# ''The importance of monitoring serum calcium level in patients with Traumatic brain injury''

***[1] Dr.Harsha Gupta***
*Associate Professor, department of Anesthesia*
*chirayu medical college and hospital Bhopal*
*harshagupta13@yahoo.com*

***[2] Dr Manish Singh, Assistant Professor***
*Dept of General surgery*
*Birsa munda government medical college shahdolmp*
*drmanishsingh1977@gmail.com*

***[3] Dr. Ankush lokhande***
*Senior resident, department of general surgery,*
*NSCB medical college Jabalpur*
*Ankushlokhande562@gmail.com*

**Corresponding author**
**[4] Dr Kshama Shrivastava**
**Demonstrator Department of Pathology**
**Bundelkhand Medical College Sagar 471001**
**drkshama97@gmail.com**



Abstract

**Introduction–**Serum calcium is an important mediator of cell damage after TBI and cellular hypocalcemia may have a neuroprotective effect after brain injury Traumatic brain injury (TBI) is a leading cause of death and disability worldwide. Traumatic brain injury (TBI), sometimes referred to as acquired brain injury, significantly increases death and lifetime impairment. Traumatic brain injury accounts for 25% to 30% of all accidental deaths and 27% of trauma-related hospital deaths (TBI).

**Background:** The present study has been conducted to assess whether hypocalcaemia can be used as a prognostic factor in the outcome of traumatic braininjury.

**Materials And Methods:** This prospective study was done on 100 patients with moderate to severe traumatic brain injury in trauma centre of nscb medical college Jabalpur from 2020-2022. Serum calcium levels of patients had a Glasgow coma scale of 3–13 points following traumatic brain injury, with demonstrable intracraniallesionsincranial computed to mography were included. Student's t test, chi square test and Fisher's exact test were used for comparative analysis. Logistic regression and receiving operative curves analysis analysis were also done to assess the risk factors.

Results: Statistically significantdifference were found in the Ca2+levels on the 3rd day of admission between the patients with GOS ≤3 and the patientswith GOS>3 (P=0.029). The best level of higher sensitivity (86.27%) and specificity (68.66%) of hypocalcaemia on 3rdday was the Ca2+ value of 1.14 mmol/L.

**Conclusion:** The serum $Ca^{2+}$levels on day 3 could be useful for the prediction of mortality and disability in patients with moderate to severe traumatic brain injury.

**DOI Number: 10.48047/nq.2024.22.2.NQ24022 NeuroQuantology 2024; 22(2):206-210**

## INTRODUCTION:

Traumatic brain injury (TBI) is a leading cause of death and disability worldwide Traumatic brain injury (TBI), sometimes referred to as acquired brain injury, significantly increases death and lifetime impairment. Traumatic brain injury accounts for25%to 30% of all accidental deaths and 27% of trauma-related hospital deaths (TBI). Each year, 1.5 to 2 million people areinjured and over a million people die in India. [1]RTAs (60%) are the most common cause of TBIs, followed by falls (20%–25%) and violence (10%). Alcohol usage is recognizedto be a confounding factor in 15%–20% of TBIs at the time ofinjury. [2,3]Around the world, 3.2 million people are thought to be living with long-term disability brought on by TBI.[4] Although there have been significant advancements in the field of TBI research, the number of outcome predictors arenothugeinnumber. We still have to rely on the MRI findings to evaluate the long term prognosis.[5-7]

$Ca^{++}$ is an important mediator of cell damage after TBI and cellular hypocalcemia may have a neuroprotective effect after brain injury. We hypothesized that early hypocalcemia might have an adverse effect on the neurological outcome of patients suffering from isolated severe TBI. In this study, we aimed to evaluate the relationship between admission $Ca^{++}$ level and the neurological outcome of these patients.

aim- The present study has been conducted to assess whether hypocalcaemia can be used as a prognostic factor in the outcome of traumatic braininjury.

## MATERIALSANDMETHODS:

### Sampling

This prospective study was done on 100 patients with moderate to severe traumatic brain injury in trauma centre ofnscb medical college Jabalpur from 2020-2022. The ethical approval was obtained from the institutional ethical committee. A total of 100 patients whowere 18-75 years old and had a Glasgow Coma Scale score of3-13 following the TBI with demonstrable intracranial lesionsin cranial computed tomography were include in the study. Patients with the following characteristics were excluded: TBI older as 3 days, intake of medications, conditions or diseasesaffecting calcium metabolism (such as hyperparathyroidism,acute pancreatitis, massive blood transfusion, and treatment with hydrochlorothiazide), multisystem trauma, exposed fracture, lacerated spleen, liver, great vessels or hypovolemicshock III–IV, lesions in the brainstem as an isolated finding, previous treatment in an other clinic, pregnancy, hyperphosphatemia (>1.32mmol/L), hypomagnesemia (<0.61mmol/L), alcoholism, hypoalbuminemia at the hospitality admittance and prior disability toTBI.

### Methodology

The patients admitted to the emergency room were managed according to the Advanced Trauma Life Support guidelines. After stabilization, tests were conducted to measure hematicbiometry, blood chemistry and serum electrolytes (sodium, potassium, calcium, and ionized calcium). Additionally, arterial blood gases were taken. To provide further care for the patients, a crystalloid solution, gastric protector, analgesic, and sedative (if needed) were administered. Forintubation, propofol and rocuronium were used. At hospitaladmittance,

theclinicalvariablesincludedage, sexandpupillary reaction noted. Additionally, respiratory rate andheart rate, along with systolic blood pressure, diastolic bloodpressure, and mean arterial pressure were measured. All the parameters wereevaluated.

**Statistical Analysis**

The data was tabulated in Microsoft excel and analysed with SPSS v.24 software. The continuous variables are presentedwith mean and standard deviation. The categorical variablesare presented with frequency and percentage. Student's t-testwas used for the comparison of the continuous variables andChi-squared test or Fisher's exact test were used for thecomparison of the categorical variables. Logistic regressionwas used to identifythe potentialrisk factors. Receivingoperative curves (ROC) were also used to evaluate sensitivity, specificity, predicted positive value and negative predicted negative value for different points of clinical interest. The p value ≤0.05 is considered as statistically significant.

**RESULTS**:

-Among the 100 patients, 54 had Glasgow Outcome Score(GOS)≤3 and 46 had GOS>3. There were 64 males and 36females with age ranging from 18-75 years.

-The demographicand clinical variables are presented in Table 1. There were statistically significant difference between the GOS≤3 and GOS>3 groups in terms of GCS at admission, GCS at discharge, mean arterial tension(mmHg),presence of presence of anisocoria and pupillary reactivity, which can be noted as a risk factors.

Table 1: Demographic And Clinical Variables

| Parameters | GOS≤3(n=54) | GOS>3(n=46) | pvalue |
|---|---|---|---|
| Gender (male/female) | 31/23 | 33/13 | 0.136 |
| Age(years) | 38.2(18-72) | 42.7(20-75) | 0.144 |
| GCS atadmittance | 10.08±3.64 | 8.64±3.71 | 0.039 |
| GCSatdischarge | 9.08±1.02 | 13.65±5.02 | 0.006 |
| ICUdays | 24.02±14.21 | 21.25±13.07 | 0.377 |
| Meanarterialtension(mmHg) | 109.50±21.10 | 115.82±17.20 | 0.031 |
| Cardiac frequency | 110.46±31.33 | 109.30±31.37 | 0.914 |
| Respiratoryfrequency | 17.71±2.03 | 18.28±2.15 | 0.223 |
| pH | 7.40±0.07 | 7.37±0.05 | 0.183 |
| pHday3 | 7.45±0.08 | 7.42±0.09 | 0.207 |
| Isocoria (%)Yes<br>No | <br>38/54(70.4%)<br>16/54(29.6%) | <br>41/46(89.1%)<br>5/46(10.9%) | 0.010 |
| Pupillary reactivity (%)Yes<br>No | <br>29/54(70.4%)<br>25/54(29.6%) | <br>37/46(80.4%)<br>9/46(19.6%) | 0.004 |

- the difference in the blood parameters between the GOS≤3 and GOS>3 groups on three intervals which were day 0, day 3 and day 7. On the day 0, patients with GOS≤3 had higher total leukocytes count, potassium level and lower hematocrit, hemoglobin, sodium, calcium and glucose levels in comparison to the patients with GOS>3 but there were no statistically significant differences. On the day 3, patients with GOS≤3 had higher total leukocytes count, haematocrit, haemoglobin and lower sodium, potassium, calcium and glucose levels in comparison to the patients with GOS>3 and only the difference in the calcium level between the groups was statistically significant. On the day 7, patients

with GOS≤3 had higher total leukocytes count, haematocrit, haemoglobin, glucose and lower sodium, potassium, calcium levels in comparison to the patients with GOS>3 but there were no statistically significant differences.

Logistic regression analysis for the GOS≤3 group showed that, pupillary reactivity and hypocalcaemia on day 3 were the significantly potential risk factors.
- The ROC analysis showed that, the best level of higher 2+ sensitivity (86.27%) and specificity (68.66%) of Ca on 3rd day was the value of 1.14 mmol/L.

**DISCUSSION:**

The results of the study found that the Ca2+ values in serum on the third post-traumatic day to be a prognostic factor for mortality and morbidity in moderate/severe TBI, with a level of significance (p=0.012). A similar result was seen the study done by Vinas-Rios et al demonstrating a significant difference for serum hypocalcaemia at day 3 after TBI between [8] survivors and non-survivors. Neuronal death leads to hypocalcaemia that results in the development of cerebral edema. Patients with poor outcome had an impaired pupillary reactivity which was an indirect sign. Impaired pupillary reactivity is an important clinical sign for raised intracranial pressure with imminent risk of cerebral herniation/cerebral ischemia [9,10] correlating with bad prognosis. In the present study there was an association of hypocalcemia at3 day in ionized calcium after moderate/severe TBI with bad outcome (GOS ≤3). A variety of mechanisms have been postulated to be involved in TBI such as neuroinflammation, neuronal hypoxia, loss of cerebral vessel autoregulation, and brain edema with MRI as a reliable prognostic marker. [11-14]

Based on the level of hypocalcemia, there is significant variation in the risk for the patient to die or suffer moderate/severe disability. A worst or poor outcome, defined as death or moderate/severe disability respectively, was 2+ evident in all patients with a level of Ca of 0.98 mmol/L or lower. In addition, our results demonstrated that 86.27% of patients had an unfavorable outcome, consisting of death or moderate/severe disability when the serum hypocalcemia level was lower than 1.14 mmol/L.

**CONCLUSION:**

From the results of this study, this can be concluded that, theserum levels of $Ca^{2+}$on day 3 as well as impaired pupillaryreactivity are significantly associated with higher mortalityand disability rates in patients with moderate and severe TBI.The value of $Ca^{2+}$of 1.14 mmol/L can be utilized as a reliablecut-off point. Therefore, the medical professionals shouldhave a keen eye on the hypocalcemia in the patients withtraumatic brain injury to have a clear understanding abouttheirmorbidityandmortality.

REFERENCES:

1. Kirankumar MR, Satri V, Satyanarayana V, Ramesh Chandra VV, MadhusudanM, Sowjanya J. Demographic profile, clinical features, imaging and outcomesin patients with traumatic brain injury presenting to emergency room. J ClinSciRes2019;8:132-6.
2. JennettB.Epidemiologyofheadinjury.ArchDisChild1998;78:4036.
3. Gururaj G. Epidemiology of traumatic brain injuries: Indian scenario. NeurolRes2002;24:24-8.
4. Corrigan JD, Selassie AW, Orman JA. The epidemiology of traumatic braininjury.TheJournalofheadtraumarehabilitation.2010;25(2):72–80.
5. Woischneck D, Lerch K, Kapapa T, Skalej M, Firsching R. Predictive quality ofthe injury severity score in the systematic use of cranial MRI. Z OrthopUnfall2010;148:548-53.
6. Di Battista AP, Rhind SG, Baker AJ. Application of blood-based biomarkers inhumanmildtraumaticbraininjury.Fr

ontNeurol2013;4:44.
7. HästbackaJ,PettiläV.Prevalenceandpredictivevalueofionizedhypocalcemiaamongcriticallyillpatients.ActaAnaesthesiolScand2003;47:1264-9.
8. Manuel VRJ, Martin SA, Juan SRJ, Fernando MAL, Frerk M, Thomas K, et al.Hypocalcemiaasaprognos!ticfactorinmortalityandmorbidityinmoderateandseveretraumaticbraininjury. AsianJNeurosurg2015;10:190-4.
9. Dickerson RN, Morgan LM, Martin A. Croce treatment of moderate to severeacute hypocalcemia in critically trauma patients. JEPN J ParenterNutr2007;31:228.
10. Zivin JR, Gooley T, Zager RA, Ryan MJ. Hypocalcemia: A pervasive metabolicabnormalityinthecriticallyill.AmJKidneyDis2001;37:689-98.
11. DiasCR,LeiteHP,NogueiraPC,BrunowdeCarvalhoW.Ionizedhypocalcemiais an early event and is associated with organ dysfunction in children admitted to the intensive care unit.JCritCare2013;28:810-5.
12. Murillo-Rodriguez. Head trauma in the child and teenager. ModerateTraumaticBrainInjury.1sted.México,D.F:McGrawhill;2007.p.46-7.
13. Buriticá E, Villamil L, Guzmán F, Escobar MI, García-Cairasco N, Pimienta HJ.Changes in calcium-binding protein expression in human cortical contusiontissue.JNeurotrauma2009;26:2145-55.
14. LucasSM,RothwellNJ, GibsonRM.Theroleofinflammation in CNS injury and disease. BrJ Pharmacol 2006;147 Suppl1:S232-40.
15. Balbino M, Capone Neto A, Prist R, Ferreira AT, Poli-de-Figueiredo LF. Fluidresuscitation with isotonic or hypertonic saline solution avoids intraneuralcalcium influx after traumatic brain injury associated with hemorrhagicshock.JTrauma2010;68:859-64.
16. ZhangM,ShanH,GuZ,WangD,WangT,WangZ,etal.Increasedexpressionofcalcium/calmodulin-dependent protein kinase type II subunit d after rat traumatic brain injury.JMolNeurosci2012;46:631-43.