Chapter 3
Contusion and Diffuse Injury

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Clinical Scenario

A 25-year-old female presents after a pedestrian versus auto collision in which she struck the back of her head. Her Glasgow Coma Scale (GCS) score was 13 (E3V4M6) both in the field and in the emergency room. It is unknown whether she had loss of consciousness (LOC). She cannot remember the incident, indicating post-traumatic amnesia (PTA).

3.1 History and Neurologic Exam

On clinical examination, our patient is alert but only oriented to her name. She moves all extremities equally. She has clear signs of trauma, including abrasions and an occipital subgaleal hematoma.

Cerebral contusion and diffuse traumatic brain injury describe a spectrum of traumatic brain injury (TBI) that initially presents without a space-occupying mass lesion. Contusions can evolve to become space-occupying lesions, causing increased intracranial pressure and mass effect. Pure diffuse axonal injury (DAI) does not typically follow this course. DAI is associated with contusions, as the force required to generate a contusion usually leads to some degree of diffuse injury as well. This has been confirmed when examining TBI patients with MRI [1].

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A diffuse traumatic brain injury is to be suspected in any patient with altered mental status after a traumatic event. If the event is witnessed, the presence of LOC, PTA, alteration of consciousness (AOC), or seizures should be determined. These clinical "biomarkers" help the physician establish the severity of the injury. Additionally, the clinician should determine what the GCS was for the first responders in the field, as well as on arrival to the emergency department. A worsening GCS could portend an expanding mass lesion that requires emergent neurosurgical intervention. A low GCS—out of proportion to imaging findings—should raise concerns for DAI. If witnesses are able to describe the mechanism of trauma or even the site of impact to the head, this information can prove valuable.

In the patient with suspected but unwitnessed trauma (the "found down" patient), physical examination and imaging findings can help clue the examiner in to the presence of a diffuse traumatic brain injury. External signs of trauma out of proportion to what might be expected for a ground level fall point to trauma as an etiology rather than a syncopal event. Regardless of whether the event was witnessed, patients with diffuse traumatic brain injury often present with a degree of altered mental status that makes reliable history taking impossible. Instead, history must be obtained by speaking with relatives or caregivers and investigating any available medical records.

Relevant factors beyond the acute injury to be investigated include:

- *Use of medications that could increase bleeding risk (antiplatelets or anticoagulants) or history of a bleeding disorder*. This can include chronic liver or kidney disease. The reason for the antiplatelet or anticoagulant use must also be ascertained, as holding or reversing these medications also entails risk.
- *History of conditions which may preclude a patient from having a "normal" GCS at baseline*. For example, a patient with a history of stroke or spinal cord injury may have focal neurologic deficits at baseline, while a patient with dementia, mild cognitive impairment, or schizophrenia might have baseline confusion (GCS14, E4V4M6).
- *Drug or alcohol dependence*. Withdrawal from certain toxic substances, notably alcohol and methamphetamine, can mimic a neurologic decline from a mass lesion. This is especially relevant in the TBI population, which has a high rate of drug and alcohol use [2].
- *Systemic conditions that may complicate mechanical ventilation or fluid resuscitation*. These include congestive heart failure, kidney failure, and interstitial lung disease.
- *Advanced directive or durable power of attorney documentation*. It is important to provide patient-centric care, respecting any previously articulated wishes a patient may have. Obtaining an advanced directive or speaking with a designated decision maker can help guide clinical interventions.

The presence of polytrauma can alter intervention priorities. Other injuries may be acutely life threatening and influence treatment options for TBI. For example, hypotension from another injury is a contraindication for the administration of mannitol. A patient needing emergency surgery for an extracranial injury would likely

need intracranial pressure monitoring since the neurologic examination cannot be used to monitor for an enlarging mass lesion while under general anesthesia.

Neurologic examination should include evaluation of the distinct components of the GCS. This includes the best motor, verbal and eye-opening response. In an obtunded patient, the clinician should examine the pupillary response to light (ideally using a pupillometer) along with the corneal, cough, and gag reflexes. The examiner should attempt to assess any facial asymmetry and any gross differences in limb strength. If the patient can follow commands, a pronator drift can be assessed. Lastly, the clinician should examine the head for external signs of trauma, such as abrasions, subgaleal hematomas, scalp lacerations, otorrhea, rhinorrhea, raccoon's eyes, and Battle's sign. Signs of trauma to the posterior scalp could clue the examiner in to the presence of the classic frontotemporal *contrecoup* cerebral contusions.

3.2 Differential Diagnosis

The differential diagnosis for our patient would include diffuse traumatic brain injury, such as DAI, contusions, subarachnoid hemorrhage, and intraventricular hemorrhage. Concern for a mass lesion, such as a subdural or epidural hematoma, is also high.

In a patient with a witnessed trauma, the diagnosis is typically clear. However, the case can occasionally be murky, especially when a patient is found down. Even if the trauma is witnessed, it is possible that another event precipitated the trauma, especially in the case of a patient who fell. A syncopal event could cause a patient to fall. If a patient suffers one of these events while operating a motor vehicle, it would likely result in a crash. Thus, the pathogenesis of the patient's altered mental status could be from the underlying event alone, or in combination with the resultant traumatic brain injury. History here is important, as a patient who was seen acting inappropriately prior to the trauma is likely to have had a precipitating event.

Differentiating between a diffuse injury and an extra-axial hematoma is difficult on clinical exam. They often occur together. Lateralizing signs could suggest an extra-axial mass lesion, but this is nonspecific. For a patient with a declining GCS, worsening somnolence or unilaterally decreasing pupil reactivity to light could be signs of an enlarging mass lesion needing emergent surgical intervention.

Post-traumatic seizures can also be a cause of neurologic decline after traumatic brain injury. If an expanding mass lesion has been ruled out by CT scan, seizures should be considered. Rhythmic movements, eye deviation, and vital sign changes could signify seizures, but EEG must be performed to confirm this suspicion.

Toxic-metabolic encephalopathy can also mimic a diffuse brain injury. Again, history is important. A patient who fell or was found down may be suffering from acute intoxication. Knowing if the patient has a history of substance use disorder, takes medications with sedating side effects, or has a severe metabolic disorder would help make this diagnosis.

3.3 Diagnostic Evaluation

Our patient's initial labs are unremarkable, aside from a sodium of 135. A noncontrast CT of the head shows extensive bifrontal contusions, bitemporal contusions, and diffuse subarachnoid blood, along with a nondisplaced occipital bone fracture (Fig. 3.1a, b).

Noncontrast CT head will be the primary diagnostic modality to determine the nature and extent of injury in the acute setting and will facilitate rapid triage for pathology potentially requiring emergent intervention. Diffuse traumatic brain injury findings on CT include contusions, subarachnoid blood, intraventricular hemorrhage, as well as petechial hemorrhages at the gray-white matter junction and along the white matter tracts (corpus callosum, corona radiata, and/or brainstem).

CT is often negative in cases of diffuse injury. MRI, while not appropriate for first-line investigation in the acute setting, is more sensitive, especially for microhemorrhages [1, 3, 4]. Sequences that detect extravascular blood, such as gradient echo, are most sensitive for diffuse injury and DAI. MRI should only be pursued once the patient has been stabilized. A trauma survey CT scan should also be performed to rule out other injuries.

Diagnostic evaluation will be the primary which should include an examination of laboratory results. A basic metabolic panel (BMP), complete blood count (CBC), and coagulation studies (PT, PTT, INR) should be reviewed. Abnormal sodium or creatinine levels may help guide hyperosmolar therapy. While platelet count and coagulation studies are mainstays in the workup of any trauma patient, thromboelastography should also be strongly considered. Antiplatelet agents and newer anticoagulant agents will not always result in abnormal coagulation studies, but these can be detected on thromboelastography [5]. Additionally, traumatic brain injury

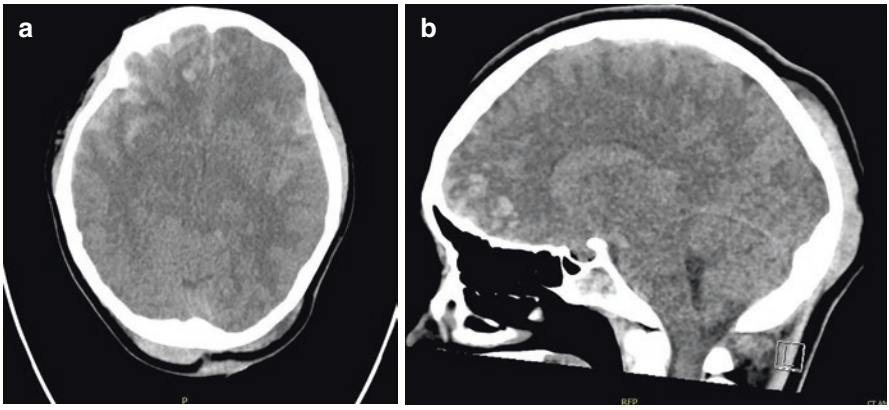

Fig. 3.1 Noncontrast CT head (**a**) axial and (**b**) sagittal images demonstrating the array of findings that might be associated with diffuse brain injury, including bilateral inferior frontal and temporal contusions, as well as traumatic subarachnoid blood. The occipital bone fracture is not well visualized on the brain tissue window

itself can result in a unique coagulopathy which is better characterized by thromboelastography [6]. Blood alcohol level and toxicology screening (both blood and urine) should also be performed in all traumatic brain injury patients [7].

3.4 Clinical Decision-Making and Next Steps

Initial clinical decision-making is guided by principles of Advanced Trauma Life Support (ATLS). Patients with diffuse brain injury may decline rapidly, so clinicians must ensure close monitoring of airway and breathing, even after initial stabilization. Hypotension should be avoided, and adequate fluid resuscitation continued.

The patient in the current clinical scenario was admitted to the neurocritical care unit with hourly neurologic checks, including pupillometer assessment. Antiepileptic drug (AED) prophylaxis was administered, and her sodium was corrected with normal saline fluid resuscitation. Thromboelastography was also obtained and was normal. Her follow-up CT scan showed significant blossoming of her contusions (Fig. 3.2a, b).

Coagulopathy should be corrected. However, for minor injuries and if a compelling indication for anticoagulant or antiplatelet use exists, full reversal need not be performed in every patient. The risks of anticoagulant or antiplatelet reversal must be weighed against the risk of intracranial lesion progression. If the patient requires an invasive procedure, be it surgery or insertion of intracranial monitors, anticoagulant or antiplatelet drugs should be reversed. Clinicians should also consider the potency and half-life of the antiplatelet or anticoagulant drug, including the time of the last dose.

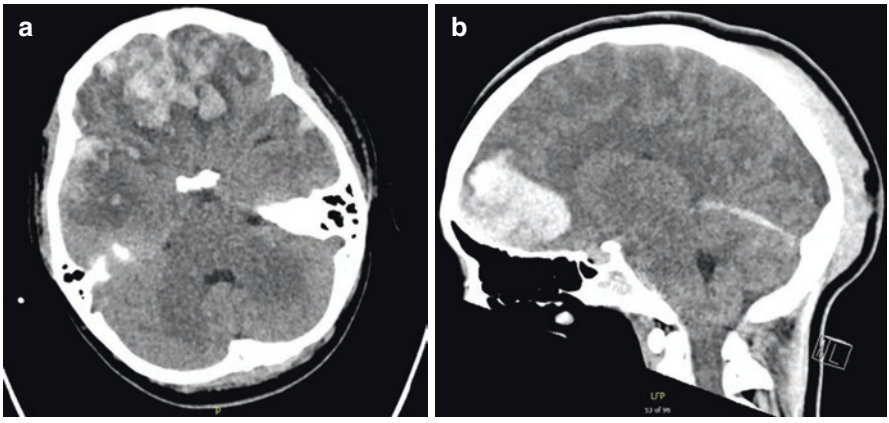

Fig. 3.2 Noncontrast CT head (**a**) axial and (**b**) sagittal images, performed at an interval of approximately 6–8 h from initial presentation, demonstrating generalized progression of the bilateral inferior frontal and temporal contusions, resulting in increased mass effect

All patients should undergo close neurologic monitoring with frequent neurologic checks and pupillometer assessment, along with repeat CT scan (typically 6–8 h after the initial scan). All patients with cerebral contusions or subdural hematoma should receive a loading dose of antiepileptic medications [8], followed by 7 days of administration. The head of bed should be elevated (30–45°). Adequate analgesia and sedation should be maintained in intubated patients. If there is expectation that vasopressors or hypertonic solutions will be administered, a central line should be placed. Arterial line monitoring should be established if cerebral perfusion pressure (CPP) will be monitored.

Much of the general treatment of TBI is outlined in the most recent guidelines released by the Brain Trauma Foundation (BTF) and the American College of Surgeons (ACS) TBI Best Practices Guidelines [9–19]. The BTF recommends intracranial pressure (ICP) monitoring to reduce in-hospital mortality after TBI while the ACS recommends monitoring patients with structural brain injury on CT and a GCS $\leq$8. This should include monitoring of cerebral perfusion pressure. The authors recommend the use of an external ventricular drain (EVD), as this offers the added therapeutic benefit of cerebrospinal fluid (CSF) drainage to lower elevated intracranial pressure [14]. The BTF guidelines recommend continuous CSF drainage for elevated ICP, and since an EVD must be clamped to monitor pressure, the authors also recommend placement of a parenchymal ICP monitor. A dual lumen bolt allows placement of both the parenchymal monitor and a brain tissue oxygenation monitor [20, 21].

Unless ICP is acutely elevated, hyperosmolar therapy is not recommended. Prophylactic use of mannitol or hypertonic saline should not be used [10]. Likewise, prophylactic hyperventilation and hypothermia are not recommended [11, 18]. The clinician should aim to achieve normonatremia, normothermia, and normocarbia.

Most patients with CT-positive diffuse traumatic brain injury will require hourly neurologic assessment, which typically necessitates an admission to an intensive care unit (ICU). While patients with TBI are treated in various ICU types (neuro, surgical, trauma, or medical ICUs), they require specialized nursing care with expertise in ICP monitoring, pupillometer use, and neurologic examination.

In the case of elevated intracranial pressure, a tiered strategy should be used and can be continued in the ICU [22]. Tier one includes ensuring venous return from the head must be unobstructed (neutral head position and removal of cervical collar if cleared). Analgesia and sedation can be increased, intermittent hyperosmolar therapy utilized and CSF drainage to maintain a minimum CPP of 60–70 mmHg.

For the acute patient in the trauma bay, escalation past the first tier of ICP control measures is rarely needed and, if required, would be an unfavorable sign Uncontrolled ICP would be indicative of a mass lesion requiring evacuation or a devastating injury with little chance of survival. However, if escalation beyond the first tier is needed, the second tier involves performing a cerebral autoregulatory challenge [22–24]. This is performed by elevating the mean arterial pressure (MAP) under direct physician supervision. If the ICP increases at a 1:1 ratio with the MAP, the patient is not able to autoregulate cerebral blood flow and future therapies should be directed at lowering ICP, rather than increasing CPP.

If autoregulation is intact, the second tier of ICP management includes using vasopressors or fluid boluses to increase CPP. Regardless of autoregulation status, other second tier interventions are a trial of neuromuscular paralysis and mild hypocapnia (32–35 mmHg). Tier three interventions are barbiturate coma and hypothermia to 35 °C.

It is rare that a diffuse injury, or even large contusions, will cause elevated ICP acutely. The intracranial hypertension typically develops over the next few days as the contusions blossom and the brain become edematous. However, a large contusion causing mass effect and midline shift may require surgical intervention. A declining neurologic exam, pupil asymmetry, or refractory elevated ICP are indications for surgery with a unilateral mass lesion. The operative approach is a large frontotemporoparietal decompressive hemicraniectomy no smaller than 12 × 15 cm. The contusion itself should not be evacuated, as this can lead to uncontrollable intraoperative hemorrhage. The surgical goal is bony decompression alone. Conversely, bifrontal decompressive craniectomy for intracranial hypertension secondary to diffuse injury without a focal mass lesion is not recommended based on the DECRA study [25].

Patients with a pure DAI without contusions rarely develop increased ICP, and monitoring can typically be discontinued after 2–3 days. We typically remove the parenchymal monitor after this trial, which allows the patient to undergo an MRI to help with prognosis. The EVD is MRI compatible and may be left for a few more days to ensure no ICP elevations develop. If the clinical and radiographic pictures indicate the patient will need an early tracheostomy and percutaneous gastronomy tube, keeping the EVD allows for continued ICP monitoring during these procedures.

The patient in the clinical scenario had a neurological exam remained stable. On the second night, her pupil reactivity index began trending downward. She responded to mannitol 50 g with a return to baseline. However, she progressed to develop a waxing and waning neurologic exam, prompting placement of triple monitoring (EVD/parenchymal monitor and brain tissue oxygen monitor). She had a prolonged ICU stay, with intermittent episodes of elevated ICP. These were treated with alternating CSF drainage and hyperosmolar therapy. She was eventually able to return to work 4 months after her accident.

3.5 Clinical Pearls

- Acute intracranial pathology is to be suspected in any patient presenting with altered mental status after trauma. Contusions and diffuse brain injury are typically apparent on initial imaging workup, but more advanced imaging, such as MRI, are required to show the full extent of the damage.
- ATLS protocols should guide initial management for an obtunded patient. Once stabilized. Invasive intracranial monitoring—preferably by external ventricular drain—should be initiated. Management of elevated ICP should follow a tiered protocol.

- Mass lesions causing elevated intracranial pressure should are candidates for surgical intervention. However, two large trials have failed to show improved outcomes with decompressive craniectomy in the setting of refractory ICP elevations.

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