International Journal of Science and Research (IJSR)
ISSN: 2319-7064
SJIF (2022): 7.942

Effect of Traumatic Brain Injury on Serum Magnesium

Dr. Tahreem Fatima[1], Dr. Muhammad Irshad[2], Dr. Muhammad Rizwan Sarwar[3], Dr. Rahmatullah Salah[4], Dr. Abdul Ghaffoor[5], Dr. Freshta Asekzai[6]

[1]Department of Neurosurgery, MHL, Lahore, Pakistan

[2]Department of Neurosurgery, MHL, Lahore

[3]Khawaja Muhammad Safdar Medical College, Sialkot

[4]Department of Neurosurgery, MHL, Lahore

[5]Mayo Hospital Lahore

[6]Kabul University of Medical Sciences, Afghanistan

[1]Author correspondence: *reemfatima528[at]yahoo.com*

Abstract: ***Objectives: To assess the effect on serum magnesium levels after traumatic brain injury. Methods: The study was conducted in the Neurosurgery Ward of Mayo Hospital Lahore, Pakistan. A total of 51 patients were included in the study. All the patients who presented with traumatic brain injury and were admitted for management in Neurosurgery Ward in the last six months of 2020 were included in this study. The blood sample for serum magnesium level was taken and recorded. A control group of similar age group with no disease was also included and their samples of serum Mg were taken for comparison with the test group. The post - admission trend of serum magnesium levels of all the participants after TBI was extrapolated and evaluated. Reference range of Normal serum magnesium level is 1.6 to 2.6mEq/L according to ABIM (American Board of Internal Medicine) Laboratory Test Reference Ranges January 2021. Results: A total of 51 participants with a mean age of 32 ± 15SD were included in the study. Among them, 41 were males and 10 were females. The most common cause of traumatic brain injury was found to be road traffic accident (RTA).40 out of 51 patients had a history of RTA making it up to 78%. Second most common cause was the presentation after a fall i. e.9out of 51; mostly involving elderly people. The serum magnesium level in all the 51 participants was collected and graphed.71% patients had a drop in magnesium level from standard minimum reference value i. e.1.6 mEq/L (i. e.1.9m/dl). The mean value of serum magnesium level was calculated as 1.68 ± 0.3sd. The control group (no disease) with the same number of participants and similar age group had their mean serum magnesium level of 2.3 ± 0.2 mg/dl. The p - value was calculated to be 0.00001 which was considered significant statistically (p=<0.05). Conclusions: Serum magnesium levels decline after traumatic brain injury due to various complex cascades. This decreasing trend has a significant impact on the prognosis of the TBI patients as according to literature magnesium has a role in preventing secondary brain injury mechanisms. By extrapolating evident hypomagnesemia, this study highlights the need for conducting trials to add magnesium supplementation in managing patients with TBI.***

Keywords: Traumatic brain injury, brain trauma, serum magnesium, hypomagnesemia, head injury

1. Introduction

The advancement of technology and inculcation of this dynamic automation in our daily life has led us to face new challenges. Traumatic brain injury is a growing concern for all health officials contributing significantly to morbidity and mortality in all trauma related injuries worldwide. TBI represents any blow or penetrating injury to head affecting GCS (Glasgow Coma Scale), memory, mental status and neurological function of the individual. [(1)] The global incidence of all - cause, all - severity TBI is estimated at 939 cases per 100, 000 people; thus, an estimated 69.0 million people worldwide will suffer TBI each year. [(2)] Each year, 80, 000 - 90, 000 people experience the onset of long - term or lifelong disabilities associated with TBI. The mortality rate for TBI is 30 per 100, 000 making up to 34 percent of all traumatic deaths. [(1)]

Keeping in view the grave outcome of TBI we need to formulate a radical management approach to cater its deteriorating sequelae. Treating raised intracranial pressure and promoting adequate cerebral perfusion are considered the cornerstones of current therapy dealing with primary injury impacts. Hence, the secondary neurological sequelae that leads to continued brain injury after TBI is not addressed. [(3)] Depending on severity of injury, a complex disruptive cascade of processes are activated which continuously lead to endogenous changes affecting cellular systems. Secondary brain injury is reflective of the prognosis of TBI, so efforts are being done to halt these complex cascades therapeutically after identifying the secondary injury culprit factors. [(3, 4)]

Magnesium is considered to be a crucial diagnostic marker of neurotrauma with important implications in cytotoxic and reperfusion pathways of secondary brain damage. [(4)] Numerous studies show that there is a profound decline in total tissue and intracellular magnesium levels of brain or spinal cord after trauma [(5)] and magnesium supplementation have influenced the degree of post traumatic cellular damage. [(4, 6)] A comprehensive analysis of neurometabolic cascade after brain injury also revealed that intracellular

magnesium levels reduce immediately after TBI and remain low for up to 4 days. Hypomagnesemia may lead to neuronal dysfunction as both glycolytic and oxidative generation of ATP is impaired when magnesium levels are below normal. [7] In addition, low magnesium effectively unblocks the NMDA receptor channel more easily, leading to greater Ca2+ influx which has been shown to disrupt neurofilaments and microtubules impairing post - traumatic neural connectivity. [6] More calcium than magnesium in brain neurons is never a good thing—excess calcium causes ceaseless neuron stimulation, leading to cell death. [8]

Measuring magnesium ion levels in the blood after TBI can be of both diagnostic and prognostic value in treating brain injury. [8] To add on to our treatment armamentarium, we decided to evaluate the role of magnesium. In order to observe its valuable effects on prognosis we needed to highlight the fact that levels of magnesium are affected after traumatic brain injury. So we measured and recorded the serum magnesium levels and extrapolated its trend as TBI patients. We believe that the results will provide us with tangible evidence for infusing magnesium salts and give a way forward in researching its effects on improved outcomes as proposed in previous literature.

2. Objectives

To analyse the level of serum magnesium after traumatic brain injury.

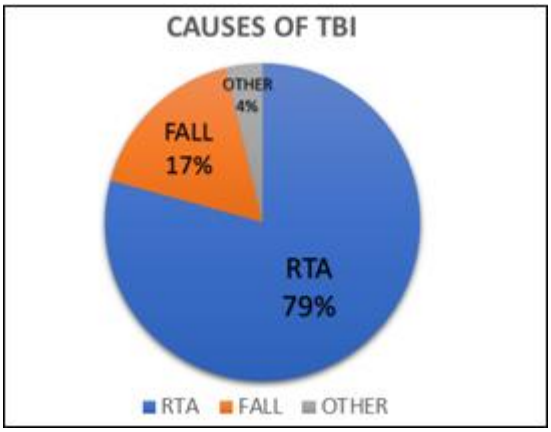
3. Materials and Methods

The study was conducted in the Neurosurgery Ward of Mayo Hospital Lahore, Pakistan. A total of 51 participants were included in the study. All the patients who presented with traumatic brain injury and were admitted for management in our Neurosurgery Ward in the last six months of 2020 took part in the study. The included participants were admitted within 12 hours of injury. The patients who had multi - organ involvement, prolonged shock on presentation and bilateral absent pupillary reflex were not included in the study.

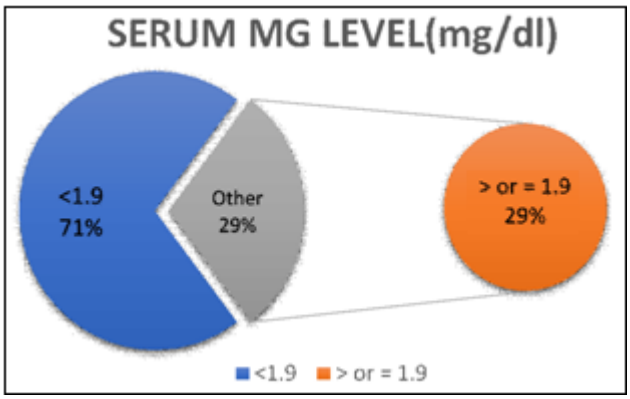
The blood samples including the serum magnesium levels of the patients were taken and recorded. A control group of similar age group with no disease was also included and their samples of serum Mg were taken for comparison with the test group. The serum magnesium measurement was carried out in a fully automated computerized micro - analyzer. The post - admission trend of serum magnesium levels of all the participants was extrapolated and evaluated. The Reference range of Normal serum magnesium level is 1.6 to 2.6mEq/L (1.9mg/dl to 3.1mg/dl) according to ABIM (American Board of Internal Medicine) Laboratory Test Reference Ranges January 2021.

4. Results

A total of 51 participants with a mean age of 32 ± 15SD were included in the study. Among them, 41 were males and 10 were females. The most common cause of traumatic brain injury was found to be road traffic accident (RTA).40 out of 51 patients had a history of RTA making it up to 78%. Second most common cause was the presentation after a fall i. e.9 out of 51; mostly involving elderly people.

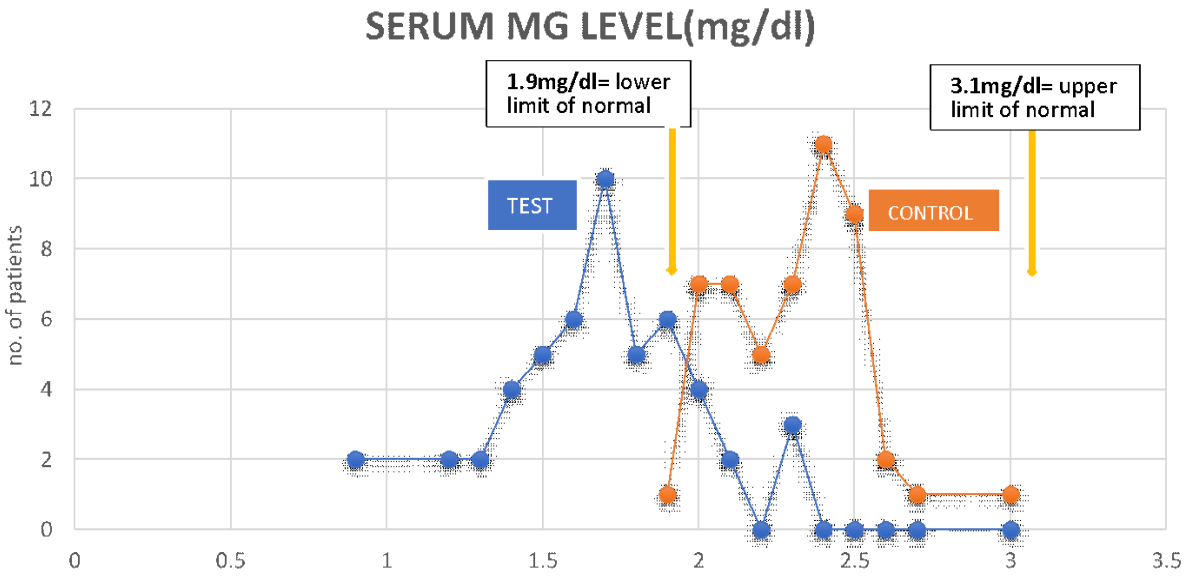


The serum magnesium level in all the 51 participants was collected and graphed.71% patients had a drop in magnesium level from standard minimum reference value i. e.1.6 mEq/L or 1.9mg/dl.12% of the individuals had their serum level in the lower limit of normal range.



The mean value of serum magnesium level of the test group (TBI patients) was 1.68 ± 0.3sd mg/dl. The control group (no disease) with the same number of participants and similar age group had their mean serum magnesium level of 2.3 ± 0.2 mg/dl.

The p value of <0.05 was considered significant. The difference of means of the test and control groups results were considered statistically significant as p - value was calculated to be 0.00001.



5. Discussion

When we start to untangle the relationship of magnesium and brain health, we touch upon every single biological mechanism which requires magnesium for its vitality. Magnesium is the fourth most abundant cation and is vital for more than 300 enzymatic reactions in the body. [9] Its importance depict how crucial it is to have its normal levels. Measuring magnesium levels will help us identify and maintain this biochemical balance. Previous studies have emphasized its role of being a critical biochemical marker for traumatic brain injury. [10]

This study clearly reveals that magnesium levels drop below normal after any traumatic brain injury. Almost 71% of the patients had their serum magnesium level decreased. Some of them had their Mg level in the lower limit of normal range which made upto 12%. Comparing the mean of serum magnesium levels of the control group the results were significant. The evident fall in magnesium showed that in most of the patients this electrolyte was reduced after trauma supporting the previous literature. Fall in this most important cation of the body is a red flag which demands radical approach to prevent detrimental effects of hypomagnesemia promoting secondary brain injury.

The research also propped up the most common cause of TBI being the road traffic accidents. The statistics support the fact that technological revolution has led to an increase in TBI patients. [1, 2] Traffic safety measures should be promulgated with strict surveillance to avoid this fatal aftermath. This also explains more percentage of male being affected by TBI as they are mostly involved in RTAs. Second most common cause found was history of fall which was mostly seen in elderly patients. Many causes have been proposed for fall in old age patients including comorbidities, increasing age, medication use and cognitive impairment. Special attention should be given to geriatic health. [13]

The main mechanism postulated for beneficial effects of magnesium ions is it being the gatekeeper between NMDA neuroreceptors and the cell membranes. Without adequate magnesium, there are essentially no 'soldiers at the gate' to block the free flow of ions, and this can lead to cell death. As explained in the introduction, the more the calcium influx, the more the disruption of microcellular environment leading to irreversible fatal outcome. [6, 7, 8]

It has been established back in 1987, that traumatic brain injury in rats lead to a 70% decline in intracellular free magnesium concentration. [5] They also proposed that this profound decline following CNS trauma may be a critical early factor in the development of irreversible brain injury. [11] Moving forward, nuclear magnetic resonance (NMR) studies of CNS trauma have shown that intracellular free magnesium concentration declines following injury and is associated with a decrease in brain total tissue Mg concentration. Thus putting light to the fact that Mg plays a central role in determining the degree of neurologic deficit expressed following a traumatic brain injury. [12]

The gravity of this fact is grave that magnesium levels are significantly effected after TBI and yet its not being acknowledged. If we are unaware of its trends, how can a directed approach be taken for its correction? Hence ignorance from this knowledge causes poor outcomes in TBI patients causing the burden of guilt on our shoulders. This study spotlights the momentous electrolyte of our body i. e. magnesium by bringing up its decreasing trends after TBI and hence emphasizes the need of its correction due to the documented critical role of magnesium in cellular mechanisms.

6. Conclusion

Magnesium levels are affected by the traumatic brain injury. The decreasing trend is evident in this study and supported by most of the past research. The previous data also delineate the implications of hypomagnesemia in prognosis of TBI. Hence, by shedding light to the explicit hypomagnesemia observed in TBI patients, this study probes further trials for addition of magnesium supplementation in the management of TBI to add evidence for its mandatory inclusion in standard therapy.

References

[1] Agarwal, N., Thakkar, R. and Than, K., 2020. *Traumatic Brain Injury – Causes, Symptoms and Treatments*. [online] Aans. org. Available at: <https: //www.aans. org/Patients/Neurosurgical - Conditions - and - Treatments/Traumatic - Brain - Injury> [Accessed 24 February 2021].

[2] Dewan, M. C., Rattani, A., Gupta, S., Baticulon, R. E., Hung, Y. C., Punchak, M., Agrawal, A., Adeleye, A. O., Shrime, M. G., Rubiano, A. M. and Rosenfeld, J. V., 2018. Estimating the global incidence of traumatic brain injury. *Journal of neurosurgery*, *130* (4), pp.1080 - 1097.

[3] McConeghy, K. W., Hatton, J., Hughes, L. and Cook, A. M., 2012. A review of neuroprotection pharmacology and therapies in patients with acute traumatic brain injury. *CNS drugs*, *26* (7), pp.613 - 636.

[4] Dhandapani, S. S., Gupta, A., Vivekanandhan, S., Mahapatra, A. K. and Mehta, V. S., 2005. Serum ionic magnesium in traumatic brain injury. *Indian J Neurotrauma*, *2* (02), pp.103 - 106.

[5] Vink, R., McIntosh, T. K., Demediuk, P. and Faden, A. I., 1987. Decrease in total and free magnesium concentration following traumatic brain injury in rats. *Biochemical and biophysical research communications*, *149* (2), pp.594 - 599.

[6] Saatman, K. E., Bareyre, F. M., Grady, M. S. and McIntosh, T. K., 2001. Acute cytoskeletal alterations and cell death induced by experimental brain injury are attenuated by magnesium treatment and exacerbated by magnesium deficiency. *Journal of Neuropathology & Experimental Neurology*, *60* (2), pp.183 - 194.

[7] Giza, C. C. and Hovda, D. A., 2001. The neurometabolic cascade of concussion. *Journal of athletic training*, *36* (3), p.228.

[8] Dean, C., 2018. Head Injury & Magnesium [WWW Document]. Dr. Carolyn Dean MD ND. URL https: //drcarolyndean. com/2018/11/head - injury - magnesium/ (accessed 2.24.21).

[9] Fox, C., Ramsoomair, D. and Carter, C., 2001. Magnesium: its proven and potential clinical significance. *Southern medical journal*, *94* (12), pp.1195 - 1202.

[10] Mendez, D. R., Corbett, R., Macias, C. and Laptook, A., 2005. Total and ionized plasma magnesium concentrations in children after traumatic brain injury. *Pediatric research*, *57* (3), pp.347 - 352.

[11] Vink, R., McIntosh, T. K., Demediuk, P., Weiner, M. W. and Faden, A. I., 1988. Decline in intracellular free Mg2+ is associated with irreversible tissue injury after brain trauma. *Journal of Biological Chemistry*, *263* (2), pp.757 - 761.

[12] Vink, R., 1991. Magnesium and brain trauma. *Magnesium and trace elements*, *10* (1), pp.1 - 10.

[13] Fuller, G. F., 2000. Falls in the elderly. *American family physician*, *61* (7), p.2159.