

Penetrating Traumatic Brain Injury: A Review of Current Evaluation and Management Concepts

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Abstract

Penetrating traumatic brain injury (pTBI) remains one of the most devastating and lethal forms of trauma. Prognosis is generally poor and, for those who survive long enough to make it to the hospital, the management of penetrating brain injury presents complex challenges to medical and surgical providers in the civilian sector. Recent experiences in Operation Iraqi Freedom and Operation Enduring Freedom have provided opportunities to study and refine the surgical and medical management of pTBIs that may impact civilian evaluation and management of similar traumas. These experiences demonstrated that aggressive pre-hospital and emergency department resuscitation, followed by immediate surgical management and post-operative intensive care to monitor for and intervene on surgical and medical complications, could significantly improve patient outcomes. We begin with a brief case vignette that will introduce a comprehensive discussion on the epidemiology, pathophysiology, evolution of current surgical and medical therapies, complications, and prognostic indicators that may improve outcomes in these challenging cases.

Keywords: Traumatic brain injury; Neurosurgery

Introduction

Case Review

A 31 year-old female was the victim of a gunshot wound to the head from a 9mm caliber handgun sustained during a home invasion. A 2 cm entry wound was identified in the right frontotemporal region with no exit wound; cerebral tissue was noted to be protruding through the entry wound. The patient was intubated and cervical spine immobilization was implemented. She was unresponsive and non-verbal with sluggishly reactive pupils at 5 mm bilaterally. She had intact corneal and gag reflexes and withdrawal to pain on her left side. Her initial Glasgow Coma Score was recorded as 6 on scene and 3T in the emergency department (ED) following intubation and sedation provided during transportation. A CT scan of the head demonstrated multiple metallic fragments in the scalp tissue overlying the entry wound as well as within the right frontal lobe extending into the frontal horn of the right ventricle. Right intraventricular extension of the hematoma was present. Additional metallic and bony fragments were noted in the right parietal lobe and the region adjacent to the right Sylvian fissure. Extensive intraparenchymal hematoma was noted in the right frontal lobe and extending to the right parietal region measuring 4.1 x 2.0 cm. A 3 mm subdural hematoma was observed overlying the right cerebral hemisphere with subarachnoid hemorrhage within the right Sylvian fissure and the sulci of the right frontal lobe. There was evidence of diffuse sulcal effacement with a higher degree of focal edema in the right frontal lobe. The right lateral ventricle was severely narrowed and there was 9mm of right to left midline shift with narrowed basilar cisterns and evidence of early uncal herniation (Figure 1). Mannitol 100g intravenously (IV), levetiracetam 1000mg IV, and ceftriaxone 2g IV were administered in the ED.

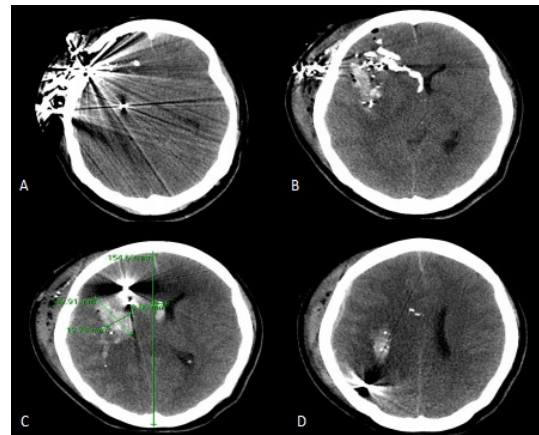


Figure 1: Axial CT brain showing the bullet entry wound in the right frontotemporal region; the bullet did not exit the intracranial cavity (A). Several bullet fragments are lodged in the tissue of the right frontal lobe. At the level of the lateral ventricles there is evidence of bony fragments in the overlying scalp tissue and right frontal lobe extending into the right lateral ventricle (B). A large intraparenchymal hematoma is present in the right frontal lobe adjacent to additional bullet and bone fragments with approximately 9mm of midline shift (C). Additional bullet fragments are appreciated in the right parietal lobe with a small subdural hematoma overlying the right cerebral hemisphere (D). Diffuse sulcal effacement is appreciated in all images.

Neurosurgery was consulted and the patient was taken to the operating room for emergent decompressive hemicraniectomy.

An intraparenchymal monitor was placed intraoperatively due to cerebral edema and elevated intracranial pressures and a subgaleal drain was placed to reduce blood accumulation in the subgaleal space post-operatively. She was taken to the neurointensive care unit where her post-operative course was initially complicated by elevated intracranial pressures.

These stabilized by post-operative day 3 allowing for the removal of the ICP monitor and subgaleal drain, but digital subtraction angiography on post-operative day 4 noted a pseudoaneurysm involving the M4 branch of the right middle cerebral artery that required embolization. By this time, the patient was following commands and communicating via hand signals. Extubation was attempted, but she required re-intubation due to inability to clear secretions that eventually led to the placement of a tracheostomy and percutaneous endoscopic gastrostomy. Hyponatremia manifested on post-operative day 6 that continued to fluctuate through post-operative day 10. She was continually reassessed by physical, occupational, and speech therapy throughout her hospitalization with noted functional improvement. After successful decannulation on post-operative day 20, she was transferred to inpatient rehab in stable condition.

Epidemiology

Penetrating traumatic brain injury (pTBI) is the most lethal form of traumatic head injury. Approximately 70-90% of these victims die before arriving at the hospital, and 50% of those who survive to reach the hospital die during resuscitation attempts in the ED [1-3]. Of the 333,169 US military TBIs recorded between 2000-2015, 4,904 were classified as pTBI [4]. Approximately 32,000-35,000 civilian deaths are attributed to penetrating brain injury each year, with firearms-related injuries being the leading cause of mortality in this group [2,5,6]. Less than 20% of civilians who reach a trauma center will undergo a neurosurgical procedure [3]. Approximately half of pTBIs involving firearms are suicides [7]; these injuries have a higher mortality due to the close ranges at which the firearm is discharged [1]. In 2010, total healthcare costs attributed to TBI were reported by the CDC as \$76.5 billion with severe TBI accounting for 90% of the cost [8]. Although direct comparison of morbidity and mortality in military vs. civilian pTBIs is complicated, the recent data described below suggest both higher rates of neurosurgical intervention and improvement in survival with the early and aggressive resuscitation implemented by the military [2].

Mechanisms of Injury

Traumatic brain injury is the result of energy being transferred from an object to the human skull and underlying brain. The penetrating object has kinetic energy that is proportional to the projectile equal to the mass times the square of its velocity ($E_k=1/2mv^2$) [5]. Most non-bullet penetrating objects, such as nails or knives, impart less damage to the skull and brain because they have less kinetic energy to transfer on impact. Bullets tend to have less mass, but travel at higher velocities, though bullet velocities can vary based on a number of factors, such as the muzzle velocity, travel through the air, and travel through the impacted target. Modern firearm projectile velocities can range from 200m/s in handguns to more than 1000 m/s in some rifles [9,10]. High velocity bullets fired from rifles have the potential to do more damage than bullets fired from handguns, though some large caliber handgun rounds are of sufficient mass to make up for their lower velocities with regard to kinetic energy. Other factors to consider include the distance

from which the round was fired, the yaw, or tumbling that occurs when the bullet impacts soft tissue, and whether the bullet design causes it to deform or fragment on impact. Bullets fired from closer ranges often cause more damage than when the same bullet is fired from a longer distance. Non-deforming projectiles have a tendency to yaw inside tissue, which increases penetration and results in a moderate wound cavity size, while deforming rounds have more superficial penetrations, but form larger wound cavities [9-12]. Figure 2 demonstrates some of these ballistic features in several different types of commonly used modern ammunition.

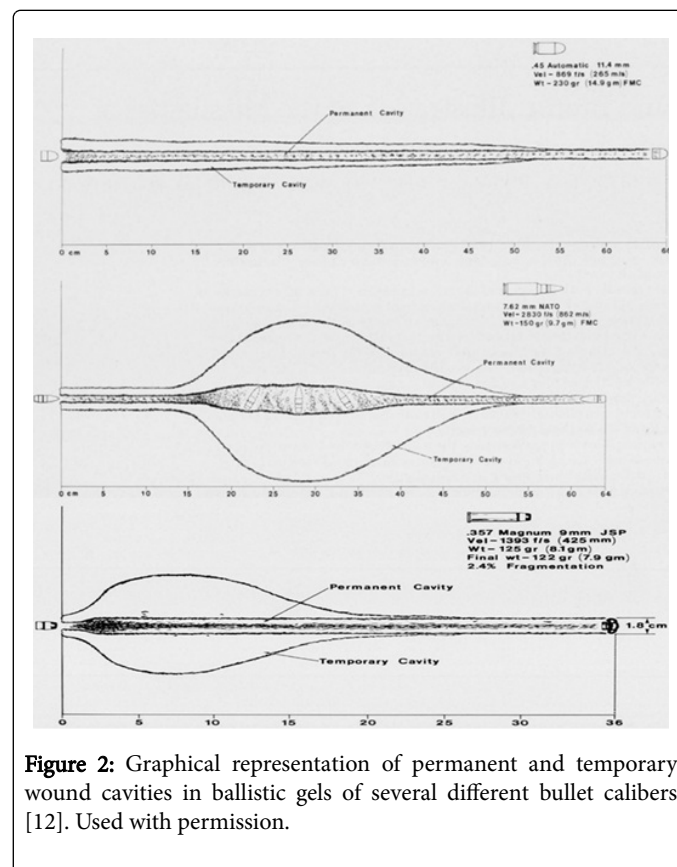


Figure 2: Graphical representation of permanent and temporary wound cavities in ballistic gels of several different bullet calibers [12]. Used with permission.

Wounds resulting from pTBI can be classified into five categories: tangential, penetrating, perforating, ricochet, and careening. Classifying the type of pTBI can assist providers in surgical planning, in anticipating complications, and with prognosis. Tangential wounds are projectile injuries that do not penetrate through the skull. This occurs when the projectile impacts the head at an angle and results in superficial damage to the scalp. While often associated with the best prognosis, tangential injuries can cause underlying injury to the skull, meninges, and cortex, including skull fractures, cerebrospinal fluid (CSF) leaks, epidural and subdural hematomas, and cortical contusions. Careening is an unusual injury pattern where the projectile penetrates the skull, but travels along the periphery of the cortex without penetrating brain parenchyma. This type of injury has a risk of venous sinus injury and, therefore, subdural hematomas or intracranial hemorrhages (ICH). Penetrating injuries are those where the projectiles penetrate the skull and brain parenchyma and remain there. Perforating injuries, the most devastating of all pTBIs, which are usually the result of high-velocity projectiles or those fired at close range, penetrate the skull and then exit at a site distal from the entry point. Injuries associated with penetrating and perforating injuries

include cerebral contusions, hematomas, CSF leaks, pseudoaneurysms and arteriovenous fistulas. Projectiles may penetrate the skull and brain and ricochet off the inner aspect of the skull. This occurs most often with low-velocity projectiles and creates a new wound tract in otherwise uninjured brain tissue [9,13]. Figure 3 provides an illustrated depiction of the various types of pTBI.

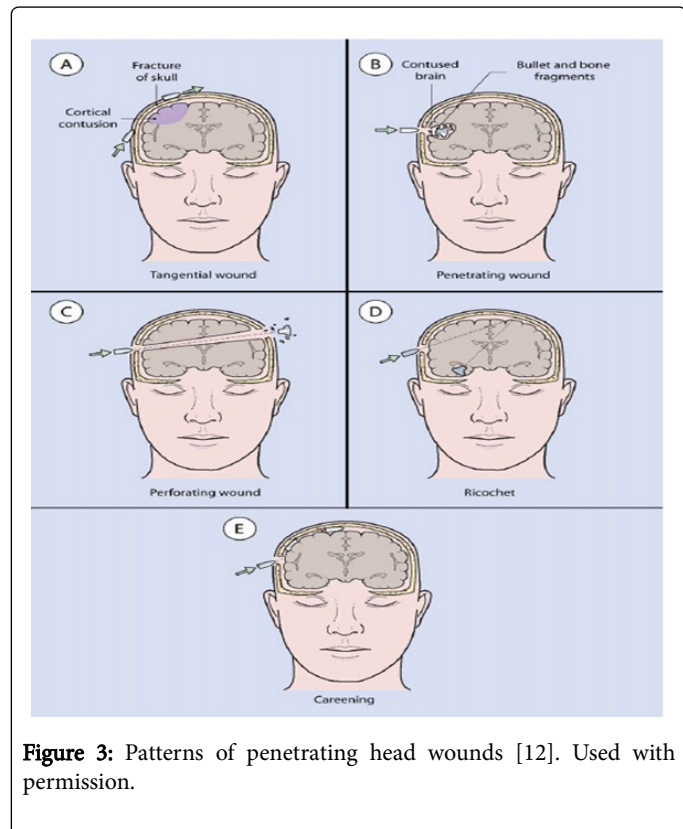


Figure 3: Patterns of penetrating head wounds [12]. Used with permission.

Pathophysiology

Immediate intracranial injury occurs as the result of neuronal and vascular destruction caused by the projectile traveling through intracranial tissues. Once the projectile strikes the head and transfers its kinetic energy to extra and intracranial tissues, destruction occurs in tissue both in the projectile's path as well as in distant tissues outside the projectile's trajectory. Permanent cavitation occurs in tissues directly in the projectile's path, but sonic waves followed by pressure waves of as much as 30 atmospheres produce temporary cavitation [7]. Expansion and retraction of the temporary cavities cause distant punctate hemorrhages and neuronal membrane disruption. The result is a rapid rise in intracranial pressure (ICP) as hematomas enlarge, and cerebral edema increases as early as 30 minutes after the initial injury [13,14]. Concurrently, cerebral perfusion pressure decreases and infarction can follow. If severe, herniation can occur.

The exact pathophysiology of secondary tissue injury associated with pTBI is poorly understood and differs from that of closed-head injuries. Gajavelli et al. recently described reduced regional cerebral oxygen and consumption and concurrent global glucose uptake in animal models. Referred to as the penetrating ballistic-like brain injury (PBBi) model, they noted that lesion core and peri-lesional areas showed similar metabolic impairment, but with less severe metabolic impairment and less neurodegeneration occurring in the latter.

Coupled with the knowledge that mitochondrial damage in the cavitation zone results in the release of various neuro-inflammatory mediators that cause tissue necrosis and neuronal apoptosis in distant tissues, this information provides evidence that peri-lesional regions should be amenable to acute surgical and medical therapies and yet-to-be identified neuroprotectants [15] (Figure 4).

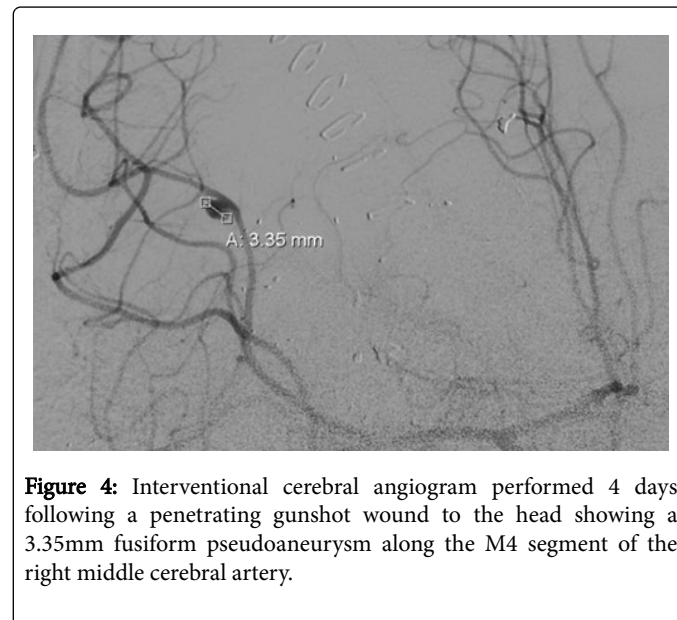


Figure 4: Interventional cerebral angiogram performed 4 days following a penetrating gunshot wound to the head showing a 3.35mm fusiform pseudoaneurysm along the M4 segment of the right middle cerebral artery.

In contrast to non-penetrating TBIs, hemorrhagic shock and cardiac arrest can occur secondary to blood loss. The scalp's risk of vascular supply and hemorrhage from meningeal vessels, parenchymal vessels, and venous sinuses can result in death from exsanguination [16]. Acute traumatic coagulopathy due to release of tissue thromboplastin from the brain resulting in diffuse intravascular coagulation (DIC) may also contribute, particularly in the setting of polytrauma, but also in isolated head injuries. Early identification of related bleeding complications and emergent resuscitation is an important factor in decreasing mortality in these patients [1].

Pre-hospital Care

Pre-hospital management has developed an increasingly important role in the care of pTBI patients and has the potential to significantly impact outcomes. Pre-hospital care is focused on minimizing secondary injury and delivering the patient to a trauma center alive. This is achieved through effective airway maintenance and optimizing oxygenation, ventilation, and cerebral perfusion. General measures to achieve this include elevating the patient's head to 30 degrees and maintaining the head in a midline position. Systolic blood pressures <90 mmHg have been associated with poor outcomes in traumatic brain injury patients, and an SBP >90mmHg should be targeted and maintained. Additionally, oxygen saturation >90%, a PCO₂ between 35-40 mmHg (if capnography is available) are common pre-hospital and resuscitation goals [17].

Historically, pre-hospital management has focused on rapid scene clearance and transport to definitive care. However, combat experiences in Iraq and Afghanistan have shifted the focus toward emphasis on and early management of hypotension, hypoxia, hypocarbia, and/or hypercarbia. The military instituted guidelines for the pre-hospital management of combat-related TBI in 2005, and

implementation of these practices has been associated with improved outcomes [17,18]. A compilation by the Joint Theater Trauma Registry compared 604 combat-related TBIs to similar civilian TBIs in the American College of Surgeons National Trauma Databank and showed improved mortality among military penetrating TBI casualties as compared to civilian pTBIs (5.6% vs. 47.9%, $p < 0.001$). The authors concluded that the reduced mortality was due in part to a stronger emphasis on prevention of secondary brain injury in the pre-hospital setting. This conclusion appears to be supported by a 2014 review by Chowdhury et al. in which the authors cite a study showing that air transportation of severe TBI patients, despite having longer pre-hospital times when compared to ground transport (76.1 min vs. 43.5 min), had no significant impact on mortality or neurologic outcome at 6 months. Additional analysis determined that patients transported by air crews had higher rates of intubation and received more IV fluids than those transported by ground. Likewise, this group concluded that emphasis on pre-hospital management of hypoxemia and hypotension, the two strongest predictors of mortality, resulted in better survival and long-term outcomes for the patients [19].

Emergency Department Care

Aggressive resuscitation following pTBI has been associated with improved survival [2]. Furthermore, it is recommended that aggressive therapy be continued through the resuscitation phase, even in patients with initially low GCS scores, as patients who may benefit from hyperosmolar therapy and surgery may be overlooked based on the initial poor neurological exam upon presentation. Treatment in the emergency department should include correction of hypotension and hypoxia, airway maintenance to include placement of a surgical airway if there is co-existing oromaxillofacial trauma, control of any associated hemorrhaging (i.e. packing facial wounds), hyperosmolar therapy with mannitol or hypertonic saline, correction of traumatic coagulopathy, placement of a cervical immobilization device, an urgent CT scan of the head and neck, and tetanus and antibiotic prophylaxis. Plain films are not necessary if a CT is obtained. Seizure prophylaxis is typically started in the emergency room. Excessive crystalloid should be avoided, and colloid is contraindicated given the association with elevated ICPs [20]. Steroids, including recent trials evaluating the use of progesterone in severe TBI, have either shown no benefit or increased risk of death and should also be avoided [21,22]. Blood products should be available for transfusion, as well as cryoprecipitate, prothrombin complex concentrates and, in rare cases, recombinant Factor VIIa to help control bleeding [1,2,23]. In recent military conflicts, Factor VIIa was administered in the helicopter along with hypertonic saline (Personal communication G.G). Tranexamic acid is currently being evaluated for bleeding associated with TBI in the CRASH-3 trial [24]. Following the initial resuscitation, it is also important to ensure that a detailed secondary assessment is completed. The presence of powder burns on the scalp indicates that the projectile was fired from a close range [13]. Scalp injuries may be obscured by hair, and both entrance and exit wounds may have been missed, particularly in the retromastoid/posterior auricular, occipital and suboccipital areas.

Neurosurgery should be emergently consulted for evaluation in the emergency room and surgical management. An intracranial monitoring device, either parenchymal or intraventricular, would often be placed, although very little data exists on the prognostic role of ICP monitoring in civilian pTBIs. While there is sufficient data to support ICP monitoring for prognosis in severe closed head injury, the only evidence of its use in pTBI comes from military settings. Nevertheless,

elevated ICPs are believed to be predictors of poor outcome based on the work of Nagib et al., and Crockard, both of whom demonstrated that elevated ICPs were associated with higher mortalities in pTBI patients [25,26].

Surgical Management

The surgical treatment of penetrating brain injury has evolved significantly over the past century. Prior to 1889, pTBI patients did not typically undergo surgery due to ineffective hemostasis and poor post-operative infection control [27]. Dr. Harvey Cushing was the first to develop a formal approach to the management of pTBI, and advocated complete removal of metallic and bone fragments, as well as craniectomies to relieve ICP. Radical debridement continued to be standard throughout World Wars I and II, the Korean War, and the Vietnam War [9,27]. In the 1980s during the Israeli-Lebanon conflict, however, a shift was made toward conservative debridement in an effort to preserve as much cerebral tissue as possible. The results, according to Brandvold et al., were similar with regard to acute and chronic outcomes as compared to soldiers who underwent radical debridement in the Korean and Vietnam conflicts [28]. The most recent conflicts in Afghanistan and Iraq have resulted in further refinements to surgical management with a trend toward early decompression with conservative debridement and duraplasty. This approach appears to yield improved survivability not seen in prior conflicts. [3,9,29-32].

A challenging aspect to the surgical management of pTBI is the selection of appropriate surgical candidates. There is extensive literature that has attempted to identify which patients may benefit from surgery. Poor prognostic indicators have previously been identified as old age, low admission GCS, abnormal pupil reactivity, bi-hemispheric involvement, path of the projectile, and loss of the basal cisterns on imaging [1,3,9]. A GCS of 3-5 and/or a projectile path crossing the midline at the level of the corpus callosum, through the bilateral thalami, basal ganglia posterior fossa/brainstem or through an area 4cm above the dorsum sellae containing the vessels of the Circle of Willis known as the "zona fatalis" has historically resulted in the withholding of surgical care [1,9,33]. Lateral penetrating injuries have worse outcomes when compared with antero-posterior injuries [9]. Another common indicator, the "tram track sign" or a hypodense wound track with hyperdense blood on either side has been associated with poor outcomes [1]. Tram track signs are more common with low caliber projectiles. With modern surgical and intensive care management, however, some series have shown markedly improved survival rates even in those with the low post-resuscitation GCS scores [30]. As a result, a GCS < 5 is no longer an absolute contraindication to surgery and some experts recommend urgent craniotomy in the setting of an intracranial mass lesion [1].

In those patients where surgery is deemed appropriate, intervention should be undertaken as soon as the patient is stabilized and preferably within 1 hour of arrival. When performed more than 12 hours after the initial injury, there is an increased risk for infectious complications. Small, superficial wounds without significant intracranial involvement such as may be seen with grazing injuries, quaternary blast injuries, and failed suicide attempts can be managed with local wound care and closure. For more serious injuries, acute decompression and hemorrhage control is the initial goal of surgical intervention. Nonviable scalp, skull, or dura must be identified and requires extensive debridement followed by primary closure or watertight grafting. Necrotic brain tissue should likewise be debrided followed by

the removal of any easily accessible debris. Intracranial hematomas causing mass effect should be evacuated [7,9,32,34,35]. No data exists to support the advantages of craniectomy over craniotomy with both procedures having similar morbidity and mortality, though recent data from the military suggesting improved mortality with early decompression has involved the use of craniectomy [29-31,34]. Another consideration in the combat zone is the long transport time to evacuate the patient to the next level of care. Removal of retained metallic, bony, and other debris fragments is no longer aggressively pursued unless there is evidence of migration, contact with the CSF in a cistern or ventricle, or positioning near a vascular structure [9,34]. Although associated with a lower incidence of post-traumatic epilepsy, routine exploration for removal of metallic and bony debris is no longer pursued given the association with worse functional outcomes and higher mortality [34]. The placement of drains, either subgaleal, epidural or both, are common and have shown in at least one analysis to result in a trend toward fewer post-operative complications (seizures, infections, subdural or subgaleal fluid collections, hematomas) when compared to those who did not have a drain after craniectomy [36].

Complications

There is an extensive list of delayed complications that can arise from pTBI. Vascular complications are among the most common and most devastating and can include cerebral vasospasm when subarachnoid hemorrhage is present, and traumatic intracranial aneurysm formation. Intracranial pseudoaneurysm formation occurs at a rate of 2-33% following pTBI; early identification and repair of traumatic aneurysms portends a better prognosis than intervention following rupture. Subarachnoid hemorrhage can result in delayed cerebral vasospasm and delayed cerebral ischemia. The highest incidence of vasospasm occurs between days 5-11, but can occur anywhere between 3-21 days after injury. Daily transcranial Doppler ultrasound can help screen for vasospasm, but if there is high suspicion in the setting of sudden clinical deterioration, the patient should undergo urgent catheter angiogram. Other potential complications include arterial and venous sinus occlusions and arteriovenous fistulas [7,9]. Identification of vascular complications can be achieved by early catheter angiogram following injury and should be performed in any patient with penetrating injury involving the orbitofrontal or pterional regions, known cerebral vessel injury, penetrating injury related to a blast, TCD evidence of vasospasm, or unexplained drops in brain-tissue oxygen tension [37].

Risk of infectious complications increases following the first week post-injury with recent post-surgical infectious complications occurring at a rate of 5-23% [7]. Despite the heat associated with the projectile, wound contamination can occur via rapid expansion and contraction of temporary wound cavities that suck in debris. Most of the contaminating organisms are skin flora such as *Staphylococcus epidermidis*, but *Staphylococcus aureus* and Gram-negative bacilli are also common pathogens. Effective debridement can reduce the risk of infection from such contaminants. Other risk factors for infection following pTBI include air sinus involvement, ventricular involvement, low GCS score, severe tissue involvement, and CSF leakage [7,9,12]. There is no consensus in the literature regarding prophylactic antibiotics following pTBI. Bayson et al. found the evidence for prophylactic antibiotics to be weak and anecdotal [38]. Lin et al. reported antibiotic coverage with vancomycin, gentamycin, and metronidazole for 2-3 days [39], while recent US military guidelines recommend cefazolin for 5-7 days and also note high rates of multi-

drug resistant organisms such as *Acinetobacter* [40]. Prophylactic antibiotic use should be determined on an individual basis considering the patient's overall risk for infection and an infectious disease consult should be strongly considered. In general, broad-spectrum antibiotics that cover *Staphylococci* and Gram-negative bacilli are appropriate. If dirt, debris or clothing contaminates the wound, expansion to anaerobic coverage with metronidazole is recommended [1].

CSF leaks are a complication that arise from laceration of the dura and are most common with orbitofrontal and transtemporal patterns of injury where basilar skull fractures or orbitonasal injuries are most prevalent. CSF is contaminated as the projectile path pulls skin, bone, clothing, and other contaminants into the wound. Leaks lead to contaminated CSF draining from the nose, auditory canal, or the entrance or exit wounds and have a markedly increased risk of meningitis [41]. Cerebritis, ventriculitis, and abscess formation are also potential infectious complications related to CSF leaks. CSF leaks must be sealed with direct closure of the dura or utilizing grafting materials. Repair of associated fractures of the skull base may also decrease the incidence of CSF leak. Distant CSF leaks may require CSF diversion via ventriculostomy or lumbar drain, though these have a substantial association with subsequent *Acinetobacter* infections [42].

Post-traumatic epilepsy is a common complication and can occur as early or late manifestations related to pTBI. Early seizures, occurring within the first week after injury, are often generalized and occur in 2-8.9% of pTBI patients. Late-onset seizures, occurring up to 15 years following the injury, can present as focal seizures or focal seizures with secondary generalization in as many as 50% of patients. Early seizures following TBI can be prevented with anti-convulsant therapy that is typically instituted during the first week following pTBI, though there is no evidence that early anti-convulsant therapy reduces the incidence of late post-traumatic seizures, nor does it affect functional outcome. Risk factors associated with seizures in pTBI include injury severity, infection, retained projectile or bone fragments, hematomas, aphasia, hemiparesis, visual field defects, and cognitive dysfunction [7,9,12,43-44].

Other complications have been described with pTBI and may depend on the pattern of injury. Traumatic optic neuropathy, for example, has been reported in 0.7-2.5% of blunt or penetrating TBI cases [45]. The oculomotor, trochlear, and abducens nerves are also at risk when there is penetrating injury involving the face, and transtemporal injuries can lead to facial or acoustic nerve injury [9,12,40]. Lead or copper toxicity from retained bullet fragments is a rare complication, and levels should be monitored if large fragments are retained [1].

Post-Operative Medical Management

Post-operative management of pTBI patients is critical to improving survivability and functional outcomes and requires a multidisciplinary approach. For approximately two weeks following the onset of injury, close monitoring of intracranial dynamics allows secondary injury to be identified and for prompt intervention when this occurs. Intracranial hypertension is common, and may be associated with, decreased cerebral perfusion pressures (CPP), cerebral ischemia, seizures, vasospasm, arteriovenous fistula formation, or traumatic aneurysm rupture as a direct result of pTBI. Maintaining ICP <20-25mmHg and CPP >60mmHg using general measures (head mid-line, head of bed elevated to 30 degrees, control of pain and temperature) hyperosmotic therapy, sedation, neuromuscular blockade, and induced hypothermia may improve outcomes by

limiting secondary injury. TCD can help monitor cerebral blood flow as well as assess for evidence of developing cerebral vasospasm in the setting of traumatic subarachnoid hemorrhage [12].

Patients who have sustained pTBI are at higher risk for the development of non-neurologic complications as well. For example, pTBI patients are at high risk for Acute Respiratory Distress Syndrome (ARDS). This condition is also associated with the use of fluids and vasopressors used to maintain an adequate CPP. When this occurs, patients may require extracorporeal membrane oxygenation or high frequency oscillations since hypercapnea and prone positioning can worsen secondary brain injury. Patients also require observation of their cardiovascular status (to include cardiac rate, rhythm, and blood pressure), monitoring for the development of diffuse intravascular coagulopathy, infection, kidney injury, and skin breakdown. A number of endocrine abnormalities may present in pTBI to include hyperglycemia, diabetes insipidus, cerebral salt wasting, or syndrome of inappropriate diuretic hormone. Frequent monitoring of blood sugars, urine output, electrolytes, osmolality, and specific gravity will help identify these disorders promptly. Ongoing assessments of dietary needs and nutrition should be started early via nasogastric or orogastric tube if possible, or via percutaneous endoscopic gastrostomy tube if facial injuries preclude the use of the former methods. Stress ulcer prophylaxis and a bowel regimen should be instituted on admission to the ICU. Critically ill patients are at high risk for deep venous thrombosis (DVT), and prophylaxis with pneumatic compression devices and/or heparinoids should be started as soon as it is safe to do so. Ancillary services, such as physical therapy, occupational therapy, and speech pathology should be ordered as soon as possible to help mitigate the many risks that accompany immobility in these patients [12].

Prognosis

Clinical factors associated with poor outcomes include low post-resuscitation GCS, older age, large and unreactive pupils or pupil asymmetry, hypoxia, and hypotension (SBP<90 mmHg). Other factors include time to reach a neurosurgeon and weapon ballistics, including caliber and close proximity to the projectile's source. GCS is generally considered to be the best single predictor of good or bad outcome following pTBI [1-3,5,9,18].

Survival in civilian gunshot pTBIs has typically been linked to GCS upon presentation. Rosenfeld et al. reported survival at 0-8.1%, 35.6% and 90.5% for GCS scores of 3-5, 6-8 and 9-15 respectively [46]. Aarabi et al. reported similar findings in the GCS 3-5 category in their analysis with survival rates of 5%. However, their 6-8 and 9-15 categories were similar, with survival rates of 83.3% and 84.6% respectively [3]. The improvement in the GCS 6-8 category can be attributed to improved access to neurosurgeons as well as more aggressive resuscitation and post-operative care. There appears to be improved mortality in the GCS 3-5 category as well, with some recent military studies showing 32-38% of patients living independently at 2 years following aggressive intervention [9,30]. The overall trend supports aggressive pre-hospital and ED resuscitation with rapid surgical intervention for optimizing outcomes in pTBI patients to include those who have clinical and imaging predictors of poor outcome.

Conclusion

The patient in the introductory vignette continued to improve following discharge from the hospital to acute inpatient rehab. After

approximately two months of intensive rehabilitation she was discharged to home and requiring supervision for wheelchair propulsion and contact guard assistance with short distance transfers, showering, toileting, and stand sink grooming. She advanced to a dysphagia level 3 diet and her gastrostomy tube was removed. She was recently seen in the outpatient neurosurgery clinic to discuss a custom-made cranioplasty where she remained hemiparetic, but was ambulating without assistance.

Penetrating traumatic brain injury remains a devastating form of trauma associated with high mortality. For those that do survive, there is a high probability for permanent neurologic dysfunction. Nevertheless, experience in recent military conflicts allow, for a degree of optimism for those pTBI patients who survive the initial trauma. Pre-hospital and emergency management has improved survivability by limiting the development of secondary brain injuries, and rapid, aggressive surgical intervention has been shown to improve outcomes in even the most severely injured patients. Emergency medical personnel should undergo proper training and be able to treat pTBI patients in the field using treatment guidelines that are similar to those provided to military medics. All patients who survive long enough to make it to the hospital should undergo aggressive resuscitation, and neurosurgery should be notified without delay so those patients who may benefit from decompression and debridement can be promptly identified and taken to surgery. Based on data provided by military medical providers, this should include those with low GCS scores between 3-5 as we now have evidence that some of these patients seems to benefit from early surgical care. These patients should be managed post-operatively in dedicated neurological intensive care units to provide the best opportunities for short and long-term survival and functional outcomes. Additional research and experience is needed to determine optimal parameters for limiting secondary brain injury, and the search for effective neuroprotective agents must continue.

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