Adult Traumatic Brain Injury

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Introduction

Epidemiology

Traumatic brain injury (TBI) is defined by the Centers for Disease Control (CDC) as any shearing, blunt or penetrating injury to the head that alters normal function [1, 2]. According to the CDC there has been a 53% rise in TBI visits and deaths from 2006 to 2014 with 2.87 million affected in 2014 [1, 3]. This is likely an underestimate as nearly 25% of the population has had self-reported head injury and many do not seek medical care [4]. TBI are typically graded mild to severe. All grades of TBI can have lasting effects on patients and their families. Long term consequences can include memory loss, chronic headaches, neurologic deficits, anxiety, depression, post-traumatic stress disorder and cognitive delays [5–9]. In 2005 3.17 million Americans were living with TBI related disability [10]. Patients

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and families also have significant financial and social impact from TBI. Unfortunately, statistics suggest an upward trend in rates of TBI and TBI related death. TBI related deaths account for 155 deaths per day [1]. The population most likely to suffer a lethal TBI is those 75 years and older [1].

TBIs have a variety of etiologies with most common varying by age group. However, falls account for 48% of TBI related ED visits, being struck by or against an object are 2nd accounting for 17% [1]. Falls are more likely than other etiologies to lead to hospitalization with motor vehicle collision in a distant second [1]. Self-harm related TBI accounts for nearly a third of TBI related deaths despite not being in the top 3 etiologies of TBI; this is likely secondary to the lethality and intent associated with these injuries. This population has also, unfortunately, seen the largest rise in occurrence from 2006 to 2014, rising by 60% [1].

Pathophysiology

Traumatic brain injury can be broken up into two components, primary and secondary brain injury. Primary brain injury is a result of the direct forces and acceleration and deceleration [10–13]. Following this initial trauma secondary brain injury occurs. This is the more complicated and less understood process that is believed to contribute many of the long-term complications and variability in patient symptoms and outcomes [10–14]. Secondary injury involves activation of inflammatory cascades, increased metabolic demand, ischemia, and edema. Secondary injury is a frequent cause of morbidity and mortality in TBI patients. Fatima et al. describes the alterations in cerebral blood flow during secondary injury. Day 0 presents with hypoperfusion followed over days 1–3 with hyperemia and then the remainder of the first 2 weeks the patient is at risk for vasospasm. Risk of elevated intracranial pressure (ICP) is also most prevalent during this time [11, 12].

Defining the Disease

GCS the Glasgow Coma Scale is a commonly utilized neurologic assessment in trauma patients. It is utilized by nursing, prehospital providers, and physicians to communicate the global neurologic state of the patient.

The components of the GCS are motor, verbal, and eye opening. The scoring is demonstrated in Table 1. Patients can get a minimum score of 3 and a maximum score of 15. Score is made based on best effort, even if unilateral. Traditionally ranges of GCS have been used to grade TBI [13, 14]. However, recent literature proposes that this may not be sufficient [15–21].

Mild – Moderate TBI

Mild and moderate TBI account for at least 80% of traumatic injuries that present to the ED [1, 22, 23]. Mild TBI is GCS 14-15. Moderate TBI is GCS 9-13 [18, 24, 25].

Severe TBI

Severe TBI is diagnosed by GCS < 9 [18, 24–26].

Table 1 Glasgow Coma Scale

Eyes	Verbal	Motor
1 = will not open eyes	1 = Nonverbal	1 = No response
2 = opens to painful stimuli	2 = Incomprehensible sound	2 = Decerebrate Posturing
3 = opens to voice	3 = Inappropriate words	3 = Decorticate Posturing
4 = spontaneously opens eyes	4 = Confused	4 = Withdraws from pain
	5 = Oriented	5 = Localizes Pain
		6 = Follows commands

Signs and Symptoms

The signs and symptoms of TBI vary greatly depending on the severity of the injury. Patients may present with headache, confusion, nausea and vomiting with mild TB. Patients with more severe TBI may have focal neurologic deficits, and decreased level of consciousness. The most severe TBIs will present unresponsive and may even display signs of herniation such as a fixed dilated pupil, Cheyne stokes respirations, and decorticate or decerebrate posturing.

Complications

Given the wide range of severity of TBI, complications too can vary significantly. Short term complications of TBI can include difficulty concentrating, aspiration, neurologic deficits, airway compromise, elevated intracranial pressure, cerebral and cerebellar herniation. Long term complications can include memory loss, short and long term, mood disturbances, cognitive delay, chronic headaches, pituitary dysfunction, sleep dysfunction, permanent neurologic deficits and death [5, 27–30].

Differential Diagnosis

Altered level of consciousness and headache are common presentations of TBI. These two presentations can have a broad spectrum of differential diagnoses .

TCD Findings

Transcranial Doppler allows for in-vivo monitoring of ICP and cerebral perfusion pressure (CPP) in patients with TBI. Often these patients are critically ill and are not optimal candidates to be transported to and from other imaging modalities [12, 31]. TCD

monitors cerebral blood flow via mean blood flow velocity (MFV) and ICP via pulsatility index of the MCA and other vessels. It also allows for monitoring of vasospasm. All of this can be done on serial exams, and is a non-invasive, low cost, low risk, bedside available imaging modality for real time assessment [31].

TCD has been proposed as a useful tool for predicting neurologic outcomes with all grades of TBI, see Table 2 [23, 32–34]. Neurologic outcomes after TBI are dependent on a number of variables including initial injury severity, concomitant injuries, comorbidities, associated organ dysfunction and extent of secondary brain injury [22, 23, 34].

TCD has been shown to be a good screening tool for secondary neurologic deterioration in patients with initial head CT that does not show signs of severe injury [22].

In one study by Bouzat et al. they found that the two most predictive factors for secondary neurologic deterioration (SND) were mean diastolic blood flow velocity of <25 cm/s and pulsatility index >= 1.25 in the MCA [22, 23].

In Mild TBI these findings had a sensitivity of 91%, and a specificity of 80%, 100% NPV, 15.6% PPV for neurologic decline. In moderate TBI the aforementioned measurements had a sensitivity of 67%, specificity of 74%, 94% NPV, and 26% PPV for neurologic decline [23]. These numbers suggest that a normal TCD should be reassuring for likely neurologic stability. An abnormal TCD measurement is more useful in mild TBI for predicting neurologic decline [23].

Another study by Fatima et al. found several measures that were associated with poor neurologic outcomes. These measures

Table 2 Grades of TBI

Grade of TBI	GCS	Altered LOC Duration	Post Trauma Amnesia	Mortality
Mild	13–15	<30 minutes	<24 hours	0.1%
Moderate	9–12	30 minutes-24 hours	1–7 days	10%
Severe	<9	>24 hours	>7 days	40%

include MCA MFV <35 cm/s within 72 hours of head injury, moderate basilar artery vasospasm (MFV >60 cm/s), and severe basilar artery vasospasm (MFV > 85 cm/s) which were all associated with poor neurologic outcomes. In addition, this study also found that MCA PI >= 1.56 was associated with 83% rate of poor outcome whereas PI <=1 was associated with 71% rate of good neurologic outcome at 6 months [12].

TCD also has a role in severe TBI. In the first 24 hours, low CBF (MFV < 40 cm/s) can be a surrogate marker of ischemia and can be acted upon to minimize secondary brain injury. This measurement becomes less reliable after the first 24 hours [35]. Ract et al. evaluated resuscitation of TBI patients guided by TCD. Early TCD to screen severe TBI patients with signs of decreased CPP or increased ICP was beneficial. Patients with these abnormal TCDs were given treatment to increase cerebral blood flow early. In response, they saw that ICP was still higher at the time of pressure monitoring but CPP and jugular venous oxygen saturation were the same as in the normal TCD group. From these findings they concluded that early TCD in those with compromised CPP could possibly decrease in secondary brain injury [36]. In their study Vd < 20 cm/s and PI > 1.4 were the best predictors of decreased CPP.

In severe TBI patients serial TCD in conjunction with ICP monitoring is useful in TBI patients after decompressive hemicraniectomy to monitor for decreased CPP or increase in ICP using FVd, MFV, and PI. Those with more regular monitoring and early detection of these changes had more favorable outcomes at 6 months than the traditional ICP only monitoring group [37].

Severe TBI is often associated with high morbidity and mortality. However, one study showed that 80% of patients with normal TCD measurements can expect good outcomes, those with hypoperfusion have a 90% chance of brain death and 98.6% chance of overall mortality. However, 14% of those with normal TCD expired prior to discharge, 4 from brain death [38]. While this was a single study, one could take away that normal TCDs are reassuring of better outcomes but prognosis should still be guarded as a significant percent of those with initially normal TCDs died.

Table 3 TCD Findings Associated with Poor Neurologic outcome

Vd < 20 cm/s
PI > 1.4
MFV < 40 cm/s @ 24 hours
MCA MFV < 35 cm/s @ 72 hours
Basilar Artery MFV > 60 cm /s
MCA PI > 1.56

Table 3 summarizes the various measurements from the aforementioned studies that are associated with poor neurologic outcomes.

Other Imaging

As mentioned earlier in this chapter just as GCS is not an adequate tool in isolation neither is TCD for diagnosis, management and prognosis of TBI patients. CT, MRI and EEG all have a role in monitoring and guidance of care of these patients.

Computed Tomography (CT)

CT is often the initial imaging modality in the developed world for moderate and severe TBI. It is readily available and more affordable than MRI. However, CT does have some associated risks such as radiation, and typically requires transport out of the critical care unit. According to CDC guidelines, CT imaging is indicated under conditions of: GCS < 15, polytrauma, neurologic deficit, coagulopathy, severe headache, age > 65 years, dangerous mechanism, or signs of basilar skull fracture [39–44].

Magnetic Resonance Imaging (MRI)

MRI has sensitivities approaching 100% for intracranial injury [44]. In addition, MRI unlike CT has no radiation. However, MRI is more costly and has limited availability. In addition MRI takes significantly more time than CT requiring potentially ill patients to be off the critical care unit and unable to receive certain treatments and intensive monitoring [44]. MRI also has better ability to assess the skull base and brainstem. Additionally, MRI has the capability to assess perfusion, diffusion, and proton spectroscopy which further enhance its sensitivity.

Positron Emission Tomography (PET)

PET scan is an even more highly limited imaging modality. It evaluates tissue metabolism. PET can detect non anatomic lesions and areas with potentially reversible insults that were not detected on CT, MRI, or EEG [44].

Electroencephalography (EEG)

EEG is one of the oldest modalities used in monitoring of TBI patients however in the past 2 decades has been surpassed by other modalities. However, new research is looking into the value of EEG in determining prognosis in TBI patients.

Treatment

Treatments for TBI are constantly evolving as the pathophysiology of the secondary injury is being revealed. Several treatments exist with varying levels of evidence. These include decompressive craniotomy/craniectomy, targeted temperature management, steroids, glucose, hypertonic saline, amphetamines, bone marrow transplants, mannitol.

Summary

Traumatic Brain Injury is a prevalent disease in the United States and across the world. Patients with even minor TBI are prone to long term sequelae including but not limited to cognitive delay, emotional disturbances, interpersonal and professional challenges. The more severe TBIs face the aforementioned sequelae in addition to the potential for neurologic deficits. TCD has a significant role in screening TBI patients and aiding in management and prognostication. In these patients TCD is best if used in conjunction with other modalities such as invasive monitoring, physical exam findings, EEG, and neuroimaging.

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