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STRESS HYPERGLYCEMIA AS A PROGNOSTIC MARKER IN ACUTE ISCHAEMIC STROKE

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ABSTRACT

OBJECTIVES: To evaluate the outcome in acute ischemic stroke patients with stress hyperglycemia in terms of mortality and functional recovery based on National Institute of Health Stroke Scale (NIHSS). **MATERIALS AND METHODS:** This is a case control study. 100 patients with CT evidence of acute ischaemic stroke meeting the inclusion and exclusion criteria's admitted to R.L. Jalappa Hospital and Research center attached to Sri Devaraj Urs Medical college, Tamaka, Kolar during may2014 to September 2015 were included in the study. The data was collected based on detailed history and clinical examination done as per the proforma along with few investigations like CT brain, RBG levels at admission, FBS, PPBS, HbA1c and RBG at discharge. Neurological status was assessed using NIHSS scale. Functional recovery was assessed based on the difference between NIHSS score on day of admission and 7th day. **RESULTS:** The maximum number of patients in our study were in the age group between 71 and 80 years. The mean age in both sexes was 62.71±14.09 years. male is to female ratio M:F is 1.38:1. Of the total100 patients 53 patients had stress hyperglycemia (RBG>140mg/dl) and 47 were normoglycemic (RBG<140). mean RBG value in stress hyperglycemic patients on admission was 183.06 ± 35.99 and mean RBG value in normoglycemic patients was 121.55 ± 14.03. Functional recovery which was better in normoglycemia patients compared to stress hyperglycemia patients. There was mortality in 5 patients with stress hyperglycemia. **CONCLUSION:** Functional recovery was better in the patients with normal RBG on admission than in patients having stress hyperglycemia. This suggests a possible association between stress hyperglycemia and poor outcome with stroke.

KEYWORDS: stress hyperglycemia, Acute ischaemic stroke.

INTRODUCTION

Stroke is the third most leading cause of death worldwide after coronary heart disease and cancer especially ischemic infarcts, comprise one of the most common devastating disorders.[1] They cause about 200,000 deaths each year in the united states and are a major cause of disability. The incidence of these cerebrovascular events increases with age and the number of strokes is projected to increase as the elderly population grows, with a doubling in stoke deaths in the united states by 2030. Most cerebrovascular diseases manifest by the abrupt onset of a neurologic deficit, as if the patient was ‘’struck by the hand of God’’.[2]

Hyperglycemia and Diabetes mellitus are more common in the hospital setting. In the 1989 National Health Survey, it was found that 24% of adults with diabetes and hyperglycemia are being hospitalized atleast once in the year.[3]

Of all acute ischemic stroke patients 20% to 50% have stress hyperglycemia at presentation.[4] Admission hyperglycemia in acute ischaemic stroke patients have been associated with longer in-hospital stay, increased cost and mortality.[5]

Large number of patient’s suffering from acute stress conditions like stroke may develop stress hyperglycemia, even in the absence of a preexisting diabetes.[6] Both human and animal studies suggest that stress hyperglycemia is associated with a high risk of mortality after stroke.[7]

Hyperglycemia is common in the early phase of stroke. The prevalence of stress hyperglycemia has been observed in two thirds of all ischemic stroke sub types on admission including lacunar strokes.[8] Hyperglycemia is not only common in the hospitalized patient, but is also being recognized as a marker for in-hospital mortality.
Factors contributing to stress hyperglycemia in the hospital setting are stroke, myocardial infarction, infections, corticosteroid therapy, medication or insulin omission, insulin errors.[9]

Identifying hyperglycemia as a marker for poor functional recovery and inhospital mortality has provided a rationale for the pursuit of tight glucose control. Benefits of tight glucose control include reduced mortality and decreased infection rates. Stroke patients who have stress hyperglycemia at admission have been associated with three fold higher risk of poor functional recovery and death.[10] Mortality risk was greater in patients who had hyperglycemia without a diabetes (representing stress hyperglycemia)than in those with diabetes.[11]

In our study we systemically reviewed the literature to summarize and assess the strength of the association between stress hyperglycemia and both short-term mortality and functional recovery in acute ischemic stroke patients.

STRESS HYPERGLYCEMIA IN STROKE

Diabetes and hyperglycemia are common in the hospital setting. In the 1989 National Health Interview Survey, 24% of adults with diabetes reported being hospitalized at least once in the previous year.[12]

In 1997, diabetes was the fourth most common comorbid condition in hospital discharges, and the prevalence of diabetes was 29% among cardiac surgery patients in 2001.[13]

From 1980 to 2003, the number of hospital discharges with diabetes as any-listed diagnosis more than doubled (from 2.2 to 5.1 million discharges)-an increase of 234.[14]

Hyperglycemia was present in 38% of adult non critically-ill medical and surgical patients admitted to one community teaching hospital, of whom 26% had a known history of diabetes and 12% were without prior diagnosis or recognition of diabetes.[15]

Stress hyperglycemia (also called stress diabetes or diabetes of injury) is transient elevation of the blood glucose due to the stress of illness. It usually resolves spontaneously, but must be distinguished from various forms of diabetes mellitus.[16] A high proportion of patients suffering an acute stress such as stroke or myocardial infarction[17] may develop hyperglycemia, even in the absence of a preexisting diagnosis of diabetes.

The definition of stress hyperglycemia also varied among studies. Most studies did not specify whether whole blood or plasma glucose was measured.

A random glucose level drawn on admission was used to define stress hyperglycemia in 10 of the 32 studies (with cutoffs ranging from 6 to 10 mmol/L [108 to 180 nwidL]). Another 9 studies based the definition of stress hyperglycemia on fasting glucose level the morning after admission (ranging from 6.1 to 7.8 mmol/L (110 to 141 mg/Dl).

In present study stress hyperglycemia was defined as random blood glucose levels >140mg/dl. Recognition of hyperglycemia as a marker for in-hospital mortality has provided a rationale for the pursuit of tight glucose control.

The American Association of Clinical Endocrinologists (AACE) Consensus Conference recommended blood glucose be maintained below 110 mg/dL in intensive care unit (ICU) patients, preprandial levels be maintained below 110 mg/dL and peak postprandial levels be maintained below 180 mg/dL in noncritically ill patients 128. Similarly, the American Diabetes Association (ADA) has proposed target blood glucose of about 110 mg/dl, 90-130mg/(11 and <180mg/dI for these three categories of hospitalized patients.[18]

Hyperglycemia is a common and costly health care problem in hospitalized patients. In hospital hyperglycemia is defined as any glucose value >7.8 mmol/l (140mg/dl). The American Diabetes Association and American Association of Clinical Endocrinologists consensus on inpatient hyperglycemia defined stress hyperglycemia or hospital-related hyperglycemia as any blood glucose concentration > 7.8 mmol/l (140 mg/dl).[19]

HISTORY

In multivariate analysis, as admission blood glucose increased, the odds for neurologic improvement decreased with an OR of 0.76 per 100mg/dl increase in admission glucose (95% CI 0.61-0.95), P = 0.01.[20]

Pulsinelli et al. 60 reported worse outcomes for both patients with diabetes and hyperglycemic patients without an established diagnosis of diabetes compared with those who were normoglycemic. Stroke-related deficits were more severe when admission glucose values were >120 mg/dl (6.7 mmo1/1).

Demchuk et al.[21] studied the effect of admission glucose level and risk for intracerebral hemorrhage or infarct when treatment with recombinant tissue plasminogen activator. The authors reported admission blood glucose and/or history of diabetes as the only independent predictors of hemorrhage.

Kiers et al.[22] prospectively studied acute stroke patients and threshold blood glucose for euglycemia was defined as fasting blood glucose <140 mg/di (7.8 mmo1/1). Patients were divided into four groups: euglycemia with no history of diabetes, patients with "stress hyperglycemia" (blood glucose >140 mg/dl, 7.8 mmo1/1 and HbA lc <8%), newly diagnosed diabetes (blood glucose >140 mg/dl, 7.8 mmol/l and HbA lc >8%) and known diabetes. No difference was found in the type or site of stroke among the four groups. Compared with the

euglycemic, nondiabetic patients, mortality was increased in all three groups of hyperglycemic patients.

Williams et al.[23] reported on the association of hyperglycemia and outcomes in a group of 656 acute stroke patients. 52% percent of the cohort had a known history of diabetes. Hlyperglycemia, defined as a random blood glucose >=130 mg/dl (7.22 mmol/l), was present in 40% of patients at the time of admission.

Hyperglycemia was an independent predictor of death at 30 day (RR 1.87) and at 1 year (RR 1.75) (both P <= 0.01). Other outcomes that were significantly correlated with hyperglycemia, when compared with normal blood glucose, were length of stay (7 vs. 6 days, P = 0.015) and charges ($6,611 vs. $5,262, P < 0.001).

Parsons et al[23] reported a study of magnetic resonance imaging (MR1) and MRS in acute stroke. Median acute blood glucose was 133.2 mg/di (7.4 mmo1/1), range 104.4-172.8 mg/dl (5.8-9.6 mmo1/1). A doubling of b71-eod glucose from 90 to 180 mg/dl (5-10 mmo1/1) led to a 60% reduction in penumbral salvage and a 56 cm3 increase in final infarct size.

Hala El Kawas study found that Acute hyperglycemia predicts increased risk of poor neurological and functional outcome. Measures to normalize blood glucose level in the setting of acute stroke could be of value in improving stroke outcome.[24]

Sagarbasu study suggests that stroke severity is the most important predictor of stroke outcome, with high sugar level as a marker of stroke severity.[25]

Bogdan timar study in 2013 concluded that: T2DM is a major risk factor for stroke. Plasma glucose level at admission was correlated positively with stroke mortality, both in patients with T2DM and in those without previously diagnosed T2DM, independently of other related factors.[26]

STRESS HYPERGLYCEMIA

DEFINITION

Stress hyperglycemia has been defined as hyperglycemia in previously euglycemic patients that corrects once the acute process resolves. Hyperglycemia occurs in 60% of the cases with acute stroke and in 12- 53% cases without the prior diagnosis of diabetes.

It imposes a range of adverse effects like abnormal immune function[27], hemodynamic and electromyocardial disturbances and increased infection rate.[28] Various studies have shown a direct relationship between the extent of stress hyperglycemia and severity and outcome of stroke, including mortality. Hyperglycemia in both diabetic and non- diabetic (i.e., stress hyperglycemia) patients is associated with poor prognosis both in terms of mortality and functional recovery, irrespective of patient's age, severity of condition or stroke sub- type.[29]

The WHO criteria of blood glucose for diagnosis of diabetes mellitus are used to define the minimum cut- off point for hyperglycemia as RPG ≥ 140 mg/dl. HbA1c cut point of 6.5% is used according to the recommendation of International Expert Committee in 2009.[30]

Hyperglycemia is a common and costly health care problem in hospitalized patients. In hospital hyperglycemia is defined as any glucose value >7.8 mmol/l (140mg/dl). The American Diabetes Association and American Association of Clinical Endocrinologists consensus on inpatient hyperglycemia defined stress hyperglycemia or hospital-related hyperglycemia as any blood glucose concentration > 7.8 mmol/l (140 mg/dl).[31]

PATHOPHYSIOLOGY

Hyperglycemia may be directly toxic to the ischemic brain. Accumulation of lactate and intracellular acidosis in the ischemic brain (produced through anaerobic cerebral glucose metabolism).[32] promotes and accelerates ischemic injury by enhancing lipid peroxidation and free radical formation[33] and impairing mitochondrial function.[34] These neurotoxic effects may be particularly important in the ischemic penumbra where neurons are injured but still viable.[35]

Hyperglycemia facilitates the development of cellular acidosis in the ischemic penumbra and results in a greater infarct volume, thus promoting the recruitment of potentially salvageable neurons into the infarction.

Hyperglycemic patients are relatively deficient in insulin. This leads to both reduced peripheral uptake of glucose (increasing the amount of glucose available to diffuse into brain) and increased circulating free fatty acids. Free fatty acids may impair endothelium-dependent vasodilation.[36]

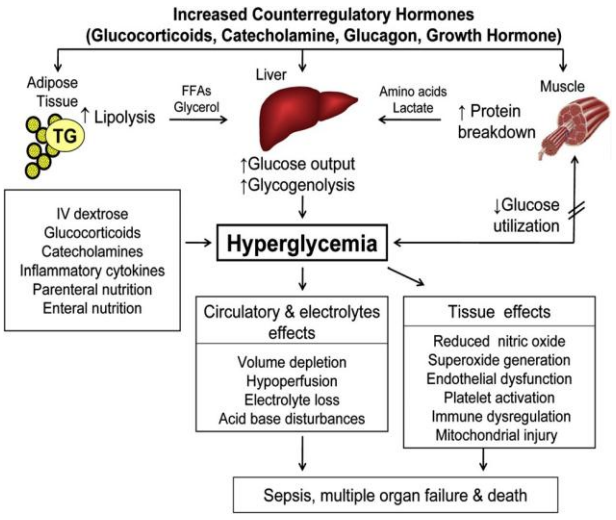

Fig 5. Showing pathogenesis of stress hyperglycemia.

Pathogenesis of stress hyperglycemia Stress hyperglycemia results from increased hepatic glucose production and impaired glucose utilization in peripheral tissues. Excess counterregulatory hormones (glucagon, cortisol, catecholamines and growth hormone) increases lipolysis and protein breakdown (proteolysis) and impaired glucose utilization by peripheral tissues. Hyperglycemia causes osmotic diuresis that leads to hypovolemia decreased glomerular filtration rate and worsening hyperglycemia. At the cellular level increased blood glucose levels results in mitochondrial injury by generating reaction oxygen species and endothelial dysfunction by inhibiting nitric oxide production.

Hyperglycemia increases levels of pro-inflammatory cytokine such as TNFa and IL-6 leading to immune system dysfunction, also increases plasminogen activator inhibitor-1 and fibrinogen causing platelet aggregation and hypercoagulable state. These changes can eventually lead to increased risk of infection, impaired wound healing, multiple organ failure, prolonged hospital stay.

Stress hyperglycemia patients are likely to have dysglycemia (ie, blood glucose level above the normal range but below the threshold for diabetes or undiagnosed diabetes[38] when not stressed. These patients have a higher risk of vascular disease than patients with normal blood glucose level.[39] These patients could sustain more ischemic damage at the time of infarction as a result of more extensive underlying cerebral vasculopathy compared with those who do not develop stress hyperglycemia. Hyperglycemia is an important determinant of the widespread changes in both small cerebral blood vessels[40] and large extracranial vessels seen in diabetic patients.[41]

Hyperglycemia may disrupt the blood-brain barrier[42] and promote hemorrhagic infarct conversion.[43] Higher admission serum glucose level is associated with a higher risk of hemorrhagic conversion of the infarct, with a substantial rise in risk with levels >8.4 mmol/L.[44]

Stress hyperglycemia may be a marker of the extent of ischemic damage in patients with stroke.

Hyperglycemia-Associated Reduction in Perfusion

Hyperglycemia causes 24% reduction in regional blood flow, reduction in blood circulation to the marginal ischemic areas and converts ischemic penumbra to infarct.[46] CO2-induced increase in cerebral blood flow is decreased in diabetics.[47] CO2-induced cerebral vasodilatation is mediated through NO and diabetics are known to have decreased endothelial NO production.[27]

Hyperglycemia and Thrombosis

Multiple studies have identified a variety of hyperglycemia-related abnormalities in hemostasis, favoring thrombosis.[48] Human studies in patients with type 2 diabetes have shown platelet hyperactivity indicated by increasedthromboxane biosynthesis Hyperglycemia-induced elevations of interleukin (IL)-6 levels have been linked to elevated plasma fibrinogen concentrations and fibrinogen Mrna.[49]

Increased platelet activation as shown by shear-induced platelet adhesion and aggregation on extracellular matrix has been demonstrated in patients with diabetes.[49]

In the healthy state, the vascular endothelium maintains the vasculature in a quiescent, relaxant, antithrombotic, antioxidant and antiadhesive state. Acute hyperglycemia may directly alter endothelial cell function by promoting chemical inactivation of nitric oxide, triggering production of reactive oxygen species (ROS) or activating other pathways.[37]

MANAGEMENT OF STRESS HYPERGLYCEMIA IN ACUTE ISCHEMIC STROKE

It is reasonable to treat patients with acute ischemic stroke according to American Diabetes Association inpatient glycemic control guidelines, initiating therapy to achieve glucose targets of 140to 180mg/dl if fasting glucose is greater than 140mg/dl or random glucose is constantly higher than 180mg/dl.

Lower glucose targets (<140mg/dl) may be appropriate for patients with well controlled diabetes and those with stress hyperglycemia. Lowering glucose levels less than 80mg/dl should be avoided.[50]

Patients who present with extreme or persistent hyperglycemia, are critically ill and should be started on intravenous insulin to improve blood glucose control for atleast 24 to 48hrs of hospitalization. They should then be transitioned to a subcutaneous insulin regimen that incudes basal long acting insulin and short acting insulin.[50]

MATERIALS AND METHODS

SOURCE OF DATA

- Hundred patients satisfying inclusion criteria presenting to RLJHRC are included in the study.
- Sample size is calculated with 80% power, 95% confidence interval, α error of 0.05, according to the formula.

$$n = \frac{2\,S^2\,(Z\alpha/2 + Z\beta)^2}{(d)^2}$$

METHOD OF COLLECTION OF DATA

- Informed consent has been obtained from all subjects.
- Baseline clinical data includes clinical history, examination. Data from those patients satisfying inclusion and exclusion criteria are collected.
- Patient status was evaluated by NIHS Scale on the day of admission and 7th day of admission and functional recovery was assessed based on difference of score.

INCLUSION CRITERIA FOR CASES

- Age> 18 yrs.
- Patients with CT evidence of acute ischemic stroke or progressive stroke < 24 hrs of onset.
- RBS >140 mg/dl at the time of admission.

INCLUSION CRITERIA FOR CONTROLS

- Age > 18 yrs.
- Patients with CT evidence of acute ischemic stroke or progressive stroke < 24 hrs of onset.

EXCLUSION CRITERIA FOR CASES

- Previously treated outside.
- Head injury, CT showing any space occupying lesion, bleed.

EXCLUSION CRITERIA FOR CONTROLS

- Previously treated outside.
- Head injury, CT showing any space occupying lesion, bleed.

INVESTIGATIONS DONE

- CT brain in 24hrs of admission.
- Glycated Haemoglobin levels.
- RBS at the time of presentation and on 7th day of admission.
- FBS, PPBS in next 24 hrs.

Statistical Methods

Data was entered into Microsoft excel data sheet and was analyzed using SPSS 22 version software. Categorical data was represented in the form of Frequencies and proportions. Chi-square was used as test of significance. Continuous data was represented as mean and standard deviation. Independent t test was used as test of significance to identify the mean difference between two groups. Paired t test was used to find the mean difference on day 1 and day 7 values. p value <0.05 was considered as statistically significant.

RESULTS AND DISCUSSION

The maximum number of patients in our study were in the age group between 71 and 80 years. The mean age in both sexes was 62.71±14.09years.male is to female ratio M:F is 1.38:1. Of the total100 patients 53 patients had stress hyperglycemia (RBG>140mg/dl) and 47 were normoglycemic (RBG<140). mean RBG value in stress hyperglycemic patients on admission was 183.06 ± 35.99 and mean RBG value in normoglycemic patients was 121.55 ± 14.03. Functional recovery which was better in normoglycemia patients compared to stress hyperglycemia patients. There was mortality in 5 patients with stress hyperglycemia.

DISCUSSION

This study was aimed at evaluating the outcome in acute ischemic stroke patients with stress hyperglycemia in terms of mortality and functional recovery based on National Institute of Health Stroke s Scale [NIH SS].[12] 100 patients were included in the study. These subjects were non diabetic patients.

CASE CHARACTERISTICS

AGE DISTRIBUTION OF SUBJECTS

The age of patients in this study ranged between 20 and 99 years. The maximum number of patients in this study were in the age group between 71 and 80 years. The mean age of both sexes was 62.71 + 14.09. this correlates with the findings of other authors.

STUDIES	PRESENT STUDY	FRAMINGHAM STUDY	SRIDHARAN STUDY
AGE in years	62.71 + 14.09	65 - 74	60 - 70

AII studies have shown that the rate of stroke increases with age. The Framingham study showed that only 1/5th of atherothrombotic strokes occurs below 65 years. The study also demonstrated a sharp increase of stroke after 65 years and a peak incidence of stoke between 65 – 74 years of age.[20]

Among the Indian studies, Sridharan[53] found a peak incidence of stroke between 60 – 70 years and Agarwal et al[58] showed a peak age incidence in the 6th decade. Abraham, Daniel and sunder Rao[116] study on South Indian subjects described a sharp increase in the prevalence of stroke after 60 years in both sexes with a peak prevalence above 70 years in males.

Nagaraja and Pratap chand[45] in their study from NIMHANS found that the peak incidence of stroke was in the 6th decade.

The WHO collaborative stroke study group from Rohtak, Haryana in 1974 reported that the prevalence of stroke increases from 86.96 /100,000 below 60 years to 622.22/100,000 above the age of 60.[14]

SEX DISTRIBUTION

In the present study male subjects were 58% and female subjects were of 42%.

In Kamel Abdelaziz mohamed study in 2013 male patients were 61% and female patients were (39%).[37]

In Abdu Hameed AI Kassir study in 2012 male patients were 67.6% and female patients were 32.4%.[38]

In Hala El Kawas study in 2006 male patients were 56.6% and female patients were 43.3%.[39]

PERCENTAGE	PRESENT STUDY	KAMEL ABDELAZIZ MOHAMED STUDY 2013	ABDUL HAMEED AI KASSIR STUDY 2012	HALA EL KAWAS STUDY 2006
MALES	58%	61%	67.6%	56.6%
FEMALES	42%	39%	32.4%	43.3%

MALE: FEMALE RATIO

The male: female ratio (M: F) is 1.38: 1 in the present study. As in the other studies, there is male excess of strokes.[107]

	PRESENT STUDY	AGARWAL STUDY	SRIDHARAN STUDY
M : F RATIO	1.38 : 1	5.8 : 1	2.3 : 1

In India a wide variation in male sex prominence is seen. Agarwal noted a male: female ratio of 5.8: 1 and Sridharan.[50]

PR0P0RTIONS OF STRESS HYPERGLYCEMIA

In our study.

We, have total of 100 patients of which 53 patients had **stress hyperglycemia** with RBS> 140 mg/dl at admission who were included as cases. Remaining 47 patients who had RBS < 140 mg/dl at admission were included as controls.

So in our study proportion of stress hyperglycemia was 53%.

In Kamel Abdelaziz mohamed study in 2013 proportion of stress hyperglycemia was 31%.[37]

In Abdul Hameed AI Kassir study in 2012 proportion of stress hyperglycemia was 36.8%.[38]

In Hala El Kawas study in 2006 proportion of stress hyperglycemia was 24%.[39]

PROPORTION OF STRESS HYPERGLYCEMIA	PRESENT STUDY 2015	KAMEL ABDELAZIZ MOHAMED STUDY 2013	ABDUL HAMEED AI KASSIR STUDY 2012	HALA EL KAWAS STUDY 2006
PERCENTAGE	53%	31%	36.8%	24%

We, can see that there is an increase in proportion of stress hyperglycemia, may be due to the modern life style changes.

ODDS RATIO

	PRESENT STUDY	BOGDAN TIMAR STUDY 2013	ALVAREZ –SABIN et al study
Odds ratio (OR)	1.979	3.63	8.4

The odds ratio for the present study was 1.979 less compared to the other studies.

MEAN RANDOM BLOOD GLUCOSE LEVELS AT ADMISSION

	PRESENT STUDY	ABDUL HAMEED AI KASSIR STUDY 2012	ACUTE STROKE TREATMENT TRIAL
MEAN RANDOM BLOOD GLUCOSE LEVELS AT ADMISSION	183.06 + 35.99	163 +40.3	144 + 68

The mean RBG on admission in the present study is higher compared to that of other studies.

NIHS SCORE

NIHS score was more in stress hyperglycemia patients (cases) than normoglycemia Patients (control).

Mean NIHSS score on day 1 in cases was 12.208 ± 2.34 and in controls was 9.404 ± 1.59.

	STRESS HYPERGLYCEMIA	NORMOGLYCEMIA
MEAN NIHSS SCORE ON ADMISSION	12.208 ± 2.34	9.404 ± 1.59.

NIHSS Score on 7th day of admission was also high in stress hyperglycemic patients compared to normoglycemic patients.

	STRESS HYPERGLYCEMIA	NORMOGLYCEMIA
MEAN NIHS SCORE on 7th day	8.98 ± 2.17	4.62 ± 1.63

Mean NIHSS score on day 7 in cases was 8.98 ± 2.17 and in controls was 4.62 ± 1.63. This difference was statistically significant. I.e. cases had higher scores of NIHSS than controls.

FUNCTIONAL RECOVERY BASED ON DIFFERENCE IN NIHSS SCORE

Mean Difference in NIHSS score was 4.79 in Normoglycemic patients.

Mean difference in NIHSS score was 3.16 in stress hyperglycemic patients.

	STRESS HYPERGLYCEMIA	NORMOGLYCEMIA
MEAN DIFFERENCE IN SCORE	3.16	4.79

More the difference in NIHS score, better is the functional recovery. So, if we see difference in the NIHS score was more in normoglycemic patients (controls) when compared to that of stress hyperglycemic patients.

Hence functional recovery was more in normoglycemic patients than stress hyperglycemic patients.

	MEAN RBG ON ADMISSION	NIHS SCORE ON DAY 1	NIHSS SCORE ON DAY 7	FUNCTIONAL RECOVERY(Difference between the score)
STRESS HYPERGLYCEMIA	183.06 + 35.99	12.208 ± 2.34	8.98 ± 2.17	3.16
NORMOGLYCEMIA	121.55 ± 14.03	9.404 ± 1.59.	4.62 ± 1.63	4.79

OUTCOME

In 53 patients of stress hyperglycemia 5 patients had mortality.

Percentage of mortality was 9.4%.

There was no mortality in normoglycemic patients.

	PRESENT STUDY	KAMEL ABDELAZIZ MOHAMED STUDY 2013	SAGAR BABU STUDY 2006
MORTALITY RATE IN STRESS HYPERGLYCEMIA PATIENTS	9.4%	25.8%	89%

Mortality was less in our study because of adequate treatment of stress hyperglycemia with insulin since the time of admission.

NUMBER OF DEATHS	MEAN RBS ON ADMISSION IN THESE PATIENTS	MEAN FBS IN NEXT 24HRS IN THESE PATIENTS	MEAN PPBS IN NEXT 24 HRS IN THESE PATIENTS	MEAN GLYCATED HAEMOGLOBIN
5	255	157.4	190.8	5.02

All these 5 patients had high RBG, FBS and PPBS values.

HbA1C was normal in these patients. Hence, this shows that stress hyperglycemia is associated with poor out come in terms of mortality compared to normoglycemic patients. Hence stress hyperglycemia has an impact on acute ischemic stroke patients and helps in accessing the prognosis in terms of functional recovery and outcome in terms of mortality.

It has been postulated that in cerebrovascular accidents an elevated blood glucose concentration is directly harmful to the ischemic brain. numerous studies have demonstrated that stress hyperglycemia augments the extent of ischemic brain damage.[74] mediated by anaerobic metabolism and consequent acidosis. hyperglycemia intensifies the risk of cerebral edema and mortality after stroke[71] this has given rise to the association between serum glucose concentration

immediatedly after stroke and subsequent morbidity and mortality.[71]

Williams et al 66 0ld stress hyperglycemia was present in 40% of patients at the time of admission, hyperglycemia was an independent predictor of death at 30 days.

Hala El Kawas study found that Acute hyperglycemia predicts increased risk of poor neurological and functional outcome. Measures to normalize blood glucose level in the setting of acute stroke could be of value in improving stroke outcome.[39]

Sagarbasu study suggested with high sugar level as a marker of stroke severity.[90]

Bogdan timar study in 2013 concluded that Type2DM is a major risk factor for stroke. Plasma glucose level at admission was correlated positively with stroke mortality, both in patients with T2DM and in those without previously diagnosed Type2DM.[91]

CONCLUSION

The maximum number of patients in the present study were in the age group between 71 to 80 years. Male patients were of 58% and female patients were of 42%. The male: female ratio (M: F ratio) is 1.38: 1. Stress hyperglycemia was noted in 53 patients and 47 patients were normoglycemics. Functional recovery (based on difference in then NIHSS score on day one and 7th day) was more in the normoglycemia patients compared to the stress hyperglycemic patients.

There was mortality in 5 patients (9.4%) with stress hyperglycemia patients. There was no mortality in normoglycemic patients. This suggest a possible association between stress hyperglycemia and bad outcome with stroke. Functional recovery and Outcome was better in our study compared to other study because of adequate treatment of stress hyperglycemia with insulin.

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