ACADEMICIA
An International Multidisciplinary Research Journal
(Double Blind Refereed & Peer Reviewed Journal)

DOI: 10.5958/2249-7137.2021.02374.0

AN OVERVIEW ON TRAUMATIC BRAIN INJURIES

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ABSTRACT

Traumatic brain injury (TBI) may manifest itself in a variety of ways, ranging from minor changes in awareness to a permanent vegetative condition and death. In the most severe form of TBI, a diffuse kind of damage and edema affects the whole brain. Treatment options range from regular cognitive therapy sessions to drastic surgery such as bilateral decompressive craniectomies, depending on the degree of the damage. Guidelines for the best treatment of TBI have been established, but they must be considered in the context of the situation and cannot be used in every case. We have described the present state of TBI therapy in clinical practice and fundamental research in this review paper. We have included a short review of the different subtypes of traumatic injuries, optimum medical treatment, noninvasive and invasive monitoring methods, as well as surgical procedures that may be required in certain cases. We've reviewed the most significant advances in fundamental research in the quest for TBI treatment methods. From an experimental standpoint, we've also addressed the future path of TBI therapy development.

KEYWORDS: *Intracranial Hypertension, Management, Treatment Strategies, Traumatic Brain Injury.*

1. INTRODUCTION

TBI continues to affect millions of people every year all around the globe. The overall combined rates of TBI-related emergency department visits, hospitalizations, and fatalities have risen throughout the decade 2001–2010, according to the Centers for Disease Control. Individually, however, the incidence of TBI-related fatalities has dropped during the same time period, owing in part to greater awareness, more structured management and standards, and substantial technical advances in current treatment regimens. We must also recognize that a certain proportion of TBIs never get medical attention, implying that total TBI rates are likely underreported. TBI is most common in children (0–4 years old), as well as teenagers and young adults (15–24 years old). Another surge in occurrence occurs in those over 65 years old. Falls and motor vehicle accidents are the two most common causes of TBI. We have a rising group of people living with severe impairments directly linked to their TBI as a consequence of an overall increasing number of TBIs but a reduced incidence of associated fatalities[1]–[4].

Path Physiology of TBI:

TBI pathogenesis is a multi-step process that begins with a primary injury and progresses via subsequent injuries to result in temporary or permanent brain impairments. The main deficiency is directly linked to the brain's major external effect. The secondary injury, which may occur minutes to days after the initial hit, is caused by a molecular, chemical, and inflammatory cascade that causes further brain damage. The release of excitatory neurotransmitters like glutamate and aspartate causes depolarization of the neurons, which leads to a rise in intracellular calcium. Intracellular calcium triggers a cascade of events including the activation of enzymes such as caspases, calpases, and free radicals, resulting in cell death either directly or indirectly through the apoptotic process. This deterioration of neuronal cells is accompanied by an inflammatory response that destroys neuronal cells even more and causes a breach in the blood-brain barrier (BBB), resulting in more cerebral edema. Through various mediators, this whole process is both up and down controlled. After the second damage phase, the healing period begins, which includes morphological, molecular, and functional remodeling[5], [6].

Concussion:

Concussions are often regarded as mild TBIs that do not result in any severe structural damage as a result of a non-penetrating TBI. They typically occur as a result of direct strikes to the head, with following acceleration and deceleration forces. A concussion usually leaves the victim with various degrees of temporary altered mental status, ranging from mild disorientation to complete unconsciousness for a few minutes. Routine neuroradiographic imaging, such as computerized axial tomography scans (commonly known as CT scans) and magnetic resonance imaging (MRI) reveal no abnormalities right away. Newer MRI imaging methods such as diffusion tensor imaging and functional MRI, on the other hand, may lead to an earlier diagnosis of concussion. Even in the face of a mild TBI, modest degrees of axonal damage have been hypothesized to occur.

Chronic Traumatic Encephalopathy (CTE):

Mild TBI may lead to CTE, which is a delayed manifestation of mild TBI. This entity has received widespread public attention since one of the terrible outcomes of CTE is mental

problems, which have resulted in suicidal conduct in a number of high-profile players in professional sports. Dysarthric speech, tremors, trouble paying attention, memory and executive function impairments, incoordination, and pyramidal symptoms are some of the other clinical indications of CTE. CTE is most likely the consequence of increasing neuronal loss.

Extra-axial Hematomas:

Epidural hematomas (EDH) and subdural hematomas (SDH) are both types of extra-axial hematomas (SDH). EDH is caused by a direct hit to the temporal area, which may sometimes result in a skull fracture and a rupture of the middle meningeal artery. However, venous injuries, such as transverse sinus disruption, have also been linked to more posteriorly directed EDH. Once a threshold level of intracranial pressure (ICP) is achieved, EDH may quickly increase in size, allowing a person to present with basically normal mentation, followed by worsening along the herniation syndromes cascade.

Traumatic Subarachnoid Hemorrhage and Contusions:

In most cases, contusions occur as a consequence of coup and countercoup forces. Coup injuries happen at the point of impact, while contrecoup injuries happen on the other side of the impact, most often injuring the frontal lobe and anterior temporal lobe. Trauma is the most common cause of subarachnoid hemorrhage, which occurs when tiny capillaries rupture and leak blood transiently into the subarachnoid region. Because blood is pushed into the subarachnoid space under artery pressure in spontaneous aneurysmal subarachnoid hemorrhage, traumatic subarachnoid hemorrhage is often not as serious a brain damage as spontaneous aneurysmal subarachnoid hemorrhage.

Diffuse Axonal Injury (DAI):

The most severe kind of axonal shearing damage is DAI. For such an injury to occur, significant rotational acceleration/deceleration forces are usually needed. Radiographically, it appears as modest hemorrhagic foci in the corona radiata, corpus callosum, internal capsule, brainstem, and thalamus on T2 and gradient echo sequences. Patients may appear with different degrees of clinical symptoms depending on where the axonal shearing occurs. A minority of DAI patients may have altered consciousness for a few days, while others may develop hemiparesis as a result of internal capsule involvement. Others never recover consciousness because portions of the reticular activating system have lost axonal integrity.

TBI Medical Interventions:

i. Height of the Head:

Raising the head of a person who has suffered a traumatic brain injury usually has immediate consequences. The displacement of CSF from the cerebral compartment, as well as the stimulation of venous outflow, both lower ICP. Although ICP is decreased and cerebral blood flow (CBF) is unchanged by head of bed elevation, mean carotid pressure is reduced.

ii. Hyperventilation:

ICP is reduced by hyperventilation because the intra-arterial carbon dioxide partial pressure ($PaCO_2$) is reduced, resulting in vasoconstriction. The decrease of cerebral blood volume is the

end consequence of this sequence of events. Because vasoconstriction decreases CBF, prophylactic hyperventilations are not usually advised. Focal regions of ischemia may develop in areas where autoregulation is maintained. In the case of severe TBI, hyperventilation is typically only required for a short time during acute neurological deterioration.

iii. Hyperosmolar Therapy:

In the event of a TBI, hyperosmolar treatment may be given as a bolus or as an infusion. The initial effects of mannitol have been demonstrated to be caused by changes in blood rheology. A rise in CBF occurs when blood rheology improves and blood becomes less viscous. Transient vasoconstriction is the body's autoregulatory reaction to this, which eventually lowers CBF. Although mannitol has osmotic diuretic characteristics, this method for lowering ICPs is believed to occur after the main impact.

iv. Cooling for Therapeutic Purposes:

TBI is believed to cause oxidative stress as a side effect. Therapeutic hypothermia has been demonstrated to reduce oxidative damage in babies and children. The cerebral metabolic requirement reduces when the body temperature drops. Changes in blood sugar, platelet count, and coagulation factors are all hazards associated with this kind of treatment. When someone is brought to a hypothermic condition, their platelet count and coagulation factors must be evaluated before any invasive treatment. Therapeutic cooling has had inconsistent success in the treatment of severe TBI and is now considered a second-tier treatment option.

v. Monitoring of the ICP:

In the case of ICP monitoring in brain-injured patients, several indications have been proposed as recommendations. Some individuals have clinical symptoms of severe neurological impairment but no obvious signals that they need emergency surgery. In patients with a severe TBI, a GCS of 3 to 8, and an abnormal CT scan of the head, there is Level II evidence for using an ICP monitor. If two or more of the following are observed at admission: age over 40 years, unilateral or bilateral posturing, or systolic blood pressure of 90 mm Hg, Level III evidence supports putting an ICP monitor in patients with a severe TBI and a normal CT scan of the head.

Regeneration of the Neurovascular System:

It's been suggested that neuronal and vascular regeneration play a part in brain healing after a head injury. Neurogenesis has been discovered in the subgranular zone of the hippocampus's dentate gyrus (DG) and the subventricular zone of the adult brain. TBI has been shown to promote neurogenesis in the cerebral cortex, DG, and CA3 in animal models. In cells, Thymosin 4 (T4) is an essential G-actin-sequestering molecule. Tb4 injection promotes NPC growth in animal models. Tb4 also promotes NPC differentiation and increases angiogenesis. There is a specific population of astrocytes in the subventricular and subgranular zones that may divide and differentiate into new neurons. After a TBI, these newborn neurons are thought to play a role in replacing neurons in the olfactory bulb, brain, and hippocampus. The number of regenerated neurons in young animals is higher than in older animals, according to animal models. The process of NPC proliferation and differentiation peaks 2 to 5 days after TBI, but some investigations go as far as 14 days[7], [8].

2. LITERATURE REVIEW

H. Dash et al. discussed about Management of Traumatic Brain Disease[9]. Traumatic brain injury (TBI) has been dubbed the modern-day "hidden pandemic" since it is the greatest cause of death and morbidity in children and young people in both industrialized and developing countries. TBI therapy has experienced a paradigm change in recent years. The Brain Trauma Foundation's protocol-based recommendations are suitable for the treatment of severe TBI. Prophylaxis and early treatment of intracranial hypertension and secondary brain damage, preservation of cerebral perfusion pressure, and providing sufficient oxygen supply to injured brain tissue are the goals and objectives of its management. The authors of this paper address protocol-based methods to the treatment of severe TBI in accordance with current recommendations.

W. Peeters et al. discussed about Traumatic Brain injuries[10]. Traumatic brain injury (TBI) is a major public health and socioeconomic issue throughout the globe, necessitating epidemiological surveillance of TBI incidence, prevalence, and outcomes. The goal of this study was to characterize the epidemiology of traumatic brain injury in Europe and to assess incidence study methods. A comprehensive review and meta-analysis of publications reporting the epidemiology of TBI in European nations were conducted. The keywords epidemiology, incidence, brain injur*, head injur*, and Europe were searched in the PubMed electronic database. Only papers published in English between 1990 and 2014 that reported on data gathered in Europe were included. In all, 28 epidemiological studies on TBI were found in the literature, representing 16 European nations. Between studies, there was a lot of variance in case definitions and case selection. The two most common causes of TBI were falls and road traffic accidents (RTA), with falls being recorded more often than RTA. TBI incidence peaked in the oldest age groups in the majority of studies. An overall incidence rate of 262 per 100,000 for hospitalized TBI was calculated in the meta-analysis. Differences in inclusion criteria and case ascertainment make it difficult to interpret published epidemiologic research. Despite this, changes in epidemiological trends have been discovered: falls are now the most frequent cause of TBI, particularly in the elderly. For accurate monitoring of epidemiological trends and to guide proper targeting of preventive efforts, the quality of standardized data collection for TBI must be improved.

3. DISCUSSION

Traumatic Brain Injury (TBI) is a disturbance in brain function caused by a blow, bump, or jolt to the head, the head striking an item abruptly and forcefully, or an object piercing the skull and entering brain tissue. Mild TBI, often known as a concussion, Moderate TBI, and Severe TBI are the three major forms of TBI. Concussions are a kind of TBI that is relatively mild. Mild types produce just transient symptoms that go away within a few days or weeks. TBIs with the most severe consequences may result in irreversible brain damage, coma, or death. TBI is most often caused by falls from a bed or ladder, down stairs, in the bath, and other falls, especially in elderly people and children.

4. CONCLUSION

A severe hit or jolt to the head or body typically causes traumatic brain damage. Despite the fact that there is no effective therapy for TBI rehabilitation today, attempts to create therapeutic

methods for TBI recovery have been ongoing for decades. In the acute treatment of TBI patients, standard medical and surgical procedures are always important. The number of TBI survivors with different impairments has grown owing to the increasing population of TBI survivors as a result of improved acute treatment recommendations in the acute phase of TBI. This necessitates a transition in TBI research to the fields of neurorestoration and neurorehabilitation.

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