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ORIGINAL ARTICLES

Peripheral Neuropathy in Environmental Lead Exposed Children

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Abstract:

Background and objectives: *Peripheral neuropathy has been considered as an infrequent occurrence in children with lead intoxication. This study looked for detection of peripheral neuropathy in environmental lead exposed primary school children.* ***Methods:*** *Blood lead level (BLL) was measured in 107 primary school children in an urban industrial area of Dhaka. BLL range was from 3.6 to 31.9 µgm/ dl. Out of 107, 15 (14%) had normal BLL (<10 µgm/ dl) with mean BLL 6.81 (±2.08) µgm/ dl and 92 (86%) had elevated BLL (e"10 µgm/ dl) with mean BLL 17.76 (±3.89) µgm/ dl.* ***Results:*** *None of the 107 participants had clinical features of neuropathy. Electrophysiological evaluation was done in 31 subjects. Out of 31, 7(22.6%) had normal BLL with mean BLL 7.0 (±2.12) µgm/ dl and 24(77.4%) had elevated BLL with mean BLL 19.8 (±4.79) µgm/ dl. All parameters of NCS were normal in 31 participants both with normal and elevated BLL. Statistical analysis also did not show any significant difference in NCS between the two groups (p value >0.05).* ***Conclusions:*** *This study did not find any clinical or electrophysiological evidence of peripheral nerve dysfunction in the environmental lead exposed children. Because of wide ranges of values of the NCS in the normal subjects and no consistent abnormalities in the NCS of the lead exposed, evaluation of NCS seems to be impractical for early detection of lead neuropathy specially in children with mildly elevated BLL.*

Key words: *Peripheral neuropathy, environmental lead exposure, children*

Introduction:

Lead poisoning is a medical condition caused by increased level of the heavy metal lead in the body. Diagnosis of lead exposure is based on blood lead level measured in micrograms of lead per deciliter of blood (µgm /dl). The United States Centers for Disease Control and Prevention and the World Health Organization (WHO) state that a blood lead level (BLL) of 10 µgm /dl or above is abnormal and a cause for concern[1]. BLL <10 µgm/ dl is considered as normal.

Lead poisoning can occur from both occupational and environmental source[2]. Lead is toxic to many organs and tissues including nervous systems[3]. Lead is much more harmful to children than adults because it can affect children's developing nerves and brains[4]. Peripheral nervous system effects are more prominent in adults and central nervous system effects are more prominent in children[5]. While heavy exposure to inorganic lead is capable of inducing symptomatic neuropathy, subclinical neuropathy is more common[6-9]. The effects of occupational exposure to lead on the nervous system have been described in several previous studies[10-27]. No unanimity has, however been reached, on the lowest exposures at which such effects are found. Traditionally lead poisoning is described as a purely motor disorder[10-14]. Sensory abnormalities have not been a noted feature of poisoning with lead, despite their prominence in other heavy metal poisoning due to arsenic, mercury or thallium[15-21].

Dhaka, Bangladesh, was discovered to have one of the highest air lead level in the world. It was about 453 ngm/m3 in 2000[22]. In one study in 2001, almost 90% of the sampled children had BLL e" 10 ìg/dl in Dhaka[23]. Another study in 2008 found BLLs at the urban industrial area of Bangladesh were significantly higher than those at the rural and urban nonindustrial areas (24.58 ± 10.32, 7.24 ± 6.31, and 2.47 ± 3.32 ìg/ dL, respectively)[24]. Those studies designed to look for level of lead without any clinical correlation. The purpose of this study was to evaluate the peripheral nerve function of environmental lead exposed children of urban industrial area of Dhaka. Cohort study will be performed in the future in these students to find out the long term effect of lead on peripheral nerve.

Materials and methods:

The study was conducted from January 2009 to December 2010 in BSMMU. Study population was

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selected from a primary school, where the tannery industries of Dhaka are located. Blood lead level (BLL) was measured in 107 children of 6 to 12 years age. Patients suffering from diabetes mellitus or hypothyroidism, children with family history of peripheral neuropathy were excluded from the study. Clinical evaluation was done among these 107 students. Despite repeated counseling 31 children agreed to do electrophysiological evaluation. Denial of consent to perform NCS was the primary reason for not performing the test.

BLL was measured by using a portable Lead Care instrument (ESA Inc., Chelmsford, MA, USA) from venous blood (0.5 ml). The instrument has been approved by the US Food and Drug Administration (FDA), and is being used in many health centers in the United States.

A structured questionnaire was used to obtain information from the children regarding symptoms of neuropathy. Clinical examination includes higher psychic functions, cranial nerves, muscle bulk, tone, power, tendon reflexes, plantar response, light touch, pain, vibration sense and blood pressure in the recumbent and standing position.

Nerve conduction studies were performed by a single neurophysiologist by NIHON KOHDON (Neuropack E) machine. Sensory nerve action potentials were recorded using an antidromic stimulus. The amplitudes were measured from the base line to the peak. Surface electrodes were used for recording and for stimulation. NCS was done in cross limb (right upper and left lower limb).

All data was recorded systematically in preformed data collection form and quantitative data was expressed as mean and standard deviation. Statistical analyses of the results were obtained by using window based computer software devised with Statistical Packages for Social Sciences (SPSS-16). Statistical tests for significance of difference were done using student's t test.

Observation and Results:

BLL range was from 3.6 to 31.9 µgm/ dl. Out of 107 clinically evaluated participants, 15 (14%) had normal BLL with mean BLL 6.81 (±2.08) µgm/ dl and 92 (86%) had elevated BLL with mean BLL 17.76 (±3.89) µgm/ dl. Electrophysiological evaluation was done in 31 participants. Out of 31, 7 (22.6%) had normal BLL with mean BLL of 7.0 (±2.12) µgm/ dl and 24 (77.4%) had elevated BLL with mean BLL of 19.8 (±4.79) µgm/ dl. There were no statistical significant difference between the two groups in respect of age, sex and socioeconomic status.

Clinical evaluation for evidence of peripheral neuropathy was done in all 107 students. Symptoms related to peripheral nerve dysfunction were found in none of the subject. Clinical signs of peripheral neuropathy were also absent in all the children.

Motor nerve function was evaluated by motor conduction velocity (MCV), distal motor latencies (DML) and motor amplitude measured in median, ulnar, peroneal, and tibial nerves. All the recordings were within normal level in all groups of students. Statistical analysis also did not find any significant difference in motor NCS between the children with normal and elevated BLL. The results are shown in table I.

Sensory nerve function was evaluated by distal sensory latencies (DSL), sensory amplitudes (SNAP) and sensory conduction velocity (NCV) measured in median, ulnar and sural nerves. All the recordings were within normal level in all groups of students. Statistical analysis also did not find any significant difference in sensory NCS between the children with normal and elevated BLL. The results are shown in table II.

Table-I

Comparison of mean (SD) motor Motor nerve function study between the children with normal and elevated BLL.

Name of nerve	Distal motor latency (ms)			Motor amplitude (mv)			Motor conduction velocity (m/s)		
	BLL <10 µgm/dl	*BLL >10 µgm/dl*	*P value*	*BLL <10 µgm/dl*	*BLL >10 µgm/dl*	*P value*	*BLL <10 µgm/dl*	*BLL >10 µgm/dl*	*P value*
Median	2.77 (0.24)	2.87 (0.41)	0.251 NS	13.50 (4.02)	13.76 (4.50)	0.550 NS	60.23 (6.53)	60.93 (5.75)	0.712 NS
Ulnar	1.89 (0.29)	2.14 (0.38)	0.249 NS	7.47 (1.37)	8.25 (1.86)	0.201 NS	60.56 (5.53)	60.08 (7.79)	0.290 NS
Tibial	3.00 (0.22)	2.92 (0.29)	0.330 NS	19.91 (4.93)	17.87 (4.66)	0.962 NS	51.29 (4.04)	49.76 (4.08)	0.935 NS
Peroneal	3.74 (0.73)	3.64 (0.76)	0.686 NS	4.69 (1.78)	4.81 (1.69)	0.808 NS	58.44 (9.05)	52.98 (7.67)	0.559 NS

NS = not significant, ms = mili second, mv = mili volt, m/s = meter/ second

Table-II

Comparison of mean (SD) sensory nerve function study between the children with normal and elevated BLL.

Name of nerve	Sensory latency (ms)			Sensory amplitude (μv)			Sensory conduction velocity (m/s)		
	BLL <10 μgm/dl	*BLL >10 μgm/dl*	*P value*	*BLL <10 μgm/dl*	*BLL >10 μgm/dl*	*P value*	*BLL <10 μgm/dl*	*BLL >10 μgm/dl*	*P value*
Median	1.97 (0.19)	2.03 (0.22)	0.458 NS	32.42 (7.63)	31.42 (8.35)	0.890 NS	57.44 (8.89)	55.83 (7.86)	0.519 NS
Ulnar	1.70 (0.20)	1.70 (0.22)	0.585 NS	30.57 (7.46)	29.46 (7.82)	0.473 NS	58.77 (2.53)	56.70 (5.86)	0.602 NS
Sural	1.88 (0.16)	1.84 (0.21)	0.187 NS	34.43 (6.48)	32.66 (8.59)	0.458 NS	54.37 (3.30)	53.36 (3.19)	0.708 NS

NS = not significant, ms = mili second, μv = micro volt, m/s = meter/ second

Discussion:

Traditionally the neuromuscular disorder associated with lead poisoning has been purely motor. But most previous studies of exposed workers with raised lead concentrations have not noted clinical neuropathic abnormalities, despite mild electrophysiological abnormalities[18-22]. Some studies showed neuropathic features different from those traditionally attributed to lead toxicity. Their finding of mild sensory and autonomic polyneuropathy contrasts markedly with the traditionally described pure motor disorder, which particularly picks out over used muscle groups such as the wrist extensors[25, 26].

There have been increased abnormal NCVs in lead workers who had BLL within the range of 30-70 μgm / dl in the past. Minor changes in distal motor nerve conduction or CMAP amplitudes have been noted when the level of safe chronic occupational exposure was exceeded despite lack of symptoms[10,18,19,26,27].

After a critical review and meta-analysis on NCS in 32 patients asymptomatic lead workers, Davis and Svendsgaard concluded that there were no substantive differences in NCS between the asymptomatic workers and normal controls[28]. This result is consistent with present study findings as no substantive differences was found in NCS between the asymptomatic rise of BLL and normal controls.

Jeyaratnam et al proposed that the distal segment of the peripheral nerves should show the earliest change[29]. He also suggested that the distal latency is a sensitive indicator of lead neuropathy. Jiann-Horng Yeh (1995)[30] reported that early conduction abnormalities in lead neuropathy are manifested by prolonged distal latencies rather than a slowing of NCV. In this study no abnormalities were found in distal latency. But all the participants were asymptomatic and BLL of them were not as much elevated compared to their study.

Joel Schwartz[21] performed clinical and electrophysiological evaluation in 202 five to nine year-old children living near a lead smelter in Idaho. Blood lead levels ranged from 13 to 97 μgm /dl. There was no clinical feature of neuropathy in those children. But asymptomatic increase of BLL was associated with slowing of nerve conduction velocity in motor nerves.

In this study, the age group was nearly similar (6 to 12 year). BLL was much lower (3.6-31.9 μgm /dl) in the study comparison to previous study. No clinical feature of neuropathy was found in the children; which is similar to other study finding. No difference was found in electrophysiological evaluation; which is contrary to previous study finding. One explanation to this finding is that, BLL of this current study subjects were significantly low.

NCS findings between the normal and elevated BLL group was compared in this study. But none of Joel Schwartz's[21] study group had normal BLL. So he did not evaluate the relationship between normal and elevated BLL with NCS as this study have discussed.

The rise of blood lead level may or may not produce symptoms. Symptomatic neuropathy usually develops

after decade of exposure and with high blood lead level. But the exact time duration and blood lead level causing peripheral neuropathy is not yet established. Most of the previous studies done in subjects with asymptomatic increase of blood lead level did not find any electrophysiological abnormalities comparing to normal subjects. The findings of this study are similar to their findings in this sense.

Conclusion:

The neurotoxic effects of long term lead exposure in asymptomatic lead workers has become a major concern in past decades. Overt lead poisoning is now uncommon in developed countries due to an improvement in occupational hygiene and medical surveillance. But in a developing country like Bangladesh, lead poisoning seems to be a matter of concern both in occupational and environmental aspects. In this study no significant association was found between BLL and neuropathy probably because the BLL of the environmental lead exposed children was not that much raised and the duration of exposure was small to cause neuropathy. There is a plan to reevaluate this cohort in the future periodically.

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A Study on Clinical and Electrophysiological Pattern of Polyneuropathy and their Relationship

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Abstract:

Background: *Polyneuropathy has many different cause and clinical and electrophysiological correlation play an important role in its management. Methodology: This retrospective cross sectional study included purposively selected 80 patients from the department of Neurology during the period of January 2009 to June 2010. History and clinical examination compatible with polyneuropathy underwent electrophysiological examination; patient's symptoms, signs and electrophysiological alterations were graded and analyzed.* ***Results:*** *Mean age of the patients was 34.5 ±6.8 and M: F was 1.8:1. Students, laborer and cultivators were the most affected people. Fifty five percent patients were acute cases and 35% patients were chronic Cases. Thirty percent patient had no known risk factor for neuropathy, 25% patient had antecedent infection, 15% had diabetes mellitus, 7.5% were exposed to drugs/toxins or solvents and 5% had family history of neuropathy. Most of the patient presented with tingling, paresthesia, weakness and difficulty in gait. Deep tendon reflex was hypo/areflexic in 87.5%, loss of muscle power in 75% but only 25% had loss of pain sentation. Thirty seven & half percent patients were in motor type, 10% pure sensory type and 52.5% mixed sensorimotor type. In electrophysiological study following parameter like – MDL, CMAP, MNCV, F wave latency, SDL, SNAP and SNCV considered and median, ulnar, tibial, peroneal and sural nerves were studies. Regarding the electrophysiological type 47.5% were axonal, 27.5% demyelinating and 25% mixed axonal and demyelinating and electrophysiological motor, sensory and mixed sensorimotor category were 47.5%, 7.5% and 45% respectively..In the comparison of clinical and electrophysiological features symptoms and signs of severe and non-severe (mild and moderate) grade were 20(25%), and 60(75%) respectively and electro physiological grades of severe and non-severe (mild and moderate) were 22(27.5%) and 58(72.5%) respectively. Classifications were related for motor and mixed sensorimotor type but not for sensory type. Severities of clinical and electrophysiological grades were not also related and weakness distributions were not also related for demyelinating or axonal type.* ***Conclusion:*** *Variations and relations were found among the clinical and electrophysiological features in polyneuropathy patients.*

Key words: *Peripheral neuropathy, electrophysiology.*

Abbreviation: *MDL (motor distal latency), CMAP (compound motor action potential), MNCV (motor nerve conduction velocity), SDL (sensing distal latency), SNAP (sensory nerve action potential), SNCV (sensory nerve conduction velocity)*

Introduction:

Polyneuropathy is the disorder in which the function of numerous peripheral nerves are affected at the same time and leads to distal and symmetrical deficit with loss of tendon reflexes except when small fibers are selectively affected[1]. It is a relatively common syndrome which is often distressing and sometime disabling or even fatal[2]. Polyneuropathy has an estimated incidence of 25-200/100,000 persons per year and a prevalence of about 5%[3].

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Peripheral nerves have motor, sensory and autonomic component. Nerve fibers (axons) can be classified as either small fibers or long fibers and the distinction has a direct bearing in peripheral neuropathy in terms of symptoms and signs. Large nerve fibers:- are long nerve fibers that are myelinated and enable very fast conduction of impulses to the brain. They carry non-nociceptive information and are not normally associated with pain. Lesions or injury to large fibers can affect many functions including - motor function, vibration perception, position sense, perception of temperature. Symptoms associated with large fibers neuropathy includes -numbness, tingling, weakness, pain, loss of deep reflexes. Small nerve fibers: – some are myelinated and some are not myelinated and each type involves different sensation. They contain nociceptors which are highly sensitive to pain and paresthesia. Small fiber type neuropathy is common in people over 50 yrs of age. Most are idiopathic and are very painful. Symptoms of small fiber neuropathy are many and includes—pain describes as burning, stabbing, prickling, jabbing or lancinating (piercing), sensation of broken glass, burning sands, or ice pick in the bone, tight band like pressure, insensitivity to heat or cold and autonomic dysfunctions related to the organs.

Sensory nerves damage produce symptoms such as pain, numbness, tingling, burning or loss of sensation or feeling. The pain normally begins in feet or hands and progresses the trunk. Lack of sensation can produce cuts or burns unnoticed and ulcer or poor wound healing. The nerve damage in sensory peripheral neuropathy may be found in either large fiber or small fiber. Motor nerves damage results in decreased movement or control of muscle. Since movement is important for health, damage to motor nerve function can also lead to abnormal change in muscle and (wasting) bone, skin other organs. Symptoms usually begin as weakness or heaviness of the hands and or feet and may deteriorate over time. Damage to autonomic nerves affects involuntary body functions such as impaired ability to regulate body temperature, blurred vision, orthostatic hypotension, decrease sweating, dizziness, bowel/ bladder dysfunction, sexual dysfunction etc[4].

Polyneuropathy (PN) as a syndrome has many different causes and worldwide diabetes mellitus is the commonest cause other being are hereditary neuropathy, deficiency of vitamins, B_1, B_{12}, uremia, autoimmune neuropathies, infections including leprosy and HIV, drugs and toxins, porphyria ,paraneoplastic state and 25% cases are idiopathic[5, 6]. About 80% of polyneuropathies are axonal and the remaining 20% are demyelinating. Most of the axonal polyneuropathies are either purely sensory or mixed sensory motor type[2].

Electrophysiological studies play a critical rule firstly to confirm the diagnosis of PN and then to classify it as axonal or demyelinating variety and thereby directing the search for cause[5,7]. Furthermore electrophysiology is quite sensitive to detect sub clinical involvement of either motor or sensory component of apparently pure sensory or pure motor PN giving further clue to the aetiology[8,9].

PN are also classified into acute and chronic form. Acute forms PN are those that have relatively dramatic onset and usually recovers within six weeks. The classical and commonest example of that group is Guillaine Barre Syndrome (GBS). The other less common causes of acute PN are vasculitic, drugs and toxins, porphyria diphtheria, acute idiopathic sensory neuropathies. Chronic PN are those which usually develop over several months. Most of the classical chronic PN which presents with signs symptoms of distal symmetrical way falls into this category. The causes of chronic PN are diabetes mellitus, uremia, alcoholism and other toxins, drugs, underlying neoplasm, hereditary (Charcot Marie tooth disease) and idiopathic[10-12]. After exclusion of common causes of PN routine and specific investigations is the first stage of screening, neurophysiological studies in the form of nerve conduction study (NCS) and electromyogram (EMG) becomes the vital and important way of approach to the underlying cause in the second stage. The subsequent investigation in the third stage depends on the findings of NCS which distinguishes demyelinating from axonal polyneuropathies and divides axonal type into purely sensory, pure motor and mixed sensory motor groups. The cause of chronic demyelinating neuropathies are limited and includes CMTD type-1, chronic inflammatory demyelinating polyradiculoneuropathy (CIDP), multifocal motor neuropathy (MMN) and paraproteinemic demyelinating neuropathy. CIDP is the commonest form of acquired demyelinating neuropathy and affects about 2/100,000 population[13]. Chronic axonal PN has many causes. After NCS confirmation further approach in the third stage should includes investigations to identify cases of diabetes mellitus that were not detected by the

screening 1st stage[14] tests and to show the less common medical condition. Even after extensive investigations about 25% of cases remain idiopathic, which occur mostly in elderly and is often indolent, predominantly sensory and unlikely to become severely disabling[15].

Materials and Methods:

Study design: Cross sectional observational study.

Place of Study: The study was carried out in the department of Neurology and Medicine inpatients and outpatient department of Dhaka Medical College and Hospital, Dhaka.

Study Period: From January 2009 to June 2010 for duration of one and half years.

Sample Size: A total of 80(eighty) subjects were included purposively in this study.

Inclusion Criteria:

1) Patients of 18 yrs to 60 yrs of age.
2) Patients with symptoms and signs of polyneuropathy
3) Electrophysiological investigation compatible with peripheral neuropathy.

Exclusion Criteria:

1) Patient with mononeuropathy, traumatic or entrapment neuropathy.
2) Conditions such as confusional state, pregnancy, skin diseases, oedema, prosthetic device that interfere with electrophysiological investigations.
3) Neuropathy symptoms mimiking specific disease like motor neuron disease, myopathy, muscular dystrophy, spinal muscular atrophy or neuromuscular junction disorder.
4) Patient who were not willing to participate in the study.

Methods:

The study was conducted in the electrophysiology section of neurology department of Dhaka Medical College Hospital. All the study cases were underwent meticulous history (Appendix I) and asked for neuropathy symptoms and risk factors of polyneuropathy and physical examination were done by standard methods. Those Patients who presented within four weeks were defined as acute cases, those presented within 4 – 8 weeks were sub-acute cases and those presented beyond 8 weeks were defined as chronic case. Patients who presented with weakness, wasting and cramps were categorized clinically into motor type and those with tingling, numbness or paresthesias were categorized into sensory type and combination of sensory and motor features were classified into mixed type. Michigan neuropathy screening instrument questionnaire (Appendix II) with scoring system were used for quantitive assessment of symptoms and signs and were graded as mild, moderate and severe.

Quantitative Assessment of symptoms by scoring was as:-

- Sensation felt – Burning/ tingling/ numbness (2 point)

  Fatigue (weakness)/ cramping/ aching (1 point)
- Location of symptoms—Feet (2 point) Calves (1 point)

  Elsewhere (no point)
- Timing of symptoms – Worse at night (2 point) present day and night(1 point) present only during the day (no point)
- How the symptoms relieved – walking around (2 point) standing (1 point) sitting or lying (no point)

  (Maximum 2 point in each)

The total symptoms score were graded as:-

0—2 = normal, 3—4= mild, 5—6= moderate, >7=severe

Quantitative assessments of physical findings were as:-

- Achilles tendon reflex—absent (2 point for each foot) present with reinforcement (1 point for each foot)
- Vibration sense – absent or reduced (1 point for each foot)
- Pin prick sensation— absent or reduced (1 point for each foot)
- Temperature sensation— absent or reduced (1 point for each foot)

The neurological signs were graded as:-

0-2= normal, 3-5= mild, 6-8= moderate, >9= severe

Electrophysiological study and relevant other investigations were done to find out the cause.Nerve conduction study was carried out using standard techniques by Neuro pack II of 'Nihon Kohden' MEB

9200 machine (Japan). Skin temperature was maintained between 32^0C to 34^0C. The studies included motor and sensory nerve conduction in at least cross limbs or one arm and two legs. Electrophysiological study was set as a gold standard test for neuropathy assessment. For electrophysiological classification operational definition were set as below:

Operational Definition at Electrophysiology:-

Demyelinating Polyneuropathy

- Markedly reduced conduction velocity
- Prolonged terminal latency
- Conduction block and temporal dispersion
- Prolonged or absent F wave latency

Axonal Polyneuropathy

- Absent or reduced compound muscle action potential
- Normal terminal latency and conduction velocity

Electrophysiological study was done in neurophysiology laboratory of Dhaka Medical College Hospital. Electrophysiological study was conducted by the investigator and reviewed by designated consultant neurophysiologist in the department. General guidelines for performing nerve conduction study were followed. Electrophysiological study was done irrespective of duration of illness. Patients underwent testing for median, ulnar, common peroneal, tibial and sural nerve. A monopolar needle electromyogram (EMG) was performed in most patients.

All measurements were performed with surface electrodes and measurements were recorded in a computerized form. Nerves were stimulated using 1ms electrical pulse at a repetition rate of 1/sec with intensity sufficient to elicit maximum amplitude of compound muscle action potential (CMAP) and sensory nerve action potential (SNAP). Recording electrode was placed in muscle belly and non-recording electrode was placed in muscle tendon. For median and ulnar motor nerve study stimulation at wrist for median and ulnar nerve was 6-8 cm from the recording electrode and 11- 14 cm for sensory assessment. Recording electrode was placed in muscle belly and non-recording electrode was placed in muscle tendon. Abductor policis brevis, abductor digiti minimi were chosen for median and ulnar nerve respectively. Base to peak amplitude were measured. Tibial and peroneal nerve were stimulated at ankle with a distance of 6-10 cm from the recording electrode and abductor halucis and extensor digitorum brevis muscles were chosen respectively. Sural nerve was stimulated with a distance of 10-14 cm. In addition to distal latency, amplitude, and nerve conduction velocity (NCV) and F wave were measured.

Nerve conduction abnormalities were graded as mild, moderate and severe according to the study of ulnar and common peroneal nerve. Parameters considered were ulnar conduction velocity, CMAP amplitude, F wave latencies; peroneal CMAP amplitude, conduction velocity; spontaneous activity (fibrillation and positive sharp wave) in the muscles supplied by the ulnar or peroneal nerves.

Severity of polyneuropathy based on electophysiological alteration:

Class	Electrophysiological signs
Mild polyneuropathy	At least two parameters slightly altered. Fall in CV between 1-10% of normal value, fall in CMAP 1-20% of normal value, prolongation of F wave 1-20% of normal value and occasional fibrillation or positive sharp wave.
Moderate polyneuropathy	At least two parameters moderately altered. Fall in CV between 10-30% of normal value, fall in CMAP 20-50% of normal value, prolongation of F wave 20-50% of normal value and frequent fibrillation or positive sharp wave.
Severe polyneuropathy	At least two parameters severely altered. Fall in CV between >30% of normal value, fall in CMAP >50% of normal value, prolongation of F wave >50% of normal value and presence of fibrillation or positive sharp wave always.

CV= conduction velocity, CMAP= compound muscle action potential

Motor nerve conduction study

Name of nerve	Distal latency(ms)	Amplitude (mV)	Conduction velocity (m/s)	F wave latency (ms)
Median	4.2	4.4	50	31
Ulnar	3.4	6	50	32
Peroneal	5.5	5	40	NA
Tibial	6	3	40	56
Sensory nerve conduction study				
Median	2.5	20 µV	50	NA
Ulnar	2.5	15 µV	50	NA
Sural	3	6 µV	40	NA

NA= not applicable

Axonal Polyneuropathy:-

Mild: Ulnar - motor amplitude < 4.8 mV, sensory amplitude <12 µV, Peroneal motor amplitude < 4 mV. Occasional fibrillation or positive sharp wave in EMG

Moderate: Ulnar - motor amplitude 3- 4.8 mV, sensory amplitude 7.5-12 µV, Peroneal motor amplitude 2.5-4 mV. Frequent fibrillation or positive sharp wave in EMG

Severe: Ulnar - motor amplitude < 3 mV, sensory amplitude <7.5 µV, Peroneal motor amplitude < 2.5 mV. Presence of fibrillation or positive sharp wave all the time in EMG

Demyelinating Polyneuropathy:

Mild: Ulnar – conduction velocity 45-49 m/s, F wave latency 25.6 - 31 ms. Peroneal nerve conduction velocity 36-39 m/s.

Moderate:Ulnar– conduction velocity 35 - 44.9m/s, F wave latency 16 – 25.5 ms. Peroneal nerve conduction velocity 28 -35.9 m/s.

Severe: Ulnar– conduction velocity <35 m/s, F wave latency <16 ms. Peroneal nerve conduction velocity <28 m/s.

Mixed axonal and demyelinating:

Combination of axonal and demyelinating parameters:

Mild: presence of no moderate or severe feature

Moderate: presence of at least one moderate feature

Severe: presence of at least one severe feature

To see the association between weakness distribution and electrophysiological type proximal and distal weakness grading were compared with demyelinating category and distal to proximal weakness were compared with axonal type.

Data Collection & Data Analysis

Data was collected by semi-structured questionnaire by the investigator. Face to face interview, medical history and clinical examination and subsequent laboratory investigations were done. Proper permission was taken from the concern departments. All the patients (cases) were informed about the about the nature of the study and their informed written consent were taken in a consent form before collecting data. Data was analyzed with the help of computer SPSS program version 12.0 software facility. The chi-square test and Fisher's exact test were used to evaluate the proportion. A p-value of less then 0.05 was considered as statistically significant. Agreement tests (reliability) were done to see the fair or poor agreement.

Results:

A total of eighty patients of polyneuropathy (acute, subacute and chronic) were included in the study. Meticulous history and clinical examination was undertaken before neurophysiological investigation. The findings of study obtained from data analysis are presented below.

Table-I

Patients Characteristics (n=80)

	No. of patients	Percentage (%)
Men	52	65
Women	28	35
Age in years		
18 – 30	32	40
31 – 40	28	35
41 – 50	8	9
51 – 60	12	16
Mean age ±SD=34.4±6.8		
Range of age = 18- 60		
Occupation		
Service	12	15
Business	6	7.5
Student	10	12.5
Labourer	14	17.5
Cultivator	10	12.5
House wife	8	10
Unemployed	10	12.5
Retired	4	5
Others	6	7.5
Mode of onset/Duration		
Acute/upto 4 weeks	44	55
Subacute / 4 to 8 weeks	08	10
Chronic / > 8 weeks	28	35
Risk factor distribution		
Diabetes	12	15
Connective tissue disease	2	2.5
Hypothyroidism	4	5
Hereditary/ Family history	4	5
Preceding illness- diarrhea or RTI	20	25
Drugs/Toxins/Solvents exposure	6	7.5
Deficiency (vitamin)	4	5
Malignancy	2	2.5
Heavy metal(lead)	2	2.5
Not known (Idiopathic)	24	30

SD= standard deviation, RTI= respiratory tract infection

Table-1 shows the age distribution of the patient in to four groups. Ages of the patient ranged from 18 – 60 years. Most of the patient fell into 18 – 30 and 31-40 years age group and are 32 (40%) and 28 (35%) respectively. Lowest 8 (9%) was in 41 – 50 years age group. The mean age was 34.4 years with a standard deviation of 6.8. Patients were divided into male and female gender. Out of them 52 (65%) were male and 28 (35%) were female patients. M: F=1.8: 1. In occupation distribution service category comprised 15%(12), in business category 7.5%(6), student 12.5%(10), laborer 17.5%(14) which was the highest category, cultivator 12.5%(10), house wife 10%(8), unemployed comprises 12.5%(10), and retired 5%(4) which was lowest category and other not specified were 7.5%(6). Distribution of the patients according to the duration of illness i.e. mode of onset most of patient presented acutely which were 44(55%), chronic onset were 28(35%) and rests 8(10%) were in sub-acute category. Risk factors distribution of disorder causes polyneuropathy, highest number of patient were in not known or idiopathic group which comprises 30%(24), next in preceding illness of diarrhea or respiratory tract infection were 25%(20), history of diabetes were present in 15%(12), drugs/ toxins/solvents in 7.5%(6), hypothyroidism in 5%(4), hereditary or family history of neuropathy also in 5%(4).

Table-II

Clinical features of the study population (n=80)

Clinical feature	No. of Patient affected	Percentage (%)
Symptoms		
Paresthesia	70	87.5
Tingling	72	90
Numbness	24	30
Lack of feeling	20	25
Weakness	60	75
Wasting	20	25
Cramps	24	30
Signs		
Cranial nerve palsy	26	32.5
Loss of muscle power	60	75
Loss of Pinprick	20	25
Loss of Vibration sense	10	12.5
Deep tendon reflex hypo/areflexia	70	87.5
Autonomic dysfunction (any level)	10	12.5
Gait abnormality (any level)	70	87.5
Nerve thickening	2	2.5
Weakness Distribution		
Proximal and distal	40	50
Distal to proximal	20	25
No weakness	20	25
Clinical type of Polyneuropathy		
Motor	30	37.5
Sensory	8	10
Mixed	42	52.5

The multiple response table II shows that most of the patient had tingling, paresthesia and weakness which ranges from 75% to 90%, numbness in 30% patient, wasting in 25% patient, loss of muscle power were observed in 75%, deep tendon hypo or areflexia were in 87.5%, cranial nerve palsy in 32.5%, pinprick loss in 25%, loss of vibration in 12.5%, autonomic dysfunction at any level in only 12.5% and nerve thickening in only two patients. Distribution of weakness in study subject, 50% patient there were both simultaneous proximal and distal weakness, distal to proximal weakness were in 25% patient, and 25% patient were with no weakness at all. Clinical types of polyneuropathy among study population. Out of 80 patients, 42(52.5%) had mixed sensory motor neuropathy, followed by 30 (37.5%) had motor neuropathy and only 10% had sensory neuropathy.

Table-III

Results of NCS in study population (n=80)

Nerve	Variable	Range	Mean	SD
Median nerve(MN)	MN MDL	NR – 14.2	5.84	1.8
	MN CMAP	NR – 18	5.24	4.2
	MN MNCV	NR - 82	48.4	8.7
	F wave latency	NR- 45.3	32.42	7.62
	MN SDL	NR – 9.2	3.2	2.4
	MN SNAP	NR -30	6.4	9.2
	MN SNCV	NR – 60	52.2	10.4
	Total	MN = 80		
Ulnar nerve (UN)	UN MDL	NR -10.8	4.52	2.8
	UN CMAP	NR – 8.6	2.88	2.42
	UN MNCV	NR -70	46.2	14.2
	F wave latency	NR -42.2	30	8.9
	UN SDL	NR -9.2	4.4	2.6
	UN SNAP	NR -7.44	3.2	2.4
	UN SNCV	NR -68	53.6	14.2
	Total	UN = 80		
Tibial Nerve (TN)	TN MDL	NR -12.4	7.8	3.2
	TN CMAP	NR – 5.2	1.6	1.4
	TN MNCV	NR – 54	38.2	10.2
	F wave latency	NR - 65	54.4	12.4
	Total	120		
CommonperonealNerve (CPN)	CPN MDL	NR -12.6	7.9	3.3
	CPN CMAP	NR -4.6	1.8	1.6
	CPN MNCV	NR – 52	38.4	10.1
	Total	130		
SuralNerve (SN)	SN SDL	NR- 10.6	6.6	3.6
	SN SNAP	NR - 16	5.4	2.8
	SN SNCV	NR – 50	40.2	12.6
	Total	140		

MDL=motor distal latency, NR=not recordable, CMAP=compound muscle action potential, MNCV= motor nerve conduction velocity, SDL= sensory distal latency, SNAP= sensory nerve action potential, SNCV= sensory nerve conduction velocity

The above table III shows the results of the different neurophysiological parameters of the patients. Total 80 median nerves and 80 ulnar nerves were examined. The mean of the MN MDL, MN SDL, UN MDL and UN SDL were found to be 5.84, 3.2, 4.52 and 4.4 milliseconds (ms) respectively. The mean of median nerve CMAP = 5.24 mV, MN MNCV = 48.4 m/s, MN SNAP = 6.4 micro volt, UN CMAP = 2.88 mV, UN MNCV = 46.2 m/s UN SNAP = 3.2 micro volt. The range and standard deviation are also shown in table. Mean F wave latency of median and ulnar nerves were 32.42ms and 30 ms respectively. In the lower limb 120 tibial nerve, 130 peroneal nerve and 140 sural nerves were examined. The mean of the tibial, peroneal and sural distal latency were 7.8 ms, 7.9 ms and 6.6 ms respectively. TN CMAP= 1.6 mV, CPN CMAP=1.8 mV, SN SNAP=5.4 microvolt, TN MNCV= 38.2 m/s, CPN MNCV = 38.4 m/s and SN SNCV = 40.2 m/s were observed. The range and standard deviation are also shown in table and the mean F wave latency in tibial nerve was 54.4 ms.

Table-IV

Electrophysiological classification of neuropathy in study population (n=80)

Type	No. of patients	Percentage (%)
Axonal	38	47.5
Demyelinating	22	27.5
Mixed axonal & demyelinating	20	25
Total	80	100
Motor	38	47.5
Sensory	6	7.5
Mixed sensorimotor	36	45
Total	80	100

The above table IV shows the electrophysiological category of polyneuropathy in study population. Axonal varieties were highest and comprised 47.5 %(38), mixed variety were lowest 25 %(20) and demyelinating category were 27.5 %(22). Types determined by electrophysiological examination into motor, sensory and mixed sensorimotor polyneuropathy were 47.5 %(38), 7.5% (6) and 45% (36) respectively.

Table-V

Statistical analysis of Clinical and electrophysiological relationship of polyneuropathy

	Chi-square value	p value	Agreement test, k value
Motor polyeuropathy	9.59	0.0019	0.5 (fair agreement)
Sensory polyneuropathy		0.1597 (Fisher's exact)	0.19 (poor agreement)
Mixed sensorimotor polyneuropathy	5.681	0.0032	0.44 (fair agreement)
Severity of polyneuropathy	1.53	0.2166	0.03 (poor agreement)
Weakness distribution of demyelinating type	1.726	0.1892	0.06(poor agreement)
Weakness distribution of axonal type	2.678	0.1018	0.14 (poor agreement)

The above table V shows the statistical analysis of clinical and electrophysiological relationship. For motor type of polyneuropathy, statistical analysis shows there is significant relation as the p value is < 0.05 and k value = 0.5 (fair agreement). For sensory type of polyneuropathy, statistical analysis shows there is no relation as the p value is > 0.05 and k value = 0.19 (poor agreement). For mixed sensorimotor type of polyneuropathy, statistical analysis shows there is significant relation as the p value is < 0.05 and k value = 0.44 (fair agreement). Comparison between clinical electrophysiological severity grades, statistical analysis shows that clinical and electrophysiological grades are not comparable as the p value is >0.05 and k value = 0.03 (poor agreement). Association of weakness distribution of proximal and distal with demyelinating category, statistical analysis shows that the association is not significant as the p value is >0.05 and k value = 0.06 (poor agreement). Association of weakness distribution of distal to proximal with axonal category, statistical analysis shows that the association is not significant as the p value is >0.05 and k value = 0.14 (poor agreement).

Discussion:

Polyneuropathy is relatively common and often a distressing chronic condition. It has many diverse underlying causes and in different diseases the incidence of PN varies considerably[16]. This cross sectional study was designed to correlate the clinical and electrophysiological features of polyneuropathy patients. This study also addressed the clinical and electrophysiological pattern of polyneuropathy patient.

In this study patients of all age group ranging from 18-60 years were included. Majority of the patients 32 (40%) were in 18 to 30 years of age with mean ±SD = 34.4 ±6.8. In Rosenberg et al[6] age ranges from 26-93 years with a mean age of 62 years, in a local study [16] age ranges from 10 to >60 years and most of the patient were in 40-50 years. The mean age in this study differ from the other study might be due to that in this study age ranging from 18-60 years were included.

In this study 65 % were male and 35 % were female with M: F = 1.8: 1. In one local study[16] the M: F was 1.88: 1 and in another local study [17] the M: F ratio was 1.9:1 which resembles with the present study and it is observed that polyneuropathy is about two times more common in male. McLeod et al[18]. also found an overall predilection for men (3:1). In this study polyneuropathy were widely distributed in different occupations, labourers, cultivator and students were affected more. In this regard there are a few studies elsewhere. In the study ofChistee, [19] more or less similar findings were observed but in his series cultivators were less affected but housewives were more affected as well as labourer and students.

It was observed in this study that 55% patienst presented acutely and 35% had chronic onset and 10% patients had subacute onset. Study on polyneuropathy patients comprising acute, subacute and chronic cases are few. In the study of Rizvi et al.[18] 35 patients were GBS cases among 110 polyneuropathy patients but they did not categorize them into acute, subacute or chronic and in another local study Chistee[19] of 50 polyneuropathy cases GBS cases were 50% and the findings were similar with the present study.

In this study majority (30%) patients had no known history of risk factors i.e. idiopathic, antecedent infections (preceding illness either diarrhoea or RTI) was the next common risk factors (25%), next was diabetes mellitus (15%), followed by combined drugs & toxins (7.5%). In a study of chronic polyneuropathy by Vrancken et al[3]. idiopathic were 43%, diabetes mellitus 32%, alcohol abuse 14%, paraproeinaemia 9%, deficiency of vitamin 6% and autoimmune or systemic disease 4% were observed. In a Dutch study on chronic polyneuropathy, Rosenberg NR et al[6]. observed 60(57.1%) patients of diabetes mellitus, followed by HIV infection in 21(20%) patients, alcoholism in 11(10.5%) patients; drug induced in 7(6.7%) patients and renal failure in 6(5.8%) patients in a study of 105 chronic polyneuropathy cases. In Lubec et al[20]. frequency of causal factors in 124 cases were : - diabetes mellitus in 26(21%) cases, alcohol in 20(16.1%) cases, vitamin deficiency in 13(10.5%) cases, GBS in 9(7.3%) cases, paraproteinamias in 6(4.8%) cases, hypothyroidism in 5(4.03%) cases, borreliosis in 6(4.8%) cases, paraneoplasia in 4(3.2%) cases, CIDP in 5(4.03%) cases, hereditary in 3(2.4%) cases, hyperthyroidism in 3(2.4%) cases, critical illness in 2(1.6%) cases, vasculitis in 3(2.4%) cases, and each one(0.8%) case of sarcoidosis, vincristine, azathioprine, Refsum's disease, Sneddon's syndrome, Ehlers-Danlos syndrome, crohn's disease inflammatory polyarthritis and solvent. In an Asian study of 124 cases of chronic polyneuropathy Habib and Ferdousi[16] observed diabetes were 45.2%,

idiopathic 45.2%, hereditary 5.7% and CIDP in 3% cases. So the distribution of polyneuropathy in different diseases varies worldwide. In this study less diabetic and infectious cases were observed as because major bulk of diabetes mellitus are cared by internationally reputed separate diabetic hospital and infectious disease hospitals[17].

The features of polyneuropathy may be exclusively motor, sensory, autonomic or combined. Most PN present with mixed sensory motor symptoms. Sensory symptoms were usually the presenting features. These were tingling, pins and needles, burning sensation, pain and numbness in the extremities. Motor symptoms were usually those of weakness and wasting[21]. This is reflected in the present study where paresthesias were present in 87.5 %, tingling in 90%, numbness in 30% cases. Weakness in this study was in 75% cases, deep tendon reflex hypo/areflexia in 87.5% and abnormality of gate at any level were also 87.5%. Similarity was observed in the study of Habib and Ferdousi[16] also. A relative lack of muscle wasting in relation to the degree of weakness, weakness of proximal muscle as well as distal muscle, disproportionate loss of joint position and vibration sensation compared to relative preservation of pain and temperature are suggestive of demyelinating neuropathy[22]. In this present study proximal and distal weakness was in 50% cases and distal to proximal weakness was observed in 45% cases.

One of the most important aims of the study was to detect the clinical and neurophysiological type of polyneuropathy. In Rosenberg et al.[6] 77.5% were mixed sensorimotor type, 13.75% were pure sensory type and 8.75% were pure motor type. In Konig et al[23]. 42% were mixed sensory motor, 30% sensory, no case of motor type. In the study of Konig et al[23]. cases of mononeuropathy and mononeuritis multiplex were included. In Macleod et al[18]. 64% were mixed sensory motor type and 27% pure sensory type and 9% were pure motor type. In our present study mixed sensory motor types were 52.5%, motor types were 37.5% and pure sensory types were only 10%. Though in this present series mixed sensory motor type was the most common, the high motor type reflects the inclusion of significant acute polyneuropathy cases.

In this study of 80 polyneuropathy cases either cross limbs or both the lower limbs and an upper limb nerves were examined electrophysiologically. 80 median nerves, 80 ulnar nerves, 120 tibial nerves, 130 common peroneal nerves and 140 sural nerves were studied. Regarding the neurophysiological parameter MDL, CMAP, CV, F wave latency, SDL, SNAP and sensory CV were measured and compared with normal value. Similar parameters were measured for electrophysiological study in Rubens et al[24]. also in Franssen and Van Den Bergh[25]. There are no internationally accepted criteria for axonal polyneuropathy. However, in most publication, axonal polyneuropathy is associated if there are decreased distal CMAPs & SNAPs and no evidence of demyelination[26,27]. However, in some axonal neuropathy such as diabetic neuropathy or CMT type 2 SNAPs are with in normal limits, so that other evidences are required for diagnosis of polyneuropathy[28, 29]. Criteria for demyelination required nerve conduction to be slower than a given percentage beyond the limit of normal; criteria were defined for MCV, distal motor latency and F wave latency[13,30,31]. Globally a MCV below 38m/s in an arm nerve and below 30m/s in a leg nerve are consistent with demyelination, provided that the distal CMAP exceeds 1 mV. A distal CMAP duration of about 9-10ms or more indicates demyelination[32, 33]. A CMAP area reduction proximal versus distal of 50% or more indicates conduction block (CB). Very detailed criteria for CB were defined by American Association of Electrodiagnostic Medicine (AAEM) Onley[34] et al. In this study electrophysiological types of polyneuropathy were axonal type 47.5%, demyelinating type 27.5% and mixed type 25% which were near similar with Vrancken[3] et al. Where axonal type was 57%, demyelinating type 13% and not specified were 31%. In another European study[6] (Rosenberg NR et al.) of 56 chronic polyneuropathy cases, axonal types were 87.5% and demyelinating type were 12.5% and the findings resembles the present study. In a Bangladehi study by Habib and Ferdousi[16] 26.6% were axonal, 16.1% demyelinating and 31.5% were mixed axonal and demyelinating. The above mentioned local study does not match with our study due to the fact that 25.8% patient were not labeled in any particular pathological type.

Finally, to see the relation between the clinical and eltectrophsiological features of polyneuropathy and to compare the severity measured in both clinically and eletrophysiologically, parameters of both the clinical features and electrophysiological parameters were graded as mild, moderate and severe and

statistically analyzed by chi-square/ Fisher's exact test and agreement test. Highly significant association was seen in motor and mixed sonsorimotor type of clinical and electrophysiological classification. In sensory polyneuropathy distribution in clinical and electrophysiological types varies. Comparison of the severity of polyneuropathy in clinical and electrophysiological grade there were poor agreement among them. The weakness distribution such as distal to proximal with axonal and distal and proximal with demyelinating polyneuropathy, statistically significant association were not found.. To determine the relation between neurophysiological data and clinical examination Lefaucheur[35] et al. observed that clinical and neurophysiological classifications and severity scores were correlated whatever the type of neuropathy. These differences with the present study might be due to that Lefaucheur et al.[35] studied the sensory neuropathy according to fiber type involvement. Latov et al.[36] observed that the number and type of demyelinating abnormalities in patients with polyneuropathy vary with the clinical phenotype. Rajabally et al.[37] in their studied patients with CIDP demonstrated the predominance of demyelination in upper limbs nerves, of axonal loss in lower limbs nerves and presence of about 50% of demyelinating – range abnormalities in clinically unaffected territories. Vittadini et al. [38] found significant correlation between alcoholic polyneuropathy, the duration of alcoholism and the type of alcoholic beverage consumed. So, from the present study, there are some variations and there are some relations among the clinical and electrophysiological spectrums of polyneuropathy. Motor and mixed sensorimotor type of Clinical and electrophysiological classification of polyneuropathy are related but the sensory types of polyneuropathy are not related. Severities of polyneuropathy in clinical and electrophysiological parameters are not also related. And weakness distributions are not also associated with axonal and demyelinating type.

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Epileptic Patients Attending in a Tertiary Care Hospital: A 4 (four) Years Study

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Abstract:

This was a prospective study done in out patient department in Bangabandhu Sheikh Mujib Medical University from January 2008 to June 2010. Patient who diagnosed an epilepsy by history and EEG were included in this study. Patients were followed up every 3-6 months in this study period. Patients were assessed by history and EEG findings, CT or MRI of brain. Structured questionnaire were used to follow up the improvement.

Introduction:

Epilepsy is a disorder characterized by recurrent seizures of cerebral origin, presenting with episodes of sensory, motor or autonomic phenomenon with or without loss of consciousness. Epilepsy is second most common chronic neurological condition seen by neurologist. The global prevalence of epilepsy is generally 5 to 10 cases per 1000 persons[2,3]. Epilepsy is a condition of chronic recurrent seizures and its most disabiliting aspect is unpredictability of when and where the next seizures will occur. Its etiology is complex and heterogeneous. Its prevalence varies in relation to ethnicity, geography, age and sex[3-5]. Seizure frequency, type, duration, family history, triggers for epileptic seizure and other neurological, behavioral and scholastic characteristics are

important characteristics of epilepsy in a population. There peculiarities are known to affect other neurological, behavioural and scholastic characteristics. The understanding of clinical profile to epilepsy patients from different human populations is important to broaden the available knowledge and to provide baseline data for cross-cultural comparisons. This would also be important for adopting strategies in effective and better health planning. It is important to record that no previous study was done for clinical profile of epilepsy in Bangladesh. With these major objectives in view, the present study was undertaken.

Methology:

Data was collected prospectively from a sample of 120 epileptic patients (60 idiopathic and 60 symptomatic) attending the neurology epileptic clinic of Bangabandhu Sheikh Mujib Medical University, Dhaka, Bangladesh from January 2008 to June 2010. The patients were referred to the clinic by neurologists, general practitioners and from hospitals of Bangladesh. Both old and new patients attending the clinic were included. All the patients were clinically examined and underwent one Electroencephalogram (EEG) and Computed Tomography (CT) scan or MRI of brain were done who can afford the test after clinical evaluations and were diagnosed as Idiopathic Epilepsy (IE) or Symptomatic Epilepsy (SE) on the basis of these reports. Mental retardation was diagnosed clinically. Behavioural disorders were diagnosed by history taking and clinical interview. Informed consent was obtained as per institute ethical committee guidelines.

Statistical analysis: The data so generated was subjected to statistical tests like mean, t test and chi-square test to draw inferences.

Results:

The results are summarized in Table-1 to Table-10 and Fig. 1. Most of age group were 10-30 years, mean (SD) age of the respondents was 24.89 (±11.63) years (Table-1). Prevalence of generalized tonic clonic was 38.3%, generalized myoclonic 5%, generalized atonic 1.7%, generalized absence 5% and partial with secondary generalized (simple) 20% and partial with secondary generalized (complex) 30% (Table-II). In this study 58.3% patients were male and 41.7% were female (Table-3). Only 7% have positive family history ((Table-IV). Place of residence revealed that most of

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the patients epilepsy were from Dhaka (Table-V). The majority of epileptics were unable to perceive any triggering factor, but when probed further by gaining their confidence, many of them attributed seizures to supernatural forces. Exertion was reported as a major triggering factor 55%, followed by sleep deprivation 21.7%. Some female patients linked epilepsy with their menstrual cycles (Table-VI). In this study 83.3% patients have no neurological abnormalities and 5% patients were mentally retarded (Table-VII). Neurological investigations revealed abnormal electroencephalography (EEG) in 58.33% patients (Table VIII). Computed Tomography (CT) Scan was abnormal in 15% patients and MRI was abnormal in 18.3% patients (Table-VIII).

Table-I

Age distribution of the patients (n=120)

Age (year)	Frequency	Percent
Up to 10	8	6.7
11-20	40	33.3
21-30	48	40.0
31-40	12	10.0
>40	12	10.0
Total	120	100.0

Mean (SD); Range: 24.89(11.63); 5.5-65

Table-II

Distribution of the patients by types of epilepsy (n=120)

	Frequency	Percent
Generalized (Absence)	6	5.0
Generalized (atonic)	2	1.7
Generalized (myoclonic atonic)	4	3.3
Generalized (myoclonic)	2	1.7
Generalized (Tonic clonic)	46	38.3
Simple partial seizure	24	20.0
Partial secondary Generalized	36	30.0
Total	120	100.0

Table-III

Gender distribution of the patients (n=120)

	Frequency	Percent
Male	70	58.3
Female	50	41.7
Total	120	100.0

Table-IV

Distribution of the patients by family history of same disease (n=120)

	Frequency	Percent
Positive	14	11.7
Negative	106	88.3
Total	120	100.0

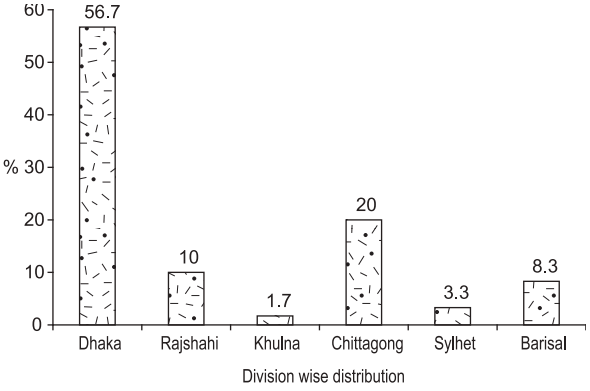

Fig.-1:

Table-V

Division wise distribution of residence of the patients (n=120)

	Frequency	Percent
Dhaka	68	56.7
Rajshahi	12	10.0
Khulna	2	1.7
Chittagong	24	20.0
Sylhet	4	3.3
Barisal	10	8.3
Total	120	100.0

Table-VI

Precipitating factor of epilepsy (n=120)

	Frequency	Percent
Exertion	66	55.0
Sleep deprivation	26	21.7
Watching TV	14	11.7
Menstrual Cycle	10	8.3
Hyperventilation	2	1.7

Table-VII

Neurological/Morphological/Behavioral abnormalities (n=120)

	Frequency	Percent
No	100	83.3
Mental Retardation	6	5.0
Hemiplagia	8	6.7
Weakness	4	3.3
Aphasia	2	1.7
Total	120	100.0

Table-VIII

Laboratory findings of the patients (n=120)

	EEG	CT scan	MRI
Normal	50 (41.67)	42 (35.0)	32 (26.7)
Abnormal	70 (58.33)	18 (15.0)	22 (18.3)
Not advised	-	10 (8.3)	8 (6.7)
Not done	-	50 (41.7)	58 (48.3)

Discussion:

Epidemiological studies of epilepsy are affected by methodology. Major limitations are differences between the definite classification of epilepsy, inclusion criteria, case ascertainment methods, selection bias, underreporting, small population studies, descriptive rather than analytic nature of the studies, high cost of studies, limited resources, geographical and cultural differences, political and social atmosphere and variant public health priorities[1,2,6-9,10-16]. The profile of epilepsy varies across various cultures and the review shows that in western countries about two–third of the epileptic patients have partial seizures[17,18]. Similar trend has been shown in some developing countries like Nigeria[19]. In this study among symptomatic epilepsy 20% patients were simple patient and 30% patients were diagnosed as partial with secondary generalized, the results being comparable with the previous study reported from Northwest India[20].

Seizure frequency is another important characteristic of epilepsy in a population. In developed countries, it has been estimated that among those with active generalized tonic-clonic seizures undergoing treatment, about 15% have seizures less than once a year, 60% have seizures frequency ranging between on per month and one per year and 25% have seizures occurring at a frequency of more than one per month[21]. In the present study median attack was 4 in last one year. In some previous study the age of onset of epilepsy has been reported in the first two decades of life in 75-80% patients[22,23]. In the present study, the majority of cases, the age of onset of epilepsy was less than 20 years.

Most studies of epilepsy in industrialized countries report that males are more frequently affected than females[20,24,25]. Our study shows male preponderance in epilepsy cases (58.3%). The association of epilepsy with changes in cognition, personality and behavior has been discussed for centuries[20,26-28].

In the present study, behavioral disorders or acute changes have been noticed in 4% cases and mental retardation in 1.7% cases, which is compatible with previous studies[20]. In the present study 82% cases had abnormal EEG. Which was slightly higher then previous study in India[20]. In the present study 18% cases with abnormal CT Scan and 22% patients with abnormal MR were noted only in symptomatic epilepsy, which were slightly higher then some previous study[8].

Conclusion:

The study demonstrates differences in the type of seizures between idiopathic and symptomatic epilepsies. Additional studies of epileptic populations across various cultures and geographical areas allow broad generalizations and the differences in the magnitude are due to the factors unique to each populations.

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Prognosis of Severe Head Injury after Surgery- An Observational Study

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Abstract:

Background: *Severe head injury patients with low initial score of Glasgow coma scale (GCS), abnormal motor responses, loss of brainstem reflexes, high intracranial pressure, mass lesions and their effects and midline shifts shown by Computerized tomography (CT) scan are the factors which have been associated with poor progress. With proper indications, early surgery may reduce the mortality and morbidity in these patients.*

Aims and Objective: *The aim of this study was to determine the prognosis of severe head injury after surgery.*

Materials and methods: *This was an observational study carried out in the Department of Neurosurgery, Square Hospitals Limited, Dhaka, Bangladesh.*

The study of 36 patients was conducted during March 2007 to May 2010. The patients were categorized according to age, sex, type of pathology requiring surgery and post operative outcome at three months using GOS. Only the patients who underwent surgery for severe head injuries were included in this study.

Results: *Majority of the patients were male (86%). Most of the victims were young adults (50.1%) within the age groups of 21-40 years. Most of the patients were operated for acute subdural hematoma (41.7%), followed by traumatic intracerebral hematoma (36%) and hemorrhagic brain contusion (19.5%). In this study the least number of the patients (5.6%) sustained extradural hematoma. Compared with other studies the death rate of this study was significantly low (16.7%) and a significant number of patients had good recovery (38.9%).*

Conclusion: *Although the number of cases was not very high, the study concluded that early diagnoses and surgical interventions were related to more favorable outcome.*

Key words: Severe head injury, prognosis, surgery for severe head injury.

Abbreviation: *GOS (glasgow coma scale), CPP (cerebral perfusion pressor), ICP (intracranial pressure), GOS (glasgow outcome scale), ICU (intensive care unit).*

Introduction:

Head injury refers to trauma of the head. This may or may not include injury to the brain[1]. However, the terms traumatic brain injury and head injury are often used interchangeably in medical literature[2].

TBI (Traumatic brain injury) severity is most commonly graded by the initial Glasgow Coma Scale score. The GCS rates the patient's best verbal response, best motor response and the stimulus needed to elicit eye opening. Scores range from 3-15, with score d" 8 representing coma. 'Mild' head injury (accounting for ~80% of cases) is manifest by a 30 minute post-injury and GCS of 13-15. 'Moderate' head injury consists of immediately altered or loss of consciousness for > 30 minutes and 6 hour post-injury GCS of 9-12. 'Severe head injury' involves immediate loss of consciousness for > 6 hours with residual GCS of 3-8[3].

Head injury is a major public health problem and the leading cause of death and disability worldwide[4]. Despite the modern diagnosis and treatment, the

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prognosis for patients with severe head injury remains poor. While severity of primary injury is the major factor determining the outcome, the secondary injury caused by physiological insults such as hypotension, hypoxemia, hypercarbia, hypocarbia, hyperglycemia and hypoglycemia, etc. that develop over time after the onset of the initial injury, causes further damage to brain tissue, worsening the outcome in head injury.

Pathophysiology of head injury involves primary and secondary injuries to the brain. Primary injury is the damage caused by the initial trauma involving mechanical impact to the brain tissue and skull due to acceleration-deceleration or rotational forces, resulting in skull fracture, brain contusion, expanding intracranial hematoma or diffuse axonal injury[5]. The primary injury then initiates inflammatory process, edema formation and excitotoxicity, resulting in further increase in intracranial pressure (ICP) and reduced cerebral perfusion pressure (CPP). Severity of primary injury is the major factor determining the outcome of head injury patients. Secondary injury is a consequence of physiological insults that develop over time after the onset of the initial injury, causing further damage to the brain tissue and worsening the outcome in head injury patients. Two major factors that cause secondary injury are hypotension and hypoxemia.

Brain surgery following a head injury is not that common. The purpose of brain surgery after severe head injury is for controlling pressure inside the skull or for alleviating brain swelling or for repairing blood vessels and removing blood clots or foreign matter.

Perioperative period may be particularly important in the course of head injury management. While surgery and anesthesia may predispose the patients to new onset secondary injuries which may contribute adversely to outcomes, the perioperative period is also an opportunity to detect and correct the undiagnosed pre-existing secondary insults, to prevent against new secondary insults and is a potential window to initiate interventions that may improve outcome of head injury.

The Glasgow Outcome Scale (GOS) is a 5-point score given to victims of traumatic brain injury at some point in their recovery[6]. It is a very general assessment of the general functioning of the person who suffered a head injury. The scores of GOS are- 1- dead, 2- vegetative State (meaning the patient is unresponsive, but alive; a “vegetable” in lay language), 3- severely Disabled (conscious but the patient requires others for daily support due to disability), 4- moderately Disabled (the patient is independent but disabled) and 5- good Recovery (the patient has resumed most normal activities but may have minor residual problems).

At Square Hospitals Ltd. Dhaka, we performed a study on the prognosis of severe head injury after surgery.

Materials and methods:

This observational study was carried out in the Neurosurgery Department of Square Hospitals Ltd. during March 2007 to May 2010. A total of 36 patients who under went surgery for severe head injury were included in this study. The patients were further categorized according to age, sex, type of pathology requiring surgery and post operative outcome at three months using GOS. Study period was 3 years 3 months. The patients with head injury who do not fulfill the definition of severe head injury were excluded from this study. Moreover, patients who refused to take part in the study were also excluded from this study.

Aim of the study was to observe the prognosis of patients who underwent surgery for severe head injury. Diagnosis was confirmed by detailed history, clinical examination and imaging studies, which included CT scan of brain. After confirmation of diagnosis as severe head injury, patients’ data were collected by questionnaire. Severe head injury was determined by Glasgow Coma Scale (GCS d” 8) and the presence of lesion requiring surgery was determined by CT scan of brain and its clinical correlation. With appropriate indications surgeries were performed within 6 hours of the admission. Indications of surgery were intracranial extra axial hematoma (extradural and subdural hematoma) of more than 30cc volume or center point thickness of an extra axial hematoma e” 1cm; if the hematoma causing a midline shift of e” 5 mm; if the traumatic intracranial mass lesion causing impending brain harniation and rapid deterioration of the patients clinical condition and for intracerebral hematoma of e” 50 cc or cerebellar hematoma of e” 5 cc in volume with clinical correlation. In this study we followed the indications of surgery according to the scientific work of Olumide AA et al[7]. Surgeries primarily performed were craniotomy to evacuate an intracranial mass lesion (extradural hematoma, acute subdural hematoma, intracerebral hematoma or hemorrhagic brain contusion) to normalize raised intracranial pressure and to prevent farther damage of brain parenchyma and fatal outcome. After the surgery all the patients were kept in mechanical ventilation with

full muscle paralysis and sedation for at least 72 hours followed by gradual weaning from mechanical ventilation whenever possible. After stabilization of vital signs and neurological status patients were shifted out of the ICU. Patients' condition were noted at the time of discharge. Follow ups were performed at the end of three months after discharge using the GOS.

All the surgeries were performed by a single team of surgeons, anesthetists and scrub nurses. Perioperative cares were also provided by the same ICU team. A master sheet was formed and all the questionnaires were arranged at the end of data collection and were tabulated according to different parameters.

Result:

This observational study was carried out on 36 patients of severe head injury. Data were tabulated according to different parameters.

Table-I

Distribution according to sex (N=36)

Sex	No. of case	percentage	Male female Ratio
Male	31	86	6.2:1
Female	05	14	

Table-I shows the distribution according to sex. Most of the patients were male. They were 86% of total number of cases. Male is to female ratio was 6.2:1.

Table-II

Distribution according to age group (N=36)

Age frequency (years)	No. of case	percentage	Mean age
< 20	04	11	38.9
21- 30	11	30.6	
31-40	07	19.5	
41-50	03	8.3	
51-60	04	11	
>60	07	19.5	

Table-II demonstrates the distribution patients according to age group. Young adults were the most frequently affected group; about half (18; 50%) of the studied subjects fall under this age group i.e. between 21 to 40 years. Below 20 and from 51 to 60 age group had same number of patients which were approximately one tenth (11%) of total patient number. Total number of patients between 41-50 age group was only three (8.3%). Near about one fifth patients were found in >60 age group.

Table-III

Distribution according to types of injury (N=36)

Types of injury	No. of case	Percentage
Extradural hematoma	02	5.6
Acute subdural hematoma	14	41.7
Intracerebral hematoma	13	36
Hemorrhagic contusion	06	19.5
Acute subdural hematoma with brain contusion	1	

Table-III shows type of head injury. More than 40% of patients had acute subdural hematoma while traumatic intracerebral hematomas were found in 36% cases. Radiological examinations (C.T. scan of brain0 shows brain confusion (Fig.-1) and subdural hematoma (Fig.-2).

Table-IV

Outcome at 3 months follow up according to GOS (N=36)

GOS	No. of case	percentage
1. Dead	06	16.7
2. Vegetative state	03	8.3
3. Severely disable	09	25
4. Moderately disable	04	11
5. Good recovery	14	38.9

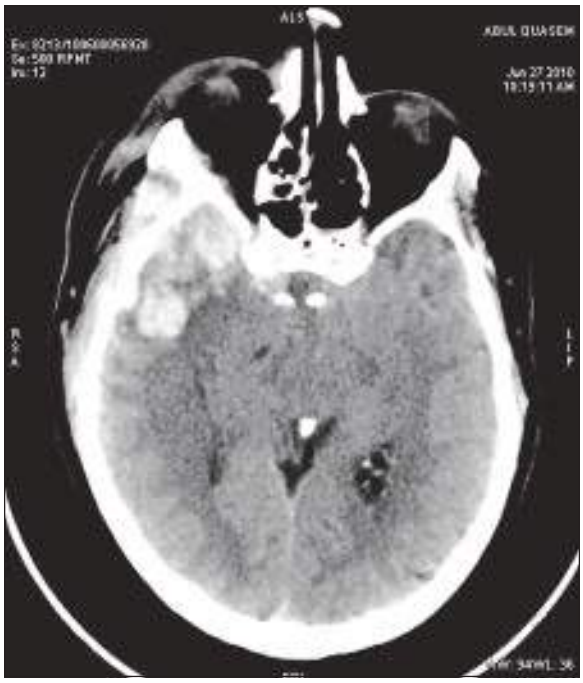

Fig.-1: *CT scan of right temporal lobe contusion*

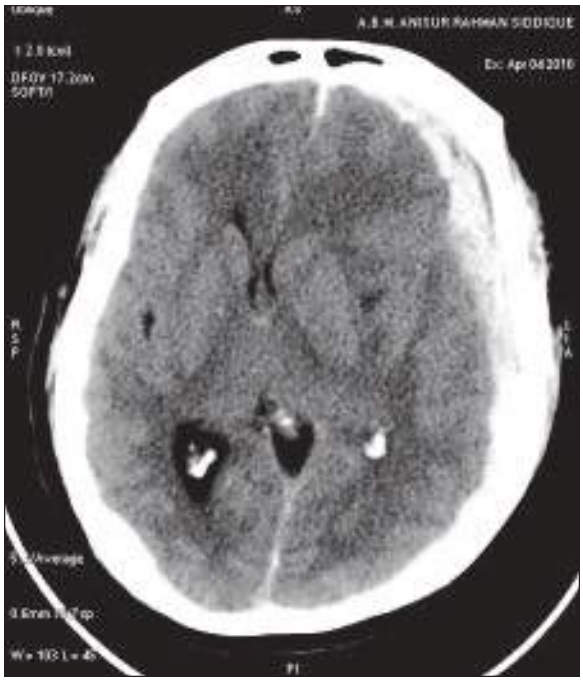

Fig.-2: *CT scan of left sided acute subdural hematoma.*

Table-IV illustrates the distribution of all the post operative severe head injury cases according to Glasgow outcome score at 3 months after discharge from hospital. In this study 38.9% of cases (total number 14) had good recovery, one quarter of the patients were severely disabled, four patients (11%) had moderate disability. Another three patients passed to vegetative state. Death rate in this study was 16.7% (total 6 patients).

Discussions:

A study of 36 patients with post operative severe head injury (GCSd" 8) was conducted at a tertiary care hospital to identify the prognosis of such injuries after surgery at 3 months follow up. According to GOS 38.9% of patients had a good recovery, 25% of the patients classified as severely disabled, 11% of the patients had moderate disability and 8.3% of the patients passed to persistent vegetative state. Moreover the death rate of the study population was 16.7%.

The presence of intact brainstem reflexes 24 hours post injury in comatose patients with head injury is a prognostic sign for a good recovery. Favorable clinical signs include pupillary reactivity intact oculovestibular reflexes and motor responses such as localization to pain. Poor outcome was associated with nonreactive pupils, absent oculovestibular reflexes and motor response of extension or no response at all. Most frequent outcome category for survivors at discharge was severe disability, but at the end of 3rd month 38.9% of the total patient population improved to good recovery category.

According to the study of Levati A et al[8]. the mortality rate was 39.5%. Of the survivors, 59.2% made a good recovery, 18.4% remained moderately disabled, 6.1% were severely disabled, and 1.5% was in a persistent vegetative state. The most reliable predictive criteria were: absence of brain-stem reflexes, neurological status, abnormal motor patterns, arterial hypotension, and presence of mass lesions. In this study mortality rate was 16.7%. Good recovery was 38.9% and 25% of patients had severe disability at follow up after 3 months.

The study of Donald et al [9] was on the outcome from severe head injury followed a standardized protocol in 160 patients. Of these patients, 36% made a good recovery, 24% were moderately disabled, 8% were severely disabled, 2% were vegetative and 30% died.

It was observed that vigorous surgical and medical therapy, by preventing or reversing secondary cerebral insults, enables some patients who would have died to make a good recovery without increasing the proportion of severely disabled patients. In the present study good recovery was almost the same (38.9%) but the mortality was almost the half (16.7%) of the study described by Donald et al.[9]

Sousa J et al[10] presented the outcome of consecutive 166 patients with severe head injury retrospectively. Their aim was to analyze the long term outcome in these patients and identify the other significant prognostic factors. Of the 166 patients, 42 patients (25.30%) had a functional outcome good recovery in 10.24%, moderate disability in 15.06%, and 124 (74.69%) had a poor outcome (death in 58.43% and severe disability in 16.26% of cases). There were 45 patients with polytrauma and 24 of these patients (53.33%) succumbed to the injuries. At follow up, most of the patients with a functional outcome showed a significant improvement in their motor function but continued to have neuro-behavioral and cognitive deficits. The present study has fairly appreciable outcome than this study with mortality of 16.7%, good recovery 38.9%, severe disability of 25%, moderate disability of 11% and patient passed to vegetative state are 8.3%.

Mwang'ombe NJ et al[11] published their study on factors influencing the outcome of severe head injury. Six hundred and seventy seven patients with severe head injuries were treated in 5 years. Three hundred and eighty one patients died while undergoing treatment, 56.2% overall mortality. Age specific mortality was 35.7% in patients below 13 years, 44% in age group 14-25 years, 56% in age group 26-45 years. Patients with admission GCS of 3-4 had a mortality of 88%, those with GCS 5-6 had a mortality of 60% and those with admission GCS 7-8 had a mortality of 52%. Ninety per cent of the patients who had bilaterally dilated pupils not reacting to light on admission died and 66% of the patients with bilaterally constricted pupils at the time of admission died. Only 20% of patients with severe head injury who had normal pupillary reaction to light at the time of admission died. Mwang'ombe NJ et al's study also had significant high mortality than the present study (16.7%). "Both the studies shows majority of patients are young adults".

There were few limitations of the study. Number of cases was not very high. Few patients had some co-existing problems which influenced the outcome of the patient. Etiology and mode of severe head injuries were not included. Prognosis of different age group was not observed. Combined efforts at a tertiary care hospital which include early diagnosis, surgical management, critical care management, good nutrition and hydration, physiotherapy and rehabilitation therapy and social support- all contributed to good prognosis.

Conclusion:

Choosing the right patient for surgery was always important. Deeply unconscious (GCS<5) patients with bilaterally dilated and/or non reactive pupils had poor prognosis. Combined efforts preferably at a tertiary care hospital which include early diagnosis, surgical management, critical care management, good nutrition, physiotherapy and rehabilitation therapy and social support- all contributed to good prognosis.

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Outcome of Postoperative Discitis at the Department of Neurosurgery at Chittagong Medical College Hospital: A Study of 20 Cases

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Abstract:

Background: *Postoperative discitis is a burning issue of Neuro & Spine Surgeon. Objective: Purpose of the study was to see the outcome of postoperative discitis patients with different modalities of treatment.* ***Method:*** *This was a prospective study, which was carried out at the Department of Neurosurgery, Chittagong Medical College Hospital from March 2006 to December 2009. Twenty patients, who had postoperative discitis were included in this study.* ***Results:*** *Among these patients, majority were age group 21-40 years 12(60%). 15(75%) patients were male. It was documented that the commonest causes of disc prolapse were degenerative 12(60%). The culture showed 15(75%) had no growth, 03(15%) had staphylococcus, 01(5%) had E. coli and 01(05%) had pseudomonas. It was evident that the 3(15%) patients had treated by flucloxacillin, 01(05%) ciprofloxacin, 01(05%) was by injection amikacin and 07(35%) patients were treated by Injection Vancomycin+Rifampicin. One patient needed reexploration. After three months follow up 17(85%) of patients improved and went to their daily works two (10%) of patients partially improved and 01(05%) did not improve.* ***Conclusion:*** *Postoperative discitis is a troublesome suffering of patients. The management is a challenge for the neurosurgeons. Treatment with inj. Vancomycin with Rifampicin can relief the patients suffering.*

Key words : *Discitis, Prolapsed lumber intervertebral disc, magnetic resonant imaging, discitis, rifampicin, vancomycin.*

Abbreviation: *MRSA= Methicillin resistant staphylococcus aureus, LP= Lumber puncture, CRP = C Reactive protein, MRI= Magnetic resonance imaging, VRE= Vancomycin resistant enterococci, CT scan= Computed tomography scan*

Introduction:

Incidence of discitis after lumbar discectorny is 0.2-4%. It may also occur after lumber puncture (LP), myelogram, cervical laminectomy, lumbar sympathectomy, chemonucleolysis, discography, fusions and other procedures. Risk factors include: advanced age, obesity, immuno-suppression, systemic infection at the time of surgery.[1]

There is some controversy as to whether these cases of post-operative discitis are not infectious, an autoimmune process has been implicated in some of these so-called “avascular” or chemical” or “aseptic” discitis cases. These cases are less common than infectious ones. Erythrocyte sedimentation rate (ESR) and C. reactive protein (CRP) abnormalities may be less pronounced in these patients, and biopsy of the disc space fails to grow organisms or show signs of infection on microscopy.[2]

Treatment of postprocedural spine infections should be initiated in a timely fashion. Both nonsurgical and surgical management have a role in selected cases. Identification of a microbial pathogen and administration of specific antibiotics are essential. In cases of discitis after microdiscectomy or spinal injections, CT-guided biopsy and aspiration may be sufficient to establish the diagnosis and start antibiotics. Blood cultures may also be obtained to help guide the antimicrobial therapy. While cultures are pending, broad-spectrum antibiotics with antistaphylococcal coverage may be instituted. The cornerstones of nonoperative management of postprocedural discitis are immobilization/bracing and organism-specific antibiotic therapy.[3] The exact length of the antimicrobial treatment is based on clinical, laboratory, and radiographic responses. In the

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nonoperatively managed patients, serial clinical examination and laboratory surveillance tests with WBC count, ESR, and CRP are performed. Special considerations for the management of a surgical site infection involving antibiotic-resistant organisms include minimizing patient contact and reduction of the spread of the organism by adhering to universal precautions[4] and the use of organism-specific antimicrobials. However, with the increasing prevalence of resistant organisms, standard antibiotic regimens are becoming less effective. Fortunately, new classes of antibiotics with activity against MRSA and vancomycin-resistant enterococci (VRE) are available.[5] In addition to systemic parenteral antibiotic therapy, local wound care along with the use of topical antimicrobial agents may also be of benefit in eradicating resistant organisms. Wright et al.[6] showed that silver in the form of silver-coated dressing is a useful prophylactic or therapeutic agent for the prevention of wound colonization by organisms that impede healing, including antibiotic-resistant bacteria.

Absence of improvement after 6 weeks of antibiotics and bracing may necessitate an open biopsy, surgical debridement, and stabilization.[7] Other indications for open surgical debridement and management of post-procedural spine infections are as follows: drainage from or dehiscence of the incision, clinical sepsis, neurological deficits secondary to fluid collection or mass effect, a spinal or epidural abscess, and instability from bone destruction or implant/fixation failure.[7,8] The goal of surgery is to debride all necrotic and nonviable tissue followed by stabilization of the spine to prevent deformity and/or neurologic injury.

Results:

Our study showed that the commonest age group were 21-40 years, 12(60%), followed by second common age groups were 15-20 years, 5(25%) (Table-I).

Sex distribution of patients showed male 15(75%), predominant the female (Table-II).

It was evident in our study that day labourer 8(40%) were the highest occupational group, service holder, and farmer belonged to 5(25%) and 4(20%) (Table-III).

The commonest causes of prolapsed lumber intervertibral disc were degeneration 12(60%). Others were road traffic accident 3(15%), fall from height 1(5%) and lift of heavy weight 3(15%) (Table-IV).

The commonest site of compression were L4/5 space 15(75%). MRI of lumbo sacral spine showed hypeintence area at disc space and at margin of end plate of body (Figure 1, 2 and 3).

It was documented that organism that causes infection were staphylococcus, 3(15%), E. coli (Escheresiacoli) 1(5%) and pseudomonas belonged to 1(5%) (Table-VI).

The majority of patients 08(40%) were treated by flocloxacillin +levofloxacin.

Others were by rifampicin+vancomycin 7(35%) (Table-VII).

In our study after 3 months follow up 12(60%) of patients improved excellently, 5(25%) of patients had good outcome and 2(10%) of patients had fair outcome. 1(5%) of our patients had poor outcome after 3 months followup who needed additional operative intervention due to radiation of pain.

Table-I

Distribution of patients by age (n=20)

Age in years	Number	Percentage
15-20 years	05	25.00
21-40 years	12	60.00
41-60 years	02	10.00
>60 years	01	05.00

It was evident that highest age group was (60%) 21-40 years.

Table-II

Distribution of the patients by sex (n=20)

Sex	Number	Percentage
Male	15	75.00
Female	05	25.00
Total	20	100.00

It was documented that male predominate the female 15(75%).

Table-III

Distribution of the patients by occupation (n=20)

Occupation	Number	Percentage
Day labour	08	40.00
Service holder	05	25.00
Farmer	04	20.00
House wife	02	10.00

The most commonest sufferer were day labourer 08(40%).

Table-IV

Distribution of patient by causes of prolapsed lumber intervertebral disc (n=20)

SL	Causes	Number	Percentage
1	Degenerative	12	60.00
2	Road Traffic accident	03	15.00
3	Fall from height	01	05.00
4	Lift of heavy weight	03	15.00
5	Unknown	01	05.00

It was documented that the commonest cause of disc prolapse were degeneration.

Table-V

Distribution patients by site of compression (n=20)

SL	Site	Number	Percentage
1	L4/5	15	75.00
2	L5/S1	04	20.00
3	L3/4	01	05.00

It was evident that L4/5 space was the commonest space.

Table-VI

Distribution of patients by type of organism

Type organism	No	Percentage
Staphylococcus	03	15.00
E. coli	01	05.00
Pseudomonas	01	05.00
No growth	15	75.00

The most common organism of discitis was staphylococcus aureus.

Table-VII

Distribution of patients by type of treatment with antibiotic

Type antibiotic	Number	Percentage
Flucloxacillin	03	15.00
Ciprofloxacin	01	05.00
Amikacin	01	05.00
Flocloxacillin+levofloxacin	08	40.00
Rifampicin+vancomycin	07	35.00

It was documented that the commonest organism of discitis was staphylococcus aureus.

Table-VIII

Modified Macnab criteria to assess the clinical outcome after treatment

	Number	Percentage
Excellent (free of pain, no restriction of mobility, able to return to normal work and activities)	12	60.00
Good (Occasional nonradicular pain, relief of presenting symptoms, able to return to modified work	5	25.00
Fair (Some improved functional capacity, still handicapped and/or unemployed)	2	10.00
Poor (Continued objective symptoms of root involvement, additional operative intervention needed at the index level, irrespective repeat or length of postoperative follow up)	1	5.00

This most 12(60%) of the patients were improved after treatment.

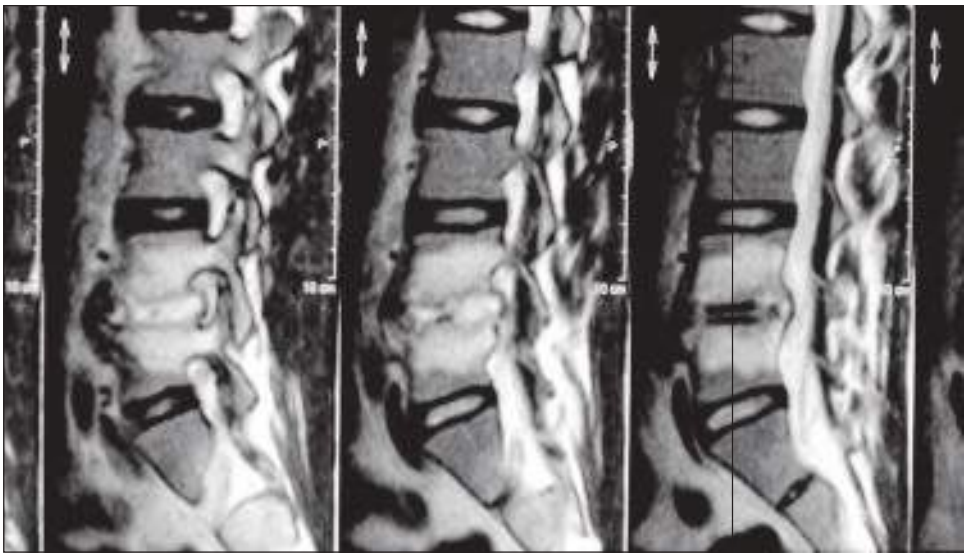

Fig.-1: *Postoperative discitis of L4, 5 space*

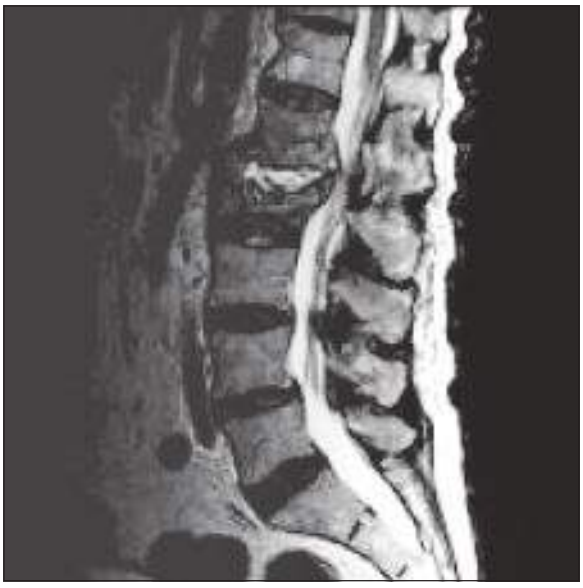

Fig.-2: *Post operative discitis of L1,2 space*

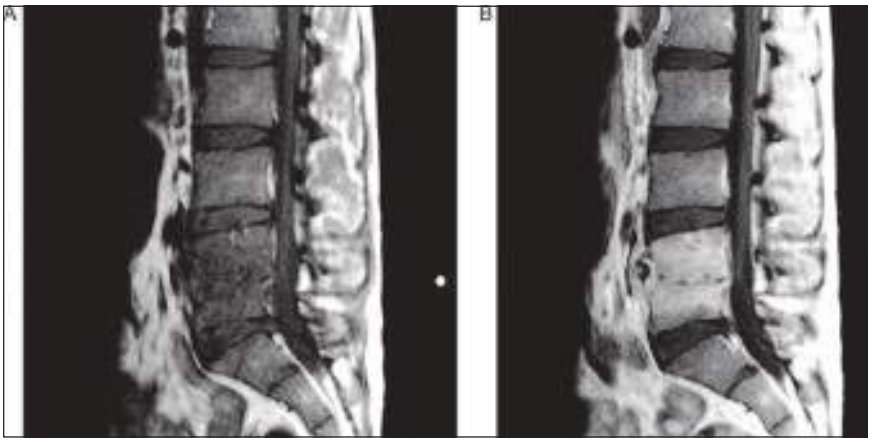

Fig.-3: *Postoperative discitis with fusion of L4, 5 space*

Discussion:

Moderate to severe back pain at the site of operation was the most common symptom, exacerbated by virtually any motion of the spine. Infection (discitis) is important problem following operation as literature reports only 30-50% can present with chills and fever.[9]

Pain radiating to hip leg, scrotum, groin, abdomen or perineum, true sciatica were uncommon. Signs of paravertebral muscle spasm was in all patients and limiting range of motion of the spine was found.[9]

An elevated ESR that never decreases after surgery was a strong indicator of discitis. C-reactive protein (CRP), an acute phase protein synthesized by hepato-cytes that may be a more specific indicator of post-op infection than ESR be-cause of rapid decomposition.[10] In our series CRP and ESR is not documented.

In our study revealed that 03(15%) had staphylo aureus, 01(5%) had E. coli, 01(5%) had pseudomonas & 15(75%) had no growth.

Most studies report staph epidermidis is the most common causes of postoperative discitis.[1] Other studies report staphylococcas aureus common identified organism of discitis followed by other staph species. Also reported: Gram-negative organisms, including *E. coili, Strep.* viri-dans, streptococcus species anaerobes, TB and fungi.[1] So our study correlated with previous study.

In our study 08(40%) were treated by flucloxacillin+levofloxacin empirically and those patients who were not responding with this combination with treatment of 3 weeks were treated by injection vancomycin+rifampicin 07(35%), for 4 weeks to 8 weeks.

In our series 3(15%) patients were treated by flucloxacillin, 01(5%) treated by ciprofloxacin and 01(5%) by injection amikacine.

Previous studies showed discitis were treated by analgesics + muscle relaxants+ antibiotics. IV antibiotics for 1-6 wks or until ESR decreases. Most start with anti-staphylococcal antibiotics (initial empiric therapy: van-comycin + rifampin) and a broad spectrum antibiotic (e.g. cefizox), mod-ify based on sensitivities if positive cultures are obtained activity restriction one of the following used, usually until significant pain relief[11]. In our study those patients who did not respond to other study were treated by Inj. Vancomycin rifampicin. So our study had similar sensitivity to antibiotic to previous study.

In our series after 3 months follow up 12(60%) patients improved excellently, 05(25%) patients had good improvement, 02(10%) had improve fairly and 01(5%) patients had poor improvement. One patient needed re-exploration.

In previous studies reported, all patients eventually became pain free. This is not the case in all series, where some report 60% were pain free at follow up. others found slight back pain in most patients, and yet others report severe chronic low back pain in 75%. No difference in outcome was found for the various activity restrictions specified, ex-cept for earlier pain relief with first two types listed above[11]. In our study those patients who did not respond to other drug were treated by Inj. Vancomycin and rifampicin. So our study had similarity of antibiotic therapy with previous study.

In certain cases of refractory discitis/osteomyelitis after less invasive procedures, minimally invasive techniques entailing percutaneous disk debridement and fusion have been described[8,12,13]. This approach is appropriate only in the absence of a significant superficial or deep collection and would not be used for an open draining wound. Minimally invasive techniques have the advantages of less postoperative pain, shorter hospital stay, and possibly an accelerated healing response[8,12,13]. The success in the lumbar spine is overshadowed by the high complication rate of 33% in the thoracic spine. Complications include the need for conversion to open procedures to address inadequately drained epidural abscesses[8].

Contrary in our study reflect the good outcome. In this study modified Macnab's criteria showed the clinical outcome after treatment. Excellent out come in 60% cases, good outcome in 25% cases, fair outcome in 10% cases, poor outcome in 5% cases and no improvement with medicine and needed reexploration. After reoperation patient improved clinically.

Conclusion:

Postoperative discitis is a burning problem for the neurosurgeon. Common cause of discitis are staphylococcus aureus and normal skin flora, stapylo epidermidis. Staphylo-epidermydis is sensitive to combination of rifampicin and vancomycin. Those patients whose do not respond to conventional antibiotic can be treated by this combination empirically.

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Outcome of Extradural Hematoma in Pediatric Age Group after Surgery

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Abstract:

Aim: *Out experiences in the management of extradural haematoma (EDH) in pediatric patients which involved an aggressive diagnostic approach, prompt surgical evacuation results in an excellent outcome.* ***Material and Methods:*** *Sixty five EDH patients who underwent surgery in the department of neurosurgery in Dhaka medical College hospital and private hospitals from December 2009 to December 2010 included in this prospective study. Each patient evaluated in term of age, sex, mode of injury, localization of haematoma, clinical presentation, CT findings, operative measures and outcome.* ***Results:*** *Out of 65 cases 72% (47) were boys and 28 % (18) were girls. The boys to girls ratio was 2.58: 1. Age ranged from 1 to 18 years with a mean age of 11.59 years. Most of the victims are in the 11-18 years age group 65% (42). The most common mode of injury was fall 37% (24) followed by Road traffic Accident (RTA) 32% (21) .The most common clinical presentation was altered sensorium 65% (42), followed by headache and/or vomiting 52% (34). The mortality rate was 8%.* ***Conclusion:*** *EDH is recognized as one of the most rewarding neurosurgical emergencies. It must be diagnosed in the early period of the trauma and evacuated immediate to prevent potential mortality and morbidity. Different factors affect the outcome of extradural haematoma like surgery including age of the patient, presence of cranial fractures, associated brain lesions and pre-operative neurological condition of patient, duration of time interval between onset of coma and surgical intervention. Better prognosis is seen in patients less than 11 years of age.*

Keywords: *Extradural haematoma, Surgical management, Neurosurgical emergencies*

Introduction:

Extradural haematoma (EDH) is an acute crisis after head injury, if diagnosed early then promptly treated. The incidence of EDH among traumatic brain injury (TBI) patients has been reported to be in the range of 2.7 to 4%[1]. It has been estimated that EDH represents 2–3% of all head injuries in the pediatric population[2]. The mean age of pediatric patients harboring EDH is between 6 and 10 years and it is even rarer among infants under the age of 12 months[1,2,3]. It is recognized that EDH in children differ from EDH in adults that the haematoma may follow a trivial injury, the symptoms are different, the course is more insidious, associated skull fracture is infrequent [16] and these make it difficult to diagnose and often challenging to manage. Furthermore, the criteria for utilizing surgical evacuation vs conservative management have remained ill-defined. Thus, the lack of any guideline regarding the appropriate management of EDH in pediatric patients and particularly in infants makes the management of this specific group of patients all the more complicated[2]. The reported mortality rates of EDH in children are quite different varying between 0% and 17%[4]. These considerable variations in the outcome and lack of data that provide a clear cut picture of EDH in children prompted us to carry out this study. We present our experiences with sixty five cases of acute epidural haematoma in children less than 18 years of age. Here, we also discuss the age-related findings and the differences with adults.

Material and Methods:

From December 2009 to December 2010, sixty five patients were surgically managed at the Department of Neurosurgery, Dhaka Medical College hospital & other private hospitals. This study includes these patients and categorized them according to age and were divided into four groups, for analysis and comparison of their presentation and management. A meticulous physical examination, with an emphasis on neurological function, was performed on admission.

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The diagnosis of haematoma was confirmed by CT scanning. In addition, standard epidemiological data including age, sex and mode of injury, presence of skull fracture, presenting clinical features from injury to surgery, preoperative Glasgow Coma Score (GCS) and pupillary reaction to light, presence of other injury, CT findings, localization of haematoma, the effect of injury and timing from injury to surgery were recorded. Surgical decision was determined by the following clinical and radiologic parameters: 1) Unconscious or deterioration of neurological status in patients with a extradural hematoma larger than 1 cm thickness, 2) Volume of the hematoma 30 ml even if the patient is awake and free from neurological deficit, 3) Volume of the hematoma 20 ml when located in critical sites, such as posterior cranial fossa or temporal base, 4) midline Shift > 0.5 cm with deterioration of level of consciousness,5) Increase in the hematoma size. Asymptomatic patients, patients presenting only minor symptoms with small haematoma (thickness less than 20mm), patients admitted 24 or more hour after trauma with neurological stable condition were selected for conservative management. These patients were very closely observed clinically by monitoring level of consciousness, focal neurological signs, and vital signs and follow up CT scan was done to asses clot size. We had to operate on 3 such cases when patient's level of consciousness and GCS were deteriorated and repeat CT scan showed increase in size of haematoma. All the cases were operated on emergency basis. Surgical management consisted of craniectomy and craniotomy under general anesthesia and removal of the underlying hematoma. Outcome was assessed on discharge and an outpatient basis within two month. The Glasgow Outcome Scale (GOS) and clinical condition was utilized for evaluating the outcomes.

Observations and Results:

The results were summarized in the table which illustrates the epidemiology and clinical features of the pediatric patients.

Table-I

Sex distribution of the patients

Sex	Number of patients	Percentages
Male	47	72
Female	18	28
M:F	2.58:1	

Table-1 summarized the age distribution of the patients. Out of 65 patients, 72% (47) were boys and 28% (18) were girls. The boys and girls ratio was 2.58:1.

Table–II

Age distribution of the patients

Age in years	Number of patients	Percentages
0-5	5	7
6-10	18	28
11-15	26	40
16-18	16	25

Table-II shows the age distribution of the patients. Age at the time of the accident ranged from 1 to 18 years with a mean age of 11.59 years. We found that the frequency of acute traumatic extradural haematoma in pediatric patients increases with age, and comparatively elder children (age group 8-18 years) were the commonest victims.

Table-III

Mode of injury

Mode if injury	Number of patients	Percentages
fall from height	24	37
RTA	21	32
Assault	14	22
Trauma to head by heavy weight	06	9

Table-III shows the mode of injury among 65 patients. The most common mode of injury was fall from height 37 % (24) followed by road traffic accident (RTA) 32% (21).

Table-IV

Common site of hematoma(n=65)

Common site of hematoma	Number of patients	Percentages
Temporoparietal	22	34
Frontal	18	28
Parietal	10	15
Temporal	9	14
Posterior fossa	4	6
Occipital	2	3

Table-V

Presentations of the patients

Presentations	Number of patients	Percentages
Clinical Presentation		
Altered sensorium	42	65
Headache / Vomiting	34	52
Lucid interval	17	26
Neurodeficit (hemiparesis)	13	20
Seizure	9	14
Cerebral oedema	23	36
Associated injuries		
Skull Fracture	39	60
Contusion / ICH Acute	17	26
Subdural Haematoma	4	6
Extra cranial injuries sustained		
Long bone fracture	12	18
Maxillofacial injury	8	12

Table-V shows the common site of hematoma. According to CT finding, temporoparietal region was involved in 34 % (22) followed by frontal and parietal region in 28 % (19) and 15% (10) respectively. The posterior fossa involvement was very few, just 6 % cases (4).

Table-VII

Time interval between trauma and operation

Time interval between trauma and operation (Hours)	Number of patients	Percentages
0 - 12	9	14
13 - 24	35	54
25 – 48	21	32
J. Mortality rate	5	8

Table- VI summarized the presentation among 65 patients. The most common clinical presentation was altered sensorium 65% (42), followed by Headache / Vomiting 52% (34). Aside from these, 20% (13) patients had neurological deficit. Early seizures were recorded in 14 % (9) children presenting with EDH. The classically described “lucid interval” i, e. a patient who is initially unconscious, then wakes up and secondarily deteriorates, was observed in 28 % (18) cases. 60% (39) patients showed skull fractures demonstrated either by x-ray, or CT scan or discovered at the time of operation. Associated extra cranial injuries were present in 31 % (20) cases.

Table-VII

Time interval between trauma and operation and mortality rate.

Time interval between trauma and operation (Hours)	Number of patients	Percentages
0 - 12	9	14
13 - 24	35	54
25 – 48	21	32
Mortality rate	5	8

All the cases were operated on emergency basis and two third of cases (68 %) were operated on within the first 24 hours. Five patients (8 %) died after surgery. Among these 5 patients, 2 had associated brain injuries, 3 cases were deeply unconscious and 4 of these had fixed pupil / pupils at the time of admission.

Discussion:

Only a small number of articles of pediatrics extradural haematomas based on surgical experiences and prospective analysis exist in the literature. Traumatic extradural haematoma (EDH) in children have some unique features when compared with those in the adult population. Traumatic extradural haematoma (EDH) in children accounted for 11 % of the all patients with an epidural hematoma operated in our institution in the same period. This lower incidence of acute epidural hematoma in the children compared with adults is also reported in other series and is attributable to the tight adherence of the dura mater to the inner table of the skull[5,4,6,7]. In our series, the age of the patients ranged from 1 years to 18 years with a mean age of 11.59 years. There were 72% males and 28% female patients; the ratio between them was 2.58:1. Our analysis has identified that EDH is more frequent in 11 years of age or above among children (60%). Only 5% of the victims were 0-5 years. These data is correlated with other reported series of EDH in children [9,20,11,6]. This is due to the high-velocity trauma mechanism in the older age group[12]. In this series majority of patients were boys (72%). It is similar to other reported series of EDH in children[5,10,13,14]. It was reflecting the natural tendency of boys to indulge in risky play activities. In our series, falls were the predominant modes of injury (37%) followed by RTA (32 %). Out of 24 victims of fall, (11/24) were in domestic, (9/24) in sports and recreation; and (4/24) were in work. Similar results have been reported by

other authors[5,6,7,10,13,] but Dhellemmes found that 64% patients with EDH had been involved RTA and other causes had occurred less frequently[5]. RTA is the commonest mode of injury in adult also[8,4]. So, this series showed a significant difference in the mechanism of injury between children and adults. The classical presentation of traumatic extradural haematoma, as described in the majority of text books was the exception rather than rule. In this series, most patients (65 %) had altered sensorium and we strongly believe that this is the most significant sign of EDH in Children. It is supported in other reported series also[5,10,11]. 52 % Children had headache associated with persistent vomiting. It is nonspecific but important clinical sign. In our series we evaluated other clinical signs such as hemiparesis (20%) and pupil dilatation (21%). The classically described "lucid interval" was observed in 26 % cases in our series. It is almost similar to Ersahin et al[15] who had found lucid interval in 37% cases but was contradictory to Hanci et al[10] who discovered it was only in 11.125 cases. Epilepsy is a recognized complication of EDH, especially in the presence of associated intradural (ID) damage[4]. In our series, early seizure was noted in 14% cases. Our data is almost double than Ben Abraham et al.[3] who reported early seizure in 8% of pediatrics patients only. In this series, there was a significant difference between children and adults in the clinical course. Simpson et al.[12] pointed out that diagnosis of EDH in a child may not be made until early clinical feature of raised ICP is present. Young children can tolerate an acute increase in intracranial pressure better than adults because they have unfused cranial sutures, open fontanelles, large extracerebral spaces and basal cisterns, and moreover the origin of EDH in children is often venous whereas in adults, it is mainly caused by an arterial bleeding[16]. In our series, we evaluated clinical signs as expected. Accordingly early detection of the lesion is critical. The sign / symptoms were nonspecific in majority of cases. Based on clinical findings early diagnosis was established only in small percentage of cases. Evolution over time influences the treatment of EDH significantly. We believe that CT scan of head should be done routinely in suspected case as early diagnosis of EDH is mandatory for good recovery as radiological changes always occur earlier than clinical changes and should be monitor to predict the clinical progression[12,17,18]. In this series, the temporoparietal region (34%) was the commonest site followed by frontal region. It is similar to Hanci et al[10] who had opinion that EDH originating in the fronto temporal region does not spread to the frontal region. A possible explanation is the adherence of dura at the suture line[19]. In our series, as regard location, frontal haematomas have shown a better prognosis and a slower course than convexity haematomas. It is contrary to Mohanty et al[20] who found that the site of haematoma had no correlation with the final outcome. We also found that the GCS score and the associated parenchymal injuries had a strong correlation with the outcome both in adult and children. It is in strong agreement with Mohanty et al. [23] experiences. In our series, cranial fractures were present in 60 % patients' with significantly lower mortality rate. In many reported series, cranial fractures were in between 70-95%[1,21,22,23]. The impact of fracture on outcome is controversial. Kuday et al. (18) observed a significant relationship between cranial fractures and adverse outcome in 115 patients undergoing surgery for EDH. Lee et al. [18] did not see this relationship in a series of 200 patients managed similarly. But Rivas et al. [24] reported a significantly lower mortality rate in patients with cranial fractures which is in strong agreement with our findings. However, incidence of skull fracture in children in this series is lower than adults[9]. The rate and type of associated extracranial injuries (31%) in our series is higher than in the study by Duthie et al [12]. 5% of children in their study had associated extracranial injuries. The presence of CT scan evidence of a concomitant intradural (ID) injury is recognised as a poor prognostic factor (16). In our series, associated brain injuries discovered in 26 % cases. These are predominantly contusions, intra-cerebral haemorrhage and subdural haematomas. The incidence of associated lesion in reported series is less in pediatrics age group[7, 8,16,21,23]. SDH and / or parenchymal injuries in association with EDH lower the chance of good outcome[7]. Like our series, in children the most common concomitant intradural abnormality was brain edema, while in adults it was hemorrhagic contusion[16]. Despite a steady decline in mortality, in this series the mortality rate was 8%. Among these, 40% had associated brain injuries and 60 % victims of these had fixed pupil / pupils and deeply unconscious at the time of admission. Mazza et al.[19] discovered associated brain lesions in 40% of his cases, over all mortality rate was 17%, with 14% operative mortality. Ersahin et al. [13] found 10% overall mortality, with mortality rate in the CT and plain x-ray

groups 6% and 16% respectively. In this series, we observed that outcome showed an age-related discontinuity because the prognosis worsen with age increases. The mortality rate in children was less than in adult series [9] but 0% mortality as proposed by Ammirati [1] and Bricolo [6] should be the goal of EDH surgery.

Conclusion:

Extradural haematoma in pediatric age group is one of the most rewarding neurosurgical emergencies that must be recognized and treated immediately. An extradural haematoma must be considered in any child whose condition does not improve rapidly following a relatively mild head injury and it can be enlarged while child is under supervision. It is not rare for EDH to develop and present in delayed fashion. Timely diagnosis and prompt surgical evacuation of the haematoma results in excellent outcome. Our experiences of sixty five surgically managed cases produced such a result. Many factors affect the outcome of extradural haematoma surgery. In addition, presence of cranial fractures, associated brain lesions and pre-operative neurological status of patient, duration of time interval between onset of coma and surgical intervention determine the outcome of patient. Morbidity and mortality have also been shown to be affected by age, with better prognosis in patients under 10 years of age.

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Experience of Carpal Tunnel Syndrome Surgery- Clinical Study of 64 Cases

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Abstract:

Aim: *In our neurosurgical practice, releasing the carpal tunnel for Carpal Tunnel Syndrome (CTS) is the most common surgical procedure. In this study, we aimed to analyze the outcome of patients operated on for carpal tunnel syndrome using small skin incision under local anesthesia.* ***Material and Methods:*** *From January 2007 to December 2010, we performed 75 carpal tunnel releasing procedures for 64 patients. There were 57 (89%) female and 7 (11%) male patients with a mean age of 34 (21-62). 41 operations were performed for the right hand and 34 for the left hand.* ***Results:*** *Each patient was evaluated by history, physical examination and nerve conduction velocity test (NCV). All patients had brachialgia paraesthetica nocturna showed severe CTS by NCV. Pre- and post-operative Visual Analog Scale (VAS) was used for clinical evaluation. There was no complication such as bleeding or nerve injury in the operated patients. The follow up period was 1 to 3 months. The mean VAS score was 8.1 pre-operatively and 2.2 post-operatively. One patients underwent re-operation because of persistent of symptoms. There was no procedure-related complication during the follow-up period.* ***Conclusion:*** *The small skin incision is a safe and effective procedure for releasing the carpal tunnel.*

Keywords: *Carpal Tunnel Syndrome, Small Skin incision, Minimally invasive*

Abbreviation: *NCV-Nerve Conduction Velocity, CTS-Carpal Tunnel Syndrom, VAS-Visual Analogue Scale.*

Introduction:

Carpal tunnel syndrome (CTS) is the most common entrapment neuropathy in the upper extremity. The median nerve is compressed within its course through the carpal tunnel just distal to wrist crease [1]. It results in symptoms of dysesthesia and burning pain in the hand and it mainly affects the middle-aged female population [2]. CTS accounts for 90% of all entrapment neuropathies and its incidence is approximately 1% in the general population [3]. Palmer et al. reported in 1995 that between 400.000 and 500.000 cases require surgical treatment per year in the United States [4]. History, physical examination and electrophysiological results must be evaluated for the diagnosis of CTS [5]. Patients with mild symptoms of CTS can be managed with conservative treatment including non-steroid anti-inflammatory drugs, vitamin B6, local steroid injections or hand braces [2, 3]. Surgical treatment is generally required in patients with moderate and severe symptoms [6]. Various methods have been described for the surgical treatment of CTS. Standard open carpal tunnel release with a long palmar curvilinear incision still remains to be the preferred surgical procedure for many departments and neurosurgeons [7], but this procedure has many complications including pillar pain, scar tenderness, cosmetic problems, loss of grip and pinch strength or time losses due to inability to work [8]. Endoscopic techniques and different limited skin incisions are described in the literature to minimize these complications [9]. The aim of this study was to analyze the results of patients who were operated by using small skin incision under local anesthesia.

Material and Methods:

From January 2007 to December 2010, we performed 75 carpal tunnel releasing procedures on 64 consecutive patients. There were 57 (89%) female and 7 (11%) male patients with a mean age of 34 (21-62).

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41 operations were performed for the right hand and 34 for the left hand. Each patient was evaluated with his/her history, physical examination and nerve conduction velocity test (NCV). Brachialgia paraesthetica nocturna and numbness were observed in all patients. Additional symptoms and signs included positive Phalen's test in 55 patients (86 %) and positive Tinel's sign in 59 patients (92 %). The pain status of the patients were pre and postoperatively assessed with the Visual Analog Scale (VAS). All the patients and their families were informed about the operation technique before the operation. Every operation was performed in the operating room under sterile conditions in the supine position. Before surgery, the affected hand, wrist and forearm were cleaned with povidone iodine solution. The area to be operated was covered with a sterile draping sheet. Local anesthesia was performed with 10 ml of 2 % lignocaine and no tourniquet was used. Operation was done with or without microscope. The hand should be in extended position. This position allows a good surgical route. After these routine operation preparations, a longitudinal 1 to 3 cm long incision was performed at the wrist region, proximal to the distal flexion crease, between the third and fourth finger (Fig 1). The incised skin was retracted with the help of a mini retractor and subcutaneous fat tissue was dissected laterally. The proximal part of the carpal ligament was passed with a surgical blade, and then the ligament was cut with scissors (Fig 2). After the irrigation and homeostasis, the skin was sutured with 3/0 prolene and it was removed on 10 to 12th POD (Fig 3). The mean operation time was 20 to 25 minutes (ranging between 15-25 minutes). The mean hospital stay was maximum 1 hour.

Fig-1: *A longitudinal incision mark was performed at the wrist region.*

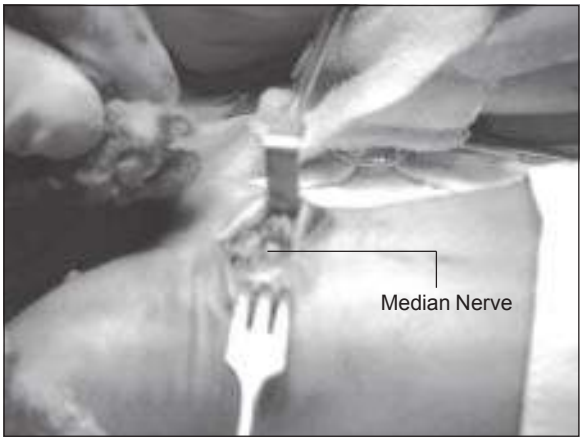

Fig-2: *Peroperative appearance of median nerve after releasing the carpal tunn*

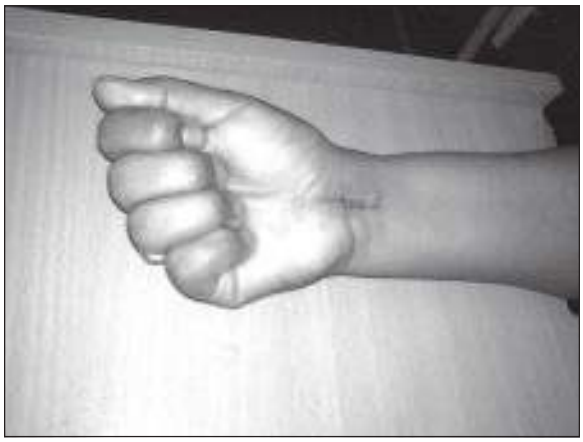

Fig-3: *The skin incision appearance after an operation performed 1 month ago.*

Results:

Table-I
Sex Distribution

Sex		Percentage
Male	7	11
Female	57	89
Total	64	100

Table I Shows 89%(64) were female and 11%(7) were male

Table-II
Result of Pain Score

Before operation	After operation
8.1	2.2

Table II Shows pain score (VAS) before operation was 8.1 and after operation 2.2

In this study, 75 carpal tunnel release operations were performed on 64 patients (table-I). There were no complications during the operations such as bleeding or nerve injury. The follow up period was 1 to 3 months and no procedure related complication was observed such as skin infection and palmar tenderness. The mean pre-operative VAS score for pain was 8.1, which decreased to 2.2 postoperatively (table-II). In this study, 2 patients had temporary paraesthesia and 1 patients were reoperated because of pain recurrence and paraesthesia. Patient was reoperated with the same technique. No problem occurred during the follow-up period.

Discussion:

CTS is a common condition causing burning hand pain, paresthesia and dysfunction. It affects mainly middle-aged female population [2, 3]. In this study, just like in the literature, 89% of the patients were female [5]. In moderate and severe cases, surgery is the only treatment option that provides cure and there are many surgical techniques for releasing carpal tunnel. Until recent years, standard incision with a long curvilinear incision was the most performed technique by many neurosurgeons. This technique is safe and effective as reported by authors, but it has some complications [10]. Early complications including incomplete release of carpal ligament, injury to the palmar cutaneous and recurrent motor branch of median nerve or injury to the superficial palmar arch and ulnar artery are rare because the operation is performed under direct vision [2, 3]. Late complications, on the other hand, have a relatively high incidence. These are hypertrophic scar formation, scar tenderness, pillar pain, loss of grip strength and sympathetic dystrophy resulting in the delay of returning to daily activities or work and emotional distress [11]. To reduce these complications, various limited incisions or endoscopic techniques are described by authors [12-15]. During the last two decades, endoscopic carpal tunnel release procedures have become popular and have been widely used by surgeons. In spite of the many advantages of endoscopic techniques, there are also some disadvantages. In 2008, Nazzi et al. drew attention to the disadvantages of endoscopic techniques; the difficulty of inserting a relatively large device through a narrow tunnel, nerve ischemia due to the use of tourniquet for a long time, performing transverse incision that might damage the superficial palmar arch and the experience needed for such an operation [16]. Also it is reported that the most common complications of these techniques are paresthesia of the median and ulnar nerves, tendon lacerations and injury to the arteries [3]. Some authors have also reported multiple limited mini open incision techniques to decrease the postoperative morbidity observed in standard open techniques [13, 17]. Mini open procedures have been performed using either a longitudinal incision on the wrist and/or palmar surface, or a transverse wrist incision. In 1996, Franzini et al. reported a minimally invasive technique with a small longitudinal incision of 1 cm, proximal to the wrist crease. They performed 473 operations by using that incision and transillumination technique with 90 % complete remission [11]. In this study, we aimed to analyze the outcome of patients operated for carpal tunnel syndrome using small skin incision under local anesthesia with or without microscope. This technique is the combination of Franzini's 1 cm. longitudinal incision and the technique reported by Aydýn et al. that allows the use of an operative microscope [4, 11]. The incision is performed at the wrist region and the proximal part of distal flexion crease where the skin is thinner than the distal side and palmar surface; therefore, we thought that the complications including cosmetic problems, palmar tenderness and scar sensitivity would be less. In this study, we did not see any complication caused by the type of incision and when the patients were return to daily routine activities, palmar tenderness and scar sensitivity. In 2008, Nazzi and Franzini reported a technical note about their experience of three different non-endoscopic minimally invasive surgical techniques for carpal tunnel release [16]. They obtained 90 % complete remission of symptoms with their first technique, which is the same as with our incision type. In our study, there were 1 reoperations because of the recurrence of symptoms and 2 patients had temporary paresthesia. 59 patients (92%) had complete remission of pain and acceptable remission of numbness during the follow-up period. In the literature, it is reported that either the recurrent thenar branch or palmar arteries have a potential risk of injury during all endoscopic and limited incision techniques [2]. The recurrent thenar branch may leave the median nerve from various anatomic places. These variations may cause difficulties for the surgeon to release or protect the recurrent thenar branch. The palmar arteries, and particularly the superficial palmar

arch, also have a potential risk due to the difficulties in visualization with these limited incisions or endoscopic techniques. However, just like the studies of Franzini and Aydýn et al., we also did not experience any artery, nerve or tendon injury [4,5,16].

Conclusion:

The technique for carpal tunnel release using small skin incision is a safe and effective surgical procedure. It can be used in the surgical treatment of CTS to achieve better palmar appearance, excellent cosmetic results and to reduce the complications of other standard techniques.

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Outcomes following Purely Endoscopic, Endonasal Resection of Pituitary Adenomas

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Abstract

Background: *The use of endoscope for the management of pituitary adenoma is not new. The better magnification and illumination provided by the endoscope gives better outcome than microscopic pituitary surgery.* ***Objectives:*** *The purpose of the study to find the benefits of endoscope in relation to microscopic surgery.* ***Methods:*** *We did 45 cases of pituitary adenoma surgery by endoscopic endonasal approach from July 2008 to July 2010.* ***Results:*** *Among 45 cases underwent endoscopic transsphenoidal approach gross total removal was done in 35 cases; rest 10 cases subtotal removal was done. Residual tumour were seen in 10 cases (22%) in postoperative follow-up MRI scan.Visual improvement was satisfactory, and hormonal improvement of functional adenoma was nice. Postoperative visual acuity and visual field was improved in 75% cases. There were 37% cases of temporary D.I. and about 4.5% cases of permanent D.I. The average duration of follow-up was 20 months. One patient was required re-exploration to correct visual deterioration in the immediate postoperative period. There were 4.5% cases of C.S.F. leak and 6.6% mortality. Mortality was from electrolyte imbalance and from improper management of infection and hydrocephalus.* ***Conclusion:*** *Endoscopic endonasal pituitary surgery now become a gold standard surgery for most of the pituitary adenoma because of its better advantages in relation to microscopic surgery and less complications and less hospital stay.*

Key Words: *Endoscopic pituitary surgery, Endoscopic transsphenoidal approach, Endonasal endoscopic approach.*

Introduction:

Pituitary adenomas are slow growing tumours that constitute about 10-15% of all intracranial neoplasms. They can produce compression symptoms when enlarged or give rise to hormonal disturbances. These tumours are often diagnosed late or remain undiagnosed. Radiology is the best tool for diagnosis along with hormonal assays[1]. Since the late 1970, the transsphenoid approach has been the preferred procedure for removal of these tumours[1, 2]. With the advent of endoscopic surgery the endoscopes have now been applied to access these tumours with favourable result. The better magnification and illumination provided by the endoscopes has helped in precise delineation of the tumour and has ensured completeness of tumour removal[2]. It has also greatly reduced the postoperative morbidity.[2] Embryologically Pituitary tumour is formed partly from brain tissue itself (the posterior lobe or neurohypophysis) and partly from upward extension of the Rathke's pouch, (anterior lobe or adenohypophysis)[1,2,3]. The average size of the pituitary gland is 12mm (transverse) x 8mm (sagittal) x 6mm (vertical). It secretes various hormones required to maintain normal metabolic and cellular functions within the body[3]. The gland has important relations with the optic chiasm and the cavernous sinus. The hormones secreted by the pituitary gland are, from

the Anterior Lobe : Secrets Thyroid Stimulating Hormone (TSH), Gonadotropins, Follicle Stimulating Hormone (FSH), Luteinizing Hormone (LH), Prolactin (PRL), Growth Hormone (GH), ACTH, Alpha Melanocyte-Stimulating Hormone (a-MSH), from the Posterior Lobe: Secrets Oxytocin, Vasopiesin. Classically, pituitary tumors are divided into two groups: functional (secretory) and non-functional (non-secretory), the non-functional tumour usually do not present until reaching a sufficient size to cause mass

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effect or compression on the surrounding neurovascular structures, mainly the optic nerves. The functional frequently present at an earlier stage caused by the physiological effects of the excess hormones they secrete. This distinction is not always adhered to as "secretory" tumors may grow large enough to cause symptomatic mass effect and "non-secretory" tumors, can grow and destroy the normal pituitary gland leading to a decrease in the secretion of some or all of the pituitary hormones, a condition known as panhypopituitarism which includes hypothyroidism, hypocortisolism, hypogonadism[4,5].

Methods:

We did 45 cases of pituitary adenoma surgery by endoscopic endonasal approach from July 2008 to July 2010. in BSMMU and some in private clinics. Patient characteristics (age, sex, follow-up), tumour factors (size, position extension, previous surgery), and outcomes (visual, endocrine, and surgical morbidity) were defined.

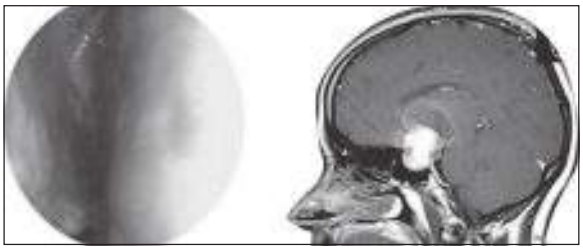
Steps of Surgery: The surgery was the done under general anaesthesia.

1. Position: The patient was supine on the operation table with the head resting on a horseshoe. The head and trunk raised 10^0 and the head slightly flexed and rotated 10^0 towards the surgeon.

2. Disinfection of the nasal cavities: Using a small killian-type nasal speculum, cotton pledgests soaked in 50% polyvidone-iodine are placed along the floor of the nasal cavities and in the space between the nasal septum and the turbinates, and they are allowed to take effect for approximately 5 minutes.

3. Choice of the Nostril: We usually choose both the nostril.

4. Nasal Stage: During this stage, a 0^0 –telescope, 4mm in length, was used freehand. Once the telescope has been inserted into the chosen nostril, the inferior and middle turbinates, and the nasal septum were identified. With a tilleys forceps, long cotton pledgets, soaked in diluted adrenaline (1/10,000) or xylomethazoline hydrochlorate, were inserted in the space between the middle turbinate and the nasal septum to achieve a vasoconstrictive effect particularly at the relevant, richly vascularized areas. The middle turbinate is gently medialized or removed to make sure that the surgical pathway, that passes between the nasal septum and the turbinate itself (Fig.1), is wide enough. Once the cotton pledgets were removed, there will be allowing for adequate inspection of the posterior portion of the nasal cavity, where the choana, the spheno-etmoidal recess and the sphenoid ostium are identified (Fig.1A)



A B

Fig.1A: Shows the middle turbinate and nasal septum.

Fig.1B: Shows the pituitary adenoma sagittal view and the base of middle turbinate correspond with sphenoidal air sinus.

5. Sphenoid Stage: The sphenoid stage of the procedure begins with coagulation of the sphenoetmoidal recess. The target site of coagulation is located approx. 0.5 cm above the roof of the choana, or from the base of the middle turbinate up to the superior margin of the nasal cavity. (Fig.2)

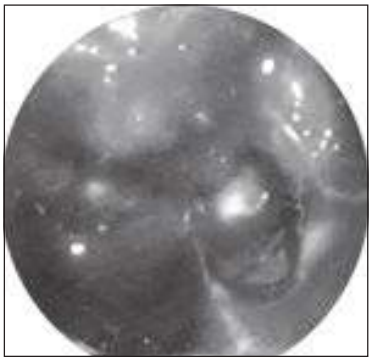


Fig.2: *Shows following sphenoidectomy sellar floor is seen.*

6. Sellar stage: To free both of the surgeon's hands and thus allow simultaneous use of two operating instruments during this stage of surgery, the 0^0-telescope (4 mm in diameter and 18 cm in length) was holded by assistant (Fig. 3). The consistency of the sellar floor depends on the type of lesion present in the sellar cavity; it is nearly always intact in microadenomas, while it is frequently thinned-out and/ or eroded in the case of pituitary macroadenomas.

Therefore, depending on its condition, the sellar floor may be intially opended by diamond drill. The opening may be enlarged with kerrison bone punches. The dural opening is made by a cross, an "X", or a rectangular incision (Fig.3).

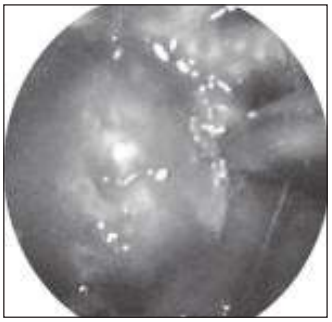


Fig.3: *Shows bulged pituitary dura following sellar bone removal.*

7. Tumour Removal: Following durotomy, the tumour was removed by suction and curettes. Sometimes by two sucker technique.

A B C

Fig.-4: *Shows tumour removal by pituitary microrounger (4A-B) and opening of suprasellar arachnoid after tumour removal (4C).*

8. Closure: Sellar reconstruction was conducted most of the cases, using various materials. Care have to be taken to avoid over packing which involves the risk of damage to the optic chiasm. If a CSF leak becomes evident during the operation, it was necessary to close the sellar floor by intradural, extradural or intraextradural ("sandwich") technique (Fig.5)

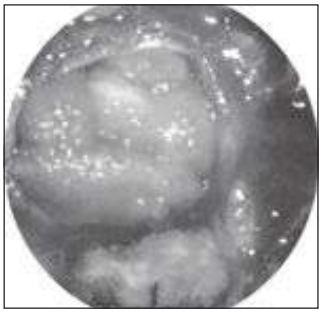


Fig.5: *Shows falling of the sellarfloor and sphenoidal air sinus by gel foam.*

Results:

We did endoscopic endonasal operations in 45 patients from July 2008 to July 2010 in BSMMU and some in private clinics. Among them 25 cases were women and 20 were men (Table-1). Male female ratio was 1:1.5. Age ranges were from 22 to 55 years (median, 33.5 years) (Table-1). Among 45 cases of pituitary adenoma there were 36 cases (82%) macroadenoma and 9 cases (18%) were microadenoma. Functional adenoma were 11 cases (25%) which includes acromegaly 6 cases (54%) cushing 4 cases (36%) and prolactinoma 1 case (10%) and rest 34 cases (75%) of the tumour were nonfunctional adenoma (Table-II).

Table-I

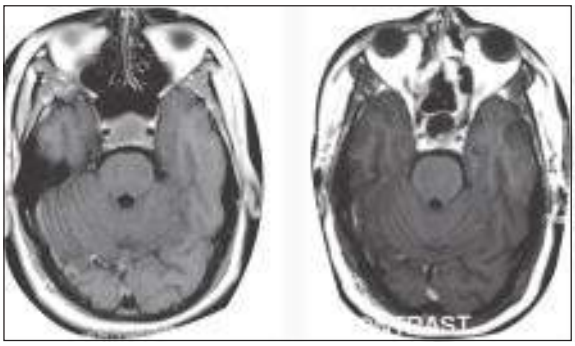
Shows sex and age distribution. (N-45)

Sex	No. of Cases	Percent	Age	No. of Cases	Percent
Male	20	44.44%	10-20	3	6.66%
Female	25	55.55%	21-30	6	13.33%
			31-40	10	22.22%
			41-50	18	40%
			51-60	8	17.77%
Total	45	100%	Total	45	100%

Table-II

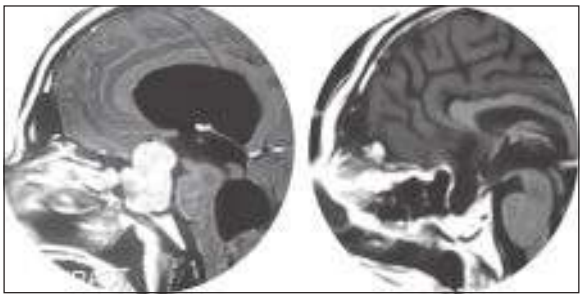
Shows distribution of pituitary adenoma.(N-45)

Pituitary Adenoma	Number of Cases	Percent
Pituitary macro adenoma	36	82%
Pituitary micro adenoma	9	18%
Total	45	100%



A B

Fig.-6: *Shows preoperative pituitary adenoma in axial view (A) and postoperative (B) total clearance following endoscopic endonasal surgery*



A B

Fig.-7: *Shows preoperative pituitary adenoma in surgical view (A) and postoperative (B) total clearance following endoscopic endonasal surgery*

There were 33 cases (73%) sellar enlargment found out of 45 cases (Table-III). Most of cases were having supersellar extension. Hydrocephalus were present in 5 cases (11.11%) (Table-III). Only 6 cases (13%) presented with pituitary apoplexy (Table-III).

The average duration of follow-up was 20 months and one patient was required re-exploration to correct the visual deterioration in the immediate postoperative period.

Ophthalmological results

Preop visual Presentation: Typical chaismal syndrome (bitemporal hemianopia) 25 cases (55%) or junctional syndrome (unilateral blindness and contralateral temporal field defect due to involvement of Von willebrand fibre of opposite optic nerve) - 18 cases (40%) Bil. Upper quadrentanopia 2 cases (5%).

Visual outcome were assessed postoperatively. Visual improvement was satisfactory Postoperative visual acuity and visual field was improved in 34 cases (75%) (Table-IV). All patients with visual field and/or visual acuity defect improved except two patients. Postoperatively, visual acuity was normal or improved in 75% of the eyes and the visual fields were normal or improved in 75%. (Fig.8-A, 8-B, 9-A, 9-B) (Table-IV) The visual outcome (for both acuity and fields) was better in younger patients and those with a shorter duration of symptoms. Patients with lesser degrees of preoperative visual acuity compromise had better postoperative visual acuity outcome. However, the severity of preoperative visual field defects did not seem to predict postoperative field outcome, and even patients with severe preoperative field defects often had striking postoperative improvement. Patients who had undergone prior operation were less likely to have good visual acuity improvement.

Endocrine results

Pre op Endocrine Presentation: Typical endocrinological findings are hypocortisolism, 12 cases (26%) hypothyroidism and hypogonadism-17 cases (37%) in Non functional adenoma. In Functional adenoma–Acromegaly-6 cases (13%)/Prolactinoma-1 cases (2%) Cushing syndrome- 4 cases (8%) are most commonst presentation.

Post op Endocrine outcome: Postoperative anterior pituitary dysfunction did improved in 17 cases (37%) out of 45 cases. There were 37% cases of temporary D.I. and about 5% cases of permanent D.I. Regarding functional adenoma-all patients of Acromegaly, Cushing and Proloctinoma improved except 1 case of acromegaly who died following surgery.

Extent of Resection and Recurrence: Among 45 cases underwent endoscopic transsphenoidal approach gross total removal were done in 35 cases, (Fig.6-B, 7-B) rest 10 case subtotal removal were done (Table-VI). Residual tumour was in 10 cases (22%) in postoperative follow-up MRI scan.

Table-III

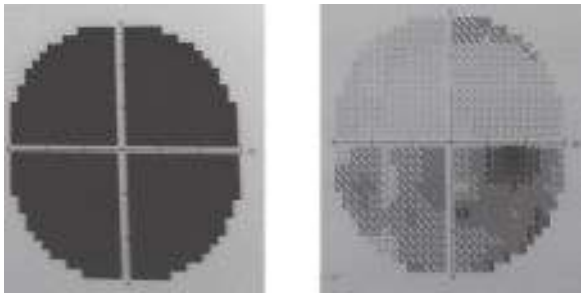
Shows distribution of hydrocephalus, pituitary apoplexy and sellar size.(N-45)

Hydrocephalus	No. of Cases	Percent	Pituitary Apoplexy	No. of Cases	Percent	Sellar Size	No. of Cases	Percent
Present	5	11.11%	Present	6	13.33%	Normal	12	26.66%
Absent	40	88.88%	Absent	39	86.66%	Enlarged	33	73.33%
Total	45	100%	Total	45	100%	Total	45	100%

Table-IV

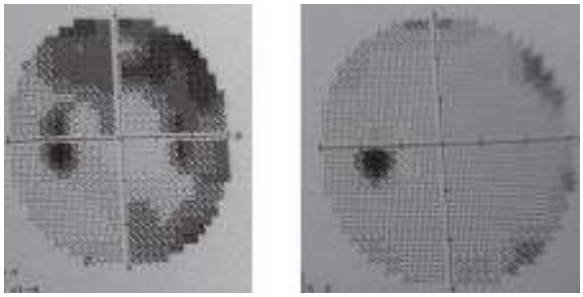
Distribution of post-operative visual status (N-45)

Post-operative visual status	No. of Cases	Percent
Improved	34	75.55%
Not improved / static	08	17.77%
Deteriorated	02	04.44%
Normal	01	02.22%
Total	45	100%



A B

Fig.-8: *Pre op (A) and Post op (B) picture of visual field of right eye showing significant improvement. Following endoscopic endonasal approach.*



A B

Fig.-9: *Pre op (A) and Post op (B) picture of of visual field of left eye showing significant improvement. Following endoscopic endonasal approach*

Table-V

Shows distribution of extent of tumour removal.(N-45)

Extent of removal	Number of Cases	Percent
Total	35	77.77%
Sub total	10	22.22%

Postoperative Mortality and Morbidity: There were 4.5% cases of C.S.F. leak and 2 cases had severe pneumocephalus. 3 cases had meningitis and 2 patient developed post meningitis hydrocephalus. 1 patient developed subarachnoid haemorrhage (Table-VII).There were 6.6% mortality. Mortality were from electrolyte imbalance and from improper management of infection and hydrocephalus

Table-VII

Shows distribution of morbidity.(N-45)

Complications	No. of Cases	Percent
Permanent D. I.	2	4.44%
Temporary D. I.	17	37.77%
Meningitis	3	6.66%
CSF Leak	2	4.44%
Pneumocephalus	2	4.44%
Hydrocephalus	2	4.44%
S.A.H	1	2.22%

Discussion:

Endoscopic management of pituitary adenoma offers, not only the advantage of improved visualization, but also magnification, and a panoramic perspective of the important relationships of the sella turcica. The disadvantages of endoscopic pituitary surgery when compared to microscopic surgery are that endoscopic images are two dimensional monitor generated. The clearness and sharpness of the endoscopic images are little reduced then microscopic images. Endoscopic video-images are still inferior to those of direct

microscopic visualization. Digitally enhanced cameras have improved the picture quality to some degree. High-definition cameras and monitors will further improve the quality of endoscopic views. Another disadvantage is the learning curve for neurosurgeons who are already well trained in conventional microscopic surgery[7]. Postoperative CSF leakage has been a major potential complication in transsphenoidal surgery[8]. Occasionally it needs lumbar drainage, fluid therapy for its management. It may develop in early or late postoperative period. Meningitis, Hydrocephalous, Pneumocephalous are a sequle of C.S.F leak. Arterial bleeding, Venous bleeding, Subarachnoid haemorrhage are other potential complications[8]. Regarding endocrine problem early diabetes incipidious is a common problem which was managed by injection pitresin or minirin (Desmopressin) nasal spray. Hypocortisolism, Hypothyroidism, Hypogonadism were common endocrine abnormality which were usefully managed by hormone replacement therapy. In a study of Tabaee etal 2009, endoscopic pituitary surgery done on 821 patients where the overall mortality were 0.24%, permanent diabetic insipidus 1% and C.S.F. leak were 2%[9]. In our study, we are having high rate of mortality, diabetic insipidus and C.S.F. leak due to learning curve and improper post operative management.

Conclusion:

Now a day's endoscopic pituitary surgery remains the main line of treatment for pituitary adenoma. The panoramic exposure, magnification and flexibility of the endoscope combined with the absence of skin incisions, brain retraction and cranial nerve dissection, gives better outcome in endoscopic pituitary surgery. Endoscopic pituitary surgery now not a fashion but an ongoing demand from the patient side for its greater advantages, maximum tumour removal, less complications, less hospital stay and no scar.

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CASE REPORTS

Iatrogenic Entrapement Neuropathy of Ilioinguinal and Genitofemoral Nerve – A Rare case Report

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Abstract:

Entrapement neuropathy can occur in different nerves. It causes special features of according to the nerve involvement. Iatrogenic entrapement neuropathy of ilioinguinal and genitofemoral nerve can occur after herniotomy and herniorrhaphy and patients present with severe pain at the inguinal region.

A 18 years young male patients had been admitted in Private Hospital, Dhaka with history of bilateral indirect inguinal hernia. Bilateral herniotomy and herniorraphy was done two years back by an urologist. Subsequently he developed atrophy of testes and decreased libido. Postoperative period he developed severe burning pain at the left inguinal region and upper part of the left scrotum. We gave local anesthesia at left inguinal region and diagnosed the case of entrapement neuropathy of ilioinguinal and genitofemoral nerve. He was clinically diagnosed as entrapement neuropathy of iloinguinal & genitofemoral nerve. Pain was not relieved by analgesic. Neurolysis of left ilioinguinal and neurectomy of genitofemoral nerve was done. Subsequently this patient relieved from pain. This was a very rare case report with proper diagnosis and treatment can relief the patients.

Key word: *Entrapment neuropathy, ilioinguinal, genitofemoral, neurectomy, neurolysis*

Introduction:

The iliohypogastric nerve is rarely injured in isolation. The most common causes of injury are surgical procedures. These include transverse lower abdominal incisions, as in hysterectomies, or injuries from procedures such as inguinal herniorrhaphy and appendectomies. The injuries mainly occur if the incision extends beyond the lateral margin of the inferior rectus abdominis fibers. The damage can result from direct surgical trauma, such as passing a suture around the nerve and incorporating it into the fascial repair, or postoperative entrapment in scar tissue or neuroma formation. Sports injuries, such as trauma or muscle tears of the lower abdominal muscles, may also result in injury to the nerve. Injury may also occur during pregnancy, owing to the rapidly expanding abdomen in the third trimester. This is called the idiopathic iliohypogastric syndrome and is rare[1].

The ilioinguinal nerve arises from the fusion of T12 and L1 nerve roots and emerges from the lateral border of the psoas muscle; it traverses the anterior abdominal wall to the iliac crest just inferior to the hypogastric nerve. Adjacent to the anterior margin of the iliac crest, the nerve pierces the transversus abdominis and internal oblique muscles (providing neural branches to these) and sending neural branches to the iliohypogastric nerve. The nerve then supplies sensory branches to supply the pubic symphysis, the superior and medial aspect of the femoral triangle, and either the root of the penis and anterior scrotum in the male or the mons pubis and labia majora in the female[2].

Symptoms may include hyperesthesia or hypoesthesia of the skin along the inguinal ligament. The sensation may radiate to the lower abdomen. Pain may be localized to the medial groin, the labia majora or scrotum, and the inner thigh. The characteristics of the pain may vary considerably. Patients may be able to associate their pain clearly with a traumatic event or with the surgical procedure[2].

Pain and tenderness may be present with application of pressure where the nerve exits the inguinal canal in up to 75% of patients. Sensory impairment is common in the above-noted distribution of the nerve supply. Symptoms usually increase with hip extension (patients walk with the trunk in a forward-flexed posture). Pain may also be reproduced with palpation medial to the anterosuperior iliac spine (ASIS)[3].

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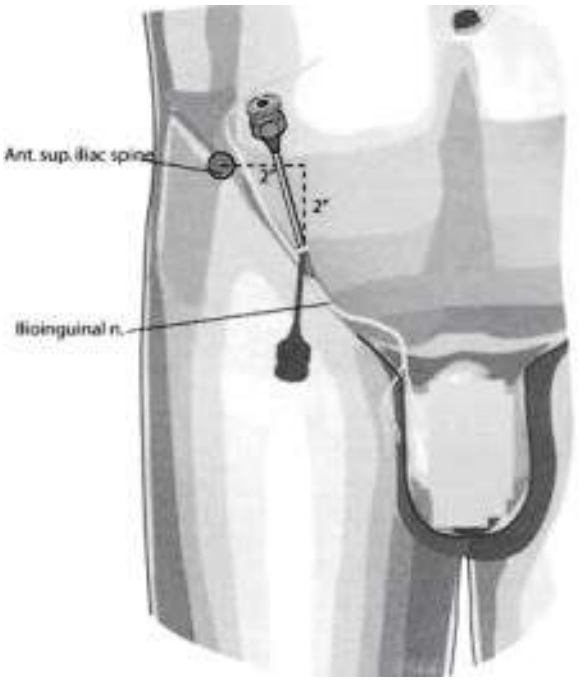

Fig.-1: *Ilioinguinal nerve*

The diagnosis can be made on the basis of local infiltration of anesthetic with or without steroid, which should result in relief within 10 minutes. Unfortunately, no electrodiagnostic techniques are readily available to test this nerve. Abdominal needle electromyography may be helpful in determining the severity of nerve injury, but electromyography is not sensitive or specific[4] (Fig.-1).

Treatment includes local injection of an anesthetic, physical therapy, or oral medications. Types of medications may include antiseizure medications, such as gabapentin (Neurontin), carbamazepine (Tegretol), or lamotrigine (Lamictal), as well as NSAIDs, tricyclic antidepressant medications (amitriptyline [Elavil], doxepin), capsaicin cream, topical lidocaine (Lidoderm patches), or tramadol (Ultram). Ice or possibly a TENS unit may be used with physical therapy. When conservative measures are not successful, surgical excision may result in relief of pain with few potential complications[5].

The genitofemoral nerve or its branches (genital or femoral branches) can be entrapped throughout its course. Nerve injury occurs most commonly as a complication of lower abdominal surgeries[2].

The genitofemoral nerve arises from the L1 and L2 ventral primary rami, which fuse in the psoas muscle. The nerve then pierces the anterior surface of the psoas major muscle at the level of L3-4 and descends on the fascial surface of the psoas major muscle past the ureter. It then splits into the genital and femoral branches near the inguinal ligament[2] (Fig.-2).

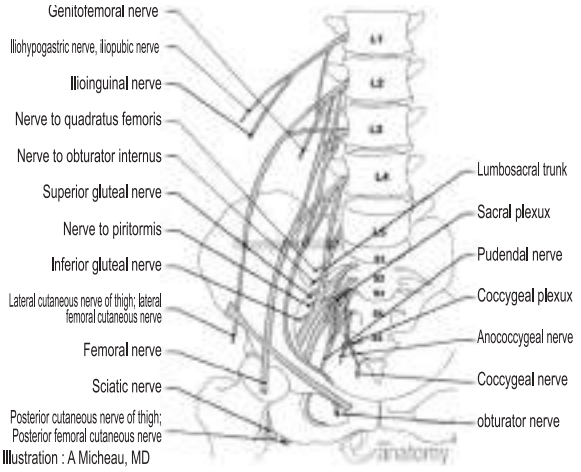

Fig.-2: *Ilioinguinal & genitofemoral nerve*

The genital branch continues along the psoas major to the deep inguinal ring and enters the inguinal canal. It supplies the cremaster muscle, spermatic cord, scrotum, and adjacent thigh in males. In females, it travels with the round ligament of the uterus and provides cutaneous sensation to the labia majora and adjacent thigh. The femoral branch lies lateral to the genital on the psoas major and travels lateral to the femoral artery and posterior to the inguinal ligament to enter the proximal thigh. There, it pierces the sartorius muscle distal to the inguinal ligament and supplies the proximal portion of the thigh about the femoral triangle just lateral to the skin that is innervated by the ilioinguinal nerve[2].

Nerve injury may result from hernia repair, appendectomy, biopsies, and cesarean delivery. Injury may also result from intrapelvic trauma to the posterior abdominal wall, retroperitoneal hematoma, pregnancy, or trauma to the inguinal ligament. Fortunately, injury to this nerve is rare, even with open herniorrhaphy[2].

A prospective study was performed to evaluate the genitofemoral nerve electrophysiologically in children with inguinal hernia repair. Latency of the genitofemoral nerve was found to be prolonged after inguinal hernia repair possibly because of surgical-related injury[2].

Injury to the femoral branch causes hypoesthesia over the anterior thigh below the inguinal ligament, which is how it is distinguished from the iliohypogastric and

ilioinguinal nerve. Groin pain is a common presentation of neuralgia from nerve injury or entrapment. The pain may be worse with internal or external rotation of the hip, prolonged walking, or even with light touch. Differential diagnoses include injury to the ilioinguinal and genitofemoral nerves as well as L1-2 radiculopathies. Some anatomic overlap may exist with the supply of the ilioinguinal and genitofemoral nerves, which makes the diagnosis somewhat difficult to establish.

Unfortunately, no reliable electrodiagnostic test exists that can be used for diagnosis of injury to this nerve. Oh has discussed a side-to-side sensory comparison study, but this test is technically difficult to perform[3]. Diagnosis typically is made using anesthetic nerve blocks. Injection of the ilioinguinal and iliohypogastric nerves anteriorly should leave the pain or abnormal sensation unchanged. A block of the L1 and L2 roots should then result in relief. This should help to determine the diagnosis and to prevent unnecessary surgical exploration of an uninjured nerve.

The above-mentioned blocks are diagnostic and therapeutic. Avoidance of aggravating activities should be emphasized. Treatment may also consist of antiseizure medications, such as gabapentin (Neurontin), carbamazepine (Tegretol), or lamotrigine (Lamictal), as well as tricyclic antidepressant medications (amitriptyline [Elavil], doxepin). Other medications include capsaicin cream, topical lidocaine (Lidoderm patches), NSAIDs, or, possibly, tramadol (Ultram). A trial with a TENS unit may also be beneficial.

If conservative treatment fails, surgical excision of the nerve is the treatment of choice. Some authors describe a transabdominal approach to the nerve (Magee and Lyon) with satisfactory results[4,5]. The complications of this procedure include hypoesthesia of the scrotum or labium majus and of the skin over the femoral triangle, as well as loss of the cremasteric reflex. This usually will not result in notable morbidity. According to Harms and colleagues, an extraperitoneal approach should result in fewer operative complications[6].

The term *entrapment neuropa*thies refers to isolated peripheral nerve injuries occurring at specific locations where a nerve is mechanically constricted in a fibrous or fibro-osseous tunnel or deformed by a fibrous band. In some instances the nerve is injured by chronic direct compression, and in other instances angulation or stretching forces cause mechanical damage to the nerve. Common examples of nerve compression in a fibro-osseous tunnel are the carpal tunnel syndrome and ulnar neuropathy at the cubital tunnel. Angulation and stretch injury are important mechanisms of nerve injury for ulnar neuropathies associated with gross deformity of the elbow joint ("tardy ulnar palsy") and neurogenic thoracic outlet syndrome. Recurrent compression of nerves by external forces may also cause focal nerve injuries such as ulnar neuropathy at the elbow and deep branch lesions of the ulnar nerve in the hand. Although these latter neuropathies do not satisfy the strict definition of "entrapment neuropathies", they are often considered in a discussion of the topic. The pathological features of all of these isolated neuropathies include a varying combination of focal demyelination and wallerian axonal degeneration[7,8].

Case report

A 18 years old young patients had been suffering bilateral indirect inguinal hernia for 4 years. Bilateral herniotomy & herniorraphy were done 2 years back by an urologist. After surgery he developed severe burning pain at left inguinal region and left upper part of scrotum. Pain was not relieved by any analgesic. He could not sleep for pain. His testes became atrophy, bilaterally & decreased libido. With this complaints he admitted at a private hospital of Dhaka. A medical board was done with urologist & neurosurgeon. Medical board took decision if the patient pain relieved by local anaesthesia, the clinical diagnosis was entrapment neuropathy of left sided ilioinguinal & genitorfemoral nerve. The patient would cure by neurolysis, neurectomy of ilioinguinal & genitofemoral nerve. Pain was subsided by pushing injection lignocaine and adrenaline. Under general anaesthesia re-exploration of previous wound was done. Left sided ilioinguinal nerve and it two branches were found adhesion with surrounding structure. Neurolysis of ilioinguinal nerve and its branches were done. Left sided genitofemoral nerve also found entrapped. Neurectomy of genitofemoral nerve was done (Fig.-3).

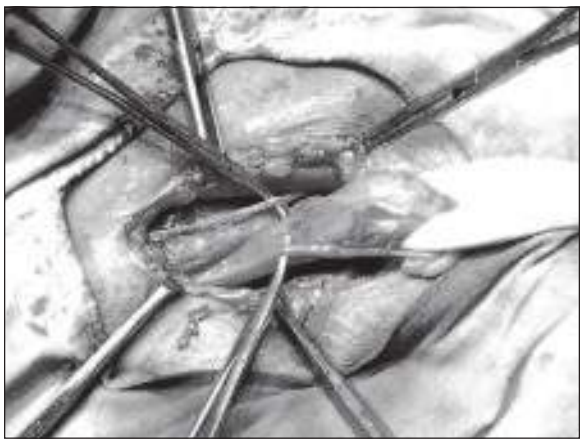

Fig. 3: *Entrapped left ilioinguinal nerve*

Proper hemostasis was done and wound was closed in layers. Patient's pain was relieved after operation. Follow up after one months patients had no symptom. But his testes remain same size and did not increase sexual function. It was a rare case report. Proper diagnosis and treatment and can relief patients symptoms.

Discussion:

The ilioinguinal nerve entrapment syndrome has received little attention in the literature and seems to be little known[9,10,11]. The incidence cannot be determined from our study but seems not to be rare. Mummenthaler et al. reported on seven patients seen during a period of several months[11]. Another report described 23 cases following common lower abdominal surgical procedures[12]. The preponderance of right fossa localization (75% ofour cases) might be explained by referral bias caused by the suspicion of appendicitis in case of right fossa pain. Chronic left fossa pain will easily be ascribed to irritable bowel. Analysis of the predisposing and aggravating factors does not explain the enormous female preponderance in this series. The weaker abdominal muscles and more downward angulation of the pelvis in females, increasing the distance the nerve has to travel, might predispose to nerve damage[10]. However, surgery of the lower abdomen, mainly appendicectomy, hernia repair and hysterectomy, predisposes to damage of the ilioinguinal nerve[11].

Entrapment neuropathy following surgical procedures frequently has to be treated by neurectomy[11,12]. The differential diagnosis of ilioinguinal nerve entrapment includes entrapment of the neighbouring iliohypogastric(Ll-Th12) and genitofemoral(Ll-L2) nerve and the rectus abdominis muscle syndrome[12,13,14,15].

In one study the incidence of nerve entrapment in laparoscopic heniorrhaphy was reported to be as high as 4.2%, while in open herniorrhaphy it was only 1.8%. The genitofemoral nerve was the most commonly affected nerve in laparoscopic herniorrhaphy(2%), then comes the lateral femoral cutaneous nerve(1.1%) and the ilioinguinal nerve(1.1%). However in a review of more than 14,000 laparoscopic inguinal hernia repairs, the lateral femoral cutaneous nerve was the most commonly affected nerve in 58% of cases of nerve injury, then the femoral branch of the genitofemoral nerve in 31% of cases[16].

This is a very rare case report, very few author described about this disease previously in literature or text book. Though indirect inguinal hernia is a common disease of young people. During surgery should carefully procedure for avoid undue complication. Entrapment neuropathic pain is a severe pain which does not relieved by ordinary analgesia. Proper diagnosis and release the nerve only relief the patients. Our patient had both vascular insult bilaterally as well as neurologic involvement. Iatrogenic symptoms made the patients more interesting.

Conclusion:

Iatrogenic entrapment of two nerve made the case more interesting. Very few author described about this insult previously. So proper diagnosis and treatment relief the many lives.

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Unusual Case of Angular Epidermoid Association with Facial Cleft Defect: A Rare Case Report

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Abstract

Facial cleft defect is one of the important cranio-facial anomaly and hypertelorism is associated with few of the facial cleft defects. Epidermoid is a benign tumor that may arise when retained ectodermal implants are trapped by two fusing ectodermal surfaces. Common sites are calvaria, suprasellar region, sylvian fissure, Cerebellar Pontine Angle (CPA) region. We discuss this case because there is rare association between epidermoid, hypertelorism and facial cleft defect in a two years and six month years old boy who was operated in All India Medical Institute, Delhi, India.

Keywords: *Epidermoid cyst, Hypertelorism, Facial cleft, One-stage correction, Medial orbital advancement*

Introduction

Epidermoid contain stratified squamous epithelium, keratin cellular debris and cholesterol. Usually arise from ectoderm trapped within or displaced into the CNS. It has predilection for CP angle, fourth ventricle, suprasellar region and spinal cord. It also known as cholesteotoma, may produce Mollaret's meningitis[1].

Kitlowski (1959) presented a case in which a median cleft of the primary palate, bifid nose, hypertelorism, and a cleft palate were presumably due to a congenital dermoid cyst of the nose[2].

Encephaloceles are usually associated with facial cleft defect and hypertelorism. In this case there is rare association of epidermoid, hypertelorism and absence of ala of nose due to facial cleft defect.

A Case Report

A two years and six month old male patient was admitted in the hospital with the complaints of deformity of nose since birth and progressively increasing swelling at right frontonasal region for last 2 years. There was no history of CSF rhinorrhoea, seizure, unconsciousness, abnormal increase in head size or delayed developmental milestones. Birth history was prolong due to obstructed labour, which lead to cesarian section. He was the first child of his parent. Family history of parents was normal. Patient underwent surgery at some other hospital 1 year back for correction of nasal deformity but only partial correction was achieved.

On examination

All neurological parameters were normal, no other systemic abnormalities

Local examination

Patient had swelling at the right fronto-orbito nasal region (just above medial canthus) which was almost spherical in shape, soft, non-tender, non-fluctuating and 2.5 x 2.5 cm in diameter. Trans- illumination test, cough impulse was negative. The swelling was non-pulsatile. OFC (occipito frontal circumference) was 52 cm and antero and posterior frontanele were closed. Gross nasal defect was found at the right side in form of absence of ala and lateral wall of nose on right side. There was hypertelorism (Fig. 1). Intercanthal distance was 5 cm.

Radiological investigation

MRI of head shows hyper intense mass at right fronto-orbito-nasal region. CT scan of head shows angular epidermoid (Fig. 2).

Per operative diagnosis

Angular epidermoid with medial facial cleft defect associated with hypertelorism (Fig. 1).

Surgical procedure

A bicoronal skin incision was made to gain wide exposure. Pericranial graft was reflected to expose frontal bone. Both supraorbital rims, and the nasal bridge were exposed. Bifrontal craniotomy and bilateral supraorbital orbitotomies were made. The

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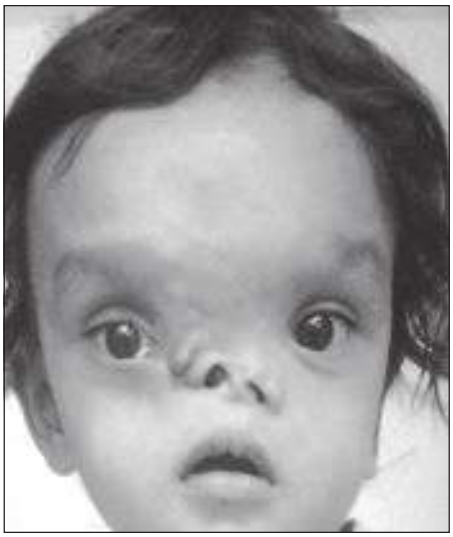

Fig.-1: *Preoperative photograph showing nasal deformity with hypertelorism and angular epidermoid*

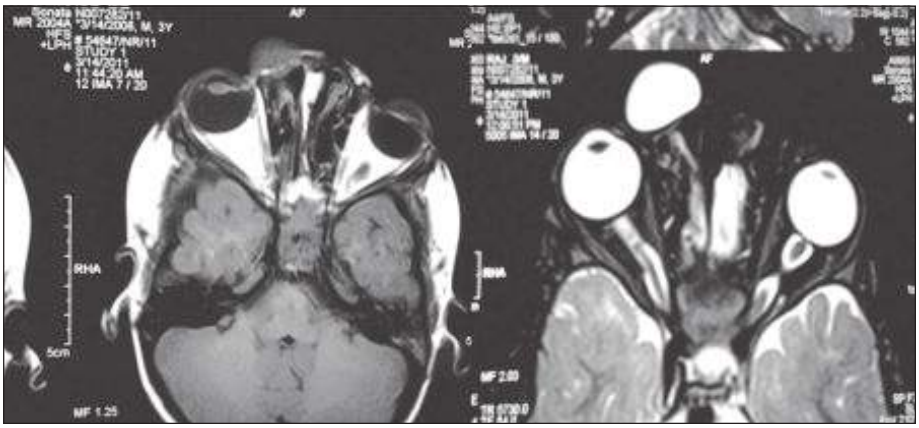

Fig.-2: *MRI of head showing angular epidermoid*

frontal bone flap was made separately and both supraorbital ridges with part of frontal bones were taken as a single piece separately. The intercanthal distance was about 5 cm. About 2.5 cm of intercanathal bone was removed which was used as the bone graft over the mid of supraciliary margine after bilateral hemiorbital advancement to correct the hypertelorism. All bones were fixed with mini plate (Fig. 3). Excision of epidermoid was done by separate skin incision.

Fig.-3: *Per operative photograph showing correction of hypertelorism and fixation of bone with mini plates*

Discussion:

Epidermoid usually arise from ectoderm trapped within or displaced into the CNS. It has predilection for: CP angle, 4th ventricle, suprasellar region, spinal cord. It also known as cholesteatoma (not to be confused with cholesterol granuloma). Epidermoid grows at linear rate and may produce Mollaret's (aseptic) meningitis. Mollaret's meningitis is a rare variant of aseptic meningitis which includes the finding of large cells in the CSF that resemble endothelial cells[3].

Basal encephalocele may be associated with other craniofacial deformities, including: hypertelorism, cleft lip, bifid nose, optic-nerve dysplasia, coloboma and microphthalmia, hypothalamic pituitary dysfunction[4].

Recently, Mahapatra had reported advancement of the medial half of the orbit medially and placing a supraorbital bone graft on either side. By the above procedure the operating time can be reduced on an average by 90-100 minutes[5,6].

In our case there was rare association between hypertelorism, nasal defect and angular epidermoid. There is usual association with fonto basal encephalocele with hypertelorism. We removed 2.5 cm bone in the middle to correct hypertelorism and advanced the orbit medially. The orbital ridges bony gap was covered by bone graft and bones were fixed by titanium mini plate. Thus the hypertelorism was corrected.

The aim of surgical treatment is to correct hypertelorism with appropriate osteotomies and reconstruction and excision of epidermoid through separate incision.

Conclusion:

Angular eopidermoid associated with facial cleft defect is a rare case. Though epidermoid is a benign tumour; the association of epidermoid with hypertelorism and facial cleft defect is a rare association. Yet there are few cases reported like this. Though this is a complicated case and treatment modality which was done successfully. This report will bring attention to world community about rare association of angular epidermoid with hypertelorism and facial cleft defect.

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