Original Contribution

Clinical Audit of Stroke Mortality

Identification of Mortality-related Predictive Factors in Hospitalized Patients with Ischemic Stroke

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Abstract

Objective: This prospective study was planned to study the prognostic value of time to presentation to the hospital, clinical scales and examination, hematological and biochemical parameters as predictive factors of mortality in ischemic stroke. **Materials and Methods:** In this study, 188 consecutive patients of ischemic stroke were included. The patients were subjected to relevant history taking, clinical evaluation, blood investigations (total leukocyte count [TLC], capillary blood sugar, potassium, high-sensitivity C-reactive protein (hs-CRP), troponin I (Trop I)), electrocardiogram (ECG) and neuroimaging. The patients were followed up till their final outcome in the hospital, and patients who expired were grouped as 'Mortality Group' and the rest as 'Discharged Group'. Logistic regression analysis was carried out among the significant parameters to identify independent predictors of mortality in cases of ischemic stroke. **Results:** After statistical analysis, it was found that late presentation to the hospital, pyrexia (temperature >99°F), low diastolic blood pressure at the time of admission, hypoxia (saturation of oxygen [sPo2] <94%), National Institute of Health Science scale (NIHSS) score >15, modified Rankin score (MRS) greater than 3, Glasgow coma scale (GCS) less than 8, hyperglycemia (random blood sugar >200 mg/dL), raised total leukocyte count and hs-CRP (>10 mg/L), are positive predictive factors of mortality in cases of ischemic stroke. **Conclusion:** Late presentation to the hospital, pyrexia (temperature >99°F), low diastolic blood pressure at the time of admission, hypoxia (sPo2 <94%), more severe stroke (NIHSS score >15, MRS >3, GCS <8), high TLC estimated at the time of hospitalization, are the most important predictive prognostic factors of in-hospital mortality in cases of ischemic stroke.

Key words: In-hospital mortality, ischemic stroke, predictive factors

Historical Background

Stroke burden in India is rising in the past few decades, in contrast to developed countries where it has reached a plateau or decreased. The average annual incidence of stroke in India currently is 145 per 100,000 population, which is higher than the Western nations. In India 10–15% of strokes occur in people aged below 40 years. Up to 80% strokes in India are ischemic in nature, among which intracranial atherosclerosis is the most common mechanism. There are 4,700,000 stroke survivors alive today.[1,2]

Of a total of 9.4 million deaths in India, 619,000 deaths were due to stroke. This gives stroke a mortality rate of 73 per 100,000 (estimated total population 849 million). Approximately 8–12% of ischemic strokes are fatal (37–38% hemorrhagic strokes are fatal). In the same year, the number of deaths due to stroke were 22 times that due to malaria, 1.4 times that due to tuberculosis, 4 times that due to rheumatic heart disease, and almost equal to ischemic heart disease. Stroke mortality rates have been found to be higher in Indians, compared with developed nations.[1,2]

Stroke recently declined from the third to the fourth leading cause of death in the United States, its first rank transition among sources of American mortality in nearly 75 years. Historically, stroke transitioned from the second to the third leading cause of death in the United States in 1937, but stroke death rates were essentially stable from 1930 to 1960. Then a long, great decline began, moderate in the 1960s, precipitous in the 1970s and 1980s, and moderate again in the 1990s and 2000s. By 2008, age-adjusted annual death rates from stroke were three-fourths less than the historic 1931 to 1960 norm (40.6 vs 175.0 per 100,000). Total actual stroke deaths in the United States declined from a high of 214,000 in 1973 to 134,000 in 2008.[3]

Introduction

Cerebrovascular diseases include some of the most common and devastating disorders: Ischemic stroke, hemorrhagic stroke, and cerebrovascular anomalies such as intracranial aneurysms and arteriovenous malformations. Cerebral

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ischemia is caused by reduction in blood flow lasting longer than several seconds. 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 and the site of occlusion.[4]

Mortality after stroke is still high, with stroke ranked as the second most common single cause of death in the developed world after ischemic heart disease, or third if all neoplastic diseases are considered as a group.[5]

Complications after ischemic stroke comprise medical and neurological complications. Neurological complications include brain edema, hemorrhagic transformation, seizures and epilepsy, recurrent stroke, and delirium. These complications are less frequent than medical complications but occur earlier in the course of stroke progression – within 48–72 h of stroke onset rather than within the first few weeks of stroke. Results from some studies have indicated that deaths within the first few days of stroke are usually the direct consequence of brain damage from neurological complications. Similarly, autopsy series of early stroke fatalities have indicated that death within the first few weeks after stroke is mainly attributable to the direct effects of stroke, such as brain edema with transtentorial herniation. In a study of neurological worsening during the acute phase of ischemic stroke in 1964 patients, 33.6% of patients deteriorated because of progressive stroke, 27.3% as a result of brain swelling, 11.3% owing to recurrent ischemic stroke, and 10.5% because of parenchymal hemorrhage. The remaining 17.3% deteriorated because of pyrexia, hyperglycemia, and hypertension, which are abnormal physiological variables or medical complications.[5]

There is a need to initiate a systemic data collection on predictive factors of mortality due to stroke since at such a significant mortality rate, stroke consumes a major part of limited health resources in a developing country like India.[2]

Few systemic evaluations are available for association of factors with in-hospital mortality in stroke patients in India. Thus, this study was planned to find out various predictors of in-hospital mortality. In this prospective study, we have evaluated the significance of time to presentation to the hospital, examination scales, blood parameters for in-hospital mortality.

Materials and Methods

The study was carried out in the Department of Medicine at the Himalayan Institute of Medical Sciences, Swami Ram Nagar, Dehradun over a period of 19 months. All consecutive patients of ischemic stroke of age greater than 18 years were included after obtaining informed written consent.

The time of onset of the stroke was defined as the time when the patient or observer first became aware of the symptoms. After inclusion, each patient was subjected to a detailed history, and clinical evaluation. The patients were assessed as per the National Institute of Health Science scale (NIHSS), modified Rankin score (MRS), and the Glasgow coma scale (GCS). Informed consent was taken for each patient. Subsequently, the patients were treated according to the standard treatment protocols as per American Academy of Neurology guidelines, 2007. The study in no way interfered with the treatment. The patients were subjected to a computed tomography (CT) scan, and blood investigations. The patients were followed up till their final outcome in the hospital, and patients who expired were grouped as 'Mortality Group' and the rest as 'Discharged Group'.

In-hospital mortality is defined as, death within 30 days of admission, which is used both in Medicare Mortality Predictive System and in Medicare Hospital Mortality Information 1986.[6]

Factors recorded were:

- Duration of time between the onset of symptoms and presentation
- History of a previous stroke or transient ischemic attack (TIA)
- NIHSS score at the time of admission
- MRS score at the time of admission
- GCS score at the time of admission
- Blood pressure at the time of admission
- Temperature at the time of admission
- Mean reading of oxygen saturation for 10 min at the time of admission
- Random blood sugar at the time of admission
- Electrocardiogram (ECG) at the time of admission
- Total leukocyte count (TLC) at the time of admission
- Potassium at the time of admission
- Radiological Imaging at the time of admission
- CRP (HS) levels at the time of admission
- Troponin I (Trop I) levels at the time of admission.

Blood samples were collected immediately after admission, before starting any intravenous infusion. Blood samples were immediately sent to the laboratory for evaluation.

Interpretation and analysis of the obtained results were carried out using standard statistical tests of significance.

Results

The study included 188 patients [Table 1] over a period of 1.5 years. Among these, 47 (25%) patients expired, whereas 141 (75%) patients survived and were discharged from the hospital. Among the 188 patients of acute ischemic stroke, 126 patients were males and 62 patients were females. Among 126 male patients, 36 patients died and remaining 90 patients were discharged from the hospital. Among the 62 female patients, 11 patients died and remaining 51 patients were discharged from the hospital ($P = 0.1518$).

Among the 47 patients who expired during their stay in the hospital, the average duration from the onset of symptoms of stroke to the presentation at the hospital was 47.510 ± 39.727 h.

Table 1: Table of Factors Included and Their Results

Factors considered	Mortality	Morbidity	*P*
Population	47	141	
Males	36	90	
Females	11	51	
Onset of symptoms of stroke to the presentation (hours)	47.51±39.73	22.55±43.07	0.0006
Prior stroke	5	32	0.1929
Prior TIA	0	3	
Prior history of hypertension	23	75	0.8875
Prior history of diabetes	14	36	0.803
NIHSS score at the time of admission	32.659±0.938	22.269±4.878	0.0001
MRS	5±0	4.014±0.609	0.0001
GCS scores	3.787	11.3	0.0001
Systolic blood pressure (mm of Hg)	130.212±42.859	135.602±24.064	0.2848
Diastolic blood pressure (mm of Hg)	75.531±29.177	82.056±14.318	0.044
RBS (mg/dl (mmol/L))	184.53±83.66 (10.26)	149.29±71.38 (8.26)	0.0001
Temperature >99 F	9	8	
sPo2 ≤94%	23	12	0.0001
Atrial fibrillation	3	7	1
TLC (/cmm)	15258.51±6951.62	11475.12±4954.69	0.0001
Potassium (mEq/L)	4.416+0.699	4.115+0.633	0.007
hs-CRP (mg/L)	13.82±4.18	4.76±4.36	0.0001
Trop I positive	3	3	0.36

TIA: Transient ischemic attack, TLC: Total leukocyte count, MRS: Modified Rankin score, GCS: Glasgow coma scale, CRP: C-reactive protein

In contrast, amongst the 141 patients who were subsequently discharged following treatment, the average duration from the onset of symptoms of stroke to the presentation at the hospital was 22.553 ± 43.067 h ($P = 0.0006$).

Among the 188 patients, 37 patients were found to have suffered from stroke previously. Of these, only 5 patients expired. The remaining 32 patients were discharged. Only three patients reported having suffered from transient ischemic attack previously. All the three patients were discharged ($P = 0.1929$).

All of the 188 patients were assessed as per the NIHSS score at the time of admission. The mean NIHSS score in the mortality group was 32.659 ± 0.938. The mean NIHSS score in the discharged group was 22.269 ± 4.878 ($P = 0.0001$).

Of the 188 patients, 47 patients expired. All the 47 patients were admitted in the severe category as per the MRS system. In contrast, 141 patients, who were admitted and later discharged, 18 patients presented with the mild score (score in the range of 1–3), 100 patients presented with a moderate score (score of 4), and 23 presented with the severe score (score of 5). The mean MRS score in the mortality group is 5 ± 0, while the mean MRS score in the patients that were discharged was 4.014 ± 0.609 ($P = 0.0001$).

Of the 188 patients that were included in the study, 47 expired and all the 47 patients were in the severe category of GCS scores.(3–8) In contrast, in the discharged group, 20 patients were in the severe category (3–8) at the time of admission, 84 patients were in the moderately severe category (9–12), and the remaining 37 patients were in the mild category (13–15) as per the GCS ($P = 0.0001$). Mean GCS in mortality group (3.787) was significantly low in comparison to the other group (11.3, $P = 0.0001$).

The mean of systolic blood pressure of the patients in the expired group was 130.212 ± 42.859 mmHg. The mean of systolic blood pressure of the patients in the group that was discharged was 135.602 ± 24.064 mmHg ($P = 0.2848$). The mean of diastolic blood pressure of the patients in the mortality group was 75.531 ± 29.177 mmHg. The mean of diastolic blood pressure of the patients who were discharged was 82.056 ± 14.318 mmHg ($P = 0.044$).

These patients of ischemic stroke were assessed for factors like hypertension. Ninety-eight of these 188 patients were found to have hypertension. Of the 98 patients who were found to be hypertensive, 23 patients were in the mortality group and 75 patients could be discharged ($P = 0.8875$).

Patients of ischemic stroke were assessed for factors like diabetes. Among the 188 patients included, 50 patients were found to be having diabetes. From the 50 diabetic patients, 14 patients were in the patients who expired, whereas 36 patients were discharged ($P = 0.803$).

Of the 188 patients, 20 patients among the patients who expired, and 26 patients among the patients who were discharged, were brought with a RBS of more than 200 mg%. Among the patients who expired, 11 patients who were brought with a raised RBS were diabetics. In contrast, 20 patients among the patients who were discharged were diabetic and presented with high RBS. Nine non diabetic patients presented with hyperglycemia in the mortality group, and six non diabetic patients presented with hyperglycemia in patients who were discharged ($P = 0.008$). The mean RBS of the patients who expired was 184.53 ± 83.66 mg/dl (10.26 mmol/L), whereas that of the patients who were discharged was 149.29 ± 71.38 mg/dl (8. 26 mmol/L) ($P = 0.0001$).

Among the 47 patients who expired, and 141 patients who were discharged, 9 and 8 individuals, respectively, had temperature >99°F each ($P = 0.02$).

Among the 47 patients, 23 patients had a saturation of oxygen (sPo2) ≤94%, while 12 patients of the 141 patients who were discharged had a sPo2 ≤94% ($P = 0.0001$).

ECGs were studied for changes of atrial fibrillation. Among the total of 188 patients, 10 patients presented with features of atrial fibrillation. Of the 10 patients who presented with atrial fibrillation, 3 patients expired, while 7 patients survived and were discharged ($P = 1.00$).

Of the 47 patients who expired, 2 patients had a low TLC (<4000/cmm), 36 patients had raised TLC (>12,000/cmm), while the remaining 9 had a TLC within the normal range (4000–12,000/cmm). In contrast, of the 141 patients who were discharged from the hospital, 2 patients had a low TLC (<4000/cmm), 59 had a raised TLC (>12,000/cmm), while 80 had a normal TLC (4000–12,000/cmm) ($P = 0.02$). The mean TLC among the patients who expired was 15258.51 ± 6951.62/cmm, whereas that among patients who were discharged was 11475.12 ± 4954.69/cmm ($P = 0.0001$).

Of the 47 patients who expired, only 1 patient had presented with hypokalemia. Of the 47 patients who expired, 16 patients had presented with hyperkalemia. In contrast, 16 and 27 of the 141 patients presented with hypokalemia and hyperkalemia, respectively. The mean potassium in mortality group was 4.416 + 0.699 mEq/L, and the mean potassium in the patients who were discharged was 4.115 + 0.633 mEq/L ($P = 0.007$).

Of the 47 patients, who expired, 1 had left sided anterior cerebral artery (ACA) infarct, none had a right sided ACA infarct. Nineteen of them had left sided middle cerebral artery (MCA) infarct, while 13 had right sided MCA infarct. Nine of the 47 patients had a left sided posterior cerebral artery (PCA) infarct, while 5 had right sided PCA infarct. Of the 141 patients who were discharged, 3 had a left sided ACA infarct, 4 had right sided ACA infarct, 57 had left sided MCA infarct, 36 had right sided MCA infarct, 32 had left sided PCA infarct, and 9 suffered right sided PCA infarct. Of all the territories involved, MCA territory was the one most commonly affected (125) in either groups (32 in the mortality group and 93 in the discharged group). This was followed by PCA territory (55), in either group (14 in mortality group and 41 in discharged group). This was followed by ACA territory (8), in either group (1 and 7 in mortality and discharged group, respectively) ($P = 0.67$).

Among the patients who expired, the volume of infarct of the patient with left ACA infarct was 6 ml. There were no patients with right ACA infarct. The mean volume of infarct in the patients with left and right MCA infarct were 74.321 ± 10.586 and 76.535 ± 9.896 ml, respectively. The mean volume of infarct in the patients with PCA infarct was 12.521 ± 5.126 and 13.127 ± 5.586 ml, respectively, for left and right side. Among the patients who were discharged, the mean volume of patients with left and right ACA territory stroke were 6.418 ± 2.138 and 6.422 ± 2.146 ml, respectively (*P* value could not be calculated due to low sample size). The mean volume of patients left and right MCA territory stroke were 76.615 ± 12.375 and 77.087 ± 12.589 ml, respectively (*P* value 0.4715 and 0.8872 on left and right side, respectively). The mean volume of patients with left and right PCA territory stroke were 14.148 ± 6.655 and 14.348 ± 6.857 ml, respectively (*P* value 0.5025 and 0.7508 on left and right side, respectively).

All the 188 patients were subjected to evaluation of hs-CRP at the time of admission. Of the patients who expired, 40 of the 47 had a level of more than 10 mg/L and 7 had levels less than or equal to 10 mg/L. In contrast, 25 patients of the 141 patients who were discharged had hs-CRP levels of more than 10 mg/L. Of the 144 patients, 116 who were discharged had hs-CRP levels of less than or equal to 10 mg/L. The mean of hs-CRP levels of patients who expired was 13.82 ± 4.18 mg/L, while that of the patients who were discharged was 4.76 ± 4.36 mg/L ($P = 0.0001$).

Of the 47 patients who expired, 3 were found to have Trop I positive. Similarly, of the 141 patients who were discharged, 3 were found to have Trop I positive at the time of admission. All six patients had electrocardiography changes suggestive of inferior wall myocardial infarction ($P = 0.36$).

Discussion

The mortality of ischemic stroke remains high in developing nation like India. In this study, 25% patients expired. Similar to another Indian study, mortality in our patients with acute cerebral infarction was 25%.[7]

Male gender is an independent risk factor for stroke. In our study, males were twice as common as females (126:62). Similar to a previous study,[8] gender was not found to be a predictive factor for increased mortality in acute ischemic stroke. Low incidence of stroke in females was explained on the basis of the protective effect of estrogen in females.[9]

Late presentation to the hospital after onset of ischemic stroke resulted in a poor outcome and mortality in cases of ischemic stroke. In this study, patients who presented late to the hospital had a higher mortality rate as compared with those who presented early.

A history of diabetes, hypertension, and previous ischemic event are known independent risk factors for the occurrence of another ischemic stroke. A history of diabetes (strict glucose control is associated with minimization of the amount of damage due to glycosylation[10]), hypertension (in part because of the fact that currently we have efficient tools to lower blood pressure[11]), and previous ischemic event do not portend the possibility of mortality in ischemic stroke.

Clinical scales evaluated in our study were NIHSS, MRS, and GCS. Similar to previous studies[12-14,8] our study found that:

- More than 25% (47/175) patients who presented with NIHSS score >15 expired while none of the patients with NIHSS score <15 expired
- More than 25% (47/170) patients who presented with MRS score of more than 3 expired
- Nearly 70% patients (47/67) who had GCS <8 expired, while in patients who had GCS more than 8, none expired.

This is because high NIHSS, MRS, and a low GCS indicate a more severe stroke. Therefore they predict a higher mortality.

Clinical parameters – a low diastolic blood pressure, temperature >99°F, and sPo2 ≤94% – are found to be significant

factors that predict mortality in acute ischemic stroke. High diastolic blood pressure may have a beneficial effect in maintaining brain perfusion after acute ischemia.[15] Fever can be caused by an impairment of central thermoregulation. Fever can exacerbate neuronal injury by increasing metabolic demands on injured brain tissue.[16] Tissue surrounding the core region of infarction is ischemic and is referred to as the ischemic penumbra and will eventually infarct if no change in flow occurs.[4] Oxygen therapy might protect the ischemic penumbra by suppressing harmful processes such as oedema, inflammation and apoptosis.[17]

Blood investigations – raised TLC, poststroke hyperglycemia, and hsCRP >10 mg/L – were found to be predictive factors of in-hospital mortality in cases of acute ischemic stroke. Several mechanisms by which leukocytes may be implicated in parenchymal brain injury include vessel plugging, release of hydrolytic enzymes, oxygen free radicals or initiation of thrombosis. Leucocytosis might also be a manifestation of some common causes of fever (e.g., pulmonary or urinary tract infections, sepsis, or pulmonary embolism from deep vein thrombosis).[8] Hyperglycemia increases growth of the infarct core in patients with surrounding hypoperfusion, suggesting that hyperglycemic blood is toxic to ischemic brain.[17] Inflammatory markers predict incident ischemic events, including myocardial infarction and stroke. Levels of high-sensitivity C-reactive protein (hs-CRP), an acute-phase reactant, are strongly associated with stroke severity. Because stroke severity is also strongly associated with mortality after stroke, it is not surprising that hs-CRP is also associated with mortality.[18]

Atrial fibrillation, one of the most common causes of cerebral embolization, may not be a significant factor to predict mortality in ischemic stroke. Myocardial infarction is a cause of mortality in the cases of ischemic stroke. Trop I is one of the most sensitive markers of a myocardial infarction. In our study, the sample size in the study was too small to be statistically evaluated.

The MCA territory infarct, followed by PCA territory infarct, followed by ACA territory infarct are the most commonly affected territories in our patients, in that order. The territory does not predict the mortality in acute ischemic stroke. The volume of the infarct was not found to be a significant factor in the mortality of stroke. The reason may be that a small infarct in a crucial area may be more significant than a large infarct in a less significant area.

Conclusion

Males were more commonly affected, and MCA territory infarct was most common territory involved in ischemic stroke, followed by the PCA territory.

Past history of diabetes, hypertension, and previous ischemic events, though independent risk factors, do not predict mortality in cases of acute ischemic stroke.

After statistical analysis, it was found that late presentation to the hospital, pyrexia (temperature >99°F), low diastolic blood pressure at the time of admission, hypoxia (sPo2 <94%), NIHSS score >15, MRS score greater than 3, GCS less than 8, hyperglycemia (RBS >200 mg/dL), raised TLC, and hs-CRP >10 mg/L are positive predictive factors of mortality in cases of ischemic stroke.

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