TO STUDY THE EFFECT OF HYPERGLYCAEMIA IN TRAUMATIC BRAIN INJURY

Palanisamy S[1], Gunalan S[2], Mahendran J. V[3]

[1]Assistant Professor, Department of Neuro Surgery, Government Mohan Kumaramangalam Medical College and Hospital, (GMKMC), Salem, Tamil Nadu.
[2]Assistant Professor, Department of Neuro Surgery, Dharmapuri Medical College, Dharmapuri, Tamil Nadu.
[3]Retd. Professor, Department of Neuro Surgery, Madras Medical College, Chennai, Tamil Nadu.

ABSTRACT

BACKGROUND

Traumatic brain injury (TBI) is the most common cause of mortality in the young- and middle-aged group in developing countries. As there is an increasing incident of type 2 diabetes mellitus, it plays an important role in any disease outcome.

MATERIALS AND METHODS

It is a comparative study of two groups of patients who had sustained traumatic brain injury with blood sugar of >150 mg/dL and <150 mg/dL. Each group had 30 patients. They were followed for 14 days with respect to outcome and complications.

RESULTS

The mean age was 36 ± 12 years, the mean GCS on admission was 9 ± 2 & the mean blood glucose level in the first 24 hours was 132 ± 52 mg/dL. GCS improvement was defective and mortality higher in blood sugar more than 150 mg/dL group when compared to the other.

CONCLUSION

In our study to determine the effect of hyperglycaemia in traumatic brain injury, there was a significant difference with reference to outcome of patient with hyperglycaemia at admission, with improvement with regard to GCS, increased length of stay and increased mortality rate when compared to the patients with normal glucose level.

KEYWORDS

Traumatic Brain Injury, Hyperglycaemia, GCS.

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BACKGROUND

Traumatic brain injury (TBI) occurs when an external force traumatically injures the brain. TBI can be classified based on severity, mechanism (closed or penetrating head injury), or other features (e.g., occurring in a specific location or over a widespread area). Head injury usually refers to TBI but is a broader category because it can involve damage to structures other than the brain, such as the scalp and skull. TBI is a leading cause of death and disability.

Although the head, face and neck comprise only 12% of the total body surface area exposed during an accident, these areas sustain disproportionally more injuries leading to death at the time of a crash.[1] Males are more at risk than females. The increased frequency of head injuries observed in men is consistent with the existing literature with a male to female ratio 6.1:1.[2]

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Corresponding Author:
Dr. S. Palanisamy,
Assistant Professor,
Department of Neurosurgery,
GMKMCH, Salem- 636007, Tamil Nadu.
E-mail: drpalanisamy72@gmail.com
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Brain trauma can be caused by a direct impact or by acceleration alone. In addition to the damage caused at the moment of injury, brain trauma causes secondary injury, a variety of events that take place in the minutes and days following the injury. These processes, which include alterations in cerebral blood flow and the pressure within the skull, contribute substantially to the damage from the initial injury.

Traumatic brain injury induces a complex pathophysiological cascade of cellular events. Central components of this response include increase in cerebral glucose uptake, reductions in cerebral blood flow, indiscriminate excitatory neurotransmitter release, ionic disequilibrium and intracellular calcium accumulation. Restoration of homeostasis requires significant increase in glucose metabolism. Experimental models have shown that TBI results in a significant increase of glucose utilization within the first 30 minutes post-injury, after which glucose uptake diminishes and then remains low for about 5-10 days.[3,4] The initial hyper glycolysis described above results from disruption of ionic gradients across the neuronal cell membrane, activating energy-dependent ionic pumps.[5]

Hyperglycaemia aggravates underlying brain damage and influences both morbidity and mortality in critically ill patients[6] by inducing tissue acidosis, oxidative stress, and cellular immunosuppression[7] which in turn promote the

development of multiorgan failure.[8] Hypoglycaemia impairs energy supply causing metabolic perturbation[9] and induces cortical spreading depolarization's.[10]

Studies have mentioned that intensive glycemic control is essential following traumatic brain injury for a better clinical outcome; controversially the majority of currently available clinical and preclinical evidence does not support tight glucose control (maintenance of blood glucose levels below 110-120 mg/dL) during the acute care of patients with severe TBI.[11]

Aims and Objectives

1. To determine the effects of hyperglycaemia on outcome in patients with traumatic brain injury.
2. To identify the relationship between admission hyperglycaemia and neurological outcome of patients with traumatic brain injury classified based on the Glasgow Coma Scale.

MATERIALS AND METHODS

Sixty consecutive traumatic brain injury patients admitted in the department of Neurosurgery, between the period of November 2015 to February 2016 were studied.

Inclusion Criteria

1. Patients above 13 years with traumatic brain injury.
2. Admission GCS between 6 and 15.

Exclusion Criteria

1. Patients with other causes of brain injury like ischemia, hypertension, and infection.
2. GCS less than 6
3. Age <13
4. Patients with severe life threatening musculoskeletal/ spine/ thoracoabdominal injuries
5. Patients who were administered glucose or exogenous steroids during the admission or transport
6. Patients with evidence of brain stem dysfunction
7. Patients not consenting to participate in the study

Methodology

Patients admitted with TBI were selected from neuro ICU and post- traumatic head injury wards in the Department of Neurosurgery, Government Mohan Kumaramangalam Medical College based on the above inclusion and exclusion criteria. Patients and their attenders were informed about the study and informed consent was obtained for enrolment in our study. Data regarding the demographic characteristics like age, sex, mode of injury, clinical evaluation pertaining to GCS, pupils and computed tomography (CT) finding/ diagnosis were recorded. Laboratory investigations including random blood sugar and HbA1c.

Values were done. Patient's GCS and admission blood glucose levels were registered. According to the blood glucose levels, 2 groups were formed among the 30 TBI individuals.

- Persons with <150 mg% of blood glucose
- Persons with >150 mg% of blood glucose

First 30 patients in this study with admission glucose of <150 mg% forms the study group. Second 30 consecutive patients with blood glucose of >150 mg% as the control group. In the study group, those with raised HbA1c were considered as previously uncontrolled diabetics. Those with normal HbA1c were considered to be having stress induced hyperglycaemia or newly diagnosed diabetics. In all these patients, admission GCS was recorded. These patients were studied for 14 days and were observed for daily variations in the GCS, random blood sugar. Hyperglycaemic patients were treated according to their blood glucose levels with human insulin (short acting) as advised by the diabetologist to strictly control blood sugar levels. Treatment for the head injury and its consequences was carried out as per the Institute's protocol. After 14 days, the outcome was assessed as discharged, continuing as inpatient or dead during the study period. The improvement or deterioration in GCS was also noted on the 14th day.

RESULTS

Age	No. of Patients	%
Below 20	07	11.6%
21-30	18	30%
31-40	11	18.3%
41-50	14	23.3%
Above 50	10	16.6%
Total	60	100%

Table 1. Age Wise Distribution

Sex		No. of Patients	%
Male	Female		
5	2	07	11.6%
13	5	18	30%
6	5	11	18.3%
6	8	14	23.3%
6	4	10	16.6%
36	24	60	100%

Table 2. Sex Wise Distribution

Age	Glucose Level				No. of Patients
	Below 150	151-250	251-350	Above 350	
Below 20	5	1	1	-	7
21-30	2	3	5	-	10
31-40	8	2	2	-	12
41-50	8	7	2	1	18
Above 50	7	-	5	1	13
Total	30	13	15	2	60

Table 3. Blood Sugar Level at the Time Admission

Glycaemic Range	No. of Patients
Non-diabetes	45
Known type 2 diabetes	15
Total	60

Table 4. Previous Glycaemic Range with History

Comorbid Conditions	No. of Patients
Diabetes+ Hypertension+ IHD	04
Diabetes + Hypertension	09
Hypertension	06
Diabetes	02
Others (hypothyroidism, COPD)	03
Total	24

Table 5. Comorbid Conditions with History

Types of Trauma	No. of Patients
Isolated head trauma	49
Poly trauma	11

Table 6. Types of Trauma

Causes of Trauma	No. of Patients
Traffic accident	34
Assault trauma	09
Falling down	09
Patient Found on public road	08
Total	60

Table 7. Causes of Trauma

In the present above study we divided the group A and group B.

- Group A: Below 150 Glucose level
- Group B: Above 150 glucose level

	Group A	Group B
Random Blood Sugar	Below 150	Above 150
GCS at Admission	10 ± 3	9 ± 2
HbA1c	5.74 ± 1.23	7.84 ± 1.87
GCS @ 7th Day	11 ± 3	20 ± 5
GCS @ 14th Day	12 ± 4	10 ± 3
Mortality	4 patients	8 patients
Still on Treatment	2 patients	14 patients

Table 8. Comparison between Group A and Group B

DISCUSSION

- A total of 60 patients were identified with TBI. Of these patients, 60% (36 patients) were males and females were 40% (24 patients).
- In our study, the most affected age group was 21-30 (30%). The available evidence indicates a mortality of 35% to 42% due to TBI, especially in patients between 15 and 25 years.[12-14]
- The mean age was 36 ± 12 years, the mean time of remission was 5 ± 8 hours, the mean GCS on admission was 9 ± 2 and the mean blood glucose level in the first 24 hours was 132 ± 52 mg/ dL. Multiple studies have evaluated the relationship between hyperglycaemia and the evolution of TBI patients and found that hyperglycaemia is associated with increased morbidity and mortality in patients with TBI. In a retrospective study by Jeremitsky et al., 77 patients who were admitted in ICU with severe TBI were followed with measurements of serum glucose level for 5 days, finding that among patients who died, the value of mean glucose level was greater than survivors (187.0 mg/dL vs. 153 mg/ dL). In the same undertaken study, the hyperglycaemia value was ≥170 mg/dL and patients who had 2 or more measures of hyperglycaemia had less survival.[15] Liu-DeRyke et al.,[16] developed a retrospective study of 380 ICU patients with TBI and found that levels ≥160 mg/dL in the first 24 hours of admission were associated with a poor outcome in terms of increased mortality or severity of injury. Donald et al., did a retrospective cohort study on ICU patients with severe TBI who survived at least 12 hours, and measured the fasting glucose level in the first 10 days. They found that levels of hyperglycaemia ≥11.1 mmol/L (≥200 mg/dL) were associated with a 3.6-fold increased risk of death compared to those with lower levels. Rovlias et al.,[17] in their prospective study on 267 patients with moderate or severe TBI, who underwent surgery, reported that patients with severe TBI had higher levels of hyperglycaemia. Hyperglycaemia was detected in 25% (15 patients).
- In our study patients affected with co morbid conditions like, Diabetes+ Hypertension+ IHD (4 patients), Diabetes + Hypertension (9 patients), Hypertension (6 patients), Diabetes (2 patients) and others (3 patients).
- Isolated head trauma was present in 49 patients (81.6%) and polytrauma in 11 patients (18.3%) of all patients.
- According to the causes of trauma, the most frequent ones were traffic accidents, 34 patients (56.6%) followed by assault trauma 9 patients (15%), falling down 9 patients (15%) and Patient found on public road 8 patients (13.3%).
- In our study we made two groups like Group A and Group B. Comparison between two groups with blood glucose level (Below 150 vs. Above 150), GCS level at the admission (10.30 ± 2.972 vs 9.4 ± 1.89), HbA1c level (5.74 ± 1.23 vs 7.84 ± 1.87), Mortality rate (4 patients vs. 8 patients) and Still on treatment (2 patients vs. 14 patients).

CONCLUSION

In our study to determine the effect of hyperglycaemia in traumatic brain injury, there was a significant difference with reference to outcome of patient with hyperglycaemia at admission, with improvement with regards to GCS, increased length of stay and increased mortality rate when compared to the patients with normal glucose level.

REFERENCES

[1] Carey ME. Learning from traditional combat mortality and morbidity data used in the evaluation of combat medical care. Military Medicine 1987;152(1):6-13.

[2] Jager TE, Weiss HB, Cohen JH, et al. Traumatic brain injuries evaluated in U.S. emergency departments 1992-1994. Acad Emerg Med 2000;7(2):134-140.
[3] Yoshino A, Hovda DA, Kawamata T, et al. Dynamic changes in local cerebral glucose utilization following cerebral conclusion in rats: evidence of a hyper- and subsequent hypo-metabolic state. Brain Res 1991;561(1):106-119.
[4] Hovda, D.A. Metabolic Dysfunction. In: Narayan RK, Wilberger JE, Povlishock JT, eds. Neurotrauma. San Francisco: McGraw-Hill 1996:1459-1478.
[5] Rosenthal M, LaManna J, Yamada S, et al. Oxidative metabolism, extracellular potassium and sustained potential shifts in cat spinal cord in situ. Brain Res 1979;162(1):113-127.
[6] Jeremitsky E, Omert LA, Dunham CM, et al. The impact of hyperglycemia on patients with severe brain injury. J Trauma 2005;58(1):47-50.
[7] Aronson D. Hyperglycemia and the pathobiology of diabetic complications. Adv Cardiol 2008;45:1-16.
[8] Sperry JL, Frankel HL, Vanek SL, et al. Early hyperglycemia predicts multiple organ failure and mortality but not infection. J Trauma 2007;63(3):487-493.
[9] Vespa P, Boonyaputthikul R, McArthur DL, et al. Intensive insulin therapy reduces micro-dialysis glucose values without altering glucose utilization or improving the lactate/pyruvate ratio after traumatic brain injury. Crit Care Med 2006;34(3):850-856.
[10] Strong AJ, Hartings JA, Dreier JP. Cortical spreading depression: an adverse but treatable factor in intensive care? Curr Opin Crit Care 2007;13(2):126-133.
[11] Marion DW. Optimum serum glucose levels for patients with severe traumatic brain injury. F1000 Med Rep 2009;1:pii: 42.
[12] Engel DC, Mikocka-Walus A, Cameron PA, et al. Pre-hospital and in-hospital parameters and outcomes in patients with traumatic brain injury: a comparison between German and Australian trauma registries. Injury 2010;41(9):901-906.
[13] Engberg A. Severe traumatic brain injury--epidemiology, external causes, prevention, and rehabilitation of mental and physical sequelae. Acta Neurol Scand Suppl 1995;164:1-151.
[14] Arabi YM, Haddad S, Tamim HM, et al. Mortality reduction after implementing a clinical practice guidelines-based management protocol for severe traumatic brain injury. J Crit Care 2010;25(2):190-195.
[15] Jeremitsky E, Omert LA, Dunham CM, et al. The impact of hyperglycemia on patients with severe brain injury. J Trauma 2005;58(1):47-50.
[16] Liu-DeRyke X, Collingridge DS, Orme J, et al. Clinical impact of early hyperglycemia during acute phase of traumatic brain injury. Neurocrit Care 2009;11(2):151-157.
[17] Rovlias A, Kotsou S. The influence of hyperglycemia on neurological outcome in patients with severe head injury. Neurosurgery 2000;46(2):335-342.