Traumatic Brain Injury – A Neurologist's Approach

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Traumatic brain injury (TBI) is defined as brain injury caused by an external force. It remains a major global health concern, contributing significantly to death and disability across all age groups. In the United States, there were approximately 214,000 TBI-related hospitalizations in 2020 and 69,000 TBI-related deaths in 2021, averaging over 586 hospitalizations and 190 deaths per day. Individuals aged 75 years and older exhibited the highest rates of TBI-related hospitalizations and deaths. Males were nearly twice as likely to be hospitalized and three times more likely to die from a TBI than females.[1]

TBI is increasingly recognized not only as an acute insult but also as a chronic disease process that evolves over time. It carries the potential for long-term cognitive, emotional, and physical disability, underscoring the need for a comprehensive, multidisciplinary approach to care – from the point of injury through rehabilitation.

TBI is typically classified by severity into mild, moderate, and severe categories using the Glasgow Coma Scale (GCS). A GCS score of 13–15 indicates mild TBI, 9–12 indicates moderate, and 3–8 signifies severe injury. Further stratification involves structural imaging (CT/MRI), duration of loss of consciousness (LOC), alteration of consciousness, and post-traumatic amnesia (PTA).[2]

Contemporary classification schemes, including the Mayo Classification System[2] and the use of biomarkers and advanced imaging techniques, aim to provide a more nuanced characterization of injury severity and potential outcomes. Increasingly, the emphasis is shifting from static grading to dynamic, physiology-informed classification systems that account for evolving intracranial pathophysiology.

PRE-HOSPITAL EVALUATION AND MANAGEMENT

The pre-hospital phase is critical in the management of TBI, as early interventions can significantly influence outcomes. Following primary injury, secondary insults from hypoxia, hypoperfusion, and/or ischemia may occur in the pre-hospital setting. Key priorities include ensuring airway patency, providing adequate ventilation to prevent hypoxia, and maintaining cerebral perfusion by avoiding hypotension. Cervical spine precautions should be implemented until spinal injuries are ruled out. Rapid transport to a facility equipped to manage TBI is essential. Pre-hospital providers should perform frequent pupillary response assessments and report GCS score every 30 minutes or with any change in mental status, which could indicate early signs of herniation or increased intracranial pressure (ICP). Administration of hyperosmolar therapy for prophylactic treatment of suspected elevated ICP, with or without signs of herniation, in the pre-hospital setting is not recommended.[3]

IN-HOSPITAL INITIAL EVALUATION AND MANAGEMENT

Upon arrival at the hospital, patients with suspected TBI undergo a comprehensive assessment following Advanced Trauma Life Support (ATLS) protocols.[4] This includes a primary survey focusing on airway, breathing, circulation, disability (neurological status), and exposure. Neurological evaluation involves determining the GCS score and assessing pupil reactivity. A non-contrast head CT scan is the imaging modality of choice for detecting intracranial hemorrhages, contusions, and skull fractures. Laboratory evaluations may include coagulation profiles, blood glucose levels, and arterial blood gases. Early neurosurgical consultation is warranted for patients with mass lesions or deteriorating neurological status.[5]

The 4th Edition Guidelines for the Management of Severe Traumatic Brain Injury provide evidence-based recommendations for both treatment and monitoring strategies specific to adult patients with severe TBI.[5] Most treatment strategies are aimed at reducing intracranial pressure which can be elevated following severe TBI. Decompressive craniectomy (DC) is the most definitive and rapid means of reducing or relieving elevated intracranial pressure. Large fronto-temporo-parietal DC is recommended (Level II A) for improved mortality and neurological outcomes in select patients. However, early bifrontal DC, while effective in lowering intracranial pressure (ICP), reducing ICU days, and lowering mortality, was associated with more unfavorable outcomes at six months and did not show six-month functional improvement as measured by the Glasgow Outcome Scale–Extended (GOS-E).[6,7] Evidence supports the use of hypothermia as standard of care for neuroprotection after cardiac arrest from acute coronary syndromes.[8] When hypothermia is induced early after injury and prior to intracranial pressure elevation, it is termed "prophylactic". Prophylactic hypothermia lacks

sufficient evidence for a strong recommendation, as current studies are highly heterogeneous, preventing definitive conclusions.[5] Hyperosmolar therapy also reduces intracranial pressure and remains a mainstay of ICP management, but no single agent, such as mannitol or hypertonic saline, is clearly favored based on current data.[5] Cerebrospinal fluid (CSF) drainage is acknowledged for its utility in reducing ICP, though the evidence base is still developing.[5, 9,10]

Ventilation therapies (previously with emphasis on hyperventilation) are approached with caution. The emphasis is on tailored ventilation strategies that reduce ICP without compromising cerebral perfusion.[5] The use of anesthetics, analgesics, and sedatives in severe TBI remains guided largely by clinical judgment due to the low quality of available evidence.[5] Corticosteroids, particularly high-dose methylprednisolone, are not recommended (Level I), given strong evidence of harm.[5] Nutritional support should be initiated early – preferably within five to seven days post-injury – as evidence suggests a positive impact on recovery.[5] Infection prophylaxis now focuses on targeted strategies like oral care and management of ventilator-associated pneumonia (VAP). Prophylaxis against VAP has been previously supported by ANTHARTIC trial (patients after cardiac arrest)[11] and most recently by PROPHY-VAP which focused on patients with acute brain injury (including stroke, subarachnoid hemorrhage, TBI), this showed a decreased risk of VAP, decreased ventilation days, decreased prolonged ICU and hospital stay, and decreased mortality.[12]

For deep vein thrombosis (DVT) prophylaxis, a Level III recommendation supports the use of low molecular weight heparin (LMWH) or unfractionated heparin (UFH) in combination with mechanical prophylaxis, provided the hemorrhagic risk is acceptable. Finally, clinicians routinely prescribed antiseizure medications of post-traumatic seizure (PTS) prophylaxis despite lacking clear clinical evidence or supporting guidelines. There is modest effectiveness in PTS prophylaxis in mild to moderate TBI.[13] Phenytoin is recommended (Level II A) for early seizure prophylaxis for post-traumatic seizures (PTS) as it is effective in reducing seizures within the first seven days post-injury, though not for preventing late-onset seizures in severe TBI.[14] However, other antiseizure medications such as levetiracetam may pose less risks. It is important to mention that up to one-quarter of patients are inappropriately discharged with antiseizure medications after failure to stop prophylactic medications after seven days.[15] Prolonged and unnecessary antiseizure medication usage may also inhibit recovery from TBI, especially in moderate and severe TBI.[15]

Looking ahead, multimodal monitoring (MMM) represents a shift toward precision neurocritical care and is increasingly being employed with the goal to improve outcomes in patients with severe TBI.[16] ICP monitoring remains a foundational component of TBI management. The BEST:TRIP trial, however, highlighted the shortcomings of relying on ICP monitoring alone and emphasized the importance of using integrated monitoring strategies.[17] Cerebral perfusion pressure (CPP) monitoring is similarly supported with a Level II B recommendation. CPP-guided therapy has been shown to lower two-week mortality, although the overall quality of evidence remains limited.[5] Advanced cerebral monitoring (ACM) techniques are gaining interest as adjuncts to traditional methods.[16] Rather than applying a one-size-fits-all approach, MMM supports individualized treatment strategies based on real-time physiologic data. Future advancements include the development of validated multimodal algorithms, less invasive technologies like near-infrared spectroscopy, and the integration of artificial intelligence for real-time data interpretation and clinical decision support. While not yet standard practice, MMM offers a promising framework for improving outcomes in patients with severe TBI.

EARLY COMPLICATIONS OF TBI

Early complications following TBI can significantly influence patient outcomes, so they require close monitoring and timely intervention. TBI experimental animal models are used to replicate human pathophysiology and clarify aspects of primary and secondary brain injury.[17]

Early damage in TBI often follows from an ischemic cascade and disruption of normal metabolic energy processes such as decreased glucose utilization, lactic acid accumulation, reduced ATP usage, excitotoxicity, and cellular death.[18] One of the most critical concerns is elevated ICP, which can progress to brain herniation – a life-threatening emergency that demands immediate treatment. Seizures are another common complication, with approximately 10% of individuals hospitalized for moderate to severe TBI experiencing post-traumatic seizures, most often within the first few days to weeks after injury.[19] Coagulopathy is also frequently observed, as TBI can disrupt the coagulation cascade and cause platelet dysfunction akin to disseminated intravascular coagulation (DIC), which increases the risk of both intracranial hemorrhage and cerebral ischemia.[20-21] Additionally, neurogenic pulmonary edema may develop because of acute brain injury, posing significant challenges for respiratory management.[22] Infections such as ventilator-associated pneumonia and surgical site infections are prevalent among TBI patients due to factors such as prolonged hospitalization, mechanical ventilation, and compromised immune responses.[5] Prompt recognition and management of these early complications are essential to improving short- and long-term outcomes in patients with severe TBI.

REHABILITATION STRATEGIES BEGINNING IN THE HOSPITAL

Early initiation of rehabilitation is essential for long-term recovery in almost all types of injuries. For TBI, hospital-based

cognitive rehabilitation offers little to no effect on return-to-work rates, but post-acute care becomes vitally important.[23] Compared to hypoxic-anoxic ischemic brain injury, better functional outcomes can be achieved after traumatic brain injury.[24] Post-acute therapy recommendations differ with severity of TBI, with mild severity needing minimal therapy for likely return to premorbid daily functioning while severe TBI patients have indeterminate and variable outcomes. Mild TBI (mTBI) can be further characterized into uncomplicated and complicated, the latter referring to patients with findings on CT. The Collaborative European NeuroTrauma Effectiveness Research in TBI (CENTER-TBI) showed that complicated mTBI had poorer outcomes than uncomplicated mTBI and that greater duration of therapy predicted poorer outcomes, as patients with more severe injuries needed more intensive treatment.[25] A greater number of transitions of care and pre-morbid psychiatric illnesses also predicted poorer outcomes for mTBI.[23]

In contrast to mild TBI, greater duration of therapies for severe TBI predicted a more favorable prognosis. Recommended therapies include physical (PT), occupational (OT), speech language pathology (SLP), psychiatric (PSY) and cognitive rehabilitation. In physical therapy, greater patient effort and more complex therapy, rather than length of therapy, are associated with improved functional outcomes.[26] Earlier and more intensive occupational therapy has been shown to improve outcomes.[27] Social communication approaches have been shown in a systematic review to be the most effective approach for SLP intervention for moderate to severe TBI.[28]

Overall, TBI functional outcomes are influenced more by patient and injury characteristics than time spent in therapies. For example, mechanism of TBI (assault), CT abnormalities, and premorbid alcohol use predicted worse outcomes on the Glascow Outcome Scale Extended (GOS-E).[29] The GOS-E is an assessment of physical, social and cognitive function that categorizes TBI patients in one of eight levels, from death to upper good recovery.[30] While the GOS-E is widely accepted for TBI outcomes, more complex, structured assessments are often used to quantify TBI outcome after rehabilitation to capture more of the nuanced improvements. One such outcome measure is the Functional Independence Measure (FIM) Cognitive score, which measures 13 motor and five cognitive items and rates patients from one (total assistance) to seven (complete independence).[26] A lower FIM score on admission to rehabilitation centers is associated with patients who had more in-hospital days prior to rehabilitation, Medicaid as primary payer, increased levels of agitation, and younger age.[26] Lower FIM scores on admission for rehabilitation were associated with a longer length of stay and decreased effort with OT/PT/SLP. Patient effort level (as rated by clinicians) during therapies was strongly associated with post-rehab placement, with those showing higher effort more likely to be discharged to a private residence.[26]

Current medical interventions in the post-acute TBI period affect outcomes less than therapies.[26] Though there is data to support anti-seizure medications (ASM) in prevention of early post-traumatic epilepsy, late seizures (greater than six months post-injury) and mortality are not modified by ASMs.[31] Similarly, neuro-protective agents such as magnesium sulfate did not show benefit.[32] In mild TBI, methylphenidate improves cognition, n-acetyl cysteine within 24 hours of injury helps with faster recovery, and galantamine improves episodic memory, but these findings cannot be extrapolated to those with moderate or severe TBI.[33] For severe TBI, amantadine may hasten recovery in the first four weeks after injury, but overall recovery at six weeks after a two-week washout period was not significantly different from placebo.[34]

LATE COMPLICATIONS OF TBI

Most patients with severe TBI will have long-term disability in health, behavior and functional status. The United States Traumatic Brain Injury Model Systems of Care, which has followed individuals with moderate-to-severe TBI for over 30 years, has shown that TBI increases rates of hospitalization and decreases life expectancy compared to the general population.[35] Deficits may not be at peak at diagnosis, either, with evidence for decline overtime. For example, in the United States, the TRACK-TBI LONG study found that additional functional decline occurred in 29% of mild TBI and 23% of moderate to severe TBI to seven years post-injury.[35] Older age and lower acute functional status were associated with higher rates of post-injury decline. Rates of psychotic disorders, attention deficit hyperactivity disorder (ADHD), suicide, and depression are also increased post-TBI compared to a general population, with a relative risk of ADHD as high as 6.49 in the severe TBI cohort.[36]

Chronic traumatic encephalopathy (CTE) has been an increasingly researched entity, thought to occur from repetitive mTBIs. Official diagnosis requires demonstration of tauopathy on autopsy. Traumatic encephalopathy syndrome (TES) has been coined to describe the progressive symptoms associated with presumed CTE. Patient-specific targeted rehabilitation for cognition, executive functioning and emotional control in TES has been shown to improve patient-reported outcomes, with mixed objective significance.[37] Physical exercise has shown to be beneficial for motor function, balance and cognition in tauopathies,[38] and this has been extrapolated to treatment of TES.

Challenges with TBI rehabilitation research include the lack of standardized scoring as well as the observational and longitudinal nature of studies. As discussed previously, GOS-E is the most widely used outcome measure because of its simplicity and flexibility of administration including low administration time, but it may fail to capture symptoms and quality of life after TBI. Because long-term outcome

research requires the passing of time, studies are more logistically demanding and subject to error from patient loss to follow-up.

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None

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