Head Injuries: Neurosurgical and Orthopaedic Strategies

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Michael A. Flierl, Kathryn M. Beauchamp, and Philip F. Stahel

Contents

M.A. Flierl
Department of Orthopaedic Surgery,
Denver Health Medical Center,
University of Colorado Denver School of Medicine,
777 Bannock Street, Denver, CO 80204, USA

K.M. Beauchamp
Department of Neurosurgery, Denver Health Medical Center,
University of Colorado Denver School of Medicine,
777 Bannock Street, Denver, CO 80204, USA

P.F. Stahel (✉)
Department of Orthopaedic Surgery and Department
of Neurosurgery, Denver Health Medical Center,
University of Colorado Denver School of Medicine,
777 Bannock Street, Denver, CO 80204, USA
e-mail: philip.stahel@dhha.org

6.1 Pathophysiology of TBI

Despite modern intensive care strategies, the clinical outcome of severely head-injured patients remains poor [1–3]. The high mortality rates in this patient population are often attributed to the development of secondary insults to the injured brain [4–6]. While *primary brain injury* is a result of the mechanical forces applied to the skull at the time of impact, *secondary brain injury* evolves over time and thus cannot be detected on initial CT imaging studies [7, 8]. Evidence of secondary brain injury has been found on autopsy in 70–90% of all fatally head-injured patients [7, 9]. Secondary brain injury is initiated by a trauma-induced, host-mediated inflammatory response within the intracranial compartment [10–14], and is aggravated by hypoxia, metabolic acidosis, cerebral fat emboli from the fracture site, injury-triggered activation of the coagulation system, and development of cerebral edema [9, 15–19].

The immuno-patho physiological sequelae of Traumatic Brain Injury (TBI) are highly complex, and involve numerous brain-derived pro-inflammatory mediators, such as cytokines, chemokines, complement anaphylatoxins, excitatory molecules, electrolyte disturbances, and blood-derived leukocytes which are migrating across the blood–brain barrier [11, 20–24]. The resulting complex neuro-inflammatory network leads to a pro-inflammatory environment with brain edema and brain tissue destruction by leukocyte-released proteases, lipases, and reactive oxygen species. In addition, these events culminate in the breakdown of the blood–brain barrier and allow neurotoxic circulating molecules to enter the brain. As a result, the traumatized brain is highly susceptible to secondary injuries caused by intracerebral inflammation, as well as systemic neurotoxic molecules, which are normally "blocked" under physiological conditions (Fig. 6.1).

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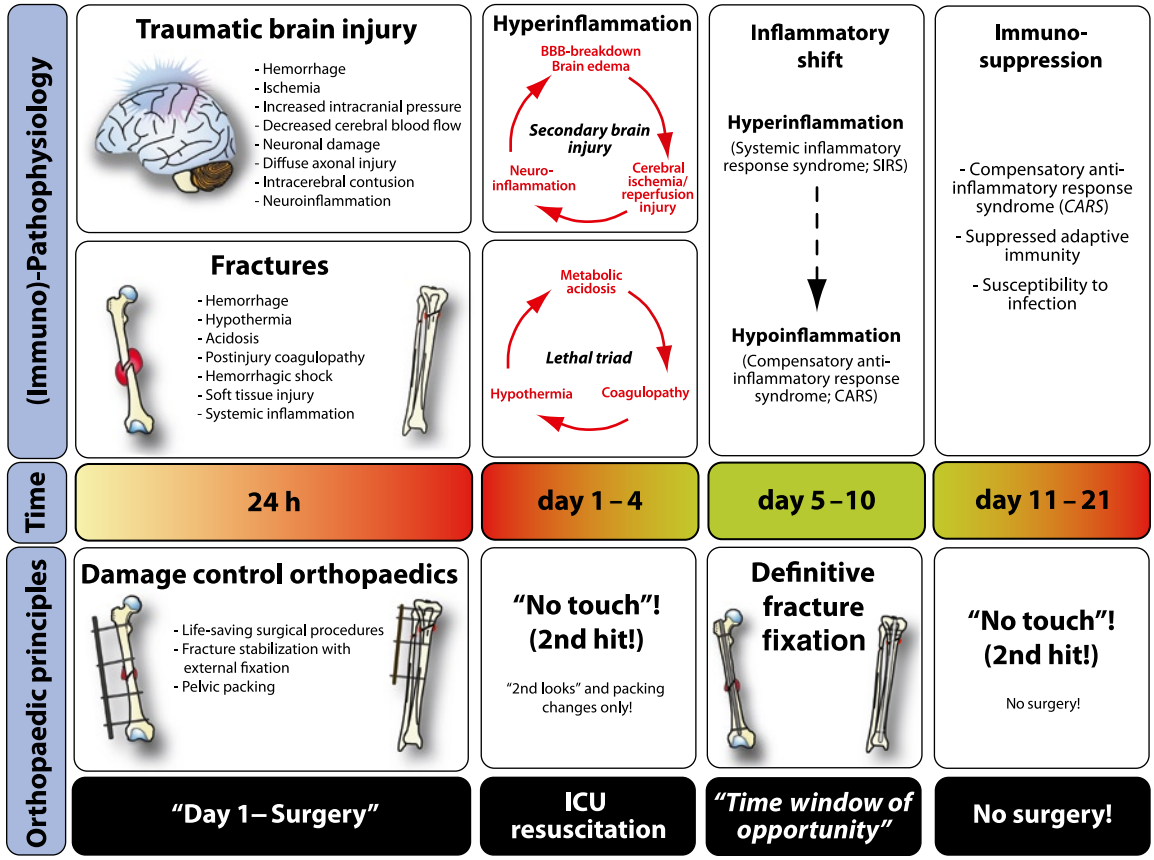

Fig. 6.1 Schematic of priorities in the management of associated orthopedic injuries in patients with severe head injuries, based on the understanding of the underlying immunological pathophysiology

In TBI patients who have sustained concomitant major trauma to the musculoskeletal system, a profound *systemic* inflammatory response is also triggered in parallel, involving cytokines/chemokines, complement activation products, the coagulation system, stress hormones, neuronal signaling, and numerous inflammatory cells [25–30]. To date, we have an incomplete understanding of how the cerebral and systemic inflammatory responses interact and inter-communicate with each other, and whether there is "spill-over" from the intracerebral into the systemic "compartment" and vice versa, exacerbating the inflammatory response.

Consequently, the treating surgeon must be aware of the neuropathology of TBI as well as the systemic inflammatory events when deciding on the optimal management approach in this vulnerable patient population, as inappropriate treatment may result in an iatrogenic secondary insult to the brain [14, 31].

6.2 Clinical Assessment of TBI

On hospital admission, all head-injured patients are systemically assessed and resuscitated according to the American College of Surgeons' *Advanced Trauma Life Support* (ATLS®) protocol. Closed head injury is typically diagnosed by (1) the history of trauma, (2) the clinical status, and (3) computed tomography (CT) scan. The neurologic status is assessed after stabilization of vital functions [17]. The level of consciousness is rapidly evaluated by the Glasgow Coma Scale

(GCS), which grades the severity of TBI as mild (GCS 14/15), moderate (GCS 9–13), and severe (GCS 3–8) [17]. The post-resuscitation GCS score is of clinical importance due to the significant correlation with patient outcome [32]. A head CT should be obtained under the following circumstances: (1) altered level of consciousness with GCS <14 (moderate or severe brain injury), (2) abnormal neurological status, (3) differences in pupil size or reactivity, (4) suspected skull fracture, (5) intoxicated patients. The CT should be repeated whenever the patient's neurologic status deteriorates [17]. During the initial management of TBI, hypoxemia, hypotension, hypercarbia, and hypoglycemia must be avoided or rapidly corrected to minimize the development of secondary brain damage [17]. Hemodynamic stability should be maintained using an isotonic electrolyte solution [33]. Maintenance of an adequate cerebral perfusion pressure (CPP = mean arterial pressure [MAP] – intracranial pressure [ICP]) above 70–80 mmHg is recommended in the early phase post trauma [17, 34, 35]. Cerebral edema should be rapidly addressed using osmotic drugs, and intracranial volume may be reduced by draining cerebrospinal fluid (CSF) via intra-ventricular catheters, or surgical hematoma evacuation.

Augmentation of the intravascular volume with osmotic therapeutics, such as mannitol, results in a transient increase in MAP and CPP and induces cerebral vasoconstriction, reducing the intracranial volume [17]. Osmotic therapy using mannitol or hypertonic saline is currently recommended if the patient displays clinical signs of trans-tentorial herniation, progressive neurological deterioration, or bilaterally dilated and nonreactive pupils [17]. Results of the Corticosteroid Randomization after Significant Head Injury (CRASH) trial have made the use of gluco-corticoids obsolete and contrast indicated in severely head-injured patients [36, 37].

Intra-ventricular catheters are used to monitor intracranial pressures and allow CSF drainage to decrease the ICP [35]. Current guidelines recommend continuous ICP monitoring in patients (1) with *severe* head injury (GCS < 9) and abnormal admission CT scan; (2) with *severe* head injury (GCS < 9) and normal initial CT scan, but with a prolonged coma >6 h; (3) requiring evacuation of intracranial hematomas; (4) with neurological deterioration (GCS < 9) in patients with initially mild or moderate head injury; and (5) head-injured patients requiring prolonged mechanical ventilation, unless the initial CT scan is normal [17, 34, 38, 39].

Craniotomy with evacuation of intracranial mass lesion must be undertaken as soon as possible in patients with clinically relevant and surgically accessible hematomas [92]. Elevated intracranial pressure is one of the most common causes of death and disability in the severely brain-injured patient. When intracranial hypertension is refractory to medical treatment, many patients undergo decompressive craniectomy. However, the indication, timing, and method of decompressive craniectomy are not well defined [40, 41]. Recent literature suggests that hinge craniectomy achieves similar outcomes with respect to ICP control and early outcomes when compared to standard decompressive craniectomy [40]. An additional benefit of this modality is that patients will not have to undergo a delayed cranioplasty for skull defect closure.

6.3 Strategies of Fracture Fixation in TBI Patients

Choosing the ideal timing and modality of fracture fixation in head-injured patients is of paramount importance to avoid a detrimental "second hit" injury to the brain [14, 42]. However, selecting a "safe" treatment modality of the multiply injured patient with concomitant TBI can be difficult.

The initial assessment and management of any trauma patient with TBI should follow the ATLS® algorithm [25, 43]. Hypoxemia and hypotension, the "lethal duo of TBI" [44], need to be avoided at all times, as both will exacerbate post-traumatic edema with potentially detrimental consequences [33]. Adequate oxygenation, appropriate fluid resuscitation, and maintenance of the CCP above 70 mmHg are of paramount importance [33, 45].

To best achieve these goals in multiply injured patients with TBI, modern ventilation strategies and damage control resuscitation principles have been developed, aiming to avoid overly aggressive fluid management exacerbating cerebral edema, respectively [46–50]. Glucose-containing crystalloid fluids should be avoided in TBI patients, as hyperglycemia induces local acidosis and oxidative stress, promotes edema formation, impairs nitric oxide-mediated vasodilation, and

triggers inflammation [51–53]. Arterial blood glucose values between 6 and 8 mmol/L are currently considered ideal in patients with severe TBI [54].

During the first 24 h post trauma, any unnecessary surgical interventions may negatively alter the patient's MAP and lower the CCP [55]. As a result, surgical intervention should be limited to emergency procedures and "damage control" procedures for hemodynamically unstable patients in extremis, such as emergent laparotomy, pelvic external fixation, and pelvic packing [25, 43, 55].

Definitive fracture management in traction is currently considered obsolete, as any non-stabilized fracture greatly increases the "antigenic load" (pain and stress) of trauma. Such a management approach exacerbates systemic inflammation and intracranial edema, and allows fat emboli from the fracture site to circulate, aggravating the patient's secondary brain injuries [25, 26, 56, 57]. Moreover, the lack of adequate positioning and mobilization put the patient at risk for pressure sores, thrombo-embolic events, and pulmonary complications. Thus, modern treatment strategies favor early fracture fixation, to reduce the "antigenic load," allow early mobilization and adequate positioning options in the ICU, and help prevent pulmonary complications [43, 58]. Nevertheless, the ideal timing of definitive femur fixation remains a topic of lively debate and no authoritative, evidence-based guidelines exist [59, 60].

To date, there are two conflicting philosophies for the ideal timing of operative fracture fixation once hemodynamic stabilization is achieved and trauma-induced neuropathology is addressed. Several groups favor an "early total care" approach with immediate definitive fracture fixation within the first 24 h [64]. Others advocate the staged "damage control" notion that supports immediate temporary fracture stabilization by means of external fixation and delayed definitive fracture fixation once resuscitation endpoints are met [43, 86, 91]. This operative "window of opportunity" for definitive fracture fixation is currently considered to be between day 5 and 10 after injury [25, 26, 43, 56]. However, the pertinent literature fails to identify an evidence-based ideal strategy for the optimal time-point of definitive fracture fixation in patients with associated TBI. Several groups report that timing of definitive fracture fixation in patients with TBI and associated femoral fractures does not influence morbidity, mortality, or neurological outcome [61–67].

Authors advocating the "early total care" concept report decreased mortality and superior outcome if definitive fracture fixation occurs within the first 24–48 h of admission [58, 68], even in the co-presence of associated combined chest and head injuries [69, 70]. In addition, the incidence of complications and pulmonary morbidities, and ICU length of stay were all found to be reduced when patients were definitively treated within 24 h [58, 71–74].

In contrast, supporters of "damage control orthopaedics" (DCO) base their surgical decision making on a patho-physiological standpoint, and consider the inadequately resuscitated patient already in jeopardy of the "lethal triad," i.e., metabolic acidosis, hypothermia, and coagulopathy [75]. As a result, DCO supports a staged surgical approach in TBI patients with associated fractures, with immediate external stabilization as a bridge toward eventual definitive fixation during the "window of opportunity" once the patient is hemodynamically stable, fully resuscitated, and the hyper-inflammatory response subsides [31, 43, 55, 76]. Furthermore, advocates of DCO claim that an "early total care" approach prolongs operative times, significantly increases intracranial pressures [77, 78], decreases cerebral perfusion pressures [79–81], and causes cerebral micro-emboli due to immediate fracture care [82]. These patho-physiological changes may result in an iatrogenic amplification of secondary brain injury [77, 79, 80, 83, 84]. Moreover, early intramedullary fracture fixation creates a higher pulmonary embolic load than primary external fixation [85], and may cause serious pulmonary complications [86, 87]. Based on these observations, the DCO concept favors immediate temporary stabilization <24 h via external fixation, early transfer to intensive care, hemodynamic stabilization, and acute resuscitation [31, 55]. Definitive surgical fracture fixation is then planned on an elective basis once the patient meets the following "endpoints of resuscitation" [43]:

- Stable hemodynamics without the need for vasoactive or inotropic stimulation
- No hypoxemia or hypercapnia
- Serum lactate < 2.5 mmol/L
- Normal coagulation (INR, TEG)
- Normothermia
- Normal urinary output (>1 mL/kg/h)

These parameters are usually reached within the "window of opportunity" (day 5–10 post trauma), and favorable results have been described when external

fixation is converted to definitive fracture fixation within the first 2 weeks [88].

6.4 Conclusion

Head-injured patients with concomitant fractures represent a highly vulnerable patient population. An initially undiagnosed TBI can result in secondary evolution of intracranial hemorrhages or cerebral edema, as exemplified by the patient who "talks and dies" [17]. Involving multiple disciplines, including trauma surgeons, neurosurgeons, ICU anesthetists, as well as orthopaedic trauma surgeons allows for a best possible outcome. The current literature is conflicting and fails to identify an unequivocal management strategy [89, 90], underscoring the pressing need for large, randomized multicenter trails to evaluate the concept of DCO in TBI patients with fractures vs. "early total care."

When the patient with combined orthopedic and neurosurgical injuries is evaluated in the emergency department, several questions need to be answered. A rapid neurologic exam should be performed to assess the severity of brain injury. If the patient is hemodynamically stable, then a non-contrast CT brain should be performed. If the patient is hemodynamically unstable, then an ICP monitor (either fiberoptic or ventricular) may be placed in the ED. In the patient with lateralizing signs, an air ventriculogram may be helpful to evaluate for mass lesions (may be performed in the ED or OR).

Any patient with a suspected brain injury who needs to be taken to the operating room and will be unable to undergo follow up neurologic examination requires ICP monitoring. The exact ICP threshold of when not to proceed to the operating room is unknown, though sustained pressures beyond 15 mmHg should be an indication to proceed to the ICU for resuscitation. Any patient with a progressively worsening neurological exam is also at high risk as is the patient with unexplained changes in ICP. Hypoxia and hypotension significantly increase mortality in the patient with brain injury.

Until clear-cut, evidence-based recommendations are established, the clinical approach needs to be based on knowledge of physiology, logic and the accumulated experience. We recommend the following management strategy for orthopaedic injuries in head-injured patients [91]:

1. *Early total care* in all patients with *mild* TBI (GCS 14/15 points), and normal craniocerebral CT scan, unless IMN contraindicated (e.g., severe chest injury, traumatic-hemorrhagic shock, etc.).
2. *Damage control orthopaedics* by means of external femur fixation in all patients with *severe* TBI (GCS ≤8 points, presence of significant intracranial pathology on CT scan, such as edema, midline shift, sub-/epidural bleeding, open head injury with intracranial air). Concomitant procedures may be performed (emergency craniotomy at same time as DCO).
3. Consider *damage control orthopaedics* in all patients with *moderate* TBI (GCS 9–13 points), or patients with GCS of 14 or 15 with "minor" intracranial pathology on CT scan (e.g., traumatic subarachnoid bleeding, extra-axial hematomas that warrant observation only).
4. No additional operation for patients with refractory intracranial hypertension or unexplained change in neurologic exam,
5. *Conversion from external to internal fixation* in TBI patients who recovered from a comatose state and are awake and alert (GCS >12) or comatose patients with a stable ICP (<20 mmHg) and CPP (>80 mmHg) for more than 48 h.

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