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ORIGINAL ARTICLES

Headache Associated with Epileptic Seizures: Epidemiology and Clinical Characteristics

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Summary:

This is a cross sectional observation study conducted in the Department of Neurology, Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka; Bangladesh from January 2007 to December 2008. Three hundred seventy six (376) epileptic patients were initially enrolled purposively as sample size which was determined by considering the prevalence rate and 170 patients fulfilled the inclusion criteria. Face to face interview was performed with a semistructured questionnaire. Mean age of the respondents was 23.44 ± 5.49 years. It was found that most of the respondents (80%) had generalized tonic clonic seizure (GTCS), 11.2% had partial seizure. Most of the respondents (80.6%) suffered from postictal headache and rest from preictal headache. Most of the postictal headache was both sided (82.5%) and frontal headache was more frequent (48.9%). A large number of respondents (n-87) suffered moderate intensity of headache, where as ipsilateral spread of headache was more (n-81). Among preictal headache, both sided headache was common (n-28), which was more in frontal and temporal areas (84.8%). Like postictal headache, a large number of respondents (n-24) experienced moderate preictal pain.

In this study it is found that headache is very common in patients with epileptic seizures. Seizures often triggered postictal headache with migraine features and also caused preictal migraine. Headache associated with epileptic seizures caused both sided headache in most of the cases. The comorbidity of migraine and epilepsy should receive ample clinical attention, and the headache may need specific treatment.

Introduction:

Epilepsy is common among two extreme age groups. Epidemiological studies indicate an association of headache with an increased prevalence of migraine in patients with epilepsy and vice versa[1]. This association is well known but poorly understood[2]. Patients with epilepsy often exhibit headache during the postictal period[3,4] but preictal headache has also been less frequently encountered[5,6]. Postictal headache is a common symptom after generalized tonic–clonic seizures[7,8] and also occurs after complex partial seizures originating in the temporal area[9]. Patients are usually more concerned by their seizures, and headache associated with epilepsy are often neglected, not only by the patient, but also by the physician which is one of the most common complaints of epileptic patients.

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Aim of the study:

To identify epidemiology and clinical characteristics of headache associated with epileptic seizures among Bangladeshi patients.

Methods:

Consecutive new adult patients aged from 18 to 36 years with active epileptic seizures attending in epilepsy clinic of neurology department of BSMMU, Dhaka were recruited for this study. We didn't found any previous data on headache associated with epilepsy among Bangladeshi patients. Frequency of seizure-associated headache was 43% in a study conducted by Forderreuther and his colleagues[6]. We have determined sample size (n-376) by considering prevalence rate 43%. During the study period, data was collected from January to December 2008 with a semistructured pre-tested questionnaire in patients who gave written informed consent were included. The study was approved by the university ethical committee before conducting research. Patients were assessed by a neurologist to rule out secondary causes of seizures. Questions about headache in relation to seizures, side, location, spread, intensity and character of headache were asked. Inclusion criteria were the age between 1836 years with definite diagnosis of epilepsy and those who were given consent to participate in this study.

Headache was diagnosed according to the International Headache Society (IHS) criteria (Headache Classification Committee of the International Headache Society 2004). Headache was defined as chronic if present for more than 15 days per month for the previous 3 months and episodic if present less than 15 days per month. The headaches were categorised as preictal and postictal. Preictal headache was defined as headache starting not more than 24 hours prior to the seizure and lasting until the onset of a seizure. Postictal headache was defined as a headache starting within three hours after a seizure and ceasing within 72 hours after the attack. All patients were asked to grade the usual headache intensity as mild (maintaining normal activities without problems), moderate (maintaining normal activities with difficulty), severe (must give up normal activities and lie down). Data were analysed as mean, standard deviation, frequency distribution and percentage by using statistical analyses and were performed with the Statistical Package for the Social Sciences (SPSS) version 13.0.

Results:

Among 89 male and 81 female (Table-I) respondents 52.4% were within 20 years age group followed by 17.6% within 21 to 25 years and 20.0% within 26 to 30 years (Table-II). Mean (SD) age of the respondents was 23.44 ± 5.49 years. All patients were within 18 to 36 years age range. Prevalence of epileptic generalized tonic clonic seizure (GTCS) was found in 80% of cases, where as 8.8% had complex partial seizure (CPS), simple partial seizure (SPS) was in 5.9% of cases, another 5.3% was due to other causes (Figure-1). Preictal headache was reported by 19.4% of cases whereas most of the respondents suffered from postictal headache (80.6%) [Table-III] but none reported interictal headache. Clinical characteristics of the headaches are given in Table-IV.

Sex	Number	Percent
Male	89	52.4

Female	81	47.6

Table-I

Distribution of the respondents by sex (n-170)

Total	170	100.00

Out of all respondents 52.4% were male and 47.6% were female. Male and female ratio was 1.10:1.

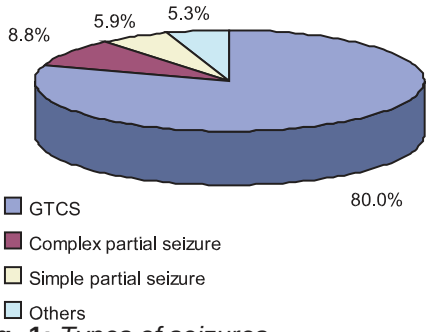

Fig.-1: *Types of seizures*

Table-II

Distribution of respondents by age (n-170)

Age (in years)	Number	Percent
≤ 20	89	52.4
21-25	30	17.6
26-30	34	20.0
31-35	08	04.7
≥ 36	09	05.3
Total	170	100.00

Table-III

Distribution of respondents by types of headache (n-170)

Headache	Number	Percent
Preictal	33	19.4
		80.6
		100.00
Postictal	137	
Total	170	

Table-IV

Characteristics of the ictal headache (n-170)

Characteristics	Postictal (n=137)		Preictal (n-33)	
	Number	Percent	Number	Percent
Side of headache				
• Right	9	6.6	2	6.1
• Left	15	10.9	3	9.1
• Both side	113	82.5	28	84.8
Location of headache				
• Frontal	67	48.9	14	42.4
• Occipital	23	16.8	5	15.2
• Temporal	47	34.3	14	42.4
Spread of headache				
• Ipsilateral	81	59.1	21	63.6
• Contralateral	56	40.9	12	36.4
Headache intensity (VAS)				
• Mild	36	26.3	9	27.3
• Moderate	87	63.5	24	72.7
• Severe	14	10.2		

Characters of headache				
• Pulsating	71	51.8	16	48.5
• Pressure/tightening	58	42.3	16.	48.5
• Alternate pulsating /tightening	8	5.9	1	3.0

3

Table-V

Distribution of the patients by accompanying symptoms in ictal headache (n-170)

Presenting symptoms*	Postictal (n-137)		Preictal (n-33)	
	Number	Percent	Number	Percent
Worsening on activity	39	28.5	20	60.6
Photophobia	47	34.3	7	21.2
Phonophobia	32	23.4	17	51.5
Vomiting	41	29.9	4	12.1
Nausea	84	61.3	15	45.5

* Multiple responses

Postictal headache:

Of the 137 patients in this group, the headache was graded (Table-IV) as mild in 36 (26.3%), moderate in 87 (63.5%) and severe in 14 (10.2%) patients. One hundred and thirteen (113) respondents suffered both sided headache, in which frontal headache was found among 48.9% of respondents followed by more than 34% faced temporal headache. Ipsilateral headache was found more than contralateral headache (59.1% vs. 40.9%). Respondents experiencing postictal pulsating headache was in 51.9% of cases, where as 42.3% had pressure or tightening and only 5.9% suffered alternate pulsating or tightening characters of headache.

Preictal headache:

Thirty three (33) respondents had preictal headache (Table-IV) which was categorised as mild in 27.3% and moderate in 72.7% of cases. Twenty eight (28) respondents were suffered both sided headache, in which frontal and temporal headache was more (84.8%) than occipital headache (15.2%). Ipsilateral headache was found more than contralateral headache (63.6% vs. 36.4%). Most of the respondents suffered pulsating and pressure or tightening nature of headaches (n-32) where as, only one respondent faced alternate pulsating and tightening nature of headache.

Among 137 respondents (Table-V) with postictal headache, 28.5% had worsening during activity, 34.3% had photophobia, 23.4% had phonophobia, 29.9% had vomiting and 61.3% had nausea. While, among 33 respondents (Table-V) with preictal headache, it was found that in 60.6% cases there was worsening of headache during activity, 21.2% had photophobia, 51.5% had phonophobia, 24.2% had vomiting and 45.5% had nausea.

Discussion

Age range of the respondents was from 18 to 36 years where mean (±SD) was 23.44 ± 5.49 years. In one study, average age was found 35.2 years[6] and in another study, mean age of the study population was 24 ± 11.9 years[9], which is consistent with the present study.

In the present study 80.0% had GTCS, 8.8% had CPS, 5.9% had SPS and 5.3% had some other types of seizures. KaraaliSavrun F et al identified 6.66% had SPS,

22.22% had CPS, 22.22% had CPS+ GTCS, 29.62% had secondarily GTCS, 14.81% had primary GTCS and 4.44% had primary nonconvulsive seizure (NCS) among the study subjects[9].

We found incidence of postictal headache in 80.6% of cases among our study subjects. One study mentioned 26% of patients with postictal headache[10] and it was reported 44% in another study[11]. Postictal headache was also found 41-43% in some other studies[5,7,12]. Forderretuther et al. also mentioned that 47(43%) out of 110 consecutive patients suffered headache associated with epileptic seizures and majority of them (n-43) had exclusively postictal headache[6]. The present study also showed the similar findings.

In this study more than 19% of preictal headache patients presented with epileptic seizure. In a study among 100 consecutive patients with pharmacologically refractory partial epilepsy, reported preictal headache in 11% of cases[13]. Another study mentioned preictal headache occurred in 10% of the respondents[10].

Both sided postictal headache (82.5%) was found in most of our respondents which also matched with another study which was 76%[6]. It was observed that frontal (48.9%) and temporal (34.3%) headache were more common whereas, ipsilateral (59.1%) spread of headache was observed in large number of respondents. In our study, more than 63% suffered moderate intensity of headache where as in one study among 48 patients of postictal headache mentioned moderate intensity of headache in 29% of cases[11]. The study also revealed 25% of mild and 38% of severe intensity headache which was similar with our findings.

Several researchers[4-7], have pointed out that the postictal headache has similar characteristics like migraine such as pounding pain and accompanying nausea, photophobia and phonophobia in a significant proportion (41–56%) of patients.

In the present study among the respondents with postictal headache 28.5% had worsening of headache with activity, 34.3% had photophobia, 23.4% had phonophobia, near about 30% had vomiting and more than 61% had nausea. Out of all respondents with preictal headache 60.6% had worsening with activity, over 21% had photophobia, 51.5% had phonophobia, 12.1% had vomiting and 45.5% had nausea. Leniger T et al. stated symptoms associated with seizure headache which was phonophobia in 83 (72%), hemicrania in 52 (45%), throbbing pain in 42%, photophobia in 41%, nausea in 41% and vomiting in 21% of cases[5]. The present study also showed nearly similar result.

Limitations of study: It was a cross sectional study, we couldn't compare with control groups. The sample size was small and was not representative.

We did not performed cooperative analysis with the present data. Time constraint was another limitation for this study.

Conclusion:

Patients with epilepsy often suffer from troublesome headaches that may contribute to a reduced quality of life. Postictal headache is common; preictal headache is less common. There is a strong association between headache and seizures. This comorbidity is important, as headaches often receive less attention than the more acute and dramatic symptoms of seizures. The results of the present study

revealed the frequency of epilepsy related headache in our epileptic population and the frequency of accompanying symptoms of epilepsy related headache. This study also showed the characteristics of epilepsy related headache which could help to conduct a study with larger sample size to confirm our findings.

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Smoking - A Risk Factor for Ischemic Stroke

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Abstract:

This case control observational study was conducted in Neurology Department of Dhaka Medical College Hospital (DMCH) to determine the relation of smoking with ischemic stroke. Fifty cases & 50 controls were selected following certain inclusion & exclusion criteria. The results of the study showed that mean age of ischemic stroke was 63.58 + 10.22 years. Ischemic stroke was more common in male (M:F=2.57:1). Majority of the ischemic stroke patients came from middle economic class. Habit of smoking was equally present in case & control groups (54% & 52% respectively). However duration of smoking was significantly longer (P< 0.01) in ischemic stroke patients (35.26 ± 9.97 years.) than the control group (24.12 ± 7.32 years.). The number of cigarette sticks smoked per day was 15 ± 1.2 in stroke patients and 6 ± 1.5 in the control group with a significant difference between them (P< 0.01). Longer duration & increased number of cigarettes smoked per day were significantly associated with ischemic stroke (P< 0.01 & P< 0.0 1).

Introduction:

Stroke is the third common cause of death in the developed countries. It is also an important cause of mortality and morbidity in our country. Stroke has been defined as focal, at times global, neurological deficit of sudden onset lasting for more than 24 hours or leading to death of the patient with no apparent cause other than that of vascular origin[1]. Stroke has modifiable risk factors like hypertension, diabetes mellitus, dyslipidemia, obesity & smoking[2].

Successful modification of these risk factors may play important role in primary or secondary prevention of stroke. Smoking is a common personal habit among

Bangladeshi people. It is an important cause of atherosclerosis[3]. The present study was conducted to see the prevalence of smoking & it's role in ischemic stroke patients.

Methods & Materials:

This was a case control study conducted in the Neurology department of DMCH from January 2005 to March 2006. Cases were ischemic stroke patients admitted in Medicine & Neurology units of DMCH proved by computerized tomography (CT) scan of brain. Controls were age & sex matched healthy volunteers from the community. Fifty cases & 50 controls were selected randomly following inclusion & exclusion criteria.

Inclusion criteria: Age above 40 years, first attack of stroke, patient admitted within 7 days of onset of illness & those who or their legal attendant gave consent to be included in the study.

Exclusion criteria: Age below 40 years, focal neurological deficit due to other cause like vasculitis, infection & intracranial space occupying lesion (ICSOL), those who did not give consent.

The variables included in the study were age, sex, economic status, presence of smoking habit, duration of smoking & amount of cigarattes smoked per day. Detailed history taking and clinical examination were done. Some relevant investigations were also done. Data were analyzed with the help of computer using SPSS (Statistical package for social science). Statistical significance was set at 0.05 level & confidence interval at 95%.

Results:

The mean age of ischemic stroke was 63.58 ± 10.22 years (Table- V). Majority (80%) of patients belonged to the age group of 59 to 79 years (Table- II). Seventy two percent of the ischemic stroke patients were male & 28% female. Most of the patients (80%) came from middle class income group (Table-III). Fifty four percent of the cases and 52% of the control were smoker (TableIV). Mean duration of smoking in the case and control groups was 35.26 ± 9.9 years and 24.12 + 7.32 years respectively (TableV). The average number of sticks of cigarettes smoked per day was 15 ±1.2 in case group and 6 ± 1 .5 in the control group.

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Table-I

Comparison of age between case and control groups.

Age in years	Study Group		T value	df	P value
	Case	Control			
40-49	46.33 ± 1.53	44. 33 ± 1,16			
50-59	54.60 ± 2.85	53.87 ± 2.59			
60-69	62.73 ± 3.06	61.13 ± 1.60	0.226	98	0.822
70-79	71.50 ± 2.42	72.50 ± 2.72			
80-89	80.71 ± 1.89	81.71±12.36			
Total	63.58 ± 10.22	63.10 ± 11.01			

The mean age of ischemic stroke patients was 63.58 ± 10.22 years.

Table-II

Age and sex distribution among case and control group

Age in Years	Study group

	Case		Control	
	Male (%)	Female (%)	Male (%)	Female (%)
40-49	2(4)	1 (2)	2(4)	1(2)
50-59	12 (24)	3(6)	12 (24)	3(6)
60-69	8(16)	7(14)	8(16)	7(14)
70-79	8(16)	2(4)	8(16)	2(4)
80-89	6(12)	1(2)	6(12)	1(2)
Total	36(72)	14(28)	36(72)	14(28)

72% of the stroke patients were male and 28% were female. Male: Female ration was 2.57:1. Eighty percent of the stroke patients were between the ages of 40-79 years.

Table-III

Economic status of cases and controls

Economic Status	Case (%)	Control (%)	χ^2 value	df	P value
Upper	6(12)	4(8)			
Middle	40(80)	38(76)	1.785	2	0.410
Lower	4(8)	8(16)			
Total	50(100)	50(100)			

Most of the patients (80%) of ischemic stroke came from middle economic class while 12% were of low and 8% were of high economic class.

Table-IV

Comparison of smoking habit between case and control groups.

Study group	Smoker (%)	Non- smoker (%)	Total (%)	χ^2 value	df	P value
Case	27(54)	23(46)	50(100)			
Control	26 (52)	24(48)	50(100)	0.04	1	0.841
Total	53(53)	47(47)	100(100)			

Fifty four percent of ischemic stroke patients and 52% of the controls were smoker. There was no significant difference between the two groups regarding the presence of smoking habit ($p>0.05$).

Table-V

Comparison of duration of smoking between case and control groups.

Group	Mean duration of smoking in years.	t value	df	p value
Case (n - 27)	35.26 ± 9.97	4.623	51	0.001
Control (n - 26)	24.12 ± 7.32			

Mean duration of smoking in the case and control groups was 35.26 ± 9.7 years and 24.12 ± 7.32 years respectively. There was significant difference between the two groups regarding the duration of smoking habit (P< 0.01).

Table-VI

Comparison of daily cigarette smoking between case and control groups

Group	Mean number of sticks smoked per day.	t value	df	p value
Case (n - 27)	15 ± 1.2	24.17	51	0.001
Control (n - 26)	06 ± 1.5			

The average number of sticks of cigarette smoked per day was 15 ± 1.2 in case group and
6 ± 1.5 in the control group.

Discussion

Smoking has been found to be an important risk factor for ischemic stroke[4]. It is a risk factor for chronic obstructive airway disease, lung cancer, carcinoma esophagus, coronary heart disease, peripheral vascular disease & ischemic stroke[5]. This study was conducted to see the smoking habit of patients with ischemic stroke & to find any association of smoking with ischemic stroke. The results showed that the mean age of stroke patients was 63.58 ± 10.22 years. Previous two studies[4,6] showed mean age of ischaemic stroke patients were 55.56 ± 13.14 years and 59.61 ± 13.20 years respectively. Most of the patients in this study belonged to the age group of 5th & 6th decade (60%). The mean age of ischemic stroke in this study is higher than previous studies. The mean age of ischemic stroke is increasing in our country due to increased life expectancy because of improvement in education, public awareness & improved health care facilities. Male: female ratio of ischemic stroke patients in this study was 2.57:1 indicating that ischemic stroke is a male predominant disease. The male: female ratio in previous two studies[6,7] was higher (8.9:1.1 & 8.56:1.44 respectively). The cause of higher prevalence in male in addition to other factors may be that female patients with ischemic stroke get less admitted in hospital due to religious grounds, less number of allocated beds for female & less attention by the family. Eighty percent of ischemic stroke patients in this study came from middle economic class. This result does mean that ischemic stroke is more common in middle class people. This hospital based study may not reflect association of economic status with ischemic stroke. Patients of ischemic stroke with higher economic status usually seek health service in private hospital or clinics on payment in a better environment while poor people usually avail government health care facilities free of cost. Previous two hospital based studies[6,7] showed that 57% & 61% patients of ischemic stroke came from middle economic class. The present case control study revealed that 54% of cases & 52% of controls were smoker indicating that smoking is a common habit in Bangladeshi people. Mere presence of smoking habit showed no significant association with ischemic stroke ($p>0.05$). However duration of smoking & amount of smoking showed significant relation. The mean duration of

smoking in ischemic stroke patients was 35.26 ± 9.97 years which was longer than the control

group (24.12 ± 7.32 years). Longer duration of smoking showed significant association with ischemic stroke ($p<0.01$), because prolonged & heavy smoking have cumulative effect in causing atherosclerosis & ischemic stroke. Chronic smoking increases fibrinogen, platelet adhesiveness & increases blood viscosity and reduces blood flow contributing to pathogenesis of ischemic stroke[8]. The number of cigarette sticks smoked per day in the case group was 15±1.2 while it was 6 ± 1.5 per day in the control group. There was significant difference between the two groups regarding the amount of smoking per day ($P< 0.01$). A previous long term prospective study revealed that daily cigarette smoking increases the risk of fatal stroke three & half times i.e. an increased risk was found in relation to increased consumption[9].This finding is supported by a other study[10]. The relative risk of stroke in a heavy smoker (>40 cigarettes/day) was twice than that of light smoker (<10 cigarettes/day). The stroke risk reduced significantly after cessation of smoking for 2 years & was at the level of non-smoker after cessation of smoking for 5 years[10].

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CASE REPORTS

Cirsoid Aneurysm of The Scalp: Report of Two Cases

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Abstract

Cirsoid aneurysms of the scalp are a type of arteriovenous malformation of the scalp and are rarely encountered in neurosurgical practice. Different modalities of investigations to diagnose and different options to treat them are described in the literatures. They are difficult to treat in the sense that they have a high rate of recurrence as well as have higher complication rate in the form of skin necrosis at the site of surgery. Here we are presenting two cases of cirsoid aneurysms of the scalp that we have treated at the department of Neurosurgery, Bangabandhu Sheikh Mujib Medical University, Dhaka, Bangladesh of which one developed button hole skin necrosis following surgery. We are presenting the cases for their rarity with literature review.

Introduction

Cirsoid aneurysms, a variant of arteriovenous malformations (AVM) of the scalp are rare vascular lesions and are infrequently encountered in neurosurgical practice[1-4]. They are notoriously difficult lesions to manage because of their complex vascular anatomy, high shunt flow and cosmetic disfigurement[3,5,6]. These lesions present as an innocuous looking subcutaneous scalp lump or a large, grotesque, pulsatile mass with a propensity to massive hemorrhage[4]. Various treatment options that have been adopted to treat these lesions include: surgical excision, ligation of feeding vessels, transarterial and transvenous embolization, injection of sclerosant into the nidus and electro thrombosis[1,4,6]. A proper diagnostic evaluation is essential for the successful management of these lesions[5].

Case Reports

Case 1: A 37 year old male presented with a gradually increasing pulsatile soft swelling in the left fronto-parietal region for one and a half year that developed following a blunt trauma on that region three years back. The swelling was tender and he also complained of generalized headache for about one year. He was very anxious about the disfiguring swelling. He also had one episode of profuse haemorrhage from the swelling following a trivial trauma to it. On examination the swelling was soft, compressible, pulsatile, felt like a bag filled with worms and was warmer than the surrounding areas. There was a healed ulcer on it. The left superficial temporal artery (STA) was dilated and tortuous. There were some other engorged vessels around the swelling

(Fig 1). On auscultation it had bruit. CT scan of head revealed a soft tissue mass in the left fronto-parietal region with dilated vascular channels in it (Fig 2). It had no intracranial extension. MRA revealed no abnormality of the intracranial vessels. All his blood pictures and biochemical parameters were within normal range. We excised the lesion totally with a large horse-

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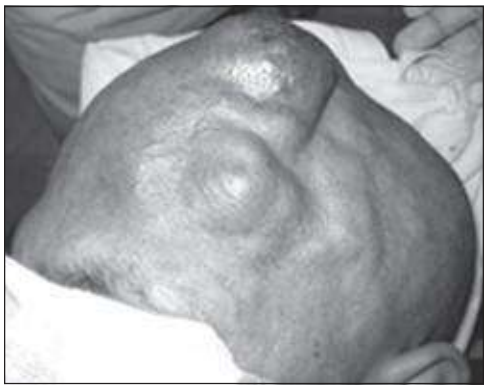

Fig.-1: *Preoperative picture of the patient case 1 showing the swelling and dilated scalp.*

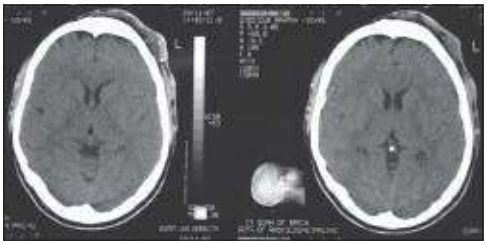

Fig-2: *CT scan of head of case 1 showing soft tissue mass with dilated vascular channels.*

tortuous vessels.

shoe shaped skin incision. After skin incision we identified the left STA and ligated it after securing it with sutures to diminish the blood flow to the nidus. There were numerous dilated vascular channels in the subcutaneous fatty layer that also involved the pericranium to some extent (Fig 3). All the vascular channels were excised by meticulous dissection and bipolar coagulation. Post-operatively he developed a button hole necrosis which was repaired with skin graft later on (Fig 4). Till ten months of follow-up he was doing well without any recurrence. After that he was lost to follow-up.

Fig.-3: *Peroperative picture of case 1 showing dilated worm like vessels in the of*

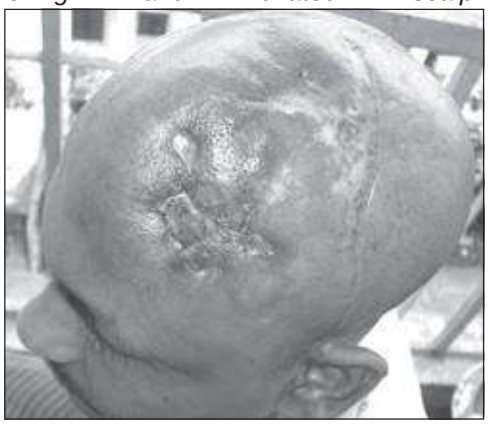

Fig.-4: *Postoperative picture of case 1 showing healed scalp after skin graft on the button hole necrosis.*

Case 2

A 21 year old male presented with a mass on the left frontoparietal region for about seven years, which was like a small nodule in the beginning and started to grow larger for the last one year following a blunt trauma to head on that region to become a disfiguring mass now. On examination the swelling was soft, compressible, had the feeling like a bag of worms. The skin over it was unhealthy and fixed to it but was free from the underlying bone. It was well

the scalp having high vascular flow mimicking an arteriovenous fistula that had two feeders from both the STAs and three

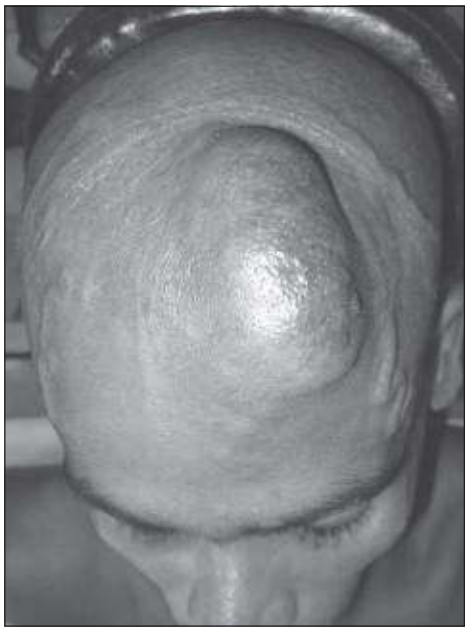

Fig.-5: *Preoperative picture of case 2 showing the cirsoid aneurysm with dilated and tortuous bilateral STA*

to four indistinct drainers. We planned to dissect it out from the scalp. Accordingly pulsatile having bruit on auscultation. Both the superficial temporal arteries (STA) were dilated and tortuous (Fig.-5). As the patient was poor, so only a duplex doppler ultrasonography was done and that revealed a cystic lesion over the left frontal region of the skin flap. Post-operatively the skin healed well without any necrosis (Fig 6). The patient is still under follow-up without recurrence or any complication for the last seven months.

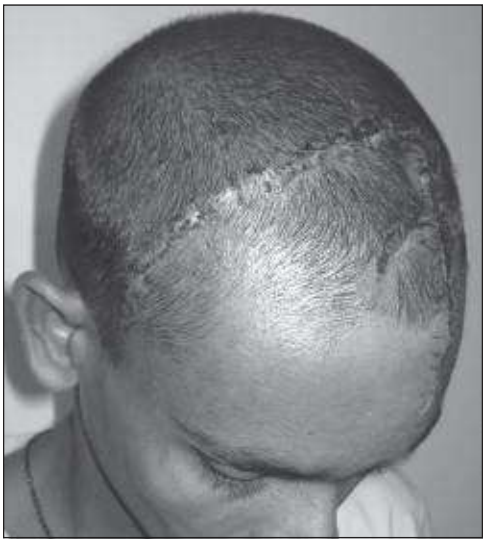

Fig.-6: *Post-operative picture of case 2 showing disappearance of the aneurysm and good healing of the scalp.*

Discussion

we proceeded with a bicoronal skin incision and started to dissect it out meticulously. But eventually we found that the dilated and tortuous vascular channels were so superficially placed near the skin that we decided to excise

the swelling along with the skin. And we did so with an elliptical incision saving the vascularity of the rest of

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The vascular malformation of the scalp is an abnormal arteriovenous communication situated within the subcutaneous fatty layer of the scalp with the feeding arteries derived from the vessels supplying the scalp. Cirsoid aneurysms are discussed in the literature under various titles. Virchow believed them to be tumours of blood vessels and called them racemose aneurysms. Dupuytren considered them to be arterial varices, while according to Virchow, it was Breschet who suggested the term cirsoid from the Greek “kirsos” meaning varix because of the characteristic variceal dilatation of the draining veins in 1833. Various names being used to describe this vascular malformations of the scalp include arterial angiomas, aneurysms by anastomosis, aneurysma serpentinum, plexiform angioma, arteriovenous fistula and arteriovenous malformation[1,4,7.]

The genesis of Arteriovenous Malformations (AVMs) is faulty differentiation of the primitive vessel complex. As a result of abnormal haemodynamics they become progressively dilated and tortuous. The arterial system that supplies an AVM frequently is multiple and complex. The main sources of blood supply to the scalp are located in the subcutaneous tissue and originates from the external carotid, occipital and supraorbital arteries. The superficial temporal artery is frequently involved in traumatic cirsoid aneurysm due to its long unprotected course[1,3,4]. In our patient of case 1 the left STA and in case 2 both the STAs were the main feeders to supply the nidus. We could not identify the drainers well with our limited facilities of investigations. The location of scalp arteriovenous fistulas is roughly evenly distributed among the frontal, temporal, parietal and occipital regions[3,4]. Both the our cases had the nidus in the left frontoparietal regions which is one of the commonest sites. The etiology of these lesions is still controversial. However, it is generally accepted that it may be either of congenital or traumatic origin. About 10 to 20% of scalp arteriovenous malformations develop following penetrating or nonpenetrating trauma to the scalp. Some authours believe that inflammation plays a prominent role in their production[1,4,7]. In case 1 the aneurysm definitely developed following a trauma. But in case 2 it seems that it might be a congenital one which was not noticed before until it started to grow larger. Trauma seems to be an inciting factor that caused the rapid growth later.

Most of the patients reported in the literature had a history of progressive increase in the size of the lesion and had become symptomatic in the third decade of life[4]. Our first patient presented in his fourth decade. However, the second patient presented in his third decade. Clinically the aneurysm is easily diagnosed. Beneath the bluish tinged skin, the cirsoid aneurysm appears as an irregular, diffuse, elevated mass which is not only soft but easily compressible. There are usually visible pulsations, and a thrill synchronous with the arterial beat can be palpated. A bruit can be heard on auscultation over the aneurysm. The impression imparted to the examining hand is that of a mass of worms[7]. Lesions of both the patients were

pulsatile, had bruit on auscultation, felt like bags of worms and were soft and compressible. The clinical manifestations relate primarily to the size of the fistula, and they can cause headaches, excessive bleeding when traumatized, epilepsy, tinnitus, and deformity[4,7,8,9]. The first patient had profuse haemorrhage once following a trauma to the lesion and had generalized headache. Both the patients had complained of disfigurement by the lesion and the engorged dilated vessels around the lesion.

The radiological findings are important for patient management. The different modalities for diagnosis are CT, MRI, MRA, digital subtraction angiography (DSA) and duplex doppler ultrasound studies. DSA is the gold standard for diagnosing these lesions. The feeders and the drainers are well visualized in DSA and is very helpful to plan the surgery[3,8,10]. Magnetic resonance angiography can show the feeders of the malformation well, but it can not define the draining veins satisfactorily[5]. For case 1, we did CT scan of head that revealed a soft tissue swelling over the left frontoparietal region with multiple dilated vascular channels with no intracranial extension. His MRA showed normal intracranial vessels and the extracranial vessels were not delineated. In case 2, we only did duplex doppler ultrasound study and that was sufficient enough to reveal the characteristic nature of the lesion with delineation of the feeders from both the STAs.

Management of scalp arteriovenous malformation is difficult because of its high shunt flow, complex vascular anatomy and cosmetic problems[3,4]. The indication of treatment includes cosmetic relief of the pulsatile mass, prevention of hemorrhage and other symptoms such as headache and tinnitus. The treatment options include surgical excision, ligation of feeding vessels, transarterial and transvenous embolization, injection of sclerosant into the nidus and electrothrombosis[1,4,6]. Surgical excision is the most common and successful method of dealing with scalp arteriovenous malformation[2,4,7]. We did surgical excision in both the cases. Various techniques have been used to control the hemorrhage during surgery including percutaneous sutures of the feeding vessels, interlocking suture along the line of incision, and use of tourniquet and intestinal clamp over the base of the flap[4]. For prevention of

haemorrhage during surgery we secured and ligated the feeders just after the skin incision - the left STA and both the STAs for case 1 and case 2 respectively.

Conclusion

Although, it carries some risk from severe haemorrhage and a tendency for the occurrence of necrosis of skin at the site of surgery, surgical resection seems to be the most effective treatment with complete surgical removal and good cosmetic result is feasible in patients of cirsoid aneurysms.

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REVIEW ARTICLE

Role of Strength Training on Post-stroke Hemiparesis

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Summary

Stroke is the leading cause of activity limitation and participation restriction all over the world and weakness presents a more serious compromise to movement and function in poststroke hemiplegia than spasticity. In this article we have gone through publications on functional consequences of poststroke paresis and effect of resistance training in effective stroke rehabilitation and reviewed the clinical and functional phenomena of paresis in stroke survivors, effects of highresistance training on improvement of strength and functional capacity. By examining the various facets of weakness, we can understand the specific nature of motor impairment and can identify potential strategies to mitigate it's effects, functional improvement and participation in activities. The specific aims were to emphasis the importance of strength training in the functional improvement of patients suffering from stroke with paralysis.

Introduction

Stroke is a leading cause of long-term disability in the western world, with a prevalence of approximately 900 per 100,000 persons. Because of remarkable development in the acute management of stroke, the majority of patients now survives and recovers, experiencing only a modest decrease in life expectancy[1]. In Bangladesh we do not know the actual prevalence and impact of stroke but it may approximate or exceed the statistics of the western world. During the last decade, the number of stroke survivors has increased 30 percent worldwide. As a result considerable number of stroke survivors present with partial to complete loss of function and social burden. Though the incidence of stroke doubles with each decade beyond 60 years of age, the incidence of stroke has increased dramatically in younger individuals in the recent years[2]. This changing landscape for persons surviving stroke underscores the critical importance of providing effective rehabilitation with the potential to optimize recovery of function, minimize long-term disability and enable functional independence so that quality of life and employment is ensured. In a poor country like Bangladesh the socioeconomic burden of disabled stroke survivors is much higher.

The sequelae of stroke: The sequelae of stroke are multifactorial and depend heavily on the mechanism, extent, and location of the vascular lesion. The primary concern addressed in physical rehabilitation is restoration of the requisite motor function to perform the myriad of tasks encountered in daily life. These tasks range from grasping, reaching, and manipulation to more

physical demanding transitional movements and complex coordinated

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movements, such as locomotion. Common to these motor tasks is control of muscular force, which becomes compromised with central nervous system damage and manifests as impaired intersegmental coordination, hyperreflexia or spasticity, and unilateral weakness[3].

Traditional perspectives: Spasticity presented the most significant limitation to recovery of normal motor function. Physical exertion was clinically observed to exacerbate spasticity. Therapeutic activities using forceful contractions became restricted for persons with nervous system injury. One prominent approach to treatment of adult hemiplegia thus centered on the concept of managing muscle hypertonia. The general goal of neurorehabilitation treatment is to focus on improving control, and especially the quality of movement. Meta-analyses that examined the effects of commonly used interventions for rehabilitation of both the upper and lower limb in poststroke hemiplegia reported a lack of compelling evidence that any of the existing approaches to neurorehabilitation have demonstrated superior efficacy for promoting recovery of motor function[4,5].

Resistance training: Currently emerging evidence suggests that paresis may be directly responsible for compromised motor function[6.7,8]. Positive effects of resistance exercise have been demonstrated in persons with poststroke hemiplegia, and in some cases, concomitant influences on performance of functional tasks have been observed. High-intensity activities, including resistance training, could form an important component of rehabilitation programs for persons with poststroke hemiplegia.

Role of agonists and antagonists in functional improvement:

Weakness following stroke is referred to as either hemiparesis (mild to moderate degree of weakness) or hemiplegia (severe or complete loss of motor function) on one side of the body. However, evidence is now emerging that weakness also occurs on the ipsilesional side (traditionally termed the “nonparetic”), within a short time frame postacute stroke[9]. In the literature, poststroke weakness has been described not only as impaired force magnitude[10] but also as a more broadly defined phenomenon, including slowness to produce force, a rapid onset of fatigue[14], an excessive sense of effort[11,12] and difficulty with producing force effectively within the context of a task[15]. Throughout this review, we use the term “poststroke weakness” to include all aspects of weakness following stroke.

Cocontraction of antagonist muscles has also been found to interefere with force magnitude, rate of force production, and intersegmental coordination by acting as an “antagonist restraint”[16]. Significant impairment of agonist activation has been demonstrated in the paretic limb[7,17]. Such observations lead predictably to questions of whether and how agonist activation can be improved and whether such improvement in

physiologic function leads to clinically and functionally important differences in motor performance.

Poststroke paresis and functional impairment:

Poststroke hemiplegia is associated with significant impairments of motor function that are believed to compromise activity of daily living (ADL) performances and lead to loss of independence. However, a direct causal relationship between strength or weakness and motor function has not been established. Traditionally, a strong bias has existed against quantifying strength in hemiplegic persons. As a result, the majority of clinical research in this population has focused on outcome measures at the activity and participation levels[18]. Bohannon and Andrews observed that gait performance in 17 hemiparetic persons was significantly correlated with knee extensor torque but not with spasticity and knee extension muscle performance measured either isometrically or isokinetically correlated significantly with gait velocity[19]. Nakamura and co-workers also observed that spasticity was unrelated to locomotor impairments[20,21]. Isokinetic knee extension strength in the paretic limb was strongly associated with self-selected walking speed (SSWS). Lindmark and Hamrin observed a moderate relationship between SSWS and either motor scores or knee extension torque, which improved in predictive power when examined in a multivariate statistical model[22]. Pohl and co-workers observed that the combination of peak isometric knee extension force and rate of force acquisition explained a significant 12 percent of variance related to gait speed in hemiparetic adults[23]. When plantar flexion strength was added to the model, its explanatory power increased such that it became possible to predict maximal gait speed[24]. Each of the investigations just described focused on isolated muscle groups or actions. However, functional movement involves simultaneous activation and coordination of multiple muscles. This disparity may contribute in part to failure to demonstrate a direct relationship between strength and function[25].

Strengthening Exercise Induced Neurological Recovery.

Because poststroke weakness involves both neural and muscular changes, it seems appealing to suggest an analogy with other physiological conditions, such as aging, for which very clear benefits of strength training have been demonstrated[26]. Currently, available evidence regarding strengthening in hemiplegia indicates that significant strength gains are attainable in persons with poststroke hemiparesis at acute, subacute, and chronic stages of recovery[27-29]. However, the physiological mechanisms responsible for these therapeutically induced improvements have not been demonstrated. Strengthening exercises may influence neural drive at either the supraspinal or spinal level. **Power reaching or skilled reaching training:**

Recent efforts for stroke rehabilitation have been directed toward functional

and task-specific therapies that focus primarily on ADL and on grossly related precursor activities[30,31]. A common element to these more recent approaches is substantially increased therapeutic intensity relative to traditional approaches. Increased intensity is defined by a substantially increased volume of therapeutic participation[32], an increased amount of direct participation in therapeutic activities, or performance of activities at a higher level of the subject's functional capacity[33]. Current controversy thus centers around whether the critical variable for therapeutic efficacy is the task specificity or the intensity of effort involved in therapeutic activities. Thus, while skillbased, task-specific interventions clearly promote important use-dependent cortical reorganization, resistance training apparently can promote additional, beneficial plasticity elsewhere in the neuraxis. In all likelihood, the most effective therapeutic intervention involves a combination of elements.

The Ultimate Goal of Rehabilitation
The ultimate goal of rehabilitation following stroke is to promote improvements in function, activities, and participation. Collective efforts are thus required to design effective and efficient rehabilitation interventions. Weakness is not the only impairment in poststroke hemiplegia but weakness plays a significant contributory role to motor disability. By examining the various facets of weakness such as low force production, fatigability, excessive sense of effort, ineffective task-dependent force production etc. we can understand the specific nature of motor impairment and can identify potential strategies to mitigate it's effects, functional improvement and participation in activities.

Future research: Important area for future research is developing a greater understanding of the mechanisms underlying poststroke weakness. Without this information, we are restricted in our efforts to design appropriate rehabilitation interventions to counteract compromised function associated with poststroke weakness. Recent research evidence indicates that "task-specific" therapy[34-37] produces superior outcomes as compared to traditional therapeutic approaches[38-41]. However, there is also evidence that increased intensity of therapy leads to more significant functional outcome. There is a need to establish the effectiveness of strength training in relation to task-specific therapies because it may be the case that strength training is an efficient means for delivering high-intensity therapy. To define and implement suitable protocols of strength training into stroke rehabilitation programs, future research should explore the specific factors such as the types of exercise, the frequency, intensity and time spent in strength training, and the number of specific exercises. Moreover, the long-term effects, both longterm training and retention of training, need to be understood. Finally, once gains in strength have been achieved we need to understand how they translate to

functional gains and how they are best maintained.

Precaution: One must recognize that post stroke resistant training may not be suitable for all hemiparetic persons. In this regard, we recommend exercise should be idividualized on clinical judgment appropriate for any rehabilitation setting. High-intensity resistance training is certainly contraindicated in any case before the patient is neurologically stable. Other significant contraindications would involve postsurgical patients and persons with severe osteoporosis, acute orthopaedic or joint injuries. While the patient or client is exercising, his or her blood pressure should be monitored, and precautions should be taken to avoid conditions leading to a valsalva maneuver.

Conclusion

While the number of studies is limited, emerging evidence suggests that persons with poststroke paresis can improve strength through resistance exercise in the absence of negative side effects, including exacerbation of hypertonia. Moreover, these improvements in strength appear to transfer to functional improvements. Despite increases in strength, improvements in functional performance may not occur in hemiplegic persons with low strength and low performance. It is entirely possible that vigorous strength training promotes positive effects on other aspects of physiologic function in this type of at-risk population. In elderly patients, strength training has been demonstrated to decrease depression and improve sleep patterns, influence bone mass, decrease insulin resistance (Type II diabetes), and normalize blood pressure[42]. Still, many unresolved issues remain. The potential for strength training to improve the overall outcomes of rehabilitation for persons with poststroke hemiplegia warranty further investigations.

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Treatment of Traumatic Carotid Cavernous Fistula – Experience of Three Cases in BSMMU

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Summary

A variety of surgical techniques have been developed to treat the traumatic carotidcavernous fistulae. The carotid occlusion or the carotid preservation techniques are two basic approaches available. Here we reported three cases of traumatic carotidcavernous fistulae (CCF) two of whom were treated by carotid occlusion technique and other one resolved spontaneously. On admission all patients had pulsatile proptosis, chemosis and bruit over orbit. The diagnosis of CCF was made clinically and on computed tomography or magnetic resonance angiography of brain and confirmed by digital subtraction angiography of carotid arteries. Cross circulation of the carotid artery was checked clinically (Matas Test) and angiographically. Two patients were treated by ligation of internal carotid artery in cervical region and C2 segment of the same intracranially. In one patient fistula was resolved spontaneously. No major complication developed in two patients treated by carotid occlusion. The carotid occlusion technique is old and less popular in view of the great advances of endovascular techniques which preserve the patency of the carotid artery. But due to less availability of facilities and poor socio-economic status, this procedure is still an appropriate therapeutic option, as reflected by the success at our hospital. However, more affluent patients have options to be treated in other centres for carotid preservation techniques like detachable balloon embolization.

Introduction

Abnormal communications between the cavernous sinus and the carotid artery can be classified by etiology (traumatic; spontaneous; iatrogenic), by flow dynamics (low flow; high flow) and by anatomy (direct versus dural; internal carotid artery versus external carotid artery versus both). A clinically useful classification is the anatomical-angiographic classification by Barrow DL et al in which fistulas are divided in four types. Type A fistulas are direct shunts between the internal carotid artery (ICA) and the cavernous sinus. Types B-D are dural shunts between meningeal branches of the ICA, ECA or both and the cavernous sinus [1]. Traumatic fistulas are almost always type A fistula and

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rarely resolve without treatment. In type A fistula trauma produces a rent in the intracavernous portion of the internal carotid artery allowing arterial blood to enter the cavernous sinus and its main tributaries, the orbital venous complex, and the inferior petrosal sinus [2]. A bruit, proptosis and chemosis are most common symptoms. Some would also suffer from headaches, diplopia and visual failure [3,4].

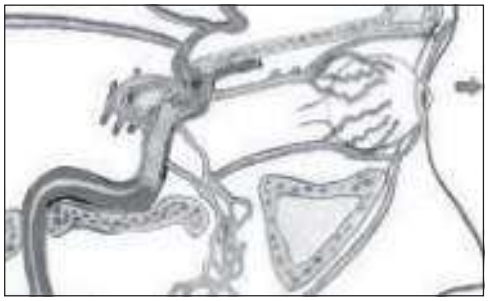

Fig.-1: *Schematic representation of a direct CCF.*

Treatment of a direct CCF is mandatory for immediate orbital symptom relief and to prevent the development of intracranial venous hypertension. Most direct CCFs can be treated electively, urgent treatment is necessary only in patients with progressive visual loss, corneal exposure, severe proptosis with pain and high intraocular pressure, intolerable bruit, epistaxis, sphenoid sinus aneurysm, severe retro-orbital pain, cortical venous drainage or coma [5,6]. When the fistula is small and asymptomatic, further treatment is not indicated. The goal of treatment is the occlusion of the fistula while maintaining carotid artery patency, which is mandatory when the collateral blood flow via the contralateral ICA is insufficient[7]. The treatment of choice for direct CCFs is transarterial embolization with detachable balloons[8-10]. In some patients this approach is not possible and a transvenous approach via the superior ophthalmic vein or inferior petrosal sinus may be successful[11].

An alternative for balloon embolization is detachable platinum coils. Only recently, covered stents suitable for delivery in the cavernous segment of the carotid siphon became available [7]. Very rarely, if both the transarterial and transvenous approaches fail, pterional craniotomy with a Dolenc approach is a possibility, but this approach is technically difficult [12].

However, in areas where endovascular facilities are unavailable carotid occlusion techniques are still of value. Carotid occlusion techniques include muscle embolization, ligation of the cervical internal carotid or trapping the fistula between cervical and intracranial occlusions of the internal carotid – either as single or combined procedures [13, 14].

Case Reports Case 1

A 25 year old man was referred for headache, progressive pulsatile exophthalmos and gradual dimness of vision in right eye. He had sustained a road traffic accident 2 years back. Few days after the accident he noticed a small swelling in right supraorbital region. Two months later there was protrusion of right eye ball and both the supraorbital swelling and eyeball became pulsatile. One month later he developed visual dimness in right eye ball and headache. His symptoms continued to increase up to the time of admission to the hospital. Visual acuity examination revealed no PL/PR in right eye and 6/6 in left eye. Chemosis and abducens nerve palsy were present on

right side and intraocular pressure in the right eye was elevated (24 mm Hg). A bruit was audible over the right orbit. Fundoscopy showed primary optic atrophy and dilated vessels in right eye with normal fundus in left side. The diagnosis of a traumatic direct CCF was considered. CT scan of head showed dense lobulated lesion in the right retrobulbar region and right temporal region with contrast enhancement. MR angiography showed an enlarged, hyperintense right cavernous sinus with dilated venous channels, including ipsilateral superior ophthalmic vein (SOV) and inferior petrosal sinus. DSA revealed a rupture of the cavernous segment of the ICA proximal to the ophthalmic artery and early retrograde opacification of the superior ophthalmic vein and inferior petrosal sinus.

contralateral carotid artery. He was treated by ligation of the right internal carotid artery in cervical region and intracranially at C2 segment. Regression of ocular symptoms was obtained after treatment. No immediate postoperative complication was observed.

Case 2

Five months after road traffic accident, a 22 year old male patient presented with right sided pulsatile exophthalmos, chemosis and dilated supraorbital vein. There was no visual disturbance. Intraocular pressure was slightly elevated. Bruit was present over right orbit and temporal fossa.

CT scan of head showed enlarged enhancing right cavernous sinus and superior ophthalmic vein (SOV) and inferior petrosal sinus. Intracerebral perfusion and collateral circulation

Before operation *After operation*

Fig.-2: *Patient of CCF showing chemosis, proptosis and supraorbital sweelling. Immediate after operation, proptosis and supraorbital swelling reduced but chemosis still persisted*

We planned to treat this case by trapping operation of right ICA. Collateral blood flow from opposite ICA was checked clinically by the Matas test and confirmed on DSA. The Matas test was performed with external compression of the

showed no anomalies. Digital subtraction angiography (DSA) revealed a right sided, direct, high-flow CCF draining anteriorly into the SOV and posteriorly into the basilar plexus and inferior petrosal sinus.

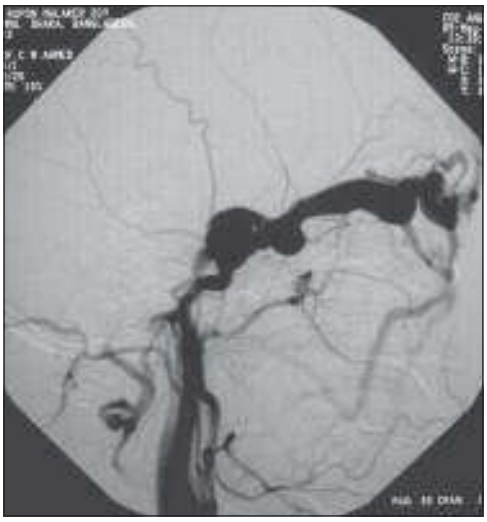

Fig.-3: *DSA shows a direct fistula between the intracavernous segment of the right ICA and the cavernous sinus with dilated superior ophthalmic vein and inferior petrosal sinus.*

He was treated by the same procedure as that of the first case. Post-operatively proptosis and pulsation were reduced but radiological evaluation could not be done.

Fig.-4: *Proptosis due to CCF*

Case 3

A 12 year old girl was referred for headache, pulsatile exophthalmos and chemosis in left eye following blunt head trauma. She had no visual disturbance. Bruit was present over left orbit and dilated veins were visible in left upper eyelid. CT scan of head, MR angiography and DSA revealed left carotid cavernous fistula draining into in left SOV and left inferior petrosal sinus.

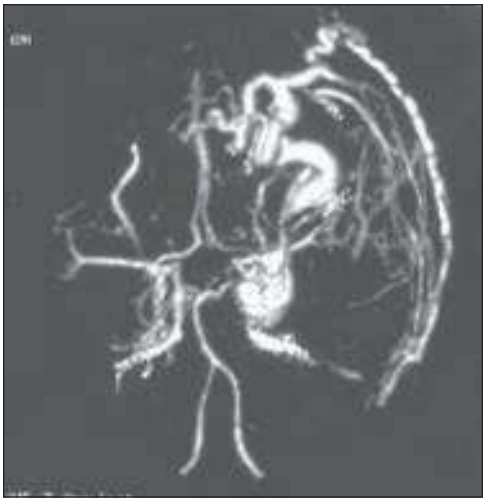

Fig.-5: *DSA shows a direct fistula between the intracavernous segment of the left ICA and the cavernous sinus with dilated superior ophthalmic vein and inferior petrosal sinus.*

She was treated conservatively and over one month period exophthalmos and

chemosis regressed spontaneously. However radiologically CCF persisted.

Discussion

We have treated two cases of traumatic CCF by ligating the internal carotid artery both in the neck and in the supraclinoid portion thus isolating the fistula between an intracranial carotid ligation and a cervical carotid ligation – the so called 'trapping' operation. It is one of the carotid occlusion techniques used for the treatment of traumatic CCF. This carotid occlusion procedure is now less common and susceptible to impairment of the cerebral circulation on the side of the occluded carotid artery. However, considering limited availability of endovascular facility and the patient's low socio-economic status we still can give benefit using this procedure. In Udon Thani Center Hospital, Thailand 12 patients during the period from 1997 to 2002 were treated by carotid occlusion technique and success rate was 91.6% [14].

Spontaneous remission may occur in 5-10% of patients[15]. In our experience, clinical features resolved spontaneously in one patient. None of the patients has recurrence till now but long term follow up is necessary.

Conclusion

In BSMMU we have treated two cases of traumatic CCF by carotid occlusion technique with no major complication. Though endovascular technique is now the standard treatment for this type of CCF, carotid occlusion technique is still an alternative method in centres with technical and financial limitations.

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Malignant Thymoma in a Patient with Myasthenia Gravis - A Case Report

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Summary :

Invasive thymomas and thymic carcinomas are relatively rare tumours, which together represent about 0.2% to 1.5% of all malignancies. We are reporting a 48 years old patient who was a businessman, nondiabetic, nonhypertensive, admitted in neurology department of Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka, Bangladesh with the complaints of dropping of both upper eye-lids, difficulties in swallowing, mastication, & movement of eyeballs, and blurring of vision. The patient was operated on with the diagnosis of myasthenia gravis to remove the thymus. Histological examination of the tumour specimen revealed lymphocyte rich thymoma which showed thick fibrous band and focal calcification.

Introduction

Thymomas are epithelial tumours of the thymus, which may or may not be extensively infiltrated by non-neoplastic lymphocytes. The term, thymoma, is customarily used to describe neoplasms that show no overt atypia of the epithelial component. A thymic epithelial tumour that exhibits clear-cut cytologic atypia and histologic features no longer specific to the thymus is known as a thymic carcinoma (also known as type C thymoma)[1].

Invasive thymomas and thymic carcinomas are relatively rare tumours, which together represent about 0.2-1.5% of all malignancies[2]. Thymic carcinomas are rare and have been reported to account for only 0.06% of all thymic neoplasms[3]. In general, thymomas are indolent tumours with a tendency toward local recurrence rather than metastasis. Thymic carcinomas, however, are typically invasive, with a high risk of relapse and death[4, 5].

Most patients with thymoma or thymic carcinoma are aged 40 through 60 years[6]. The etiology of these types of tumours are not known. In about 50% of the patients, thymomas/thymic carcinomas are detected by chance with plain-film chest radiography[6]. Ninety percent occur in the anterior mediastinum[7].

Approximately 30% of the patients with thymoma/thymic carcinoma are asymptomatic at the time of diagnosis[6]. In other cases, the presenting clinical signs

(excluding thymic carcinoma) were found to be associated with myasthenia gravis[17]. Although the oncologic prognosis of thymoma is reported to be more favourable in patients with

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of these types of tumours may include coughing, chest pain, and signs of upper airway congestion. Paraneoplastic autoimmune syndromes associated with thymoma include myasthenia gravis, polymyositis, lupus erythematosus, rheumatoid arthritis, thyroiditis, and Sjögreen syndrome, among others[6,8,9]. Autoimmune pure red cell aplasia and hypogammaglobulinemia affect approximately 5% and 5-10%, respectively, of patients with thymoma[7]. Thymoma-associated autoimmune disease involves an alteration in circulating T-cell subsets[10, 11]. The primary T-cell abnormality appears to be related to the acquisition of the CD45RA+ phenotype on naive CD4+ T cells during terminal intratumourous thymopoiesis, followed by export of these activated CD4+ T cells into the circulation[12]. In addition to T-cell defects, B-cell lymphopenia has been observed in thymoma-related immunodeficiency, with hypogamma-globulinemia (Good syndrome) and opportunistic infection[13-15]. In contrast to thymoma, the association of thymic carcinoma and autoimmune diseases is rare[8, 16].

In a large retrospective study, approximately 47% of thymoma cases

myasthenia gravis than in patients without myasthenia gravis[7], treatment with thymectomy may not significantly improve the course of thymomaassociated myasthenia gravis[18, 19].

Thymoma and thymic carcinoma should be differentiated from a number of nonepithelial thymic neoplasms, including neuroendocrine tumours, germ cell tumours, lymphomas, stromal tumours, tumour-like lesions (such as true thymic hyperplasia), thymic cysts, metastatic tumours, and lung cancer[1, 20]. Standard primary treatment for these types of tumours is surgical with enbloc resection for invasive tumours, if possible[4, 6, 7, 21]. Depending on tumour stage, multimodality treatment includes the use of radiation therapy and chemotherapy with or without surgery[6, 22].

Thymoma has been associated with an increased risk for second malignancies, which appears to be unrelated to thymectomy, radiation therapy, or a clinical history of myasthenia gravis[19, 23, 24].

Computed tomography (CT) may be useful in the diagnosis and clinical

staging of thymoma, especially for noninvasive tumours. CT scan is usually accurate in predicting tumour size, location, and invasion into vessels, the pericardium, and the lung. CT scan cannot predict, however, invasion or resectability with accuracy[25, 26].

Case Presentation

A 48 years old patient who was a businessman, non-diabetic, nonhypertensive, got himself admitted in neurology department of Bangabandhu Sheikh Mujib Medical University, Dhaka, Bangladesh with the complaints of dropping of both upper eyelids with difficulty in swallowing, mastication, movement of eyeballs and blurring of vision for 6 months. The patient also reported some sorts of general weakness. The complaints that expressed were very mild in early morning, but became more more worse with the advancement of the day. The patient also reported about dyspnoea seven days back, and double vision for last 3 months.

After getting admission in the hospital, thorough clinical examinations and

laboratory tests were done according to symptoms and history as mentioned below. On neurological examination we found bilateral ptosis, diplopla, normally reacting pupils and normal cognitive function.

In the examination of motor system, muscle tone and power were reduced where as bulk was normal. All reflexes and cerebellar function were normal.

Initial laboratory data showed a haematocrit value of 44.00%, the white cell count was 12,000/mm^3 with 84% neutrophils, 13% lymhocytes, 2% Monocytes, and 1% eosinophil. Blood chemistry findings were all within normal limits. Serum FT_4 and TSH levels were normal.

Antiacetylcholine receptor antibody (Anti achRab) level was not done.

In the CT scan of brain there was mild atrophic changes in the brain with secondary effects in the ventricles & other fluid containing spaces.

CT scan of the chest - showed an enlarged thymic mass in the supervisor mediastinum.

The patient was operated on with the diagnosis of myasthenia gravis to remove the thymus. A standard median transsternal thoracotomy was performed. Thymus was found highly adhesive with pericardium and pleura. Partial excision of the thymus was done and sent for histopathological examination.

Histological examination of the specimen revealed lymphocyte rich thymoma which showed thick fibrous band and focal calcification.

In addition to partial resection of the tumour, the patient was treated with anticholinesterase, local radiotherapy, and chemotherapy. Now the patient is on anticholinesterase medication only.

Investigations done on 15.5.2009 showed Hb-15.2 gm/dl, Tc- 7300/cm^3, P-68%, L29%, E-2%, M-1%, ESR - 27 in first hour. At present patient is getting tab. Pyridostigmine Tab. (60 mg) 6 hourly.

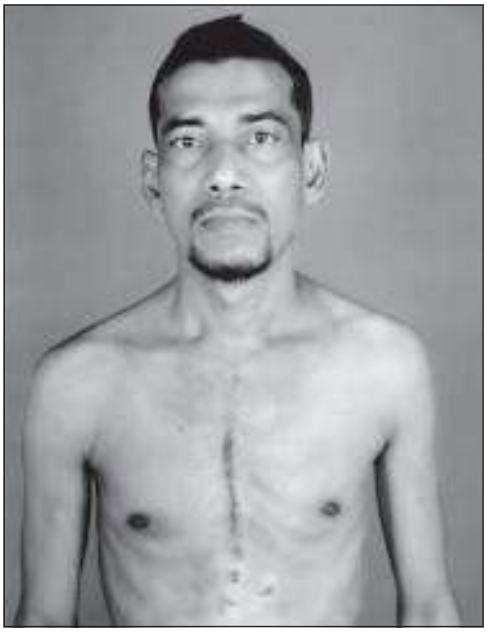

Fig.-1: *Showing standard median transsternal thoracotomy scar in a patient of myastheina gravis with malignant thymoma.*

Discussion

Approximately half of all thymomas never produce symptoms, and are discovered

incidentally, either on a routine chest X-ray or at autopsy[27].

The symptoms of myeasthenia gravis (MG) usually precede the discovery of a thymoma. Reports indicate the association of MG with thymoma to be about 15% but increase to 35% in older patients[28]. Ocular symptoms like diplopia and ptosis are the commonest clinical presentations in MG seen in more than 50% of patients[29].

Thymus develops from the ventral portion of the third and fourth pharyngeal pouches and then descends into the anterior mediastinum by the sixth week of gestation. Thymic ectopia results from failure of this migration. Aberrent nodules of thymic tissue are found in approximately 20% of humans. Although the majority of aberrent nodules are located laterally in the neck, ectopic thymic nodules also have been reported in the base of the skull, in the mediastinum and at the root of the bronchus[30].

As we suspected this as a case of thymic growth, we undertook various imaging procedures, among which, CT scan was more informative. Keen and Libshitz reported that the presence or absence of intervening fat planes on CT accurately predicts vascular and/or pericardial invasion[31]. However Fon GT et al. reported that although the presence of preserved fat planes between a thymoma and the adjacent vascular structures reliably excludes macroscopic invasion, the absence of such fat does not consistently indicate an invasive tumour[32]. Chiou GT et al[33]. defined three categories of mediastinal fat planes; complete preservation, partial obliteration or preservation, and complete obliteration[34, 35].

The patient was operated with the diagnosis of myasthenia gravis due to thymic growth (hyperplasia or tumour) to remove the thymus. Thymus was found highly adhesive with pericardium and pleura.

Pericardial involvement by thymic tumours is usually a late stage observation in primary mediastinal thymomas. The development of massive pericardial effusion associated with pericardial metastasis of thymoma is described by various authours. In some cases it was the first clinical sign of an orthotopic thymic tumour[36-39]. Thymomas originating primarily in the pericardium, however, are rarely reported neoplasms. A review of literature reveals a total of nine cases of primary intrapericardial thymoma: four in the AFIP series[40], two reports addressing diagnostic procedures and follow-up issues[41,42], and three incidental autopsy cases[43, 44]. Patients were mostly elderly women and presented clinically with symptoms of right heart congestion. The histopathology of the thymic tissue after operation showed malignancy.

The two basic elements of the thymus gland are the epithelial cells and the lymphocytes. The large majority of the tumours designated as thymomas are composed of a mixture of these two elements, the relative proportions varying considerably from case to case and even in different areas of the same

tumour. The designition of thymoma is restricted to neoplasms of the thymic epithelial cells, regardless of the presence or absence of a lymphoid component. It thus follows that identification of epithelial cells constitutes a sine qua non for the histopathologic diagnosis of thymoma. Occasionally, however, differential diagnosis from other tumours, particularly malignant lymphoma and germinoma (seminoma), may be impossible under these circumstances. In some instances, the newly developed immunologic technics may provide a definitive answer. Kodama T et al[44]. reported that most thymomas are focally or diffusely stained with monoclonal antibody.

Leu-7, and all thymomas are stained for keratin in varying degrees. This makes a good marker for differential diagnosis between thymoma and lymphoma or seminoma[45, 46].

The prognosis of patients with thymoma is related to the stage of the tumour. Several other factors also influence prognosis, including degree of surgical resection and the presence of parathymic syndromes.

Although the presence of myasthenia gravis in patients with thymoma had been regarded as an indicator of poor prognosis, recent evidence suggests that this is not the case. There are reports of improved survival rates for patients with myasthenia gravis with thymoma[47,48]. Indeed, prognosis may be improved because the thymomas are diagnosed at an earlier stage. In addition, the recurrence rate of patients with myasthenic thymoma is lower than that of patients with nonmyasthenic thymoma[49].

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Relation between Hydrocephalus with Vertical Dimension of The Suprasellar Nonpituitary Space Occupying Lesions

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Abstract:

To evaluate the association of the vertical dimension of the suprasellar nonpituitary space occupying lesions (LOC) with occurrence of hydrocephalus and to find out vertical dimension of the suprasellar nonpituitary space occupying lesions as well as to find out the existence and extent of the hydrocephalus.

Background: Patients of suprasellar space occupying lesions presented with headache, visual disturbances and hormonal imbalance and consulted with medicine, eye specialist and sometimes endocrinologist. In many situations it seems that by the time Computed tomography (CT) scan and Magnetic resonance imaging (MRI) diagnosis has made, the patient has already became blind. Sometimes the patient present with features of raised intracrainal pressure (ICP) due to obstructive hydrocephalus. A patient with acute hydrocephalus has rapid deterioration of his/her vision. Few studies have been done on the effect of suprasellar space occupying lesions on the cerebrospinal fluid(CSF) pathways in some center but in our country this type of study has not been done before.

When the tumours grow upward and elevates the floor of the third ventricle, the pathways of CSF flow is obstructed between lateral, third and fourth ventricle. But if tumours are small in size then hydrocephalus (HCP) does not occur. So we felt upward growth of the non - pituitary suprasellar space occupying lesions is an important factor for development of HCP. This can lead to severe morbidity and poor outcome of treatment and may be the predictor of treatment results. Therefore we undertook the study.

Methods:The study was a cross sectional study. The study was done on patients with CT scan or MR imaging diagnosis of suprasellar lesions, whose histopathological study revealed that the SOL was non-pituitary in nature.

Results: A total of 35 patients with suprasellar non-pituitary space occupying lesions were studied. Majority (74.29%) of them were craniopharyngiomas. Other were menigiomas 17.14%. Gliomas 5.71% and Rathke's cleft cyst 2.86%. 71.53% of the patients had vertical dimension more than 4 cm and 28.57% had below 4 cm. Hydrocephalus were present in 25 cases and absent in 10 cases.

Introduction:

Tumours of the suprasellar region such as adenomas of the pituitary gland, craniopharyngiomas, non-neoplastic cystic lesions (especially Rathke's cleft cysts) and meningiomas are frequently encountered in neurosurgical practice[1]. Patients with lesions in the suprasellar regions may present with hydrocephalus. In larger suprasellar tumours, effacement and compression of the 3rd ventricle may be seen. Distortion of interventricular foramina may result in unilateral or bilateral ventricular dilatations. This is more common in craniopharyngiomas than in pituitary adenomas[2]. Most of the craniopharyngiomas occupy the suprasellar space and often extend into parasellar, intrasellar or retrosellar space. Symptoms reflect the tumour's close proximity to the optic apparatus, hypothalamic pituitary axis and ventricular systems. Upward growth into the region of the hypothalamus gives these tumours the potential to affect overall endocrine control. If these lesions impinge on the pituitary stalk, they can effectively disconnect the hypothalamus from the neurohypophysis. Symptoms of increased intracranial pressure may also result if these growths disturb the ventricular system and results in hydrocephalus[3]. Pituitary tumours are common lesions believed to account for 10-15% of all intracranial tumours. Prolactinoma is the most common of the hormone secreting pituitary tumours. Most of them are microprolactinoma (<1cm in diameter) and few are macroprolactinoma (>1cm in diameter). The term giant prolactinoma is used for tumours larger than 4 cm in diameter. Clinical manifestations are attributed to marked hyperprolactinemia, gonadal failure, and neurological symptoms. The development of symptomatic hydrocephalus due to a pituitary adenoma is an exceptional event. Only a limited number of cases have been reported so far[4]. Suprasellar meningiomas originate from the arachnoid layer connected to the dura of the anterior or posterior clinoidal process, or the tuberculum, dorsum or diaphragma sellae. Tumours originating from the pituitary stalk are rare. Such lesions may include germinomas, astrocytomas, histiocytosis X, hamartomas, and sarcoidosis.

Meningiomas frequently occur in the suprasellar region and require a different therapeutic strategy[1]. Suprasellar meningiomas arise in close proximity to the optic chiasma, displacing it posteriorly and superiorly and stretching it. So visual symptoms are early and common[5].

Rathke's cleft cysts (RCCs) are benign, epithelium-lined intrasellar cysts believed to originate from remnants of the Rathke's pouch. RCCs are commonly round, ovoid, or dumbbell

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shape. Because of their cystic nature, most of the RCCs expand into the suprasellar cistern through the cleft of the diaphragma sella. Patients with symptomatic RCC, the cyst was found in intrasellar and suprasellar locations[6]. Other tumours of the suprasellar region include chiasmatic gliomas, germinomas, haemangiomas, epidermoids, dermoids, lymphomas and metastatic ones[7].

Materials and Methods:

The study was done in the department of neurosurgery, Bangabandhu Sheikh Mujib Medical University, Dhaka, Bangladesh on patients with CT scan or MRI imaging diagnosis of suprasellar lesions, whose histopathological study revealed the SOL was non-pituitary in nature. The duration of study was from October 2007 to April 2009. Fourteen patients were recorded from OPD where they came for follow up and rest 21 patients were studied during their hospital stay. All the patients were evaluated on the basis of history, clinical examination, neuroimage (CT and MRI) findings and histopathological reports.

Results:

A total of 35 patients with suprasellar nonpituitary space occupying lesions were studied. Majority (74.29%) of them were craniopharyngiomas. Others were menigiomas 17.14%. Gliomas 5.71% and Rathke's cleft cysts 2.86%. 71.53% of the patients had vertical dimension more than
4cm and 28.57% had below 4cm. Hydrocephalus was present in 25 cases and absent in 10 cases.

Table-I

Distribution of the patients by age (n-35)

Age groups in years	Frequency	Percentage
0-10	10	28.57%
11-20	8	22.86%
21-30	10	28.57%
31-40	6	17.14%
41-50	0	0%
51-60	0	0%
61-70	1	2.86%
Total	35	100%

Table-I shows age distribution of craniopharyngiomas. Majority (28.57%) of them were in the group of 0-10 years followed by age groups 11-20 years and 21-30 years, 31-40 years (17.14%) and 6170 years (2.86%). The mean age was 18.87 years. The minimum age of the cases was
5 years and maximum age was 65 years.

Table -II

Distribution of the patients by sex (n-35)

Sex	Frequency	Percentage
Male	24	68.57%
Female	11	31.43%
Total	35	100%

Table-II shows the distribution of patients by sex. 68.57% of the patients were male and 31.43% of them were female. Male to female ratio was 2.18:1.

Table-III

Distribution of suprasellar non-pituitary space occupying lesions by types (n-35)

Types	Frequency	Percentage
Craniopharyngioma	26	74.29%
Suprasellar meningioma	06	17.14%
Suprasellar glioma	02	5.71%
Rathke's cleft cyst	01	2.86%
Total	35	100%

Table-III shows distribution of suprasellar non-pituitary space occupying lesions by types. Majority (74.29%) of them were craniopharyngiomas. Others were menigiomas 17.14%, Gliomas 5.71% and Rathke's cleft cyst 2.86%.

Table-IV

Distribution of patients by presenting symptoms (n-35)

Symptoms	Frequency	Percentage
Headache	33	94.29%
Visual Disturbance	26	74.28%
Vomiting	25	71.43%
Endocrine abnormalities	22	62.86%

Table-IV shows the presenting symptoms among the patients with suprasellar nonpituitary space occupying lesions. More than 90% presented with the complaints of headache, 74.28% of the patient had visual disturbances, 62.86% had endocrine abnormalities and 71.43% had occasional vomiting. Other minor symptoms were present in 34.29% of cases.

Table-V

Vertical dimension of suprasellar nonpituitary space occupying lesions (n-35)

Vertical dimension	Frequency	Percentage
>4cm	25	71.53%
$\leq$ 4cm	10	28.47%
Total	35	100%

Table-V shows the vertical dimension of the suprasellar non-pituitary space occupying lesions. 71.53% of the patients had vertical dimension more than 4 cm and 28.47% had less than 4 cm.

Table-VI

Distribution of patients with hydrocephalus in suprasellar non-pituitary space occupying lesions (n-32)

SOL	HCP Present	Percentage
Crainopharyngioma	21	60%
Meningioma	2	5.71%
Glioma	2	5.71%
Rathke's cleft cyst	0	0%
Total	25	71.42%

Table-VI shows HCP present in the suprasellar non-pituitary space occupying lesions. 60% were in craniopharyngiomas, 5.71% were in

menigiomas and gliomas respectively. No HCP was seen in Rarhke's cleft cyst in this series. Over all 71.42% of the patients had HCP.

Table-VII

Distribution of patients without hydrocephalus in suprasellar non-pituitary space occupying lesions.

SOL	HCP absent	Percentage
Cranopha-ryngioma	5	14.29%
Meningioma	4	11.43%
Glioma	0	
Rathke' cleft cyst	1	2.86%
Total	10	
		28.58%

Table-VII shows HCP absent in the suprasellar non-pituitary space occupying lesions. HCP was absent in 14.29% cases of craniopharyngiomas, in 11.43% cases of menigiomas and in 2.86% cases of Rarhke's cleft cyst. Total 28.58% of the patients had no HCP.

Table-VIII shows the association between vertical dimensions of the suprasellar nonpituitary space occupying lesions with hydrocephalus. Pearson (χ^2) test value was 6.776 with degree of freedom[1]. p value was found <0.01, which was significant to establish the association between upward growth of suprasellar non-pituitary space occupying lesions with hydrocephalus.

Table-VIII

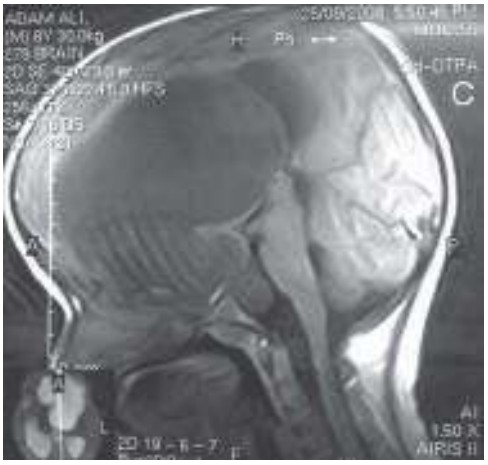
Association between vertical dimensions of suprasellar non-pituitary space occupying lesions with hydrocephalus (n-35)

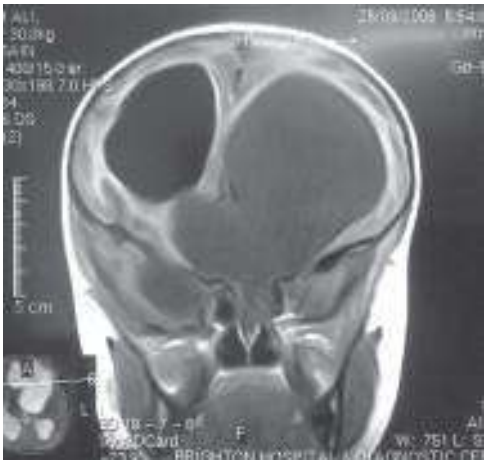
Vertical Dimension of suprasrellar SOL	Hydrocephalus		Total
	Present	Absent	
>4cm	21	4	25
≤ 4cm	4	6	10
Total	25	10	35

Pearson Chi-Square (χ^2) test value 6.776 df=

1

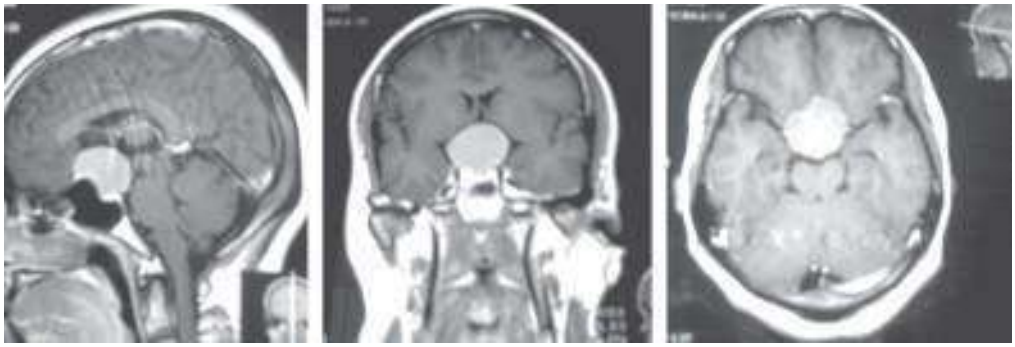
P value <0.01 (p value <0.05 was considered significant in 95% confidence limit)



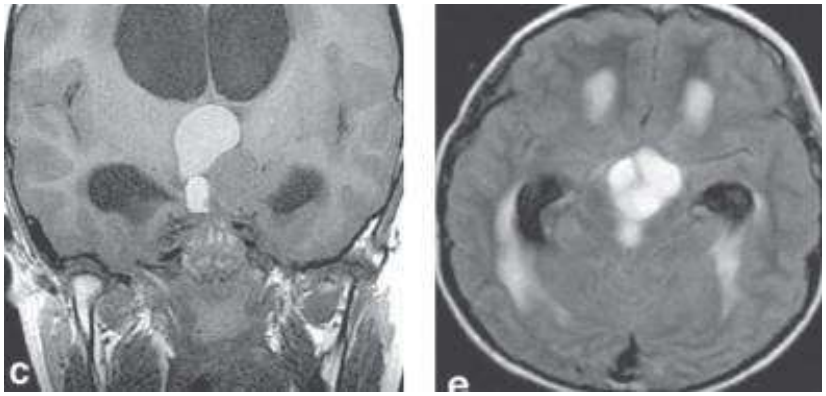


MRI of brain with contrast sagittal view shows upward growth of cranio- craniopharyngioma. This occupied the third

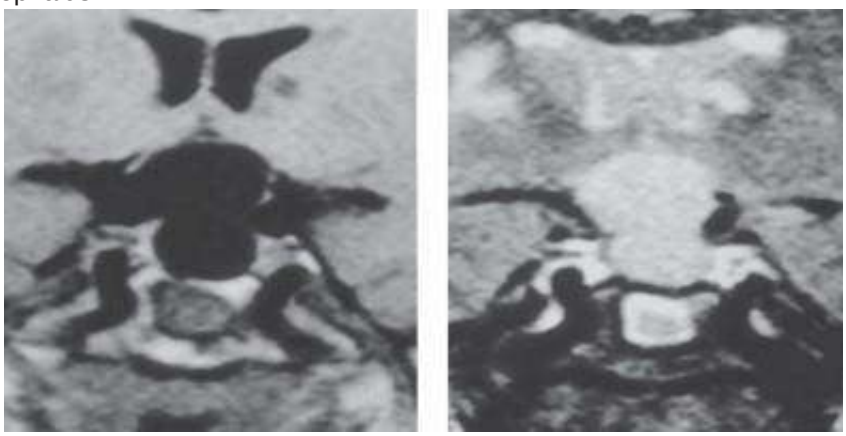
MRI of brain with contrast, coronal section shows upward growth of pharyngioma. and part of left lateral ventricle, Right lateral ventricle is also shown dilated.



MRI of Brain with contrast shows suprasellar meningioma in sagittal, coronal and axial views without ventricular dialtaion.



MRI of brain, coronal (contrast) and axial (FLAIR) films shows craniopharyngioma (Multiple cystic) which elevates the floor of the third ventricle and shows hydrocephalus.



T1WI and T2W2 coronal view images of MRI revealed supra-sellar homogeneous ovoid mass-Rathke's cleft cyst, low intensity in T1WI, and high intensity in T_2WI.

Discussion:

Our study was carried out in the department of neurosurgery, Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka, Bangladesh during the period of October 2007 to April 2009 to find out the relation between hydrocephalus with vertical dimension of the suprasellar non-pituitary space occupying lesions.The study subjects were 35 patients with suprasellar non-pituitary space occupying lesions. Their age range was between 5 to 65 years. Mean age was 19.18 years. The highest incidence was craniopharyngioma in the age group of 0 to 10 years, that means these tumours mostly occur in children which is similar to Erain et al. 2005[8] study. Median age of craniopharyngioma was 18.87 years, because most of the patients came to hospital in advanced stage. Majority of

suprasellar meningioma was seen in 21 to 30 years age group, glioma in 0 to 10 years age group of patients. Only one Rathke's cleft cyst was found and his age was 21 years.The male to female ratio was 2.18 to 1 in this series, probably due to fact that the male bed number of the department of neurosurgery, BSMMU, is two times greater than that of female and males are more privileged in our society. In our series craniopharyngiomas were found in highest number (74.29%) then suprasellar meningiomas (17.14%), suprasellar gliomas (5.71%) and Rathke's cleft cyst (2.86%). Headache, visual disturbances, endocrine abnormalities and vomiting were the most common clinical presentations for suprasellar space occupying lesions. In our study, headache was more than 90%, visual disturbance was 74.28%, endocrine abnormality was 62.86% and vomiting was

71.43%, which is almost similar to Halac and Zimmerman, 2005 study[9]. Vertical dimension was measured from mid-sagittal sections of MRI and CT scan (sagittal section). Upward growth of the suprasellar non-pituitary space occupying lesions elevate the floor of the third ventricle, obstruct the foramen of Monro and leads to hydrocephalus. In our series vertical dimension of more than 4cm was in 25 (71.53%) cases and less than 4cm was in 10 cases (28.57%). 21 out of 25 and 4 out of 10 cases presented as symptomatic HCP. 60% patients of craniopharyngiomas presented with HCP which was nearly similar with the study of Hukin et al. 2005[10]. They reported incidence of HCP in their series was 57%. 5.71% suprasellar meingiomas, and 5.71% suprasellar gliomas presented with HCP. Over all HCP was present in 71.42% and absent in 28.58% of cases. This is due to fact that the patient came to the hospital with late presentation and took more time for confirmation of the diagnosis in our country. Most of the patients had difficulty to afford expenses for investigations like CT scan or MRI. Though very few studies reported relation between HCP and suprasellar nonpituitary space occupying lesions.Ershahin et al. 2005[8] reported that if craniopharyngiomas is >4cm in size, then HCP was present. In this series we found 21 cases out of 25 having vertical dimension >4cm presented with symptomatic HCP. 25 cases out of 35, presented with HCP in this study, which is a less significant association (p=0.01 in 95% confidence limit) between hydrocephalus and upward growth of suprasellar non-pituitary space occupying lesions.

Conclusion:

In our study we found that there is a significant relation between hydrocephalus and vertical dimension of suprasellar nonpituitary space occupying lesions. Vertical dimension is the key factor for the development of symptomatic hydrocephalus. A patient with acute hydrocephalus has rapid deterioration of his/her vision. In this study we found most of the HCP was developed when vertical dimension was larger than 4cm. So, upward growth of suprasellar space occupying lesions is

the important factor for patients prognosis.

Recomendations:

1. There should be appropriate referral system.
2. There should be adequate facilities to diagnose suprasellar space occupying lesions and its risk factor in the primary level hospitals that will help the patients for early treatment and decrease the morbidity and mortality rate.
3. A large sample study with fixed time interval between initial symptom and presentation should be done.

Limitations of the study:

- The study was carried out at a tertiary level specialized hospital. So, actual incidence and all types of cases were not represented.
- Sample size was small. So, it was not possible to generalize the findings of the study to reference populations.
- Randomization of the sample was not done. So, there may be a sample bias.
- All data were collected from the patients at the time of admission but there are variations of the time interval from the onset of symptoms and admission in the hospital between the patients.

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Fluxetine in Migraine Prophylaxis - A Prospective Study

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Abstract

This was a prospective study done in outpatient department of Bangladesh Medical College from 1.1.06-1.12.08. Patients who met the criteria for migraine were included in this study. They were given cap fluxetine an antidepressant of SSRI group 20 mg daily. Patients were followed up for 6 months. Pain was assessed by universal pain assessment scale. Structured questionnaire were used to follow the improvement. Eight hundred & seventy five patients completed the study. Fluxetine showed significant improvement in both severity and frequency of migrainous attack.

Introduction

Migraine is a painful condition characterized by periodic intense headache occupying one or both side of the head often accompanied by vomiting, photophobia and phonophobia, continuing for 4-72 hours[1]. Its prevalence is about 11% and females are affected more (about 15-18%) and males are about 6%[2,3,4]. The median frequency of attack is 1.5 per month and median duration is 24 hrs[2]. Classic migraine is associated with aura, usually runs in the family in about 60-80% and common migraine without aura, is less frequent in the family. Migraine reduces in severity or may disappear during menopause[1].

In addition to being an important cause of pain and suffering, migraine shares a significant portion of medical cost, and loss of productivity. Some study showed lost of productivity of 313 dollars per attack with annual cost of 3309 dollers per sufferer in USA. Total medical cost was about one billion dollar in USA for treating this condition in 1994[5-7].

Although pathophysiology of migraine is not well understood, vasoconstriction followed by vasodialation was the 1st possible explanation for migraine[8]. Cortical spreading depression leading to suppression of neurological activity in an area of cortex results in failure of brain ion homeostasis & efflux of excitatory amino acid[9]. The theory of hyperexcitability to stress and other triggers results in abnormal chemistry of brain. Migraine patients have chronically low systemic 5 HT predisposing them to migrainous headache. Stimulation of vascular 5HT-1 receptors probably located in the dural vascular bed may alleviate headache. Stimulation of these yet unidentified receptors result in vasoconstriction, inhibiting depolarization of sensory perivascular afferents within the trigemino –vascular system & thus stopping headache[10]. High oestrogen level interacts with serotonin transport system, compromising serotonin transport

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system can explain increased frequency of attacks during child bearing age . Stress leads to sympathetic over activity and increased attack[11].

Preventive therapy should be introduced when more than two attacks occur per month with disability lasting three or more days per month, failure or contraindication or adverse events of abortive treatment, use of abortive medication more than twice per week and uncommon migraine conditions with prolonged aura.

The goal of preventive therapy is to improve patients quality of life by reducing migraine frequency, severity and duration and by increasing the responsiveness of acute migraine therapy.

A full therapeutic trial may take 2-6 months .After successful therapy migraine should be reduced by 50% or more, then the medication should be continued for 6-12 months, then discontinuation of therapy may be considered[12].

Fluxetine is an antidepressant of SSRI class and an approved drug for the treatment of depression. It acts by inhibiting reabsorption of serotonin. In a study by AAN determines a bidirectional association between depression and migraine. It is thought that some people are more susceptible to changes and abnormalities of serotonin regulation. Serotonin causes blood vessels in the head to contract and lowers pain threshold. Before a migraine episode serotonin becomes unusually high and during attack it becomes very low leading to vascular dilatation and pain . Most drug treatment influence serotonin level in some way.

Fluxetine is the third most prescribed antidepressant in USA in 2007[13]. Fluxetine was shown to be effective for depression in a 6 weeks long double blind controlled trial showed alleviation of anxiety and improved sleep. It is better than placebo for the prevention of recurrence[14].

Materials and Methods:

It is an open prospective study done in the Neurology OPD of Bangladesh medical college from 1.1.06-1.12.08. All patients undergone proper physical examination along with thorough neurological examination. Routine investigations including blood and urine examinations were done to exclude any systemic infection. Eight hundred and seventy five (875) patients completed the study. 23 patients left the study due to intolerability of the drug. Structured questionnaire was used .Patients were prescribed to take fluxetine 20 mg daily and advised to attend the OPD after one month, first visit, 2nd visit after two months and last visit after six months. Pain was assessed by universal pain assessment tool.

Inclusion Criteria

(1) Patients who met the criteria for migraine were included in this study.

(2) Patient of all ages and both sexes.

Exclusion Criteria

(1) Any patient having focal neurological deficit or optic fundui abnormality.

(2) Any patient having uncontrolled hypertension, ischemic heart disease, nephropathy, heart failure and hepatic impairment.

(3) Any patient having any evidence of any systemic infection.

Observation and Results Among 875 patients 48.34%(n-423) patients were between 21-30 years of age (Table-I), 67.54% (n-591) patients were female (Table-I). Housewives were mostly affected (Table-II), 55.08 % (n-482). Sleep disturbance was present in 59.77% (n-523) cases (Table-III). Duration of pain varied from 4-72 hours (Table-IV), mostly occurred more than 24 hours in 64% of cases (n-560) & frequency of pain was more than 3 attacks per month in 49.71% (n-435) of cases. Aura was present in only 9.4% (n-83), vomiting was present in 72.11% (n-631), photophobia was present in 77.48% (n-678) of patients. 211 (35.70%) patients out of 591 were using contraceptive pills or injection (Table-III). Stress affected most patients (Table-IV) as precipitating factor (82.62%, n-723) . Journey and hot environment affected more than 70% of patients .There was gradual improvement in patient who were continuing the medication .After one month frequency reduced to 16% and duration reduced to 23.09%. In the 3rd month duration reduced to 48.54% and frequency reduced to 32.56%. After 6 months duration reduced to 68% and frequency reduced upto 79.40%.

Table -I

Demographic Characteristics of the patients

Age in years	Number (n-875)	Percentage
<20	255	29.14
21-30	423	48.34
31-40	109	12.45
41-50	68	7.77
>50	20	2.28
Sex		
Male	284	32.45
Female	591	67.54

Table -II

Distribution of patints with occupational significance (n-875)

Occupation	Number	%
Housewife	482	55.08
Student	263	30.05
Service	53	6.05
Business	35	4
Teacher	27	3.08
Driver	18	2.05

Table-III

Distribution of patients with predisposing factors (n-875)

Sleep disturbance	Num3ber	%
Present	523	59.77
Absent	352	40.22
Contraceptive pill/inj (n=591)	211	35.70

Table-IV

Distribution of patients with characteristics of pain (n-875)

Characteristics of pain		Number	%
Duration of pain	<24hrs	180	20.57
	>24hrs	560	64

	>48hrs	135	15.42
Photophobia	Present	678	77.48
	Absent	192	21.94
Frequency of pain	>2 attack per month	239	27.31
	>3 attack per month	435	49.71
	>4 attack per month	201	22.97
Aura	Present	83	9.4
	Absent	792	90.51
Vomiting	Present	631	72.11
	Absent	244	7.88

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Table-V

Distribution of patients with precipitating factors (n-875)

	Number	%
Stress	723	82.62
Journey	633	72.34
Environmental change- Hot	659	75.31
Cold	216	24.68
Premenstrual	202	34.17
Food	79	9.02

Table-VI

Distribution of patients with side effects of fluxetine (n-23)

	Number	%
Insomnia/restlessness	15	65.21
Tremor	8	34.78

Discussion

Migraine sufferers were five times more likely to develop major depression in a study conducted by the Henry Ford Health system. Those who started the study with depression were three times to develop migraine. BRESLAU and colleagues interviewed 496 adults with migraine and followed up for two years, concluded that both disorders are biologically linked and suggested to look for another while treating one[15]. Tyramine test revealed presence of major depression in 16 patients among 40 migraine patients[16].

A case controlled study with 141 migrainers and 109 control, stress was measured using perceived stress questionnaire and anxiety and depression using hospital anxiety and depression scale. Results indicated that stress and anxiety were higher in migraine group and were above clinical level. Stress is a primordial factor in the triggering and perpetuation of migraine attacks. So, it is necessary to manage stress to improve daily life of migrainers[17]. Another study revealed depression by Beck Depression Inventory in episodic and transformed or chronic migraine when analyzed qualitatively by dependence analysis[18]. In another study among 37 males (36.27%), and 65 females (67.7%), 45.9% patients showed depression with migraine. Life time prevalence of major depression is about three times higher in migraine than control[19]. As migraine headache is linked to anxiety and depression the condition may share common biological causation. Serotonin agonists may be used to combat depression and thereby migraine[20].

Another report involving 1032 patients in headache clinic in USA, 90% diagnosed as migraine and found that depression is more than 32 fold in patients with severe migraine[21]. Few studies showed effectiveness of fluxetine in alleviation of migraine as well as depression. A double blind study done in headache centre, neurological institute, 2nd university of Naples Italia followed up 52 patients for 6 months. Total pain index showed significant reduction ($p<.05$) of pain starting from 3rd month[22]. Another double blind study done in the department of psychiatry in Lousiana state university school of medicine, Shrevepor; followed up patients for 8 weeks, obtained migraine headache score showed significant reduction of headache score starting from 3-4 weeks of treatment[23]. Another small study (n-18) showed fluxetine is useful in migraine[5]. SFluxetine were used in 65 patients in a phase-II double blind placebo controlled study showed reduction of 1.7 attacks per 28 days or 52% on active therapy than placebo and statistically significant in 2nd month ($F= 4.93$; $p=0.033$) and on 4th month ($F=4.55$;$p=0.04$). There was no serious side effect[24]. The possible efficacy of fluxetine in migraine prophylaxis was supported by an open trial. In September 19,1995 Sepracor announced results of phase-II study of S- fluxetine for prevention of migraine. The study demonstrated a statistically significant decrease in attack frequency by Fluxetine. These results were presented on September 17th 1995 at 7th international headache congress in Toronto, Canada .These double blind placebo controlled study were conducted parallelly in 3 sites of UK. All patients received placebo for 1st 4 weeks, following that patients were divided into two groups. One group was having placebo and another group was getting fluxetine 40 mg daily for 12 weeks.

Conclusion

Though fluxetine is not approved by FDA, it is found to be effective by many clinical concensus[25]. Review of literature showed fluxetine is the only SSRI studied for prophylaxis of migraine[26]. Most of other drugs used for prophylaxis of migraine should be used with caution for there side effects like cardiovascular problems, aplastic anaemia, foetal abnormality etc., at least sleepiness is a troublesome complaint for many persons including drivers.On the other hand depression is biologically correlated to migraine, it might be useful to try to combat depression to get rid of migraine. Fluxetine has got no known dangerous side effect. So, it needs larger study to find out the efficacy and possible side effects of fluxetine.

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