Indian Journal of Health Care, Medical & Pharmacy Practice Vol 5; Issue 1, Jan-Jun 2024, ISSN 2583-2069

Review Article

TRAUMATIC BRAIN INJURY AND ITS MANAGEMENT

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DOI: https://doi.org/10.59551/IJHMP/25832069/2024.5.1.170

Received: 16 May, 2024, Decision for Acceptance: 12 June, 2024

Abstract

A prevalent illness with a high morbidity and death rate is head injuries. Early detection and evacuation are crucial for serious cerebral hemorrhages in order to optimize the likelihood of independent recovery. It is the primary cause of death for children and young people and a significant medical and socioeconomic issue. The Brain Trauma Foundation's "Guidelines for the Management of this disease" are a major source of inspiration for critical care management of this injury. The primary goals are to maintain cerebral perfusion pressure (CPP), optimize cerebral oxygenation, and prevent and cure intracranial hypertension and secondary brain injuries. The care management of (TBI) will be covered in this review, with particular attention paid to monitoring, preventing and minimizing subsequent brain insults, and optimizing cerebral oxygenation and CPP. The influences of worrying mind damage can be profound and lengthy-lasting, affecting all aspects of someone's life. However, with suitable assist, rehabilitation, and adaptive techniques, many individuals with TBI are capable of lead pleasant and significant lives.

Keywords: Traumatic Brain Injury, Head Injury, Head Trauma, Critical Care

1. Introduction

Any harm to the head is referred to as a head injury, which can include brain injuries, fractures of the skull and face bones, and harm to one's particular senses. Adult head injuries most frequently result from falls (37%) and auto accidents (40%). Children are more likely to fall (24%), have incidents where the child is a pedestrian (36%), and both[2]. Head injuries, which have an incidence of 150–450 cases/100,000/year, cause of morbidity and mortality worldwide at all ages[3,4]. These patients should be initially assessed and managed in accordance with the Advanced Trauma Life Support algorithm[1]. Pupil size and reaction, focused neurology, and the Glasgow Coma Score should be the main areas of focus during a neurological assessment.

A serious and difficult issue in critical care medicine is severe (TBI), which is defined as head trauma linked to a Glasgow Coma Scale (GCS) score of three to eight. A great deal has been learnt and progress has been made in the critical care management of brain injury over the last 20 years. The first recommendations for treating (TBI) were released by the Brain Trauma Foundation (BTF) in 1996[2].

The third edition was released in 2007 after the next edition was released in 2000 and updated in 2003. The effects of using guidelines- procedures

for this injury on patient care and results have been documented in a number of research. These studies have unequivocally shown that following guidelines and putting procedures into practice for the treatment of (TBI) is linked to significantly improved results in terms of death rate, functional outcome scores, duration of hospital stay, and expenses[3]. Nonetheless, there is still a great deal of institutional diversity in the way that individuals with severe TBI are treated.

Generally speaking, there are two distinct phases of (TBI): primary and secondary. The initial state of this disease is caused by physical damage to the parenchyma (tissue and vessels) after a traumatic event, which causes the surrounding brain tissue to shear and compress. In the hours and days that follow the initial brain injury and compound it, a complicated process leads to the secondary brain injury. Many additional brain insults, such as extra-cranial, intracranial, and systemic ones, which worsen the initial brain injury, might result in secondary brain injury.

Therefore, it is now evident that the primary brain injury, which is irreversible and cannot be changed, is only partially responsible for the brain damage caused by head trauma. On the other hand, secondary brain damages can frequently be avoided or reversed. Patients with (TBI) require dynamic intensive care management, which begins at the accident scene during the pre-hospital phase. Before being sent to the (ICU), patients may receive initial hospital care in a number of settings, such as the radiology department, emergency department, or operating room[5]. It is important to guarantee the continuity of acute treatment during the "GOLDEN HOUR," from the moment of damage until the onset of definitive care, and to base this on the previously mentioned principles and suggestions. The essential concepts of care for patients with (TBI) during their ICU stay are described in this study.

2. Background

Globally, (TBI) is one of the main causes of death and disability. In order to improve neurological outcomes and death rates, who have traumatic brain injuries frequently need emergency neurosurgery to remove post-traumatic mass lesions and/or invasive neuro monitoring to guide and customize therapy. Taking into account the aforementioned, patients suffering from (TBI) ought to be admitted to a hub trauma center, which is a hospital equipped with neurosurgical capabilities. This will facilitate the prompt provision of appropriate care in a specialized setting. Due to patient- or geography-related reasons, severe TBI patients are occasionally admitted to a spoke hospital—a hospital lacking neurosurgery capabilities[6].

At a typical hospital, there is currently a dearth of knowledge on the best ways to treat patients with severe solitary traumatic brain injuries. This consensus's particular goal is to offer guidance on the prompt treatment of severely isolated patients who are admitted to spoke hospitals and during their transfer to hub hospitals. Specifically, we are referring to patients who are admitted to an urban hospital in a high-income nation that does not have neurosurgery capabilities but does have an (ICU), an (OR), and a (CT) scan available. For initial therapy in cases of (TBI) with extracranial lesions or hospital admissions with limited resources, readers can consult a number of published publications and guidelines.

3. Practice Essentials

(TBI) is a leading cause of disability and death. It happens when an abrupt trauma ruins brain tissue and interferes with regular brain function. TBI may have significant effects on one's physical, mental, emotional, cognitive, and social well-being[7].

4. Classification

Primary and secondary injuries:

A. Primary Injury: Primary injury is caused by two basic mechanisms: contact (e.g., an object impacting the head, or the brain striking the inside of the skull) and acceleration-deceleration. Contact is induced by mechanical force and occurs at the instant of injury.

B. Secondary Injury: Not caused by a machine; it

could happen later after impact, and it could add damage to a brain that has already been harmed by a machine.

- Focal and diffuse injuries

These injuries are commonly found together; they are defined as follows:

A. Focal Injury: Includes scalp injury, skull fracture, and surface contusions; generally caused by contact[8]

B. Diffuse Injury: Includes diffuse axonal injury (DAI), hypoxic-ischemic damage, meningitis, and vascular injury; usually caused by acceleration-deceleration forces

5. Complications[9]

Complications include the following:

A. Physical Headaches: TBI can lead to numerous physical problems, together with complications, dizziness, fatigue, seizures, muscle spasticity, stability and coordination problems, and sensory disturbances consisting of changes in vision, listening to, taste, or scent.

B. Cognitive Headaches: TBI may also cause difficulties with questioning, memory, attention, and government functions (consisting of making plans, problem-fixing, and judgment). These cognitive impairments can impact daily functioning and excellent of lifestyles.

C. Emotional and Behavioral Headaches: TBI survivors regularly experience emotional and behavioral changes, consisting of mood swings, irritability, despair, anxiety, aggression, impulsivity, and social dis inhibition. These changes can strain relationships and have an effect on social interactions.

D. Communication Issues: TBI can bring about difficulties with speech, language, and verbal exchange. This can also encompass aphasia (problem speaking or understanding language), dysarthria (trouble with speech articulation), or cognitive-communication deficits.

E. Sensory Deficits: TBI may result in sensory impairments, together with lack of imaginative and prescient, listening to, or sensation. These deficits can affect one's capacity to understand and interact with the environment.

F. Sleep Disturbances: TBI survivors often experience sleep problems, which include insomnia, immoderate daylight hours sleepiness, sleep apnea, or changes in sleep-wake cycles. Poor sleep can exacerbate different TBI symptoms and impair healing.

G. Post-annoying Epilepsy: Some individuals develop epilepsy after experiencing a worrying mind damage. Seizures may occur straight away after the damage or increase later, every now and then years afterward.

H. Degenerative Brain Illnesses: There is proof linking TBI to an expanded hazard of growing neurodegenerative sicknesses later in lifestyles, consisting of Alzheimer's disease, Parkinson's disease, or chronic disturbing encephalopathy (CTE).

I. Secondary Accidents: TBI also can predispose people to secondary injuries, which includes infections, hydrocephalus (accumulation of fluid inside the mind), or intracranial hemorrhage.

J. Psychosocial Complications: TBI survivors regularly face challenges in returning to work or faculty, retaining relationships, and adjusting to changes of their skills and lifestyle. These psychosocial elements can drastically effect recovery and rehabilitation.

6. Management of TBI

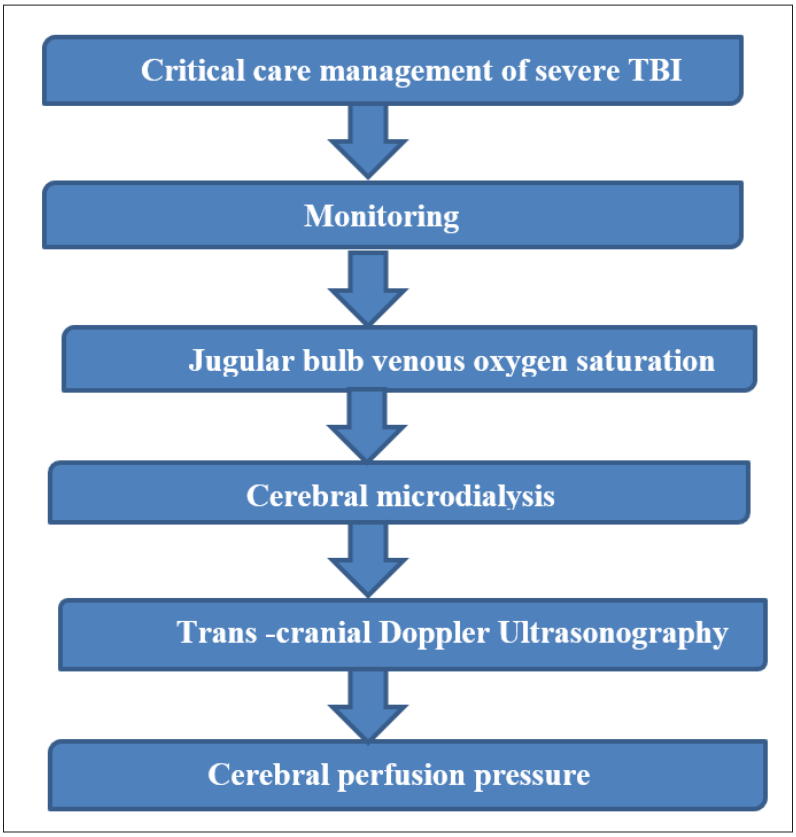

Figure 1: Flow chart for Management of TBI

6.1 Care management of severe TBI

Patients who suffering, typically seen, revived, and stabilized in an emergency room or operating room before being sent to the intensive care unit. Following their transfer to the intensive care unit (ICU), patients with serious head injuries are managed with a combination of high-quality general care and techniques to maintain hemostasis, such as: - Stabilization of the patient, if still unstable - Avoiding intracranial high blood pressure - Preserving a sufficient and steady cerebral perfusion pressure (CPP) Preventing systemic (SBI) and optimizing cerebral oxygenation and hemodynamics[11].

6.2 Monitoring

It is crucial to monitor individuals suffering from this disorder in order to guide and optimize treatment. The justification for analyzing the CSF for culture and sensitivity, protein, glucose, cell count, and Gram stain. ICP thresholds greater than 20 mm Hg should be the starting point for intracranial hypertension treatment. ICP monitoring has become a crucial component of the treatment of patients with severe traumatic brain injury in the majority of trauma centers. In addition to ICP readings, monitoring enhances outcome or supports its usage as standard. On the other hand, there is conflicting data regarding whether ICP monitoring enhances results. ICP monitoring has been shown in multiple trials to lower the overall death rate from this disease.

ICP monitoring has not been found to be beneficial in other trials. Furthermore, a few studies have shown a correlation between ICP monitoring and a decline in survival. ICP monitoring malfunction, bleeding, infection, blockage, or malposition are possible side effects. Our recent analysis revealed that ICP monitoring in patients with severe traumatic brain injury did not correlate with a lower hospital death rate. However, it did significantly increase the duration of mechanical breathing, the necessity for tracheotomy, and the length of stay in the intensive care unit.A recent systematic review of the Cochrane

database yielded no RCTs that shed light on the function of ICP monitoring in acute coma, whether caused by trauma or not.

Raised ICP was linked to higher odds ratios (OR) of mortality compared to normal ICP (< 20 mm Hg). The odds ratios (ORs) for death or a poor neurological outcome increased three to four times in correlation with elevated but reducible ICP. A significant rise was linked to a refractory ICP pattern.

6.3 Jugular bulb venous oxygen saturation

Jugular venous oxygen saturation (SjvO2), which represents the relationship between cerebral blood flow (CBF) and cerebral metabolic rate of oxygen (CMRO2), is a measure of both cerebral oxygenation and cerebral metabolism. For SjvO2 monitoring, an internal jugular vein (IJV) retrograde catheterization is utilized. Since the right IJV is typically prominent, cannulation to represent the global cerebral oxygenation often uses it. SjvO2 can be intermittently monitored with repeated blood samples or continuously monitored with a fiberoptic device. Cruz, in a prospective trial of patients with intracranial hypertension and severe acute brain trauma, found that continuous SjvO2 monitoring was linked to better results.

In a normal awake subject, the normal average of the SjvO2 is 62%, which is irrespective of the severity of the injury, low CPP, and excessive ICP. After a severe traumatic brain injury, PbtO2 might be a crucial therapeutic target. It has been shown that PbtO2 is more effective at detecting cerebral ischemia than SjvO2, regional transcranial oxygen saturation, and near infrared spectroscopy. Although it is not commonly used or readily available, PbtO2 monitoring is a promising, safe, and therapeutically useful approach in patients with severe traumatic brain injury. ICP/PbtO2 intra-parenchymal monitoring combinations are significant and beneficial methods in the treatment of severe traumatic brain injury.

6.4 Cerebral microdialysis

A new invasive laboratory technique called cerebral microdialysis (MD) uses a bedside monitor to examine the biochemistry of brain tissue. In order to measure biochemical changes in the part of the brain most sensitive to secondary insults, an MD catheter is typically placed into "susceptible" brain tissue. Dialysate concentrations can be determined using a variety of assays, including those that detect glycerol, glutamate, pyruvate, lactate, and glucose. The lactate:pyruvate ratio (LPR) typically rises significantly in cases of cerebral hypoxia or ischemia. An LPR of 20–25 is thought to be a threshold for cerebral ischemia and is linked to a poor prognosis in traumatic brain injury[13]. The utilization of MD, a well-respected instrument that offers extra support in the treatment of patients with this disease, is quite restricted.

6.5 Transcranial Doppler Ultrasonography

CBF velocity can be measured non-invasively using transcranial Doppler (TCD). It is being used more frequently in neurocritical care, which includes TBI. The detection of problems such as vasospasm, significant increases of ICP and decreases in CPP, carotid dissection, and cerebral circulatory arrest (brain death) that may occur in patients with TBI can be aided by this clinically valuable technique. Before post-traumatic vasospasm manifests clinically, TCD can anticipate it. TCD has been proposed as a non-invasive substitute method for ICP and CPP measurement because ICP monitoring is an intrusive operation with possible side effects. TCD has an overall specificity of 98% and sensitivity of 75% to 88% for confirming brain death. While TCD is a well-recognized monitoring technique in neurocritical care, there is insufficient data to justify its routine application for ICP/CPP management in patients with severe traumatic brain injury.

6.6 Cerebral perfusion pressure

After this disease, cerebral ischemia is thought to be the most significant subsequent event influencing the prognosis. CPP should be avoided if it is less than 50 mm Hg. CPP is calculated as the MAP minus ICP (CPP = MAP - ICP). Brain regions that already have ischemia may be at risk due to a low CPP, and raising

CPP may help prevent cerebral ischemia. At least 60 mm Hg should be maintained as the CPP value to goal above the ischemia threshold. After a severe traumatic brain injury (TBI), maintaining a CPP higher than 60 mmHg is a treatment approach that may be linked to a significant decrease in mortality and an improvement in quality of survival.It also likely to improve perfusion to ischemic areas of the brain.

There is no proof that actively maintaining CPP above 60 mmHg with normalized intravascular volume or causing systemic hypertension increases the incidence of intracranial hypertension, morbidity, or mortality[14]. The literature lists two values for the threshold at which CPP should be maintained: 60 mm Hg and 70 mm Hg. When cerebral ischemia is not present, the CPP should be kept at least 60 mm Hg, and when it occurs, it should be kept at least 70 mm Hg. It has been proposed to use PbtO2 monitoring to determine each person's ideal CPP. The risk of ARDS makes vigorous measures to maintain CPP above 70 mm Hg with fluids and vasopressors unnecessary in the absence of cerebral ischemia.

7. Critical Care Management Guidelines

Critical care management of excessive traumatic brain injury (TBI) necessitates a meticulous and multidisciplinary technique geared toward minimizing similarly brain harm, optimizing cerebral perfusion, and assisting the patient's normal physiological balance. Upon admission to the extensive care unit (ICU), immediate interest is directed in the direction of securing the airway, ensuring adequate oxygenation, and retaining hemodynamic balance. This commonly involves endotracheal intubation and mechanical air flow, as well as the administration of fluids and vasopressors to obtain appropriate perfusion stress. A thorough neurological assessment is conducted, encompassing assessment of awareness level using the Glasgow Coma Scale (GCS), pupillary reactivity, and evaluation of motor responses. Emergent neuroimaging with computed tomography (CT) is performed to perceive any acute intracranial pathologies together with hematomas or contusions.

Continuous tracking of intracranial strain (ICP) is initiated to guide healing interventions and prevent secondary mind damage. Various modalities for ICP tracking are available, along with intraventricular catheters, intraparenchymal monitors, and subdural or epidural sensors. Elevated ICP is managed aggressively through a mixture of scientific and surgical interventions. Pharmacological strategies consist of osmotic retailers which include mannitol or hypertonic saline to reduce cerebral edema, sedatives to decrease metabolic call for, and neuromuscular blockade to prevent will increase in ICP due to patient agitation. If medical management fails to govern ICP thoroughly, surgical interventions including decompressive craniectomy may be considered to relieve intracranial hypertension.

Optimizing cerebral perfusion strain (CPP) is paramount in stopping secondary mind harm. CPP is calculated as the distinction between imply arterial pressure (MAP) and ICP. Current recommendations advise keeping CPP inside more than a few 60-70 mmHg to make sure adequate cerebral blood flow while minimizing the risk of cerebral ischemia or hyperemia. Achieving this target may additionally involve aggressive blood strain control with vasopressors or inotropes to elevate MAP within the setting of accelerated ICP.

Preventing and managing complications related to severe TBI is critical for optimizing effects. Measures to save you secondary insults encompass retaining normothermia to minimize metabolic call for, stopping hypotension and hypoxia, and avoiding hypercapnia and hyperglycemia. Seizure prophylaxis with antiepileptic tablets can be considered, particularly in patients with cortical contusions or penetrating head injuries. Early initiation of enteral or parenteral vitamins is essential to fulfill the increased metabolic needs of the injured brain and support standard healing.

In addition to medical management, early mobilization and rehabilitation interventions are

initiated to save you complications of immobility and promote practical recuperation. Physical remedy, occupational therapy, and speech therapy are crucial additives of the rehabilitation procedure, aimed at maximizing the patient's independence and quality of life submit-injury.

Psychological aid for each the affected person and their circle of relatives participants is a essential component of essential care management in severe TBI. The psychological impact of TBI may be profound, and addressing the emotional wishes of patients and their cherished ones is critical for promoting coping and resilience in the course of the recovery manner. Clear and compassionate communique with family participants, supplying ordinary updates on the affected person's circumstance and analysis, helps to foster believe and collaboration in the care crew.

7.1 Temperature Modulation

At 32°C to 34°C, there is moderate systemic hypothermia, which lowers cerebral metabolism and CBV, raises CPP, and lowers ICP. There was conflicting evidence about how mild hypothermia affected TBI patients' prognosis.research initially indicated that moderate hypothermia, as determined upon admission, was associated with a considerably better prognosis. However, no effect of moderate hypothermia on outcome following traumatic brain injury has been shown in a major RCT.

Patients with severe TBI who were included in the National Acute Brain Injury Study: Hypothermia II clinical trial between two and five hours after injury were part of a multicenter, randomized clinical trial. Patients were randomized to either normothermia or hypothermia (cooling to 33° C for 48 hours). The hypothermia and normothermia groups did not significantly differ in their results[17]. The efficacy of hypothermia as the main neuroprotective tactic for individuals with severe traumatic brain injury was not supported by the experiment. In patients with suffering with this, however, fever should be aggressively treated and the temperature should be regulated. If the ICP is uncontrolled and refractory, moderate hypothermia may be utilized.

7.2 Antiseizure prophylaxis

There are two types of post-traumatic seizures: those that happen early, within 7 days of the injury, and those that happen later, after 7 days of the injury. It is not advised to use preventive medication (phenytoin, carbamazepine, or phenobarbital) to stop late post-traumatic seizures. However, prophylactic therapy was advised by the BTF for TBI patients who are at high risk of having seizures in order to prevent early post-traumatic seizures. GCS score less than 10, cortical contusion, depressed skull fracture, infections, and septic sequelae are among the risk factors.

Patients who suffer from (TBI) often experience dysphagia for a variety of reasons, including as irregular stomach emptying, altered stomach function as a result of elevated intracranial pressure (ICP), and opioid use. Tolerance is enhanced by prokinetic drugs like erythromycin or metoclopramide. Higher calorie and nitrogen intake is possible with postpyloric feeding, which also prevents stomach intolerance. A increasing amount of research points to the advantages of consuming less calories, even though the BTF advised replacing 100% of resting metabolic expenditure in patients who are paralyzed and 140% of resting metabolic expenditure in people who are not.

7.3 Glycemic control

Glycemic manage in excessive disturbing mind harm (TBI) is a crucial element of control geared toward optimizing effects and reducing the hazard of secondary brain damage. Traumatic brain injury frequently leads to dysregulation of glucose metabolism, ensuing in hyperglycemia, that can exacerbate neuronal damage, increase cerebral edema, and worsen neurological outcomes. Conversely, hypoglycemia can impair cerebral strength metabolism and exacerbate secondary brain injury. Therefore, maintaining euglycemia is crucial for minimizing in addition harm to the injured brain.

Current pointers recommend tight glycemic control in sufferers with extreme TBI, commonly focused on blood glucose levels within the range of one hundred forty-180 mg/dL (7.Eight-10 mmol/L). This goal variety balances the want to keep away from hyperglycemia-caused neuronal harm even as minimizing the threat of hypoglycemia-associated headaches. Continuous glucose tracking or frequent factor-of-care glucose testing is often utilized inside the ICU putting to closely monitor blood glucose stages and manual insulin therapy titration.

Insulin remedy is the mainstay of glycemic manage in severe TBI patients with hyperglycemia. Continuous intravenous insulin infusion allows for unique manipulate of blood glucose levels even as minimizing the chance of hypoglycemia. Insulin infusion protocols are normally protocolized and titrated based on frequent glucose measurements to reap and keep goal glucose ranges. Close tracking of serum electrolytes, particularly potassium and phosphorus, is essential for the duration of insulin remedy to save you electrolyte imbalances.

In addition to insulin remedy, other measures may be hired to optimize glycemic manage in extreme TBI patients. These consist of early initiation of enteral or parenteral nutrition to offer glucose substrates and save you catabolism, in addition to avoidance of immoderate dextrose-containing fluids to prevent hyperglycemia. Corticosteroids, that could exacerbate hyperglycemia, are usually prevented in the management of extreme TBI unless indicated for precise reasons together with spinal twine harm.

7.4 Predicting outcome after TBI

Predicting outcomes following Traumatic Brain Injury (TBI) entails a multifaceted assessment thinking about various factors. The Glasgow Coma Scale (GCS) serves as a fundamental tool, gauging the initial severity of harm via measures of cognizance. Higher GCS scores often correlate with better prognoses. Age performs a crucial function, with more youthful people normally showing greater resilience and recovery ability in comparison to older opposite numbers. The severity and nature of the harm itself, encompassing factors like length of unconsciousness and neuroimaging findings, significantly impact results.Moreover, the vicinity of the injury in the brain and its precise kind, whether contusion, hemorrhage, or diffuse axonal damage, contribute to prognostic assessments. Complications, which includes infections or seizures, can obstruct recovery trajectories. Pre-existing fitness situations further complicate prognoses, whilst rehabilitation offerings and robust help systems appreciably impact practical effects and satisfactory of existence publish-damage. Despite these concerns, predicting outcomes remains challenging due to the elaborate nature of mind damage and character variations in response to treatment and rehabilitation. Thus, prognostic assessments necessitate the know-how of healthcare specialists well-versed in TBI control, spotting the inherent variability in recuperation trajectories.

8. Impacts of (TBI)

(TBI) can have significant and varied impacts on individuals, both physically and mentally. Here are some of the impacts associated with TBI:

8.1 Impacts

1. Physical consequences: TBI can result in a range of bodily signs, such as complications, dizziness, fatigue, motor coordination problems, and sensitivity to mild and sound. These signs and symptoms can effect an character's potential to carry out day by day activities and may require ongoing medical treatment. [17]
2. Cognitive impairments: TBI frequently consequences in cognitive deficits which include memory troubles, trouble concentrating, impaired judgment, and slower processing pace. These impairments can have an effect on work, school, and interpersonal relationships.
3. Emotional and behavioral changes: TBI can adjust someone's temper, behavior, and character. Depression, tension, irritability, impulsivity, and temper swings are commonplace emotional consequences of TBI. These modifications can

strain relationships and make it tough to preserve social connections.

4. Social and vocational demanding situations: TBI survivors may additionally face difficulties reintegrating into society and preserving employment. Social isolation, stigma, and discrimination can similarly compound these challenges. [18]
5. Financial burden: The cost of medical treatment, rehabilitation, and ongoing take care of TBI can be good sized. Additionally, individuals might also experience lack of earnings due to incapacity or unemployment, similarly exacerbating economic stress.

9. Conclusion

In order to prevent secondary mind insults, maintain an ok CPP, and maximize cerebral oxygenation, the control of excessive traumatic brain harm (TBI) revolves around cautious and complete intensive care. This care consists of a multi-version, protocolized approach concerning cautious hemodynamic assist, respiratory care, fluid control, and different components of therapy. A multidisciplinary team of neurointensivists, neurosurgeons, bedside nurses, respiratory therapists, and other scientific crew participants is obviously had to put in force this strategy. Given the age of the victims and the socioeconomic effect of the issue, such management can be difficult, but it is also very gratifying. The influences of worrying mind damage can be profound and lengthy-lasting, affecting all aspects of someone's life. However, with suitable assist, rehabilitation, and adaptive techniques, many individuals with TBI are capable of lead pleasant and significant lives.

Conflict of Interest: None

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Cite this article Pal U et al, Traumatic Brain Injury and its Management. Indian Journal of Health Care, Medical & Pharmacy Practice.2024; 5(1) 134-143.