**ORIGINAL ARTICLE**

# Effectiveness of Magnesium Sulfate for the Treatment of Severe Traumatic Brain Injury

SAFDAR HUSSAIN ARAIN[1], NAJMUS SAQIB ANSARI[2], HAMID AKBAR SHAIKH[3], MUMTAZ ALI NAREJO[4], RUQAIA KANWAL[5]

*[1]Associate Professor/Head, Dept of Neurosurgery, Gambat Medical College Pir Abdul Qadir Shah Jeelani Institute of Medical Sciences*
*[2]Assistant Professor, Department of Neurosurgery, Ghulam Muhammad Mahar Medical College Sukkur*
*[3]Associate Professor, Department of Neurosurgery, Peoples University of Medical & Health Sciences for Women, Nawabshah Shaheed Benazir Abad*
*[4]Senior Registrar, Department of Neurosurgery, Khairpur Medical College, Khairpur Mirs*
*[5]Consultant, Gynaecologist & Obstetrician/Women Medical Officer, Sindh Government Hospital Liaqatabad Karachi*
*Correspondence to Dr. Safdar Hussain Arain E-mail: drsafdararain@gmail.com Cell 0321-2318923*

## ABSTRACT

**Aim:** To determine the effectiveness of magnesium sulfate for the treatment of severe traumatic brain injury.
**Study design:** Prospective study
**Place and duration of study:** Department of Neurosurgery, Gambat Medical College Pir Abdul Qadir Shah Jeelani Institute of Medical Sciences Gambat from 1st January 2020 to 30th June 2020.
**Methodology:** Fifty five patients of both genders were included in this study. Enrolled patients were aged between 20-60 years. Patients detailed demographics age, sex and body mass index were recorded after taking written consent. Glasgow coma score were recorded in this research at first day of admission in hospital and at 5th day.
**Results:** Thirty nine (70.91%) patients were males and 16 (29.09%) patients were females. Mean age of the patient was 40.02±8.78 years with mean body mass index 26.21±9.52 kg/m$^2$. The mean duration of the post traumatic brain injury was 7.19±1.23 hours. Mean Glasgow coma score without magnesium sulfate at first day was 6.64±1.23 but at the 5th day Glasgow coma score was 10.14±1.65. Significantly difference was observed with p value <0.0126. But significantly difference was not observed in Glasgow coma score with respect to gender and duration of diseases.
**Conclusion:** The use of magnesium sulfate was effective for the treatment of traumatic brain injury among patients. Glasgow coma score among patients were improved by using magnesium sulfate at the 5th day.
**Keywords:** Magnesium sulfate, Traumatic brain injury, Glasgow Coma Score (GCS)

## INTRODUCTION

Traumatic brain injury (TBI) is caused by a sudden brain shock. The tuberculosis affects everyone of any age and demonstrates high morbidity and mortality. After TBI, in patients with morbid disabilities, financial, mental and social effects for their lives become permanently damaged. Magnesium deficiency has been discovered in the brain and the blood of animals after cerebral damage has taken place. The use of magnesium minimized the neurobehavioral and pathological transition in animal models of brain injury. Nevertheless, two clinical trials on a magnesium-based neuroprotective agent revealed conflicting findings in TBI patients.[1,2] With secondary brain insults and other parameter adverse effects of the clinical results, those with brain damage were identified and may have adverse effects on results of clinical studies on magnesium-based therapeutic efficacy in care.

Pharmacokinetic and pharmacodynamic experiments in normal rats have shown, but pharmacokinetic research in humans with brain insults has shown, after systemic management, that magnesium cannot reach the brain[3,4], that the parenteral administration of magnesium in cerebrospinal fluid (CSF) has not resulted in a simultaneous increase in magnesium. Blood–brain barrier permeability for peripheral magnesium, which may have been a limiting factor in TBI patients' influence, can be limited by control of the brain and the CSF by the central nervous system.[5-7]

In the brain degeneration process that follows TBI, several biochemical pathways are involved. A single agent treatment may cause a lack of knowledge at a safe dose or adverse effects at a therapeutic or repeated dose. A neuroprotective therapy that is clinically effective must be structured to manipulate these pathways across many agents to achieve synergy. Apart from magnesium, other pharmacological agents[8,9] and physiological intervention are now being investigated for the treatment of tuberculosis, such as hyperoxia and hypothermia (examined somewhere else in this issue). Dexanabinol and progesterone were tested in clinical trials among the pharmacological agents. In a Phase III analysis, Dexanabinol was safe but not successful.[10] We assume that increasing the bioavailability of the brain of mannitol magnesium, pharmacology cotherapy and physiological treatments at the lowest safe and efficient dose may contribute to a clinically effective TBI treatment neuroprotective scheme.

## MATERIALS AND METHODS

This prospective study was conducted at Department of Neurosurgery, Gambat Medical College Pir Abdul Qadir Shah Jeelani Institute of Medical Sciences Gambat from 1st January 2020 to 30th June 2020 and comprised of 55 patients. Patients detailed demographics age, sex, body

Received on 14-10-2020
Accepted on 03-01-2021

mass index were recorded after taking written consent. The age of the patients was between 20-60 years. Patients with ay metabolic diseases, pregnant women and patients less than 20 years of age were excluded. The CT scantest were performed on each patients. The periodic mode of therapy for TBI was the implementation of a typical predictor treatment (reinforced brain trauma directives, the transition of night time percussion, intravenous fluids, Individual, treatment, food, depression, operation when presented, required surgery) and the ingestion of patients addition to the therapy treatments contain magnesium sulfate. The starting loading doses after the trauma resulted in 50mg/kg magnesium sulfate, then 15 mg/kg TDS after a threatened duration for care directives. Glasgow Coma score (GCS) were recorded in this research at first day of admission in hospital and at the 5$^{th}$ day. T test was used to compare the results significantly. Complete data was analyzed by SPSS 24.

## RESULTS

There were 39 (70.91%) male patients and 16 (29.09%) female patients. Mean age of the patients was 40.02±8.78 years with mean BMI 26.21±9.52 kg/m$^2$. Mean duration of the post traumatic brain injury was 7.19±1.23 hrs (Table 1).

Mean GCS without magnesium sulfate at first day was 6.64±1.23 but at the 5$^{th}$ day GCS was 10.14±1.65. Significantly difference was observed with p value <0.0126. Glasgow coma score was 8.96±4.65 at age <40 years and it was 10.92±1.75 in age > then 40 years. But significantly difference was not observed in GCS with respect to gender and duration of disease (Table 2)

Table 1: Demographics of the traumatic patients

| Variable | No. | % |
|---|---|---|
| **Gender** | | |
| Male | 39 | 70.91 |
| Female | 16 | 29.09 |
| Mean age (years) | 40.02±8.78 | |
| Mean BMI (mg/m$^2$) | 26.21±9.52 | |
| Mean post traumatic injury (hrs) | 7.19±1.23 | |

Table 2: Distribution of GCS pre/ post using of magnesium sulfate

| Variable | Glasgow coma score | P value |
|---|---|---|
| **Magnesium sulfate** | | |
| At first day | 6.64±1.23 | 0.0126 |
| At 5$^{th}$ day | 10.14±1.65 | |
| **Age** | | |
| <40 years | 8.96±4.65 | 0.56 |
| > 40 years | 10.92±1.75 | |
| **Gender** | | |
| Male | 9.40±3.80 | 0.92 |
| Female | 7.96±4.48 | |
| **Duration of disease** | | |
| At 5hours | 9.40±3.98 | 0.92 |
| >5hours | 7.96±4.76 | |

## DISCUSSION

This research presents data on the effectiveness of magnesium sulphate therapy in disorderly headache patients. This research has contrasted GCS with related GCS sulphate feedback on 5th before and after magnesium sulphate at the start of day management. Tests will substantially monitor an increase in GCS magnesium sulphate treatment. The effectiveness and safety models in the CSF for magnesium concentrations have to be monitored by human studies.[11] In one randomized, controlled trial provided magnesium therapy efficacy evidence for patient GCS and GOS evaluations. They analyzed the test results with a team not caused by magnesium. The result was not enough from usual brain injury care.[12]

In this study, a total of 55 patients enrolled were 70.91% of the patients. Patients' average age was 40.02±8.78, mean BMI 26.21±9.52 kg/m2 The mean post-traumatic brain injury length was 7.19±1.23 hours. Our findings were close to those of Li et al.[13] The enrolled patients were aged in our sample between 20-60 years. The mean GCS for the first day was 6.64±1.23 but the mean GCS for the fifth day was 10.14±1.65. There was a substantial difference of $p < 0.0126$. More evidence about the outcome of combination therapy in patients with magnesium and hypothermia were discovered. This demonstrated that magnesium sulphate was beneficial for traumatic injury and analysis of the success of combination therapies using magnesium and hypothermia.[14]

An I/V infusion study of magnesium sulphate resulted in a merely insignificant rise in magnesium sulphate CSF levels.[15,16] Significant improvement in this evaluation has been indicated by the threshold results for therapies for patients with standardized TBI.[17] No combination effects in people with TBI with magnesium and hyperoxia have been reported. Through his study, Ali et al[18] also provided the very results of the more effective use of magnesium sulphate for traumatic patients. Glasgow coma scale has improved significantly in patients whose disease has a positive effect, following treatment with magnesium sulphate. Patients with TBI should have a better outcome. Further study must therefore be carried out to generalize the findings.

## CONCLUSION

The use of magnesium sulfate was effective for the treatment of traumatic brain injury among patients. Glasgow coma scale among patients was improved by using magnesium sulfate at the 5$^{th}$ day.

## REFERENCES


1. Temkin NR, Anderson GD, Winn HR, et al. Magnesium sulfate for neuroprotection after traumatic brain injury: a randomised controlled trial. Lancet Neurol 2007;6:29-38.
2. Dhandapani SS, Gupta A, Vivekanandhan S, Sharma BS, Mahapatra AK. Randomized controlled trial of magnesium sulphate in severe closed traumatic brain injury. Indian J Neurotrauma 2008; 5:27-33.
3. Hallak M, Berman RF, Irtenkauf SM, Evans MI, Cotton DB. Peripheral magnesium sulfate enters the brain and increases the threshold for hippocampal seizures in rats. Am J Obstet Gynecol 1992;167:1605-10.
4. Hoane MR. Assessment of cognitive function following magnesium therapy in the traumatically injured brain. Magnes Res 2007; 20:229–236.
5. Fuchs-Buder T, Tramèr MR, Tassonyi E. Cerebrospinal fluid passage of intravenous magnesium sulfate in neurosurgical patients. J Neurosurg Anesthesiol 1997;9:324-8.

6. McKee JA, Brewer RP, Macy GE, et al. Analysis of the brain bioavailability of peripherally administered magnesium sulfate: a study in humans with acute brain injury undergoing prolonged induced hypermagnesemia. Crit Care Med 2005;33:661-6.
7. Thurnau GR, Kemp DB, Jarvis A. Cerebrospinal fluid levels of magnesium in patients with preeclampsia after treatment with intravenous magnesium sulfate: a preliminary report. Am J Obstet Gynecol 1987;157:1435-8.
8. Stoica B, Byrnes K, Faden AI. Multifunctional drug treatment in neurotrauma. Neurotherapeutics 2009;6:14-27.
9. Vink R, Nimmo AJ. Multifunctional drugs for head injury. Neurotherapeutics 2009;6:28-42.
10. Maas AI, Murray G, Henney H 3rd, et al. Efficacy and safety of dexanabinol in severe traumatic brain injury: results of a phase III randomized, placebo-controlled, clinical trial. Lancet Neurol 2006; 5:38-45.
11. Coronado VG, Thomas KE, Sattin RW, Johnson RL. The CDC traumatic brain injury surveillance system: characteristics of persons aged 65 years and older hospitalized with a TBI. J Head Trauma Rehab 2005;20(3):215-28.
12. Boake C, McCauley SR, Levin HS, Pedroza C, Contant CF, Song JX, Brown SA, Goodman H, Brundage SI, Diaz-Marchan PJ. Diagnosticcriteria for postconcussional syndrome after mild to moderate traumatic brain injury. J Neuropsychiatr Clin Neurosci 2005;17(3):350-6.
13. Li W, Bai YA, Li YJ, Liu KG, Wang MD, Xu GZ, et al. Magnesium sulfate for acute traumatic brain injury. J Craniofac Surg 2015;26(2):393-8.
14. Wang F, Wang Y, Sun T, Yu HL. Hyperbaric oxygen therapy for the treatment of traumatic brain injury: a meta-analysis. Neurol Sci 2016;37(5): 693-701.
15. Zhu H, Meloni BP, Bojarski C, Knuckey MW, Knuckey NW. Post-ischemic modest hypothermia (35°C) combined with intravenous magnesium is more effective at reducing CA1 neuronal death than either treatment used alone following global cerebral ischemia in rats. Exp Neurol 2005;193:361-8.
16. Dutca LM, Stasheff SF, Hedberg-Buenz A, Rudd DS, Batra N, Blodi FR, et al. Early detection of subclinical visual damage after blast-mediated TBI enables prevention of chronic visual deficit by treatment with P7C3-S243. Investigative Ophthalmol Visual Sci 2014;55(12):8330-41.
17. Nortje J, Coles JP, Timofeev I, et al. Effect of hyperoxia on regional oxygenation and metabolism after severe traumatic brain injury: preliminary findings. Crit Care Med 2008;36:273–81.
18. Ali K, Irfan M, Abbas R. Effects of magnesium sulfate therapy on GCS scores in patients with severe traumatic brain injury. Pak J Neurol Surg 2020; 24(4): 322-7.