Introduction

Prognosis is the cornerstone of clinical medicine, because all diagnostic and therapeutic actions aim to improve patient's prognosis and outcome. Advances in statistical modeling and the availability of large databases have made it possible to consider diagnosis and prognosis in terms of probabilities rather than vague prophecies Probability estimates can be applied to clinical decision making, research, and assessment of the quality of health care. Such quantitative estimates are of particular relevance to heterogeneous conditions such as traumatic brain injury (TBI).

TBI poses a major public-health problem, with an estimated annual incidence of up to 500 per100 000 population and more than 200 hospital admissions per 100 000 admissions in Europe each year.[1,2]TBI is heterogeneous in terms of cause, pathology, severity, and prognosis, which poses diagnostic challenges. Furthermore, comparison of results between studies is difficult because case mix and treatments may vary substantially.

The majority of TBI is classified as mild, and around 8–10% is classified as moderate or severe.[3, 4] Patients with mild TBI have a good prognosis providing treatable complications are not missed. Overall mortality in this group is around 0.1% and is associated predominantly with missed intra-cranial hemorrhage.[5] Although many patients may return to work after mild TBI[6], around 50% of survivors have moderate or severe disability as assessed by the Glasgow Outcome Scale (GOS) or the disability outcome scale;[3,6,7]

this represents significant morbidity. For the minority of patients presenting with more severe TBI, the prognosis is much worse. Approximately 30% of patients admitted to hospital with Glasgow Coma Scale (GCS) score < 13 will ultimately die; mortality for those with GCS ≤ 8 after resuscitation may be as high as 50%.[8]And also long-term outcome among survivors of severe TBI is worse than in those with mild TBI.

For patients with moderate and severe TBI, prediction of clinical outcome is also highly relevant and typically, most studies have defined clinical outcome as mortality or functional outcome assessed with the Glasgow outcome scale (GOS)as their endpoint.[9]

Because the traumatic brain injury is a leading cause of death and disability worldwide (Every year, about 1.5 million affected people die and several millions receive emergency treatment [10, 11] and most burdens (90%) is in low and middle income countries) [12], clinicians treating patients often make therapeutic decisions based on their assessment of prognosis. According to a 2005survey, 80% of doctors believed that an accurate assessment of prognosis was important when they made decisions about the use of specific methods of treatment such as hyperventilation, barbiturates, or mannitol.[13]

A similar proportion considered that this was important in deciding whether or not to withdraw treatment. Assessment of prognosis was also deemed important for counseling patients and relatives. Only a third of doctors, however, thought that they accurately assessed prognosis.[13]

Much research has been done to identify early predictors of mortality and functional outcome, as assessed by the GOS on admission, after moderate or severe TBI. The GOS is usually dichotomized into good recovery and mild disability versus severe disability, vegetative state, and mortality. This is a limitation because we cannot assume that predictors differentiate death from survival as well as they can differentiate good recovery from worse outcomes. A large body of evidence supports the relation between some predictors and outcome, but for other predictors the relation is less well established. Information obtained during the subsequent clinical course may further contribute to outcome prediction.[9]

Traumatic brain injury has evolved in the last two decades; this may be a result of a more thorough understanding of the physiologic events leading to secondary neuronal injury after TBI, as well as advances in care of the critically ill patients.

The pathophysiology of the primary brain injury can be divided into focal and diffuse lesions. Focal injuries impact morbidity and mortality based on their location, size and overall progression. The most common types of primary head injuries are skull fractures, epidural hematoma, subdural hematoma, subarachnoid hemorrhage, intraventricular hemorrhage, brain contusions and diffuse axonal injury.[14]

Secondary brain injury occurs after initial trauma and is defined as the damage to neurons due to systemic physiologic responses to the initial injury [15]. A number of biochemical substances have been postulated to play a role in the propagation of neural injury following TBI. The release of these substances initiates a deleterious cascade of continued cell membrane break-down and ionic shift that further harm the injured brain.[16]

The myocardium of the head trauma patients is exposed to physiological and pharmacological stresses resulting in an imbalance between myocardial oxygen supply and demand. Oxygen demand is increased by tachycardia, increased contractility and increased left ventricular end diastolic pressure. Anemia, hypoxia and hypotension can further diminish the oxygen supply, and approximately 50% of TBI patients are reported to be in the hypoxic field.[17]

Cardiac troponin I (cTnI) is a regulatory protein that controls the calcium mediated interaction of actin and myosin.[18] It is used as a biochemical marker of myocardial injury and has proven to be more sensitive and specific than creatine kinase.[19]Besides its use in diagnosing acute coronary syndromes, cTnI elevation has been observed in a variety of non-coronary conditions such as pulmonary embolism, sepsis, and chronic renal failure.[18, 20, 21]Troponin elevation has also been observed in acute non traumatic head injury including subarachnoid (SAH) hemorrhage, stroke, and intracerebral hemorrhage [22, 23].

In fact up to 50% of patients with SAH have abnormal admission cTnI levels [24, 25]. Elevation of troponin after non traumatic cerebral insult has been associated with an increased risk of cardiopulmonary complications and mortality.[22, 24]

The cause of elevated troponin after these neurologic events is poorly understood, but may be because of elevated circulating levels of catecholamine after brain injury.[26, 27, 28]

Troponin elevation has been observed in as many as 30% of the critically injured patients [29]. Recent reports suggest that elevation of troponin in trauma may be more related to the degree of the physiologic stress, not to the mechanical chest trauma [29]. There is very little written about the association between the traumatic brain injury and the troponin elevation; since the catecholamine surge seen in the SAH is present after TBI [28, 30]elevated cTnI should be present as well.

So the purpose of this prospective study is to analyze the elevation of the cardiac troponin I in the traumatic brain injury patients and to evaluate the association between the elevated cardiac troponin I level and the poor outcome of the patients with TBI as classified according to the Glassco outcome score into five groups; good recovery, mild disability, major disability, vegetative state and death.

Hence the assessment of the predictive value of the serum troponin I in traumatic brain injury patients.