Introduction

Brain injury resulting from traumatic, ischemic and/or hemorrhagic etiology is a significant international health concern, representing a potentially catastrophic debilitating medical emergency with poor prognosis for long-term disability. (1)

Neurological events like brain trauma, haemorrhage, and ischemic stroke result in primary damage of neurons and glia that in combination with secondary insults due to hypoxia, hypotension, seizures, sepsis or central fever can have an adverse effect on brain integrity and neurological outcome. (2)

Clinical assessment of brain function [neurological examination, Glasgow Coma Scale for level of consciousness (GCS), pupillary reactivity and neuro-imaging techniques [Computed Tomography (CT) and Magnetic Resonance Imaging (MRI) constitute the common ways for diagnosis and assessment of brain damage. (3)

Despite the substantial recent progress in cerebral neuro-monitoring it remains difficult to quantify the extent of primary brain injury as well as the ongoing secondary damage as it is well known that a prompt diagnosis of neurological deterioration or complication remains a major challenge in clinical practice. (4)

Therefore, and because of previous limitations; assessment of brain injury severity has been of considerable interest in the development and the use of biochemical measures of brain cell damage. [4]

It is well established that some serum biochemical markers offer valuable information regarding the diagnosis, severity and course of many diseases as troponin for myocardial infarction, PSA for prostate cancer, creatinine for renal failure, CEA for colon cancer and amylase for pancreatitis. But others are not specific such as CRP. [5]

Recent studies have assessed the serum level of a Varity of brain markers that have high specificity and sensitivity for brain injury with no age or sex variability as well as rapid appearance in serum. They also show a predictable relationship between the serum concentration and the tissue injury. [6]

Many serum biochemical markers have been proposed as potential brain injury indicators as: [7]

- Creatine Kinase (CK) [CK-BB],
- Lactate Dehydrogenase (LDH),
- Glial Fibrillary Acidic Protein (GFAP),
- Myelin Basic Protein ,
- S-100B protein ,
- Neuron Specific Enolase (NSE)

Neuron Specific Enolase (NSE) is a glycolytic enzymes family (enolases) that is located in the cytoplasm of neurons and is involved in chloride levels balance during the onset of neural activity. It serves as a

marker of neuronal damage in a patient with a Varity of neurological conditions including status epilepticus. Increased concentration of NSE can be measured in the cerebrospinal fluid (CSF) and in peripheral blood after neuronal damage. [8, 9]

Another biochemical serum marker currently under investigations is S-100B protein which is a member of the calcium biding protein hat is found mainly in the cytosol of glial cells. It is assumed that S-100B is released after glial cell damage and can be found in increased concentration in CSF. [10]