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## CONTENTS



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# Bangladesh Journal of Neuroscience



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## ORIGINAL ARTICLES

# Electrocardiographic Findings and Mortality in Ischemic Stroke

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**Abstract:**

*This study was carried out in the Department of Neurology, Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka from July 2006 to October 2008. The objective of this study was to compare mortality among ischemic stroke patients having normal and abnormal electro-cardiogram (ECG).*

*A total of 72 patients with acute ischemic stroke were selected by purposive sampling method and diagnosed by history, clinical findings and was confirmed by CT scan of head. The clinical details, investigations of the respondents were reviewed. Data were recorded in predesigned data collection sheet. Majority of the subjects were in 7th decade 12(33.3%) and 6th decade 9(25%) with the male to female ratio was 1.25:1. Among the patients with abnormal electrocardiographic findings 19.4% patients each had myocardial ischemia, atrial fibrillation, and myocardial infarction; 16.7% had non-specific ST changes, 13.9% had ventricular hypertrophy, 11.1% patients each had conduction block, and ventricular arrhythmias and 2.8% had atrial dilatation.*

*In this study 14(38.9%) and 5 (13.9%) patients expired having abnormal and normal ECG findings respectively at the end of 6 months from admission.*

**Introduction:**

Stroke is the third commonest cause of death after ischaemic heart disease and cancer in developed countries and is responsible for a large proportion of physical disability[1].

It is also the commonest cause of morbidity and mortality among adult population, one year case fatality being 42 percent[2].

The main types of stroke and their relative oc-currences are: Ischaemic stroke - 85% and Haemorrhagic stroke -15%[3].

Coronary heart disease and ischaemic stroke share the same risk factors and may co-exist in the same patient and in most of the patients with ischaemic stroke, the mortality may be related to the underlying coronary heart disease[4].

ECG changes are common in patients with ischaemic stroke. Studies have shown that the frontal lobe, insular cortex and

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amygdale play an important role in the regulation of heart via the sympathetic and parasympathetic systems and cardiac involvement is more common in patients with cerebral lesion involving these areas[5,6].

In financial terms, stroke represents 6% of hospital running costs and 4.6% of all National Health Service costs.

About 40-50% of beds are occupied by stroke patients in neurology ward which is reported in a developing country like ours[8,9,10].

Ischaemic stroke, which is perhaps the commonest subtype of stroke, is associated with ECG changes; some of these changes have been thought to be due either to the stroke itself or pre-existing heart disease." Controversies exits in the exact cause of mortality in ischaemic stroke. Most common cause of death in patients with stroke is cardiac. Cardiac states and also ECG changes in patients with acute ischaemic stroke are associated with higher mortality[12].

Because ECG is rapid, noninvasive and low cost, successful detection of cardiac complications early in the course of acute ischaemic stroke could have an impact on clinical management.

In Bangladesh, the exact situation regarding mortality with electrocardiographic changes in ischaemic stroke patients is not known. Therefore, this study was performed among Bangladeshi patients with ECG changes in ischaemic stroke and their mortality to delineate the general demographic data as a prognostic factor.

**Aims and Objectives:**

Many studies are available from other countries, especially from Japan, India but there has been no such study from Bangladesh. The aims of the study was to see the mortality difference among ischemic stroke patients having normal and abnormal ECG findings in Bangladeshi population. Chi-square test and unpaired students 't' test has been done in this study.

**Materials and Methods:**

This was an observational study carried out in the Department of Neurology, Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka. Study subjects were collected from admitted patients in neurology ward in Bangabandhu Sheikh Mujib Medical University followed upto discharge in the neurology ward and subsequent follow up were done in stroke clinic of neurology OPD from July 2006 to October 2008. A total of 72 patients with acute ischaemic stroke were selected by purposive sampling method and diagnosed by history, clinical findings and was confirmed by CT scan of head. Of them 36 patients with abnormal electrocardiographic findings with ischaemic stroke were included in Group-A and the same number (36) of age and sex matched ischaemic stroke patients with normal electrocardiographic findings were included in Group- B. Both Group- A and Group- B patients were selected from among the patients admitted in neurology ward, Department of Neurology, BSMMU, during the study period.

**Results:**

A total number of 36 ischaemic stroke patients with abnormal electrocardiographic findings (Group- A), and 36 age and sex matched respondents with ischaemic stroke with normal electrocardiographic findings (Group- B) were taken in this study(Table-I & II).

**Table-I**

*Age of the study subjects*

| Age (years) | Group- A (n=36) | | Group- B (n=36) | | P |
|---|---|---|---|---|---|
| | No. | % | No. | % | value |
| 40-49 | 5 | 13.9 | 5 | 13.9 | |
| 50-59 | 9 | 25.0 | 9 | 25.0 | |
| 60-69 | 12 | 33.3 | 12 | 33.3 | >0.50ns |
| 70-79 | 8 | 22.2 | 8 | 22.2 | |
| ≥ 80 | 2 | 5.6 | 2 | 5.6 | |
| Total | 36 | 100 | 36 | 100 | |
| Mean±SD | 62.39±10.64 | | 60.89±11.12 | | |
| Range | 42-84 | | 42-85 | | $>0.50^{ns}$ |

ns = Not significant

Group- A : Ischaemic stroke patients with abnormal ECG findings.

Group- B : Ischaemic stroke patients with normal ECG findings.

**Table-II**

*Sex distribution of study subjects*

| Sex | Group- A (n=36) | | Group- B (n=36) | | P |
|---|---|---|---|---|---|
| | No. | % | No. | % | value |
| Male | 20 | 55.6 | 20 | 55.6 | |
| | | | | | >0.50ns |
| Female | 16 | 44.4 | 16 | 44.4 | |
| Total | 36 | 100 | 36 | 100 | |

ns = Not significant

**Table-III**

*Distribution of diabetes mellitus among the study subjects.*

| Diabetes mellitus | Group- A (n=36) | | Group- B (n=36) | | P |
|---|---|---|---|---|---|
| | No. | % | No. | % | value |
| Present | 12 | 33.3 | 9 | 25.0 | |
| | | | | | $>0.10^{ns}$ |
| Absent | 24 | 66.7 | 27 | 75.0 | |
| Total | 36 | 100 | 36 | 100 | |

Chi-square test
ns = Not significant

Table – III shows that diabetes mellitus was present among 12 (33.3%) patients in group A and 9 (25.0%) patients in group B. Diabetes was absent in 24 (66.7%) patients in group A and 27 (75.0%) patients in group B and P was > 0.10 which was not significant.

**Table-IV**

*Distribution of dyslipidaemia among the study subjects.*

| Dyslipidaemia | Group-A (n=36) | | Group-B (n=36) | | P |
|---|---|---|---|---|---|
| | No. | % | No. | % | value |
| Present | 13 | 36.1 | 5 | 13.9 | <0.05* |
| Absent | 23 | 63.9 | 31 | 86.1 | |
| Total | 36 | 100 | 36 | 100 | |

* = Significant

Table-IV shows the distribution of dyslipidemia in patients among the group A 13(36%) and in group B patients 5(14%) who were suffering from ischaemic stroke. (p value was <0.05; which is significant).

**Table-V**

*Mortality at the end of 1 month.*

| Parameters | Group- A (n=36) | | Group- B (n=36) | | P |
|---|---|---|---|---|---|
| | No. | % | No. | (% | value |
| Alive | 25 | 69.4 | 33 | 91.7 | <0.05* |
| Expired | 11 | 30.6 | 3 | 8.3 | |
| Total | 36 | 100 | 36 | 100 | |

* = Significant

The mortality at the end of one month (Table-V) and mortality at the end of 6 month (Table VI) are shown below, which revealed p value <0.05 & <0.05 respectively and was significant.

**Table-VI**

*Total mortality at the end of 6 months from admission.*

| Parameters | Group-A (n=36). | | Group- B (n=36) | | P |
|---|---|---|---|---|---|
| | No | % | No | % | value |
| Alive | 22 | 61.1 | 31 | 86.1 | <0.05* |
| Expired | 14 | 38.9 | 5 | 13.9 | |
| Total | 36 | 100 | 36 | 100 | |

Chi-square test

* = Significant

The mortality at the end of one month (Table-V) and mortality at the end of 6 month (Table VI) are shown below, which revealed p value <0.05 & <0.05 respectively and was significant.

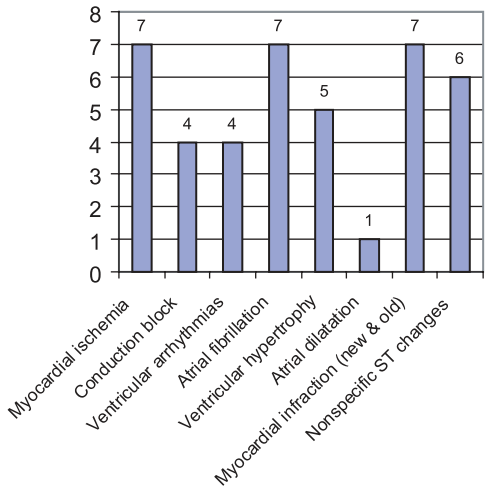


**Fig.-1:** *Distribution of abnormal ECG findings among study subjects.*

Fig.-1 shows distribution of abnormal ECG findings among ischaemic stroke patients, where 7(17.4%) patients each had myocardial ischaemia, myocardial infraction and atrial fibrillation; 6(16.7%) patients had non-specific ST changes, 5(13.9%) patients had ventricular hypertrophy, 4(11.1%) patients each had ventricular arrhythmias and 1(2.8%) patient had atrial dilatation.

**Discussion:**

This was a hospital based observational study and was carried out to see the association of electrocardiographic findings with mortality as a prognostic factor for ischaemic stroke. The study subjects were selected from the admitted patients of neurology ward, Department of Neurology, Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka. A total of 72 patients had been studied during the study period.

In this study, majority of the subjects were in 7th decade 12(33.3%) and 6th decade 9(25%). Next common age group were 8$^{th}$ decade 8(22.2%) and 5$^{th}$ decade, 5(13.9%). In a previous study among 51 stroke patients majority of them were in the age group of 50-69 years[13]. In another study among 100 stroke patients, the age range was 18-84 years and most of his cases were between 50-70 years[14]. So, this study is consistant with other studies.

In this study the mean (±SD) age of the patients in Group- A was 62.39±10.64 years and in Group- B was 60.89±11.12 years. Similar past studies had comparable age statistics of the patients[15].

In this study, the male to female ratio was 1.25:1. Male involvement was 12.5% higher than that of female. In a previous study male to female ratio was 1.2:1 which is similar to this study[12].

In this study among diabetic patients 12 (33.3%) cases presented with abnormal ECG and 9(25%) cases with normal ECG findings. In a study carried out in Dutch community on stroke patients where 29% patients were diabetic[16]. In another study among ischaemic stroke patients 30% were diabetic[17].

Independent of age, coronary heart diseases (i.e. angina pectoris or myocardial infarction) are clearly associated with ischaemic stroke. The evidences are available in postmortem[18,19], case control[20] and cohort studies[21].

In a previous study it was found that atrial fibrillation was present in 19.5% of cases[12]. In this study, 19.4% of patients with abnormal ECG had atrial fibrillation and 11.1% had ventricular arrhythmias. Therefore the present study is consistent with the findings of previous studies.

In this study, myocardial ischaemia was one of the common (19.4%) ECG findings. In some previous studies it was also found that myocardial ischaemia was a common ECG finding in stroke patients[12,22,23]. Therefore this study is consistent with many of the previous studies.

In this study, 11(30.6%) and 14(38.9%) patients expired with abnormal electrocardiographic findings in Group- A and 3 (8.3%) and 5(13.9%) patients expired with normal electrocardiographic findings in Group-B at the end of 1 and 6 months from admission respectively.

Cardiac abnormality is the most common cause of death in patients with stroke[4]. In a previous study it was found that both mortality at 30 days and mortality at 6 months were significantly high in patients with ECG changes. They showed mortality after 6 months with pathological ECG was 38.9%, where as it was 15.2% with normal ECG and the overall mortality after 1 month was 20% and after 6 months was 29.8%[12]. In another study they found mortality rate following cerebral infarcts in general at the end of 1 month was 19% and at the end of 1 year was 23%[24]. So, the present study is consistent with the findings of previous studies.

Considering all the above observations, it is established that this study showed a close relationship between abnormal electrocardiographic findings and mortality among ischaemic stroke patients.

**Conclusion**

From the statistical analysis of the results obtained in this study, it has been seen that there is presence of significant differences of mortality among abnormal electrocardiographic findings and normal electrocardiographic findings in Bangladeshi ischaemic stroke patients. So, it can be concluded that electro-cardiographic abnormality in ischaemic stroke patients is a predictor of mortality.

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# Patients with Chiari Type I Malformation have Smaller Posterior Fossa than Normal Subjects- A Cross-Sectional Comparative Study

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**Abstract**

***Background:*** *Chiari malformation is characterized by a downward herniation of the caudal part of the cerebellum and/or medulla oblongata into the spinal canal. Now-a-days, a large number of new cases of Chiari I malformation (CMI) and Chiari I-related syringomyelia are being diagnosed all over the world.* ***Hypothesis:*** *Patients with Chiari type I malformation have smaller posterior cranial fossa (PCF) than normal subjects.* ***Objective:*** *To estimate the dimensions of the posterior cranial fossa in Chiari type I malformation and to compare them with non-Chiari malformation patients.* ***Methods:*** *22 patients, whose preoperative radiological studies were available, served as subjects in this study. These patients were selected from Neurosurgery Departments of Bangabandhu Sheikh Mujib Medical University (BSMMU) and Dhaka Medical College Hospital (DMCH). A series of 22 patients who had an MRI scan of the head done for reasons other than CMI were selected from the Departments of Neurosurgery and Neurology at BSMMU. These patients were presumed to reflect the normal population.Statistical analysis was done by SPSS.* ***Results:*** *The overcrowding of PCF was estimated by some bone measurements on MRI in this study. The results showed that except for the diameter of the foramen magnum all other measurements were smaller among the Chiari group as compared to the non-Chiari group. As a matter of fact, statistically speaking, all of these measurements were significantly smaller in the Chiari group. Interestingly, the size of the foramen magnum was significantly wider in the Chiari group than the non-chiari group.* ***Conclusion:*** *Our results support that, Chiari I patients have smaller posterior fossa. The study also provides additional evidence that Chiari I is actually a result of the lack of proper development of the back of the skull, which results in crowding and a downward displacement of the cerebellum.*

**Introduction:**

Chiari malformation is characterized by a downward herniation of the caudal part of the cerebellum and/or medulla oblongata into the spinal canal. This malformation has been divided into at least two distinct groups according to the degree of herniation: Type I represents herniation of the cerebellar tonsils and Type II displays herniation of the fourth ventricle and the

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medulla oblongata as well as the caudal part of the cerebellum[1]. However, from a clinical point of view, Chiari malformation can also be divided into adult and pediatric types[2]. Adult type is mostly considered as Chiari type I (CMI) malformation and usually presents after the second or third decade of life. The signs and/or symptoms are mainly resulting from the tightness of the posterior cranial fossa (PCF) and associated syringomyelia[3]. This type is often accompanied by bone anomalies in the base of the skull such as basilar invagination, but is less frequently associated with brain abnormalities other than herniation of the cerebellar tonsils. The pediatric type mainly belongs to Chiari Type II and is characterized by myeloschisis at birth and brainstem dysfunction in the early periods after birth [4].Typical features include a small posterior cranial fossa and various neural abnormalities such as brainstem deformities and hydrocephalus[5].

The impact of new technology on the diagnosis and management of these entities has been tremendous. We are currently diagnosing these abnormalities in a very different setting from a few years ago.

**Materials and methods:**

22 patients, whose preoperative radiological studies were available, served as subjects in this study. These patients were selected from Neurosurgery Departments of BSMMU and Dhaka Medical College Hospital (DMCH). A series of 22 patients who had an MRI scan of the head done for reasons other than CMI were selected from the Departments of Neurosurgery and Neurology at BSMMU. These patients were presumed to reflect the normal population.Statistical analysis was done by SPSS.

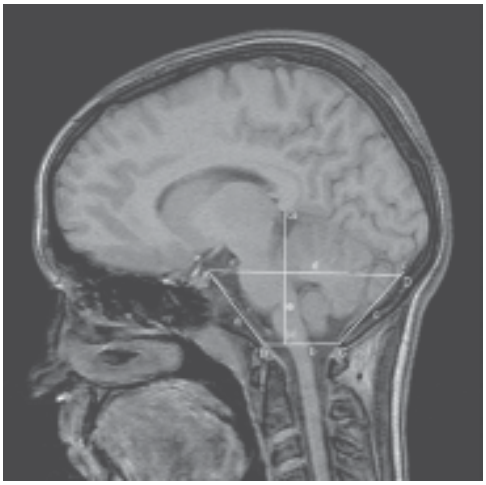

**Fig.-1:** *Demonstration of the measurements of PCF made on T1-weighted mid-sagittal MRI of healthy subjects. The length of the clivus (a) was measured along a line drawn from the top of the dorsum sella (A) to the basion (B). The anteroposterior diameter of the foramen magnum (b) was measured from the basion (B) to the opisthion (C). The length of the supraocciput (c) was measured along a line drawn from the opisthion (C) to the center of the internal occipital protuberance (D). The anteroposterior diameter of the PCF (d) was measured along a line parallel to the plane of the foramen magnum, from the top of the dorsum sella (A) to a point 1 cm above the internal occipital protuberance (F). The height of PCF (e) was measured with a line drawn from the inferior aspect of the splenium (G) perpendicular to the plane of the foramen magnum (H) (after Aydin et al. 2005).*

## Results

**Table-I**

*Comparison of age of the study subjects*

| Age (years) | Chiari (n=22) | non-Chiari (n=22) | P value |
|---|---|---|---|
| Mean±SD | 28.68±7.75 | 37.50±12.17 | 0.006** |
| Range | 19-44 | 20-57 | |

Unpaired Student's 't' test

Table-I summarizes the age distribution of the CMI group and non-Chiari group. The mean age of the CMI group was 28.68±7.75 years (range: 19-44 years) and non-Chiari group was 37.50±12.17 years (range: 20-57 years). There is statistically significant difference between the two groups regarding the age (P = .006).

**Table-II**

*Sex distribution of the study subjects*

| Sex | Chiari (n=22) | | non-Chiari (n=22) | | P value |
|---|---|---|---|---|---|
| | No. | % | No. | % | |
| Male | 18 | 81.8 | 16 | 72.7 | |
| | | | | | 0.472ns |
| Female | 4 | 18.2 | 6 | 27.3 | |
| M:F ratio | 4.5:1.0 | | 2.67:1.0 | | |

Chi square test, ns - not significant

Table II shows that the CMI group consisted of 18 men and 4 women. The non-Chiari group consisted of 16 men and 6 women. There was no significant difference in sex distribution between the two groups (P =0.47).

**Table-III**

*Duration of symptoms of CMI patients at presentation (n=22)*

| Duration (years) | Frequency | Percentage | Mean±SD (Range) |
|---|---|---|---|
| 0 2 | 13 | 59.1 | 1.01±0.57 (0.42 2.00) |
| >2 4 | 1 | 4.5 | 3.00 (3.00 3.00) |
| >4 6 | 1 | 4.5 | 5.00 (5.00 5.00) |
| >6 8 | 6 | 27.3 | 7.50±0.55 (7.00 8.00) |
| >8 10 | 1 | 4.5 | 9.00 (9.00 9.00) |
| Total | 22 | 100.0 | 3.42±3.20 (0.42 9.00) |

Table III shows the duration of symptoms at presentation i.e time elapsed between the appearance of first symptom and presentation of patients to our hospital. In this study, most of (59.1%) the patients appeared at the hospital within 2 years of the appearance of the first symptom. Out of 22 patients 6 (27.3%) presented within 6-8 years. The mean duration of symptoms before presentation was 3.42 years with SD ± 3.20 (Range: 0.42 9.00 years).

**Table-IV**

*Distribution of patients with CMI by clinical features (n-22)*

| Symptoms & signs | Frequency | Percentage |
|---|---|---|
| Headache | 11 | 50 |
| Upper limb weakness | 20 | 90.9 |
| Lower limb weakness/ gait difficulty | 13 | 59.1 |
| Sensory disturbances of upper limbs | 16 | 72.7 |
| Upper limbs muscular atrophy | 14 | 63.6 |
| Lower limbs spasticity | 13 | 59.1 |
| Lower cranial nerves palsy | 1 | 4.5 |

Table IV summarizes the mode of clinical presentation. The majority (90.9%) of patients presented with upper limb weakness followed by sensory disturbances of upper limbs 72.7%, upper limbs muscular atrophy 63.6 %, lower limbs weakness 59.1% and lower limbs spasticity 59.1%.

**Table-V**

*Distribution of patients with CMI by presence of syringomyelia*

| Syringomyelia | Frequency | Percentage |
|---|---|---|
| Present | 20 | 90.9 |
| Absent | 2 | 9.1 |
| Total | 22 | 100 |

Table V shows that syringomyelia is present in 90.9% of patients.

**Table-VI**

*Distribution of patients in the CMI group by presence of hydrocephalus*

| Hydrocephalus | Frequency | Percentage |
|---|---|---|
| Present | 2 | 9.1 |
| Absent | 20 | 90.9 |
| Total | 22 | 100 |

Table VI shows that 9.1% of the patients had hydrocephalus.

**Table-VII**

*Distribution of patients with CMI by compression of cisterna magna*

| Compression of cisterna magna | Frequency | Percentage |
|---|---|---|
| Present | 22 | 100 |
| Absent | 0 | 0 |
| Total | 22 | 100 |

From Table VII it appears that, all (100%) of the patients had compression of cisterna magna as an MRI finding.

**Table-VIII**

*Measurements of the posterior cranial fossa in CMI patients and non-Chiari patients*

| Measurements (mm) | CMI (n=22) | Non CMI (n=22) | P value |
|---|---|---|---|
| Supraocciput | | | |
| Mean±SD | 42.00±3.98 | 47.00±2.16 | 0.0001*** |
| Range | (33-51) | (43-51) | |
| Foramen magnum[a] | | | |
| Mean±SD | 32.00±3.04 | 25.00±2.16 | 0.0001*** |
| Range | (26-38) | (21-29) | |
| Clivus | | | |
| Mean±SD | 39.00±3.60 | 48.00±2.58 | 0.0001*** |
| Range | (31-47) | (43-53) | |
| Posterior fossa[b] | | | |
| Mean±SD | 60.00±5.29 | 75.00±2.16 | 0.0001*** |
| Range | (49-71) | (71-79) | |
| Height[c] | | | |
| Mean±SD | 125.00±7.84 | 141.00±3.24 | 0.0001*** |
| Range | (109-141) | (134-148) | |

Unpaired Student's 't' test, [a] Anteroposterior diameter of the foramen magnum. [b] Anteroposterior diameter of the posterior fossa. [c] Height of the posterior fossa.

Table VIII summarizes statistical results of the parameters evaluated in this study. For patients with CMI, the following statistically significant abnormalities were demonstrated: reduced mean length of the clivus (P = .0001), reduced mean anteroposterior diameter of the PCF (P = .0001), reduced mean length of the PCF (P = .0001), reduced mean height of the supraocciput ( P = .0001), and increased mean anteroposterior diameter of foramen magnum ( P = .0001).

**Discussion:**

The present study was conducted in the department of Neurosurgery, BSMMU, Dhaka during the period of October 2005 to November 2008.

In this study, we compared linear measurements of the posterior cranial fossa (PCF), as determined by MRI, in 22 adult patients with Chiari type I malformation (CMI) and 22 adult patients without CMI.

In our study where we included only adult patients of CMI, the mean age was 28.68±7.75 years (range, 19-44 years).

In a similar study showed[6] the mean age of the patients with CMI was 35.1±12.7 years (range, 25-65 years).

In this study, most of (59.1%) the patients attended at the hospital within 2 years of the appearance of the first symptom. Out of 22 patients 6 (27.3 %) presented within 6-8 years. The mean duration of symptoms before presentation was 3.42 years with SD ± 3.20 (Range : 0.42 9.00 years).

The overcrowding of PCF was estimated by some bone measurements on MRI in this study. Patients with CMI were found to have the following distinctive findings: reduced mean length of the clivus (P < .01), reduced mean length of the supraocciput (P< .01), reduced mean height of the PCF (P<.01), reduced mean anteroposterior diameter of the PCF (P < .01), and increased mean anteroposterior diameter of the foramen magnum (P < .01). The results showed that except for the diameter of the foramen magnum all other measurements were smaller among the Chiari group as compared to the non-Chiari group. As a matter of fact, statistically speaking, all of these measurements were significantly smaller in the Chiari group. Interestingly, the size of the foramen magnum was significantly wider in the Chiari group than the non-chiari group.Similar findings were also observed in a previous study[6].

Our study supports the earlier findings, that that people with Chiari malformation I have smaller posterior fossa. This study also provides additional evidence that Chiari malformation I is actually a result of the lack of proper development of the back of the skull, which results in crowding and a downward displacement of the cerebellum.

**Conclusion:**

Our results support that, Chiari malformation I patients have smaller posterior fossa. The study also provides additional evidence that Chiari I is actually a result of the lack of proper development of the back of the skull, which results in crowding and a downward displacement of the cerebellum.

**Limitation of the study:**

Sample size was small, so it was not possible to generalize the findings of the study to reference population.

Subjective variation of clinical assessment.

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# Spontaneous Supratentorial Intracerebral Haemorrhage: Relation between Level of Consciousness at Admission and Early Outcome of Surgery

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**Summary**

*This prospective study conducted in the department of neurosurgery, BSMMU, from November 2004 to May 2007. In this study we tried to findout relation of level of consciousness at admission and early surgical outcome for spontaneous supratentorial intracerebral haemorrhage (SSICH). Consecutively admitted patients of SSICH who under went surgery were included in the study. They had compatible history and CT scan evidence of SSICH .The goal of surgery was adequate decompression. Level of consciousness was measured at admission by Glasgow Coma Scale(GCS score). We measured outcome at discharge using Glasgow Outcome Scale (GOS). A total of 46 patients were included in the study based on selection criteria. Mean GCS score at presentation was 8.73 with a standard deviation of 2.36. Most of the patients (52.1%) had GCS<9. GCS score at presentation was 9-13 in 45.7% of patients. Only 2.2%had GCS >13. Mean Glasgow out come score (GOS) at discharge was 2.3 with standard deviation 1.29. 30.4% of patients were conscious but disabled (GOS-3), 19.6% of patients were discharged with moderate disability (disabled but independent, GOS-4), 2.2% of patients discharged at GOS-2. 2.2% of patients had good recovery (GOS-5) at discharge. 21.7% of patients had good outcome (GOS-4-5) and 78.3% had poor outcome (GOS 1-3) at discharge. We found the association between level of consciousness at admission and surgical outcome at discharge was satiscally significant (p=<0.001).*

## Introduction

Intracerebral haemorrhage constitutes about 13% of stroke cases in United States. In Asian population the frequency may be as high as 30%[1]. No study regarding incidence of ICH in our country is available. With a population of more than 143 million, increasing life expectancy and improvement of diagnostic facilities including availability of CT scan in many district centers contributes in increase in the number of detection of intracerebral haemorrhage patients. The place of surgery in the treatment of intracerebral haematoma is still controversial. A surgeon when confronted with an unconscious patient with a large blood clot in patient's brain has the natural inclination to remove it. The worse the condition of the patient the greater is the pressure on the surgeon from the family of patient "to do some thing". There are

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some studies suggesting that initial level of consciousness (GCS) is crucial to early clinical decision making. In some studies clinical outcome after surgery was recorded at discharge, at 3 months, and at 6 months after surgery using Glasgow outcome score(GOS). As most patients with ICH come to hospital late and due to different reasons we fail to follow up patients for long term. In our study we tried to assess whether level of consciousness at admission has any relation with outcome at discharge in spontaneous supratentorial intracerebral haemorrhage cases who under went surgery for it.

**Materials and Methods:**

It was a prospective study in the department of Neurosurgery, BSMMU, Dhaka; from November 2004 to May 2007. All consecutive admitted patients of spontaneous supratentorial intracerebral haemorrhage who underwent surgery for it were the study population. Total number of patients included in the study were 46 .

**Sample selection:**

Inclusion criteria

i. CT scan evidence of spontaneous supratentorial intracerebral haemorrhage.

ii. Those patients who underwent surgery for spontaneous supratentorial intracerebral hemorrhage.

Exclusion criteria

i. Patients treated conservatively.

ii. Patients requiring artificial ventilation.

iii. Intracerebral haemorrhage associated with subarachnoid haemorrhage.

iv. Patients who were on anticoagulant therapy.

v. Diabetic patients.

**Research approach**

This study was done on admitted patients from November 2004 to May 2007 with a CT scan confirmed spontaneous supratentorial intracerebral hemorrhage fulfilling all selection criteria. All these patients were evaluated on the basis of detailed history, clinical examination and CT scan findings. All these patients were operated upon and followed up after surgery till discharge by observing GCS, neurological improvement and Glasgow outcome score.

**Research instrument**

A checklist was prepared including the variables such as age and sex of the patients, clinical presentations, clinical findings, GCS score on admission, volume and location of hematoma on CT scan, immediate preoperative GCS score, Glasgow outcome score (GOS) at discharge. It also included history of hypertension. The data was collected by the researcher himself.

**Data collection procedure**

i. On admission, detailed history from the patients and/or from the attendant were taken, thorough general and neurological examination were carried out including GCS score evaluation on admission.

ii. Findings of CT scan were recorded carefully.

iii. Volume of haematoma was calculated from CT scan findings.

iv. Follow up examination findings were recorded at 1$^{st}$ postoperative day (POD), 3rd$^{th}$ POD and GOS at discharge.

v. Respective findings were recorded into data collection sheet.

**Variables observed**: Age of the patient, Sex of the patient, History of Hypertension,

Time elapsed since ictus to admission, Clinical features-Altered level of consciousness assessed with GCS score.

• Glasgow coma scale (GCS)[2] is as follows.

**Eye opening**: Spontaneous-4,To speech-3,To pain-2, None-1.

**Best motor response**: Obeys command-6, Localizes pain-5,Withdraws to pain-4, Flexion (decorticate)-3, Extensor (decerebrate)-2, No movement-1.

**Best Verbal response**: Oriented-5, Confused-4, Inappropriate word-3, Incomprehensible-2,

None-1.

**Imaging variables**: Volume of haematoma, Location of haemorrhage .These were measured by CT scan.

**Surgical management variables:** Type of surgery done: a) Burrhole b)Craniectomy c)Cranotomy d)External ventricular drainage (EVD)

**Surgical outcome variables:** Glasgow outcome score at discharge.

Measurement of volume of haematoma: **Volume of haematoma was measured by modified ellipsoid formula, simply by 1/2ABC$^3$.**

**CT scan assessment:** Most of the patients were admitted with CT scan already done. In rest of the patients CT scan were done in the department of Radiology and Imaging, BSMMU, Dhaka.

**Surgical outcome:** In this study early surgical outcome is used to designate the outcome at discharge and are by Glasgow Outcome Scale. Glasgow Outcome Scale[2] is as follows:

**Score 5**: Good recovery, resumption of normal life despite minor deficits ("Return to work" not reliable).

**Score 4**: Moderate disability (disabled but independent)-travel by public transportation, can work in sheltered setting (exceeds mere ability to perform "activity of daily living").

**Score 3**: Severe disability (conscious but disabled )-dependent for daily support (may be institutionalized-but this is not a criteria).

**Score 2**: Persistent vegetative state-unresponsive and speechless after 2-3 weeks. May open eyes and have sleep/wake cycle.

**Score 1**: Death

**Data analysis:** Data were collected and edited manually. A master sheet was prepared and these were analyzed by appropriate statistical test using statistical software package SPSS (Statistical Program for Social Science) version12. p value < .05 considered as significant.

**Results:**

A total number of 46 patients were included in this study. All the results of this study were shown in the tables.

**Table-I**

*Distribution of the patients by age (n-46)*

| Age in years | Frequency | Percentage |
|---|---|---|
| 30-39 | 6 | 13.0 |
| 40-49 | 7 | 15.2 |
| 50-59 | 15 | 32.6 |
| 60-69 | 13 | 28.3 |
| 70-79 | 4 | 8.7 |
| ≥ 80 | 1 | 2.2 |
| Total | 46 | 100.0 |

Mean age ± standard deviation = (54.91 ±12.40) years; range (30-80) years

Table-I shows age distribution of the cases. Majority (32.6%) of them were in the group of 50-59 years (inclusive), followed by age group of 60-69 (28.3%) years, 40-49 years (15.2%),30-39 years (13%) and 70-79 years (8.7%) respectively. Only one case was in ≥ 80years group (2.2%).The mean age was 54.91years with standard deviation of 12.4 years. The minimum age of the cases was 30 years and the maximum age was 80 years.

**Table-II**

*Distribution of patients by sex (n-46)*

| Sex | Frequency | Percentage |
|---|---|---|
| Male | 32 | 69.6 |
| Female | 14 | 30.4 |
| Total | 46 | 100.0 |

Table-II shows the distribution of the patients by sex. Majority (69.6% ) of the patients were male and 30.4% of them were female. Male to female ratio was 2.29: 1.

**Table-III**

*Preceding history of hypertension (n-46)*

| History of hypertension | Frequency | Percent |
|---|---|---|
| Present | 22 | 47.8 |
| Absent | 24 | 52.2 |
| Total | 46 | 100.0 |

Table-III demonstrates the presence of history of hypertension. In 47.8% of the patients history of hypertension was present and rest 52.2% of patients had no history of hypertension.

**Table-IV**

*GCS score at admission (n-46)*

| GCS score | Frequency | Percentage |
|---|---|---|
| Range | | |
| >13 | 1 | 2.2 |
| 9-13 | 21 | 45.7 |
| <9 | 24 | 52.1 |
| Total | 46 | 100.0 |

Mean score ± standard deviation= 8.73±2.36

Table-IV demonstrates the GCS score of patients at admission. Most of the patients (52.1%) had GCS <9, 45.7% of the patients were in GCS 9-13 group. Only 2.2% of them had GCS>13. Mean GCS score at presentation was 8.73 with a standard deviation of 2.36. The minimum score recorded was 3 and maximum score recorded was 14.

**Table-V**

*Glasgow outcome score at discharge (n-46)*

| Glasgow outcome score | Frequency | Percentage |
|---|---|---|
| 5 (Good recovery) | 1 | 2.2 |
| 4 (Moderate disability) | 9 | 19.6 |
| 3 (Severe disability) | 14 | 30.4 |
| 2 (Vegetative state) | 1 | 2.2 |
| 1 (Death) | 21 | 45.6 |
| Total | 46 | 100.0 |

Mean score ± standard deviation = 2.3 ± 1.29

Table-V demonstrates majority of the patients (45.6%) died (GOS 1). 30.4% of patients were conscious but disabled (GOS 3), 19.6% of patients were discharged with moderate disability (disabled but independent), 2.2% of patients were

discharged with GOS 2 and 2.2% patients had good recovery (GOS 5) at discharge. Mean Glasgow outcome score was 2.3 with standard deviation 1.29.

**Table-VI**

*Distribution of patients according to outcome at discharge (n=46)*

| Outcome at discharge | Frequency | Percentage |
|---|---|---|
| Good (GOS 4-5) | 10 | 21.7 |
| Poor (GOS 1-3) | 36 | 78.3 |
| Total | 46 | 100.0 |

Table-VI demonstrates 21.7% of patients had good outcome, Glasgow Outcome score (4-5) at discharge and 78.3% had poor outcome, Glasgow outcome score (1-3) at discharge after surgery.

**Table-VII**

*Distribution of patients according to volume of haematoma (n-46)*

| Volume range in cc | Frequency | Percentage |
|---|---|---|
| <30cc | 5 | 10.9 |
| 30-60cc | 31 | 67.4 |
| >60cc | 10 | 21.7 |
| Total | 46 | 100.0 |

Mean volume ± Standard deviation= 49ml ±16.10

Table-VII demonstrates volume of measured haematoma in groups. Most of the patients (67.4%) had haematoma volume between 30-60cc. 21.7% patients had volume more than 60cc, 10.9% patients had volume less than 30cc. Mean volume of haematoma was 49cc with standard deviation of 16.10.Minimum volume was 20cc and maximum volume was 85cc.

**Table-VIII**

*Distribution of patients according to location of haematoma in CT scan (n-46)*

| Location of haematoma in CT scan | Frequency | Percentage |
|---|---|---|
| Basal ganglion region | 15 | 32.6 |
| Lobar | 20 | 43.5 |
| Combined (Basal ganglion+Lobar) | 11 | 23.9 |
| Total | 46 | 100.0 |

Table-VIII demonstrates that 32.6% of spontaneous supratentorial intracerebral haemorrhage were in basal ganglion region, 43.5% were in lobar locations, 23.9% involved both basal ganglion and lobar region.

**Table-IX**

*Distribution of patients according to types of surgical intervention (n-46)*

| Surgical intervention | Frequency | Percentage |
|---|---|---|
| Burr hole | 18 | 39.1 |
| Craniectomy | 21 | 45.7 |
| Craniotomy | 4 | 8.7 |
| Craniectomy+EVD | 3 | 6.5 |
| Total | 46 | 100.0 |

Table-IX demonstrates that out of 46 patients with spontaneous supratentorial intracerebral haemorrhage in 39.1% cases decompression was done by burr hole aspiration of haematoma, in 45.7% cases craniectomy, in 8.7 %cases craniotomy and in 6.5% cases craniectomy plus external ventricular drainage were done.

**Table-X**

*Distribution of patients according to post-operative survival and death at discharge (n-46)*

| | Frequency | Percentage |
|---|---|---|
| Alive | 25 | 54.3 |
| Dead | 21 | 45.7 |
| Total | 46 | 100.0 |

Table-X demonstrates the survival and death rate in our study, where 54.3% of patients were alive at discharge and 45.7% patients had died.

**Table-XI**

*Distribution of patients according to time elapsed between ictus and admission (n-46)*

| Time in hours | Frequency | Percentage |
|---|---|---|
| ≤48 hrs | 24 | 52.2 |
| >48hrs | 22 | 47.8 |
| Total | 46 | 100.0 |

Table-XI demonstrates time elapsed between ictus and admission in our hospital, where 52.2% of patients were admitted within 48 hours and 47.8% got admitted 48 hours after the ictus respectively.

**Table-XII**

*Distribution of patients according to association between GCS at admission and GOS at discharge (n-46)*

| GCS score at admission | GOS at discharge 4-5 Favorable | 1-3 Unfavorable | Total |
|---|---|---|---|
| ≥ 9 | 10(100%) | 12(33.33%) | 22(47.83%) |
| <9 | 0(0%) | 24(66.67%) | 24(52.17%) |
| Total | 10 | 36 | 46 |

Value in parenthesis denotes corresponding column percentage.
Pearson $\chi^2$ test value after Yates correction 11.39
df=1
p value =<0.001 (p value <0.05 was considered significant)

Table-XII demonstrates association between level of consciousness at admission recorded as GCS score and surgical outcome in terms of Glasgow Outcome Scale at discharge. Patients who had GCS score 9 or more at admission had favorable outcome. p value <0.001 after Yates correction indicates that association between level of consciousness at admission and surgical outcome at discharge was significant.

**Table XIII**

*Distribution of patients according to association between level of consciousness at admission and mortality*

| GCS score at admission | Mortality of patient Dead | Alive | Total |
|---|---|---|---|
| GCS≥9 | 4(19.05%) | 18(72%) | 22(47.83%) |
| GCS<9 | 17(80.95%) | 7(28%) | 24(52.17%) |
| Total | 21 | 25 | 46 |

Value in parenthesis denotes corresponding column percentage.
Pearson $\chi^2$ test value 12.82
df =1
p value =<0.001(p value <0.05 was considered significant)

Table-XIII demonstrates that association between level of consciousness at admission and mortality. Majority of the patients (80.95%) who had GCS score <9 at admission were dead at discharge. Majority (72%) of the patients who had GCS score ≥ 9 at admission were alive at discharge. p value <0.001 indicates association between level of consciousness at admission and mortality was significant.

**Fig.-1:** *Peroperative photograph of parietal craniectomy and evacuation of haematoma*

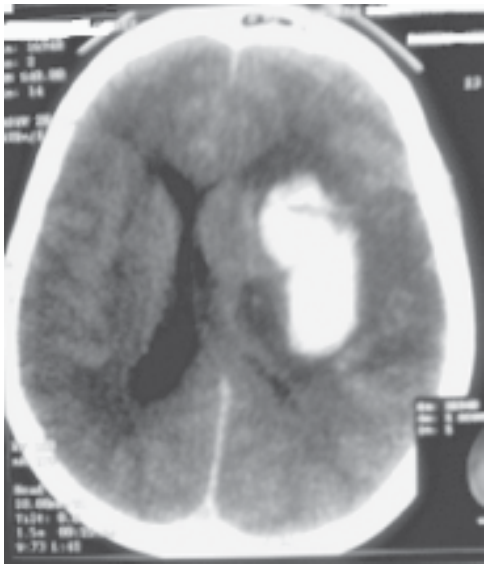

**Fig.-2:** *Plain CT scan showing left basal ganglion ICH with midline shift and perilesional oedema.*

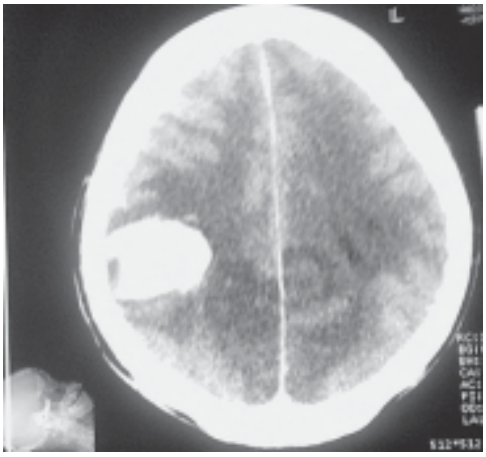

**Fig.-3:** *Plain CT scan showing right parietal ICH*

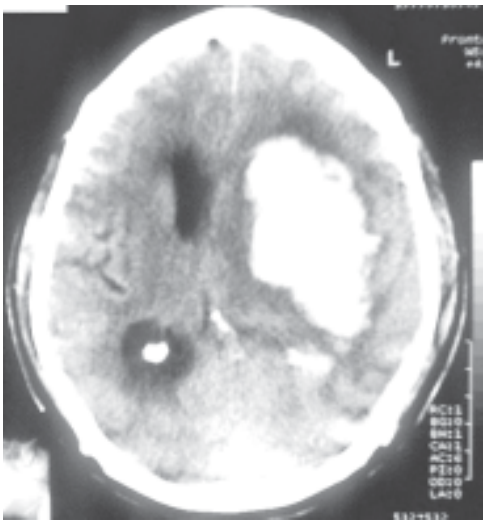

**Fig.-4:** *Plain CT scan showing left basal ganglion ICH with frontoparietal extension*

**Discussion**

Controversy exists concerning the indications for operative treatment of spontaneous ICH, and decisions should be individualized. Among the important considerations are the patients initial level of consciousness, size and location of haematoma, involvement of dominant hemisphere and deterioration of neurologic status. The single most important factor guiding the management of patient with spontaneous ICH based on nearly all literature to date, is the preoperative level of consciousness[4]. This study was carried out in the department of neurosurgery, BSMMU, Dhaka from November 2004 to

May 2007. In this study we tried to find out relation of level of consciousness at admission and early outcome of surgery for spontaneous supratentorial ICH. The study subjects were 46 patients based on selection criteria. The age ranged between 30-80 years and mean age was 54.91years, which is almost similar to the mean age of the study of Ahmed et al[5] in Pakistan (58.8 years) but is much lower than the study of Bilbao et al[6] in Spain. The age group with highest frequency was also lower level in our series than that of the series of Bilbao et al[6]. These observations reflect the higher life expectancy in western countries. The study of Kloc[7] in Poland shows mean age was 47.63 years. The male to female ratio was higher in our series (2.29:1) than other series of Bilbao[6] (1.83:1), Ahmed[5] (1.38:1) and Yen[8] (1.59:1) probably due to the fact that males are more privileged in our society and probably due to the fact that the male bed number of department of Neurosurgery, BSMMU is more than that of the female beds. In our study history of pre-existing hypertension was 47.8%, which is lower than the study of Ahmed [5]and Razzak[9], which was 66.5% and 66% respectively. In the study of Yen[8] in Taiwan, history of previous hypertension was 53.4%. In our study the range of admission GCS score was <9 in 52.1%, 9-13 in 45.7%,>13 in 2.2% cases respectively.The study of Yen et al[8] in Taiwan shows admission GCS score 32.05%, 21.02%,46.92% respectively and study of Bilbao[6] in Spain shows 24%, 29% and 47% respectively. In our study we found 52.2% of patients were admitted within 48hrs and 47.8% of patients were admitted after 48hrs from onset of symptoms. Lampl et al[10] observed all patients in their study were admitted 15 minutes to 4 hours after the onset of first symptoms and they found significant correlation between low GCS score or size of haematoma and lethal outcome (p<.001), especially during the first 48 hours(p<.007).Bilbao [6] observed in their series, 57% of patients present within 6 hour of onset of symptoms,13% between 6-24 hours,30% after 24 hours. In our study mortality was 45.7%. Bilbao[6] in their study showed overall mortality 44% within study period of 12 months. But our study period was limited till discharge or inhospital mortality following surgery. In the study of Kloc[6] mortality in surgically treated patients was 39.4% at one month. Cheung and Zou[10] observed that the death rate of patients of ICH was 22% in a hospital of Hong Kong. Due to different reasons we fail to follow up patients for longer period in our country. Regarding analysis of outcome of patients we considered Glasgow Outcome Score at discharge. Patients were discharged when they were considered neurologically stable. Only 2.2% of patients could resume normal life despite minor deficits (GOS-5) at discharge, 19.6% were disabled but independent (GOS-4), 30.4% of patients were conscious but disabled (GOS-3), 2.2% of patients were vegetative at discharge,45.7% of patients died (GOS-1).Yen et al[8] in their study showed 10.7% of patients were still capable of work,16.3% had minimal disability, 24.3%were partially disabled, 18.7% totally disabled, 13% vegetative, and 17% died. Bilbao et al[6] in their study showed that the number of independent patients increased from 14% at 30 day follow up to 45% at 6 month follow up and dependency decreased from 86% at 30 day follow up to 55%at 6 month follow up. This probably indicates better critical care

system in western countries and reflects outcome in longterm follow-up. In our study we found haematoma volume were <30cc in10.9% cases, 30-60cc in 67.4%and >60cc in 21.7% cases. Bilbao et al[6] in their study found haematoma volume 33%, 33% and 27% respectively which also relates to the better GCS level at admission in their study. The location of the haemorrhage has been shown to be an important criteria for determining early outcome of surgery [4].In our study the distribution of haematoma was lobar 43.5%, haematoma involving basal ganglion and with lobar extension was 56.5%. Patients with lobar haematoma had better GCS score at admission. Bilbao et al[6] in their study found 55% of the haematomas were deep (basal ganglia or thalamus) and 47% were lobar. They found that GCS is influenced by the site where the haematoma is located (p<0.001). The ideal goals of surgical treatment for ICH should be to remove as much clot as possible within shortest period of time with the least amount of brain trauma from the surgery itself. More complete clot removal may decrease elevated ICP and local pressure effect of the blood clot on the surrounding brain. M. Zuccarello and colleagues showed the median reduction in volume of ICH from baseline (median 35cc) to 24 hour CT (median16cc) was 44%. They performed surgery within 3 hours of randomization[11]. Goal of our surgical intervention was adequate decompression indicated by pulsation of brain and free flow of CSF at the end of surgery. Due to different reasons we could not perform postoperative CT scan to evaluate adequacy of decompression and other CT parameters. In 45.7% of our patients surgical intervention were done by craniectomy followed by Burr hole aspiration in 39.1% and Craniotomy in 8.7% cases.

The clinical severity has been considered as a potential predictor of global outcome, and specifically of mortality, in patients with stroke. The level of consciousness, measured by the GCS score, presence of coma, or a minor consciousness involvement on admission was already observed as an outcome predictor in patients with hemorrhagic stroke. Trials that used the GCS, observed an association between low GCS score and worse evolution[12].

Glasgow coma scale of $\geq$9 is predictor of favorable out come and GCS <9 is predictor of unfavorable outcome[12]. In their study Ruth et al[13] found statistically significant correlation of the level of consciousness and outcome. In our study we found significant (p<.001) association between level of consciousness at admission and surgical outcome. In our study we found GCS score $\geq$ 9 at admission had better outcome after surgery. Juvela[14] also found initial GCS score is a significant (p<0.001) outcome predictor. Association between the surgical outcome in terms of mortality and initial level of consciousness (GCS score at admission) was significant (p<0.001) in our study and similar significant association was found by Bilbao[6], Razzak and Hussain et al [9].

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# Symptoms and Signs of PLID and their Relation to the Localization of Lumbar Disc Herniation

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**Abstract**

*Background: Symptoms and signs of Protruded Lumbar intervertebral disc (PLID) are well recognized and they correlate well with the level of pathology found on MRI. Hypothesis: Level of lumbar disc herniation may be diagnosed accurately from symptoms and signs. Objective: To find out the accuracy of clinical findings of level of disc prolapse of PLID patients in comparison with MRI findings. Method: The study is a prospective observational study carried out in the department of Neurosurgery, Bangabandhu Sheikh Mujib Medical University (BSMMU), from March 2006 – October 2007. A total number of fifty nine (59) PLID patients who were admitted in the department Neurosurgery, BSMMU, were selected consecutively on the basis of inclusion and exclusion criterias. Diagnosis was confirmed by MRI. Accuracy of the clinical findings to diagnose the level of pathology was evaluated. Statistical analysis was done by SPSS. Results: A total number of fifty nine (59) PLID patients were included in the study. The mean age was 40.8 ± 11.9 years and the minimum and maximum ages were 21 and 65 years respectively. The male and female ratio was 11:1. Nearly 55% of the patients were manual workers. Over one-quarter (28.8%) of patients complained of radiation of pain to the right lower limb, 30.5% to the left lower limb and 39% to both lower limbs. About 60% of the patients had signs and symptoms of herniation of disc in between L4-5 and 35.6% in between S1-L5 vertebrae. Only 6.8% cases had herniation at the level of L3-4. Level of disc prolapse detected by MRI showed that 86.4% of the patients had disc prolapse in between L4-5 and 62.7% at L5-S1, 30.5% in between L3-4. Very few patients exhibited disc prolapse in between L1-2 (3.4%) and L2-3 (6.8%) vertebrae. Conclusion: From the findings of the study and discussion thereof, it could be concluded that the clinical diagnosis of level of disc prolapse was in little agreement with MRI diagnosis of level of disc prolapse, suggesting that level of disc prolapse cannot be established with certainty without image findings of MRI.*

**Introduction:**

Low back pain is the second leading cause of doctor visits each year, affecting over 65 million Americans. The precise cause of back pain is largely unknown, but degeneration of the intervertebral disc has been implicated as a possible source[1].

The syndrome of low back pain with or without sciatica is one of the more common

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diagnostic problems that the orthopaedist faces in his daily practice. Sciatica; formerly a syndrome of unknown aetiology has aroused the interest of research workers for almost two centuries and each generation of investigators has added to our knowledge in this field [2].

Some symptoms and signs (for example, paresis and sensory loss) indicate the level of the disc herniation and root involvement. Other clinical findings such as a positive SLR test result is not directly related to the level of the affected root. The pathoanatomical reasons for the presence of such signs in some patients and its absence in others remain largely speculative[3].

The patients with herniated lumbar disc were admitted frequently in neurosurgery ward with complains of low back pain, with or without radiation, tingling and numbness in feet, weakness and wasting of lower limbs, foot drop, bowel and bladder incontenence etc. With increasing age, the disc degenerated, the neucleous pulposus herniated through the tear of annulus fibrosus and causes compression over the nerve roots or over the Cauda-equina.

Lumbar disc herniation is one of the few causes of spinal pain that can be successfully treated surgically. Perhaps because of the insufficient knowledge of most other causes of spinal pain, it has become a popular diagnosis. The overall prognosis is favorable, with a strong tendency toward spontaneous healing; herniations shrink and may be resorbed. In most cases conservative treatment, control of pain, and awaiting the natural course are enough. However, incidence of disc surgery varies tenfold among industrialized countries, differences too conspicuous to be explained by biologic factors alone[4].

The radiographic appearance of gas collection in clefts or spaces in the intervertebral disk is described as the "vacuum phenomenon'', Fick first described this phenomenon in 1910. In 1937 Magnusson and in 1942 Knutsson correlated this radiologic phenomenon with the degenerative process in the intervertebral disk[5].

**Materials and methods:**

The study is a prospective observational study carried out in the department of Neurosurgery, BSMMU, from March 2006-October 2007.

A total number of fifty nine (59) PLID patients who were admitted in the department Neurosurgery, BSMMU were selected consecutively on the basis of inclusion and exclusion criterias. Diagnosis was confirmed by MRI. Accuracy of the clinical findings to diagnose the level of pathology was calculated. Statistical analysis was done by SPSS.

**Results:**

The total number of study subjects were fifty nine (59) PLID patients. Their the mean age was 40.8 ± 11.9 years and the minimum and maximum ages were 21 and 65 years respectively. The male and female ratio was 11:1. Nearly 55% of the patients were manual workers. Over one-quarter (28.8%) of patients complained of radiation of pain to the right lower limb, 30.5% to the left lower limb and 39% to both lower limbs. The motor power of muscles was found weak only in 6.8% cases. Level of disc prolapse detected by MRI showed that 86.4% of the patients had disc prolapse in between L4-5 and 62.7% in between L5-S1, 30.5% in between L3-4. Very few patients exhibited disc prolapse in between L1-2 (3.4%) and in between L2-3 (6.8%) vertebrale.

**Table I**

*Distribution of patients by age (n = 59)*

| Age (Years) | Frequency | Percentage |
|---|---|---|
| ≥ 20 | 0 | 0 |
| 21 - 25 | 4 | 6.7 |
| 26 - 30 | 8 | 13.5 |
| 31 – 35 | 05 | 8.5 |
| 36 – 40 | 09 | 15.3 |
| 41 – 45 | 09 | 15.3 |
| > 45 | 24 | 40.7 |
| Total | 59 | 100 |

* Mean age = (40.8 ± 11.9) years; range = (21 - 65) years.

Table I shows the age distribution of the patients. Of the 59 patients 20.3% were 30 years or below, 8.5% were in between 31-35 years, 15.3% were in between 36-40 years, another 15.3% were in between 41-45 years and 40.7% were above 45 years of age. The mean age of the patients was 40.8 ± 11.9 years and the lowest and highest ages were 21 and 65 years respectively.

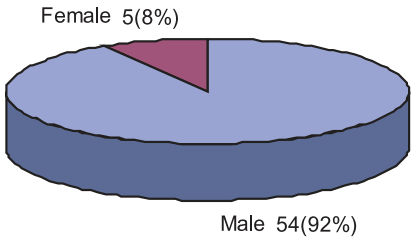


**Fig.-1:** *Distribution of patients by sex (n-59)*

Figure 1 shows the distribution of patients by sex. Majority (92%) of the patients were male and only 8% were female. The male to female ratio was 11:1.

**Table-II**

*Distribution of patients by occupation (n = 59)*

| Occupation | Frequency | Percentage |
|---|---|---|
| Manual worker | 32 | 54.2 |
| Non-manual worker | 27 | 45.8 |
| Total | 54 | 100 |

Table II demonstrates that nearly 55% of the patients were engaged with manual work and the rest 45.8% were non-manual worker in this study.

**Table-III**

*Distribution of patients by SLR test (n = 59)*

| SLR test | Right | | Left | |
|---|---|---|---|---|
| | Frequency | % | Frequency | % |
| $90^0$ (Normal) | 15 | 25.4 | 14 | 23.7 |
| $85^0 – 40^0$ (Positive) | 40 | 67.8 | 43 | 73.9 |
| $\leq 30^0$ (Strongly positive) | 04 | 6.8 | 02 | 3.4 |
| Total | 59 | 100 | 59 | 100 |

Table III shows the distribution of patients by straight leg raising (SLR) test. One-quarter of the patients were found normal on SLR test ($90^0$). Over 65% exhibited positive SLR test ($85^0 – 40^0$) and the rest 10% had strongly positive SLR test ($\leq 30^0$)

Figure 2 demonstrates that almost two-third (64%) of the patients exhibited negative crossed SLR test and over one-third (36%) positive crossed SLR test.

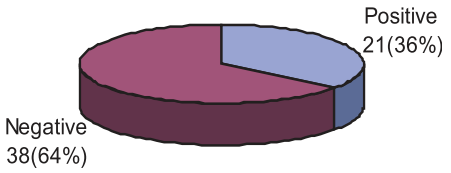


**Fig. 2** *Distribution of patients by crossed SLR (n = 59)*

Four (7%) patients had positive femoral stretch test and the rest (93%) had negative femoral stretch test (Fig. 3).

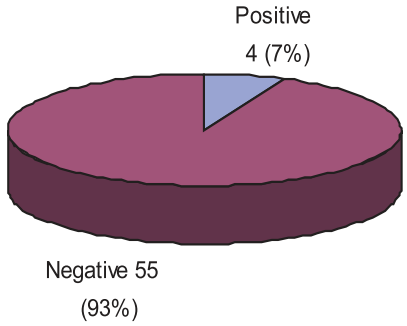


**Fig.-3:** *Distribution of patients by femoral stretch test (n = 59)*

None of the hip group of muscles was found weak on motor function test, while in 3(5.1%) cases knee group of muscles were found weak. More than half (54.2%) of the subjects exhibited weak extensor hallucis longus test, while 30.5% had weak flexor digitalis longus test (Table IV).

**Table IV**

*Distribution of patients by motor function test (n = 59)*

| Motor function test | Frequency | Percentage |
|---|---|---|
| Hip group | | |
| Weak | 00 | 0.0 |
| Normal | 59 | 100.0 |
| Knee group | | |
| Weak | 03 | 5.1 |
| Normal | 55 | 94.9 |
| EHL | | |
| Weak | 32 | 54.2 |
| Normal | 27 | 45.8 |
| FDL | | |
| Weak | 18 | 30.5 |
| Normal | 41 | 69.5 |

About 54% of the patients had sensory deficit – of them 28.8% along L5 and 25.4% along S1 dermatome. However, only 2(3.4%) had saddle anesthesia (Table V).

**Table V**

*Distribution of patients by sensory function loss (n = 59)*

| Sensory function loss | Frequency | Percentage |
|---|---|---|
| Present | 32 | 54.2 |
| L1 dermatome | 00 | 0.0 |
| L2 dermatome | 00 | 0.0 |
| L3 dermatome | 00 | 0.0 |
| L4 dermatome | 00 | 0.0 |
| L5 dermatome | 17 | 28.8 |
| S1 dermatome | 15 | 25.4 |
| Saddle anesthesia | 02 | 3.4 |

**Table VI**

*Distribution of patients by level of disc herniation detected by clinical signs and symptoms (n = 59)*

| Levels | Frequency | Percentage |
|---|---|---|
| $L_{1-2}$ | 00 | 00 |
| $L_{2-3}$ | 00 | 00 |
| $L_{3-4}$ | 04 | 6.8 |
| $L_{4-5}$ | 35 | 59.3 |
| $L_5$-$S_1$ | 21 | 35.6 |

Table-VI demonstrates that 59.3% of the patients had signs and symptoms of disc herniation in between of $L_{4-5}$ and 35.6% in between L5-S1. Only 6.8% cases had herniation in between $L_{3-4}$ vertebrae.

**Table VII**

*Distribution of respondents by level of disc prolapse detected by MRI scan (n = 59)*

| Level | Frequency | Percentage |
|---|---|---|
| L 1-2 (L2) | 02 | 3.4 |
| L 2-3 (L3) | 04 | 6.8 |
| L 3-4 (L4) | 18 | 30.5 |
| L 4-5 (L5) | 51 | 86.4 |
| L 5-S1 (S1) | 37 | 62.7 |

Level of disc prolapse detected by MRI showed that 86.4% of the patients had disc prolapse in between L4-5 and 62.7% in between L5-S1, 30.5% in between L3-4 vertebrae. Very few patients exhibited disc prolapse in between L1-2 (3.4%) and L2-3 (6.8%) vertebrae.

Fig.-4 compares the level of disc prolapse between clinical diagnoses and MRI findings. Majority (86.4%) of the level of disc prolapse was in between $L_{4-5}$ vertebrae by MRI, 60% of which was diagnosed clinically. Likewise 62.7% disc prolapse in between $L_5$ - $S_1$ vertebrae detected by MRI was matched in only 35.6% of cases diagnosed clinically. The agreement between the two modalities of diagnoses was bare minimum.

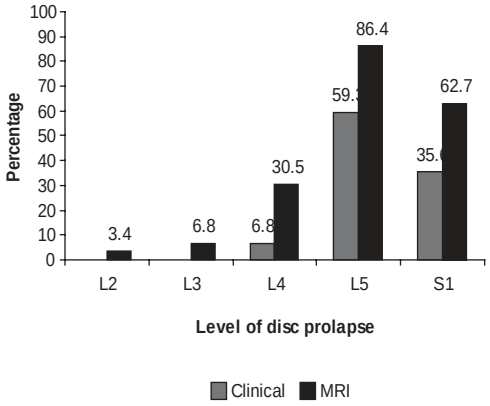


**Fig.-4:** *Comparison of level of disc prolapse between groups (n = 59)*

The strength of agreement between clinical diagnoses & MRI diagnoses regarding level of disc prolapse was evaluated using kappa statistics (K-value). The test revealed a poor agreement between the two procedures (K<0.4) suggesting that level of disc prolapse could not be correctly diagnosed without image findings by MRI (Table VII). Many patients show multiple discs prolapse clinically and in MRI findings.

**Table VIII**

*Distribution of patients agreement between clinical & MRI diagnoses (n= 59)*

| Level of disc Prolapse | Clinical Diagnoses | MRI Diagnoses | K-value | Strength of agreement |
|---|---|---|---|---|
| $L_{1-2}$ | 00 | 3.4 | …….. | …….. |
| $L_{2-3}$ | 00 | 6.8 | …….. | …… |
| $L_{3-4}$ | 6.8 | 30.5 | 0.284 | Poor |
| $L_{4-5}$ | 59.3 | 86.4 | 0.294 | Poor |
| $L_5$-$S_1$ | 35.6 | 62.7 | 0.305 | Poor |

**Discussion:**

Lumbar disc herniations occur most commonly from 3rd to 5th decade with peak incidence in the 4th decade of life and less commonly in the 6th & 7th decade. Lumbar disc herniations are most common at the level of L4 – L5 and L5 –S1 levels. Less than 5% of disc herniations occur at L1, L2 & L3 levels. The earliest evidence of root involvement in a lumbar disc prolapse is radiating pain along the course of the sciatic nerve, commonly called sciatica[6].

The present study showed that the mean age of the PLID cases was 40.8 years with lowest and highest ages were 21 and 65 years respectively which is consistent with the findings of two other studies where mean ages of the study patients were 42.9 years and 41.6 years respectively[7]. Lumbar disc herniation in childhood and adolescence is a relatively rare condition. Only 1 to 3% of lumbar disc herniation occurs in individuals under 21 years of age[8]. Sex distribution of the lumbar disc prolapse in the United States and other developed countries is almost equal[9], which sharply contrast with male, female ratio (11:1) observed in the present study. The reason of such wide variation in male to female ratio is likely due to proportionately higher bed allocation for male in the neurosurgery ward of BSMMU. Besides this, in the sociocultural context of our country female patients get less preference for shophisticated medical treatment which might be another reason of low inclusion of female patients in our study.

A positive straight leg raising test is an indication of nerve root involvement. If pain occurs when the leg is lifted between 30 – 70 degrees from horizontal position and travels down to the leg behind the knee, the test is considered positive. SLR test was usually positive in all levels of herniation (94%) and it was strongly positive under 30 degrees, more frequently in herniation of the lower discs. In our study over 65% exhibited positive SLR test ($85^0 – 40^0$) and the rest 10% exhibited strongly positive SLR test ($\leq 30^0$), while over one-third (36%) had positive crossed SLR test. In our country limitation of straight leg raising may not be seen in some patients though root compression by a prolapsed disc is proved later at surgery. This is explained by the fact that, in India, most people adopt the stooping posture to perform their many of the daily activities. Thus the nerve is perhaps already stretched and elongated and has a certain amount of elastic flexibility which permits full straight-leg

raising despite the compression of nerve roots by the disc protrusion. Femoral stretch test was positive in only 4% cases in our study, because very few patients had high level of lumbar disc prolapse.

Regarding motor function test, 54% patients showed L5 root involvement (weakness of EHL), 30% of patients showed S1 root involvement (weakness of FDL) and only 50% showed L3 root involvement (weakness of knee Flexion (Table – IV).

The diagnostic value of sensibility test for patients with lumbar disc herniation is debatable. The presence of a reduction in sensibility in such patients varies between 21 and 88%[11]. The reported frequency of loss in individuals with documented herniated lumbar discs has range from 21 to 84% (Weise et al., 1985). In our study, sensory impairment along the L4 distribution was 2%, L5 distribution 20% and S1 distribution 14% respectively.

MRI is the imaging modality of choice in evaluating patients with lumbar disc disease. Studies have shown that as many as 60% of people without low back symptoms have disc bulgings and protrusions of discs on MRI. Therefore, these findings may not correlate with the patient's symptoms.

MRI is a helpful preoperative diagnostic investigation which shows structural change in the disc and the localization and size of the disc sequestration, but there was no correlation between the imaging findings and the clinical symptoms[12]. About 60% of the patients had signs and symptoms of herniation in between L4-5, 35.3% in between L5 - S1 and only 6.8% cases had herniation in between $L_{3-4}$ vertebrae which on MRI was found to be 86.4%, 62.7% and 30.5% respectively, showing very little agreement between the two modalities of diagnoses in detecting level of disc prolapse.

**Conclusion:**

From the findings of the study and discussion thereof, it could be concluded that the clinical diagnoses of level of disc prolapse was in little agreement with MRI diagnoses of level of disc prolapse, suggesting that level of disc prolapse cannot be established with certainty without image findings of MRI.

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# Analytic Study on Ventilator Associated Pneumonia (VAP) in Neurosurgery Intensive Care Unit (NICU)

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**Abstract**

*It is an observational study. Place of study was the Neurosurgery Intensive Care Unit (NICU) of Square Hospitals Ltd. All patients who were diagnosed as a case of Ventilator Associated Pneumonia (VAP) in ICU during the 2 years study period (01.04.2007 to 31.03.2009) were included in this study. Data were collected by patient's clinical register checklist. Total number of patients were 39. Main objective of our study was to identify the organisms associated with VAP (Ventilator Associated Pneumonia) in Neurosurgery intensive care unit (NICU). Organisms were collected by tracheal swab and were identified by culture and sensitivity test. Most patients were male. Age of maximum patients was more than 70 years. Commonest isolated organism was Pseudomonas spp. Other organisms were* Acinatobacter spp*,* Staphylococcus aureus*,* Klebsiella spp, Candida*, and Methicillin Resistant Staphylococcus aureus (MRSA). Mortality was 28.2% in our series.*

***Key words:*** *Ventilator associated pneumonia, organism and Neurosurgery intensive care unit.*

**Introduction**

Ventilator-associated pneumonia (VAP) is the most common nosocomial infection in the ICU and contributes disproportionately to both poor outcomes and the high cost of care in critically ill patients[1]. Since the initial 1996 American Thoracic Society (ATS) guideline on nosocomial pneumonia, a number of new developments have appeared, mandating a new evidence-based guideline for hospital-acquired pneumonia (HAP), including healthcare-associated pneumonia (HCAP) and ventilator-associated pneumonia (VAP).

Ventilator-associated pneumonia is a common and highly morbid condition in critically ill patients[2]. Epidemiologic investigations have shown cumulative incidence rates of 10 to 25%[3], crude mortality rates of 10 to 40%[4] and attributable mortality rates of 5 to 27%[5]. Hospital length of stay and cost are both increased in patients who develop ventilator-associated pneumonia[6].

The predominant organisms responsible for infection are *Staphylococcus aureus*, *Pseudomonas aeruginosa*, and *Enterobacteriaceae*, but etiologic agents

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widely differ according to the population of patients in an intensive care unit, duration of hospital stay, and prior antimicrobial therapy. Because appropriate antimicrobial treatment of patients with VAP significantly improves outcome, more rapid identification of infected patients and accurate selection of antimicrobial agents represent important clinical goals[7].

Organisms causing ventilator-associated pneumonia generally fall into two groups: those causing early-onset ventilator-associated pneumonia (<4 days of mechanical ventilation) and those causing late-onset ventilator-associated pneumonia (≥4 days of mechanical ventilation) [3].

The most widely studied preventive strategies have focused on the prevention of oropharyngeal or gastric colonization and the prevention of aspiration of contaminated oropharyngeal or gastric secretions[8]. This evidence-based systematic review aims to identify interventions for the prevention of ventilator-associated pneumonia, critically evaluate their efficacy and adverse effects, and recommend an approach to their use.

The diagnosis of VAP is usually based on three components: systemic signs of infection, new or worsening infiltrates seen on the chest roentgenogram, and bacteriologic evidence of pulmonary parenchymal infection. The systemic signs of infection, such as fever, tachycardia, and leukocytosis, are nonspecific findings and can be caused by any condition that releases cytokines[9].

Any ventilated patient who developed clinical pneumonia along with culture positive aspirated tracheal fluid constituted VAP. Clinical pneumonia was defined as inflammation of one or both lungs with consolidation which is frequently but not always due to infection. The infection may be bacterial, viral, fungal or parasitic. Symptoms may include fever, chills, cough with sputum production, chest pain, and shortness of breath.

Aim of our study was to identify the organisms associated with Ventilator Associated Pneumonia (VAP) and to compare our study with other international studies.

**Materials and Methods:**

This observational study on ventilator associated pneumonia (VAP) was conducted at Square Hospitals Ltd, Dhaka during the period of April 2007 to March 2009. Patients who developed other infections along with VAP and patients who underwent tracheostomy were excluded from the study.

Any ventilated patient who developed fever or respiratory distress was clinically assessed by attending physician. This includes time of developing fever after intubation, Physical examination findings (auscultation findings, pattern of temperature), and chest X-ray findings. On the day of onset of fever or respiratory distress tracheal aspiration was done and the aspirated fluid was sent to microbiology laboratory for culture and sensitivity test. If bacteria were isolated in culture, the patient was evaluated to asses whether the patient fell under case definition of VAP. If patient was designated to be suffering from VAP, all relevant information was documented in a structured questionnaire.

At the end of the data collection period, all the questionnaires were complied and a master sheet was made.

**Results**

This is an observational analytical cross-sectional study. All the patients diagnosed as VAP was included in this study. Total number of cases was 39. Among these patients 11 patients expired which was 28% of the whole series. Study period was 2 years.

Among the 39 VAP patients 21 patients (54%) were male and 18 (46%) patients were female. Male : Female ratio was 1.2 : 1.

**Table-I**

*Age distribution of VAP patients (n=39).*

| Age (years) | Number | Percentage | Mean age (years) |
|---|---|---|---|
| ≥30 | 04 | 10.2 | |
| 31-40 | 02 | 5.1 | |
| 41-50 | 06 | 15.4 | 60.17 |
| 51-60 | 04 | 10.2 | |
| 61-70 | 09 | 23.1 | |
| >70 | 14 | 35.9 | |
| Total | 39 | 100 | |

Table -I shows the distribution of VAP patients according to their age frequency. Highest number of patients (36%) was in more then 70 years age group. Second highest (23%) was 61 to 70 years age group. Patients of 41-50 years and 51-60 years age groups were 15.4% and 10.2%. We had 4 patients (10.2%) in below 30 years age group and only 2 patients (5.1%) in 31-40 years age group.

**Table-II**

*Age distribution of expired VAP patients (n=11)*

| Age (years) | Number | Percentage |
|---|---|---|
| ≥ 30 | 1 | 9 |
| 31-40 | 1 | 9 |
| 41-50 | 1 | 9 |
| 51-60 | 1 | 9 |
| 61-70 | 3 | 27 |
| >70 | 4 | 36 |
| Total | 11 | 100 |

Table-II demonstrates the distribution of age among the patients who died of VAP. Age of highest number of patients (36%) was more then 70 years. Three patients (27%) were in 61-70 years age group. Number of expired patients were one each in rest of the age groups.

**Table-III**

*Distribution of organisms among VAP cases (n=39)*

| Sl. | Organism | Number | Percentage |
|---|---|---|---|
| 1. | Pseudomonas spp. | 14 | 35.9 |
| 2. | Acinatobacter spp. | 11 | 28.2 |
| 3. | Klebsiella spp. | 06 | 15.4 |
| 4. | MSSA | 04 | 10.3 |
| 5. | MRSA | 03 | 7.7 |
| 6. | Group D non enterococci | 02 | 5.2 |
| 7. | Candida | 02 | 5.2 |
| 8. | CONS | 02 | 5.2 |
| 9. | E coli | 02 | 5.2 |
| Total | | 39 | 100% |

MSSA – Methicillin Sensitive staphyloccus Aureus

MRSA = Methicilhin Resistant Staphylococcus Aureus

CONS = Coagulase Negative Staphylococcus species

In our study we found 9 organisms responsible for VAP. Single organism was isolated in 32 cases. Other 7 patients had more then 1 organism. Table-III has tabulated the list of isolated organisms in VAP cases. Pseudomonas spp. was responsible for more then one third cases (14 cases). Acinatobacter spp. was isolated in 11 cases (28%). We found Klebsiella spp. in 6 cases (15.4%). Methicillin Sensitive Staphylococcus Aureus (MSSA) and Methicillin Resistant Staphylococcus Aureus (MRSA) were found respectively in 04 and 03 cases. Group D non enterococci, Candida, Coagulase Negative Staphylococcus species (CONS) and E.coli were present in 2 cases (5.2%) each.

**Table-IV**

*Distribution of organisms among expired VAP cases (n=11).*

| Sl. | Name of the organism | Number | Percentage |
|---|---|---|---|
| 1 | Pseudomonus spp. | 05 | 45.5 |
| 2 | Klebsiella | 04 | 36.4 |
| 3 | Acinatobacter spp. | 02 | 18.2 |
| 4 | Candida | 02 | 18.2 |
| 5 | E coli | 01 | 9.1 |
| 6 | MRSA | 01 | 9.1 |

Table-IV shows the distribution of organisms among expired VAP cases. Four patients had double organisms responsible for VAP. In near about half (45.5%) of the cases Pseudomonas spp. was responsible for VAP. Klebsiella was isolated approximately in one third (36%) of the patients. Acinatobacter spp. was found in 18.2% (02 cases) of cases. Number of affected cases by Candida was 2. E coli and Methicillin Resistant Staphylococcus Aureus (MRSA) was responsible for 1 case each.

Mortality was 28.2% in this series.

**Discussion**

This study was performed on “Analytic study on Ventilator Associated Pneumonia (VAP) in Neurosurgery Intensive Care Unit (NICU)”. This was an observational analytic cross sectional study. Total number of patients was 39. Main objective of our study was to identify the organisms associated with VAP (Ventilator Associated Pneumonia) in Neurosurgery intensive care unit (NICU). All patients were diagnosed on the basis of their clinical features, radiological findings and tracheal swab culture and sensitivity. Age, sex, time of onset of VAP after intubation, primary diagnosis of patients, isolated organisms and clinical status at the time of discharge was documented as variable in data collection sheet. After collection of data we have tabulated the result and compared with that of international studies.

Kimberly et al. found mortality of VAP in between 20 - 50% in their study[9]. In this study mortality rate was 28.2%.

Marin et al. found common responsible organisms were MRSA (14.8%), *Staphylococcus aureas* (14.8%), *Pseudomonas aeruginosa* (14.3%) and other *Staphylococcus* species (8.8%) in their study[10]. In this study *Pseudomonas spp.* was found commonest (36%) followed by A*cinatobacter spp.* (28%) and *Klebsiella* (15.4%). Only 7.7% cases were found caused pneumonia by MRSA. Overall mortality was 25.1% in their series which was 28.2% in this series.

According to the study of Chastre J et al. mortality was 24 to 50% for VAP in their

series. Isolated organisms mostly were *Staphylococcus aureus*, *Pseudomonas aeruginosa* and *Enterobacteriaceae*[6]. Mortality was 28.2% in this study. *Staphylococcus aureus* (10.3%) and *Pseudomonas aeruginosa* (36%) were also common in this series.

Shaw et al. found *Staphylococcus aureus* as commonest organism in VAP cases of their series[11]. Other common organisms were *Pseudomonas aeruginosa* and *Acinobacter baumanii*. In our study commonest organism was *Pseudomonas aeruginosa* (36%) followed by A*cinatobacter* (28%) and *Klebsiella* (15.4%).

Mortality of VAP patients was 37% in the study of Rakshit et al. In their study male was 56.9% and female was 43.1%, mortality was 28.2%[12]. Rakshit et al. found that the commonest organism is Pseudomonas *aeruginosa* in their study followed by *Klebsiella pneumonae*. *Pseudomonas spp.* was also found commonest organism in this series.

Talha et al. found 25.6 % mortality in their series [14]. Pseudomonas was commonest in their study followed by Staphylococcus aureas. Highest number of VAP patient was from Neurosurgery department in that study. Mortality was 28.2% in this study and Pseudomonas was the commonest organism.

When results of this study were compared with those of international studies similarity was found in organisms, primary diagnosis and mortality.

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## CASE REPORTS

# Left CP Angle Liponeurocytoma - A Case Study

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**Abstract**

*The term "liponeurocytoma", recently adopted by the World Health Organization Working Group (WHO), replaced many other different terms used up to now to give name to this rare tumour. To our knowledge, less than 20 cases have been related up to now under different names like as lipomatous medulloblastoma, lipidized medulloblastoma, neurolipocytoma, medullocytoma and lipomatous glioneurocytoma. The new nomenclature eliminates the word "medulloblastoma", reinforces its benign character, and includes it in the category of glioneuronal tumours. We describe an additional case of this distinct clinico-pathological entity removed from the left CP angle of a 55-year-old male. With the present case report, we hope to contribute to the knowledge on the diagnostic and prognostic implications derived from the finding of mature adipose-like tissue within a medulloblastoma tumour.*

***Key words:*** *Liponeurocytoma, medulloblastoma, adipose tissue, mixed tumour.*

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## Introduction

Medulloblastoma is the most common primitive neuroectodermal tumour of the central nervous system. It occurs usually in childhood in the first decade of life[1], being also found in adults[2-4]. According to World Health Organization (WHO) it is considered a grade IV neoplasm[5]. Its heterogeneity as well as its potential to differentiate has been largely related in the literature, happening in 40% of cases[6]. Cartilaginous, rhabdomyoblastic and neuroglial variants have been reported[7-9]. The uncommon finding of adipose tissue in medulloblastomatous neoplasms has been related rarely and correlates with better prognosis[10-15]. This kind of tumour was recently included in the WHO working group for classification of central nervous system neoplasms under the name lipone-urocytoma[5]. The term adopted omits the word medulloblastoma clearly pointing to a better prognosis.

We describe here such a particular left CP angle neoplasm in a 55-year-old male, especially composed of mature adipose tissue closely admixed with undifferentiated small blue cell areas consistent with medulloblastoma.

## Case report

A 55-year-old male, developed a progressive occipital headache, vertigo, vomiting & ataxia. On neurological examination left sided cerebellar sign was positive. Plain and contrast MRI shows heterogeneous enhancing mass in the left CP angle region, displacing 4th ventricle and causing hydrocephalus. Surgical exploration with total resection of the lesion done.

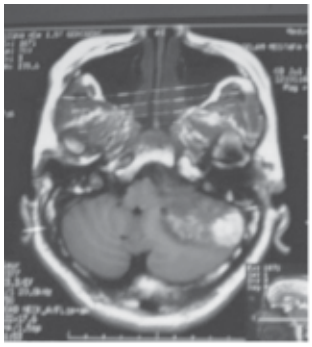

Image 1: Axial-T1WI

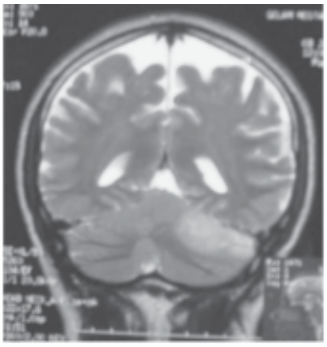

Image 2: Coronal-T2WI

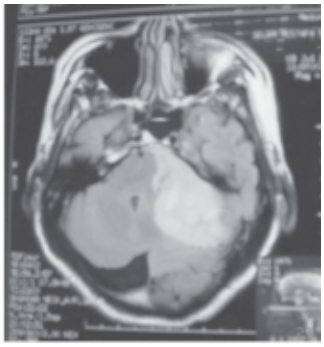

Image 3: T2FLAIR

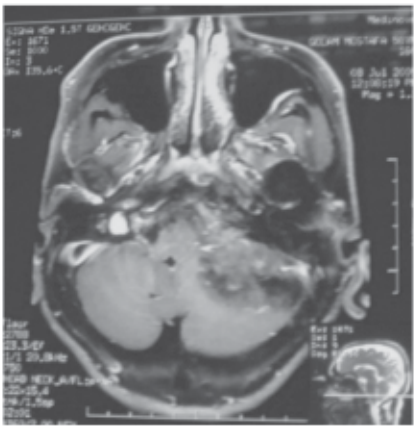

Image 4: Contrast T1WI

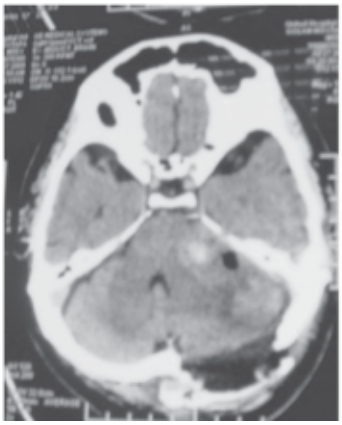

Image 5: Axial CT post-contrast

Postoperative cranial radiotherapy (5600 rads) was given. The surgically removed tumour fragments were send to the laboratory fixed in 10% formalin. They were processed for paraffin sectioning and stained with hematoxylin eosin (H&E), periodic acid-Schiff (PAS), Gomori, Fontana-Masson and reticulin.

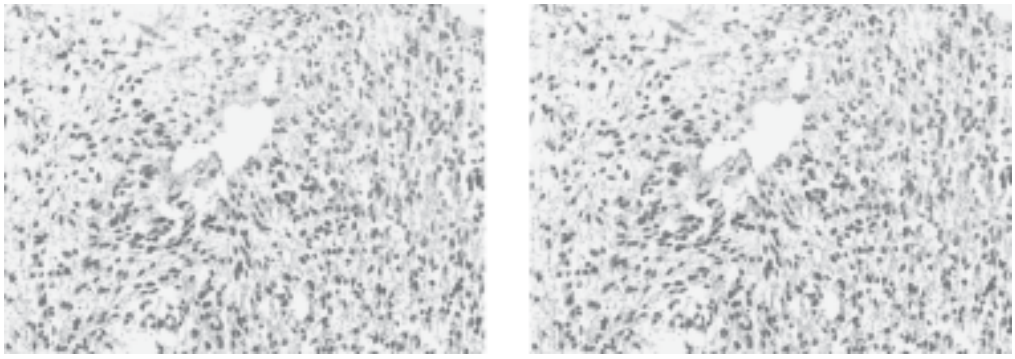

**Fig.-1:** *High power, H&E*

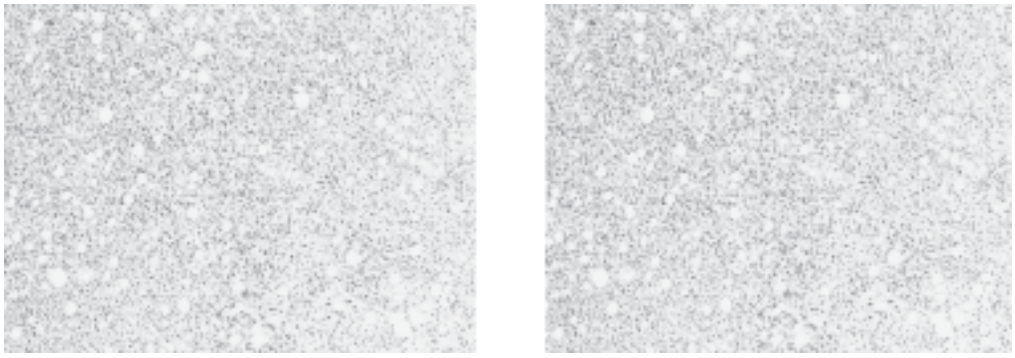

**Fig.-2:** *Low power, H& E*

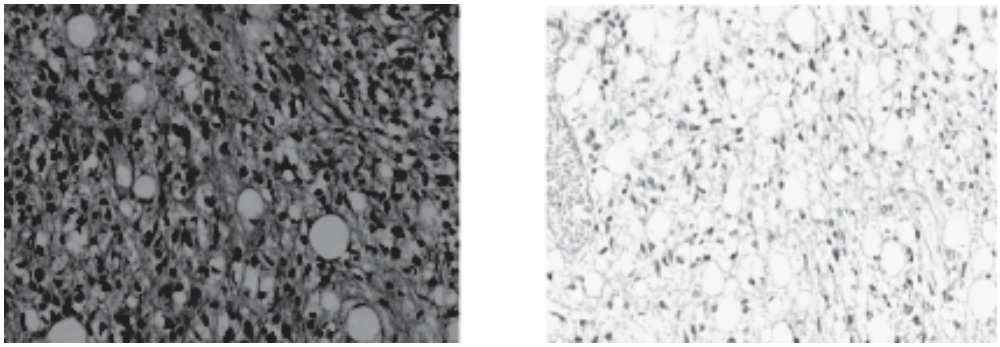

**Fig.-3:** *High power, H& E*

Immunohistochemical staining advocated but patient denied due to financial constrain.

**Pathologic findings-** Microscopically a neoplasm consisting of two distinct cellular elements were observed: They were 1) a predominant poorly differentiated component of small cells and 2) intermixed with another element indistinguishable from mature fat cells at optic microscopy examination. No distinct separation was observed between these two elements. The small cell component disclosed scanty cytoplasm and round or oval nuclei arranged in a fibrillary background. The lipomatous component was composed of grouped vacuolated cells indistinguishable from mature fat cells. Other tumour areas contained smaller vacuoles recalling an apparent transition between the two populations. In the lipomatous component there was no cellular immaturity or nuclear atypia. Only few mitosis were found in poorly differentiated areas. Recent hemorrhage as well as necrotic areas were also observed in both components. The non-tumoural cerebellar tissue circumjacent to tumour was devoid of fat cells, disclosing only hypoxic cellular alterations.

**Discussion**

Medulloblastoma, a malignant cerebellar tumour of childhood, is heterogeneous in regard to tissue pattern. Its potential of differentiation has been largely reported in

the literature, including neuronal, glial and mesenchymal elements[6-8]. Authours have attempted to correlate some histological characteristics with prognosis and this has lead, sometimes, to different conclusions[4]. We report a case that represents a rare example of cerebellar tumour in adult, exhibiting an extraordinary morphology characterized by an uncommon mixture of mature adipose tissue with medulloblastomatous areas. The presence of adipose cells in neuroglial neoplasms has been exceptionally related in the literature not being restricted to cerebellum neither to medulloblastomas[16]. It has also been described in spinal cord neoplasms[17] and in supratentorial ependymomas[18].

The origin of these adipocytic elements is still disputed and it hasn't yet been completely elucidated. Some authours consider that the presence of such cells suggests that the neoplasm has evolved in dysgenetic areas where distinct cellular elements co-exist intimately[9].

Some others have found ultrastructural evidences that there is a progressive accumulation of lipid vacuoles in the cell's cytoplasm, probably due to some degenerative or metabolic change. So, those cells would become similar to adipocytes at optic microscopy. Analyzed under immunohistochemistry their neuroglial origin manifest themselves[18]. This explains the immunoreactivity in the remaining cytoplasm ring related by some authors[14,15,18]. What mechanism was in action in these tumours is still under consideration.

Apart from histopathological considerations, it should be pointed out that this variant, characterized by adult onset, shows a much more benign evolution.

All these observations strongly suggest it is a classic pathological entity distinct from its conventional counter part.

For the last few years, many different terms have been proposed to designate these tumours, such as lipomatous medulloblastoma[15], lipidized medulloblastoma[14], and medullocytoma[13]. Most of these terms clearly point to a more benign lesion than classical medulloblastoma. In accordance to a recent publication of the WHO working group on "Classification of Tumors of the Nervous System", they were included in the category of mixed glioneuronal tumours. The term "Cerebellar Liponeurocytoma" has been proposed and should be used from now on[5]. Bearing in mind that this kind of lesion is not restricted to cerebellum[16], may be the word "cerebellar" should have been omitted.

Analyzing the postoperative approach in published cases up to now, we observed that there is no consensus about the use of complementary radiotherapy[19]. In the majority of cases that had evidence of residual tumour, patients were submitted to radiotherapy[19,20]. Despite the presumed good prognosis, in one single case tumour behaved in a more aggressive way[20]. In our case, despite the presence of large necrotic areas, factor known to be associated with a poor prognosis, the patient is doing well almost four years after surgery and postoperative radiotherapy.

**Conclusion**

We really believe the ideal post-operative conduct is still to be determined. The present report represents another step to determine whether the presence of lipomatous cells in medulloblastomatous tumours does, in fact, have a prognostic value, distinguishing them from conventional medulloblastomas.

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# Extracranial-Intracranial By-pass (EC/IC) for Symptomatic Occlusive Cerebrovascular Disease Not Amenable to Carotid Endarterectomy - A Case Report

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**Abstract:**

*EC/IC by-pass surgery was expected to solve all types of cerebral ischemia with angiographic demonstration of any degree of stenosis or occlusion where end arterectomy was not applicable. The purpose of extracranial to intracranial (EC-IC) by-pass is to augment cerebral blood flow. A 35 years old right handed young solder underwent EC/IC by-pass surgery for atherosclerotic occlusion of left ICA and MCA. It was carried out successfully with significant improvement of motor power and dysphasia.*

**Introduction:**

EC-IC bypass has been carried out as treatment for stroke for past 30 years[1]**.** The purpose of extracranial to intracranial (EC-IC) bypass is to augment cerebral blood flow[1-4.]. This procedure entails connection of the superficial temporal artery (STA), or a venous conduit, to a branch of the middle cerebral artery (MCA). Carotid end arterectomy is an alternative established and effective procedure for stroke prevention in severe stenotic lesions of the carotid artery in the neck. This procedure is not feasible for 20-30 percent of occlusive disease. These includes total occlusion of carotid above the level of the mandible and occlusion of the middle cerebral artery. The first EC-IC bypass was performed by Yasargil, in Zurich, Switzerland, in 1967.[1] Following in his footsteps, other talented cerebrovascular surgeons in the United States adopted the procedure during the 1970's, and it's use expanded to most major neurosurgical centers around the world[1,2]. Yasargil showed that anastomosis of the superficial temporal artery to a small cortical branch of middle cerebral artery was feasible[1]. The indications for EC-IC by-pass are severe stenosis or occlusion of intracranial arteries (Fig-1) with focal neurological symptoms, such as weakness or speech difficulties. This procedure is also used when an artery must be surgically occluded for the treatment of unclippable giant aneurysms[3,4.]. In children, this procedure is used to treat Moya-moya disease (a progressive narrowing of proximal intracranial blood vassels[4].

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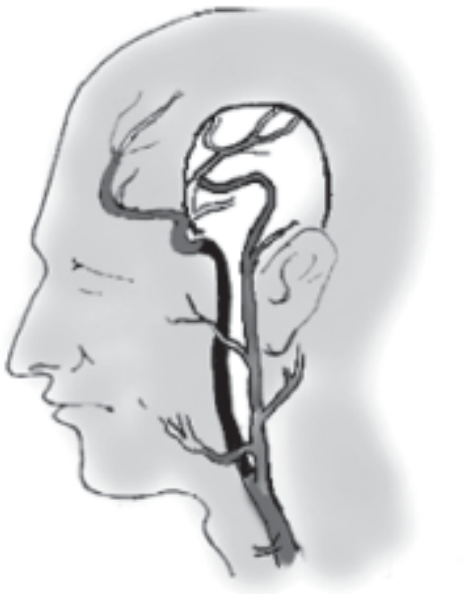

**Fig.-1:** *Diagramatic view of occlusion of ICA and EC/IC by-pass.*

**Case Report:**

A 35 years old right handed, nondiabetic, normotensive, nonasthmatic man got admitted in neurosurgery department of BSMMU with the complaints of headache, right sided weakness for 6 months, slurring of speech for 4 months and recurrent episodes of unconciousness for same duration. Headache was moderate to severe, dull aching in nature, located whole over the head specially left parietal region and relieved on taking rest and in lying position. He had no history of trauma. He gave history of episodes of unconciousness which persist for 2-3 min associated with generalized tonic clonic convulsion, occasionally associated with tongue bite and bladder bowel incontinence. He also gave h/o memory loss in the form of short term as well as long term memory. All the findings of the general examinations were within normal limit.

His nervous system examination revealed- Higher psychic function – normal except short term and long term memory loss. Speech was slurred, Gait was normal. Muscle power of right sided upper and lower limbs was grade 4, sensory functions were intact. Jerks were normal. All the cranial nerves were functionally intact. There were no bowel and bladder incontinence.

Routine blood investigations were within normal limit. X- ray of the skull was normal. CT scan of brain showed ischemic infarct of fronto – parietal region (Fig-2). CT angiogram of the brain showed complete occlusion of left internal carotid artery above the neck (Fig-3) and left MCA is partially visualized from contralateral flow (Fig-3). Transcranial color doppler study showed reduced perfusion to the left hemisphere than right (Fig-4) (Table-I).

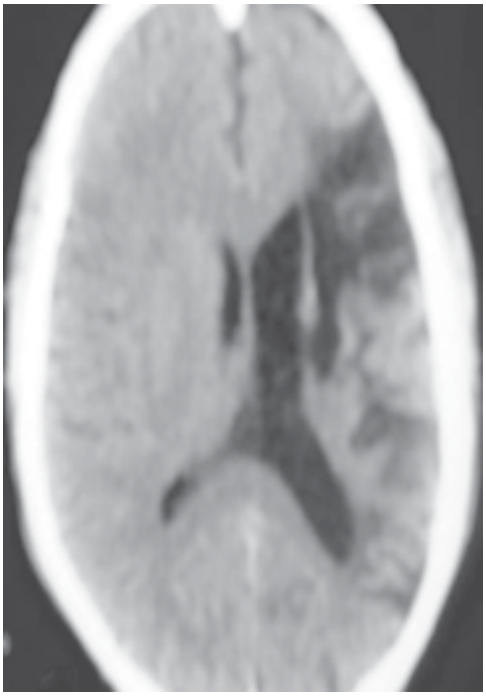

**Fig.-2:** *CT scan of brain revealed infarct of left fronto-parietal lobe.*

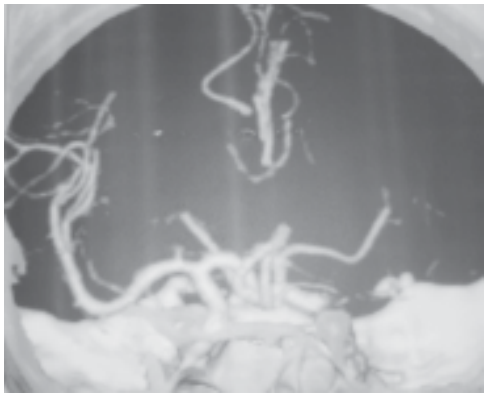

**Fig.-3:** *CT Angiogram shows the total occlusion of left ICA and stenosis and partial visualisation of left MCA by contralateral flow.*

Preoperative data:

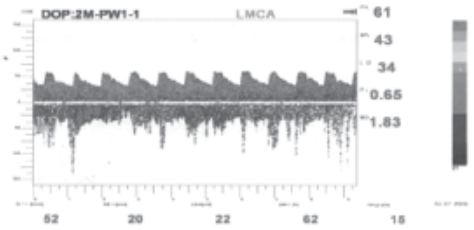


**Fig.-4:** *Preoperative MCA waveform shows reduced flow.*

**Table-I**

*Data analysis of all the major intracranial vessels shows reduced flow over left MCA, reversed flow over left ACA, no flow over the left ICA*

TCD data: (In the table, the unit of PK, MN and E.D. is cm/s Depth's unit is mm, others have no unit.)

| Vessel | DEPTH | PK | MN | E.D. | P.I. | R.I. | SBI | SD | Direction |
|---|---|---|---|---|---|---|---|---|---|
| L MCA | 52 | 81 | 56 | 44 | 0.88 | 0.48 | 0.22 | 1.84 | Toward |
| RMCA | 52 | 106 | 81 | 69 | 0.46 | 0.35 | 0.34 | 1.55 | Toward |
| LACA | 62 | 53 | 32 | 22 | 0.97 | 0.59 | 0.56 | 2.44 | Backward |
| RACA | 62 | 78 | 56 | 45 | 0.58 | 0.42 | 0.39 | 1.72 | Backward |
| LPCA | 67 | 70 | 46 | 35 | 0.75 | 0.50 | 0.49 | 2.01 | Toward |
| RPCA | 67 | 53 | 34 | 24 | 0.86 | 0.55 | 0.55 | 2.21 | Toward |
| LVA | 62 | 41 | 29 | 23 | 0.61 | 0.43 | 0.43 | 1.77 | Backward |
| RVA | 62 | 45 | 29 | 21 | 0.84 | 0.54 | 0.49 | 2.17 | Backward |
| BA | 75 | 47 | 33 | 27 | 0.61 | 0.43 | 0.40 | 1.77 | Backward |
| LOA | 47 | 39 | 25 | 17 | 0.89 | 0.56 | 0.54 | 2.27 | Toward |
| ROA | 47 | 21 | 15 | 12 | 0.62 | 0.44 | 0.40 | 1.78 | Toward |
| LCCA | 0 | 21 | 13 | 10 | 0.83 | 0.54 | 0.49 | 2.15 | Toward |
| RCCA | 0 | 33 | 14 | 4 | 2.09 | 0.87 | 0.71 | 7.93 | Toward |
| LECA | 0 | 29 | 14 | 6 | 1.62 | 0.78 | 0.61 | 4.50 | Backward |
| RECA | 0 | 67 | 34 | 18 | 1.45 | 0.74 | 0.63 | 3.82 | Backward |
| LICA | 0 | 24 | 11 | 4 | 1.81 | 0.82 | 0.78 | 5.53 | Backward |
| RICA | 0 | 47 | 31 | 24 | 0.74 | 0.49 | 0.45 | 1.98 | Backward |

**Table-II**

*Postoperative doppler data analysis revealed blood flow was increased in the left MCA in comparison to previous findings following EC/IC Bypass.*

**TCD data:**

(In the table, the unit of PK, MN and E.D. is cm/s. Depth's unit is mm, others have no unit.)

| Vessel | DEPTH | PK | MN | E.D. | P.I. | R.I. | SBI | SD | Direction |
|---|---|---|---|---|---|---|---|---|---|
| LMCA | 52 | 61 | 43 | 34 | 0.65 | 0.45 | 0.45 | 1.83 | Toward |
| RMCA | 52 | 86 | 60 | 46 | 0.66 | 0.46 | 0.46 | 1.85 | Toward |
| LACA | 65 | 106 | 74 | 58 | 0.65 | 0.45_ -. | 0.05. | 1.83_ | Toward |
| RACA | 62 | 102 | 71 | 55 | 0.66 | 0.46 | 0.03 | -1.84 | Backward |
| LPCA | 67 | 72 | 51 | 40 | 0.64 | 0.45 | 0.44 | 1.82 | Toward |
| RPCA | 67 | 53 | 35 | 27 | 0.73 | 0.49 | 0.47 | 1.96 | Toward |
| LVA | 64 | 32 | 22 | 18 | 0.64 | 0.45 | 0.43 | 1.81 | Backward |
| RVA | 62 | 41 | 29 | 23 | 0.63 | 0.44 | 0.43 | 1.80 | Backward |
| BA | 75 | 48 | 34 | 26 | 0.84 | 0.45 | 0.44 | 1.81 | Backward |
| LOA | 48 | 24 | 14 | 8 | 1.15 | 0.65 | 0.63 | 2.88 | Toward |
| ROA | 47 | 33 | 19 | 13 | 1.06 | 0.62 | 0.59 | 2.65 | Toward |
| LECA | 0 | 39 | 19 | 9 | 1.52 | 0.767 | 0.62 | 4.09 | Backward |
| RECA | 0 | 36 | 16 | 7 | 1.63 | 0.78 | 0.69 | 4.56 | Backward |
| LICA | 0 | 19 | 8 | 3 | 1.85 | 0.83 | 0.71 | 5.82 | Backward |
| RICA | 0 | 76 | 53 | 42 | 0.65 | 0.46 | 0.38 | 1.84 | Backward |

Postoperative data:

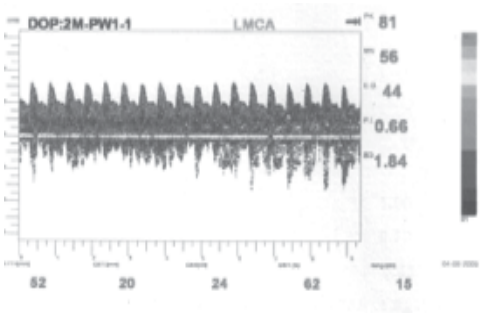


**Fig.-5:** Postoperative doppler shows the peak flow wave following EC/IC Bpass.

**Technique**

Left sided pterional craniotomy was performed to expose the intracranial vessels . The donor artery was exposed from scalp. This was performed under the operating microscope. First we identified and dissected the superficial temporal artery. Then we selected parietal branch of STA. Transsylvian approach was performed by pterional craniotomy. After gentle dissection of sylvian fissure, MCA and its bifurcation were identified. Two temporary aneurysm clip were applied in the trunk of M2 before

its division. A small hole was made along it's long axis. After that we mobilize the donar artery near to the M2. Then end to side anastomosis was performed by 7/0 prolene. Temorary clips were removed. A key hole was maintained in the dural flap and bone to prevent the collapsing of the donar artery.Finally haemostasis was secured and closed in layers. Postoperative recovery was uneventful. After 2 weeks of bypass, we did transcranial color doppler and it shows increased blood flow to the MCA and hence to left cerebal hemisphere (Table-2) and (Fig-5). Patient's motor power has improved and speech was also became normal (Fig -6).

**Fig 6:** *postoperative picture of the patient shows almost equal motor power in both hands.*

**Discussion:**

A brain by-pass is equivalent with heart by-pass. It reinitiates blood flow in a blocked or damaged or an abnormal blood vessel. As a result corresponding brain region will get adequate blood supply. As it is formed from out side to inside head it is known as Extracranial to Intracranial (EC-IC) by-pass[5]. In patients with ICA occlusion and reduced cerebrovascular reserve capacity (CVR), revealed by different technical modalities such as MRI, PET, the EC-IC by-pass can apparently prevent recurrent ischemic attacks[5,6].

There are several studies available which suggest hemodynamic failure can be predicted in patients with occlusive cerebrovascular disease, or those prior to therapeutic carotid occlusion, using SPECT scanning, Xenon CT scanning or positron emission tomography (PET)[7]. More importantly, it has been shown that EC-IC by-pass has the potential to reverse the hemodynamic failure and normalize cerebral blood flow (CBF)[7,8].

Grubb et al., have recently demonstrated that patients with an occluded ICA and ipsilateral increased oxygen extraction fraction (the fraction of oxygen in the blood that the brain extracts to maintain metabolism) by PET scanning have a significantly increased risk of future stroke[7.]This patient population for instance, may be better served by EC-IC by-pass[7-9]. We feel EC-IC by-pass has a definite role in the treatment of ischemic stroke in carefully selected patients fulfilling very strict criteria. Brain by-pass is carried out for two main reasons:

1. Symptomatic blockage or occlusion or traumatic injury of major brain artery such as the internal carotid artery or middle cerebral artery.
2. A brain aneurysm that cannot be obligated successfully using a clip or coil but instead its parent artery require to be sacrificed for the aneurysm to be effectively treated[8,9].

**Conclusion:**

The EC-IC bypass is well planned methodologically sound surgery. STA-MCA by-pass has a definitive role in the treatment of ischemic stroke.

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# Adult Stem Cells for Spinal Muscular Atrophy

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**Abstract**

*Spinal muscular atrophy (SMA) is a genetic disease that attacks the motor neurons of the spinal cord. This results in the progressive wasting of the voluntary muscles of the limbs and trunk. We present a 26 year old girl diagnosed with SMA since last ten years. The diagnosis was confirmed on the basis of muscle biopsy and electromyography (EMG) showing anterior horn cell involvement. In view of the same; the patient was given intrathecal autologous bone marrow derived stem cell therapy as part of the neuron regenerative rehabilitation therapy (NRRT) protocol. The patient showed functional improvements in her disability post therapy. A detailed case report is presented here with.*

***Key words:*** *Spinal Muscular Atrophy, autosomal, motor neurons, bone marrow, neuroregeneration.*

## Introduction

Spinal Muscular Atrophy is a recessive autosomal disorder caused by mutations of the survival motor neuron *(SMNI)* gene located on chromosome 5q13, resulting in a marked reduction in SMN protein and characterized by degeneration of motor neurons associated with muscle paralysis[1-2]. Although SMN is a ubiquitously expressed protein, mutations, in SMN specifically cause the targeted deterioration of motor neurons of the spinal cord[3]. The clinical features of SMA result from skeletal muscle denervation[4], loss of anterior horn cells in the spinal cord and cranial nerve nuclei[5] The incidence of spinal muscular atrophy is about 1 in 10,000 live births with a carrier frequency of 1 in 50. The disorder can be subdivided into three groups according to age of onset, the severity of symptoms (assessed by the achievement of motor milestones) and age at death[6-9] SMA type I and II are severe forms of SMA with an early onset while SMA type III (Kugelberg-Welander disease) is the mildest form of the disorder with a late onset. In order to be diagnosed with Spinal Muscular Atrophy, symptoms need to be present. In most cases a diagnosis can be made by the SMN gene test, which determines whether there is at least one copy of the SMNI gene by looking for its unique sequences (that distinguish it from the almost identical SMN2) in exons 7 and 8. In some cases, when the SMN gene test is not possible or does not show any abnormality, other tests such as an EMG or muscle biopsy may be indicated.

In spite of worldwide efforts, there is as yet no cure for SMA. Principal approaches to

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findout an effective treatment or indeed a cure have been aimed at either manipulating the genetic material implicated in the pathophysiology of SMA, or at restoring the lost or damaged motor neurons via cellular replacement[10-12]. Autologous hematopoetic stem cell transplantation (HSCT) has been used as a treatment for such diseases along with Physiotherapy and Occupational therapy. Before the discovery of Adult Stem Cells, Spinal Muscular Atrophy (SMA) had no cure or treatment. Normally the only thing an SMA patient can do is try to keep the status quo and try to prevent it from getting worse. However, thanks to great advances made in Adult Stem Cell research, Spinal Muscular Atrophy is now able to be improved. The case we are presenting is a good example of this. She presented with tongue fasciculations, respiratory muscle fatigue and lordotic gait with predominant abdominal weakness. The muscle biopsy and EMG suggested the diagnosis of Spinal Muscular Atrophy with anterior horn cell involvement.

**Case report**

We present a 26 year old girl suffering with Spinal Muscular Atrophy with a history of frequent falls while walking and progressive left extremity weakness and difficulty while getting up from squatting position. She had tongue fasciculations and respiratory muscle fatigue. She had a lordotic gait and predominant abdominal muscle weakness. She also had a difficulty in overhead activities and occasional misarticulation of few words was also observed. Her genetic tests did not show any abnormality. Hence, her Muscle Biopsy and EMG were carried out. The Quadriceps Muscle Biopsy indicated Denervation Atrophy from spinal origin and EMG showed very chronic motor axon degeneration affecting all the four limbs predominantly proximal muscles. Lower limb proximal muscles and trunk muscles were most affected. The site of involvement was the Anterior Horn Cell level which indicated Spinal Muscular Atrophy.

**Materials and Methods:**

NRRT Protocol: Patients selection and protocol design has been based on the inclusion criterion as per paragraph 32 of the World Medical Associations Helsinki declaration[13]. The protocol had been reviewed and approved by the Institutional committee for Stem cell Research and therapy (IC-SRT). The patient' was informed about the procedure and a duly filled informed consent form was obtained from her. Blood Tests, MRI were performed one week before the transplantation. The investigations were repeated prior to the second transplant as well. G-CSF injections were administrated 48 hours and 24 hours before Bone Marrow derived Stem Cell Transplantation. Autologous bone marrow derived MNCs were transplanted according to the NRRT protocol. Bone marrow (100ml) was aspirated from the iliac bone. Mononucleocytes (MNC) were obtained after density gradient separation. Viable count of the isolated MNCs was taken. The MNCS were checked for CD34+ by FACS analysis. Approximately 64 x $10^6$ MNC were immediately injected post separation, intrathecally in L4-L5 intervertebral space using a lumbar puncture needle and catheter. Along with Neuroregeneration, Neuro Rehabilitation, Physical Therapy and Occupational Therapy were also given to the patient. This therapy emphasizes the role of stem cells

in taking advantage of the brain's capacity for repair & recovery. Rehabilitation interventions seek to promote recovery & independence through neurofacilitation.

**Observation:**

*Clinical improvements*

After the stem cell therapy, the patient had no side effects and was evaluated at a regular time period. On follow up, improvement was observed in the strength of the upper extremity. Her hand grip had improved significantly bilaterally. Her stamina had improved along with the sitting balance. Improvement was observed in her walking and frequency of falling had decreased. She could now climb stairs better with minimal support. Her speech had also improved significantly. Her over all muscle strength had improved. She has become more confident in doing her daily activities. According to her the quality of life has improved significantly.

**Discussion:**

Spinal Muscular Atrophy (SMA) is a neuromuscular disease characterized by degeneration of motor neurons[14], resulting in progressive muscular atrophy (wasting away) and weakness. The clinical spectrum of SMA ranges from early infant death to normal adult life with only mild weakness. In all of its forms, the primary feature of SMA is muscle weakness, accompanied by atrophy of muscle. This is the result of denervation, or loss of the signal to contract, that is transmitted from the spinal cord. This is normally transmitted from motor neurons in the spinal cord to muscle via the motor neurons axon, but either the motor neuron with its axon, or the axon itself, is lost in all forms of SMA.

Since, many potential therapies have not been successful in treating the disorder, Adult Stem Cell (Autologous bone marrow derived MNC's) transplantation was carried out. The adult bone marrow stem cells per se have no side effects and are very safe. This treatment showed significant improvements in the case. She could walk better and her muscle strength had improved significantly. She was advised to do physical therapy to enhance the benefits of the stem cells even more. While the stem cells may not have cured her completely, they have definitely helped and hopefully she is now on the road to a full recovery.

The present data provide clear evidence that Autologous Hematopoietic Stem Cell Transplantation along with Neuro-rehabilitation can result in significant improvements in the case of Spinal Muscular Atrophy with no side effects.

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