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Original Research Paper

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ROLE OF SERUM ALBUMIN AS A PROGNOSTIC FACTOR IN TRAUMATIC BRAIN INJURY

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ABSTRACT

Background: Traumatic brain injury still remains a serious concern and one of the leading causes of death and disability, particularly among young adults. Early determination of prognosis after traumatic brain injury is a priority for relatives and physicians involved in the care of these patients. New prognostic information beyond the clinical examination, patient demographics and radiological imaging from admission is needed to allow early prediction of short, mid, and long term outcome of patients with moderate and severe traumatic brain injury.

We conducted our study to elucidate the role of serum Albumin as a prognostic marker in traumatic brain injury. 120 patients with moderate to severe head injury (GCS ≤ 13) admitted in the Department of Neurosurgery, PGIMER and Dr. RML Hospital, New Delhi were studied from November 2015 to March 2017.

Objectives: To elucidate the role of serum Albumin as a prognostic marker in traumatic brain injury

Materials & Methods: 120 patients with moderate to severe head injury (GCS ≤ 13) admitted between November 2015 and March 2017 were studied. Clinical parameters, radiological parameteers, details of surgical/ conservative management, duration of total hospital stay and duration of stay in the ICU were recorded. This was correlated with serum Albumin levels on admission, day 3, day 5 and day 10 following admission.

Results: S. Albumin levels on Day 1, 3, 5 and 10 were found to be significantly lower ($p<0.001$) in Severe TBI as Compared to Moderate TBI. Percentage fall in S. Albumin levels on Day 5 ($p<0.011$) and Day 10 ($p<.0005$) were found to be significantly higher in Severe TBI as Compared to Moderate TBI. Mean S. Albumin levels were found to be significantly lower among patients with an unfavourable Glasgow Outcome Score, longer ICU stay and non survival ($p < 0.005$).

Conclusion: Serum albumin levels can be considered to be a reliable prognostic indicator in patients with traumatic brain injury with no other systemic co morbidities.

KEYWORDS :

INTRODUCTION

Severe Traumatic Brain Injury (TBI) is a major cause of disability and death in the young.[1]Severe TBI initiates a complex cascade of metabolic perturbations with the primary injury triggering secondary events that evolve over many days, leading to deleterious pathophysiological and biochemical reactions.[2] Numerous studies have consistently demonstrated increased protein catabolism following TBI.[3-5]Surprisingly, only a few biochemical markers have been identified to prognosticate the patient's response to brain injury. These markers also exhibit changes over time in patients of trauma as a component of metabolic response to injury or infection (MRII), independent of the nutritional status.[6] Even though this has also been noted in patients with severe TBI,[7] detailed analysis of associated factors and their overall significance with respect to neurological outcome are is to be established.

In humans, albumin is the most abundant plasma protein, accounting for 55–60% of the measured serum protein.[8] It consists of a single polypeptide chain of 585 amino acids with a molecular weight of 66500 Da. Critical illness alters the distribution of albumin between the intravascular and extravascular compartments. There are also changes in the rates of synthesis and degradation of the protein. The serum albumin concentration will decrease, often dramatically, from early in the course of a critical illness. Endothelial cells seem to be able to control the permeability properties of the capillary membrane, possibly by altering the nature and distribution of glycoproteins in the vessel wall.It involves dysfunction of the endothelial barrier, resulting in capillary leakage and loss of protein, inflammatory cells and large volumes of fluid into the interstitial space. The altered distribution in critical illness is related to an increase in capillary leakage.[10] This occurs in sepsis and after major surgical stress.[11,12] The precise mediators of this capillary leakage are still being discovered.

Serum albumin appears to be a reliable prognostic indicator in various contexts. A recent review suggests that serum albumin could be an independent predictor of mortality in a wide range of clinical and research settings.[13]

MATERIALS AND METHODS

We conducted a prospective observational study in the Department of Neurosurgery, PGIMER & Dr. RML Hospital, New Delhi. We included all patients admitted with moderate to severe traumatic brain injury (GCS =or <13) between November 2015 to March 2017.Last patient will be taken on Dec 2016. A total 120 patients were included in the study. The following parameters were studied –

1. Clinical parameters- History, clinical examination including GCS, Pupillary reaction.
2. Radiological Parameters- CT scan head with details of intracranial injury and MRI Brain, as required.
3. Estimation of Serum albumin on admission (within 24 hrs) and 3rd, 5th and 10th day of hospital stay.
4. Management Parameters- Conservatives or surgical and if any requirement of ICU with or without ventilator support.
5. Complications- Surgical site infection, wound dehiscence, CSF leakage, UTI, RTI etc.
6. Outcomes parameters- Glasgow coma scale in follow up.

The following patients were excluded from the ambit of the study-

1. Patients less than 18 years and more than 65 years
2. Polytrauma.
3. Hepatic disease, chronic renal failure and patient with cardio respiratory diseases.
4. Patients with hypotension at admission (admission systolic BP ≤ 90 mm Hg), renal dysfunction (blood urea > 50 mg/dL) and hyperbilirubinemia (total bilirubin > 1 mg/dL) will excluded from the study.
5. Patients with history of neoplastic and immunological disorder.

Data regarding clinical and radiological status of patients was collected. Severity of traumatic brain injury was defined according to Glasgow Coma Scale (GCS) score of patients at admission (mild-14 15, moderate 9-13, and severe -8 or less). All the patients were followed up in Neurosurgery OPD after discharge at 15 days, 1 month , 2months and 3months . We assessed the outcome of our patients on the basis of Glasgow outcome scale (GOS). In GOS scale, which includes five categories, patients are classified as grade 1 (death), 2 (vegetative state), 3 (severe disability), 4 (moderate disability), and 5 (good outcome). The GOS was dichotomized as unfavorable group (grades 1, 2 and 3) or favorable group (4 and 5). Further we divided the patients in two categories as survivor (GOS grade 2,3,4,5) and nonsurvivor (grade 1). Serum albumin levels on admission and on different days were assessed in Traumatic Brain Injury patients. On the basis of admission serum albumin levels, patients with hypoalbumenimia were grouped into mild (2.8 - 3.5 g/dL), moderate (2.1- 2.7 g/dL) and severe (< 2.1 g/dL).Comparison was also done among various subgroups defined by age, sex, severity of TBI according to GCS and others.

STATISTICAL ANALYSIS

SPSS software (version 10, SPSS Inc, Chicago) was used for the statistical analyses. Continuous variables in two groups were compared by using independent-samples t-test. Continuous variables in more than two groups were compared by using One-Way Analysis of Variance (ANOVA). Proportions were compared by using Chisquare tests or Fisher's exact test, wherever appropriate. Multivariate analysis were conducted with logistic regression adjusting for age, admission GCS, systemic injury, surgical intervention, and hypoalbuminemia at admission (serum albumin ≤ 3.5 g/dL). Two sided significance tests were used throughout, and the significance level was kept at $p < 0.05$.

RESULTS

All demographic variables like age, height, weight, BMI and ASA status showed a statistically similar distribution among all patients. We noted the following results at the end of our study (Table 1.1)

	Severe TBI	Moderate TBI	P value
S. Albumin (Day 1)			
Mean ± SD	3.43 ± 0.24	3.66 ± 0.25	<0.001
S. Albumin (Day 3)			
Mean ± SD	3.27 ± 0.25	3.49 ± 0.26	<0.001
S. Albumin (Day 5)			
Mean ± SD	3.12 ± 0.26	3.38 ± 0.26	<0.001
S. Albumin (Day 10)			
Mean ± SD	2.9 ± 0.3	3.23 ± 0.34	<0.001
Percentage Fall in S. Albumin (by Day 3)			
Mean ± SD	4.81 ± 3.53	4.51 ± 3.41	<0.240
Percentage Fall in S. Albumin (by Day 5)			
Mean ± SD	8.85 ± 6.33	7.59 ± 3.9	<0.011
Percentage Fall in S. Albumin (by Day 10)			
Mean ± SD	15.65 ± 6.1	11.64 ± 6.29	<0.0005

Table 1.1 Tabulation of the summary of observations among severe and moderate TBI

Mean S. Albumin levels were found to be significantly lower among patients with an unfavourable Glasgow Outcome Score, longer ICU stay and non survival ($p < 0.005$).

DISCUSSION

Out of the total of 120 partients included in our study, 68.33% patients underwent surgical management, in comparison to 31.67% patients who were managed conservatively. The mean serum albumin levels among surgically managed patients was 3.22 (SD ±0.26) g/dL, which was significantly lower (p value < 0.001) than that among patients managed conservatively (3.45±0.26 g/dL). In fact, of the surgically managed patients, 70.73% had hypoalbuminemia which was much higher compared to conservatively managed patients (p value = 0.006). Major surgery is followed by an important metabolic stress response, which is closely related to adverse outcomes. A number of perioperative interventions allow modulating an excessive stress response, some of them having an important positive impact on clinical outcome. Therefore, reliable prediction of surgical stress response is of high interest. The *ideal marker* has to be *easy* to measure, available *early* in the perioperative course, and *inexpensive*. It should be strongly *correlated with the extent of surgical trauma* and be a *reliable predictor* of complications and prolonged hospital stay. The strong correlation between hypoalbuminemia and increased incidence of surgical management in patients of TBI suggests that Serum Albumin levels can be used as an effective prognostoic indicator in these patients.

Serum albumin can thus be used as an independent predictor of patient outcome and prognosis in cases of traumatic brain injury. We would like to however, delineate the fact that we have excluded pediatric and geriatric patients, polytrauma patients, patients with hepatic, renal or cardiovascular diseases, shock or immunological and neoplastic diseases. All these patients are bound to have an already reduced serum albumin level owing to the primary pathology, which may also in turn affect the recovery and outcome of our TBI patients. We hence, excluded these patients, so that the relationship of TBI and serum albumin could be elucidated independent of other confounding factors. Hemodilution is a potential confounder that needs to be taken into the equation of postoperative albumin decrease while dealing with patients with shock who have undergone or are undergoing massive fluid resuscitation. In the present study, patients in shock and those undergoing massive fluid resuscitation were excluded. We hence assume, that hemodilution played only a minor and subsidiary role in postoperative albumin decrease. Our findings in this study, can thus be extrapolated among patients with TBI who were otherwise healthy. The prognostic value of serum albumin among patients with other coexisting systemic diseases needs to be investigated further.

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