Traumatic Brain Injuries

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Introduction

Traumatic brain injuries belong to one of the most serious public health problems in the world. Traumatic brain injury (TBI), along with its short- and long-term physical, cognitive, and neurobehavioral consequences, contributes significantly to the mortality and morbidity rates of traumatized patients. TBI, caused by motor vehicle accidents, falls, assaults, or sports injuries affects approximately 7 million people each year. The knowledge of the pathophysiology after traumatic brain injury is necessary for adequate and patient-oriented treatment.

Definition and Classification of TBI

A.Maas, N. Stocchetti, and R. Bullock (2008) define traumatic brain injury as "damage to the brain resulting from external mechanical force, such as rapid acceleration or deceleration, impact, blast waves, or penetration by a projectile" (p. 729). The other term used for traumatic brain injury is intracranial injury. This phenomenon takes place when an external force traumatically injures the brain. TBI is a narrower category than head injury because the latter involves damage to the scalp and skull, not only the brain.

An important role in determining the patient's treatment, rehabilitation potential, and prognosis is played by the classification of TBI. There are several ways of TBI classification, which supplement each other. The most typical bases for TBI classification are severity, anatomical features of the injury, and the mechanism.

I. Moppett (2007) mentions that, classically, TBI is classified into primary and secondary injury. Primary injury is caused by mechanical force (contact and acceleration-deceleration) and takes place at the moment of injury. P. Pangilinan (2012) states that, "these forces can cause contusions, intracranial hematoma, diffuse vascular injury, and injury to cranial nerves and the

pituitary stalk" (p. 1). Contusions are distinct areas of swollen brain tissue that can be found on the poles of the frontal lobes, the inferior aspects of the frontal lobes, the cortex above and below the operculum of the sylvian fissures, and the lateral and inferior aspects of the temporal lobes. Intracranial hematomas, as the most common causes of death and clinical deterioration after TBI, are categorized into epidural hematomas, subdural hematomas, and subarachnoid hematomas.

Primary injuries on the basis of principal mechanisms of TBI are classified by C. Werner and K. Engelhard (2007) into "focal brain damage due to contact injury types resulting in contusion, laceration, and intracranial hemorrhage or diffuse brain damage due to acceleration/deceleration injury types resulting in diffuse axonal injury or brain swelling" (p.4).

Secondary injury is a kind of injury that is not of mechanical character and may superimpose injury on a brain already affected by a mechanical injury. It is usually prolonged from the moment of impact and may occur hours or days after the provoking traumatic event. Injury may result in impairment or local decreases in cerebral blood flow after a TBI. Local edema, hemorrhage, or increased intracranial pressure can cause a decline in cerebral blood flow. Such clinical conditions as arterial hypotension, hypoxemia, intracranial hemorrhage and malignant brain edema, and hyperthermia are associated with the risk of a decreased cerebral blood flow.

Severity related classification organizes TBIs into three categories: mild, moderate, and severe. As J. Burns and A. Hauser (2003) state, that the most commonly used system for classifying TBI severity is called The Glasgow Coma Scale (GCS). The GCS assesses a person's level of consciousness and neurologic functioning. A 3–15 point scale consists of three sections, constructed on verbal, motor, and eye-opening reactions to stimuli. The GCS defines the severity

of a TBI within 48 hours of injury. According to the GCS, 3 – 8 score indicates a severe TBI, 9 – 12 score indicates a moderate TBI, and 13 – 15 score designates a mild TBI.

P. Pangilinan (2012) mentions that the severity of a TBI is also measured by the duration of loss of consciousness. At a mild TBI mental status change lasts less than 30 minutes, a moderate TBI is marked by loss of consciousness for 30 minutes to 6 hours, and more than 6 hours of loss of consciousness is typical for a severe TBI.

Classification of TBIs by its pathological features presents two categories based on clinical and neuroradiologic evaluation: focal and diffusive brain injury. Focal injuries marked by extra-axial lesions include scalp injury, skull fracture, and surface contusions. They are generally caused by a contact. Diffuse injuries, occurring within the brain tissue, include DAI, hypoxic-ischemic damage, meningitis, and vascular injury. Acceleration-deceleration forces usually cause them. Focal and diffusive injuries are commonly found together.

Classification of TBI based on mechanism factors divides them into closed (the brain is not exposed) and penetrating head injury (an object pierces the skull and breaches the dura mater.

TBI’s Symptoms

Long-term physical, cognitive, and behavioral impairments are associated with TBI. These factors limit a patient's rehabilitation, his reintegration into the community, and return to work. Symptoms of TBI depend on its type (diffuse or focal), the affected part of the brain, and the injury's severity.

Fatigue, weakness, memory deficits, headache, vomiting, nausea, lack of motor coordination, dizziness, difficulty balancing, lightheadedness, blurred vision or tired eyes, ringing in the ears, bad taste in the mouth, fatigue or lethargy, and changes in sleep patterns are

typical complaints of the patient with a mild TBI. The patient, suffering from a mild TBI, may remain conscious or may lose consciousness for a few seconds or minutes. P. Pangilinan (2012) cites the study carried out by Kraus and colleagues of 235 patients. It demonstrates the most commonly reported symptoms after 6 months of mild TBI. They are fatigue (43%), weakness (43%), memory deficits (40%), headache (36%), and dizziness (34%). Behavioral or mood changes, confusion, and trouble with memory, concentration, attention, or thinking constitute cognitive and emotional symptoms. Symptoms that are typical for a mild TBI may also be present in moderate and severe injuries.

A long-lasting headache, repeated vomiting or nausea, convulsions, an inability to awaken, dilation of one or both pupils, slurred speech, aphasia, dysarthria, weakness or numbness in the limbs, loss of coordination, confusion, restlessness, or agitation are characteristics of a moderate or severe TBI. Cognitive and social deficits have long-term consequences for the daily lives of people with moderate and severe TBI. C. Werner and K. Engelhard (2007) notes, that severe TBIs are characterized by increased intracranial pressure with its manifestations of decreasing level of consciousness, paralysis or weakness on one side of the body, a blown pupil, Cushing's triad, anisocoria, and abnormal posturing.

The most common signs and symptoms related to TBI are insomnia, cognitive decline, posttraumatic headache, and posttraumatic depression. Medical complications associated with TBI include posttraumatic seizures (frequently occur after moderate or severe TBI), hydrocephalus (communicating or noncommunicating on the basis of the causative obstruction), deep vein thrombosis (with an incidence of 54% in persons with TBI), heterotopic ossification (possible during the first 3-4 months after injury), posttraumatic agitation, and others. Gastrointestinal and genitourinary disorders are the most common consequences in patients with

TBI. Stress ulcers, dysphagia, bowel incontinence, elevated levels on liver function tests, urethral strictures, urinary tract infections, and urinary incontinence are among them. Among medical problems associated with TBI, posttraumatic epilepsy, neuroendocrine, respiratory, and central autonomic dysfunction should be also mentioned.

TBI ‘s Early Management

Emergency treatment should be start as soon as possible, within the so-called "golden hour" following the injury. A treatment in an intensive care units followed by a neurosurgical ward should be proposed for people with moderate to severe injuries. Treatment depends on the stage of patient’s recovery. In the acute stage the chief perspective of the medical personnel is to stabilize the patient and focus on preventing further injury. Mass lesions such as contusions or hematomas causing a significant mass effect are considered emergencies and are removed surgically. Decompressive craniectomy is performed in the very short period following TBI during operations to treat hematomas.

I. Moppett (2007) suggests the general principles of TBI’s early management, agreed on by most clinicians, “maintenance of adequate and stable cerebral perfusion, adequate oxygenation, avoidance of hyper- and hypocapnia and avoidance of hyper- and hypoglycaemia, while avoiding iatrogenic injury” (pp. 23-25). The implementation of these principles in clinical practice differs from center to center, based largely on historical tradition, local practice, and a lack of clear evidence of benefit of any one therapeutic approach. To improve the outcomes of TBI the doctors may include in their treatment strategy such substances as calcium channel antagonists, magnesium, amino-steroids, dexanabinol, glucocorticoids, and glutamate antagonist. Rehabilitation is the main treatment strategy for the nonacute and chronic stages of recovery.

Causes of TBI

Violence, transportation accidents, construction, and sports are considered the most common causes of TBI in the world. Accidents associated with motor bikes are major causes of TBI in developing countries. Between 1.6 and 3.8 million traumatic brain injuries each year are a result of sports and recreation activities in the USA. Children in aged from two to four most frequently gain TBI because of falls, while older children get TBI because of traffic accidents. TBI is the third most common injury to result from child abuse, which causes 19% of cases of pediatric brain trauma. TBI is also caused by domestic violence and industrial accidents. TBI is the leading cause of death and disability in war zones. According to the Center for Disease Control and Prevention's National Center for Injury Prevention and Control cited by P. Pangilinan (2012), the leading causes of TBI are falls (28%), motor vehicle crashes (20%), being struck by or against objects (19%), and assaults (11%).

Epidemiology of TBI

According to E. Elovc, E. Baerga, and S. Cuccurullo (2004), TBI is the primary cause of neurologic mortality and morbidity in the United States. Moreover, the authors mention that, "trauma is the leading cause of death in ages 1 – 44 and more than half of these deaths are due to head trauma" (p. 47). TBIs that require hospitalization in the USA constitute the figure of 500, 000 per year. According to the Center for Disease Control and Prevention's National Center for Injury Prevention and Control cited by P. Pangilinan (2012), annual statistics in the United States demonstrates the following figures: at least 1.4 million people sustain a TBI; 50,000 people die from a TBI; 475,000 TBIs occur among infants, children, and adolescents aged 0-14 years; about 80,000-90,000 people experience the onset of a long-term disability due to a TBI.

As for social characteristics included in the notion of TBI, we should point out that incidence is higher in males than in females (148 – 270 per 100, 000 vs. 70 – 116 per 100, 000).

So male to female ratio is 2,5 : 1. Peak ages of TBI are 15 – 18 to 25 years. Age distribution is bimodal, with second peak in the elderly (ages 65 - 75). The latter group is characterized by a higher mortality rate. Mortality in males is 3 – 4 times higher than in females. Recent studies have confirmed that the peak ages for TBI remain infants and children aged 0-4 between, adolescents and young people aged 15–24, and over age 75 years. These three groups are at the highest risk for a TBI. Mortality rates after brain injury are the highest in people with a severe TBI. P. Pangilinan (2012)claims that, "in the first year after a TBI, people who survive are more likely to die from seizures, septicemia, pneumonia, digestive conditions, and all external causes of injury than are other people of similar age, sex, and race"(p.1). The incidence of fatal TBI is increasing globally.

Conclusions

To conclude, traumatic brain injury, as damage to the brain resulting from external mechanical force, need a serious medical attention. It carries a high morbidity and mortality. The primary causes for TBI include motor vehicle crashes, assaults, falls, and sports or recreation-related injuries. TBI classifications are based on factors of severity, anatomical features of the injury, and the mechanism. Long-term physical, cognitive, and behavioral impairments are associated with TBI. Infants, adolescents, and elderly over age 75 years are at the highest risk for a TBI. Mortality in males is 3 – 4 times higher than in females. By focusing preventive and educational efforts on these high-risk groups, it may be possible to maximize the positive impact on this significant public health problem. Early and rapid management of the initial brain injury and reduction in secondary insults can lead to improved outcomes.

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