

Magnesium Sulfate Therapy's Effects on GCS Scores in Patients with Severe TBI

SAQIB KHALIL¹, BADAR UDDIN UJJAN², MIAN AYAZ UL HAQ³, HAFIZ FAHAD ULLAH SAEED⁴, FAYAZ AHMED MEMON⁵, SANGEET⁶

¹Assistant Professor, Women medical college Abbottabad, Consultant neurosurgeon Jinnah International hospital Abbottabad

²Assistant Professor Neurosurgery, Dow International Medical College and Hospital Ojha Campus

³Assistant professor, Department of Neurology Hayatabad Medical Complex

⁴Medical Student, Department of Neurology, Mayo Hospital/ King Edward Medical University Lahore

⁵Professor Department of Medicine, Muhammed Medical College & Hospital, Mirpurkhas, Sindh.

⁶Post graduate trainee Deptt of psychiatry, Jinnah post graduation medical centre karachi

Corresponding author: Mian Ayaz ul Haq, Email: drayazulhaq@gmail.com

ABSTRACT

Objective: The main purpose of this study is to examine whether or not magnesium sulphate improves the Glasgow Coma Scale (GCS) scores of individuals who have suffered severe traumatic brain injury.

Study Design: Prospective study

Place and Duration: Women medical college Abbottabad and Department of Neurology Hayatabad Medical Complex, during from Jan, 2021 to June, 2021.

Methods: This research includes 72 participants of both sexes. Patients were between the ages of 18 and 55 when they were enrolled. All the patients had severe traumatic brain injury and magnesium sulphate treatment was administered to each patient. All of the patients' demographic information was obtained after they signed an informed permission form. In this study, the Glasgow coma score (GCS) was measured on the first and fourth days of hospitalization, respectively. The SPSS 20.0 version was used to analyze all of the data.

Results: Among 73 included patients, males were higher in numbers 41 (56.9%) than females 31 (43.1%). Presented patients had mean age 43.6±9.39 years and had mean BMI 25.8±12.83 kg/m². Post traumatic mean time of brain injury was 6.16 ±4.41 hours. At first day without magnesium sulfate mean GCS of the patients was 7.3±8.30 but at the 4th day GCS was 11.9±6.42 with p value < 0.04. GCS, on the other hand, showed no significant differences between gender and illness duration.

Conclusion: The results of this study led us to the conclusion that the use of magnesium sulphate was an effective method for treating traumatic brain injury in patients. GCS was improved among patients when magnesium sulphate was administered on the seventh day.

Keywords: Magnesium Sulfate, traumatic brain injury, Glasgow Coma Score (GCS)

INTRODUCTION

There is a direct link between traumatic brain injury (TBI) and CNS dysfunction because of an external impact to the head. To gauge the severity of a brain injury, many measures are used, the most popular of which being the Glasgow Coma Scale (GCS). [1] It is a leading cause of mortality and morbidity in the globe, particularly among those under the age of 44. It is possible for a person to lose their ability to think or move because of a severe head injury. Ages 27 to 60 are the most prevalent age group affected.

Falling from a height or being hit by an automobile are the most common ways to injure one's head, followed by workplace accidents. [2] No suggested, standard pharmacological agent for the treatment of TBI has been established, despite all of the facts and data that have been presented. In order to avoid subsequent brain damage, proper medical care must be sought out immediately. [3] In the early stages of TBI, excitotoxic processes in the brain result in the death of neurons. A number of investigations have been carried out, but there is currently no medication available to interrupt this excitotoxicity loop that leads to cell death. [4,5]

Clinical trials on healthy rats have shown that systemic injection of magnesium may reach the brain [5], but research on human brain injuries has revealed that parenteral magnesium delivery does not generate a simultaneous increase in the CSF concentration of the mineral. [6,7] The peripheral administration of magnesium may be hindered by the BBB's permeability due to central nervous system regulation, making it less effective in treating individuals with traumatic brain injury.

There are a slew of molecular processes at play in TBI-induced brain degeneration. Treatment with a single drug may result in ineffectiveness at a safe dosage or in undesirable effects at a therapeutic dose or repeated dosing. Multiple drugs must be used to provide a synergistic effect in a clinically effective neuroprotective treatment to be effective. [8,9] There are several pharmaceutical medicines and physiological therapies, such as hyperoxia and hypothermia, being tested for the treatment of TBI. Dexanabol and progesterone are among the pharmacological substances that have been examined in clinical studies. This drug

was shown to be safe but ineffective during a phase III clinical trial. [10]

Neuroprotective effects on the NMDA and calcium channel receptors have been examined in several animal studies; magnesium also reduces excitatory neurotransmitter releases. [11,12] Mean GCS in individuals with a head injury after magnesium sulphate treatment was the primary goal of the research." See how magnesium affects patient outcomes during the short- and long-terms. For the welfare of the patients, this research may provide important information from which to draw conclusions. A comparison was made between our findings and those from other countries' research that were readily accessible.

MATERIAL AND METHODS

This prospective study was conducted at Women medical college Abbottabad and Department of Neurology, Hayatabad Medical Complex, during from Jan, 2021 to June, 2021 and comprised of 72 patients. After obtaining written agreement, we collected demographic data on the patients, including their age, gender, and BMI. Pregnant women and patients under the age of 18 were not allowed to participate in this research.

Patients in the study ranged in age from 18 to 55. Each and every one of the patients got a CT scan (brain plain). An extensive preoperative workup included a full blood count and other necessary investigations such as an LFT or RFT, as well as other baseline tests. Patients with traumatic brain injury (TBI) received normal care, as well as magnesium sulphate (nasogastric/Foleys passed, intravenous fluids, Mannitol infusion, antibiotics, nutrition, antiepileptics needed, surgery if appropriate), in addition to brain trauma recommendations. Patients with a traumatic brain injury (TBI) typically received standard protocol treatment (NG/Foleys passed, intravenous fluids administered, Mannitol infusion administered, antibiotics administered, nutrition administered, antiepileptics administered, surgery administered if indicated), as well as magnesium sulphate as an add-on therapy in addition to this standard protocol treatment. Magnesium sulphate was given at an initial dosage of 50 mg/kg, followed by 15 mg/kg T.D.S. up to 48

hours following the trauma. Both at the time of admission and again on the seventh day, we measured the GCS score and the serum magnesium levels.

The variable, such as serum, is a continuous one. Magnesium levels and age were expressed as a mean and standard deviation. Qualitative variables, such as gender and Glasgow Coma Scale grades, were provided as frequency and percentages in the study. Gender and age were taken into account when stratifying the data based on an outcome metric, the Glasgow coma scale score. Findings were compared and significance/insignificance results were identified using the T test. A significant result was defined as one with a p value less than 0.05.

RESULTS

Amon 73 included patients, males were higher in numbers 41 (56.9%) than females 31 (43.1%).(fig 1)

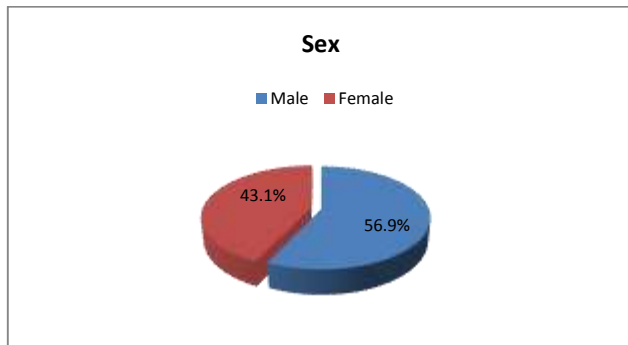


Figure-1: Sex distribution among all cases

Presented patients had mean age 43.6±9.39 years and had mean BMI 25.8±12.83 kg/m². Majority of the patients were from urban areas 45 (62.5%). Most common cause of TBI was falling followed by RTA and sports. Post traumatic mean time of brain injury was 6.16 ±4.41 hours. (table 1)

Table 1: The demographics of TBI patients

Variables	Frequency	Percentage
Mean age (years)	43.6±9.39	
Mean BMI (kg/m ²)	25.8±12.83	
Residency		
Urban	45	62.5
Rural	27	37.5
Causes of TBI		
Fall	30	41.7
RTA	25	34.7
Sports	17	23.6
Mean time of TBI (hrs)	6.16 ±4.41	

At first day without magnesium sulfate mean GCS of the patients was 7.3±8.30 but at the 4th day GCS was 11.9±6.42 with p value < 0.04. GCS, on the other hand, showed no significant differences between gender and illness duration. (table 2)

Table 2: GCS with and without magnesium sulfate

Variables	Frequency	P value
GCS		
At 1 st day	7.3±8.30	0.04
At 4 th day (Magnesium sulfate)	11.9±6.42	
Age distribution with GCS		
<35 years	9.11±6.13	0.98
>35years	9.88±12.99	
Sex Distribution with GCS		
Male	10.8±7.55	0.72
Female	9.12±4.63	
Duration of disease with GCS		
At 6 hrs	8.4±6.23	0.88
>6 hrs	9.1±3.41	

We found that 67 (93.1%) patients were satisfied and 5 (6.9%) cases were unsatisfied.(fig 2)

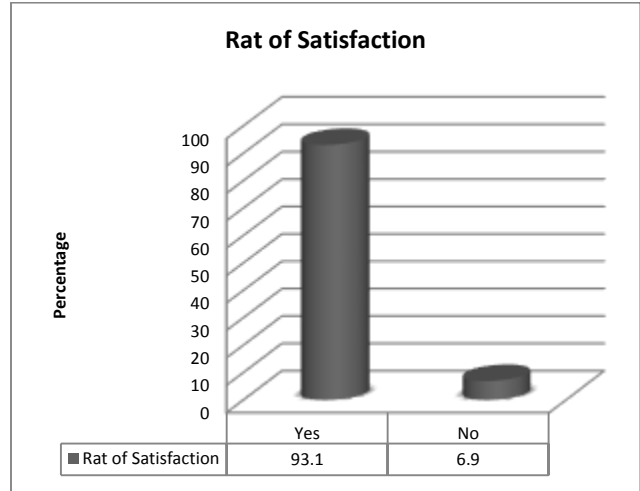


Figure-2: Rate of satisfaction among all cases

DISCUSSION

Numerous studies advocate a combination of treatments to prevent neuronal cell death after brain damage. Multiple factors are targeted by combination medicines, which have gained popularity and improved effectiveness over time. Neuroinjured individuals may benefit from the use of magnesium in conjunction with growth factors and glutamate antagonists as well as vitamin B immunomodulators and hypothermia as well as antioxidants. The combination of magnesium and hypothermia improved the outcomes of individuals with head injuries. The efficacy of magnesium and hypothermia as a combined treatment has to be studied further.[13]

The results of this research show that magnesium sulphate therapy is effective in treating individuals with a disordered headache. GCS before and after magnesium sulphate at the beginning of the day's management was compared to a comparable GCS sulphate feedback on the fifth day. Testing would be critical in determining whether magnesium sulphate therapy GCS has made a meaningful difference. It has been necessary to conduct human research in order to evaluate the effectiveness and safety of magnesium administration in the central nervous system. [14] Patients' GCS and GOS scores were assessed in a randomised, controlled experiment using magnesium therapy. Using a crew that hadn't been exposed to magnesium, they analysed the results of the tests. Normal brain damage therapy was insufficient to provide the desired result. [15]

Amon 73 included patients, males were higher in numbers 41 (56.9%) than females 31 (43.1%). Presented patients had mean age 43.6±9.39 years and had mean BMI 25.8±12.83 kg/m². Post traumatic mean time of brain injury was 6.16 ±4.41 hours. Most common cause of TBI was falling followed by RTA and sports. These results were comparable to the previous studies.[16,17] Patients' GCS and GOS scores were used to determine the efficacy of five days of magnesium treatment in one randomised controlled experiment. They contrasted the findings with those of a control group that only received normal treatment for traumatic brain injury without the addition of magnesium sulphate.[18]

In our study, At first day without magnesium sulfate mean GCS of the patients was 7.3±8.30 but at the 4th day GCS was 11.9±6.42 with p value < 0.04. GCS, on the other hand, showed no significant differences between gender and illness duration.[13]Neuroprotective effects of magnesium in preclinical models of ischemia and excitotoxic brain damage have been shown. [19,20] Magnesium shortage may shorten a victim's

lifespan, even in the case of a serious brain damage. [21] Magnesium has been shown to be safe and tolerable in a variety of clinical investigations, including those involving preeclampsia/eclampsia and myocardial infarction. The Mg²⁺ ion may influence cellular energy metabolism, vascular tone, and ion transport across the cell membrane, among other possibilities. It is common practise to utilise magnesium as an antagonist to plasma calcium.

CSF magnesium sulphate levels increased very little after an I/V infusion of magnesium sulphate. [22] Progress has been made in the evaluation of standardised TBI treatment thresholds, according to threshold results. [23] It has not been documented how TBI patients with magnesium and hyperoxia interact. In addition, Kazim Ali et al. found that magnesium sulphate was more effective in treating individuals with traumatic injury as a result of their investigation. [23] Among patients whose condition had a beneficial effect, therapy with magnesium sulphate resulted in significant improvements in GCS.[24] A better outcome is indicated for people with TBI. However, further study is needed to generalize the findings.

CONCLUSION

The results of this study led us to the conclusion that the use of magnesium sulphate was an effective method for treating traumatic brain injury in patients. GCS was improved among patients when magnesium sulphate was administered on the seventh day.

REFERENCES

- 1 Senathi-Raja D, Ponsford J, Schönberger M. Impact of age on long-term cognitive function after traumatic brain injury. *Neuropsychology*,2010;24(3):336
- 2 Mosenthal AC, Lavery RF, Addis M, Kaul S, Ross S, Marburger R, Deitch EA, Livingston DH. Isolated traumatic brain injury: age is an independent predictor of mortality and early outcome. *Journal of Trauma and Acute Care Surgery*,2002;52(5):907-11
- 3 Li W, Bai YA, Li YJ, Liu KG, Wang MD, Xu GZ, Shang HL, Li YF. Magnesium sulfate for acute traumatic brain injury. *J Craniofac Surg*. 2015;26(2):393-8.
- 4 Busingye DS, Turner RJ, Vink R. Combined Magnesium/Polyethylene Glycol Facilitates the Neuroprotective effects of magnesium in traumatic brain injury at a reduced magnesium dose. *CNS Neuroscience & Therapeutics*,2016;22: 854–859
- 5 Hallak M, Berman RF, Irtenskauf SM, Evans MI, Cotton DB. Peripheral magnesium sulfate enters the brain and increases the threshold for hippocampal seizures in rats. *Am J ObstetGynecol* 1992;167:1605–1610
- 6 Hoane MR. Assessment of cognitive function following magnesium therapy in the traumatically injured brain. *Magnes Res* 2007; 20:229–236.
- 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 ObstetGynecol* 1987;157:1435–1438
- 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 dexanabol in severe traumatic brain injury: results of a phase III randomised, placebo-controlled, clinical trial. *Lancet Neurol* 2006; 5:38–45.
- 11 Seel RT, Macciocchi S, Kreutzer JS. Clinical considerations for the diagnosis of major depression after moderate to severe TBI. *The Journal of Head Trauma Rehabilitation*,2010;25(2):99-112
- 12 Powell JM, Ferraro JV, Dikmen SS, Temkin NR, Bell KR. Accuracy of mild traumatic brain injury diagnosis. *Archives of physical medicine and rehabilitation*,2008;89(8):1550-5
- 13 Wang, F.; Wang, Y.; Sun, T.; Yu, HL. "Hyperbaric oxygen therapy for the treatment of traumatic brain injury: a meta-analysis". *Neuro Sci*. 2016; 37 (5): 693–701
- 14 Boake C, McCauley SR, Levin HS, Pedroza C, Contant CF, Song JX, Brown SA, Goodman H, Brundage SI, Diaz-Marchan PJ. Diagnostic criteria for postconcussional syndrome after mild to moderate traumatic brain injury. *The Journal of Neuropsychiatry and Clinical Neurosciences*,2005;17(3):350-6
- 15 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. *The Journal of Head Trauma Rehabilitation*,2005;20(3):215-28
- 16 Zhao L, Wang W, Zhong J, Li Y, Cheng Y, Su Z, Zheng W, Guan XD. The effects of magnesium sulfate therapy after severe diffuse axonal injury. *Ther Clin Risk Manag*. 2016 Sep 27;12:1481-1486
- 17 Shakeri M, Salehpour F, Ahmadvand A, Jafari R, Safaiyan A, Meshkini A. Effects of magnesium sulfate on the clinical course and GCS of patients with a severe diffuse axonal injury. *J Inj Violence Res*. 2012 Nov;4(3 Suppl 1):70.
- 18 Herrera-Melero MC, Egea-Guerrero JJ, Vilches-Arenas A, Rincón-Ferrari MD, Flores-Cordero JM, León-Carrión J, Murillo-Cabezas F. Acute predictors for mortality after severe TBI in Spain: Gender differences and clinical data. *Brain Injury*,2015;29(12):1439-44.
- 19 Vink R, O'Connor CA, Nimmo AJ, Heath DL. Magnesium attenuates persistent functional deficits following diffuse traumatic brain injury in rats. *Neurosci Lett*. 2003;336(1):41–44.
- 20 Heath DL, Vink R. Improved motor outcome in response to magnesium therapy received up to 24 hour after traumatic diffuse axonal brain injury in rats. *J Neurosurg*. 1999;90(3):504–509
- 21 Rowe WJ. Correcting magnesium deficiencies may prolong life. *Clin Interv Aging*. 2012;7:51–54
- 22 Dutca LM, Stasheff SF, Hedberg-Buenz A, Rudd DS, Batra N, Blodi FR, Yorek MS, Yin T, Shankar M, Herlein JA, Naidoo J. Early detection of subclinical visual damage after blast-mediated TBI enables prevention of chronic visual deficit by treatment with P7C3-S243. *Investigative Ophthalmology & Visual Science*,2014;55(12):8330-41
- 23 Kazim Ali, Muhammad Irfan, Rabia Abbas: Effects of Magnesium Sulfate Therapy on GCS Scores in Patients with Severe Traumatic Brain Injury. *Pak. J. of Neurol. Surg.* –2020–24 (4): 322-327
- 24 Moen KG, Brezova V, Skandsen T, Häberg AK, Folvik M, Vik A. Traumatic axonal injury: the prognostic value of lesion load in corpus callosum, brain stem, and thalamus in different magnetic resonance imaging sequences. *J Neurotrauma*. 2014;31(17):1486–1496.