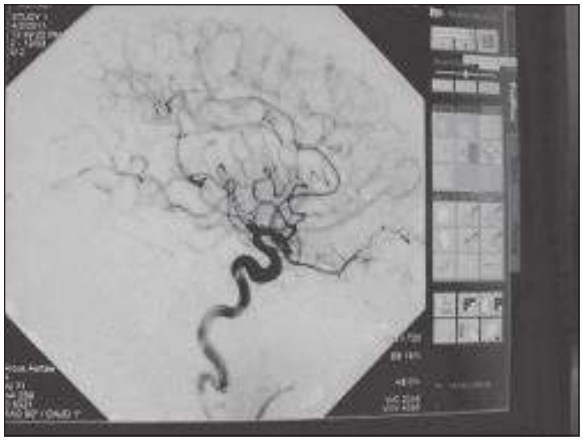

Fig.-4: *After treatment with PVA particles internal carotid injection reveals there was no evidence of fistula.*

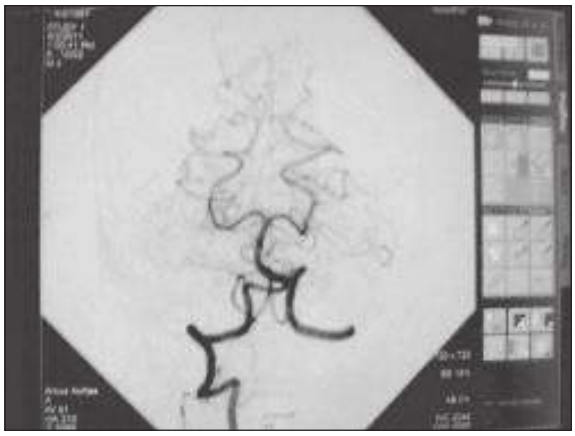

Fig.-5: *LVA injection also reveals no contribution from vertebral artey.*

3. Surgery:

Surgery for intracranial DAVFs has evolved considerably over the last three decades, from simple ligature of feeding vessels (which produced success rates of only 0–8%)[50] to blood-soaked fistula resection and packing of the venous sinus,[51]or to more elegant interruption of the draining vein, when the anatomy is favorable.[52] Although endovascular techniques are presently first-line treatments for many dAVFs, surgery remains standard for anterior fossa dAVFs.[50]

Radiosurgery

Preliminary reports of radiosurgery for intracranial dAVFs are encouraging.[53,54] In many cases embolization is combined with radiosurgery.[55] Overall rates of lesion obliteration are 58–77%.[55,56,57]

Conclusion:

Dural arteriovenous malformation which causes differenet type of neurological sequelae and even death. Along with endovascular approach, Surgical and Radiosurgical approach are also available for treatment of DAVF. Due to availability of different treatment modalities most of the lesions are now treatable with good prognosis.

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Study of Stroke and its Risk Factors Among Admitted Patients in A Tertiary Level Hospital

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Abstract:

Objective: *To observe the type of stroke and identify its association with the risk factors in northern part of the country.* ***Materials and methods:*** *500 (five hundred) stroke patients with CT scan of head were studied prospectively in the neurology department of Rajshahi Medical College Hospital from July 2004 to June 2007.* ***Results:*** *305 patients (61%) were male and remaining 195 patients (39%) were female. 16% were young stroke (age<40 years) and most patients (54%) were at and above 60 years of age. About 38% of strokes were of hemorrhagic and remaining 62% were of ischemic. Hypertension, ischemic heart disease (IHD) and smoking appeared as the most common risk factors of stroke followed by diabetes mellitus, dyslipidemia and obesity. Multiple risk factors were present in 55% of patients.* ***Conclusion:*** *Stroke types and its association with risk factors are similar with other parts of the country.*

Key words: *Stroke, Type, Risk factors*

Introduction:

WHO defines stroke as, "rapidly developing clinical signs of focal (or global) disturbance of cerebral function, with symptoms lasting 24 hours or longer or leading to death, with no apparent cause other than of vascular"[1].

Cerebrovascular disease is the second leading cause of single organ death above the age of 40 years in the world[2]. It accounts for 5 million deaths and another 15 million people suffers from non fatal stroke in a year, worldwide. About 700,000 Americans each year suffer a new or recurrent stroke. That means, on average, a stroke occurs every 45 seconds. Each year, approximately 60,000 more women than men have a stroke (GCNKSS, NINDS) [3]. However, Among men stroke incidence rates are greater than that of women at younger age but not at older age [4]. Blacks continue to have a higher stroke incidence than Whites, especially among the young. Despite advances in stroke prevention treatments during the 1990s, the incidence of hospitalized stroke did not decrease within the population being studied. Case fatality also did not change between study periods.

At about every 3 minutes, someone dies of stroke. WHO estimates, death from stroke in developing countries in 2005 accounted for 87% of stroke deaths worldwide. The number of disability-adjusted life years (DALYs) in these countries was almost 7 times that in developed countries [5].

There are several important modifiable risk factors (hypertension, diabetes mellitus, tobacco smoking and obesity) which contribute to its pathophysiology. Blood Pressure (BP) is a powerful determinant of stroke risk. Subjects with BP <120/80 mm Hg have approximately half the lifetime risk of stroke than the subjects with hypertension [6]. High BP is the biggest risk factor for stroke [7]. Incidence of stoke doubles with each 7.5 mmHg rise in diastolic pressure in western countries and 5 mmHg in Japanese and Chinese population [8-9]. Mild hypertension is of greatest risk due to its higher prevalence in the community than other risk factors. The association of stroke and high blood pressure is less in the elderly than in the middle aged group[10]. Hypertension probably increases risk of stroke by increasing extent and severity of atheroma and the prevalence of small

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vessels disease in the perforating arteries within the brain[11-13]. In Bangladesh hypertension has been found to be significantly more associated with haemorrhagic stroke than with ischemic stroke. A major portion of stroke sufferers in Bangladesh take antihypertensive drugs irregularly[14-15].

In contrast to coronary heart disease, initial studies found no overall association between plasma cholesterol concentration and stroke. Several more recent studies have found that plasma lipids and lipoproteins affect the risk of ischaemic stroke, but the exact relationship are still need to be clarified. Low high-density lipoprotein (HDL) is a risk factor for ischaemic stroke in men, but more data are needed to determine its effect in women[16]. Potential sources of embolism from the heart are associated with an increased risk of stroke. Atrial fibrillation is by far the most important because it is very common, carries a relatively high risk of stroke. In recent years identification of new risk factors for vascular diseases, including stroke, have been attempted by many investigators. Most factors are thought to operate by accelerating atherosclerosis. They include infections, inflammatory and rheological markers, plasma homocysteine concentration and various genetic polymorphisms[17]. The importance of any risk factor will depend upon both its relative risk and the prevalence of that risk factor in the population.

Stroke is largely preventable. However, it remains a challenge, to implement effective preventive programs in the population. This study was an attempt to see the spectrum of stroke with its common risk factors among the patients admitted in a tertiary level hospital representing rural population of the northern part of our country.

Materials and Methods:

This was a prospective study done in the neurology department of Rajshahi Medical College Hospital from July 2004 to June 2007. A total of 500 (five hundred) stroke patients with CT scan of head were included in this study. Detailed history of present illness, relevant past illness and personal history were noted carefully. A thorough physical examination was done and information were collected in a preformed data sheet. BMI was measured by using standard formula. Routine investigations including CBC, urinalysis, serum lipid profile, random blood sugar, serum creatinine, ECG were done. Serum electrolytes and echocardiography were requested whenever necessary depending on the patients' condition. Diabetes is diagnosed on the basis of WHO criteria. Patients who died or left the hospital before completing investigations were excluded from the study. After completion of the study, data were analyzed and arranged in tables.

Result:

Among five hundred (500) "brain stroke" patients, 305 patients (61%) were male and remaining 195 patients (39%) were female. A substantial number of patients (80) were below the age of 40 years (16%). Most of the patients (54%) were at and above 60 years of age (table-I).

Out of the 500 hundred cases 188 (37.5%) patients developed hemorrhagic stroke and 312 (62.5%) patients suffered an ischemic attack. Table-II shows incidence of stroke in relation to age, sex and type of stroke. This table came out with the findings that ischemic and hemorrhagic stroke is equally prevalent among 60-69 years age group, and ischemic stroke is more common among 40-49 years, 50-59 years and >70 years age group. Especially mentionable regarding <40 age group, hemorrhagic stroke incidence was higher in males but ischemic stroke in females.

Table-I

Age and Sex distribution of study population

Age in years	Male	Female	Total	%
< 40	45 (15%)	35 (18%)	80	16
40-49	50 (16%)	46 (24%)	96	19
50-59	40 (13%)	15 (8%)	55	11
60-69	94 (31%)	53 (27%)	147	30
>70	76 (25%)	46 (24%)	122	24
	305 (61%)	195 (39%)	500	100%

Table-II

Type of stroke in relation to age

Pattern	<40		40-49		50-59		60-69		>70	
	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female
Haemorrhagic	32(10%)	8(4%)	10(3%)	8(4%)	16(5%)	6(3%)	50(16%)	26(13%)	20(7%)	12(6%)
Ischemic	13(4.5%)	27(14%)	40(13%)	38(19%)	24(8%)	9(5%)	44(14%)	27(14%)	56(18%)	34(17%)

Among the study population 67% (356) were poor and only 4% (20) patients were from affluent families.

163 (54%) male patients were smoker while only 3 (1.5%) females were found smoker (fig-1). Obesity was predominant in female as 92 (47%) were female and 79 (26%) male were considered obese (fig-2).

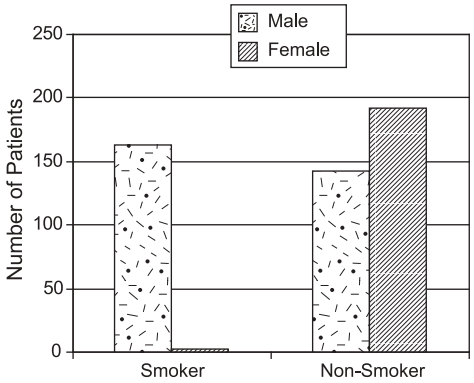

Fig.-1: *Bar Chart Showing smoking habit of study population*

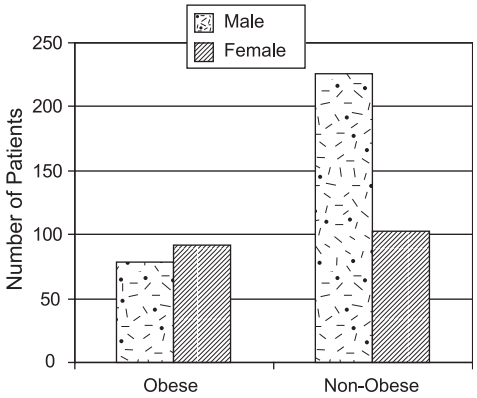

Fig.-2: *Bar Chart showing nutritional status of patients*

In fig-3 levels of blood pressure in this study revealed that, 284 (57%) patients were known to have hypertensive while 124 (25%) were unaware of their high blood pressure and 92 (18%) were found to be normotensive. Out of 284 hypertensive patients only 72 (25%) patients take their medicines regularly while remaining 212 (75%) patients undergo irregular medications.

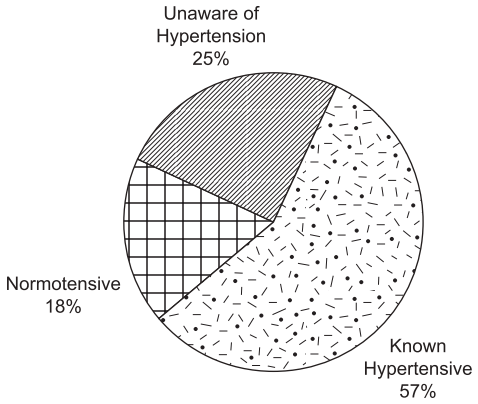

Fig.-3: *Pie chart showing status of blood pressure in study population*

Out of 188 hemorrhagic stroke cases, 100 (53%) patients were known hypertensive. However, only 15 (15%) patients were taking antihypertensive on regular basis. While among 312 ischemic stroke patients 184 (59%) were known hypertensive and of this 57 (31%) patients were complaint to their treatment (fig-4).

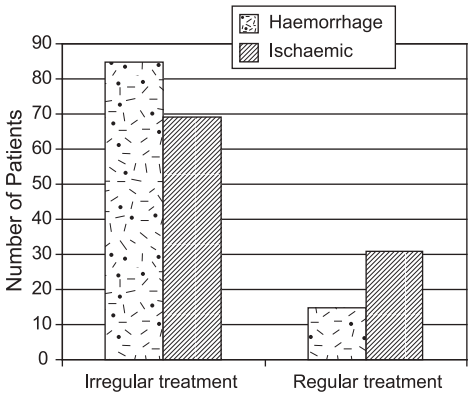

Fig.-4: *Bar chart showing type of stroke among hypertensive patients.*

Fig-5 shows around 20% patients are found to be diabetic (12% are aware and 8% are unaware of diabetes) while remainder were non diabetic. Dyslipidemia was present in 137 (27.4%) patients and slightly more common in ischemic group [(28.5% vs. 25.5%)] (Fig-6).

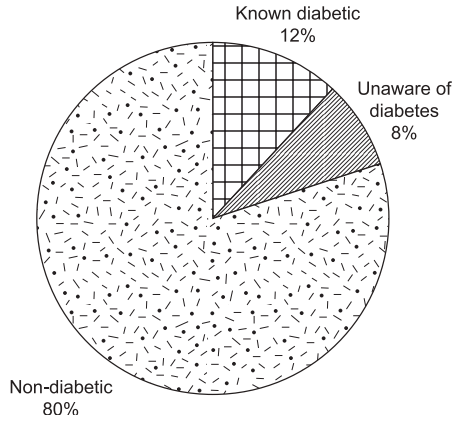

Fig.-5: *Pie chart showing incidence of Diabetes mellitus in study population*

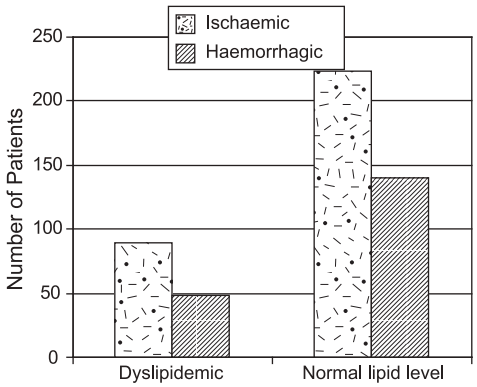

Fig.-6: *showed pattern of lipid profile in study population.*

In fig- 7 associations of stroke with ischemic heart disease (IHD) and valvular heart disease has been presented. History and investigations revealed IHD was present in 103 patients with ischemic stroke and 52 patients with haemorrhagic stroke. Number of the study subjects known to have valvular heart disease is 25 in ischemic group and 2 in haemorrhagic group.

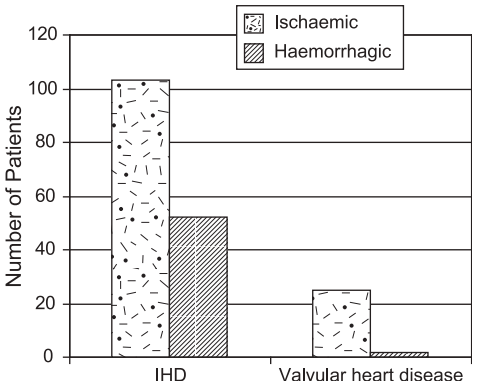

Fig.-7: *Showing association of stroke with IHD and Valvular heart disease*

Risk factors were absent in 145 patients, single factor was present in 79 (16%) patients and multiple risk factors were present in 276 (55%) patients (Fig-8).

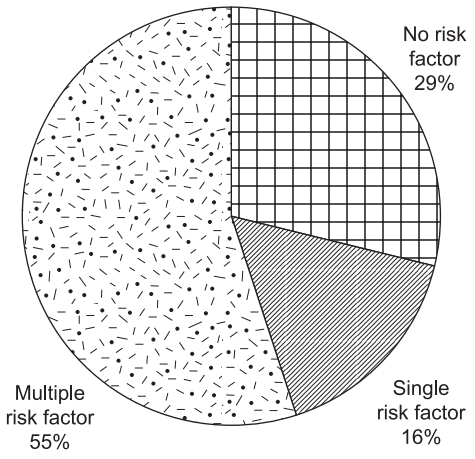

Fig.-8: *Pie chart showing association of stroke and modifiable risk factors*

Discussion:

Stroke is one of the very common neurological causes of hospital admission in our country. This study is an effort to see the spectrum of stroke among the affected patients in a government medical college hospital along with the assessment of risk factors that were usually associated with it.

Out of 500 stroke patients 305 (61%) are male and 195 (39%) are female. Most of the patients are above 60 years of age (269, 53.8%). This finding is similar with some studies but on the other hand opposes with a pretty high figure with other studies [14, 18-19].The 40-49 age group showed a substantial number of stroke incidence 96, (19%) with significant high incidence of ischemic stroke [78 (25%, vs. 18 (9.5)]. As a significant number of patients (80, 16%) were below the age of 40, they need separate and specific attention to explore the underlying etiopathology. A higher incidence of stroke in female [81(42%)) vs. 95 (31%)] in below 50 age group shows similarity with the finding published in 2003 [15] but shows a drastic incline while compared with another study [19]. This may be explained by a high incidence of rheumatic heart disease as sequelae of inadequate treatment of rheumatic fever in low income group.

In this present series 188 (38%) patients had hemorrhagic stroke and 312 (62%) patients suffered from ischemic stroke. This finding does not match

with the figures given in standard text book [24] and in a study done by Hayee et al [19] but similar to other studies [14, 20]. Ischemic stroke is more common in female (42% vs. 31%) before the age of 50, while hemorrhagic stroke is more common in male after the age 50 (69% vs. 58).

A high blood pressure is recorded in 408 (81.6%) patients on admission and blood pressure is found to be normal in the remaining cases (18%). Among the hypertensive patients 69.60% (284) were known hypertensive and 124 (30.40%) were not aware of their high blood pressure. The data showed similarity with certain study [14] but presented with a raised trend when compared with other studies [15, 18-19]. In earlier observational studies it was found that one fourth to three fourth of our hypertensive patients were ignorant of their high blood pressure [21-22]. It seems that the condition has improved, however, a significant proportion of patients (30%) are still not aware of their hypertensive status.

Among hypertensive patients one in every four patients (25%) receives medications on regular basis. Reception rate of antihypertensive therapy is less than those found in a study [14] but more than other study [15].

This study came out with the finding that hemorrhagic stroke is common in irregularly treated hypertensive group while ischemic stroke is more in regularly treated hypertensive patients. This observation is similar to a study by Jahan S et al [14].

103 (33%) patients with ischemic stroke in this study are found to have evidence of ischemic heart disease (known and/or detected in ECG). 25 (8%) patients in ischemic stroke group have evidence of valvular heart disease clinically, which later confirmed by echocardiography.

Diabetes mellitus is found in 99 (20%) patients. And of these 99 patients only 57 (12%) patients were known diabetic and the remaining patients are labeled as diabetic after admission. This observation is higher than other studies [14-15, 18-20] and seeks further exploration.

Dyslipidemia is present in 89 (28.5%) patients of ischemic stroke and 48 (25.5%) cases of hemorrhagic stroke. This finding is almost similar with other studies [14, 18-19]. Prevalence of dyslipidemia does not differ significantly between the groups of stroke.

Among the study subjects, 67% belonged to poor socio-economic class and only 4% patients are affluent. This reflects a negative tendency of patients from well to do families to receive services from government hospitals even in a life threatening condition like stroke. Same finding was established by Arif and his co-workers[15]; however, current figure is lower than other study [14]. Whether this high income group is dissatisfied with the services of government hospitals or any other region present behind this negative attitude, it needs to be analyzed with great importance.

Regarding smoking habit 54% male patients were found to be smoker showing similarity with some studies [14, 18-19] despite of country wide campaign against smoking in the recent years.

Among the study population 26% male patients were obese while in female this figure jumps to almost double (47%).

On the basis of risk factors 55% patients had multiple risk factors, 16% had single while no risk factor was found in 29% patients. The third group needs a keen study to undermine the potential etiology responsible for stroke but it is really difficult to arrange and complete such study in a government hospital with existing facilities.

Conclusion:

This prospective observational study is conducted in the northern part of the country. It came out with the finding that stroke type and its association with risk factors are very much similar with other parts of the country. Therefore a unified strategy can be taken throughout the country to prevent stroke recurrence for the greater benefit of our people.

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Prevalence of Dementia in Patients Having Chronic Obstructive Pulmonary Diseases

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Abstract:

Background: *Evidence supports that cognitive impairment is common and clinically important in patients having chronic obstructive pulmonary disease, but the mechanism is still unclear. Data from some pilot studies indicated that chronic hypoxia and hypercapnoea influence cognitive function both in human and animals.* **Objective:** *To determine the prevalence of dementia in Chronic Obstructive Pulmonary Disease (COPD) patients.* **Methods:** *It was an open random cross sectional study done among the outdoor and indoor patients with COPD in Bangladesh Medical College combindly between Neurology and Medicine department. starting from January 2008 to January 2009. COPD was confirmed by spirometry and the level of dementia was determined by Mini Mental State Examination (MMSE). The total number of patients included in the study was 275.* **Results:** *Among 275 patient's fifty patients (18.18%) had clinically insignificant dementia. One hundred and twenty patients (43.63%) had significant deficit requiring some supervision and support. Seventy five patients (27.27%) were affected moderately and may require constant supervision and five patients (1.18%) were affected markedly and requiring 24 hours supervision.* **Conclusion:** *Hypoxia and hypoxaemia in COPD affects cognitive function.*

Introduction:

Dementia literally means undoing of the mind, denotes a deterioration of all intellectual or cognitive functions with little or no disturbance of consciousness or perception. It is the gradual enfeeblement of mental powers in a person who formerly possessed a normal mind.[1] Dementia is characterized by impairment of memory and at least one other cognitive domains such as aphasia, apraxia, agnosia, and executive function. This must represent a decline from previous level of function, be severe enough to interfere with daily function and independence.[2] Dementia may be missed in daily practice. One study showed that the diagnosis was missed in twenty one percent cases in a general medical ward .[3] The normal cognitive decline associated with aging consists of mild change in memory and the rate of information processing which are not progressive and do not affect daily function. In a study of 161 community dwelling cognitively normal individuals aged between 62-100yrs, performance of learning declined uniformly with increasing age.[4] In contrast, delayed recall and forgetting remained relatively stable. Similarly, a second report found that ageing was associated with a decline in the acquisition and early retrieval of new information but not in memory retention.[5] Alzheimer's dementia (AD) is the most common form of dementia in the elderly, accounting for 80.8 % of all community residents and affecting 47.2% (95% confidence limit, 37 and 63.2) of those over 85 years. [6]

The prevalence of COPD is 1-2% in UK but exacerbation of COPD account for 10 % hospital admissions. Chronic obstructive pulmonary disease is characterized by a heterogeneous condition embracing several overlapping pathological processes including chronic bronchitis, chronic bronchiolitis and emphysema.[7] It has been confirmed that chronic hypoxia, hypercapnoea contributes a lot to the patho-physiology of COPD. Evidence supports that cognitive impairment is common and clinically important in COPD, but the mechanism is still unclear. Data from some pilot studies indicated that chronic hypoxia, hypercapnoea influences cognitive function both in human and animals, which includes some distinctive patterns of cognitive dysfunction in human beings and impairment of spatial learning memory in rats. So it can be proposed that cognitive impairment is strongly related to combination of chronic hypoxia and hypercapnoea. Chronic hypoxia and hypercapnoea induced animal models mimic cognitive dysfunction of COPD. Attempts to confirm this hypothesis may lead to a new model of cognitive dysfunction in COPD.[8] Cognitive decline in COPD might be due to hypoxaemia.

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Impairment of cognition in non hypoxaemic patient with COPD needs further study to conclude. Evaluation of cognitive function might be useful in estimation and prognostication, which may lead to better care and may halt the transition to full fledged dementia resulting in the deterioration of quality of life. [9]

Methodology:

It was an open random cross sectional study done among the admitted patients and outdoor patients with COPD in Bangladesh Medical College starting from January 2008 to January 2009. All patients underwent Spirometry examination. The patients having FEV1/FVC less than 70% were selected for Mini Mental status examination. 275 patients participated in this study.

Inclusion Criteria

1. Patients with COPD of any duration were included.
2. All age patients were included.
3. Both male and female were included.
4. Both smoker and nonsmokers were included.

Exclusion Criteria

1. Hypertensive, diabetic and dyslipidaemic patients were excluded from the study
2. Patients with history of vascular or degenerative disease: were excluded.
3. Known case of trauma or infections of brain were excluded.

Observation and Results

Among 275 patients 175 were male and 75 were female. Age level was between 40-80 yrs (Table –I). 125 patients were smoker (45.45%) others were nonsmoker. Among the smokers 20% were female. (Table-II). Period of COPD was 10-15 yrs in 125 patients (45.45%) and 15-20 yrs in 150 patients (54.45%)(Ttable –III). Mini mental status examination (MMSE) revealed the level of dementia. MMSE score was 0-10 in 5 patients (1.18%), 11-20 in 75 patients (27.27%) 21-25 in 120 patients (46.63%) 26-30 in 50 patients (18.18%) (Table -IV). Interpretation MMSE score was noted in (Table -V).

Table-I

Demographic characteristics of patients:(n=275)

Age	40-50	25	9.09%
	51-60	95	34.54%
	61-70	80	29.09%
	71-80	75	27.27%
Sex			
	Male	175	63.63%
	Female	100	36.36%

Table–I: Shows that age of the patients were between 40 – 80 years in four groups and among them 63.63% were male.

Table- II

Distribution of patients according to smoking habit

Smoker	Male	100	80%
n=125	Female	25	20%
Non –Smoker	Male	100	66.66%
n=150	Female	50	33.33%

Table-II: Shows that 125 patients were smoker (45.45%) and 80% of them were male. 150 (54.55%) patients were non smoker and among tem 66.66% were male.

Table-III

Duration of C.O.P.D (n=275)

Male	10-15yrs	100	36.36%
Female	same	50	18.18%
Male	15-20	75	27.28%
Female	same	50	18.18%

Table-III: Shows that 125 patients had duration of COPD of 10 – 15 years and 150 patients had duration of 15 – 20 years

Among 275 patient's fifty patients (18.18%) had clinically insignificant dementia. One hundred and twenty patients (43.63%) had significant deficit requiring some supervision and support. Seventy five patients (27.27%) were affected moderately and may require constant supervision and five patients (1.18%) were affected markedly and requiring 24 hours supervision.

Table-IV

MMSE score		*N=275*
0-10	5 patients.	1.18%
11-20	75 patients	27.27%
21-25	120 patients	43.63%
26-30	50 patients	18.18%

Table IV: Shows scores MMSE. Here 5 patients score of 0 - 10 and 120 patients had score of 21 – 25.

Interpretation of Results (Table-V)

Score 30	No impairment.
Score 25-30	Questionably significant .May have clinically significant mild deficit .Likely to affect only most demanding activities of daily living.
Score 25-20	Mild. Significantly affects. May require some supervision, support and assistance
Score 20-10	Moderate. Clear impairment. May require 24 hours supervision
Score 9-0	Marked impairment. Likely to require 24 hours supervision.

Table-VI

Age	Number (N)	Mean FEV1 of patients	Mean FEV1 of M (ht 1.5m) F (ht 1.4m)	
40 – 50	25	46.04% predicted	69.9% (M)	66.7%(F)
51 – 60	95	40.09% predicted	66.3% (M)	61.3%(F)
61 – 70	80	37.84% predicted	61.5%(M)	53.9%(F)
71 – 80	75	34.66% predicted	55.3%(M)	43.0%(F)

Table –VI: Shows the Mean FEV1 in different age groups in patients compared with normal male and female.

Discussion:

This was a hospital based observational study to find out the prevalence of dementia among the patients having chronic obstructive pulmonary disease. We had 275 patients both male and female having age between 40 – 80 years. Both smoker and non-smoker were included in the study. In our study we had five patients with MMSE score 0 – 10 (1.18%) affected severely, 75 patients had MMSE score 11 – 20 (27.27 %) affected moderately and 120 patients had a MMSE score of 21 – 25 (43.63%) affected mildly and other 50 patients had clinically insignificant disease. Our patients were not on domiciliary oxygen.

Mini mental state examination (MMSE) is the most widely used cognitive test for dementia in US clinical practice [10]. In 2001 practice parameter American Academy of Neurology (AAN) reviewed a number of studies of neuro -psychological testing for dementia. Five subsets including MMSE were identified to be a valid and reliable measure of cognition in normal aging and AD[11]. Eleven MMSE tasks might be completed in 7 min. Total maximum score possible is 30. A score less than 24 is suggestive of dementia. Using this cutoff value MMSE has sensitivity of 87% and specificity of 82% in population based sample. Although the cut-off median MMSE score is 29 with at least nine years of schooling, 26 for 5-8 years of schooling , 22 for 4 yr or less of schooling.[12]

Our patients were of different age starting from 40 – 80 years divided in 4 groups. All patients had FEV1/ FVC less than 70%. The mean FEV1 was less than 47% in all patients which put them in the class of severe obstructive disorder. They were not using continuous oxygen previously. In our study 200 patients had a MMSE score up to 25, which indicated clinically significant cognitive impairment. These 200 (male and female) represent 72.72% of the total (275) study population who had more than 10 years duration of COPD.

Cognitive dysfunction is common and clinically important in severe COPD. The diagnostic accuracy of the MMSE and instrumental activities of daily living scale in screening severe cognitive dysfunction in 149 patients with COPD, mean age of 69.3+8.5 yrs, FEV1-36.6+8.5, had FEVI 36.6+ 17.8% of the predicted . Patients underwent MMSE and an in depth neuro psychological assessment based upon Mental Deterioration Battery (MDB).[13] The mean age of our patients were 68.75 yrs, FEV1 39.66 of the predicted FEV1 36.6 + 17.8%.

In order to characterize the neuro psychologic profile of patients with COPD the performance of 36 patients with COPD 69+10 yrs of age (mean +SD) on 19 tests exploring eight cognitive domains was compared with 29 normal adults (69+7 yrs of age). Cognitive impairment was significantly and positively correlated with age ($p<.05$), duration of hypoxic hypercapnic chronic respiratory failure ($p<.05$). Because patients with COPD were receiving oxygen therapy from the beginning, results suggest continuous oxygen therapy does not prevent or only partly prevents cognitive decline in COPD. A distinct cognitive profile was found in a large fraction of patients

with COPD and it differs in several aspects from those of normal and demented patients.[14]

The diagnosis of C.O.P.D. is confirmed by spirometry when FEV1/FVC ratio is <70%. Both the American Thoracic Society and the European Respiratory Society recommend a simple staging system to asses COPD severity based on post bronchodilator FEV1 as percentage of predicted value.[15] COPD patient's were associated with a significant risk of cognitive impairment, compared to referent subjects (odds ratio 2.42, 95% confidence interval 1.043-6.64). Low baseline oxygen saturation was related to increased risk of cognitive impairment. (odds ratio for oxygen saturation < 88% 5.45; 95% CI, 1.014-29.2; P= 0.048). Conversely regular use of supplemental oxygen therapy decreased the risk for cognitive impairment (OR 0.14; 95% CI 0.07 – 0.27; P< 0.0001). Adults with COPD (n=1202) with referent subject (n=302) Mini mental score <24 was defined as cognitive impairment .[16]

Conclusion:

COPD is a debilitating disease costing money as well as wellbeing with deterioration of cognitive health. But if we try to prevent hypoxia and hypoxaemia the pathological process of dementia may be halted. So patient should undergo regular follow up, frequent pulse oxymetry and blood gas analysis, to avoid hypoxemia and domiciliary oxygen supply if required.

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