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A study on Neuron Specific Enolase (NSE) in cerebrovascular stroke patients.

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ABSTRACT

Background: Biochemical markers of acute neuronal injury may aid in the diagnosis and management of cerebrovascular stroke. Neuron specific enolase (NSE) is one such marker which is released in the blood in acute neuronal injury and can be estimated in the serum of patients to assess the short-term neurological outcome. This study was carried out on patients of acute cerebrovascular stroke with the aim to compare NSE levels in normal subjects with that in cerebrovascular stroke patients. **Methods:** 60 subjects investigated in the study included 30 cerebrovascular stroke patients who were admitted within 72 hours of onset of stroke symptoms, in the Emergency Department and the Department of Neurology at HIMS, and 30 healthy controls. Serum NSE levels of cases and controls were determined on day 1 and day 7 using DRG-NSE ELISA kit. Statistical analysis was performed using the unpaired 't' test on SPSS software for windows version 17.0 **Results:** There was a significant difference in the levels of serum NSE between cases and controls ($p<0.001$).The mean levels of serum NSE in controls were 2.25±2.12 ng/ml and in cases at the time of admission were 89.18±46.89 ng/ml . The normal range of serum NSE is 0-12 ng/ml. It was also observed that the levels of serum NSE showed no difference in males or females or among different age groups. **Conclusion:** This study showed that estimation of serum NSE levels can be used as an early marker of neuronal damage in acute cerebrovascular stroke patients.

Keywords: Acute neuronal injury, Cerebrovascular stroke, Neuron specific enolase.

INTRODUCTION

Cerebrovascular diseases include some of the most common and devastating disorders such as ischemic stroke, hemorrhagic stroke, and cerebrovascular anomalies. A stroke, or cerebrovascular accident, can be defined as the abrupt onset of a neurologic deficit that is attributable to a focal vascular cause. Certain biochemical markers such as Neuron Specific Enolase (NSE), can

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Indicate degree of brain damage. In humans, NSE is considered as a specific neurobiochemical marker of brain damage after brain infarction (1). It accounts to 1.5% of cell-soluble brain proteins (2). The course of plasma NSE levels is seen as a relevant parameter for assessing the prognosis of cerebral hypoxia/ischemia. Physiologically, NSE is present only in negligible amounts in the peripheral blood. Serum concentration is 8.7±3.9 ng/ml (men 8.9±3.9, women 8.3 ±4.0) (3). The biological half-life of NSE in body fluids is approximately 24 hours.

MATERIALS AND METHODS

The study was conducted in the Department of Biochemistry, Himalayan Institute of Medical Sciences (HIMS), Swami Ram Nagar, Dehradun, over a period of 12 months, and after obtaining a clearance certificate from the Ethical Committee. Subjects were recruited from patients admitted in ICU/Neurology wards, HIMS, Dehradun with a primary diagnosis of Cerebrovascular
Stroke (within 72 hours) and after obtaining written informed consent.
Study Design: Observational Analytical Study
Sample Size: 60 subjects (30 cases, 30 controls)
Inclusion Criteria: Stroke patients within 72 hours of admission
Exclusion criteria: Head injury, stroke >72 hrs.
Data Collection: Data was generated using structured study formats and subject proformas. NSE was estimated by Sandwich ELISA using DRG NSE kit. NIHSS and MRS were used for clinical assessment of patients.
Data Analysis: SPSS Software 20.0 Version and Microsoft Excel Software. Unpaired't' test and Pearson's correlation were applied.

RESULTS

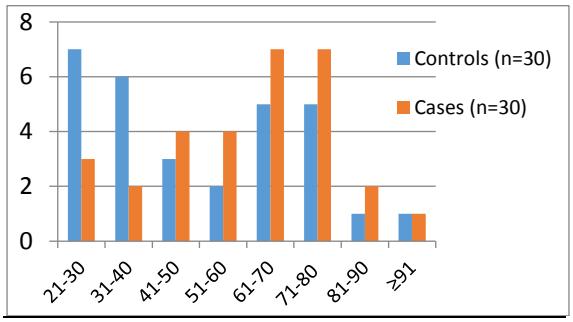

Figure 1.1: Age Group.

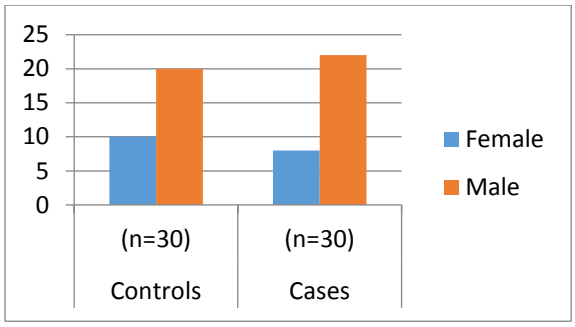

Figure 1.2: Distribution by Gender.

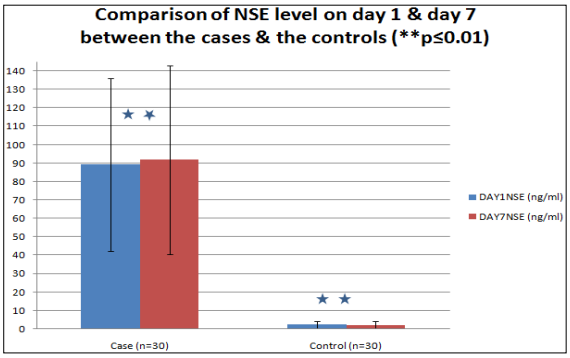

Figure 2. Distribution of Study Subjects (n=30) and Controls (n=30) by their NSE levels on Day 1 and Day 7.

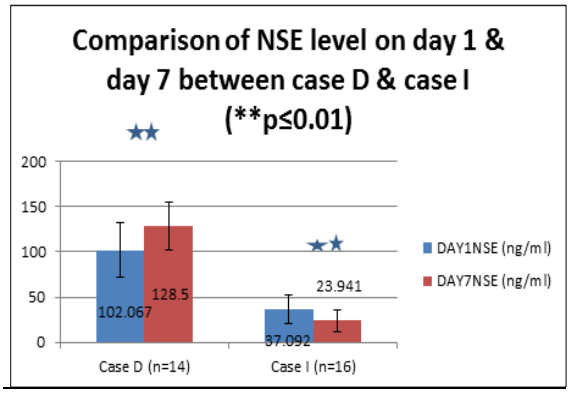

Figure 3: Comparison of the NSE levels between the Deteriorated cases (Case D) and the Improved cases (Case I) during the 7 days treatment at the hospital.

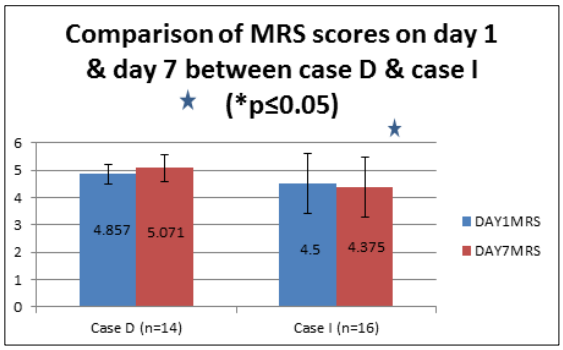

Figure 4: Comparison of the MRS Score between the Deteriorated cases (Case D) and the Improved cases (Case I) during the 7 days treatment at the hospital.

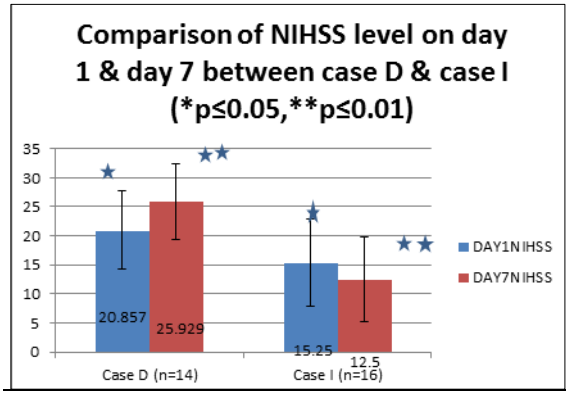

Figure 5: Comparison of the NIHSS Score between the Deteriorated cases (Case D) and the Improved cases (Case ND) during the 7 days treatment at the hospital.

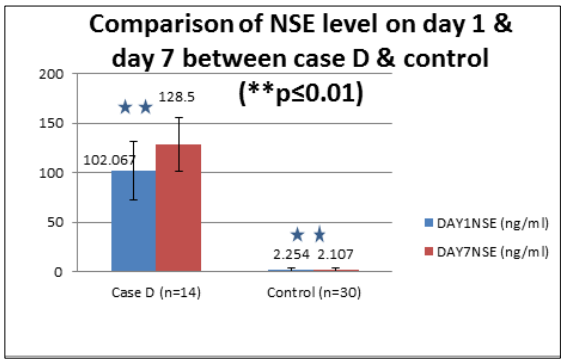

Figure 6: Comparison of the NSE levels between the Deteriorated cases (case D) and the controls.

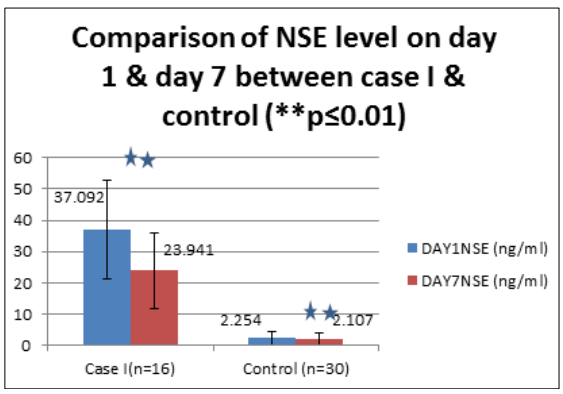

Figure 7: Comparison of the NSE levels between the improved cases (case I) and the controls.

DISCUSSION

Neuron-specific Enolase (NSE) is considered a noninvasive marker for neuronal injury which gets elevated in CSF and serum in different neurological diseases.[4] Therefore, use of neuronal biomarkers such as NSE is helpful because all ischemic brain injuries which are not detectable by CT/MRI, may be diagnosed by measuring these markers in serum/CSF.[5] In the current study, a significant positive correlation was observed between the elevated levels of serum NSE and poor neurological outcome in patients, which was clinically established by MRS ($p<0.01$) and NIHSS ($p<0.01$) scores. González-García et al had recently observed that the levels of NSE in serum samples of acute stroke patients were significantly higher than in controls.[6] In the control group, the mean serum NSE level in males was 2.46±2.27 ng/ml and in females was 1.84±1.81 ng/ml. Mean serum levels of NSE in cases were found to be 89.18±46.89 ng/ml on day 1 and 91.76±51.30 ng/ml on day 7. The observations of the present study were supported by a preliminary study conducted by Lukas et al[7] who examined and compared serum NSE level in 62 acute stroke patients. They found a significantly positive correlation of NSE levels with the extent of neurological damage in such patients ($r=+0.894$, $p<0.001$). On the basis of a rise or fall in the levels of NSE on day 7, compared to that of day 1, the cases were categorized into 2 groups- a Deteriorated cases group (n=14) and an Improved cases group (n=16). In the Deteriorated cases group, NSE levels increased on day 7 which was correlated clinically by observing deterioration in the NIHSS scores. The reverse was observed in the improved cases group. NIHSS is also found to have a strong positive correlation with the levels of serum NSE both on day 1($r=0.75$; $p \leq 0.01$) and day 7 ($r=0.79$; $p \leq 0.01$). In the present study serum NSE levels have been found to be related to neuronal injury in cerebrovascular stroke. A positive correlation was observed between serum NSE levels and neurological worsening in the study group.

CONCLUSION

A significant increase in levels of serum NSE among stroke patients was observed which also associated with greater degree of disability and neurological worsening. The association of prognosis with serum NSE levels was indicated by a strong positive correlation between NIHSS and MRS scores. NSE levels show no significant difference with differing age groups or with different gender.

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