



HEAD TRAUMA

Traumatic brain injury (TBI) represents a relevant public health problem. Most patients with TBI (75-80%) have mild head injuries; the remaining injuries are divided equally between the moderate and severe categories.

Etiology

Various mechanisms may cause TBI, the most common causes include motor vehicle accidents, falls, assaults, sports-related injuries, and penetrating trauma. TBI represents also a frequent injury occurring in sailors.

Pathophysiology

TBI may be divided into 2 categories, primary brain injury and secondary brain injury. Primary brain injury is defined as the initial injury to the brain as a direct result of the trauma. This is the initial structural injury caused by the impact on the brain, and, like other forms of neural injury, patients recover poorly. Secondary brain injury is defined as any subsequent injury to the brain after the initial insult. Secondary brain injury can result from systemic hypotension, hypoxia, elevated intracranial pressure (ICP) or as the biochemical result of a series of physiologic changes initiated by the original trauma. The treatment of head injury is directed at either preventing or minimizing secondary brain injury.

Elevated ICP may result from the initial brain trauma or from secondary injury to the brain. In adults, normal ICP is considered 0-15 mm Hg. Elevations in ICP are deleterious since if severe enough, may result in cerebral ischemia. Severe elevations of ICP are dangerous because, in addition to creating a significant risk for ischemia, uncontrolled ICP may cause herniation of brain structures.

Clinical findings

TBI may be divided into 2 broad categories, closed head injury and penetrating head injury. The clinical presentation of the patient with TBI varies significantly, from an ambulatory patient complaining of a sports-related head injury to the moribund patient arriving via helicopter following a high-speed motor vehicle accident. The Glasgow Coma Scale (GCS) developed by Jennett and Teasdale is used to describe the general level of consciousness of patients with TBI and to define broad categories of head injury. The GCS is divided into 3 categories, eye opening (E), motor response (M), and verbal response (V).

Diagnostic procedures

After the patient has been stabilized and an appropriate neurologic examination has been conducted, the diagnostic evaluation may begin. Patients with TBI do not require any additional blood tests beyond the standard panel of tests obtained in all trauma patients. A urine toxicology screen and an assessment of the blood alcohol level are important for any patient who has an altered level of consciousness because any central nervous system depressant can impair consciousness.

Imaging Studies



- Skull radiographs

Once an important part of the head injury evaluation, skull radiographs have been replaced by CT scans and are rarely used in patients with closed head injury.

- **CT scan**

- A CT scan is the diagnostic study of choice in the evaluation of TBI because it has a rapid acquisition time, is universally available, is easy to interpret, and is reliable.
- The standard CT scan for the evaluation of acute head injury is a noncontrast scan that spans from the base of the occiput to the top of the vertex in 5-mm increments.
- Three data sets are obtained from the primary scan, (1) bone windows, (2) tissue windows, and (3) subdural windows. These different types of exposure are necessary because of the significant difference in exposure necessary to visualize various intracranial structures. The bone windows allow for a detailed survey of the bony anatomy of the skull, and the tissue windows allow for a detailed survey of the brain and its contents. The subdural windows provide better visualization of intracranial hemorrhage, especially those hemorrhages adjacent to the brain (eg, subdural hematomas).
- Skull fractures may be classified as either linear or comminuted fractures. Linear skull fractures are sometimes difficult to visualize on the individual axial images of a CT scan. The scout film of the CT scan, which is the equivalent of a lateral skull x-ray film, often demonstrates linear fractures. The intracranial sutures are easily mistaken for small linear fractures. However, the sutures have characteristic locations in the skull and have a symmetric suture line on the opposite side. Small diploic veins, which traverse the skull, may also be interpreted as fractures. Comminuted fractures are complex fractures with multiple components. Comminuted fractures may be displaced inwardly; this is defined as a depressed skull fracture.
- Extra-axial hematomas include epidural and subdural hematomas.
- Subdural hematomas are located between the dura mater and the brain. Their outer edge is convex, while their inner border is usually irregularly concave. Subdural hematomas are not limited by the intracranial suture lines; this is an important feature that aids in their differentiation from epidural hematomas. Subdural hematomas are usually venous in origin, although some subdural hematomas are caused by arterial injuries. The classic cause of a posttraumatic subdural hematoma is an injury to one of the bridging veins that travel from the cerebral cortex to the dura. As the brain atrophies over time, the bridging veins become more exposed and, as a result, are more easily injured. Occasionally, the distinction between a subdural and an epidural hematoma can be difficult. The size of an extra-axial hematoma is a more important factor than whether the blood is epidural or subdural in location. In addition, a mixed hematoma with both a subdural and an epidural component is not uncommon.
- Intra-axial hematomas are defined as hemorrhages within the brain parenchyma. These hematomas include intraparenchymal hematomas, intraventricular hemorrhages, and subarachnoid hemorrhages. Subarachnoid hemorrhages that occur



because of trauma are typically located over gyri on the convexity of the brain. The subarachnoid hemorrhages that result from a ruptured cerebral aneurysm are usually located in the subarachnoid cisterns at the base of the brain. Cerebral contusions are posttraumatic lesions in the brain that appear as irregular regions, in which high-density changes (ie, blood) and low-density changes (ie, edema) are present. Frequently, 1 of these 2 types of changes predominates within a particular contusion. Contusions are most often caused by the brain gliding over rough surfaces, such as the rough portions of the skull that are present under the frontal and temporal lobes.

- CT scans may be used for classification and for diagnostic purposes.
- Magnetic resonance imaging (MRI)
- MRI has a limited role in the evaluation of acute head injury. Although MRI provides extraordinary anatomic detail, it is not commonly used to evaluate acute head injuries because of its long acquisition times and the difficulty in obtaining MRIs in persons who are critically ill. However, MRI is used in the subacute setting to evaluate patients with unexplained neurologic deficits.
- MRI is superior to CT scan for helping identify diffuse axonal injury (DAI) and small intraparenchymal contusions. .

Other Tests:

- *Initial evaluation*
 - The initial evaluation of patients with TBI involves a thorough systemic trauma evaluation according to the advanced trauma life support (ATLS) guidelines.
 - The evaluation of the spine for potential injury is critically important in patients with TBI because approximately 10% of those with severe head injuries have a concomitant spine injury. Many of these injuries are cervical spine injuries.
- *Neurologic assessment*
 - After sufficient information has been obtained regarding patient history, appropriate physical and neurologic examinations are performed.
 - The neurologic assessment begins with ascertaining the GCS score. This is a screening examination and does not substitute for a thorough neurologic examination.
 - In addition to determining the GCS score, the neurologic assessment of patients with TBI should include the following:
 - Brainstem examination - Pupillary examination, ocular movement examination, corneal reflex, gag reflex
 - Motor examination
 - Sensory examination
 - Reflex examination
 - Many patients with TBI have significant alterations of consciousness and/or pharmaceuticals present that limit the scope of the neurologic examination. When such factors limit the neurologic examination, noting their presence is important.
- *Pupillary examination*
 - A careful pupillary examination is a critical part of the evaluation of patients with TBI, especially in patients with severe injuries. When muscle relaxants have been



administered to a patient, the only aspect of the neurologic examination that may be evaluated is the pupillary examination.

- Several factors can alter the pupillary examination results. Narcotics cause pupillary constriction (meiosis), and medications or drugs that have sympathomimetic properties cause pupillary dilation (mydriasis). These effects are often strong enough to blunt or practically eliminate pupillary responses. Prior eye surgery, such as cataract surgery, can also alter or eliminate pupillary reactivity.
- Ocular movement examination
 - When the patient's level of consciousness is altered significantly, a loss of voluntary eye movements often occurs and abnormalities in ocular movements are frequently present. These abnormalities can provide specific clues to the extent and location of injury.
 - Ocular movements involve the coordination of multiple centers in the brain.
- Oculocephalic testing
 - Oculocephalic testing (doll's eyes) involves observation of eye movements when the head is turned from side to side. This maneuver helps assess the integrity of the horizontal gaze centers.
 - Before performing oculocephalic testing, the status of the cervical spine must be established. If a cervical spine injury has not been excluded reliably, oculocephalic testing should not be performed.
- Oculovestibular testing
 - Oculovestibular testing, also known as cold calorics, is another method for assessment of the integrity of the gaze centers. Oculovestibular testing is performed with the head elevated to 30° from horizontal to bring the horizontal semicircular canal into the vertical position.
 - As the level of consciousness declines, the fast component of nystagmus fades gradually. Thus, in unconscious patients, only the slow phase of nystagmus may be evaluated.
 - A normal oculocephalic response to cold-water calorics (ie, eye deviation toward the side of irrigation) indicates that the injury spares the PPRF, the MLF, and third and sixth cranial nerve nuclei. This means that the level of injury must be rostral to the reticular activating system in the upper brainstem.
- Motor examination
 - After completing the brainstem examination, a motor examination should be performed. A thorough motor or sensory examination is difficult to perform in any patient with an altered level of consciousness.
 - When a patient is not alert enough to cooperate with strength testing, the motor examination is limited to an assessment of asymmetry in the motor examination findings. This may be demonstrated by an asymmetric response to central pain stimulation or a difference in muscle tone between the left and right sides. A finding of significant asymmetry during the motor examination may be indicative of a hemispheric injury and raises the possibility of a mass lesion.
- Sensory examination
 - Performing a useful sensory examination in patients with TBI is often difficult.



- Patients with altered levels of consciousness are unable to cooperate with sensory testing, and findings from a sensory examination are not reliable in patients who are intoxicated or comatose.
- Peripheral reflex examination
 - A peripheral reflex examination can be useful to help identify gross asymmetry in the neurologic examination. This may indicate the presence of a hemispheric mass lesion. **REAT**

Medical treatment

The treatment of head injury may be divided into the treatment of closed head injury and the treatment of penetrating head injury. While significant overlap exists between the treatments of these 2 types of injury, some important differences are discussed. Closed head injury treatment is divided further into the treatment of mild, moderate, and severe head injuries.

Most head injuries are mild head injuries. Most people presenting with mild head injuries will not have any progression of their head injury; however, up to 3% of mild head injuries progress to more serious injuries. Mild head injuries may be separated into low-risk and moderate-risk groups. Patients with mild-to-moderate headaches, dizziness, and nausea are considered to have low-risk injuries. Many of these patients require only minimal observation after they are assessed carefully, and many do not require radiographic evaluation. These patients may be discharged if a reliable individual can monitor them.

Patients who are discharged after mild head injury should be given an instruction sheet for head injury care. The sheet should explain that the person with the head injury should be awakened every 2 hours and assessed neurologically. Caregivers should be instructed to seek medical attention if patients develop severe headaches, persistent nausea and vomiting, seizures, confusion or unusual behavior, or watery discharge from either the nose or the ear.

Patients with mild head injuries typically have concussions. A concussion is defined as physiologic injury to the brain without any evidence of structural alteration. Concussions are graded on a scale of I-V. A grade I concussion is one in which a person is confused temporarily but does not display any memory changes. In a grade II concussion, brief disorientation and anterograde amnesia of less than 5 minutes' duration are present. In a grade III concussion, retrograde amnesia and loss of consciousness for less than 5 minutes are present, in addition to the 2 criteria for a grade II concussion. Grade IV and grade V concussions are similar to a grade III, except that in a grade IV concussion, the duration of loss of consciousness is 5-10 minutes, and in a grade V concussion, the loss of consciousness is longer than 10 minutes.

As many as 30% of patients who experience a concussion develop postconcussive syndrome (PCS). PCS consists of a persistence of any combination of the following after a head injury: headache, nausea, emesis, memory loss, dizziness, diplopia, blurred vision, emotional lability, or sleep disturbances. Fixed neurologic deficits are not part of PCS, and any patient with a fixed deficit requires careful evaluation. PCS usually lasts 2-4 months. Typically, the symptoms peak 4-6 weeks following the injury. On occasion, the symptoms of PCS last for a year or longer. Approximately 20% of adults with PCS will not have returned to full-time work 1 year after the initial injury, and some are disabled permanently by PCS. PCS tends to be more severe in children than in adults. When PCS is severe or persistent, a multidisciplinary approach to treatment may be necessary. This includes social services, mental health services, occupational therapy, and pharmaceutical therapy.



After a mild head injury, those displaying persistent emesis, severe headache, anterograde amnesia, loss of consciousness, or signs of intoxication by drugs or alcohol are considered to have a moderate-risk head injury. These patients should be evaluated with a head CT scan. Patients with moderate-risk mild head injuries can be discharged if their CT scan findings reveal no pathology, their intoxication is cleared, and they have been observed for at least 8 hours.

The treatment of moderate and severe head injuries begins with initial cardiopulmonary stabilization" by ATLS guidelines. The initial resuscitation of a patient with a head injury is of critical importance to prevent hypoxia and hypotension. Other pharmacological and non pharmacological treatments will require specialist medical advice.

The treatment of penetrating brain injuries involves 2 main aspects. The first is the treatment of the TBI caused by a penetrating object. Penetrating brain injuries, especially from high-velocity missiles, frequently result in severe ICP elevations. This aspect of penetrating brain injury treatment is identical to the treatment of closed head injuries.

The second aspect of penetrating head injury treatment involves debridement and removal of the penetrating objects. Penetrating injuries require careful debridement because these wounds are frequently dirty. When objects penetrate the brain, they introduce pathogens into the brain from the scalp surface and from the surface of the penetrating object.

Focal neurologic deficits

Focal neurologic deficits are quite common following TBI. Cranial nerves are affected often because of their anatomic location at the base of the brain. When the brain shifts within the skull as it undergoes either acceleration or deceleration forces, significant force is often placed on the entire brain and the cranial nerves.

Anosmia caused by traumatic injury to the first cranial nerve occurs in 2-38% of patients with TBI. It is more common in those with frontal fractures and in those with posttraumatic rhinorrhea. Posttraumatic anosmia improves slowly, and as many as one third of patients do not show any improvement in olfaction.

Injuries to the fourth cranial nerve, the trochlear nerve, are also quite common. This nerve is often injured in patients with head trauma because it has the longest intracranial course of the cranial nerves. Injury to the trochlear nerve causes a positional diplopia, in which those affected experience diplopia when they look down and toward the eye in which the trochlear nerve is injured. As a result, to compensate, the head is tilted up and away from the side of the injury. Trochlear nerve injuries resolve fully in approximately two thirds of those with unilateral injury and in one fourth of those with bilateral injuries.

Facial nerve injuries often occur with head injuries in which the temporal bone is fractured. From 10-30% of persons with longitudinal fractures of the temporal bone and 30-50% of those with transverse fractures of the temporal bone have either acute or delayed facial nerve injury. Immediate facial nerve injury suggests direct injury to the nerve, while delayed injury suggests progressive edema within the nerve. In severely injured patients, a delay in the diagnosis of facial nerve injuries occurs frequently because facial nerve function is difficult to assess in obtunded patients.

Cochlear nerve injury (cranial nerve VIII) is also a common occurrence in patients with head injury, especially in patients with temporal bone fractures. In addition, vestibular disorders, including vertigo, dizziness, and tinnitus, are extremely common in patients with head injuries.



Hydrocephalus

Hydrocephalus is a common late complication of TBI. Posttraumatic hydrocephalus may present as either ventriculomegaly with increased ICP or as normal pressure hydrocephalus. In patients with increased ICP secondary to posttraumatic hydrocephalus, the typical signs of hydrocephalus are often observed and include headaches, visual disturbances, nausea/vomiting, and alterations in the level of consciousness. Normal pressure hydrocephalus usually manifests as memory problems, gait ataxia, and urinary incontinence.

Seizures

Posttraumatic seizures are a frequent complication of TBI and are divided into 3 categories. Early seizures occur within 24 hours of the initial injury, intermediate seizures occur 1-7 days following injury, and late seizures occur more than 7 days after the initial injury. Posttraumatic seizures are very common in those with a penetrating cerebral injury, and late seizures occur in as many as half of these patients.

Cerebrospinal fluid fistulae

Cerebrospinal fluid fistulae, either in the form of rhinorrhea or otorrhea, may occur in as many as 5-10% of patients with TBI. They may present either immediately or in a delayed fashion and are more frequent in patients with basilar skull fractures. Approximately 80% of acute cases of CSF rhinorrhea resolve spontaneously within 1 week. A 17% risk of meningitis exists when CSF rhinorrhea is present. Prophylactic antibiotics have not been demonstrated to decrease this meningitis risk, although very few studies have examined this issue. More than 95% of acute episodes of CSF otorrhea resolve spontaneously within 1 week, and CSF otorrhea is complicated by meningitis in fewer than 4% of cases.

Vascular injuries

Vascular injuries are uncommon sequelae of TBIs. Arterial injuries that may occur following head trauma include arterial transections, thromboembolic phenomena, posttraumatic aneurysms, dissections, and carotid-cavernous fistulae (CCF).

Arterial occlusions secondary to transections or thromboembolism following closed head injuries are uncommon occurrences.

Posttraumatic intracranial aneurysms, which are also rare, differ from congenital aneurysms because the posttraumatic aneurysms tend to be located distally, as opposed to the congenital aneurysms, which are typically proximal in location.

Arterial dissections are more common than the aforementioned arterial injuries and should be considered if significant injury has occurred to the petrous portion of the temporal bone, through which the carotid artery passes, or when an unexplained neurologic deficit is present. A cerebral angiogram is often necessary to help exclude arterial injury in these cases.

Infections

Intracranial infections are another potential complication of TBI. In uncomplicated closed head injury, infection is uncommon. When basilar skull fractures and/or CSF fistulae are present, the risk of infection is increased. In addition, if a patient has had a ventriculostomy for ICP monitoring, the



risk of infection is also increased, for either a ventriculitis or meningitis. Other intracranial infections such as subdural or epidural empyema and intraparenchymal abscesses are rare following closed head injury. As one would expect, the incidence of infection in penetrating cerebral injuries and open depressed skull fractures increases.

Brain death

Brain death can result from either massive initial injury or as the result of prolonged severe elevations of ICP. Brain death is defined as the absence of brain function. A lack of any neurologic response is not sufficient to establish brain death, and confirmatory testing must be performed.

Many methods exist for evaluating the outcome of TBI. A simple and commonly used method is the Glasgow outcome scale. This divides outcome into 5 categories, as follows: (1) good, (2) moderate disability, (3) severe disability, (4) vegetative, and (5) dead. The scale can be divided further into good outcomes (eg, good plus moderate disability) and poor outcomes (eg, severe disability, vegetative, dead).

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