Common Sequelae of Severe Traumatic Brain Injury: A Case Report

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ABSTRACT

Severe traumatic brain injuries (TBI) are caused by external forces that damage brain tissue and result in prolonged cognitive, sensory and motor deficits. This case report highlights the neurocognitive, motor, and sensory results of severe traumatic brain injury as well as several sequelae that often complicate TBI.

KEYWORDS: Brain Injury; TBI; Diffuse Axonal Injury; Heterotopic ossification; Post-traumatic Amnesia

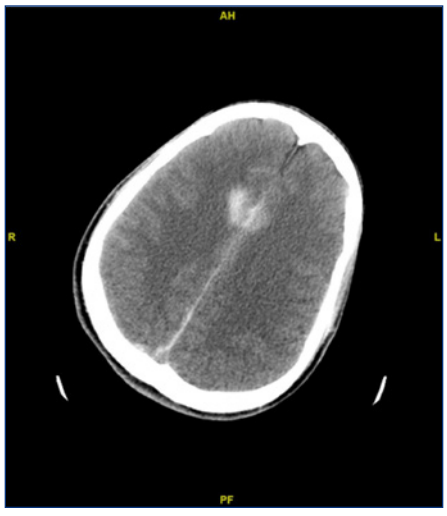
INTRODUCTION

Traumatic Brain Injuries (TBI) exist on a spectrum of severity from mild TBI/concussion with time-limited symptoms to severe traumatic brain injury with long-term, significant cognitive, motor, and sensory deficits. Severe traumatic brain injury results from an external force applied to the brain and is defined by one or more of the following criteria: loss of consciousness for greater than 24 hours, initial Glasgow Coma Scale (GCS) of 3–8, and an episode of post-traumatic amnesia lasting greater than seven days. Additionally, the clinical course of these patients usually involves complications, including dysphagia, prolonged respiratory failure, heterotopic ossification, agitation and restlessness, and concomitant orthopedic injuries. The following case describes the complex medical management and complications of a patient with a severe TBI.

CASE PRESENTATION

A 40-year-old man was the helmeted rider of a motorcycle that crashed into a car at highway speeds. His GCS in the field was 6–7, and he was intubated on arrival at the ED. Imaging demonstrated a subarachnoid hemorrhage, subdural hematoma, intraparenchymal contusions [**Figure 1**], facial fractures, bilateral rib fractures, pneumothorax, left hemothorax, left scapular fracture, right cervical ICA dissection, right scaphoid fracture, and left perilunate dislocation. Five days later, an MRI scan of the brain redemonstrated multifocal intraparenchymal hematomas, notably in the rostral corpus callosum, frontal lobes, and anterior (right greater than left) temporal lobes [**Figures 2**,**3**]. Additional findings included multifocal microhemorrhages and diffusion-restricting foci that were consistent with diffuse axonal injury (DAI) [**Figure 4**].

Figure 1. Non-Contrast CT Brain demonstrating Intraparenchymal hemorrhage in the bilateral medial frontal lobe and parafalcine subarachnoid hemorrhage.



He required a tracheostomy for prolonged endotracheal intubation. Due to severe dysphagia, a PEG tube was placed to allow enteral nutrition. His acute hospital course included multiple surgical procedures, including stabilization of facial fractures, open reduction and internal fixation (ORIF) of the right scaphoid fracture, and pinning of the left fifth metacarpal. His early acute course was notable for ongoing decreased alertness, and he was started on amantadine.[1] He developed significant psychomotor agitation, for which he started on propranolol. He had movement of the right upper and lower extremities but limited spontaneous movement on the left. With range of motion during therapy, he demonstrated significant pain with passive movement of his left upper extremity, especially at the elbow. Further evaluation revealed elevated alkaline phosphatase and heterotopic ossification (HO) in his left distal triceps on plain radiograph [**Figure 5**]. He started indomethacin[2] but did not tolerate a full course because of GI side effects.

Approximately 1.5 months following his injury, he was following simple commands but remained disoriented and had limited verbal output. Two months following his injury, he was able to vocalize spontaneously and follow simple one-step commands consistently. Approximately 2.5 months after his injury, he was transferred for acute inpatient rehabilitation to focus on intensive therapy with ongoing management of his medical sequelae.

The patient made slow but consistent progress, with improved movement of his left side, verbal output, carryover of commands, memory, and

Figure 2. MRI brain demonstrating FLAIR hyperintensity

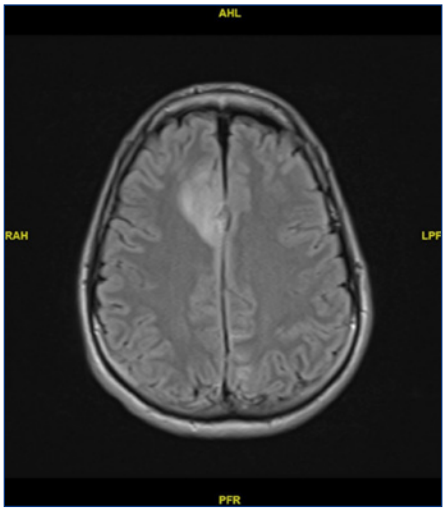


Figure 3. Intraparenchymal hemorrhage (SWI sequence which highlights areas of hemosiderin deposition) in the right medial frontal lobe.

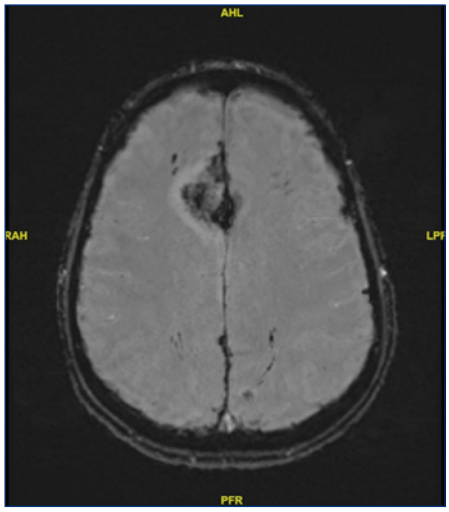
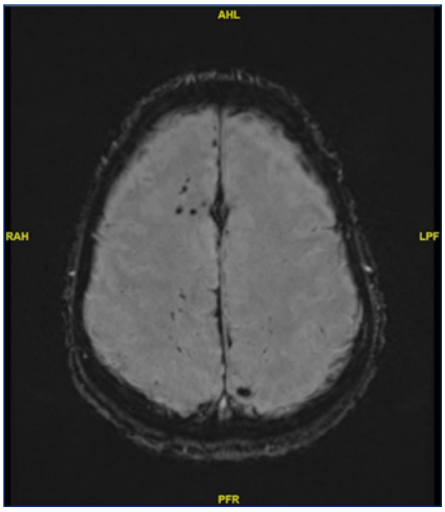


Figure 4. SWI MRI images with microhemorrhages in the bilateral cerebral hemispheres, consistent with diffuse axonal injury.

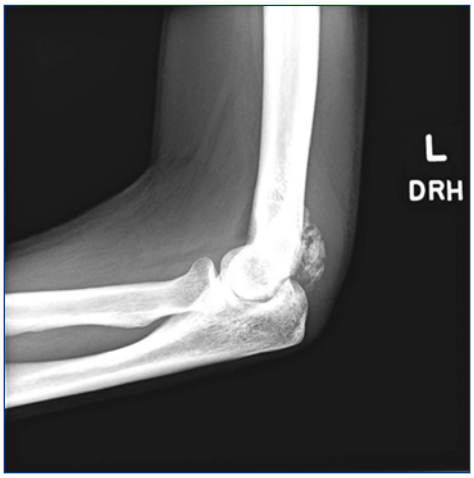


orientation. He emerged from post-traumatic amnesia approximately three months after his initial injury, evidenced by consistent orientation. His dysphagia was mainly characterized by oral apraxia, but he progressed to an oral diet; he improved to the point of meeting his nutritional needs, and his PEG tube was removed prior to discharge. Propranolol was weaned off as his psychomotor agitation improved with improving cognition. During his inpatient rehabilitation, he improved his ambulation; at the time of discharge, he needed contact guard for stand-pivot transfers and ambulation up to 150+ feet with a platform rolling walker. He continued to need minimal assistance for ADLs, including bathing and dressing. Throughout his rehabilitation course, he had significant support from his wife, who was able to provide assistance at home. She decreased barriers to his home discharge by installing a ramp at an entrance to their home and grab bars in the bathroom to improve his mobility and safety. He was eventually discharged home with his wife approximately five months following his injury.

Due to ongoing focal weakness in his left upper extremity and localized sensory loss in the ulnar aspect of the hand, there was concern for a peripheral nerve injury at the brachial plexus or ulnar nerve.

Electromyography and nerve conduction studies (EMG/NCV) demonstrated significant ulnar nerve injury, with evidence of axonal injury and demyelination localized at the elbow, which has been associated with heterotopic ossification in this location.[3] His home therapy focused on improving his independence with ADLs, fine motor function, balance, and ambulation.

Figure 5. Plain radiograph of the left elbow, with a focus of heterotopic ossification in the distal triceps.



DISCUSSION

This case report highlights a typical recovery course of a patient with a severe TBI. His neurologic imaging displayed focal structural disruptions reflected in intraparenchymal hemorrhage/contusions as well as cellular neuronal damage, or diffuse axonal injury. These different primary neurologic injuries lead to different functional outcomes. For example, focal contusions in the parietal lobe may cause hemisensory loss and frontal lobe contusions typically lead to disinhibition. Conversely, diffuse axonal injury often leads to more global symptoms such as slowed processing time, post-traumatic amnesia, or disorders of consciousness including coma and minimally conscious state.[4] This case highlights the positive outcomes but prolonged recovery after TBI, as this patient's total hospitalization was approximately four months from admission to discharge home.

This case also highlights some common sequelae of severe TBI, including dysphagia, prolonged respiratory failure, agitation and restlessness, and concomitant orthopedic injuries. Rehabilitation for TBI focuses on symptom management to allow optimal participation in physical, occupational, and speech therapy as well as maximum functional recovery.

References

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Disclosures

None

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