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CONTENTS

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ORIGINAL ARTICLES

Association of Serum testosterone Level with Depression in Parkinson's Disease

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Abstract:

Background: *Depression is a non-motor symptom in Parkinson's disease (PD) as well as elderly population due to testosterone deficiency (TD). Because of similarity between depression in PD and for testosterone deficiency, clinician may fail to recognize and treat TD in patients with PD. Objectives: Our aim was to find out the association between serum total testosterone level and depression in men with Parkinson disease.* ***Methods:*** *This was a case control study carried out in the OPD of department of Neurology, Department of Psychiatry and Department of Biochemistry, Bangabandhu Sheikh Mujib Medical University (BSMMU) on total of 64 subjects where 34 were cases and 30 were controls age ranging from 40 to 85 years of age.* ***Results:*** *This analysis showed serum testosterone levels were significantly low in PD patients with depression compared to controls (4.90±1.86 ng/ml in cases and 6.66±1.18 ng/ml in control group with Odds ratio is 2.071; 95% CI 1.587-2.704). Significantly (P <0.05) low levels of serum testosterone were noted in case suffering from depression more than one year (4.56±1.82 ng/ml vs 6.18±1.50 ng/ml). Serum testosterone level was significantly (<0.001) low in stage III PD patients with depression compared to control (4.96±1.41 ng/ml vs 7.30±1.23 ng/ml).* ***Conclusion:*** *Low serum testosterone level is an important factor causing depression in PD patients. Also progression of PD and duration of depression showed strong correlation with serum testosterone level in this study.*

Key words: *Testosterone, depression, Parkinson's disease*

Abbreviation: *PD (Parkinson's disease), TD (testosterone deficiency), OPD (Out patient department).*

Introduction:

Parkinson's disease is a chronic, progressive and degenerative disease of the nervous system affecting the basal ganglia which presents with various motor and non- motor problems. The cardinal motor symptoms are resting tremor, bardykinesia, rigidity and postural instability[1]. The non-motor symptoms of Parkinson's disease are depression, apathy, anxiety, emotional changes, fatigue with loss of energy, pain, memory difficulties with slow thinking, sleep disturbances hyposmia or anosmia and some autonomic disturbances[1,2,3]. Each person with Parkinson's disease will experiences these symptoms differently.

Parkinson's disease begins between 40 and 70 years of age. Incidences of the disease increases with age. Below 40 years of age the incidence is 3-4/100,000 population while above 70 years it is about 500/100,000 population. Males are more affected than females (M: F=3:2)[3].

Depression is a common problem which may appear early in the disease, even before other symptoms are noticed. Depression occurs for long periods of

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time in approximately 40% of persons with Parkinson disease and it can also occur for short bouts[4]. Though mechanism of depression is poorly known, an interaction among biological and psychological factors with genetic predisposition and life history may play an important role. Patients with depression were reported to have faster cognitive and functional decline, a faster progression along the Hoehn and Yahr stages of the illness and an increased need for antiparkinsonian therapy compared with age and gender comparable Parkinson disease patients without depression[5].

Testosterone, the principal hormone of the testes, is a C19 steroid. It is synthesized from cholesterol in leydig cells and formed from androstenedione secreted by the adrenal cortex. The main functions of this hormone are to develop and maintain the male secondary sex characteristics, exert an important protein-anabolic, growth promoting effect, maintenance of spermatogenesis and to exert an inhibitory feedback effect on pituitary LH secretion[6].

Testosterone deficiency occurs in various testicular disorders, disease of brain and some external factors like acute critical illness, burns, major trauma or surgery, use of some drugs like steroids, chronic disease and its treatment, alcohol abuse, and ageing. The commonest cause of testosterone deficiency is ageing. It is the androgen deficiency in adult male that is the largest under diagnosed group of all testosterone deficient individuals[7].

Testosterone deficiency due to ageing affects 20% to 25% of males over the age of 60 years in the general population. Whether due to testicular or brain or ageing, the signs and symptoms as a result of the androgen deficiency are similar. Individuals may exhibit some or all of the features like; changes in mood (fatigue, depression, anger), lethargy, decreased lean body mass, decreased muscle strength, sleep disturbances, decreased body hair (feminization), decreased bone mineral density, decreased enjoyment in life, deterioration in work performance, decreased libido and erectile quality and low or zero sperm in the semen[7].

So male Parkinson disease patients, who have testosterone deficiency, may have features resembling non-motor Parkinsonian symptoms. Because of the similarity between the non-motor symptoms of Parkinson disease and the symptoms of testosterone deficiency, clinicians may fail to recognize and treat testosterone deficiency in patients with Parkinson disease. The identification of testosterone deficiency may have a significant impact on the long term course of the disease.

This study is therefore planned to assess how common undiagnosed symptomatic testosterone deficiency is in our Parkinson disease population and how they correlate with depressive manifestations.

Materials and Methods:

Study population:

This was a case control study done in the out patient department of neurology, Department of Psychiatry and Department of Biochemistry, Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka. Study population was 40 to 85 years of age male patient of idiopathic Parkinson disease with depression as case. Age and sex matched patients of idiopathic Parkinson disease without depression was considered as control. Data were collected by filling up a semi-structured questionnaire by the investigator and also by a self reported questionnaire by the subjects. Serum total testosterone level was measured subsequently of all the subjects to evaluate the association between serum total testosterone level and depression in male Parkinson disease patients.

Statistical analysis:

All data were recorded systematically in preformed data collection form and quantitative data were expressed as mean and standard deviation and qualitative data as frequency distribution and percentage. Statistical analysis was performed by using SPSS for windows version 10.0. 95% confidence limit was taken. Probability value <0.05 was considered as level of significance and <0.001 was considered as level of highly significant.

Results and observations:

A total 64 male subjects were studied. Of them 34 were idiopathic PD with depression and 30 were idiopathic PD without depression. Thirty seven subjects were below 60 years of age (57.81%) and

27 were above 60 years of age (42.19%). The mean age was 59.09±9.45 years in cases and 58.37±10.49 years in control. This is shown in table-I

Table-I

Age distribution of the study subjects

Age (years)	Case (n=34) No.	(%)	Control (n=30) No.	(%)	P value
<60	20	(58.8)	17	(56.7)	>0.862^{ns}
>60	14	(41.2)	13	(43.3)	
Mean±SD	59.09±9.45		58.37±10.49		>0.50^{ns}
Range	41-85		40-80		

Case:Parkinson disease with depression
Control:Parkinson disease without depression
Chi square test/Unpaired Student's 't' test
ns = Not significant

In the case group 20.6% of subjects had been suffering from PD for less than one year and 79.4% were more than one year. In the control group 63.3% have their disease <1 year while rest (36.7%) were suffering for more than one year. This variation is statistically highly significant (p<0.001). The Odds ratio is 6.67 which signifies high incidence of depression in the longer duration (>1 yr) group. The mean duration of disease in case group is 2.48±1.38 years while in the control group it is 1.28±0.96 years. This is also statistically highly significant (p<0.001). This is shown in table-II.

In the case group 41.2%, 29.4% and 29.4% were in stage II, III, IV respectively. In the control group 60%, 33.3% and 6.7% were in stage II, III, IV respectively. No patient was found in stage I status in both groups. This is shown in table-III.

Mean serum total testosterone level in case group was 4.90±1.86ng/ml and in control group is 6.66±1.18 ng/ml. This is statistically highly (<0.001)

Table-II

Duration of Parkinson disease

Duration (years)	Case (n=34) No.	(%)	Control (n=30) No.	(%)	P value	Odds ratio	95% CI
>1	27	(79.4)	11	(36.7)	<0.001***	6.67	0.049 0.458
?1	7	(20.6)	19	(63.3)			
Mean±SD	2.48±1.38		1.28±0.96		<0.001***		
Range	0.17 5.00		0.17 3.00				

Chi square test/Unpaired Student's 't' test
*** = Significant

Table-III

Clinical stage of Parkinson disease (Hoehn & Yahr rating)

Stage	Case (n=34) No.	(%)	Control (n=30) No.	(%)	P value
I	0	(0)	0	(0)	
II	14	(41.2)	18	(60.0)	
III	10	(29.4)	10	(33.3)	>0.05^{ns}
IV	10	(29.4)	2	(6.7)	

Chi square test
ns = Not significant

significant. So Parkinson disease patients with depression have significantly lower mean serum total testosterone level than those with Parkinson disease without depression. Also 17.6% cases had serum total testosterone level below the normal (e"3ng/ml) biochemical range, while in the control group none was found in this range. This is also statistically significant finding ($p<0.05$). The Odds ratio is 2.071. This is shown in table-IV.

Mean serum total testosterone level in the case group who suffered for less than 1 year is 6.18±1.50 ng/ml and in those who suffered for more than 1 year is 4.56±1.82 ng/ml. This finding is statistically significant. The mean serum total testosterone level in the control group who suffered for less than 1 year is 6.60±1.09 ng/ml and in them who suffered for more than 1 year is 6.76±1.36 ng/ml. This finding is statistically not significant. On the other hand, below 1 years of disease duration, mean serum total testosterone level in the case group is 6.18±1.50 ng/ml and in the control group is 6.60±1.09 ng/ml. This finding is also statistically not significant. Above 1 years of disease duration, mean serum total testosterone level in the case group is 4.56±1.82 ng/ml while that in the control group is 6.76±1.36 ng/ml. This finding is statistically highly significant. This is shown in table-V.

The mean serum total testosterone level in the stage-II PD patients is 6.17±1.39 ng/ml in case group and 6.44±1.05 ng/ml in control group. This variation is not significant statistically. The mean serum total testosterone level in the stage-III PD patients is 4.96±1.41 ng/ml in case group and 7.30±1.23 ng/ml in control group. This variation is significant statistically. The mean serum total testosterone level in the stage-IV of PD patients is 3.06±1.28 ng/ml in case group and 5.49±0.35 ng/ml in control group. This variation is not significant statistically. This is shown in table-VI.

Table IV

Status of serum total testosterone level

Serum total testosterone (ng/ml)	Case (n=34) No.	(%)	Control (n=30) No.	(%)	P value	Odds ratio	95% CI
<3 (low)	6	(17.6)	0		<0.05*	2.071	1.587 2.704
3 (normal)	28	(82.4)	30	(100.0)			
Mean±SD	4.90±1.86		6.66±1.18			<0.001***	
Range	0.14 8.22		4.53 9.27				

Chi square test/Unpaired Student's 't' test ; */*** = Significant

Table-V

Effect of duration of Parkinson disease on serum total testosterone level

Duration of Parkinson disease	Serum total testosterone (ng/ml) level Case (n=7)	Control (n=19)	P value
<1 year			
Mean±SD	6.18±1.50	6.60±1.09	>0.10ns
Range	3.35 7.47	4.53 8.77	
>1 year	(n=27)	(n=11)	
Mean±SD	4.56±1.82	6.76±1.36	<0.001***
Range	0.14 8.22	5.24 9.27	
P value	<0.05*	>0.50ns	

Unpaired Student's 't' test

ns = Not significant, */*** = Significant

Table-VI

Effect of clinical stage of Parkinson disease on serum total testosterone level

Clinical stage of Parkinson disease	Serum total testosterone (ng/ml) level		
	Case	Control	P value
Stage II	(n=14)	(n=18)	
Mean±SD	6.17±1.39	6.44±1.05	>0.50^{ns}
Range	4.11 8.22	4.53 8.77	
Stage III	(n=10)	(n=10)	
Mean±SD	4.96±1.41	7.30±1.23	<0.001***
Range	2.79 7.28	5.93 9.27	
Stage IV	(n=10)	(n=2)	
Mean±SD	3.06±1.28	5.49±0.35	<0.05*
Range	0.14 4.96	5.24 5.73	
P value	P value		
Stage II vs III	<0.05*	>0.05^{ns}	
Stage II vs IV	<0.001***	>0.10^{ns}	
Stage III vs IV	<0.01**	>0.05^{ns}	

Unpaired Student's 't' test
ns = Not significant, */**/*** = Significant

Comparing the mean serum total testosterone level between Stage-II and Stage-III of Parkinson disease patients in the case group shows significant variation while that in the control group has not varied significantly. The mean serum total testosterone level between Stage-II and Stage-IV of Parkinson disease patients in the case group have differed highly significantly, while that in the control group have not varied significantly. Comparison of mean serum total testosterone level between Stage-III and Stage-IV of Parkinson disease patients shows significant variation in the case group while that in the control group the variation is not significant statistically.

Discussion:

This study was carried out in the outpatient department of Neurology, BSMMU, Dhaka. A total 64 patients were studied. Among them 34 subjects was Parkinson disease with depression and the other 30 subjects were Parkinson disease without depression. The aim of this study was to detect the association of serum testosterone concentration with depression in male Parkinson disease patients.

The mean age of the cases is 59.09±9.45 years while that of the controls is 58.37±10.49 years. This difference is statistically insignificant. So case and control groups are age matched. In a community study in Sweden, among 1, 47,777 inhabitants, data was analyzed of 170 Parkinson disease cases where mean age of onset was at 65.5 years [8], but that difference disclose the fact of more life expectancy in western countries from balanced nutrition and better health care facilities which likely delayed the later age of onset of Parkinson disease. But our finding is in harmony with another study who found the mean age of his subjects with Parkinson disease was 52.6±14 years [9].

But from this study it would be hard to make any final comment regarding age, as this is a hospital based study (rather than a population based data) where many patients cannot reach or come, female patients were not included and the patients below 40 years of age are excluded.

The duration of illness has affected the patients significantly. Our findings point out that patients who had been suffering from Parkinson disease for prolong period (>1 year) were more likely to suffer from depression than those who had the disease for shorter period (<1 year). The Odds ratio was 6.67 which signifies high incidence of depression in the longer duration (>1 yr) group. So Parkinson disease patients who are prolong (>1 yr) sufferers develop depression 6.67 times more than those who have

been suffering for shorter (<1 yr) period. This result is consistent with a study who found that the frequency and severity of major depression were higher in Parkinson disease patients[10]. Longer duration of Parkinson disease is significantly associated with major depression. This may have several reasons. Early in the disease the motor features respond well to anti-Parkinsonian drugs. But as time progresses, the efficacy of the drugs gradually decay. Also in the advanced stage patients understand that this is an incurable disease which will deteriorate progressively. Economic involvement due to functional loss, disease burden, cost of the medications and a hopeless future may all contribute to the occurrence of depression especially in them who are prolong sufferer. Also depression can occur as part of disease per se. Those who have the longer disease course are more vulnerable to suffer from depression.

No significant difference noted in staging of Parkinson disease between the case and control groups. So the case and control groups are matched on this ground also.

This study revealed an inverse relation with mean serum total testosterone level and severity of depression. The more is the deficiency of serum total testosterone, the more severe depression the person suffers from. The Odds ratio is 2.071 which signify depression took place twice more frequently in PD patients with testosterone deficiency than those who do not have. This finding is consistent with one study where he investigates 3987 men, aged 71 to 89 years over a period of 4 years. He found men with testosterone deficiency had three times the Odds in favor of depression compared to men with normal/ highest level of serum testosterone[11].

Increased duration (>1 yr) of Parkinson disease causes decreasing level of serum total testosterone which is significantly present in Parkinson disease patients with depression. So depression in Parkinson disease may be due to lower levels of serum total testosterone.

It is evident that patients in advanced stage of Parkinson disease have decreasing level of serum total testosterone, which is significantly present in those have depression (i.e. case group). So severity of Parkinson disease is associated with decreasing level of serum total testosterone concentration. This result is steady with the findings of Gomula A (2004) who observed testosterone deficient PD patients in advanced stage and after correction of their testosterone level, their symptoms and signs of depression improved significantly. Decreasing level of serum total testosterone concentration appears to be responsible for the depression as in the control group testosterone concentration has not decreased significantly even with disease severity, keep them out of depression.

Overall 17.6% of Parkinson disease patients with depression had low serum total testosterone level. 5.4% below 60 year age group and 23.5% above 60 year age had testosterone deficiency. Thus this study yields a potentially association between testosterone deficiency and depression.

Conclusion:

Low serum total testosterone concentration is vital for development of depression in PD patients. Duration of disease, advanced stage of PD is also important contributory factor for depression in Parkinson disease patients.

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Bangladesh Journal of Neuroscience 2011; Vol. 27 (2) : 64-68

Evaluation of the effects of Microwave Diathermy in Patients with Chronic Low Back Pain

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Abstract:

Background: *Back pain affects 60-80% of people at some time in their lives. Back pain is the most common cause of sickness-related work absence. Microwave diathermy could be an important treatment modality. Very few studies have been conducted in our country in this regard.*

Aims: *To determine the effects of microwave diathermy in relieving symptoms of chronic low back pain when applied along with other conventional therapies like drug and exercise.*

Methods: *Total 50 patients with chronic low back pain coming to OPD, DMCH were divided randomly into two groups and treated with nonsteroidal anti-inflammatory drugs, exercises, activities of daily living instructions and with or without microwave diathermy. Thereafter, the patients were evaluated weekly. After six weeks of treatment, improvements were observed in both the groups*

Results: *significant difference (P=0) in improvement was found in microwave diathermy group than in non-diathermy group*

Conclusion: *This study suggests that microwave diathermy is effective in treatment of patients with chronic low back pain.*

Introduction:

Low back pain is a symptom complex which affects the area between the lower rib cage and gluteal folds. It is chronic low back pain if it persists for more than three months[1]. Back pain affects 60-80% of people at some time in their lives[2,3]. Most patients have short attacks of pain that are mild or moderate and do not limit activities, but these tend to recur over many years. Most episodes resolve with or without treatment. However, a small percentage of low back pain becomes chronic and causes significant disability [2]. In Western countries, back pain is the most common cause of sickness-related work absence and in the UK 7% of adults consults their GP each year with back pain[1].

Significant immediate pain relief can be afforded by different therapeutic heating modalities. The proposed mechanisms include an increase in nerve conduction velocity, which may contribute to the reduced pain perception that occurs in response to increasing tissue temperature. In addition, heat contribute to reduction in muscle spasm and also reduction in ischemia as a result of vasodilatation[4].

Microwave diathermy is a physical therapy modality that produces deep heating via conversion of electromagnetic energy to thermal energy. Thermal energy is produced by increased kinetic energy of molecules within the microwave Weld. Federal Communications Commission approved frequencies for therapeutic microwave are 915-MHz (wavelength 33 cm) and 2,456 MHz (wavelength12 cm). Average temperatures of approximately 41°C at a depth of 1–3 cm have been demonstrated[2,5]. Specific contraindications to microwave diathermy can be known to be sensitive to increase cell proliferation rates or skin treated in the past 6 months with radiotherapy, ischemia, local thrombosis or defective arterial circulation, impaired cutaneous thermal sensitivity, metal implants, local infections, and

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indwelling electronic equipment, e.g., pumps or cardiac pacemakers[6]. In our study, we utilized 2456-MHz applicators available in the Physical Medicine and Rehabilitation department of Dhaka Medical College Hospital.

Considering the burden of patients attending the Physical Medicine departments and clinics of different government and private medical facilities in Bangladesh with significant low back pain, the conducted studies on this topic to date in the context of a Bangladeshi population appears to be much fewer than adequate. Although multiple high-quality studies have found that exercise results in positive outcomes in the treatment of chronic low back pain[2].

Aims:

The aim of this study was. To determine the effects of microwave diathermy in relieving symptoms of chronic low back pain *when applied along with other conventional therapies like drug and exercise*

Materials and Methods:

A total of fifty subjects (32 females and 18 males, age range 24–76 years) were enrolled in this study during the period of January 2010 to July 2010. The patients were selected randomly based on the following criteria:

Inclusion criteria: The patients of either sex, aged above 20 years and below 80 years, with complaints of significant chronic low back pain affecting activities of daily living, who consented to participate in the study.

Exclusion criteria: The patients aged below 20 years and above 80 years, having low back pain for less than three months, with traumatic, acute and inflammatory etiology, constant progressive pain, present medical history of tuberculosis, carcinoma or systemic corticosteroid use, systemic upset with any spinal deformity, muscle wasting or progressive neurological signs, patients with any complications as well as those unwilling to give consent were excluded.

The participants were initially examined by the same physician. Sociodemographic data including age (years), weight (kg), height (inch), body mass index (BMI, kg/m²), duration of symptoms (month), job and education levelwere obtained. Clinical evaluation was done giving importance to the musculoskeletal and the nervous system and necessary investigations were done. Data for other variables like pulse (beats/min), blood pressure (mmHg), Hemoglobin (g/dl), ESR (mm in the 1sthr), Schober's test etc. were thus obtained. Patient's experience of pain before starting treatment was assessed using the Lattinen test[7] score (Table I).

Figure 1 presents the overall plan of the study. The patients were divided into one of the two groups: Group A (n = 25) served as treatment group and Group B served as control group.

Treatment procedures

Group A received therapeutic microwave diathermy 15 minutes daily along with isometric back muscle exercises, 25 repetitions twice daily and NSAID in the form of Tenoxicam 20 mg once daily for 6 weeks. Group B (n = 25) received the same treatment as for group A except that they did not receive microwave diathermy. Normal activities of daily living instructions were advised to both the groups. Therapeutic exercises were demonstrated to the patients of both the groups by the same physiotherapist in the department and patients were advised to continue that at home for the specified duration.

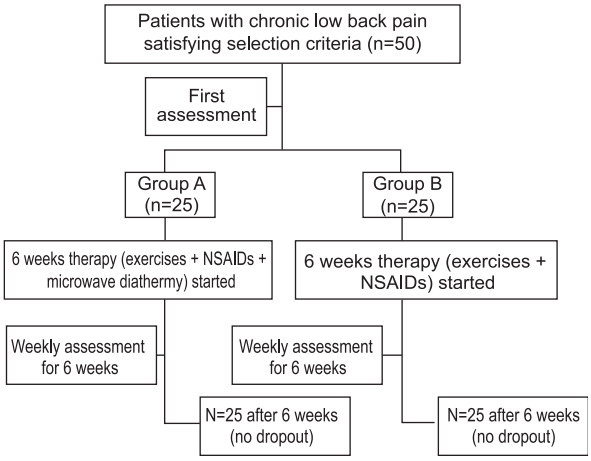

Fig.-1: *Plan of the study and Data collection procedures*

After the treatment of the patients as per schedule, the patients were followed up weekly for six weeks and the outcomes were recorded in the data collection sheet. Improvement was graded Lattinen's test score.

Statistical analysis

IBM SPSS Statistics version 19.0 software package for Windows was used to analyze and present the outcome assessment data and perform statistical tests including the Paired-Samples T test where required to determine the level of significance. The results were expressed as P value and $P<0.05$ was considered as the cutoff for significance.

Results:

There was no statistically significant difference for age, sex, BMI, educational level, jobs and duration of symptoms between the groups (P > 0.05). Basic clinical examination and baseline investigation findings of both groups were also found to be almost identical.

The treatment response assessed at weekly interval by means of Lattinen test score showed gradual reduction in the test score along with clinical improvement with increasing significance as demonstrated by gradual reduction in P value. Pretreatment combined Lattinen test scores in Group A = 9.76 ± 4.465 and in Group B= 9.28 ± 4.523. At the end of 6th week, Group A score was 3.20 ± 4.262 in comparison to Group B score of 5.52 ± 5.599. Treatment failure rate is also 4% lower with microwave diathermy (Table 2). Improvement of Lattinen test score is also significantly associated with application of microwave diathermy at the end of 6 weeks (P=0.006). As compared to conventional drug therapy and exercise, microwave diathermy significantly retards progression and improves symptoms of chronic low back pain at the end of the six weeks therapy as evidenced by more left shifting of area under curve in Figure 2 in case of Group A. Left shifting of area under curve also occurs for Group B (Figure 2), although to a lesser degree. This denotes a notable fall in Lattinen test score value as compared to the pretreatment value depicted in Figure 1, which was quite similar in both the groups.

Table-I

The Lattinen test[7*]

A. Subjective intensity	No pain	0
	Mild	1
	Uncomfortable	2
	Severe	3
	Unbearable	4
B. Frequency	Never	0
	Rare	1
	Frequent	2
	Very Frequent	3
	Continuous	4
C. Analgesics intake	None	0
	Occasional	1
	Moderate	2
	High consumption	3
	Too much	4
D. Disability due to pain	None	0
	Slight	1
	Moderate	2
	Necessary aid	3
	Total dependence	4
E. Sleep	Normal	0
	Sometimes awake	1
	Many times awake	2
	Insomnia	3
	Sedatives needed	4

*Minimum score: 0; maximum score: 20. The score from each group of questions should be added (A – E).

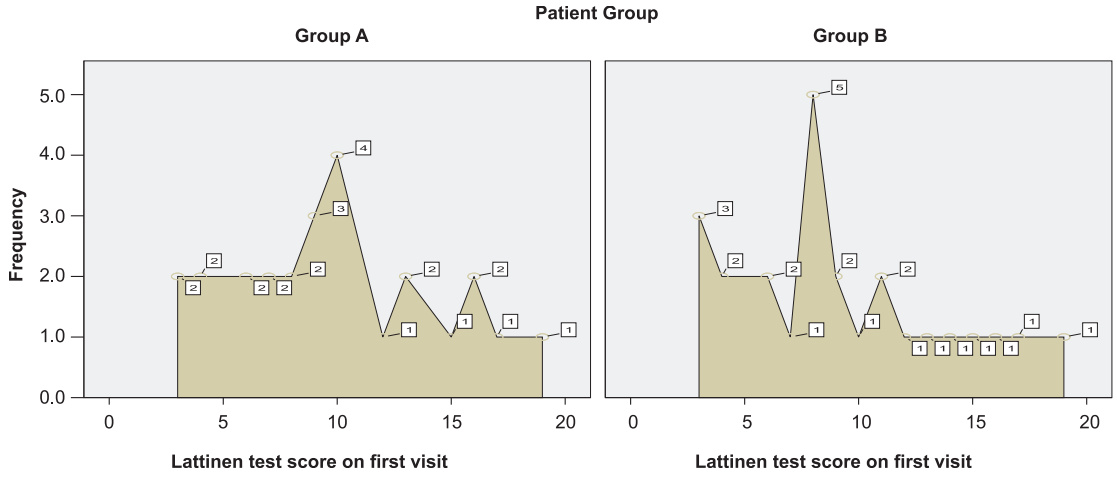

Fig.-2: *Graphical correlation of lattinen test score distribution between Group A and Group B before starting therapy.*

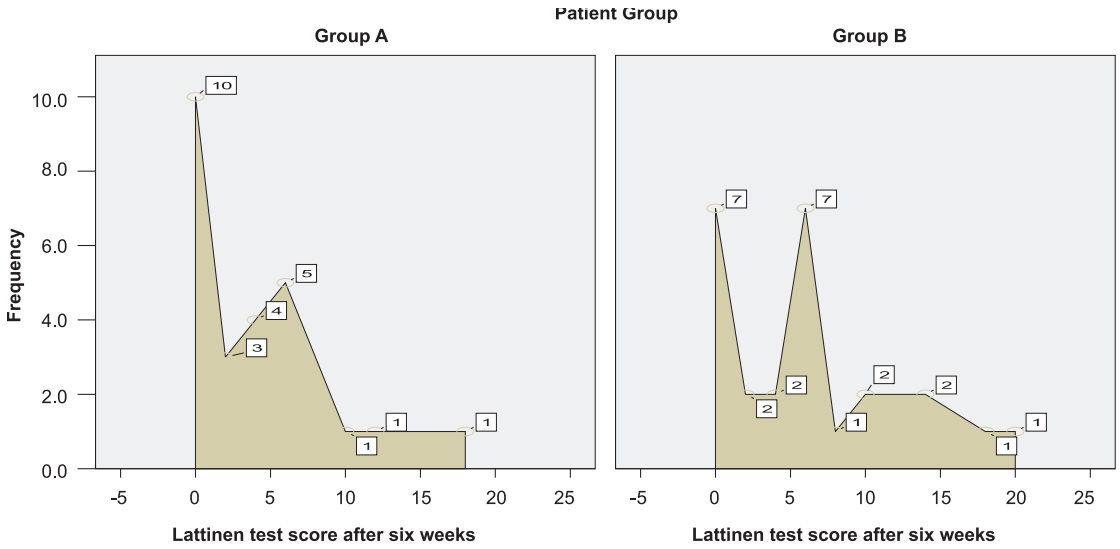

Fig.-3: *Graphical correlation of lattinen test score distribution between Group A and Group B after completing therapy.*

Table-II

Improvement of Lattinen test score with application of Microwave diathermy

	Microwave diathermy given			
	Yes (Group A)		No (Group B)	
	Count	Column N %	Count	Column N %
Improvement and lower Lettinen score	24	96.0%	23	92.0%
No improvement	1	4.0%	2	8.0%

Discussion:

The present study does not discard existing modality of treatments like exercise and drug therapy when comparing them to microwave diathermy. Rather, it shows that microwave diathermy can be an effective adjunct in improving outcome and to some degree, lessen treatment failure rate. The patients of both groups responded well to the treatment, yet a treatment failure rate of about 10% was observed in commensurate to standardized data.[1]A steady trends of improvement in symptoms was observed throughout the whole period of six weeks of study. But, in comparison, the significance of improvement in the group of patients who received microwave diathermy was better than that of non-diathermy group (p=0). The outcome of treatment was unrelated to the initial severity or duration of pain of both the groups.

There appears to be a lack of evidence base regarding use of microwave diathermy in the treatment of chronic low back pain, apparent because of the relative paucity of literature on the issue. In their study, Akyol[6] et al. found no evidence that true MD, as compared with sham MD, is beneficial when applied in addition to some commonly used interventions, including modalities such as superficial heat and exercises. Multiple researches have been conducted on a related deep heating modality namely shortwave diathermy. Zaman[8] reported in a study at IPGMR that partial or complete relief of pain was more in the patients who received shortwave diathermy than the

exercise group or placebo group. Gibson et al. studied 109 patients and significant improvements after treatment were observed in 59% patients who received shortwave diathermy[9]. Shakoor et al. found that there was significant improvement after giving shortwave diathermy on the patients with neck pain.[10] In a meticulous review, Chard and Dieppe indicated that the use of non-pharmacological interventions shortwave diathermy in osteoarthritis is essential for goodmanagement[11]. Ullah showed that improvement was better in the patients who received shortwave diathermy than that of the patients who were not treated with shortwave diathermy[12]. Kerem and Yigiter studied 60 patients and showed significant improvements in measured parameters in shortwave diathermy group after the treatment[13]. Debsarma in a study showed that deep heat modality is more effective than superficial heat in pain management in chronic low back painpatients[14].

Conclusion:

In conclusion, the present study infers that microwave diathermy can be an effective modality in the treatment of the patients with chronic low back pain. However, our effort needs to be clarified with more research works on potential application of microwave diathermy in management of chronic pains.

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Abdominal Obesity is a Risk Factor for Ischemic Stroke

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Abstract:

We carried out case control study aimed to evaluate abdominal obesity as a risk factor for ischemic stroke. Though it became established as a risk factor for cardiovascular disease, still its association with stroke is less clear. We have taken ninety cases with ischemic stroke and compare waist to hip ratio & waist circumference with same numbers of age and sex matched stroke free people as controls. There are standard markers of abdominal obesity & their cut-off values and ways of measurement were taken from International Diabetic Federation. Both increase waist to hip ratio (63.3% in cases & 26.7% in controls) and increase waist circumference (66.7% in cases & 25.6% in controls) were significant (P<.05) and showed marked strength of association (odds ratio >1) in ischemic stroke patients. After adjusting the significant risk factors in all age and sex matched cases and controls by conditional logistic regression analysis, WHR and WC still showed significant strength of association with ischemic stroke in all groups. The increase abdominal obesity markers were found to have greater association in both female and male cases in relation to their control counterparts. So, in the light of current study we may suggest that abdominal obesity defined as increase waist circumference and waist to hip ratio attribute considerably to the estimate of ischemic stroke events.

Key words: *Abdominal obesity, ischaemic stroke.*

Abbreviation: *WHR (West to hip ratio), WC (West circumference)*

Introduction:

Stroke remains the third most common causes of death worldwide, after heart disease & cancer. Two thirds of these stroke cases live in low and middle income countries such as India and this subcontinent[1]. So, we need to look stroke risk factors in Bangladeshi population. Of all stroke cases 85% are ischemic stroke The impact of obesity on public health is a growing concern because obesity is well recognized to be related to many diseases such as type 2 diabetes mellitus, hypertension, dyslipidemia, gall bladder disease, respiratory disease, sleep apnea, and cancer[2,3]. The unfavorable effect of abdominal obesity on coronary heart disease and all-cause mortality is well recognized[4.5,6,7] but its association with ischemic stroke is less clear. Zhang X et al, demonstrated that increasing levels of general or abdominal adiposity consistently predict risk of stroke in predominantly non-obese Chinese women[8], whereas Hu et al found increase association only in men[9], but Suk et al found potent risk factor for ischemic stroke in all race-ethnic groups[6]. In other large scale studies showed that BMI is not a good indicator of stroke. So we've chosen abdominal obesity and to find out its relation with ischemic stroke[6,7,10,11].

Abdominal or visceral obesity emerging as a risk factor for stroke according to various large scale studies worldwide but not the BMI[6,7]. In Bangladesh so far we know, no previous study has yet conducted to observe the association of abdominal obesity with ischemic stroke. So, we aimed to explore the importance of abdominal obesity as a risk factor of ischemic stroke.

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Objectives:
The study was performed among Bangladeshi stroke patients and healthy peoples without stroke to evaluate the association of abdominal obesity as a risk factor for ischemic stroke.

Materials and Methods:
A case control study carried out in Neurology department & other medicine units of Chittagong medical college and hospital from February 2009 to January 2010. The 90 cases were taken from the admitted cases in those departments and diagnosed as Ischemic stroke by CT scan of head. The same number of controls were taken from age and sex matched healthy attendants of the patients admitted in those departments during the study period.

We took the samples by case record form and by purposive sampling technique. We selected the cases and controls of age group >40 years as we excluded young stroke cases from our study. We excluded those people who has other causes of increase abdominal girth like ascites due to any cause, female with pregnancy or any intra abdominal mass. The following risk factors were noted- hypertension, diabetes mellitus, physical inactivity, cigarette smoking & dyslipidemia. The subjects underwent a diagnostic workup that included fasting lipid profile, blood glucose level, CT scan of head. Informed written consent was taken from each patient and control or their relatives with the approved consent form.

Sample size of this unmatched case control study determined by the software named Open Epi version 3.03.17. In both groups we compared waist circumference and waist to hip ratio as abdominal obesity markers along with other examinations and investigations. The cut-off value of waist circumference (³90 cm in men and ³80 cm in women), waist to hip ratio (³.9 in men and ³.8 in women) & waist circumference was measured in standard method, midway between costal margin and the iliac crest. All the measurements were according to International Diabetic Federation (IDF). All the information obtained were noted in a predesigned questionnaire. Collected data were compiled, processed and analyzed with the help of computer based software SPSS (Statistical Package for Social Science) version 15.

Results and observation:
A total number of 90 cases and 90 controls were enrolled in the study.

Table I showed that waist circumference (88.87±13.17 vs 80.19±7.15) & waist to hip ratio (.96±.11 vs.87±.07) are significantly higher in stroke patients than healthy controls (p=<.05). People with increase waist to hip ratio (63.3% vs 26.7%) have significant risk (p=<.05) of having ischemic stroke than those with normal ratio (odds ratio 4.75) [Table II]. Increase waist circumference (66.7% vs 25.6%) also showed significant association (p=<.05) with ischemic stroke patients than controls (odds ratio 5.83) [Table III].

Table-I

Distribution of mean & standard deviation of Waist circumference, Hip circumference and WHR by groups in all cases and controls.

	Group		p value*
	Case (n = 90)	Control (n = 90)	
Waist circumference (in cm)	88.87 ± 13.17	80.19 ± 7.15	$0.001^{\#\#}$
Hip circumference(in cm)	92.76 ± 6.68	92.43 ± 6.57	0.745^{ns}
WHR	0.96 ± 0.11	0.87 ± 0.07	$0.001^{\#\#}$

*t test was done to measure the level of significance. Data was shown as Mean ± SD.
ns = Not significant. ## = Significant.

Table-II

Distribution of WHR by groups in all cases and controls.

WHR	Group		p value*
	Case	Control	
Abnormal	57 (63.3)[#]	24 (26.7)	0.001[##]
Normal	33 (36.7)	66 (73.3)	
Total	90 (100.0)	90 (100.0)	

*Chi square test was done to measure the level of significance.
[#]Figure within parentheses indicates in percentage. Odds ratio (95%CI) = 4.75 (2.52-8.96).
[ns] = Not significant. [##] = Significant.

Table-III

Distribution of WC by groups in all cases and controls.

WC	Group		p value*
	Case	Control	
Abnormal	60 (66.7)[#]	23 (25.6)	0.001[##]
Normal	30 (33.3)	67 (74.4)	
Total	90 (100.0)	90 (100.0)	

*Chi square test was done to measure the level of significance.
[#]Figure within parentheses indicates in percentage. [##]= Significant.

Incidence of increase waist circumference & waist to hip ratio is higher in male cases than controls (p=<.05) [Table IV]. Also higher abdominal obesity markers are found in female cases than controls (p=<.05) [Table V].

Table VI we have made three age & sex matched groups adjusting hypertension in one group, hypertension & diabetes mellitus in another & added physical inactivity with previous factors in last group as they were also found significant other risk factors in our study. By applying conditional logistic regression analysis.

Table-IV

Distribution of mean & standard deviation of Waist circumference, Hip circumference and WHR by groups in male

	Group		p value*
	Case (n = 49)	Control (n = 52)	
Waist circumference (in cm)	90.55 ± 10.72	83.02 ± 7.01	0.001[##]
Hip circumference(in cm)	93.18 ± 6.48	92.67 ± 6.81	0.701[ns]
WHR	0.97 ± 0.09	0.90 ± 0.04	0.001[##]

*t test was done to measure the level of significance. Data was shown as Mean ± SD.
[ns] = Not significant. [##] = Significant.

Table-V

Distribution of mean & standard deviation of Waist circumference, Hip circumference and WHR by groups in female

	Group		p value*
	Case (n = 41)	Control (n = 38)	
WC(waist circumference)	86.85 ± 15.51	76.32 ± 5.36	0.001[##]
Hip circumference(in cm)	92.24 ± 6.96	92.11 ± 6.30	0.926[ns]
WHR	0.94 ± 0.12	0.83 ± 0.08	0.001[##]

*t test was done to measure the level of significance. Data was shown as Mean ± SD. [ns] = Not significant. [##] = Significant.

Table-VI

Association between Anthropometric variables of abdominal obesity and Stroke using all cases and controls by Conditional Logistic Regression Analysis

	{Cases/controls}	Group 1 OR (95%CI)	Group 2 OR (95%CI)	Group 3 OR (95%CI)
WHR (M=>.9, F>.8)	57/24	5.51## (2.75-11.00)	5.47## (2.78-10.77)	4.92## (2.41-10.02)
WC (M=>90cm, F=>80cm)	60/23	7.51## (3.63-15.55)	7.32## (3.45-15.54)	7.21## (3.56-14.61)

Group 1: Matched for age and sex and adjusted for Hypertension
Group 2: Matched for age and sex and adjusted for Hypertension and Diabetes mellitus
Group 3: Matched for age and sex and adjusted for Hypertension, diabetes mellitus and Physical Inactivity.
ns = Not significant. ## = Significant.

Discussion:

In this case control study ninety ischemic stroke patients were compared with same number of age & sex matched healthy controls to evaluate the impact of abdominal obesity as a risk factor for ischemic stroke.

Majority of case were 56.38 with SD ± 8.0 years and of control were 56.12 with SD ± 7.88 years respectively and maximum of cases (41.1%) and controls (42.2%) were enrolled from 50-60 years age group.

Among the stroke patients of current study, 54.4% were male and 45.6% were female. In the current study stroke patients were significantly more hypertensive (53.3% vs. 27.8%) as well as more diabetic than controls (40.0% vs. 23.3%). In our study, 21.1% of cases were smoker in comparison to controls (13.3%). Here also stroke patients were found to be physically less active. We here tried to measure & compare the strength of association of the markers of abdominal obesity i.e waist circumference and waist to hip ratio between ischemic stroke patients and age & sex matched controls, irrespective of presence or absence of other established risk factors. In our current study, waist circumference (66.7% vs 25.6%) and WHR (63.3% vs 26.7%) of ischemic stroke patients were significantly higher than those of controls. Odds ratio of waist to hip ratio and waist circumference were 4.75 and 5.83 respectively which supports the result. Hip circumference was not significantly different as it is an anatomical parameter and as we took cases and controls from almost same regional area it would expected to be in similar range. In our study both WHR and WC association with ischemic stroke was significant ($p<0.05$) when measured by groups in terms of mean and standard deviation.

In the study of Winter et al[7], with conditional logistic regression analysis method, they found strong association of abdominal obesity markers by adjusting other risk factors. Here we adjust hypertension, diabetes & physical inactivity in three combinations as we found those risk factors significant. By conditional logistic regression analysis method, odds ratio of both increase waist to hip ratio remained significant (odds ratio >1) in all three groups. Despite mild attenuation after adjustment of more risk factors, those markers still showed marked strength of association. In our study we found increase waist circumference and increase waist to hip ratio contributed significantly to the occurrence of ischemic stroke in both male and female groups. Hip circumference remained non significant in both groups.

Conclusion:

Through the study results we've found significant association of abdominal obesity with ischemic stroke patients in Bangladeshi population. Here both men and women cases showed equal association. It will be a subject to vast study that, abdominal obesity is directly or indirectly contribute to the incidence of ischemic stroke by influencing other well recognized risk factors. Here we may suggest

that peoples with abdominal obesity have greater chance of having ischemic stroke.

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Prevalence of Analgesic Induced Deafness

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Abstract:

Background: *Analgesic drugs may produce toxic effect on the cochlear system depending on the dose, duration or concomitant renal failure.* ***Objective:*** *Our aim was to find out the relationship between analgesic use and hearing loss.* ***Methods:*** *This was a cross sectional study done combindly between E.N.T. and Neurology department of Bangladesh Medical college among the patients complaining of hearing loss of different degree. One hundred and forty four patients of both sexes having sensory-neural type of hearing loss were included in this study.* ***Results:*** *Out of 944 patients, eighty eight patients were taking tab. Diclofenac (61.11%), thirty six patients were taking Naproxen(25%), and twelve were taking Paracetamol (8.33%) eight were taking Ibuprophen(5.55%). Eighty eight patients were taking the drugs more than two years (61%). All patients were taking analgesic 3-4 times per week. Intensity of damage was severe in 52.77%; moderate damage was evident in 30.55% cases and mild damage found in 16.66% cases. Severity of damage was moderate to severe in most cases of Diclofenac.* ***Conclusion:*** *Analgesic drugs may have toxic effect on hearing.*

Key Words: *Analgesic, deafness.*

Introduction:

Hearing is one of the most precious possessions of man. So prevention of deafness should be the primary target of an Otologist as well as others. Deafness refers to conditions in which individuals are fully or partially unable to detect or perceive at least some frequencies of sound which can typically be heard by members of their species. Conductive deafness indicates impaired hearing due to dysfunction of conduction of sound waves through outer ear, eardrum or bones of the middle ear .Sensory-neural deafness results from diseases of the inner ear specially the cochlea where the sound vibrates and converted into neural signals or any part of the brain which subsequently process the signals[1].

Organ of corti is the end organ of hearing wherein sound is transduced into nerve impulse. It consists of neuro-epithelial cells, called hair cells that rest on the basilar membrane. Sound causes basilar membrane to vibrate and stimulate the hair cells. This stimulation is then transmitted to the cochlear nerve. Basillar membrane vibrates at different frequencies according to the sound stimulus, so the cochlear nerve can differentiate and resolve complexes of sounds[2].

Sensory neural deafness also called neural deafness which is due to the disease of the cochlea or cochlear division of the vestibule-cochlear nerve or brain. Early Sensory-neural deafness is characterized by partial loss of perception of high pitched sounds which can be ascertained by tuning forks of different frequencies but most accurately by the use of audiometer and construction of an audiogram which reveals the entire range of hearing at a glance.

Everyday most persons are taking various types of analgesics for different reasons. Most of them along with analgesia cause significant damage to many organs of the body including hearing. Factors affecting ototoxicity include dose, duration of therapy, concurrent renal failure, infusion rate, and life time dose, co-administration of drugs having ototoxic potential and genetic susceptibility. Elderly and people with pre-existing hearing loss, ototoxic drugs should be used if no alternative is available. Lowest effective dose should be used and levels should be closely monitored[3].

Over 130 drugs and chemicals are reported to be potentially ototoxic and many cause permanent and temporary structural damage of the inner ear. Damage can be of various degree and reversibility[4].

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Materials and Method:

It was a prospective study done among the patients coming for treatment of hearing deficit in Bangladesh Medical College. Neurology and E.N.T. department combindly conducted the study during period of July 2008 to June 2009. Proper history was taken through a structured questionnaire including their job specification and the primary disease for which they were taking the analgesics. Elaborate drug history was taken including their dose, duration and frequency of use .History of concomitant nephropathy was noted. Audiometry was done in each patient and degree of hearing loss was noted.

Inclusion Criteria

1. Patient complaining of deafness was included.

2. Patient taking analgesic for at least twice weekly for six months was included.

Exclusion Criteria:

1. Patient having family H/O deafness was excluded.
2. Patient more than 70 yrs was excluded.
3. Deafness following Meningitis, Encephalitis, and Stroke were excluded.
4. Known cases of C.S.O.M. or ruptured tympanic membrane were excluded.

Results:

A total of one hundred and forty four patients were included in this study. Among them seventy six patients were more than fifty years of age (52.77%) and seventy two patients were male (50%). Forty of them were housewives suffering from headache and backache (27.77%). Thirty two of them were serving in different marketing departments, doing frequent journey mostly by motor cycle and suffering from headache, body ache and backache (22.22%). Seventy two of them were sedentary worker (50%) (Table -I). Thirty two of them were suffering from migraine (22.22%), forty had backache (27.77%), thirty two had diabetic neuropathy (22.22%) and another forty had osteoarthritis, myalgia (28.77%) (Table -II). Eighty eight patients were taking tab. Diclofenac(61.11%), thirty six patients were taking Naproxen(25%), and twelve were taking Paracetamol (8.33%) eight were taking Ibuprophen(5.55%) .Eighty eight patients were taking the drugs more than two years ((61%). All patients were taking analgesic 3-4 times per week. Intensity of damage was severe in 52.77%; moderate damage was evident in 30.55% cases and mild damage found in 16.66% cases. . Concomitant nephropathy was present in twenty patients (table-III).

Table-I

Demographic characteristics of Patients (n=144)

Age	.>50	76	52.77%
	<50	68	475.23%
Sex	male	72	50%
	female	72	50%
Occupation	Housewife	40	27.77%
	Marketing officer	32	22.22%
	Sedentary job	72	50.00%

Table-II

Primary diagnosis of affected patients (n=144)

Migraine	32	22.22%
.Low back pain	40	27.77%
Diabetic neuropathy	32	22.22%
Osteoarthritis / Myalgia	40	28.77%

Table-III

Details of analgesics used (n=144)

Name of analgesic	Diclofenac	88	61.11%
	Naproxen	36	25.00%
	Paracetamol	12	8.33%
	Ibuprofen	8	5.55%
Duration of use	>2 yrs	88	61.11%
	<2yrs	56	38.88%
Severity of Hearing loss	Mild	24	16.66%
	Moderate	44	30.55%
	Severe	76	52.77%

Discussion:

There is strong relationship between age and reported hearing loss. 18% of American adults between 45-64 yrs, 30% between 65-74 and 47% are more than 75 yrs of age having hearing impairment[4]. In 2005 about 278 million people had

moderate to profound hearing impairment, 80% of them used to live in low and middle income countries. The poor suffer more from hearing impairment, because they can't afford preventive and routine care to avoid hearing loss nor the hearing aids to manage the disability[5]. Patients with diagnosed hearing loss in 26,917 men aged 40-74 yrs at baseline in 1986 having regular use of aspirin, NSAID and acetaminophen. regular use of each analgesic was independently associated with increased hearing loss. Multivariate adjusted hazard ratio of hearing loss in regular users (2+ times/wk) compared with men < 2 times per wk, where 1.12 (95% confidence interval, 1.04-1.20 for aspirin, 1.21(95% confidence interval (1.11-1.33) for NSAID and 1.22(95% confidence interval 1.07-1.39 for acetaminophen. For NSAID and acetaminophen risk increased with longer duration and regular use and magnitude is substantially higher in younger men less than 50 yrs of age ,the hazard ratio of hearing loss was 1.33 for aspirin, 1.61 for NSAID and 1.99 for acetaminophen[6].

Study showed the average spectrum of electro-physiological cochleo-neural activity (ASECA) decreased during hrs after salicylet administration. At the end of treatment acoustic tuning of ASECA showed partially decreased sensitivity. Risk of hearing loss was assessed in 26,000 using aspirin, acetaminophen and ibuprofen and followed up every two years for eighteen yrs. Results revealed 33% more hearing loss in regular users <50 and between 50-59 but no association with >60 and older people .For NSAID regular users aged under 50 were 61% and 50-59 32% and 60 and older16% in regular users than non regular users. For acetaminophen regular users <50 99% 50 – 59 38 % and 60 and >60 16%. 6 The hearing loss is slight to moderate, bilaterally symmetrical, affect all frequencies with affection to high frequencies[7].

An n/o NSAID have been reported as inducer of aseptic meningitis specially Ibuprofen and ketorolac. A 70 yr old male presented with acute meningitis 5 times in 6 months with polymorpho-nuclear leucocytosis and raised protein without any evidence of infection or other causes of meningitis[8]. In another case report of a 66yrs old female for sudden bilateral sensory-neural deafness, she was taking Ibuprofen for three months. M.R.I. of brain revealed abnormal enhancement of the dura and basal cisterns. C.S.F. revealed evidence of asceptic meningitis. Audiogram showed new bilateral sensory-neural deafness. Hearing loss and tinnitus resolved and no abnormality was present in M.R.I. when NSAID discontinued[9]. In another case report recurrent aseptic meningitis temporarily occurred with ibuprofen in a 51 yr old white man presented with acute confusion and aphasia. C.S.F. revealed lymphocytic pleocytosis and raised protein level[10].

Side effect like aseptic meningitis is less in case of diclofenac than other NSAIDS. A survey was done in 7.6 million people world-wide by CIBA-GEIGY[11].

Aspirin and other NSAID including diclofenac is associated with tinnitus and hearing loss. In two double blind multi central study, diclofenac compared with aspirin and naproxen in patients with Rh. Arthritis. Significantly fewer patients (p< or equal to 0.05) discontinued the trial due to tinnitus and deafness in case of diclofenac compared to aspirin and naproxen[12]. A 29 yr male were reported to have sensory-neural deafness after using diclofenac for his post operative pain[13]. In one case report an otherwise healthy patient experienced permanent sensory neural hearing loss after a brief period of naproxen[14]. In another study five patients suffered from hearing loss receiving naproxen, only two recovered after discontinuation of drug[15]. Another double blind comparative study between salicylate and diclofenac for 8 weeks in 301 patients, 9 patients discontinued diclofenac due to tinnitus and hearing loss[16].

Conclusion:

Pain is very tough to tolerate specially by busy persons, so they try to get rid of it by either way. But most of them have various side effects including hearing loss. Some study showed hearing improvement after stopping the medicine and few cases not. In many cases it is unpredictable. So there should be a big campaign regarding acknowledgement of the people not to use medicine specially over the counter medicine which is not followed up by any doctor search of toxicity.

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Bangladesh Journal of Neuroscience 2011; Vol. 27 (2) : 78-82

'Invasion of Meningioma Cell in Bony Hyperostosis- an Observational Study of 34 Cases'

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Abstract:

Background: *Meningiomas are usually globular encapsulated tumors. They are extra axial tumors attached to dura and compress the underlying brain without invading it. Abnormalities of bone are frequently encountered in Meningiomas. Hyperostosis or endosotsis are certainly more common than destruction of bone.* Aims and Objective: *Aim of study was to observe the percentage of tumour cell invasion in to the hyperostosis part of intracranial meningiomas.* ***Materials and methods:*** *This is an observational analytic study. Sample size was 34. Place of study was department of Neurosurgery of Square Hospitals Ltd. All the patients with the histological diagnosis of meningioma were included in to this study.* ***Result:*** *Female patients were predominant. Highest number of patients was from 41 to 50 years age group. Convexity meningiomas were commonest (35%) followed by parasagittal meningiomas. According to histopathological subtype meningothelimatous was commonest (56%) followed by psammomatous. About one fourth meningioma patients (26.47%) presented with hyperostosis. Among the hyperostosis patients in 44.44% patient's cause of hyperostosis was due to tumour cell invasion into hyperostosis part.* ***Conclusion:*** *Tumour cell invasion is one of the causes of hyperostosis in intracranial meningiomas which was responsible in more than one third cases in this study.*

Key words: *Hyperostosis, tumour cell invasion, meningioma.*

Introduction:

Meningiomas are usually globular encapsulated tumors. They are extra axial tumors attached to dura and compress the underlying brain without invading it. Even though invasion of dura and dural sinuses are common in meningiomas, they are usually separated from the piamater. Meningiomas are defined as neoplasm composed of meningothelial cells originating from arachnoid cap cells[1].

Meningiomas can occur in any age groups. Its incidence increases with age and occur mainly in middle age and old age. The peak incidence is around 45 years. In adult meningiomas shows female predominance at a ratio of male : female = 1 : 1.4[1].

Abnormalities of bone are frequently encountered in Meningiomas. But it is very difficult to appreciate the exact frequency of bony reaction and / or invasion, because very few series mention this particular aspect. Hyperostosis or endosotsis are certainly more common than destruction of bone, and were found in 25% of Cushing cases[2]. Sosman and Putnam reported Roentgenological "osteomatous change" in 49% of their cases[3].

An extensive Hyperostosis can occur with a small Meningeal tumor, a fact already pointed by Cushing, who separated hyperostoing 'en plaque' Meningiomas from bone alterations accompanying 'global' or 'en mass' Meningiomas.

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Several theories have been put forward by Patrick J. Derome & A. Visot of department of Neurosurgery of FOCH hospital , Suresnes Cedex, France to explain bone formation and / or destruction. Slight movement of sagittal suture and bregma, including a stimulating effect on the cells of pachymeninges , explain the frequency of hyperostosing en mass meningiomas in the parasagittal area[4]. In vascular theory, a disturbance of circulation in the bone, subsequent to presence of meningioma, is responsible for hyperostosis[5]. Phemister and others suggested that the meningioma irritates the periosteum, leading to osteoblastic proliferation which leads to hyperostosis[6]. Finally , Freedman and Forster demonstrated that " the tumor cells themselves take an active part in the production of the hyperostosis is rather than acting only as relative foreign bodies to stimulate bone growth; tumor cells of meningiomas can produce fibroblasts , osteoblast and osteoclasts or act as the latter two without apparent morphological alteration[7].

Diagnosis of a hyperostosing meningioma is easily made by computerized tomography (CT) scan of brain (Fig.-1) and magnetic resonance imaging (MRI) of brain. Plain X-rays, tomography and CT scan allow the surgeon to determine the boundaries of bone involvement with relative accuracy.

Materials and methods:

This is an observational analytic study titled 'invasion of meningioma cell in bony hyperostosis- an observational study of 34 cases'. Study place was neurosurgery department of Square Hospitals Ltd. Total number of cases were 34 (N=34). All the patients with the histological diagnosis of meningioma were included in to this study. Patients who didn't consented to be included or histological diagnosis other than meningioma were not included. Study period was May 2007 to April 2010.

Aim of this study was to observe the percentage of tumour cell invasion in the bony hyperostosis in intracranial meningioma patients.

After confirmation of diagnosis as meningioma, patient's data were collected by questionnaire. Meningioma was confirmed by histopathology report and hyperostosis of bone was confirmed by CT scan of brain and intra-operative findings. Hyperostosis area of bone was drilled out and was sent for histopathology to confirm presence of meningioma cell in it.

All questionnaires were collected at the end of data collection and were tabulated according to different parameters. Study results were also compared with those of other studies.

Results:

This is an observational analytic study titled 'invasion of meningioma cell in bony hyperostosis- an observational study of 34 cases'. Total number of cases was 34. Data were tabulated according to different parameters.

Table-I

Distribution according to sex (N=34)

Sex	No. of case	percentage	Ratio
Male	13	38%	1 : 1.6
Female	21	62%	

Table-I shows the distribution according to sex. Most of patients were female. They were 62% of total cases. Male is to female ratio was 1 : 1.6

Table-II

Distribution according to age group (N=34)

Age frequency (years)	No. of case	Percentage
< 20	00	00%
21- 30	01	2.9%
31-40	09	26.5%
41-50	12	35%
51-60	07	21%
>60	05	14.7%

Table-II demonstrates the distribution patients according to age group. Highest number of patients was from 41 to 50 years age group. They were 35% of all patients. Near about one fourth patients were from 31 to 40 years age group. Next common was 51 to 60 years age group followed by more than 60 years age group. There was only one patient in 21 to 30 years age group.

Table- III

Distribution according to location (N=34)

Location of meningioma	No. of case	percentage
Convexity	12	35%
Parasagittal	08	21.5%
Sphenoid wing	05	14.7%
Tentorial	01	2.9%
Petroclival	02	5.8%
Suprasellar	02	5.8%
Olfactory groove	03	8.8%
Falcine	01	2.9%

Table-III presents distribution of all patients according to the location of meningiomas. More than one third patients were operated for convexity meningiomas. Second highest was parasagittal meningiomas which was 21.5% of all meningiomas. Number of sphenoid wing meningiomas was 5. Other locations were tentorial, petroclival, suprasellar, olfactory groove and falcine.

Table-IV

Distribution according to histopathology subtype (N=34)

Histological subtype	No. of case	percentage
Meningiothelimatous	19	56%
Psammomatous	05	14.7%
Atypical	04	11.7%
Angiomatous	04	11.7%
Transitional	01	2.9%
Malignant	01	2.9%

Table-IV illustrates the distribution of all meningiomas according to their histopathological subtypes. More than half (56%) meningiomas were of meningothelimatous subtype. Psammomatous subtype was 14.7%. Number of atypical and angiomatous subtypes was same and they were 11.7% of total cases. Transitional and malignant subtypes had only 1 case in each group.

Table-V

Distribution according to hyperostosis and tumour cell invasion

Number of case	No. of hyperostosis	Percentage of hyperostosis	Number of cases of tumour cell invasion	Percentage of cases of tumour cell invasion
34	09	26.47 %	04	44.44 %

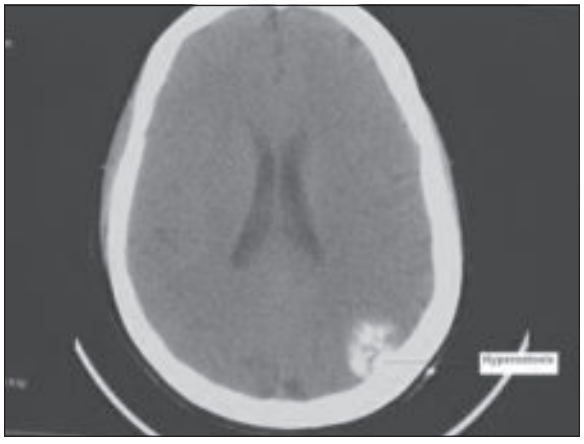

Fig.-1: *CT scan shows meningioma with bony hyperostosis.jpg*

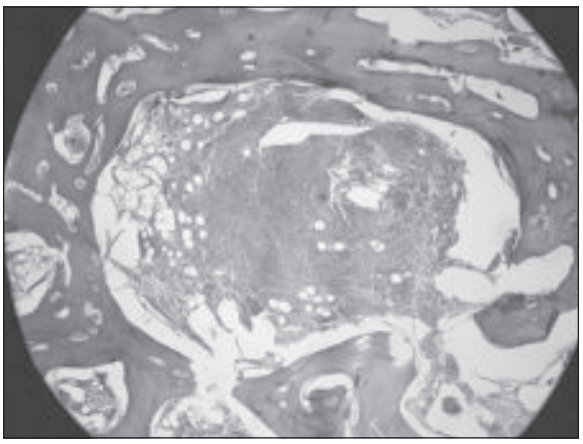

Fig.-2: *Histopathological slide shows meningioma cell inva.jpg*

Table-V shows the distribution of all 34 patients according to bony hyperostosis and tumour cell invasion in the hyperostosis area. About one quarter (26.47%) meningioma presented with bony hyperostosis. Among 9 hyperostosis patients tumour cell invasion was observed in 4 cases. That means 44.44% of bony hyperostosis was due to tumour cell invasion.

Discussions:

This study was performed to observe the percentage of tumour cell invasion into hyperostosis part of intracranial meningioma. Total number of cases was 34.

Bony reaction is a very important clinical presentation for intracranial meningiomas. Sosman and Putnam found 49% hyperostosis cases in their series[3]. Cushing and Eisenhardt found 25% hyperostosis cases in their series[6]. Their study was performed in pre-CT scan time, so confirmation of hyperostosis was done only by X-ray and intra-operative findings. According to the study of Balasubramanium et al they had 17.3% of cases presented with bony reaction[8]. In our series hyperostosis was confirmed in 26.47% of cases. Diagnosis of hyperostosis was made on the basis of CT scan, intra-operative findings and histopathology, but due to early diagnosis of meningioma and short waiting time for surgery number of hyperostosis cases may be less then other studies. More than one fourth intracranial meningioma patients presented with hyperostosis.

Study on relationship between bony reaction and age group are very few in number. Guthric et al distributed hyperostosis cases according to age group[9]. He found that in adult cases 44% of cases were presented with hyperostosis. In our study, all patients were adult to old age group and there was no pediatric age group patient and percentage of patients presenting with hyperostosis was 26.47%.

Pieper et al reported their study on 51 patients with hyperostosis. They found tumour cell invasion in 69% of cases. That means histological examination of the resected bone showed tumor invasion in 35 patients[5]. In our study tumour cell invasion in hyperostosis was observed in 44.44% cases.

There are three types of meningiomas according to malignancy grades: benign (WHO grade I), atypical (WHO grade II), and anaplastic (malignant; WHO grade III) meningiomas. About 80% of all meningiomas are slow-growing tumours of WHO grade I. Any histological variant is compatible with WHO grade I, except for the chordoid, clear-cell, papillary, and rhabdoid meningiomas, which are consistently associated with more aggressive clinical features. The histological variants most commonly diagnosed in pathology specimens are meningothelial, fibrous, and transitional meningioma. Atypical meningiomas constitute 15–20% of meningiomas. Atypical, clear cell and choroids meningiomas are under WHO grade II. Anaplastic meningiomas account for 1–3% of all

meningioma cases. Apart from anaplastic meningiomas rhabdoid and papillary meningiomas are also malignant meningiomas under WHO grade III[10].

According to the study of Jellinger et al meningothelimatous and transitional forms constituted 71.5% of intracranial tumors, fibroblastic forms 7.5% and highly vascularized meningiomas 5.2% of the intracranial tumors, while true “angioblastic” meningiomas (hemangioblastomas and hemangiopericytomas) amounted 3.1% of the intracranial meningiomas, 1.2% were “atypical” (so-called malignant) meningiomas[11]. According to this study meningothelimatous and transitional forms constituted 58.9% of all meningiomas. Angiomatous and atypical subtypes were confirmed in 11.7 % cases respectively.

When the results of this study were compared with those of other studies a fair similarity was observed. There were few limitations of this study; study sample was not very large and comparison was done by significant test.

Conclusion:

In summary it can be said that tumour cell invasion is one of the cause of hyperostosis in intracranial meningiomas which was responsible in more than one third cases in this study.

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Bangladesh Journal of Neuroscience 2011; Vol. 27 (2) : 83-86

Fontanelle as an Indicator of Hydrocephalus in Early Childhood

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Abstract:

Introduction: *The parameters of fontanelle can be very reliable and helpful source to assess the internal status of hydrocephalus in early childhood.*

Methods and Materials: *We have studied the parameters of 69 patients of hydrocephalus in early childhood to assess the status of hydrocephalus. All the patients - who were admitted in the Bangabandhu Sheikh Mujib Medical University from January 2002 to December 2003.*

Results: *All patients were evaluated clinically as well as with imaging study. The age range was from newborn to 12 months. There was male preponderance; male to female ratio was 2.6:1. Clinical evaluation of head and face showed 97.10% patients had open anterior fontanelle, 88.41% had apparent large head, 59.04% had presence of sunset sign. Analysis of anterior fontanelle showed 66.67% had bulged fontanelle, 30.43% had flat fontanelle and 2.90% had concave fontanelle. Evaluation of the clinical status of posterior fontanelle revealed that 23.19% had open posterior fontanelle. Among them 15.94% had bulged and 7.25% had flat fontanelle.*

Conclusion: *By analyzing the result we found that clinical evaluation of fontanelle gives good guidance to assess the status of hydrocephalus in early childhood.*

Key words: *Fontanelle, hydrocephalus, early childhood.*

Abbreviation: *CSF (Cerebro spinal fluid).*

Introduction:

Hydrocephalus may be defined as dilatation of ventricles and/or subarachnoid space due to increase in cerebrospinal fluid volume usually resulting from impaired absorption or flow pathway obstruction or rarely from excessive secretion[1]. It is a hydrodynamic disorder of CSF that leads to an increase in volume occupied by this fluid in the central nervous systems[2]. Theoretically hydrocephalus can result from three mechanisms a) increased production of CSF b) increased resistance of CSF flow c) increased venous sinus pressure[3].

Hydrocephalus has puzzled man since the dawn of civilization. It is one of the common conditions neurosurgeons usually encounter in their day-to-day practice. With the latest advancement of technologies we can diagnose the patients of hydrocephalus far earlier now a days. But about the exact etiology of hydrocephalus the neuroscientists are not very confident in all cases. The incidence of hydrocephalus in new born varies between .12 to 5 per 1000 live births[4]. In Liverpool series it was one per 2500 live birth in another study. The incidence increases among the older primipara family[5]. It has been suggested that 2% of all cases of congenital hydrocephalus may occur in an x-linked recessive state. [6] Cerebrospinal fluid (CSF) is normally a clear colorless fluid with a specific gravity of 1.007 and a pH of 7.33-7.35, 80% is produced by the choroids plexuses, located in both lateral ventricles (accounts for approximately 95%

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of CSF produced in the choroids plexuses) and in the 4th ventricle. Rest of intracranial production occurs mainly in the interstitial space. A small amount may also be produced by the ependymal lining of ventricles. In the spine, it is produced primarily in the dura of nerve root sleeves.

On an average the total CSF volume in the newborn is 5 ml and in adult it is 150 ml (50% intracranial, 50% intraspinal). CSF pressure in the lumber subarachnoid space in a relaxed patient in lateral decubitus position is 9-12 cm of fluid in new born, average 10 cm of fluid in 1 to 10 years children, <18-20 cm of fluid in young adult and 7-15 cm of fluid in adult. CSF production is at a rate of about 0.3ml/min which is approximately 450 ml/24 hrs, which mean that in an adult (with an average 150 ml of total CSF in their body) the CSF is turned over approximately 3 times everyday. CSF is absorbed primarily by arachnoid villi that extend into dural venous sinuses. Other sites of absorption include choroids plexuses and lymphatic. The rate of absorption is pressure dependent[5].

In developed countries the diagnosis of hydrocephalus is usually not very difficult. But in developing countries like Bangladesh it is not always possible for a patient to have hi-tech supported diagnosis due to less availability of facility and lack of economical ability. In this study patients were evaluated as per clinical findings. Assessing their status of fontanelle clinical evaluation of patients of hydrocephalus was done. This study tried to assess the status of hydrocephalus by evaluating parameters of fontanelle and establish its perfectness in the light of imaging study.

Materials and Methods:

This prospective cross-sectional study was done on 69 patients, all admitted in Neurosurgery Department of Bangabandhu Sheikh Mujib Medical University during the study period of 2 years (Jan 2002 - Dec 2003). Clinical & image proved cases of hydrocephalus having open fontanelle who got admitted in Neurosurgery Department of BSMMU during our study period were included in our study.

Thorough clinical history of the patients was taken with the help of structured questionnaire. Patients were evaluated clinically by three experienced residents remaining blind to the findings. Whenever there were dissimilar it has been reassessed by a senior and experienced neurosurgeon of consultant status. Collected data were checked for correctness and editing and coding was done and then data were entered into computer. Employing "SPSS" 11.0 software package analysis was done. Both descriptive and inferential statistics were employed for obtaining results.

Results:

Table-I

Sex distribution of patients (n=69)

Sex	Number of patients	Percentage
Male	50	72.46
Female	19	27.54
Total	69	100

A total number of 69 patients were evaluated. Table-I shows the sex distribution of the patients. In this study out of 69 patients of hydrocephalus 50 (72.46%) were male and 19 (27.54%) were female. The male to female ratio was 2.6:1.

Table-II

Age distribution of patients (n=69)

Age group (months)	Number of patients	Percentage
0-3	22	31.88
4-6	24	34.78
7-9	11	15.94
10-12	12	17.39

The age range of hydrocephalus patients in this study was from newborn up to 12 months. The peak age of hydrocephalus in this population was 4 to 6 months age group. (Table II)

Table-III

Head and Face abnormality

Abnormality	Number of patients	Percentage
Apparent large head	61	88.41
Engorged scalp vein	13	18.84
Open anterior fontanelle	67	97.10
Open posterior fontanelle	16	23.19
Presence of sutural diasthesis	19	27.54
Presence of sunset sign	41	59.42

After clinical evaluation of head and face of all 69 patients of hydrocephalus in this study it was found 67 (97.10%) patients had open anterior fontanelle, 61 (88.41%) had apparent large head, 41 (59.42%) had presence of sunset sign. (Table III)

Table-IV

Status of anterior fontanelle

Status	Number of patients	Percentage
Bulged fontanelle	46	66.67
Flat fontanelle	21	30.43
Concave fontanelle	02	2.90

In this study status of anterior fontanelle was analyzed where 46 (66.67%) had bulged fontanelle, 21 (30.43%) had flat fontanelle and only 2 (2.90%) had concave fontanelle. (Table IV)

Table-V

Status of posterior fontanelle

Status	Number of patients	Percentage
Bulged posterior fontanelle	11	15.94
Flat posterior fontanelle	05	7.25

Evaluation of the clinical status of posterior fontanelle revealed that 16 (23.19%) patients had open posterior fontanelle. Among them 11 (15.94%) had bulged posterior fontanelle and 5 (7.25%) had flat posterior fontanelle (Table-V).

Discussion:

We know that up to a certain age fontanelle remains open. These openings of the fontanelle are a useful window for an experienced clinician to evaluate the internal status of the cranial cavity. Fontanelle is used as an indicator of raised intracranial pressure and also for dehydration in clinical practice. Infants and very young children who have open fontanelle and sutures, the Monro-Kellie Doctrine does not apply because the cranial vault can expand with increased volume[7]. Very few studies were found directly related to hydrocephalus and pattern of fontanelle. We are lacking any such study in our population.

In this study sex incidence showed more than double were male babies. 72.46 per cent were male and 27.54 per cent were female. The male to female ratio was 2.6:1. Preponderance of male babies may be due to more concern and more caring of our society for male babies. As it is a government run hospital based data, which reflects patients of lower socio-economic status. May be this social classes are more careful for their male babies with relative neglect to female one.

The study population has an age range from newborn to 12 months. The peak age of hydrocephalus in this study was 4 to 6 months age group. But most of 708 cases congenital hydrocephalus reported on by Ramamurthi in 1971 were between 9 months to 3 years of age.[8] American Association of Neurological Surgeons mentioned that hydrocephalus can occur at any age but is most common in infants and adults age 60 or older[9].

This study revealed that 97.10 per cent of hydrocephalic early childhood baby had open anterior fontanelle, 88.41 percent had apparent large head and 59.42 per cent had presence of sun set sign. As described by A K Goyel and S K Pandya the characteristic feature of hydrocephalus in infant is large size of head[8].

Describing the sun set sign author mentioned that the eyeballs are rolled downward with supracorneal sclera becoming prominent producing “setting sun sign”. The appearance is primarily due to weakness of upward gaze as the dilated suprapineal recess compresses the quadrigeminal plate. [8] Analyzing the status of anterior fontanelle in this study showed 66.67 per cent had bulged fontanelle, 30.43 per cent had flat fontanelle. Tenseness and bulging of fontanelle is dependable parameter of hydrocephalus. Vidyasagar and Raju suggested a simple non-invasive technique for measuring intracranial pressure using fiber optic transducer placed over the fontanelle in the newborn[10].

The causes of hydrocephalus also determines the pattern of enlargement, the expansion of supratentorial compartment with small posterior

fossa being characteristic of aqueduct stenosis and a ballooning of whole head with prominence of posterior fossa of the Dandy Walker Malformation[8]. This study showed 23.19 per cent had open posterior fontanelle and among them 15.94 per cent had bulged post fontanelle.

Conclusion:

Hydrocephalus is one of the common problems encountered by neurosurgeons in pediatrics population. In early childhood these patients can be easily evaluated by their external parameters and physical findings. Fontanelle is a reliable parameter to understand the internal status of hydrocephalus of patients in early childhood. This small scale study enlighten us with the idea that only parameters of fontanelle could be an ideal way to evaluate these patients when expensive imaging studies are beyond reach, moreover it is a good guide to have preliminary idea before going for imaging study. A large scale study is needed to establish this fact.

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Bangladesh Journal of Neuroscience 2011; Vol. 27 (2) : 87-93

Relationship between Histopathological Subtypes of Intracranial Astrocytoma Patients

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Abstract:

Background : *The study was carried out in the department of neurosurgery, BSMMU, Dhaka during the period of July 2003 to June 2005.* ***Objective:*** *This study was done to elucidate the relationship between age groups and histopathologicas subtypes in case of intracranial astrocytoma patients. For this purpose, a total number of 44 cases were studied.* ***Results:*** *The mean age of all the patients was 33.1 years (range:1-65 years). The highest incidence was found in the age group of 20 years or below group. Male were more commonly effected than female. WHO grade I astrocytoma was the commonest type. Mean age of low grade astrocytoma (WHO grade II) and glioblastoma were nearly similar to other studies, but the mean age of presentation of grade I astrocytomas patients was little late and for anaplastic actrocytoma a little early in comparison to other study.* ***Conclusion:*** *This showed subtype of astrocytoma has definitive relation to age.*

Key words: *Histopathological subtype, astrocytoma, glioblastoma, oligodendroglyma, ependymoima.*

Introduction:

Glial cells are five to ten times more frequent than the trillion brain neurons and compose half the central nervous system (CNS) by volume[1].

Corresponding to the three histologic groups of glial cells are the following three major types of gliomas: i) astrocytoma, ii) oligodendroglima iii) ependymoma[2].

Astrocytomas do not fall within discrete, easily definable categories but instead represent a biologic continuum that ranges from hitologically well-differentiated tumor to poorly or undifferentiated neoplasms with nuclear and cellular pleomorphism, vascular endothelial proliferation and necrosis[3].

The first widely influential system was devised in 1926 by Bailey and Cushing, who divided these neoplasm into three entities according to their histological similarity to normal embryonic glia[4].

In 1949, Kernohan et al, proposed a four tiered system based on the degree of anaplasia[5].

For many years the Kernohan system was the mainstay of pathologic classification of glial tumor and it remains influential today because of its widespread use in the training of pathologists and neuro-pathologists[6,7].

In 1950, Ringertz proposed a three tiered system that was later popularized by Burger et al and used in many cooperative brain tumor clinical trials[8].

The most recently introduced grading system is that of the revised World Health Organization[9].

Pilocytic astrocytomas presumably arise from a class of astrocytes that is inconspicuous in normal brain but may become prominent in reactive gliosis and neoplasia[10].

Incidence all gliomas 5% to 10% of but account for nearly one third of pediatric neoplasm[11].

Pilocytic astrocytomas typically are tumors of children and young adults, most tumors in the cerebellum become symptomatic during the first two decades of life[12].

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Pilocytic astrocytomas are characteristically located around the third and fourth ventricles. Less common locations include the brainstem and basal ganglia[13]. The frontal lobe are the most common location of hemispheric pilocytic astrocytoma[14].

Zulch[11] showed that average age of intracranial astrocytoma was 36.15 years when it was 31.51 years in Dastur's[15] series. According to the study of Mc Kernan and Thoma[16] mean age for low grade astrocytoma was 37.4 years.

Cerebral hemispheres specially frontal (40%), temporal (25%), and parietal (25%) lobes[16]. Others (10%) include thalamus, midbrain and pons.

Anaplastic Astrocytomas are in Kernohan's grade three and in WHO grade III.

Age incidence of glioblastoma multiforme is usually in patients over 50 years and are rare in patients under 30. Glioblastoma can occasionally be found at any age.[2] According to Burger P. C.[17] median age 50 to 60 years.

Incidence of gliomas are 45% to 50%.[18] Any region of CNS possible; cerebral hemispheres predominate (40% frontal, 25% temporal, 25% parietal)[19].

Symptoms of raised intracranial pressure more common than with lower grade tumors.[20] Mental status changes and motor deficit also common. Seizure at presentation is approximately 32%[21].

Methodology:

This is a cross sectional study which was carried out at the Department of Neurosurgery, Bangabandhu Sheikh Mujib Medical University (BSMMU). The study was carried out from July 2003 to June 2005. 44 cases were studied. Inclusion criteria are patients with intracranial astrocytomas admitted in dept. of neurosurgery, BSMMU. Exclusion Criteria are patients of intracranial astrocytomas who denied to be included in the study.

A questionnaire was prepared considering the variables such as age and sex of the patient, clinical features, site of tumor, image findings, per operative findings and histopathological reports. Histopathological report of the tumor was collected and then recorded.

Data were processed through computer software SPSS version 11. Statistical calculations were performed by SPSS software. Chi-square test was applied to test for significance and conclusion was drawn at 1% level of significance.

Results:

The study was done on 44 patients of intracranial astrocytomas, all patients underwent craniotomy or burr hole biopsy to prove the histopathological diagnosis.

Table-I

Distribution of the cases according to age (N=44):

Frequency of age (years)	No. of cases	Percentage
<20	12	27.3
21-30	9	20.4
31-40	11	25
41-50	7	15.9
> 50	5	11.4
Sex		
Male	36	81
Female	8	19

Table I shows the distribution of cases according to age frequency. 12 cases (27.3%) were of 20 or below 20 years, 9 cases (20.4%) were of 21 to 30 years age group, while 31 to 40 years age group comprises of 11 cases (25%).

Table-II

Distribution of the cases by presenting symptoms (N=44)*

Presenting symptoms	No. of cases	Percentage
Headache	36	81.8%
Vomiting	29	65.9%
H/O Convulsion	26	59%
H/O altered consciousness	19	43.2%
Blurring of visual	21	47.7%
Limb weakness	20	45.5%

* Total was not correspond to 100%, because of multiple symptoms in same patient

Table II shows the presenting symptoms of the study patients. More than 80% patients presented with

headache, 65.9% of cases admitted with vomiting, nearly 60% had history of convulsion at least once during the period of illness.

Table-III

Distribution of the patients by status of consciousness (N=44):

GCS score	No. of cases	Percentage
15	38	86.3
13-14	5	11.4
7-12	01	2.3
Total cases	44	100

Table III shows the consciousness level of the patients. 38 patients (86.3%) were found conscious oriented. 5 cases (11.4%) were confused (GCS, 13-14) and 1 patient had GCS 7-12.

Table-IV

Distribution of the cases by speech pattern and gait (N=44):

Speech pattern	No. of cases	Percentage
Normal	35	79.6
Dysphasia	6	13.6
Dysarthria	3	6.8
Gait pattern		
Normal	24	54.5
Hemiplegic	19	43.2
Ataxic	01	02.3

Table IV shows the speech pattern of the patients. Put of 44 patients 35 cases (79.6%) did not have any speech defect. Out of 44 patients 24 (54.5%) had normal gait.

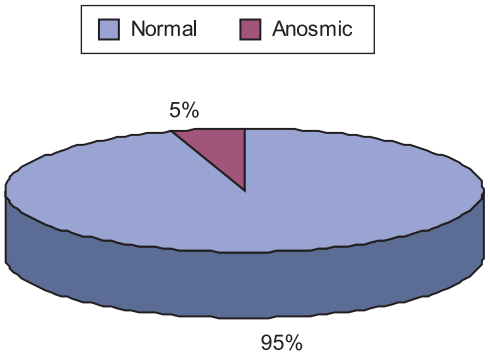

Fig.-2: *Distribution of the cases by olfactory nerve function (N=44):*

Fig.-2. shows most of the cases (95.5%) had normal Olfactory nerve function while only 2 cases (4.5%) had anosmia.

Table-V

Optic nerve function occulomotor, trochlear abducement nerve

Optic nerve function	No. of cases	Percentage
Normal	16	36.4
Defective acuity of vision	08	18.2
Defective field of vision	05	11.4
Papilloedema	17	38.6
Optic atrophy	06	13.6
Oculomotor, trochlear and abducent nerves function		
Normal	40	90.9
Abnormal size of pupil	4	9.1
Extraocular palsy	01	2.3

* Total was not correspond to 100%, because of multiple symptoms in same patient

Table V shows the distribution of patients by the status of optic nerve, 36.4% of the patients had normal optic nerve function while 38.6% had papilloedema and 13.6% had optic atrophy. The functional status of Oculomotor, trochlear and abducent nerves. 90.9% of the patients had normal function, 4 cases (9.1%) had abnormal size of pupil and 1 patient (2.3%) had extraocular muscle palsy.

Regarding the function of Trigeminal, Glossopharyngeal, Vagus, Accessory and Hypoglossal nerves all the patients were found to be normal.

Table-VI

Function of facial nerves (N-44)

Nerves function	No. of cases	Percentage
Facial nerves function		
Normal	34	77.3
Upper motor type deficit	10	22.7
Lower motor type deficit	0	0
Vestibulocochlear nerves function		
Normal	42	95.5
Hearing impairment	2	4.5
Balance impairment	0	0

Table VI shows the function of facial nerves. 77.3% of the patients were found to have normal function of facial nerves whereas 22.7% of them had upper motor type of facial palsy. The function of vestibulocochlear nerves. 95.5% of the patients were found to have normal function and the rest (4.5%) of them had hearing deficit.

Table-VII

Motor Function, sensory function and cerebellar function (N-44)

Function	No. of cases	Percentage
Motor function		
Normal	27	61.4
Hemiparetic	16	36.4
Monoparetic	1	2.2
Sensory function	44	100
Normal	43	97.7
Impaired pain and temperature	0	0
Impaired cortical sensation	1	2.3
Cerebellar function		
Normal	43	97.7
Impaired	1	2.3

Table VII shows the motor function of the patients. 61.4% of the patients did not have any motor deficit, 16 cases (36.4%) had hemiparesis and I patient presented with monoparesis. The sensory function of the patients. 97.7% of the patients did not have any sensory deficit and only 1 patient (2.3%) showed impairment of cortical sensation. The cerebellar function of the patients. 97.7% of the patients had normal coordination of movements and only 1 patient (2.3%) showed impairment of cerebellar function.

Table-VIII

Distribution of the cases according to CT / MRI findings (N-44)

CT / MRI Findings	No. of cases	Percentage
Change in attenuation, no mass effect,no enhancement	1	2.3
Change in attenuation + mass effect ,no enhancement	19	43.2
Enhancement but no necrosis	16	36.3
Enhancement with necrosis (ring enhancement)	8	18.2
Total cases	44	100

Table VIII Imaging study of 19 patients (43.2%) showed change in attenuation, mass effect without any enhancement of contrast agent. Imaging of 36.3% patients showed contrast enhancement without necrosis 18.2% of them showed enhancement with necrosis and imaging of one patient (2.3%) showed only attenuation change without mass effect and enhancement.

Table-IX

Distribution of the patients according to histopathological subtypes (N-44)

Histopathological subtypes	No. of cases	Percentage
WHO Grade I	15	34.1
WHO Grade II	10	22.7
WHO Grade III	9	20.5
WHO Grade IV	10	22.7
Total cases	44	100

Table IX presents the distribution of the patients according to histopathological subtypes. WHO grade I astrocytomas were found in 15 cases (34.1%). WHO grade II were found in 10 cases (22.7%), 9 cases (20.5%) were in WHO grade III and rest of the cases were in WHO grade IV astrocytoma. They were 10 cases (22.7%).

Table-X

Mean age of the different histopathological subtypes (N-44)

Histopathological subtypes	Mean age (years)	Standard deviation	Mean age of all cases
WHO Grade I	23.7	13.30	
WHO Grade II	30.7	5.30	33.1
WHO Grade III	36.4	19.90	
WHO Grade IV	46.5	12.02	

From table X we get the information that mean age of WHO grade I (pilocytic astrocytoma and sub-ependymal giant cell astrocytoma) patients was 23.7 years, mean age of WHO grade II (low grade astrocytoma) was 30.7 years, mean age of WHO grade III (anaplastic astrocytoma) was 36.4 years and mean age of WHO grade IV (glioblastoma multiforme) was 46.5 years. When we calculated

Relationship between age groups and histopathological subtypes:

Table-XI

Cross tabulation of age group and histopathological subtypes (N-44)

			histopathological subtypes				
			grade I	grade II	grade III	grade IV	Total
age of	<20	Observed count	9	0	3	0	12
Patients		Expected count	4.1	2.7	2.5	2.7	12.0
	21-30	Observed count	2	5	0	2	9
		Expected count	3.1	2.0	1.8	2.0	9.0
	31-40	Observed count	3	4	1	3	11
		Expected count	3.8	2.5	2.3	2.5	11.0
	41-50	Observed count	1	0	3	3	7
		Expected count	2.4	1.6	1.4	1.6	7.0
	>50	Observed count	0	1	2	2	5
		Expected count	1.7	1.1	1.0	1.1	5.0
Total		Observed count	15	10	9	10	44
		Expected count	15.0	10.0	9.0	10.0	44.0

the mean age of all the 44 patients it was 33.1 years (SD ±15.6).

Above table IX shows in 20 or less than 20 years age group highest number of cases found in Grade I subtype. In both 21-30 and 31-40 years age group highest frequency was observed in Grade II subtype, in 41-50 years age group highest frequency was in both Grade III and Grade IV and in more than 50 years age group highest number of observation was in Grade III and IV too.

After preparing the above 20 cells table Chi-square (χ^2) test was applied and value of χ^2 comes to 28.46 where degree of freedom =(4-1) X (5-1) or 12. at 12 degrees of freedom table value of is 21.13 when $p < .05$ and 26.12 when $p < .01$.

Here the calculated value of χ^2 is higher than the table value at 1% level of significance ($p < .01$). Hence the test is significant.

So there is association between age group and histopathological subtype in case of intracranial astrocytoma patients.

Discussion

Age incidence of intracranial astrocytomas is a very important variable which varies from study to study. Age incidence of astrocytoma was 31.51 years in Dastur's (1969) series[22].

According to the study of McKeran and Thomas mean age for low grade astrocytomas was 37.4 years, for anaplastic astrocytomas 45.8 years and for glioblastoma multiforme 52 year[23].

According to Youman's study, pilocytic astrocytomas occurred in childhood, low grade astrocytomas are non contrast enhancing area usually present in fourth decades[4].

According to Osborn[2] pilocytic astrocytomas typically are tumors of children and young adult.

In our study we found that mean age of low grade astrocytomas (WHO grade I) was 30.7 years. This is almost similar to other international studies. Mean age of anaplastic astrocytoma (grade III) was 36.4 years, which is little earlier than the other studies. In case of glioblastoma multiforme mean age was 46.5 years, which is near to the study of McKeran and Thomas[24]. Mean age of pilocytic astrocytomas (grade I) was 23.7 years, which is little older than the international studies where it was during the first two decades of life.

In our study mean age of all intracranial astrocytoma patients was 33.1 yaers which is very close to the result of Dastur's (31.51 years) study.

Sex incidents of intracranial astrocytomas also varies from study to study. In our study, we found a

male predominance at the ration of 4.5:1. So, if we compare our study with other international studies, we will find more male predominance.

In this study highest number of cases were found in less than 20 years group and it was 15 cases. The youngest patient was 1 year old and the oldest patient was 65 years old showing that no age group is exempted for intracranial astrocytomas.

In our study the percentage of glioblastoma multiforme was 22.7% which is surprisingly low than the international studies where it was the common of all gliomas. Probably poor prognosis and rapid deterioration may be one of the important cause of not reaching of such types of patients in hospital in proper time. Headache or seizure is sometimes the early or only symptom for intracranial astrocytoma patients which are ignored by a lot of patients or attendant vary frequently. Investigation of intracranial astrocytoma patients (CT/MRI) are very expensive which were also avoided by the patients or their relatives very frequently which is another important cause of not reaching hospital in time.

Analysis of clinical features revealed that presenting symptoms were headache, vomiting, convulsion, altered consciousness, blurring of vision, limb weakness and cranial nerve dysfunction. Most of the symptoms were related to raised intracranial pressure and mass effect by the tumor.

Jackle et al. reported seizure in 65-70% of low grade glioma and 30-50% in glioblastoma patients[24].

Tandon showed apathy, change in personality, impaired memory attention concentration and inappropriate social behavior are the common feature of fronto temporal astrocytomas[25].

In this study we observed 81.8% patients presented with headache, 59% of the patients had history of seizure, 47.7% of them had visual disturbance and limb weakness was present in 45.5% of the patients. So, the presenting symptoms of this study is almost similar to other studies.

McKeran and Thomas reported 50-70% patients with papilloedema.[16] In our study 38.6% of the patients had papilloedema which is very near to that study.

This study showed 77.3% of the patients with normal facial nerve function and 22.7% with upper motor type deficit.

In this study 79.6% patients were found with normal speech, 13.6% with dysphasia and 6.8% with dysarthria. Early dysphasia is often overlooked or confused with memory impairment[25].

In imaging (CT / MRI) study 36.4% patients had complex enhancement, 18.2% had enhancement with necrosis and rest 45.4% without enhancement. Piepmier[26] had shown in a large series that those patients whose tumors enhance on imaging had a poorer prognosis than those whose lesion did not enhance after administration of I/V contrast.

In this study, mean age of WHO grade I is 23.7 years, WHO grade II is 30.7 years, WHO grade III is 36.4 years and WHO grade IV is 46.5 years. So the histopathological subtypes of astrocytomas are related to age group.

Conclusion:

This study has shown that there is significant relationship between age groups and histopathological subtypes in case of intracranial astrocytoma patients.

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Bangladesh Journal of Neuroscience 2011; Vol. 27 (2) : 94-100

Risk Factors of Stroke in Young and Old Age Group Admitted in a Tertiary Level Hospital, Dhaka - A Comparative Study

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Abstract:

Background: *Stroke is an important cause of death and disability. Prevalence of stroke in Bangladesh differs with age. The risk factors in young differ in comparison to old age group. In this study risk factors of stroke in young in comparison to old age group were evaluated.* ***Objectives:*** *To compare the risk factors associated with stroke in young adult and to those of old age group.* ***Methodology:*** *This comparative study conducted in the department of Medicine and Neurology, Sir Salimullah Medical College & Mitford Hospital, Dhaka from January 2008 to June 2009. One hundred two stroke patient of above 15 years of age were confirmed by CT scan or MRI of brain those were included in young and old age groups. The risk factors of stroke were defined in terms of hypertension, diabetes mellitus, dyslipidaemia, ischemic heart diseases, valvular heart disease, history of transient ischemic attack or stroke, smoking and oral contraceptive pill.* ***Results:*** *Of total 102 cases 17 were young adults and 85 old patients between 19 to 100 years. Mean age young adult was 39.76 (± 6.379) and old age was 65.06 (± 11.238). 61.7% were male & 38.2% were female and the ratio was 1.6:1. Amongst male 58.8% patients were smoker in each age group and only 2.9% old patients were alcoholic. Only 17.6% had previous history of transient ischemic attack or stroke in each age group. 58.8% of young and 48.2% of old were hypertensive. 7% old patients had history of ischemic heart disease. 5.9% of young and 2.4% of old patients had valvular heart disease but no patient had vasculitis. 16.7% old patients had diabetes mellitus. 4.9% of total patients (young 11.8% and 3.5% old) were oral contraceptive pill user. Out of all patients 24.7% old patients and 5.9% young adult was dyslipidaemic. Among all patients 66.7% patients had the Ischemic stroke and 33.3% patients had the hemorrhagic stroke. 68.2% old patients and 58.8% young patients had Ischemic stroke; 31.8% old patients and 41.2% young patients had hemorrhagic stroke.* ***Conclusion:*** *In young age group smoking, transient ischemic attack or stroke, hypertension, valvular heart disease, oral contraceptive pill and in old age group smoking, transient ischemic attack or stroke, hypertension, Ischemic heart disease, diabetes mellitus and dyslipidaemia were found significant risk factors for development of stroke. So modification of risk factors may reduce the incidence of stroke.*

Key Words: *Stroke, risk factors, young, old age group.*

Introduction:

It is predicted that stroke will soon become the first cause of death world wide but now it is one of the leading cause of death and disability in developed countries as well as developing countries like Bangladesh. Still now there is no remarkable curative treatment of stroke and stroke related physical disabilities. So we should take appropriate valuable measure against its prevention. The risk factors of stroke differ significantly between young adult and

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old age group. Risk factors of stroke in Bangladeshi population differ from that of other countries. At the same time risk factors in young differ in comparison to old age group.

In a recent study in Bangladesh the prevalence rates of stroke were 2.0, 3.0, 2.0, 10.0, and 10.0 per 1000 within age groups of 40-49 years, 50-59 years, 60-69 years, 70-79 years and 80 years to above age group respectively. Prevalence rates rose with age. People with age range 70- 79 years compared to 40 - 49 years age range is 4.988 (95% CI 2.309 to 10.77) times and people with age range >80 years compared to 40 to 49 years age range is 4.798 (95% CI 1.597 to 14.416) times more likely to have suffered from stroke. Prevalence rate was higher among men compared with women 3.44 and 2.41 per 1000 respectively (Odds ratio=1.425, 95% CI. 95% CI 0.779 to 2.608)[1] .There is no cure in management of stroke but prevention is possible by early detection and reducing the modifiable risk factors for stroke. This is very much important in the context of our country where medical facilities and resource is limited and most of the people live below average condition. With this study risk factors of stroke in young in comparison to old age group will be evaluated.

Stroke is a neurological disease, which is major cause of death and disability worldwide. Stroke kills about five million people each year making this the second major cause of death worldwide. At least fifteen million others have non-fatal stroke annually and about a third are disabled as a consequence[2]. The word stroke is used to refer to a clinical syndrome, of presumed vascular origin, defined by rapidly developing signs of focal or global disturbance of cerebral functions lasting more than 24 hours or leading to death[3]. It is the outward manifestation of a localized sudden interruption of the blood supply to some parts of the brain.

Stroke is predominating in the middle and late years of life. The incidence of stroke increases with age and affects many people in their golden years. It is uncommon below the age of 40 years and more common in male. Death rate following stroke is about 25%[4]. When stroke occurs in the age group from 15 to 45 years, is called stroke in young adults[5]. Stroke in young adults is not uncommon but devastating and frequently no cause can be found. Cerebral infarction accounts for 80 to 85% of cases of stroke and 20 to 15% are caused by intracranial hemorrhages in the western world[6]. Non modifiable risk factors of stroke include age. Sex, family history, race and ethnicity. Modifiable risk factors include hypertension, diabetes mellitus, cardiac diseases (particularly atrial fibrillation), hyperlipidaemia, smoking; transient ischemic attacks, asymptomatic carotid artery stenosis, alcohol abuse and physical inactivity[7]. Stroke in young adults include a wide variety of disorders that are less frequently seen in older age groups. Though there are some overlapping in the risk factors between the two groups but there are some clearly distinct risk factors for stroke in young adults eg. oral contraceptive pill, pregnancy, connective tissue disease with vasculities, hematological variables, drug abuse, smoking, congenital heart disease, family history of stroke, some genetic diseases etc.

Western reports shows that the higher incidence was not explained by a higher prevalence of premature atherosclerotic vasculopathy. Cardioembolization and non-atherosclerotic vasculopathies are relatively important cause of ischemic stoke as compared to atherosclerotic vasculopathy and small artery occlusion.[8] It is important to find out the aetiologic factors and treatment of them adequately for preventing the recurrence. Stroke in young may influence the outcome & may have a dramatic impact on the quality of life in survivors[9]. It is the commonest neurological problems in relation to mortality and hospital admission and long-term disability in most industrialized population, which has a devastating impact on family and nation.

In Bangladesh there is no adequate data on incidence and mortality from stroke as well as young stroke. But the gravity of the situation can easily be assessed by the high incidence of hospital admission.

Methods:

This cross sectional comparative study was carried out in the department of Medicine and Neurology of Sir Salimullah Medical College & Mitford Hospital in Dhaka city from January 2008 - June 2009. For this comparative study One hundred two of above 15 years of age stroke patients confirmed by CT scan or MRI of brain were included dividing young and old age group. Young Adult patient includes age range from 15 to 45 years and old age group includes age range above 45 years. The risk factors of stroke were defined in terms of hypertension,

diabetes mellitus, dyslipidaemia, ischemic heart diseases, valvular heart disease, history of transient ischemic attack or stroke, smoking and oral contraceptive pill. Patients died or dropped out before investigations completed were excluded from the study. Detailed history was taken and thorough clinical examination was done in all cases. Some investigations like CBC with PBF, Blood Sugar, Serum Creatinine, Lipid profile, Serum Electrolytes, ECG, CT Scan of Brain were done in all cases. In Some selected cases MRI of brain, Echocardiography, Duplex study of neck vessels and ANA were done. For Statistical Analysis P value reached from Chi square test and Fisher exact tests to standard statistical analysis by using SPSS 12. Data were expressed as mean ± SD.

Results:

In this study age distribution of patient shows in Table I that out of 102 patients 17 were young adult and 85 were old age group. Ages of young adults were 39. 67 ± 6.37 years and old age group were 65.06 ± 11.24. In Table II Male in young were 41.17% and female were 58.82%, in old age male were 65.88% and female were 34.11%.

Table -III shows the difference of habitual variables in young and old age stroke patients. Many patients had more than one habits. Among 102 patients maximum 58.8% patients were smoker in each age group and only 2.9% patients were alcoholic. Smoking had no significance difference in both age groups for development of stroke.

Table IV shows that 17.6% patients in each age group had the history of TIA or stroke. History of TIA or stroke showed statistically significant in development of stroke but no significant difference between two age groups. In young age group 58.8% and in old age group 48.2% patients had hypertension. Hypertension is statistically significant for development of stroke more in young age group. In old age group 7% patients had IHD but none was in young age group. IHD is therefore significant for development of stroke in old age group. In young age group 5.9% and in old age group 2.4% patients had valvular heart disease is statistically significant for development of stroke in young age group. 20% of old age group was diabetic and young patients were nondiabetic. Diabetes had significant role in developing stroke in old age group. In young female 20% and in old female age group 10.34 % had history of OCP use. OCP showed statistically significant role in developing stroke in young age group. In young adult 5.9% and in old age group 24.7% were dyslipidaemic. Dyslipidaemia is statistically significant to develop stroke in old age group.

Table V shows among young age group 58.8% had ischemic and 41.2% had hemorrhagic stroke. In old age group 68.2% had ischemic and 31.8% had hemorrhagic stroke. Between two groups ischemic stroke is more than that of hemorrhagic stroke in both age groups.

Table-I

Distribution of patients by age (n =102)

	Young Adult	Old Age group
Nnumber of Patients (n)	17	85
Mean age (yrs)	39.76	65.06
Std. Error of Mean	1.547	1.219
Std. Deviation	6.379	11.238
Minimum age (yrs)	19	47
Maximum age (yrs)	45	100

*t test was done to measure the level of significance.

Table-II

Distribution of patients by Sex (n =102)

Sex	Young adult	Old Group	Total	Percent
Male	7	56	63	61.8
Female	10	29	39	38.2
Total	17	85	102	100.0

Table-III

Distribution of habitual variables

Parameters	Young adults (%)n=17	Old age group (%) n=85	P value
Smoking	10(58.8)	50(58.8)	<0.05
Alcohol	0	3(2.9)	<0.05

Chi square test and Fisher exact tests were done to measure the level of significant

Table IV

Distribution of risk factors

Parameters	Young adults (%)	Old age group (%)	P value
History of TIA or stroke	3(17.6)	15(17.6)	<0.001
Hypertension	10(58.8)	41(48.2)	<0.05
IHD	0	6(7)	<0.05
Valvular Heart Disease	1(5.9)	2(2.4)	<0.001
DM	0	17(20)	<0.001
OCP	2(20)	3(10.34)	<0.001
Dyslipidaemia	1(5.9)	21(24.7)	<0.001

Chi square test and Fisher exact tests were done to measure the level of significant

Table-V

Type of strokes

Parameters	Young adults (%)	Old age group (%)
Type of stroke		
Ischemic	10(58.8)	58(68.2)
Hemorrhagic	7(41.2)	27(31.8)

Discussion:

This study done on stroke patients is time demanding since it is considered as coming epidemic as labeled by WHO. In this study 17 'young adults' were included and 85 were included in 'old age group'. Of 17 young adults mean age was 39.76 ± 6.379 years with minimum 19 to maximum 45 years. Of 85 old patients mean age was 65.06 ± 11.238 years ranging from 47 to 100 years. The number of patients in old age group is five times more than that of young age group. The incidence increased with age[10]. This study also correlates with a study on the risk of subarachnoid and intracerebral hemorrhages in blacks as compared with whites that age is the most important risk factor for spontaneous ICH; incidence increases exponentially with advancing age[11].

In this study, female patients were more (10) in number than male (7) in young age group, on the

other hand male patients were almost double (56) than female (29) in old age group. Male and female ratio of stroke patients was 1.61 indicating that stroke is a male predominant disease. In a similar study done previously which was a hospital based study on risk factors for cerebral infarction in terms of hypertension, diabetes mellitus, ischaemic heart disease, smoking, dyslipidaemia, transient ischaemic attacks (TIAs), carotid artery stenosis and family history of stroke. Male were slightly predominant than female (51% vs 49%).[2.] The present hospital based study may not reflect the actual ratio in the community because of social deprivation, superstition and low socioeconomic status and male patients seek more medical attention than female.

Many patients had more than one habit. Among 102 patients maximum 58.8% patients were smoker in both age group and only 2.9% patients were alcoholic. Smoking was found to be a significant risk factor in majority of patient between the two age groups but alcohol was found to be responsible in older age group. This study is similar to that a meta-analysis of relation between cigarette smoking and stroke mentioned that cigarette smoking increases risk (RR) of ischemic stroke nearly two times, with a clear dose-response relation[12]. 17.6% patients in both age groups had the history of TIA or stroke. Previous history of TIA or stroke is significant for development of stroke in both age groups. Our study is similar to that a prospective study on reevaluation of transient ischemic attacks as a risk factor for death and described the average risk of stroke in patients with TIA is about 4%. After adjustment for major cardiovascular risk factors predisposing a patient to stroke. TIA remains a significant independent risk factor for both stroke and myocardial infarction[13]. 50% of total patients had hypertension. In young age group 58.8% and in old age group 48.2% patients had hypertension. Hypertension is statistically significant for development of stroke in young.

Out of 102 patients 5.9% had IHD and all are in old age group. In old age group 7% patients had IHD. IHD is significant for development of stroke in old age group. Myocardial disease has long been recognized as a risk factor for stroke.[10] In the Framingham Study, using multivariate analysis found that risk of stroke was increased twofold by coronary heart disease, threefold by electrocardiographic left ventricular hypertrophy, and threefold to fourfold by cardiac failure[10] .

In young age group 5.9% and in old age group 2.4% patients had mitral valvular heart disease. Mitral valvular heart disease is statistically significant for development of stroke in young age group. Prospective studies with more stringent diagnostic criteria for mitral valve prolapse suggest that the risk of stroke is low in subjects with prolapse uncomplicated by endocarditis or AF. Another valvular risk factor for stroke is mitral annular calcification. In the Framingham Study mitral annular calcification was associated with a doubled rate of stroke. As with mitral stenosis, the presence of AF and mitral annular calcification resulted in an amplification of risk for stroke. With both AF and annular calcification, stroke risk was increased fivefold, compared with a doubling in stroke risk with either factor present alone[14].

20% of old age group was diabetic and young patients were free from diabetes mellitus. Diabetes has significant role in developing stroke in old age group. Our study is similar to that in Glucose intolerance and 22-year stroke incidence that persons with diabetes have an increased susceptibility to atherosclerosis and an increased prevalence of atherogenic risk factors, notably hypertension, obesity, and abnormal blood lipids.The studies of stroke patients and prospective epidemiological studies have confirmed an independent effect of diabetes with a relative risk of ischemic stroke in persons with diabetes from 1.8 to 3.0. Among Hawaiian Japanese men in the Honolulu Heart Program, those with diabetes had twice the risk of thromboembolic stroke of persons without diabetes that was independent of other risk factors. In a population-based cohort in Rancho Bernardo, persons with diabetes had a risk factors adjusted relative risk of stroke of 1.8 in men and 2.2 in women. In Framingham study, persons with glucose intolerance have double the risk of brain infarction in comparison to nondiabetic person[15] .

Out of female stroke patients 20% young female and 10.3% old female were OCP user. OCP has statistically significant role in developing stroke in young age group[16] studied on oral contraceptive pill (OCP) and mentioned that oral contraceptives with an estrogen content >50 µg, the preparations used in the 1960s and 1970s, were strongly associated with risk for stroke. Recently a study of low-dose oral contraceptives (<50 µg estrogen) disclosed no increased risk of stroke in more than 3.6 million woman-years of observation[16].

In young adult 5.9% and in old age group 24.7% were dyslipidaemic. Dyslipidaemia is statistically significant to develop stroke in old age group. This is similar to the study stated that the association between dyslipidaemia and the risk of ischaemic stroke, specially cortical type. In the case control study among other risk factors total serum cholesterol and LDL-cholesterol levels were raised in both cortical and lacunar infarct. HDL-cholesterol levels were significantly low in cases (70%) compared to control subjects (26.7%). Serum triglyceride levels were raised in 60% of case group and 26.7% of control subjects[17].

Among 102 patients majority 66.7% had the Ischemic stroke in both age groups. In young age group 58.8% had ischemic and 41.2% had hemorrhagic stroke. In old age group 68.2% had ischemic and 31.8% had hemorrhagic stroke. Between two groups ischemic stroke is more than that of hemorrhagic stroke in both age groups. This study was consistent to similar that ischaemic stroke is more than that of haemorrhagic stroke[18] ·This study therefore reveals that smoking, TIA, Hypertension, VHD, OCP, IHD diabetes mellitus and dyslipidaemia are the risk factors for development of stroke.

Conclusion:

In this study, in younger age group smoking, TIA, Hypertension, VHD and OCP and in older age group smoking, TIA, Hypertension, IHD diabetes mellitus and dyslipidaemia are the risk factors for stroke. Among the risk factors those are modifiable need special attention for the prevention of stroke.

In this study the risk factors for stroke both in young and old age groups were found hypertension, cardiac diseases, diabetes mellitus, dyslipidaemia, smoking, oral contraceptive pill. All of these risk factors are modifiable and need attention for the prevention of stroke.

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Bangladesh Journal of Neuroscience 2011; Vol. 27 (2) : 101-104

CASE REPORTS

Madelung Deformity - A Case Report with Literature Review.

MD. RAFIQUL ISLAM [1], MOHAMMAD NAJIM UDDIN[2], HASAN ZAHIDUR RAHMAN[3]

Abstract:

Madelung deformity (MD) is a rare autosomaldominant disordercharacterized by epiphyseal growth plate disturbance in distal ulnar sidewhereina progressive ulnar and volar tilt of itsarticular surface occurs in associationwith a dorsal subluxation of the distalulna.Diagnosis of the diseaseis by correlation of clinical data with radiological findings and genetic transmission.In this study, radiographic findings ofa 16 years old female patient progressive bilateral wrist joint deformity and bowing of distal end of forearm without significant functional impairment and systemic complaints and diagnosed as Primary Madelung deformity, is reported.

Keyword: *Wrist Joint, Deformity, Bilateral, Madelung*

Introduction:

Madelung deformity (MD) is a rare inherited disorder involving the epiphysealgrowth plate of distal radial end and consequent deformity as unaffected radial and dorsalportions of the growth plate continue to grow, accounting for 1.7% of all congenital hand anomalies.[1]It can be bilateral in 50 - 66% of patients[2]. It often occurs as rare congenital deformity and does not usually manifest until 10 - 14 yearsand isprimarily found in females[2-4]. One third of cases of MD are transmitted in an autosomal dominant fashionwith variable penetrance. It may also be seen as an acquired consequence of trauma to the growth plate (e.g. Salter V fracture). Henry and Thorburn classifiedMD into 4 different etiologic groups: Posttraumatic, Dysplastic, Chromosomal or genetic (Turner syndrome), andIdiopathic or primary[4]. The underlying cause of this is unclear, with possibilities including local vascular insufficiency trauma, infection (osteomyelitis)or muscular disorders. And sometimes MD associated with Leri-Weill syndrome(autosomal dominant dyschondrosteosis & mesometric dwarfism), Turner syndrome, nail-patella syndrome, diaphyseal aclasis (hereditary multiple exostosis), Hurler mucopolysaccharidosis, achondroplasia.Madelung deformity was first described Malgaigne in 1885 and later in 1878 by OttoWilhelm Madelung, German surgeon (1846 - 1926) as “Spontaneous forward subluxation of the hand”[5-9]. It was defined in terms of radiological findings andgenetic transmission. Prompt and correct diagnosis is very important in orderto ensure correct treatment and to provide the necessary consultationservices to families. Herein, we present a case of MD in a teenage girlpresented with progressivebilateral wrist joint deformity and bowing of distal end of forearm without significant functional impairment and systemic complaints and that was diagnosedwith radiological and clinic findings, and typed by literature review.

Case Report:

A 16-year-old Muslim girl, second of two siblings of consanguineous birth to normal parents, hailing from Korban Ali’s Bari, Jamalpur, ShenbaghThana, Noakhaliand presented withshort stature and progressive bilateral wrist deformities.

She was alright 1 year back, then she developed gradual onset, progressive, painless dinner fork disfiguration of right wrist with associated outward bowing of right forearm and little bits difficulties in writing& gripping.Now for last 5 monthsit’s also involved her left hand with sparing spine & lower limb.

She had no joint pain, morningstiffness, sensory complains and no history of preceding trauma or surgery or delay in achieving mile stone of development.

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3. Associate Professor, Department of Neurology, Bangabandhu Sheikh Mujib Medical University, Dhaka.

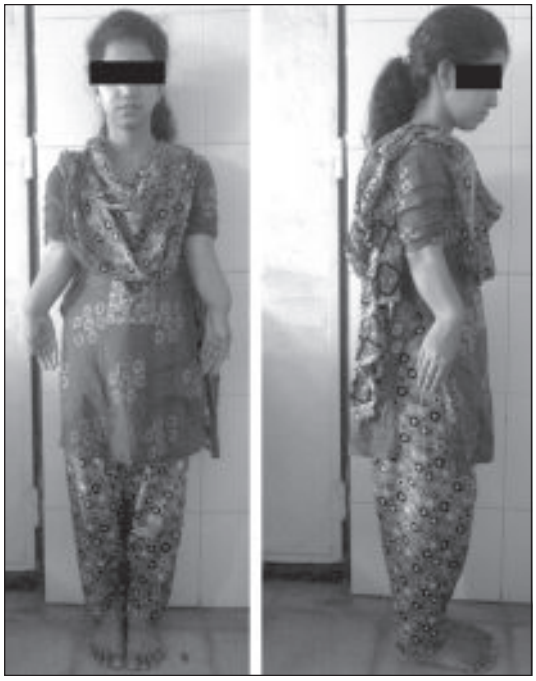

Fig.-1: *Showing mesomelic shortening of both upper limbs with lumbar lordosis.*

She was well cooperative and mentally & emotionally stable with normal vital signs. She was 141 cm tall with US: LSratio of 0.9 and total arm span 113 cm & OFC 53 cm.

She had mesomelic shortening of both upper limbs with lumbarlordosis (Fig. 1). The limbs showedwideningof wrist with garden spade deformity, fixed pronator deformity & bowing andbilateral cubitus valgus with normal warmth, bulk of muscle& no reddening & tenderness on palpation (Fig. 2). There was no limitation inmovement of joints& spine andgait with normal other systemic examination without any organomegaly.

X-ray of hand and wrist were diagnostic of Madelung deformity with lunate subsidence 22 mm (normal < 4 mm), lunate fossa angle 45^0 (normal < 40^0), palmar carpal distance 21 mm (normal < 21 mm) (Fig. 3,4). The present case had ulnar tilt 11^0 which is not fit radiologic criteria of Madelung deformity (e"33^0 in Madelung deformity).

All routine investigations were within normal limit. Some second line investigations also performed and the findings were within normal limit like Serum

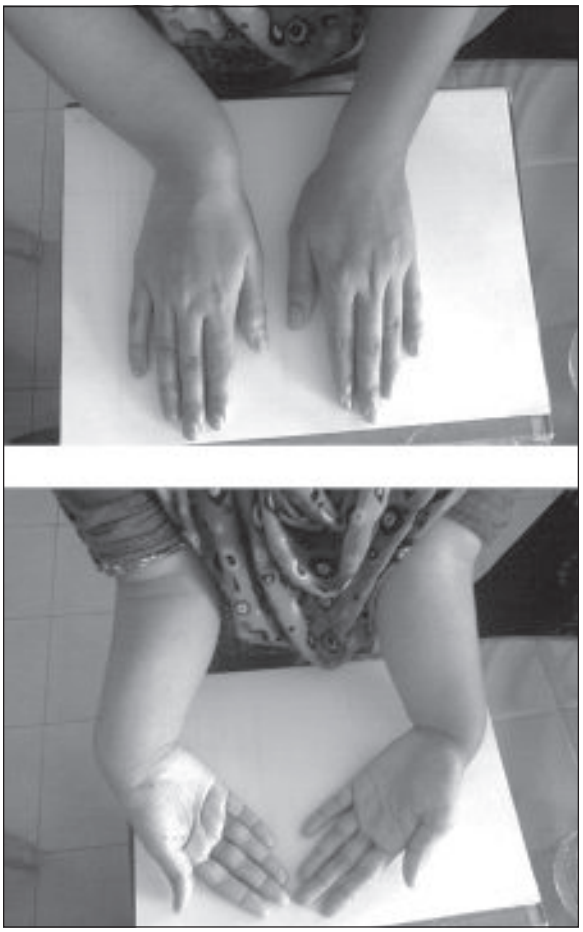

Fig.-2: *Showing widening of wrist with garden spade deformity with radial bowing &bilateral cubitus valguspredominantly in right than left.*

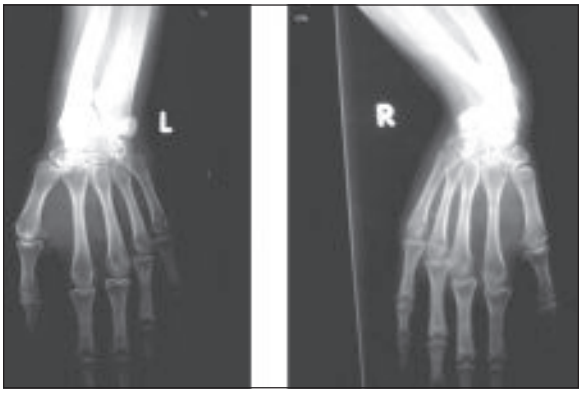

Fig.-3: *X-ray of hand and wrist shows lunate subsidence 22 mm, lunate fossa angle 450 which are diagnostic for Madelung deformity.*

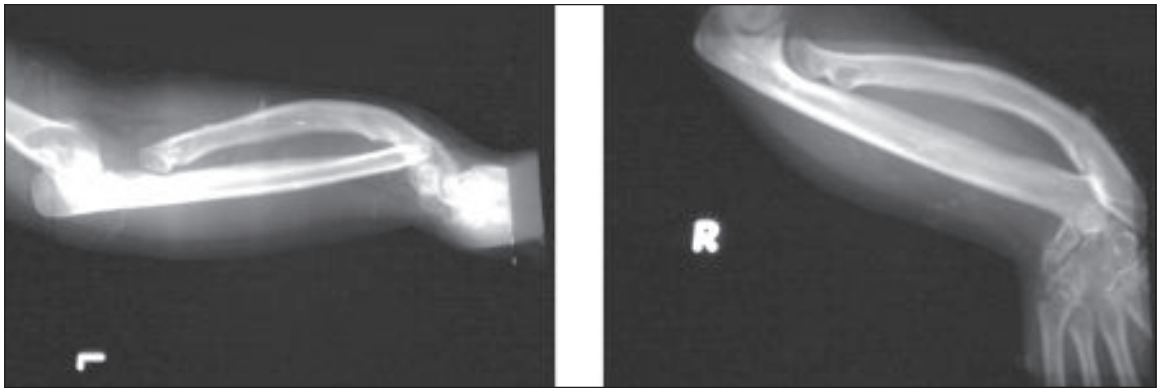

Fig.-4: *X-ray of hand and wrist shows palmar carpal distance 21 mm and dorsal & radial bowing of radius with exaggerated radial inclination.*

calcium 8.7 mg/dl, SerumInorganic Phosphate 4.5 mg/dl, Serum Alkaline Phosphatase 70 U/L, Serum PTH 51.4 pg/ml, normal Ultrasonography of whole abdomen without any organomegaly and normal thyroid status with Serum TSH 1.51 µIU/ml, F T_4 1.18 ng/dl & F T_3 3.14 pg/ml.

She was diagnosed as a case of Madelung deformity of rightwrist and initiation of similar deformity in left wrist.Orthopedics consultation was advised and preparation of surgery was undertaken. But the patients and attendant didnot give consent for operative procedure anddischargedfrom hospital with her own deformity.

Discussion:

The Madelung deformity is relatively uncommon, with prevalence less than 2% and female predominance with female: male ratio is 3 to 5[10,11].

Typically, the deformity is presentbilaterally and seldom manifests clinically before the age of7 years and typically middleto late adolescent onset of the disorder may be linked to theadolescent growth spurt[2-5].

The long-standing and progressive radial deformity graduallyworsens until it is suddenly exacerbated by the increasedgrowth rate, often occurring concurrently with a prematurephysical fusion.The present case had support the above statements.

Radiographic features of MD are characterized by[12,13]

1. dorsal and radial bowing of the radius
2. exaggerated palmar (up to 35°) and ulnar tilt (up to 60°) of the radio carpal articulation 3
3. failure of ossification of the ulnar side of the distal radial epiphysis
4. exaggerated radial inclination
5. Decreased carpal angle below 118°; normal from 118° to 139°.
6. carpal subluxation in a palmar and ulnar direction
7. lunate is gradually forced to the apex of the V-shaped radioulnaocarpal joint
8. “V-shaped” proximal carpal row = herniated proximal carpal row.
9. dorsal subluxation of the distal ulnar and positive ulnar variance
10. wedging of the carpus between the radius and ulna

Management is usually conservative.Persistent pain and/or severe deformitycall for orthopedic surgery involving radialosteotomy. In addition,ulnar shorteningin skeletally immature patients orexcision of distal ulnar head in the skeletallymature is done. Surgical prophylaxisby resection of the abnormal part ofdistal radial epiphysis and its replacementby autologous fat (also known asphysiolysis) have recently been shown torestore growth and minimize deformity.[14-17]In our patients she was asymptomatic but herdeformity was rapidly developing and also creates socialstigma. That’s may be the reason why orthopedicsdepartment wanted to operate on her.

Conclusion:

Madelung deformity is a rare condition that affects thestructure and function of the wrist.one third of disease is transmitted as autosomal dominant fashion and is commonly linked toseveral heritable factors& sometime local traumamay also be responsible. Conservative approach in asymptomaticpatient is the key but improved surgical technique forsymptomatic patients with mature bone are rewarding now a days.

Conflict of Interest: None

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Bangladesh Journal of Neuroscience 2011; Vol. 27 (2) : 105-107

A Frontal Retrobulbar Mucocele with Lytic Lesion at Orbital Plate and Orbital Ridge, Rare Case Report

HARADHAN DEB NATH[1], KANAK KANTI BARUA[2]

Abstract

Mucoceles can occurs when there is obstruction of drainage passage of mucosa of paranasal sinus. Some time it can cause pressure at eye ball and can cause proptosis. A old man admitted at Bangabandhu Sheikh Mujib Medical University with the complaints of right orbital swelling with proptosis of right eye ball. Also complaints of progressive visual bluring & headache for some time. The mucocele was operated by frontal craniotomy and excised completely. As this is a ran case and so few an reported.

Key word: *Mucocele, frontal, headache, proptosis, retrobulbar.*

Introduction:

We reported the case as it is causes unusual submucosal extending with broken orbital plate and orbital ridge.

Mucocele are cystic lesions, with a wall formed by the mucous lining of the sinuses. They arise from the paranasal sinuses when there is an obstruction to the normal draining pathways. Long-standing obstruction leads to outward expansion of the sinus. The bony wall expand and may completely disappear, but the periosteal lining persists. They occur mostly in the frontal sinuses, followed by the ethmoid, maxillary and sphenoid sinuses[1].

Extension of the mucoceles in the orbit can lead to proptosis that may or may not be accompanied by pain. They may cause others symptoms, such as visual disturbance, ocular palsy, and headache.Symptoms are seen mainly with posterior ethmoid and sphenoid mucoceles and may even be associated with neuralgias in the first and second trigeminal divisions.[2] A history of recurrent sinus infection is occasionally elicited.

On computerized tomography scan, the sinus was enlarged and the surrounding cortex appears like a thin, dense line that may be absent in some areas. On MRI, the wall enhances with contrast, whereas the contents have a variable signal, depending on their age. Initially, they have a low protein content (low and high signal intensity on T1 weighted and T2-weighted images, respectively). Subsequently, the protein concentration increase (T1-weighted signal intensity increase while T2-weighted signal intensity decrease more slowly, leading to a bright appearance on T1-weighted and T2-weighted images). With further evolution, the signal intensity drops on both sequences and eventually when the secretions desiccate, the signal becomes darker on both sequences[1].

Treatment is surgical and consists of excising the mucosal lining of the cyst and restoring the drainage pathways of the occluded sinus. Recurrences may occur[1].

Case Report:

A 60 years old male from remote area of Dhaka had been admitted at the Department of Neurosurgery at Bangabandhu Sheikh Mujib Medical University with the complaints slowly progressive swelling at right frontal resion and the swelling extended to forehead and supraorbital region. He also gave the history of dull headache and progressive visual blurring from same duration.

On examination his visual acquity was 6/60, the eye ball was pushed downward and outward. The eye ball movement restricted upward. He also had conjunctival chemosis.

Other neurological examination were normal. On local examination there was proptosis of right eye ball, a

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swelling at right frontal and supraorbital region and consistency was firm. Swelling was non tender, non pulsatile and there was no bruit or marmur over it. There was a bomy gap over the swelling.

All general parameters were normal. Contrast CT scan of head revealed non enhancing isodense lesion at the right frontal sinus extending also to right orbit. CT scan also showed bony destruction over orbital roof and posterior wall of frontal air sinus (Fig.-1). The swelling was operated with bicoronal scalp incision with right frontal craniotomy. There was a suckable tumor seem to be mucocle and was excised completely.

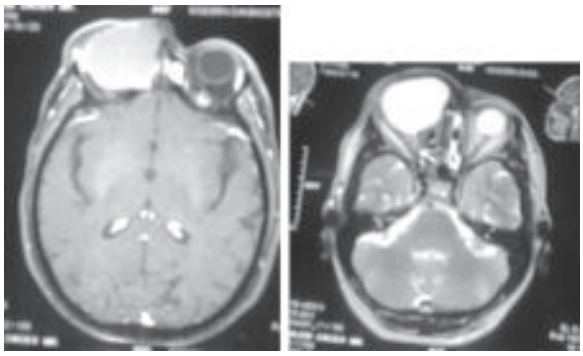

Fig.-1: *Mucocele at the right orbital region*

Sinus cavity was cleaned and packed with povidone iodine solution soaked spongastine and muscle patch and bone wax (Fig.-2 & 3). Mini carnioplasty was done by autologus bone graft from iliac crest. Proper haemostasis was done. A drain was kept in situ which was removed at 1st postoperative day and post operative period was uneventful (Fig.-4).

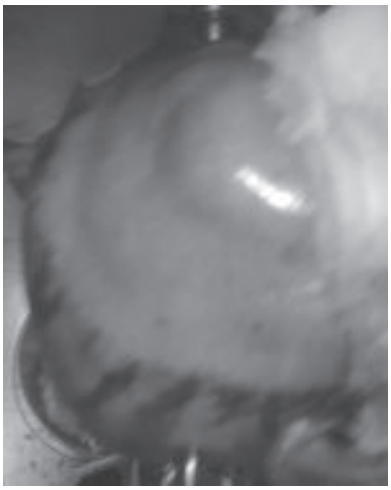

Fig.-2: *Patient under anaesthesia with fronto-orbital mucocele*

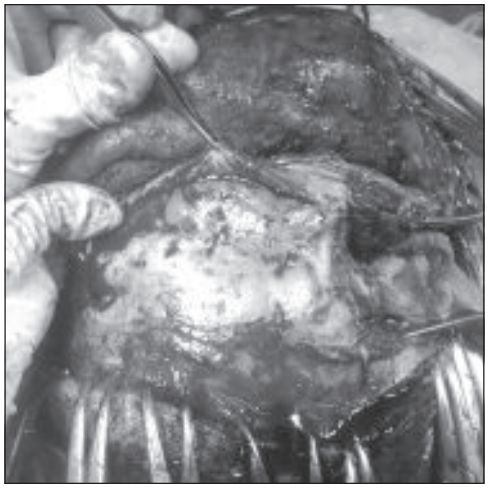

Fig.-3: *Peroperative photograph of mucocele*

Fig.-4: *Postoperative photograph*

Discussion:

Mucoceles commonly involve the frontal sinus, maxillary sinus or anterior ethmoid sinus and rarely the posterior ethmoid or sphenoid sinus[3]. Frontal sinuses are the most common site for mucoceles and these can be frontoethmoidal or frontal only, but bilateral frontal involvement is rare[4,5]. These lesions are usually observed in the fourth to sixth decade of life. No gender preference has been observed. Gradual distension, thinning and erosion of the bony wall of the sinus are caused by progressive accumulation of mucoid material. The

mucocele can extend into the orbit or intracranial compartment by eroding the bony limits and producing bony defects. This may be the most likely pathogenesis in our case secondary to a deviated nasal septum. A ruptured intracranial mucocele can present with meningitis, meningoencephalitis, brain abscess, seizures or cerebrospinal fluid (CSF) fistula.

Mucoceles can present with diminution of vision, visual field defect, diplopia, orbital swelling, retroorbital pain, displacement of eye globe, ptosis, and proptosis[6,7]. Very rarely these lesions can present as a forehead swelling. To the best of authors' knowledge, only two cases with forehead swelling caused by giant frontal mucoceles have been reported in the western literature[3,7]. Tan CSH et al.[7] reported a 33-year-old female presenting with blurring of the inferior visual field in the left eye, associated with periorbital swelling and a painless subcutaneous forehead mass. Akiyama M et al[3] reported a similar case in a 57-year-old female, who presented with a history of an asymptomatic subcutaneous tumor on the forehead for threemonths. Borkar S described mucocele presenting with forehead subcutaneous mass[8].

The swelling was 3 cm in diameter, on the mid- to left-side of forehead. Computerized tomography (CT) images showed a cystic mass demarcated from the subcutaneous area on the forehead caused by expansion of the frontal sinus by the mucocele. Computerized tomography (CT) is diagnostic for paranasal sinus mucocele[9] but magnetic resonance imaging (MRI) is useful in infected cases to find out the exact intracranial extension and to rule out lesions such as chondromyxoma, cystic hypophyseal adenoma, schwannoma and retrobulbar cyst. These are rare but lesion which can easily be treated by surgery. More can be prevented by treatment of blockage of paranasal sinus drainage.

Conclusion:

We reported the case as it is benign lesion and can easily be removed by surgery. Endoscopic sinus surgery combined with transcranial surgery is advisable in case of giant frontal mucocele. Cranioplasty can done by autologus bone graft or methyl methacrylate.

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