*Original Article*

# Role of Dexamethasone in the Management of Acute Ischaemic Stroke in a Tertiary Hospital: A Randomized Clinical Study

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**Abstract:** Bangladesh is a country of large population, a number of preventable deaths occur due to stroke in this country. The mortality rate due to stroke increased in recent years. Individuals in Bangladesh aged more than 40 years or more have increased rate of stroke prevalence. The majority of stroke patients are suffering from ischaemic stroke. Brain oedema is the usual accompaniment around the infarct or haemorrhage. It develops around the perilesional area within few hours and gets maximal within 2-4 days and then decreases to resolve usually within 2 weeks. This study determined the clinical outcome after using of injectable Dexamethasone as a treatment option in the management of acute cerebral ischaemic stroke. It is a randomized prospective clinical trial, a total of N=110 patients with acute ischaemic stroke, excluding those with having clinical suspicion of acute ischaemic stroke but no radiological evidence, Computed Tomography (CT) scan showing haemorrhage in the brain, patients of diabetes mellitus and H/O peptic ulcer disease, decrease level of consciousness due to other causes i.e. hyponatraemia, diabetic ketoacidosis, infections etc. and including those above 30 years of age admitted in Medicine units of Sher-E-Bangla Medical College Hospital, Barisal on the basis of consecutive sampling technique for the study. Following standard procedures and protocols, after taking history, doing physical examination, calculating Glasgow Coma Scale score and taking opinion about Computed Tomography (CT) Scan of brain the study was done. After getting written and informed consent two groups were selected. Group A were treated with injectable Dexamethasone and other supportive drugs and group B were treated without injectable Dexamethasone, then regular follow up was taken clinically with Glasgow Coma Score in every 1st, 3rd and 5th day after treatment. Case record form was filled in at hospital. Out of 110 patients, 70 were females and 40 males. Mean age at admission was 54.35± 8.029 years. In this study, group-A patients were 55 (50%) and group-B patients were 55(50%) in number which were randomly selected. During hospitalization, mean Glasgow Coma Scale (GCS) score of group-A was 6.44±1.65 and group-B 6.47±1.53 was. On 3rd day treatment, mean GCS score of group-A increased to 9.70±2.89 and of group-B increased to 9.48±3.35. On 5th day of treatment, mean GCS score of group-A increased to 12.57±3.46 and of group-B increased to 11.21±3.53 .On the basis of cerebral oedema seen on Computed Tomography of brain treatment group-A shows 12.73% and group-B shows 5.45% improvement in 110 patients. Overall clinical improvement in group A was 43.63% and in group B 37.27%. The Dexamethasone causes improvement in the level of consciousness as evidenced by improvement in GCS level. The improvement was more evident when the patients were suffering from cerebral oedema.

**Keywords:** Dexamethasone, Acute Ischaemic Stroke, Randomized Clinical Study

## 1. INTRODUCTION

Bangladesh is the third largest country in terms of population among south Asian countries, after India and Pakistan, with a population of 160 billion. Stroke, which is an important morbidity in the context of sustainable development goals (SDGs) determined by the United Nations, represents the leading cause of disability in the Asian population [1].Stroke is the cause of a significant number of deaths that may have been avoided in Bangladesh. Stroke is the third leading cause of mortality in Bangladesh. In last decade the mortality rate due to stroke increased from 6% to approximately 9% [2].Individuals in Bangladesh who are 40 years old or older have a stroke prevalence of 0.3%, and the incidence of strokes climbs to 1% in individuals who are 70 years old or older [3]. According to the prevalence of stroke in Bangladesh, gender and age are two key criteria to consider. A past history of transient ischemic attack (TIA) and hyperlipidemia are among the risk factors for stroke in Bangladesh. Other risk factors include diabetes mellitus, heart disease, smoking cigarettes, and using oral contraceptives [4]. In comparison to other south Asian countries, the frequencies of these risk factors are comparable. Compared to patients who suffered from hemorrhagic stroke, those who suffered from ischemic stroke had a more favorable prognosis [5]. The majority of stroke patients fall into this category of them. Bangladesh, which has a big population, does not have the necessary health infrastructure or the educated human resource that is required to deal with the high burden of stroke [6]. One of the most common types of medical emergencies is a stroke, as people get older, the incidence of the disease increases dramatically, and in many nations with lower and intermediate incomes, it is increasing in conjunction with lifestyles that are less healthful [7]. There are approximately 180–300 individuals per 100,000 people who come with a stroke each year. Of these patients, 85 percent suffer a cerebral infarction as a result of inadequate blood flow to a portion of the brain, and the majority of the remaining patients have an intracerebral hemorrhage. It is a clinical illness of abrupt onset of suspected vascular origin that is marked by rapidly increasing indications of focal or global impairment of cerebral functioning that lasts for more than twenty-four hours or leads to death [8]. The term "stroke" is used to refer to this syndrome, the majority of cases of cerebral infarction are brought on by thromboembolic illness, which is ultimately brought on by atherosclerosis in the major extracranial arteries (the carotid artery and the aortic arch) [9]. Lacunar infarctions are produced by thrombosis in situ, which is caused by intrinsic disease of small perforating vessels (lenticulostriate arteries). This causes around twenty percent of all infarctions to occur. An embolism from the heart is responsible for approximately twenty percent of all infarctions. Ischemic stroke risk factors are similar to those that are associated with the vascular disease that is the underlying cause of the stroke. Vasculitis, endocarditis, and cerebral venous disease are some of the uncommon reasons that only account for about five percent of cases [10]. Although the patient's deficit may be at its peak quickly after the arterial occlusion, the process of cerebral infarction can take many hours to complete [11]. After the closure of a cerebral artery, the opening of anastomotic channels from other arterial territories that restore perfusion to the territory of the cerebral artery can prevent an infarction from occurring during the subsequent period [12]. Similar to how a decrease in perfusion pressure causes compensatory homeostatic changes, these modifications are necessary to keep tissue oxygenation at a constant level [13]. Because of these compensatory modifications, it is occasionally possible for closure of even a carotid artery to preclude any clinically noticeable manifestation of the condition [14]. In the event that these homeostatic systems fail, however, the process of ischaemia will begin, which will ultimately result in infarction unless the vascular supply is restored [15]. There are a variety of neuronal activities that fail at different levels as the cerebral blood flow decreases. The development of neurological deficiency occurs when the blood flow drops below the threshold that is necessary for the preservation of adequate electrical activity [16]. The neurons are still alive and well at this level of blood flow; if the blood flow increases once more, function will return, and the patient will have experienced a transient ischaemic attack (TIA) [17]. But if the blood supply continues to decrease, there will be a point where permanent cell death will begin to occur. Hypoxia causes an insufficient supply of adenosine triphosphate (ATP), which in turn causes membrane pumps to fail. This ultimately results in cytotoxic oedema, which is the accumulation of sodium and water within the cell, as well as the release of glutamate, an excitatory neurotransmitter, into the extracellular fluid [18]. Glutamate is responsible for opening membrane channels, which in turn enables calcium and sodium to enter the neurons a greater quantity. Upon entering the neurons, calcium triggers the activation of intracellular enzymes, which ultimately complete the

 

process of destruction [19]. Microglia and astrocytes are responsible for the release of inflammatory mediators, which ultimately results in the death of all cell types in the region of maximum ischaemia. The production of lactic acid in anaerobic conditions and the subsequent decrease in tissue pH both contribute to the worsening of the infarction process [20]. Although there have been attempts to develop neuroprotective drugs in order to slow down the processes that lead to irreversible cell death, these attempts have been unsuccessful up to this point. Because of this, the ultimate result of the occlusion of a cerebral blood vessel is contingent upon the functional capacity of the circulatory homeostatic mechanisms, the metabolic demand, as well as the degree of severity and duration of the reduction in blood flow [21]. With a given reduction in cerebral blood flow, a higher brain temperature, such as that which occurs in fever, and higher blood sugar have both been associated with a greater volume of infarction. "Haemorrhagic transformation" refers to the process by which it is possible for subsequent restoration of blood flow to result in bleeding into the infarcted area. Patients who are receiving antithrombotic or thrombolytic medication, as well as patients who have larger infarcts, are more likely to experience this complication [22]. When viewed through the lens of radiology, a cerebral infarct can be characterized as a lesion that is composed of a combination of tissue that is already undergoing autolysis and tissue that is ischaemic and swollen but can be recovered (referred to as the "ischaemic penumbra"). After a couple of days have passed since the onset of the stroke, the infarct reaches its maximum size. It grows larger over time. It is possible that at this stage it is large enough to exert mass effect both clinically and radiologically; in some cases, a decompressive craniectomy is required. In a matter of weeks, the oedema will begin to subside, and the infarcted region will be replaced by a cavity that is clearly defined and filled with fluid [23]. Managing the condition aims to minimize the amount of irreversible damage to the patient's brain, prevent complications, lessen the patient's disability and handicap through rehabilitation, and lessen the likelihood of the patient experiencing another stroke or other vascular event in the future. Within the first few hours or days after the onset of the neurological deficits, the patient may experience a worsening of those deficits. This occurs most frequently in patients who have lacunar infarcts, but it can also happen in other patients due to the extension of the area of infarction, the transformation of the hemorrhage, or the development of oedema with a subsequent mass effect [24]. Vasogenic oedema, cellular (cytotoxic) oedema, and interstitial oedema are the three more significant types of brain oedema. In the aftermath of an ischaemic stroke, cytotoxic and vasogenic oedema manifest themselves individually. Cytotoxic oedema, also known as "intracellular" oedema, is the initial reaction to cerebral damage [25]. This oedema, regardless of whether it happens as a result of ischemic or traumatic damage, is caused by the breakdown of cellular ionic pumps, which results in water entering the cell and becoming trapped within the cellular membrane [26]. There is a lack of clarity regarding the clinical effects and response to corticosteroids of this type of oedema that occurs immediately. A damage to the blood-brain barrier causes it to become "leaky," which allows water, electrolytes, and soon protein to enter the parenchyma. Vasogenic oedema, which occurs hours later, is the result of this damage [27]. This results in a severe and clinically significant swelling of the brain, which in turn causes the brain to become distorted and herniated, leading to neurological impairment and ultimately death [28]. In brain tumors, the normally tight vascular junctions are pathologically separated, and cellular physiology (such as pinocytosis) is otherwise disturbed, resulting in severe vasogenic oedema, which is highly responsive to corticosteroids. This fundamental difference in pathophysiology between the two types of lesions may explain the apparent difference in the initial therapeutic response of the two types of lesions. Experiments conducted on a wide range of mammalian models have produced an abundance of evidence demonstrating that corticosteroids are effective in reducing ischaemic cerebral oedema, both focal and generalized [29].

## 2. MATERIALS AND METHODS

It is a randomized prospective clinical trial, conduct in department of Medicine, Sher-e- Bangla Medical College Hospital, Barisal. Acute ischaemic stroke patients who were admitted in Medicine units of Sher-e- Bangla Medical College Hospital, Barisal were the population of study. The following standard formula is widely used in determining sample size:

$$n=z^2pq/d^2$$

 

Where,

n=desired sample size

z=standardized normal deviate usually set at 1.96 which corresponds to 95% confidence level.

q=1-p

p= proportion of target population estimated to have particular characteristics. If there is no reasonable estimate then use 50%. So p=50% or 0.5 and q=1-0.5=0.5

d=degree of accuracy desired, usually set at 0.05.

Using the above formula sample size will be of, n=384.

Due to time constraint, I included 110 patients for my study.

Data has been collect with the help of convenience sampling techniques, from patients having clinical features and radiological evidence of acute ischaemic stroke, age range from 30 to 70 years. Patient impairment of the level of consciousness (persistently Glasgow Coma scale ≤ 8) & Diagnosis made and treatment started within the first 48 hours following the onset of cerebral infarction. Data were analyzed by computer based software Statistical Package for Social Science, 21st version (SPSS-21) and presented as both qualitative and quantitative data as applicable. On admission, a detailed history of illness was taken from the every patient or their attendants. The findings obtained from general & systemic examination and findings of the performed investigations, relevant associated medical conditions were recorded carefully. All the collected data were checked and verified for its consistency. The data were compiled, analyzed & then tabulated according to key variables.

## 3. RESULTS AND DISCUSSION

To find out the role of dexamethasone in the management of acute ischaemic stroke 110 acute ischaemic stroke patients were included in this study. Findings of the study are detailed below.

### 3.1 Distribution of patients by age

The age of respondents ranged from 34 to 70 years with the mean age of 54.35 (SD±8.029) years. Age distribution of patients.

**Table 01:** Distribution of patients by age (n=110)

| Age of the patients (yrs) | Number of group -A patients | Number of group -B patients | Percentage of group -A patients | Percentage of group -B patients | Total No of patients | Total percentage of group-A and B |
|---|---|---|---|---|---|---|
| 34-40 | 5 | 2 | 4.55 | 1.81 | 7 | 6.36 |
| 41-50 | 14 | 14 | 12.73 | 12.73 | 28 | 25.46 |
| 51-60 | 20 | 27 | 18.18 | 24.55 | 47 | 42.73 |
| 61-70 | 16 | 12 | 14.55 | 10.9 | 28 | 25.45 |

Mean ± SD = 54.35 ± 8.029, Range=34-70

Above table 01 shows majority of the patients incorporated in this study belongs to age group between 51 – 60 years (i.e. 42.73%), followed by 61 – 70 years group (which was 25.45%).

 

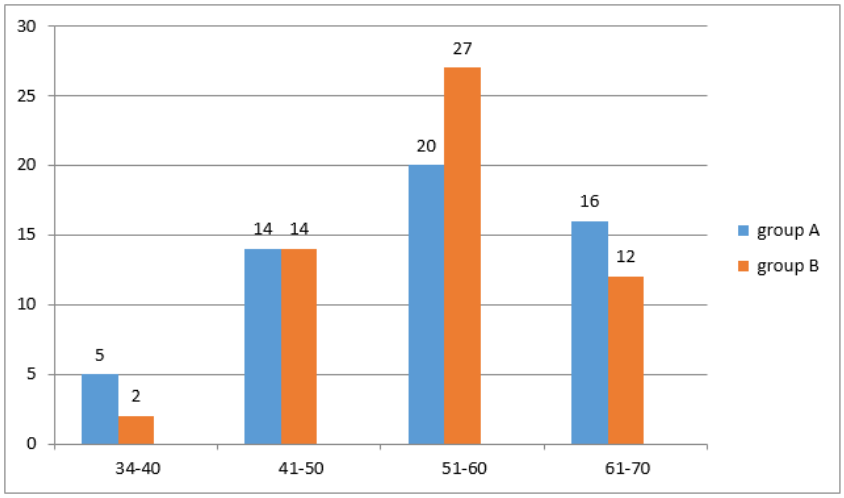


**Figure 01:** Distribution of patients by age

### 3.2 Distribution of patients according to gender

Among the patients male were 70 and female were 40 in number which were 63.64% and 36.36% of total study patients respectively.

**Table 02:** Distribution of patients according to gender (n=110)

| Gender | Number of group-A patients | Number of group-B patients | Percentage of group-A patients | Percentage of group-B patients | Total No | Total percentage |
|---|---|---|---|---|---|---|
| Female | 22 | 18 | 20 | 16.36 | 40 | 36.36 |
| Male | 33 | 37 | 30 | 33.64 | 70 | 63.64 |
| Total | 55 | 55 | 50 | 50 | 110 | 100 |

Above table depicts the distribution of respondents according to gender, it depicts the gender distribution of the patients as 63.64% of them are female and 36.36% are male. Among male Group A and Group B contains 30% and 33.64% respectively. In case of female Group A and Group B contains 20% and 16.36% respectively.

### 3.3 Distribution of patients according to geographic locations

Majority of study patients hailed from different districts of Barisal division. 24(21%) patients came from the different upazillas of Barisal district. of Patients hailed from three neighboring districts of Barisal division namely Jhalakati, Patuakhali and Barguna were 21(19.10%),18(16.40%) and 14(12.7%) respectively. Rest of the patients hailed from other districts of Barisal division namely Pirojpur and Bhola as well as from Gopalgonj and Madaripur district of Dhaka division.

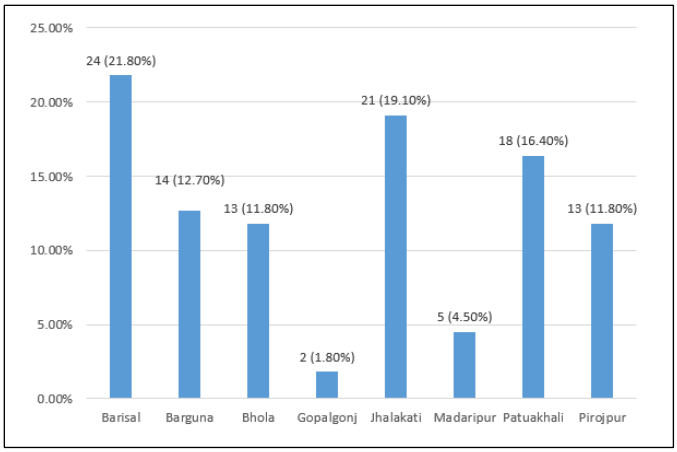


**Figure 02:** Distribution of patient by geographic locations

Above vertical bar chart depicts distribution of respondents according to their respective districts.

### 3.4 Distribution of patients according to treatment modalities

Study patients were distributed into two groups according to treatment modalities mention in table 03. Group-A patients treated with Dexamethasone which is 55(50%) and group-B treated without Dexamethasone which is also 55(50%).

**Table 03:** Distribution of patients according to treatment modalities (n=110)

| Treatment group | Treatment modalities | Total no. of patients | Percentage |
|---|---|---|---|
| A | Dexa$^{+}$ | 55 | 50 |
| B | Control | 55 | 50 |

Dexa+ means Dexamethasone

Above table shows distribution of patients according to treatment modalities

### 3.5 Glasgow Coma Scale (GCS) score during 1$^{st}$ day of hospitalization

Study subjects are categorized by GCS score which is ranged from 3 to 8 according to inclusion criteria in 1$^{st}$ day of hospitalization. The highest percentage (62.74%) of patient belongs to GCS score 7 to 8 and the lowest percentage (11.81%) of patient belongs to GCS score 3 to 4.

**Table 04**: Glasgow Coma Scale (GCS) score during 1st day of hospitalization (n=110)

| GCS score | Group A patient | Percentage | Group B patient | Percentage | Total No | Total Percentage |
|---|---|---|---|---|---|---|
| 3-4 | 7 | 6.36 | 6 | 5.45 | 13 | 11.81 |
| 5-6 | 13 | 11.81 | 15 | 13.64 | 28 | 24.45 |
| 7-8 | 35 | 31.83 | 34 | 30.91 | 69 | 62.74 |

### 3.6 Comparison of GCS score during 1st day of hospitalization in between group-A and group-B patients

Following table shows comparison of GCS score on 1st day of hospitalization in between group-A and group-B patients.

**Table 05:** Comparison of Comparison of GCS score during 1st day of hospitalization in between group-A and group-B patients (n=110)

| Patients group | Number of patients | Mean GCS ± SD | P-value |
|---|---|---|---|
| A | 55 | 6.44±1.65 | $0.92^{ns}$ |
| B | 55 | 6.47±1.53 | |

Above table shows comparison of GCS scores on 1st day of hospitalization between group-A and group-B patients.

### 3.7 GCS score on 3rd day of hospitalization

GCS score on 3rd day of hospitalization, the highest percentage (17.59%) of people from group A belongs to GCS score 11 to 12 and highest percentage (13.89%) of people from group B belongs to GCS score 9 to 10.

**Table 06:** GCS score on 3rd day of hospitalization (n=108)

| GCS score | Group A patients | Percentage | Group B patients | Percentage | Total No | Total Percentage |
|---|---|---|---|---|---|---|
| 3-4 | 1 | 0.93 | 4 | 3.7 | 5 | 5.93 |
| 5-6 | 5 | 4.63 | 5 | 4.63 | 10 | 9.26 |
| 7-8 | 15 | 13.89 | 9 | 8.33 | 24 | 22.22 |
| 9-10 | 7 | 6.48 | 15 | 13.89 | 22 | 20.37 |
| 11-12 | 19 | 17.59 | 13 | 12.04 | 32 | 29.63 |
| 13-14 | 1 | 0.93 | 0 | 0.0 | 1 | 0.93 |
| 15 | 6 | 5.55 | 8 | 7.41 | 14 | 12.96 |

Above table shows GCS score on 3rd day of hospitalization.

### 3.8 Comparison of GCS score on 3rd day of hospitalization between group-A and group-B patients

Comparison of GCS score on 3rd day of hospitalization showing table and mean GCS score increased to 9.70±2.89 for group-A and 9.48±3.35 for group-B patients.

**Table 07:** Comparison of GCS score on the 3rd day of hospitalization (n=108)

| Patients group | Number of patients | Mean pain score ±SD | P value |
|---|---|---|---|
| A | 54 | 9.70±2.89 | $0.71^{ns}$ |
| B | 54 | 9.48±3.35 | |

Above table displays comparison of GCS score on 3rd day of hospitalization between group A and group B patients.

 

### 3.9 GCS score on 5th day of hospitalization

GCS score on 5th day of hospitalization are shown, according to the table highest percentage of patient show GCS score in 15 in both group A and group B 29.52% and 18.09% respectively.

**Table 08:** GCS score on 5th day of hospitalization (n=105)

| GCS score | Group A patients | Percentage | Group B patients | Percentage | Total No | Total Percentage |
|---|---|---|---|---|---|---|
| 3-4 | 3 | 2.86 | 2 | 1.9 | 5 | 4.76 |
| 5-6 | 0 | 0.0 | 3 | 2.86 | 3 | 2.86 |
| 7-8 | 6 | 5.71 | 8 | 7.62 | 14 | 14.33 |
| 9-10 | 3 | 2.86 | 7 | 6.67 | 10 | 9.53 |
| 11-12 | 7 | 6.67 | 13 | 12.38 | 20 | 19.05 |
| 13-14 | 3 | 2.86 | 0 | 0.0 | 3 | 2.86 |
| 15 | 31 | 29.52 | 19 | 18.09 | 50 | 47.61 |

Above table displays clinical response on 5th day of hospitalization according to Glasgow Coma scale.

### 3.10 Comparison of GCS score on 5th day of hospitalization between group-A and group-B patients

Comparison of GCS score on 5th day of hospitalization showing on table 09, here mean GCS score of group-A and group-B increased to 12.57±3.46 and 11.21±3.53 respectively.

**Table 09:** Comparison of Glasgow Coma scale on 5th day of hospitalization (n=105)

| Treatment group | Number of patients | Mean GCS score ± SD | P value |
|---|---|---|---|
| A | 52 | 12.57±3.46 | 0.0488$^{s}$ |
| B | 53 | 11.21±3.53 | |

Above table depicts comparison of GCS score on 5th day of hospitalization between group A and group B patients.

### 3.11 Distribution of the patient according to the location of ischaemia on Computed Tomography of brain

According to the table highest numbers of patients were admitted with stroke on parietal region 33(30%) followed by basal ganglia 25(22.73%) and internal capsule 20 (18.18%).

**Table 10:** Distribution of the patient according to the location of ischaemia on Computed Tomography of brain (n=110)

| Site of lesion | Group A | Percentage | Group B | Percentage | Total number | Total percentage |
|---|---|---|---|---|---|---|
| Frontal | 5 | 4.54 | 1 | 0.91 | 6 | 5.45 |
| Parietal | 20 | 18.18 | 13 | 11.82 | 33 | 30 |
| Temporal | 4 | 3.64 | 7 | 6.36 | 11 | 10 |
| Occipital | 2 | 1.82 | 2 | 1.82 | 4 | 3.64 |
| Basal ganglia | 14 | 12.73 | 11 | 10 | 25 | 22.73 |
| Cerebellum | 2 | 1.82 | 5 | 4.54 | 7 | 6.36 |
| Internal capsule | 11 | 10 | 9 | 8.18 | 20 | 18.18 |

 

| | | | | | | |
|---|---|---|---|---|---|---|
| Brain stem | 3 | 2.73 | 7 | 6.36 | 10 | 9.09 |

Above table shows distribution of the patient according to the location of ischaemia on Computed Tomography of brain.

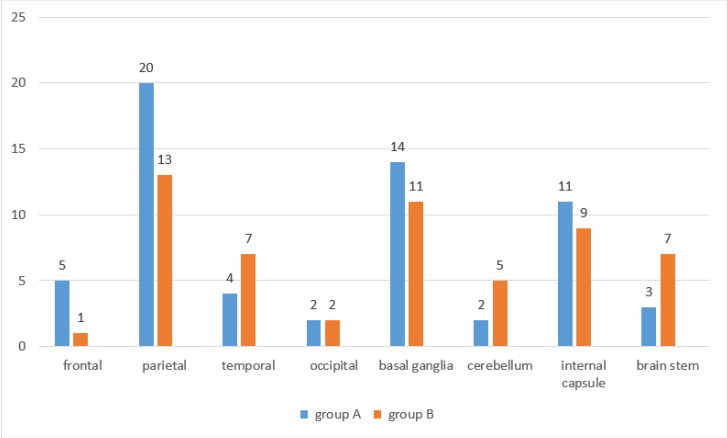


**Figure 03:** Distribution of the patient according to the location of ischaemia on Computed Tomography of brain

### 3.12 Incidence of cerebral oedema on Computed Tomography of brain on 1st day of hospitalization.

Cerebral Oedema found on Computed Tomography (CT) of brain showing Here 33 (30.01%) patients show cerebral oedema and 77 (69.99%) patients still show no cerebral oedema.

**Table3.11:** Incidence of cerebral oedema on CT scan of brain on 1st day hospitalization (n=110)

| Cerebral Oedema | Group A patients | Group B patients | Total number of patient | Total Percentage |
|---|---|---|---|---|
| Yes | 17 (15.47%) | 16(14.54%) | 33 | 30.01 |
| No | 38(34.54%) | 39(35.45%) | 77 | 69.99 |

Above table is showing number of cerebral oedema on Computed Tomography (CT) of brain on 1st day of hospitalization.

### 3.13 Comparative treatment response in between group-A and group-B patients having cerebral oedema on CT scan of brain

In this study by CT Scan 12.73% of group-A and 5.45 % of group-B patients having cerebral oedema shown improvement in terms of GCS.

**Table 11:** Comparative treatment response between group-A and group-B patients having cerebral oedema on CT scan of brain (n=110)

| Treatment Groups | Improved | Deteriorated | No change | | $X^2$ test | |
|---|---|---|---|---|---|---|
| | | | | Test result | df | P value |
| Group A | 14(12.73%) | 1(0.91%) | 1(0.91%) | 7.6004 | 2 | 0.0223[s] |

Above table displays comparative improvement of patients having cerebral oedema between group-A and group-B.

### 3.14 After 5 days of hospitalization with the comparison between the group A and group B patients

In this study the comparison between the groups A and the group B patients after 5 days of hospitalization with Dexamethasone shown. Here 48(43.63%) patients improved after having Dexamethasone treatment in comparison to 41(37.27%) patients improved in placebo group.

**Table 12:** after 5 days of hospitalization with the comparison between the treatment groups A and B patients (n=110)

| Treatment Groups | Improved | Deteriorated | No change | | $X^2$ test | |
|---|---|---|---|---|---|---|
| | | | | Test result | df | P value |
| Group A | 48(43.63%) | 3(2.73%) | 2(1.82%) | 6.3688 | 2 | 0.0321[s] |
| Group B | 41(37.27%) | 1(0.91%) | 10(9.09%) | | | |

**Table 13:** After 5 days of hospitalization with the comparison between the treatment groups A and B patients (n=110).

| Treatment Groups | Improved | Deteriorated | No change | | $X^2$ test | |
|---|---|---|---|---|---|---|
| | | | | Test result | df | P value |
| Group A | 48(43.63%) | 3(2.73%) | 2(1.82%) | 6.3688 | 2 | 0.0321[s] |
| Group B | 41(37.27%) | 1(0.91%) | 10(9.09%) | | | |

s= significant, the chi square statistic result is 6.3688. P value is 0.0321. , The result is significant as P< 0.05 is significant.

Above table showing the comparison in between the group A and the group B patients after 5 days of treatment.

### 3.15 Number of patients died or referred during the course of hospitalization.

4 patients (3.63%) died in total and 1 (0.91%) patient was referred on 2nd day from group A during 5 days of hospitalization. 1(0.91%) patient died from group A and 3(2.73%) patient died from group B.

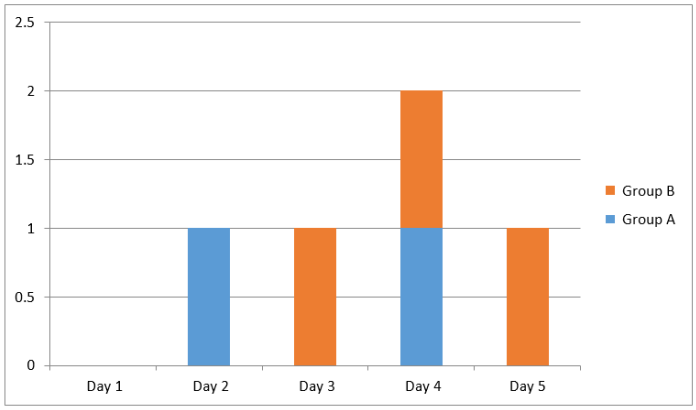


**Figure 04:** Number of patients died or referred during the course of hospitalization

Above vertical chart shows number of patients died or referred during the course of hospitalization.

### 3.16 Number of patients died or referred during the course of hospitalization having cerebral oedema.

Among 5 patients (4.55%) in who could not complete the treatment due to death or referral 1(0.91%) from group A and 2(1.82%) from group B had cerebral oedema.

**Table 14:** Number of patients died or referred during the course of hospitalization having cerebral oedema.

| Cerebral Oedema | Group A patients | Group B patients | Total number of patient | Total Percentage |
|---|---|---|---|---|
| Yes | 1 (0.91%) | 2(1.82%) | 3 | 2.73 |
| No | 1(0.91%) | 1(0.91%) | 2 | 1.82 |
| Total | 2(1.82%) | 3(2.73%) | 5 | 4.55 |

Above table shows number of patients died or referred during the course of hospitalization having cerebral oedema. Acute occlusion of an intracranial vessel causes reduction in blood flow to the brain region it supplies. The magnitude of flow reduction is a function of collateral blood flow and this depends on individual vascular anatomy, the site of occlusion, and likely systemic blood pressure. A decrease in cerebral blood flow to zero causes death of brain tissue within 4–10 min; values <16–18 mL/100 g tissue per minute cause infarction within an hour; and values <20 mL/100 g tissue per minute cause ischemia without infarction unless prolonged for several hours or days. If blood flow is restored prior to a significant amount of cell death, the patient may experience only transient symptoms, and the clinical syndrome is called a TIA. Tissue surrounding the core region of infarction is ischaemic but reversibly dysfunctional and is referred to as the ischaemic penumbra. Saving the ischaemic penumbra is the goal of revascularization therapies 31. Following ischaemic stroke both cytotoxic and vasogenic oedema occurs. Cytotoxic oedema represents the earliest response to cerebral damage. The majority of strokes are due to cerebral ischaemia. Theoretically, steroids are immunosuppressive agents, lessen the damaging effects of vasogenic cerebral edema, decrease intracranial pressure and stabilize the blood-brain barrier and can have impact on acute ischaemic stroke. A meta-analysis, based on only seven trials was performed 2–3 decades ago, using various doses and regimens of either dexamethasone or betamethasone, with CT brain imaging performed in only one trial and no common outcome scales or assessment time points. Bearing this in mind and the lack of a well-defined role for corticosteroids in experimental cerebral ischemia, some authorities believe that a further robust large RCT of corticosteroids in acute ischemic stroke is justified. It is conceivable that certain sub-groups of patients, perhaps those with massive cerebral oedema, disrupted blood brain barrier and a significant vasogenic component may derive benefit from corticosteroids.

## 3.2 Discussion

This hospital based prospective study was undertaken to establish Dexamethasone as a treatment option and to see whether it is superior or inferior to conventional therapy for acute ischaemic stroke. The role of Dexamethasone in the management of acute ischaemic stroke is not very much established. So results of Dexamethasone regarding management of acute ischaemic stroke are only compared with the results of conventional therapy of this study and also previously published. The present study findings were discussed and compared with previously published relevant studies. In this current study it was observed that the mean age was found 54.35± 8.029 years with range from 34 to 70 years. The majority of population was found to below 40 years. Out of these 110 subjects 70(63.60%) were females and 40(36.40%) were males. A study conducted suggested the mean age was 58.63±14.82 in experimental group and 60.10±16.80 was in control groups [30]. Mean age of the stroke patients was 60.6 years; the majority of patients (67.7%) were male. Only 16.5% of the patients had age greater than 70 years. Most patients (66%) had an age between 45 and 70 years. Only 72 (11%) patients (50 men and 22 women) were less than 45 years 27. Mean age of the stroke patients which is around sixty years is consistent with findings from a similar stroke registry [31]. Most of the patients from a stroke registry in USA presented with stroke at an age of 71 years [32]. In Korea the mean age of patients getting registered is around 62 years30. The lower percentage of female stroke

 

patients being registered implies either a low prevalence of stroke among females or a lower access of female stroke patients to the tertiary care hospital [33]. Most of the patients [47 out of 110 (42.73%)] under this study were diagnosed acute ischaemic stroke between the age of 51 – 60 years. At the age of 34-40 years were 7(6.36 %), between 41-50 years were 28(25.46 %), between 51-60 years were 47(42.73 %) and between 61-70 years were 28 (25.45 %) patients. These findings can be compared with a study of 51 stroke patients showed that the age range of the patients were 17-84, with majority of them between 50-69 years31 [35]. Overall male to female ratio was 1.40: 1. 30% patients have GCS level from 4-7 in experimental group and 10% patients have GCS level from 4-7 in control group. In experimental group, 46.7% patients have GCS level from 12-15 & 50% patients have GCS level from 12-15 in control group. On the first day, mean GCS level in experimental group was 10.56 and in control group it was 11.212 [36]. Incidence of cerebral oedema on Computed Tomography of brain on 1st day of admission was 33 out of 110 (30.01%). In a study the level of consciousness improved both in experimental group & control group of patients on the 3rd, 7th & 10th days of treatment. The improvement in consciousness level was more in experimental group compared to control group on the 3rd, 7th and 10th day of treatment [37]. On the 3rd day of intervention, X2 test value was 8.61 & P value was 0.033. On the 7th day, X2 test value was 9.3 & P value was 0.009. On the 10th day of intervention X2 test value was 8.60 & P value was 0.025. On 10th day of intervention, all the patients had GCS level above 7 in both the groups12. A study of 2556 ischemic stroke patients, 157 received thrombolytic therapy. Eighty of the 2556 patients (3.1%) died during hospitalization, which included 14 (8.9%) patients who received thrombolytic therapy [38].

## 4. CONCLUSIONS

This study was undertaken to establish Dexamethasone as a treatment option and to see whether it is superior or inferior to conventional therapy for acute ischaemic stroke. About one-fifth of patients with an acute stroke die within a month of the event and at least half of those who survive are left with physical disability. Steroids are considered as a group of the magic drugs and have been widely used in Neurology for more than 40 years. Dexamethasone, a synthetic steroid (glucocorticoid) lessens the damaging effects of vasogenic cerebral oedema, decreases intracranial pressure, and stabilizes the blood-brain barrier. Dexamethasone is one of the most effective and safe medicine needed in a health system. It is not expensive. It is available in most areas of the world. In Bangladesh, in the tertiary hospitals, Dexamethasone is used quite frequently but its effectiveness remains doubtful because of lack of study. So this study regarding patient's clinical improvement after intravenous Dexamethasone can be helpful in our context, majority of patients of these two groups responded to their treatment separately. This study shows Dexamethsone, a single drug has efficacy in addition of its established antioedema action. Though Dexamethasone is not superior but its efficacy showed very useful to conventional therapy. So study concludes that Dexamethasone may be another treatment addition in the management of acute ischaemic stroke. Findings of this study clearly suggest that Dexamethasone can be utilized as a treatment option in the management of acute ischaemic stroke. Dexamethasone, a single drug provides mode of action by anti-inflammatory, anti oedema, decreasing intra cranial pressure, stabilizing blood brain barrier in the management of acute ischaemic stroke. Further studies are warranted to reinforce the potential of Dexamethasone as a treatment option and to compare with other conventional therapies to establish which one is more suitable.

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