International Journal of Science and Research (IJSR)
ISSN (Online): 2319-7064
Index Copernicus Value (2013): 6.14 | Impact Factor (2013): 4.438

Impact of Serum Copper Level in Patients of Traumatic Brain Injury and its Correlation with Glasgow Coma Scale

Ankita Johary[1], Vinod Jain[2], Samir Misra[3], Vibhuti Rai[4], J. V. Singh[5]

Department of Surgery (Gen.), King George's Medical University, Lucknow

Abstract: ***Background: Trauma is the injury caused by the physical force. More often, trauma is the consequence of motor vehicle crashes, falls and assaults. Head injury is recognized as a major public health problem that is a frequent cause of death and disability in young people. Trace metals like copper is known to have its impact in traumatic brain injury patients. This study was conducted to study the impact of serum copper levels in traumatic brain injury patients and its correlation with Glasgow coma scale. Materials and Methods: It is a prospective cohort study. Blood samples were collected within eight hours of injury and subjected to biochemical assessment of serum copper. The patients were subsequently followed up and repeat observations were carried out on day 5 and day 10. Serum was separated with the help of centrifuge and quantitative analysis of copper was done by atomic absorption spectrophotometer after its complete digestion by microwave digester. Results: The results are presented in mean±sd. The levels of between cases and controls were compared by using unpaired t-test. Similarly, Cu levels between moderate and severe GCS at day 0, day 5 and day 10 were compared by unpaired t-test. The changes among follow-ups were compared by using paired t-test. The p-value<0.05 was considered as significant. All the analysis was carried out by using SPSS 16.0 version. Conclusions: The study showed a primary evidence of a relationship between and serum copper levels in traumatic brain injury patients and controls.***

Keywords: Trauma; Traumatic Brain Injury (TBI); Trace elements; Copper; Public health

1. Introduction

Trauma refers to both the experience of being harmed by an external agent as well as the response to that experience [1]. Traumatic brain injury (TBI) constitutes a major health and socioeconomic problem throughout the world. WHO has projected that, by 2020, traffic accidents will be the third greatest cause of the global burden of disease and injury. Trace elements are directly involved in maintaining and regulating many of physiological processes, especially those involved in normal carbohydrate, fat and protein metabolism and the ultimate formation of usable energy [2]. Critically ill burn and trauma patients are characterized by an increased free radical production, which is proportional to the severity of the injury. In addition, they are at high risk of negative trace element balances, which contribute to the imbalance in endogenous antioxidant capacity and the extension of primary lesions. Oxidative stress has been implicated as a potential contributor to the pathogenesis of acute central nervous system (CNS) injury. Radicals can cause damage to cardinal cellular components such as lipids, proteins, and nucleic acids, leading to subsequent cell death by modes of necrosis or apoptosis [3].Trace elements have been shown to have an association with oxidative stress [4].Thus levels of trace elements in serum of head injury patients can be an indication of the resultant oxidative stress and in turn the severity of head injury. In humans Cu is necessary for the development of connective tissue, nerve coverings, and bone. Cu also participates in both Fe and energy metabolism. Copper is present throughout the brain and is most prominent in the basal ganglia, hippocampus, cerebellum, numerous synaptic membranes, and in the cell bodies of cortical pyramidal and cerebellar granular neurons [5]. Enzymes in the central nervous system that depend on copper for their function include tyrosinase, peptidylglycine α-amidating mono-oxygenase, copper/zinc superoxide dismutase, ceruloplasmin, hephaestin, dopamine-β-hydoxylase, and cytochrome *c* oxidase [6-8]. Thus this study was conducted to study the impact of serum copper levels in traumatic brain injury patients and its correlation with Glasgow coma scale.

2. Material and Methods

It is a prospective cohort study carried out in tertiary care hospital (King George's Medical University) Lucknow. Blood samples were collected by taking informed consent within eight hours of injury from each patient by sterile disposable syringes from cubital veins and put into clean and labeled glass vials and subjected to biochemical assessment of copper. The patients were subsequently followed up and repeat observations were carried out at day 5 and day 10. Ethical approval of this study was taken from university ethics committee.

Serum was separated by centrifugation at 3000 rpm for 15 minutes. For digestion, 2ml of sample were mixed with 5ml of conc. nitric acid in pressure vessel and digested with microwave digester. Quantitative estimation of copper was done on Atomic Absorption Spectrophotometer (AAS), make Perkins Elmer, model Analyst 600 with special slit setting shown in table 1.

Statistical analysis: The results are presented in mean±sd. The level of between cases and controls was compared by using unpaired t-test. Similarly, Cu levels between moderate and severe GCS on day 0 and the follow-ups were compared by unpaired t-test. The changes among follow-ups were compared by using paired t-test. The p-value<0.05 was considered as significant. All the analysis was carried out by using SPSS 16.0 version.

3. Results

This study was carried out on total 191subjects of age 20-50 years (mean age=28) out of which 89 were cases and 102 were controls respectively. The level of Cu was significantly ($p<.001$) higher among controls (1.92±0.29) than cases (0.94±0.17) at day 0 (Table-2).When Cu levels were compared between the moderate and severe traumatic brain injury groups, the levels of copper showed insignificant difference on day 0 and day 5 in both the groups, However on day 10 the serum cu levels in moderate head injury group showed higher values than severe traumatic brain injury group and were nearly significant ($P = 0.05$) (Table-3)

4. Discussion

Head injury caused by trauma is one of the most important causes of hospital admissions and mortality. It affects an individual both physically as well as psychologically by impairing the functional ability as well as cognitive ability of an individual. The incidence of head injury is 300 of every 100,000 per year with a mortality rate of 25 per 100,000 in North America and 9 per 100,000 in Britain [9]. In India too, owing to high prevalence of road traffic accidents, head injury caused by trauma are one of the main reasons for prolonged hospital stay, mortality and morbidity [10]. In this study, the level of copper was significantly lower among the cases as compared to controls. The level of copper was significantly increased from day 0 to day 10 among moderate GCS patients in the present study. It is suggested that the correlation between the patient's outcome and serum Cu is probably due to the vital role of Cu in eliminating the oxidative stress (through metallothionin and SOD) that is the most detrimental factor in severe head injury. The patients, who have persistent low GCS score, also have persistent low serum copper levels. Therefore Serum Copper levels can be used as one of the reliable methods to prognosticate the traumatic brain injury.

5. Conclusion

The study showed a primary evidence of a relationship between and serum copper levels in head injury patients with controls. A larger sample size and longer follow up may give concrete evidence that serum copper levels can prognosticate traumatic brain injury.

References

[1] Becker D et al. Trauma and adolescence I: The nature and scope of trauma. Adolescent Psychiatry, 2003.
[2] Saraymen R, Kılıç E, Yazar S. Sweat Copper, Zinc, Iron, Magnesium and Chromium Levels in National Wrestler. İnönü Üniversitesi Tıp Fakültesi Dergisi 2004; 11(1): 7-10.
[3] Gilgun-Sherki Y, Rosenbaum Z, Melamed E, Offen D. Antioxidant therapy in acute central nervous system injury: current state. Pharmacol Rev. 2002; 54(2):271-284.
[4] Huang Y-L, Sheu J-Y, Lin T-H. Association between oxidativestress and changes of trace elements in patients with breast cancer. Clinical Biochemistry 1999; 32(2): 131-136.
[5] Madsen E, Gitlin JD. Copper and iron disorders of the brain. Annu Rev Neurosci 2007; 30:317–37.
[6] Zucconi GG, Cipriani S, Scattoni R, Balgkouranidou I, Hawkins DP, Ragnarsdottir KV. Copper deficiency elicits glial and neuronal response typical of neurodegenerative disorders. Neuropathol Appl Neurobiol 2007; 33:212–25.Medline
[7] Rinaldi AC. Meeting report: copper research at the top. Biometals 2000; 13: 9–13.
[8] Kaler SG. Metabolic and molecular bases of Menkes disease and occipital horn syndrome. Pediatr Dev Pathol 1998; 1: 85–98.
[9] Mass AIR, Stocchetti N, Bullock R. Moderate and severe traumatic brain injury in adults. Lancet Neurol 2008; 7: 728–41.
[10] Gururaj G. Epidemiology of traumatic brain injury: Indian scenario. Neurol Res 2002; 24: 24-28.