Chapter 24
Head Injury

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24.1 Introduction

While the term head injury technically refers to a traumatic insult to the scalp, skull, or brain, it often is used to describe brain injury. The term traumatic brain injury—TBI—is more precise. The US Department of Defense defines TBI as a traumatically induced structural or physiological disruption of brain function (decreased or complete loss of consciousness, amnesia, neurological deficits or alterations in mental state, intracranial lesion) as a result of an external force. The severity of brain injury is stratified into mild, moderate, or severe based on the patient's clinical presentation.

24.2 Epidemiology

Head injuries and TBIs are significant public health problems globally and continue to be a cause of thousands of deaths and disabilities worldwide. TBI is one of the leading causes of overall mortality in trauma patients and is particularly implicated in early and late deaths [1]. Accurately comparing the incidence of TBI in

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different studies is challenging because of the lack of a standardized definition in the literature [2]. According to a report released by the Centers for Disease Control (CDC) in 2014, there were 2.87 million (837,000 in children) TBI-related ED visits, hospitalizations, and deaths in the USA and TBI contributed to 56,800 deaths (2529 in children) [2]. Between 2006 and 2014, TBI-related ED visits increased 54%, hospitalization rates decreased by 8%, and death rates decreased by 6% [2]. A cross-sectional study from Europe found that in 2012 there were 1.38 million TBI-related hospital discharges and 33,415 TBI related-deaths across 25 counties in Europe [3].

In the same report published in 2014, the CDC found falls to account for roughly half of all TBI related emergency department visits. Overall, falls and motor vehicle crashes respectively were the leading causes of all TBI-related hospitalizations (52 and 20%), while motor vehicle crashes were the leading cause of TBI-related hospitalizations among adolescents and adults. Intentional self-harm was the leading cause of TBI-related deaths (33%) [2].

24.3 Emergency Room Presentation

Regardless of the anatomic location of injury, all trauma patient should be evaluated in a systematic fashion. The primary survey consisting of airway maintenance with cervical spine precautions, breathing and ventilation, and circulation with control of hemorrhage. At the end of the primary survey, a quick neurological evaluation should be performed. The most common and widely validated method includes the Glasgow Coma Scale (GCS) (Table 24.1) [4]. The scale consists of three

Table 24.1 The Glasgow Comas Scale [4]

Glasgow Coma Scale		
Eye opening	Spontaneous	4
	To voice	3
	To pain	2
	No response	1
Verbal	Alert and oriented	5
	Disoriented/confused	4
	Non-sensical speech	3
	Moaning/incomprehensible speech	2
	No response	1
Motor	Follows commands	6
	Localizes to pain	5
	Withdraws to pain	4
	Decorticate (flexor) posturing	3
	Decerebrate (extensor) posturing	2
	No response	1

components: eye response, verbal response, and motor response, and has been shown to be predictive of in-hospital mortality [5]. The addition of pupillary size and light reactivity to this quick assessment has been shown to have significant prognostic value [6].

After the initial primary survey and stabilization of patient's vital functions, a more thorough secondary survey is undertaken. Any external signs of head trauma (suspected open or depressed skull fractures, signs of basilar skull fractures), GCS < 15, multiple episodes of vomiting, any period of amnesia or loss of consciousness or dangerous mechanism of trauma should be followed up with a computed tomography (CT) scan of the head [7].

24.3.1 Principles of Early Management

- Blood pressure: Stabilization of blood pressure is crucial in early management. A single episode of systolic blood pressure < 90 mm Hg has been shown to double the rate of mortality, with current ATLS guidelines focusing on avoidance of hypotension [8]. Hypotension impairs cerebral blood flow and could exacerbate brain injury. Hypertension, on the other hand, can propagate and exacerbate hemorrhage.
- Oxygenation: O_2 saturation is crucial to maintain the increased demands in acute TBI. Hypoxia (O_2 Saturation < 90% or PaO_2 < 60 mm Hg) has been shown to increase mortality rates [9]. Early efforts to improve oxygenation, including intubation, are paramount.
- Temperature: Currently, data from randomized control trials have failed to demonstrate the benefit of hypothermia in prevention of secondary injury in TBI patients, but mild hypothermia can still be utilized for secondary benefit in lowering Intracranial pressure [10, 11]. However, it should be used judiciously owing to increased susceptibility to infections (mainly pneumonia and sepsis) and can result in coagulopathy [12, 13]. Normothermia maintenance is preferred as initial treatment in severe TBI [11].
- Hematocrit: Patients with TBI may often have concomitant injuries that lead to acute blood loss. Transfusions should be reserved for those patients who have acute blood loss or show signs of decreased tissue perfusion. However, no current research demonstrates a benefit in maintaining hemoglobin >10 g/dL or hematocrit >30% [14]. Advances in monitoring of coagulopathies using thromboelastography (TEG) and rotational thromboelastometry (ROTEM) have enabled optimization of blood product therapy [15]. However, studies demonstrating the accuracy of these technologies and correlation of their use in improving outcomes are still lacking [16, 17].
- The goal of early resuscitative measures is not so much as to reverse the initial traumatic insult, but is rather targeted at the prevention of secondary brain injury.
- Other considerations:

- Hyperventilation: While there is a role for hyperventilation in quickly reducing intracranial hypertension (by reducing the intracranial pressure, ICP), prophylactic or extended use should be avoided to prevent reduction in cerebral blood flow (CBF) that can lead to cerebral ischemia. Hyperventilation should be strictly used as a temporizing measure when patients exhibit dangerous signs of increased ICP until more sustainable efforts, such as hyperosmolar solutions or surgical decompression are possible.
- Hyperosmolar solutions: Mannitol and more recently hypertonic saline have been used as a therapeutic measure to reduce intracranial hypertension. These solutions initially increase intravascular volume causing hemodilution that leads to decreased blood viscosity and improved cerebral blood flow, although increasing osmotic gradient and reducing brain water content may also be a factor [18, 19]. Mannitol is usually given as a bolus of 0.25–1 g/kg over 20–30 minutes with peak effect in 20 minutes. However, because of mannitol's diuretic properties, failure to replace urine output with normal saline or isotonic solutions can lead to systemic hypotension and resultant cerebral ischemia. More recently, hypertonic salines (in this case referring to solutions containing at least 3% sodium chloride) have gained favor as a hyperosmolar solution as they can reduce ICP in patients without the adverse effects of nephrotoxicity and hypovolemia [20]. More recent studies have also shown hypertonic solutions to be superior to mannitol and have championed their use as a first-line medical therapy for reducing ICP [21]. The dose varies considerably, but commonly includes 30 mL boluses of 23.4% sodium chloride or 150 mL boluses of 3% sodium chloride. About 3% sodium chloride solutions may also be run as infusions with serum sodium goals of 145–150 mEq/L or higher, but not to exceed 155. It is generally felt that serum osmolality should not exceed 320 mEq/L [11].
- Antiepileptic drugs (AEDs): About 5–7% of patients with TBI experience early or late posttraumatic seizures [22]. Phenytoin is one of the most studied agents for posttraumatic seizure prophylaxis [23]. Levetiracetam has been gaining popularity as an alternative due to its ease of use without requiring drug level monitoring. In some studies, levetiracetam has been shown to be better at prophylaxis for posttraumatic seizures [24]. Prophylaxis against early posttraumatic seizures is usually administered for no greater than 7 days post-TBI, unless clinically warranted [25]. However, no AEDs to date have been shown to reduce the incidence of epilepsy following TBI [26].
- Steroids: Currently, high-dose steroids are contraindicated acutely (within 72 hours) in TBI and have shown to increase mortality rates in the CRASH trial [27].

24.4 Classification of Head Injuries

- Scalp laceration: Can be found during the secondary survey or may need to be addressed during the primary survey if they continue to be a source of major bleeding. Patients arriving in hemorrhagic shock with scalp lacerations may ini-

Table 24.2 Types of skull fractures

Skull fractures		
Location	*Considerations*	
Vault	Linear or stellate	
	Depressed or non-depressed	
	Open (compound) or closed (simple)	
Basilar	*Temporal fractures*	Longitudinal: parallel to eternal auditory canal, VII/VIII nerve sparing
		Transverse: perpendicular to external auditory canal, with potential VII/VIII nerve involvement
	Clival fractures	Longitudinal, transverse, or oblique
	Occipital condyle fractures: Anderson and Montesano classification types	Type I: Communition of the condyle following impact. *Stable*, as there is minimal fragment displacement into foramen magnum
		Type II: Results from a basilar skull fracture extending into one or both condyles. *Stable*, provided the tectorial membrane and alar ligament remain intact
		Type III: Avulsion fracture due to lateral bending and rotation. *Rule out occipitocervical dislocation*. May be *unstable*

tially have small amount of active bleeding. After resuscitative measures are completed and blood pressure normalized, the laceration may continue to bleed actively. Bleeding can be controlled with direct pressure, cauterization, or application of Raney clips in emergent situations. Prior to wound closure, a laceration should be thoroughly inspected for underlying fractures and CSF leaks.

- Skull fractures: Classification of skull fractures in Table 24.2.
  - Cranial vault fractures: Linear fractures are usually self-limiting and rarely require any surgical intervention by themselves. Figure 24.1 shows a displaced left occipital fracture with an associated subdural hematoma, as well as a fracture near the torcula causing an epidural hematoma. This patient sustained multiple injuries after being struck by a car. Depressed skull fractures generally require surgery if there are any signs of CSF leakage, underlying hematoma, neurological deficit corresponding to underlying brain parenchyma, and/or depression >1 cm. Special considerations should be made when fractures are overlying dural sinuses. The sinus may require repair, and there is a risk of significant intraoperative blood loss and air embolism. Prophylactic antibiotics and early surgical management are crucial in reducing risk of infection [28].
  - Basal skull fractures: The fractures may be associated with CSF leaks and cranial nerve palsies. Clinical signs that raise concern for basilar fractures include periorbital ecchymosis (raccoon eyes) and retroauricular ecchymosis (Battle's sign). Currently, there is no data to support the use of prophylactic antibiotics in patients with basilar fractures, with or without CSF leak, with no difference in frequency of meningitis, all-cause mortality, or meningitis-related mortality [29].

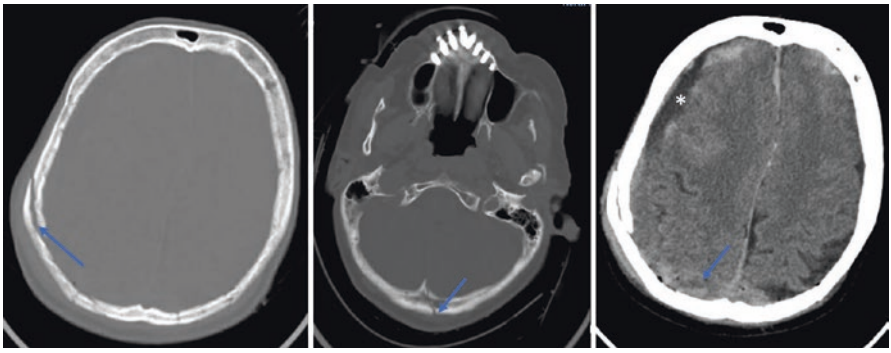

Fig. 24.1 CT scan demonstrating multiple skull fractures—a displaced right occipital fracture (left), a nondisplaced fracture near the torcula (middle), a subdural hematoma (right, *) associated with the occipital fracture, and an epidural hematoma and intracranial air associated with the sinus injury (arrow)

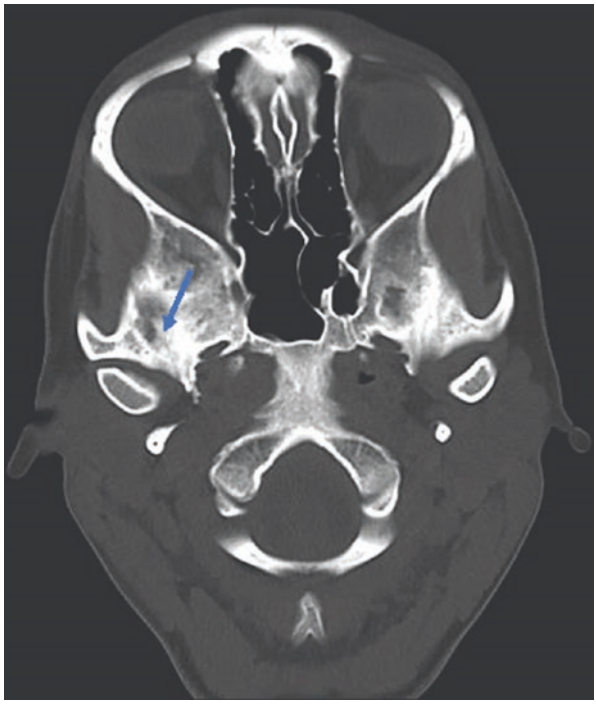

Fig. 24.2 CT scan demonstrating a nondisplaced right temporal bone fracture

– Temporal bone fractures: These may be detected with otoscopic examination of the external auditory canal (EAC). They may result in unilateral VII or VIII nerve palsies. An ear/nose/throat (ENT) consult is warranted in these patients, and steroids can be started for any new onset cranial nerve palsy. If symptoms do not improve with steroids, surgical decompression may be considered, usually in a delayed fashion. Figure 24.2 demonstrates a nondisplaced right

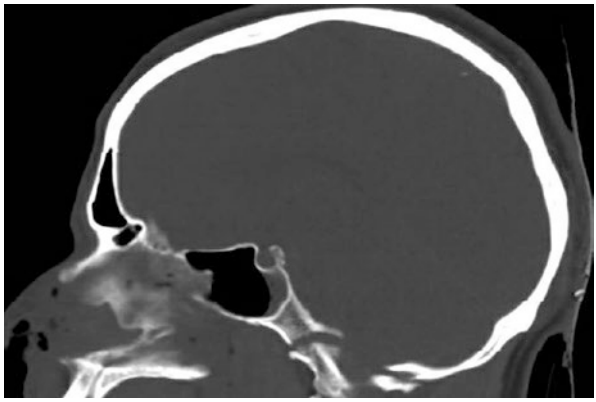

Fig. 24.3 CT scan demonstrating a transected clivus in a pedestrian struck. The patient sustained multiple skull fractures and a significant closed head injury

temporal bone fracture in a patient presenting with retroauricular ecchymosis after a fall.

- Clival fractures: These should raise high suspicion for injury to CN III-VIII, vascular dissections, or occlusions of the vertebrobasilar system and/or anterior circulation, and delayed development of traumatic aneurysms. When there is high suspicion of vascular injury, standard noncontrast CT can be followed with a CT angiogram. Figure 24.3 shows a displaced fracture of the clivus in a patient struck by a fast-moving vehicle.
- Occipital condyle fractures: These types of fractures have a very rare incidence of about 0.4–0.7% patients with major trauma [30]. Diagnosed initially on a CT scan, these should be followed up with a magnetic resonance imaging (MRI) to assess integrity of the craniocervical complex. A type III fracture that involves avulsion of the condylar fragment is usually treated with external immobilization for 6–8 weeks. Surgical fixation is usually indicated in those patients with compression of neural elements or craniocervical misalignment (occiput to cervical interval > 2 mm). Figure 24.4 shows an occipital condylar fracture in the same patient who sustained the clival transection.
- Pneumocephalus and tension pneumocephalus: These injuries results from air build up within the cranial cavity following damage to the skull and/or meninges. Persistent build up in the case of tension pneumocephalus can result in significant mass effect on the brain followed by neurological deterioration. Treatments include positioning the patient with the head down (i.e., in Trendelenburg), use of a non-rebreather mask with 100% oxygen, and in cases of tension pneumocephalus, surgery to repair the skull, dural lacerations, and close any areas where air may be pulled into the brain. Figure 24.5 shows a small amount of pneumocephalus and a small subdural hematoma adjacent to a traumatic right frontal bone fracture. This patient did not develop tension pneumocephalus and did not require any intervention.

- Intracranial hemorrhage:

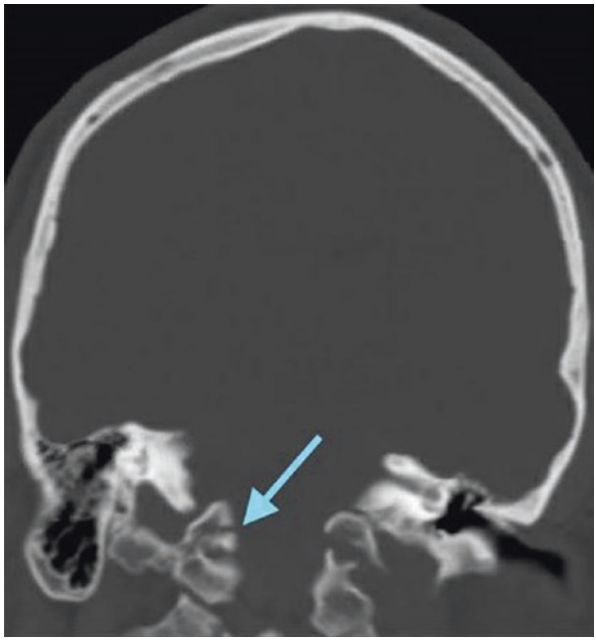

Fig. 24.4 Occipital condylar fracture (arrow). This patient also sustained a transection of the clivus, indicative of craniocervical dislocation

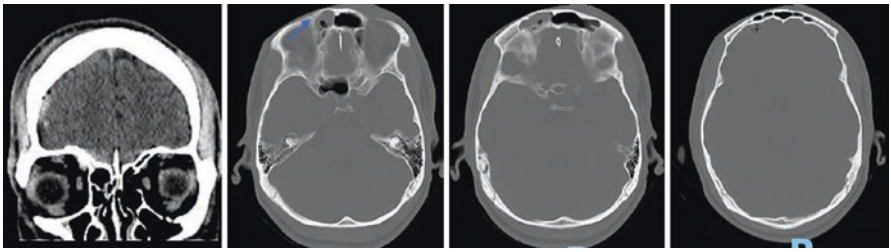

Fig. 24.5 CT scan demonstrating a small subdural hematoma and pneumocephalus resulting from a right frontal bone fracture (arrow)

– Epidural hematomas (EDH): These are relatively uncommon, especially in patients aged 2 years and younger or 60 years and older. EDHs are caused by the disruption of an arterial source of bleeding, often by temporal bone fractures disrupting the middle meningeal artery. In the case of arterial bleeding, these lesions usually become symptomatic due to mass effect causing localized pressure on brain parenchyma. As the EDH grows, it puts continued pressure on the brain and can cause uncal herniation. Patients typically can present with lucid interval (which can last hours) followed by rapid deterioration. Symptoms include contralateral hemiparesis, ipsilateral hemiparesis (Kernohan notch phenomenon [31]), ipsilateral pupillary dilation, and obtundation. EDHs are diagnosed with CT scans showing classical biconvex shape, usually with uniform density. Small, nonsymptomatic stable hematomas

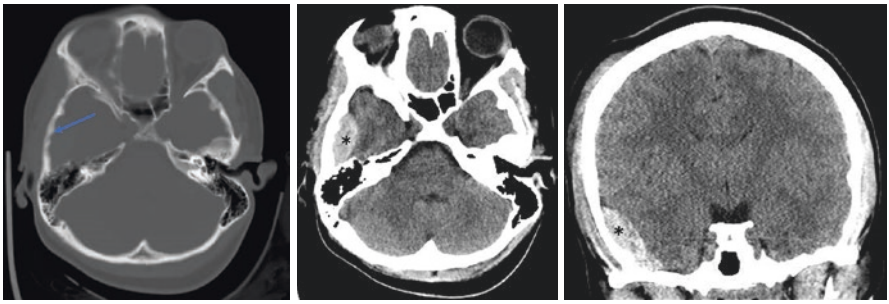

Fig. 24.6 CT scan demonstrating a temporal skull fracture (arrow) with middle meningeal artery tear causing an acute epidural hematoma (*)

(often resulting from venous bleeding) may be managed medically with serial imaging and observation. Volume > 30 cm^3, altered or decreasing GCS, and/or neurological symptoms are all indications for emergent surgical evacuation of the clot, with an emphasis on surgical hemostasis and tenting of the dura in the region to prevent reaccumulation. Figure 24.6 demonstrates an EDH in a young woman who fell and sustained a right temporal bone fracture.

– Subdural hematomas (SDH): Subdural hematomas result from disruption of bridging vessels along the surface of the brain, and are often associated with underlying damage to brain parenchyma. Use of anticoagulation and antiplatelet agents has been shown to increase the risk of developing subdural hematomas [32]. Based on timing of presentation, SDH are usually divided into acute (1–3 days), subacute (4 days–2–3 weeks), and chronic (>3 weeks). Age of the hematoma and appearance on CT scan significantly alters the management. These lesions are usually diagnosed with a CT scan and are typically divided into convexity, interhemispheric, tentorial, or posterior fossa locations.

  Acute subdural hematomas (aSDH): These can be managed medically or observed with serial imaging and clinical assessment if there is no abnormal neurological exam, the GCS score remains stable, and there is no significant midline shift. Surgical evacuation is warranted if thickness is >10 mm or midline shift >5 mm regardless of clinical presentation [33]. If possible, use of anticoagulation or anti platelet agents and their adequate reversal (Table 24.3) should be noted prior to surgery. Surgical evacuation entails craniotomy or craniectomy for adequate evacuation of acute blood clot and adequate exposure to control any active bleeding. The bone flap can be left off (craniectomy) if there is concern for significant swelling at the time of surgery. Figure 24.7 shows a thick, acute subdural hematoma with midline shift.

  Chronic subdural hematomas (cSDH): These are usually seen in elderly patients, with or without history of trauma. The fluid collection is generally motor oil in appearance and consistency. If the fluid collection is clear

Table 24.3 Anticoagulation and anti-platelet drugs and their reversal agents

Drug	Reversal agent
Antiplatelet agents	
1. Glycoprotein IIb/IIIa inhibitors (eptifibatide, tirofiban, abciximab)	Platelet transfusion (partially effective for abciximab) [49]
2. Platelet aggregation inhibitors (acetylsalicylic acid/aspirin, clopidogrel, cangrelor, integrilin, ticagrelor)	1. Desmopressin 2. Platelet transfusion (only indicated when planned surgical intervention. To be given at the time of maximal desired benefit) [50] 3. Cryoprecipitate
3. Protease-activated receptor-1 antagonist (vorapaxar)	Platelet transfusion (limited efficacy) [51]
Anticoagulation agents	
1. Vitamin K antagonist (warfarin)	1. Vitamin K (non-emergent reversal) 2. Fresh frozen plasma (FFP) 3. Prothrombin complex concentrate (PCC, 3 or 4-factor)
2. Indirect Factor Xa inhibitors (antithrombin activation) (unfractionated heparin, low molecular weight and ultra-low molecular weight heparin, enoxaparin, fondaparinux)	1. Protamine sulphate (not effective for fondaparinux) 2. Recombinant factor VII (for reversal of fondaparinux)
3. Direct thrombin inhibitors (dabigatran and argatroban)	Idarucizumab (antibody fragments against dabigatran) [52]
4. Direct factor Xa inhibitors (rivaroxaban, apixaban, edoxaban)	1. Tranexamic acid 2. Prothrombin complex concentrate [52] 3. Andexanet alfa (not indicated for reversal of edoxaban) [53]

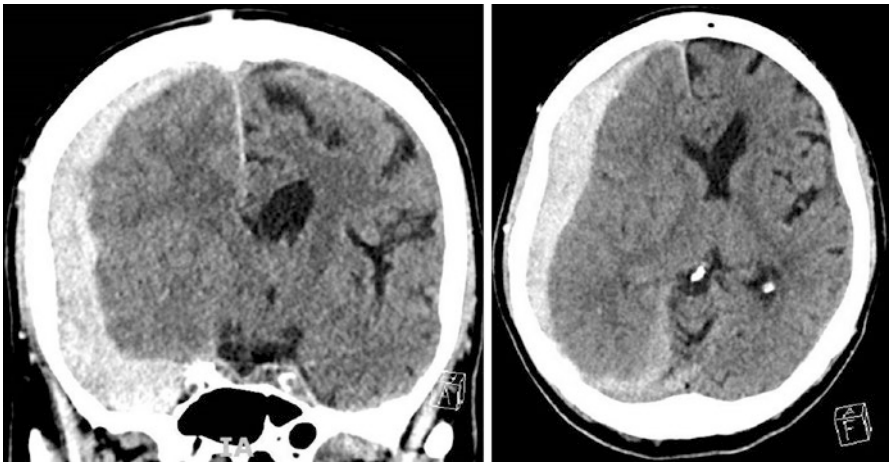

Fig. 24.7 CT scan demonstrating a large, acute right sided subdural hematoma causing sulcal and cisternal effacement with significant midline shift in a patient who fell

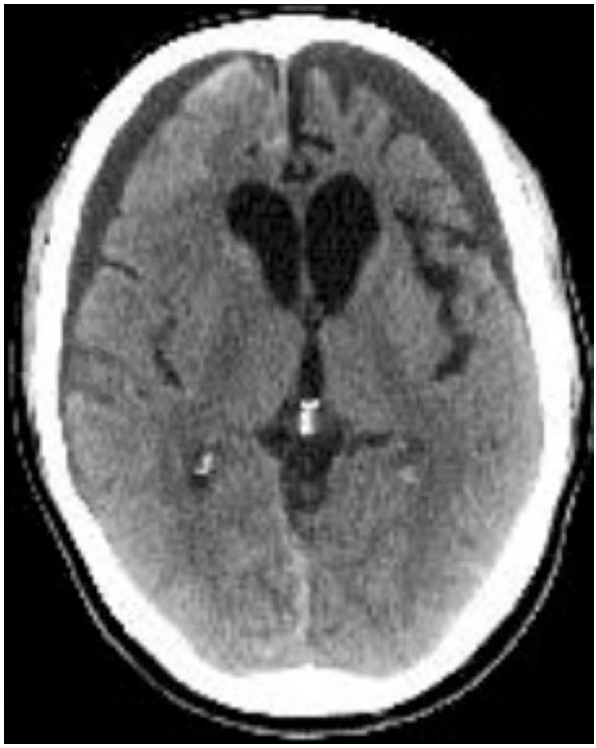

Fig. 24.8 CT scan demonstrating bilateral chronic subdural hematomas in an elderly patient

CSF, it is termed a subdural hygroma. cSDH usually are a sequalae of acute bleeds and form as a result of degradation of the blood clot and formation of neomembranes. Indications for treatment include patients who present with neurological symptoms and large or increasing size. Chronic SDH may be treated with burr holes, with or without the use of subdural drains. Complications include reaccumulation as a result of failure of re-expansion of the brain, seizures, and in some instances subdural empyemas [34]. Figure 24.8 shows the CT scan of a patient with bilateral chronic subdural hematomas.

Special considerations in treatment of cSDH

- Steroids: Dexamethasone has been used in the treatment of cSDH, either pre or postoperatively, especially in the event of recurrence. Results are mixed. The goal of steroid treatment is to reduce inflammatory mediators and inhibit the formation of neomembranes and neocapillaries, thus reducing recurrence or propagation [35]. However, one meta-analysis found an increased morbidity without any significant improvement in recurrence and cure rate with adjuvant use of corticosteroids [36]. A lack of randomized trials leaves its use reserved for the symptomatic patient until surgical drainage is possible, in those in whom surgery is associated with high risk, or at the surgeon's discretion.

- Tranexamic acid (TXA): An antifibrinolytic agent has been proposed as a stand-alone or adjuvant to surgery in treating cSDH. A recently published randomized trial showed that, while steroids used postoperatively reduced cSDH recurrence, the complication rate was higher that those patients receiving placebo [37]. One study from Japan found a significant decrease to complete cure in patients taking TXA, regardless of surgical drainage [38]. However, its role in patients taking anticoagulation has not been investigated. Currently, the use of tranexamic acid in chronic subdural hematomas (TRACS), a placebo-controlled phase IIb trial, is underway to review the safety and feasibility of its use in these patients [39].
- Other: Some studies have also investigated the use of atorvastatin and angiotensin converting enzyme (ACE) inhibitors with mixed results [35].
- Middle meningeal artery (MMA) embolization: Recently centers have investigated angiographic embolization of MMA in treatment of refractory or recurrent cSDH showing resolution, decreased rates of recurrence, and similar complication rates to surgical drainage [40–42].

– Traumatic intracerebral hemorrhage (tICH): Traumatic ICH lesions usually present in a coup-contrecoup pattern within parenchyma near bony prominences (frontal, temporal, and occipital poles) in patients suffering from head trauma. CT scan usually makes the diagnosis. Some patients present in a delayed fashion with tICH not present on the initial scan. Most can be treated nonsurgically, with interval imaging to demonstrate stability. Careful monitoring for enlargement, mass effect with increasing midline shift, with or without signs of impending transtentorial herniation may warrant surgical evacuation. Nonsurgical management includes strict blood pressure control,

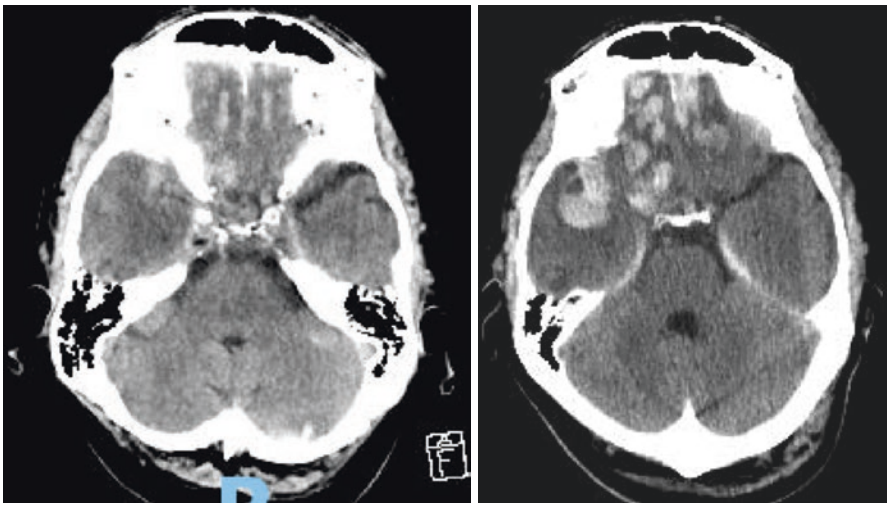

Fig. 24.9 CT head demonstrating bifrontal and a right temporal contusion (left) with blossoming of the contusions on a scan obtained 6 hours later (right)

correction of any systemic coagulopathy, serial imaging, and ICP monitoring for patients with GCS less than 8 [25]. Figure 24.9 shows a tICH that blossomed on an interval follow-up scan.

– Diffuse axonal injury (DAI): DAI results from acceleration/deceleration motions that shear axons at gray-white matter junctions. High-speed motor vehicle crashes are the most common cause. Adam's classification is used to describe the severity of DAI:

  Grade I—Mild DAI involving corpus callosum, brainstem, and cerebral cortex
  Grade II—Moderate DAI with focal lesions in the corpus callosum
  Grade III—Severe DAI with additional focal lesions in the brainstem

  DAI is suspected in patients with a persistently low GCS after resuscitative measures and/or surgical treatment of other traumatic brain injuries. Patients with mild DAI may have mild headaches and dizziness. Severe injuries may present with persistent vegetative state. These patients may present with central autonomic dysfunction such as tachycardia, tachypnea, vasoplegia, hyperthermia, and posturing [43]. CT scans are usually not helpful; diagnosis is usually made with MRI. Diffusion tensor imaging showing drop in fractional anisotropy in white matter tracts is diagnostic; microhemorrhages are often seen on other sequences, including susceptibility weighted imaging (SWI). Treatment remains primarily nonsurgical and is aimed at preventing secondary injury. Early resuscitation and maintaining cerebral perfusion by preventing hypotension, reducing ICP, and improving oxygenation are mainstays of management. Invasive neuromonitoring for ICP and monitoring of brain oxygenation is indicated in patients with GCS < 8. Prognosis in severe diffuse axonal injury remains poor. Figure 24.10 demonstrates MRI findings in a patient with DAI.

- Penetrating head injuries: These can be caused by missile (i.e., gunshot wounds) or nonmissile injuries. Primary injury occurs by damage to the scalp and surrounding facial structures (e.g., orbits). The tract formed by the bullet or object

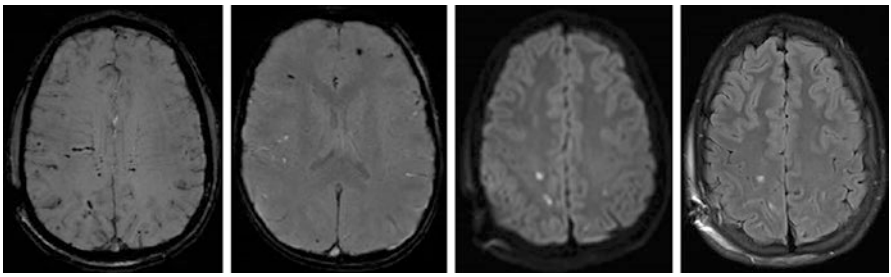

Fig. 24.10 MRI findings in diffuse axonal injury (DAI). The two left images demonstrate microhemorrhages on susceptibility weighted imaging (SWI) in two different patients. The third image demonstrates high signal on diffusion weighted imaging (DWI), and the rightmost image demonstrates the corresponding FLAIR changes

seeds bacteria into the inner parenchymal structures. Comminuted fractures of the skull damage underlying blood vessels, which can cause hematomas and direct damage to brain parenchyma. Fragmentation, richochetting, and deformation of the missile, as well as pressure waves if the weapon is at close range, can cause significant additional injuries and trauma. Secondary injury follows, with fulminant cerebral edema, sudden increase in ICP, and decrease in cerebral perfusion. Delayed complications include abscess, traumatic aneurysm, arteriovenous fistulas (AVFs), seizures, and complications associated with fragment migration (like hematomas).

- The work up begins with the identification of the entry and exit sites and x-rays and CT scans to identify the missile and extent of injury to inner structures. Angiographic imaging is indicated in patients with suspected vascular injuries. For patients with low GCS scores, surgical management is not indicated. If surgery is pursued, goals include debridement, evacuation of hematomas, removal of missile and bone fragments, and hemostasis.
- Neuromonitoring devices:
  - Electroencephalogram (EEG): EEG can detect subclinical or non-convulsive seizures. Consider EEG in patients who have a clinical picture does not match the imaging findings, and when in patients have symptoms that wax and wane. Noninvasive scalp electrodes are usually used. Besides the detection of seizure activity, cortical surface electrodes may be useful in diagnosing cortical spreading depression (CSD), or peri-infarct depolarization (when located in peri-ischemic brain tissue). Recently, use of ketamine has shown to be beneficial in inhibiting CSD in acute brain injury [44].
  - ICP monitors: ICP is recommended to be monitored in patients with a GCS 8 or less or in other patients whom a concern that intracranial hypertension may develop in the post injury period. ICP can be measured with an external ventricular drain (EVD) or a parenchymal pressure monitor, sometimes called a "bolt." EVDs allow CSF drainage; however, draining CSF requires intermittently clamping the drain in order to view the ICP. Parenchymal monitors allow continuous ICP monitoring. Since they are useful in measuring more regional cerebral pressure they should be placed in areas at greatest risk of injury. Unfortunately, values tend to drift with time and probes cannot be recalibrated. Regardless of the device, post insertion CT scan is needed to confirm placement and identify any complications (e.g., hemorrhage, incorrect position). Figure 24.11 shows an intraparenchymal pressure monitor (bolt).
  - Brain oxygenation monitors: The monitoring of brain tissue oxygen tension ($PbtO_2$) has been rapidly gaining popularity. In conjunction with ICP, the clinician may monitor and manage cerebral perfusion pressure. The probe is inserted into white matter and provides information on regional oxygen tension. Brain tissue oxygenation <20 mm Hg should trigger an algorithm to address tissue hypoxia [45].

    Global cerebral oxygenation can be assessed with $SJVO_2$ monitors that involve placement of a fiberoptic probe in the internal jugular vein. Values below 50% indicate global ischemia. They can also be used to measure oxygen content from jugular venous blood, from which arterial jugular venous

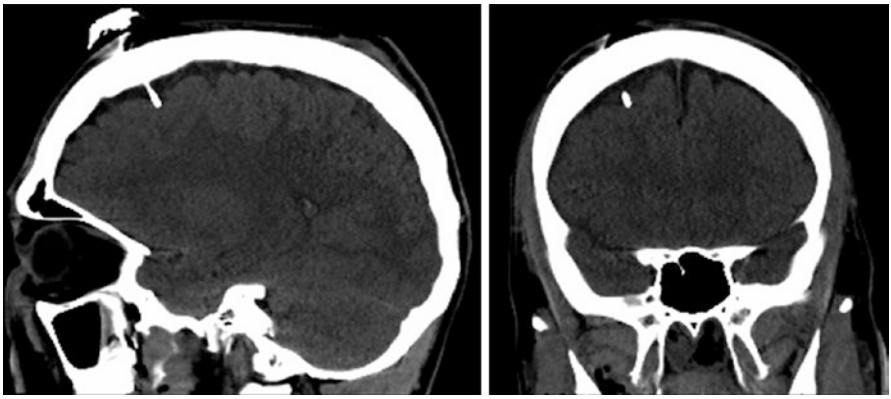

Fig. 24.11 CT scan demonstrating an intraparenchymal pressure monitor (bolt)

content difference can be calculated. Values above 9 mL/dL indicated presence of global ischemia and less than 4 mL/dL indicates hyperemia [46].

– Cerebral metabolism monitors: Microdialysis catheters allow frequent monitoring of substrates and neurotransmitters, giving insights into cerebral metabolism and informing of need for additional and/or response to therapies. The catheter has a semi-permeable membrane, and is inserted into the subcortical white matter. While current guidelines have not yet found sufficient evidence to recommend its routine use in TBI patients, it has been shown to strongly correlate with outcomes [47, 48]. Common substrates monitored include glucose, pyruvate and lactate levels. Additionally, glutamate and glycerol, markers of vasospasm and underlying ischemia, can be monitored. Placement in perilesional areas, or areas at greatest risk of injury, provides most valuable information to guide treatment.
– Other: Non-invasive imaging modalities like positron emission tomography (PET) and MR spectroscopy (MRS) may also play a role in monitoring cerebral metabolism. These technologies are not widely used in TBI management.

24.5 Conclusions

TBI remains a significant global health problem and is directly related to early and late mortality in trauma patients. Management is targeted toward early resuscitation and prevention of secondary injury.

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