Management of Medical Complications During the Rehabilitation of Moderate-Severe Traumatic Brain Injury

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KEYWORDS

• Traumatic brain injury • Rehabilitation • Complications • Agitation • Neuroendocrine • Spasticity • Post-traumatic hydrocephalus • Paroxysmal sympathetic hyperreflexia

KEY POINTS

- Medical complications after moderate-severe traumatic brain injury (TBI) are common and should be considered by the brain injury specialist in all patients.
- Many similar signs and symptoms exist for medical complications after TBI, making an understanding of differential diagnosis essential in ensuring appropriate treatment.
- Medical complications after moderate-severe TBI include posttraumatic seizures, paroxysmal sympathetic hyperactivity, spasticity, hydrocephalus, agitation, neuroendocrine dysfunction, heterotopic ossification, venous thromboembolism, and cranial nerve (CN) dysfunction.
- Caregiver support and education is essential during the acute and subacute period of time after a moderate-severe TBI.

Traumatic brain injury (TBI) in the United States is well publicized for the potential long-term effects. With incidence of TBI as defined by emergency department visits, hospitalization, and deaths rising ongoing attention from public and private organizations continues. These data show increasing rates of brain injury driven by emergency department visits gaining 56% from 2007 to 2010.[1] The rates of TBI-related hospitalization have been relatively stable over that period of time and associated deaths have decreased. It is postulated that deaths have decreased owing to a focus on primary prevention and improving acute management.

The management of moderate-severe TBI in the acute care setting is shared by critical care physicians, neurosurgeons, trauma surgeons, neurologists, and physiatrists.

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The initial goals of acute care management are prevention of the secondary injury by surgical management, management of intracranial pressures, respiratory support, and management of concurrent injuries. The other providers typically manage these conditions while the brain injury rehabilitation specialist assists in management of many other brain injury–specific complications in the acute and subacute periods. Various domains including physical, cognitive, behavioral, and somatic can be involved.

Once the patient is stabilized medically after the brain injury, transitions to the next level of care are planned. Despite decreasing numbers of patients post-TBI transferring to acute rehabilitation in the postprospective payment system,[2] acute inpatient rehabilitation is a consideration for all patients after moderate-severe TBI. Management of medical complications after TBI continues in this setting as well. This process can include the management of posttraumatic seizures, paroxysmal sympathetic hyperactivity, spasticity, hydrocephalus, agitation, neuroendocrine dysfunction, heterotopic ossification, venous thromboembolism, and CN dysfunction.

POSTTRAUMATIC SEIZURES

Posttraumatic seizures and posttraumatic epilepsy can develop after TBI with an incidence described between 4% and 53%.[3] Risk factors for seizure development include hydrocephalus, intracranial hemorrhage, depressed skull fracture, surgical hematoma evacuations, lower Glasgow Coma Scale levels, dural penetration, parietal lesions, and focal neurologic deficits.[4–6] Additionally, late seizures—those that develop after day 7 post injury—are also associated with prolonged duration of posttraumatic amnesia and a lower Glasgow Coma score. Late seizures are defined as those seizures arising after day 7. Immediate and early seizures are described as in the first 24 hours and between 24 hours through 7 days, respectively. Classic research showed that seizure prophylaxis beyond the first 7 days does not add any additional benefit in the prevention of late posttraumatic seizures. Traditionally, phenytoin was used based on the classic studies[7]; however, recently other medications were shown to have equal/better seizure control.[8–11] Late development of seizures after TBI continues at a higher rate than the general population. Based on the veterans of the Vietnam era, this increased incidence continues decades later.[12] The treatment of seizures is recommended with the development of early and late seizures. First-line treatment is antiepileptic medication; however, for medication-refractory seizures, surgical resection of the seizure focus and vagal nerve stimulators have also been described. There is lack of consensus of timing of medication discontinuation. Withdrawal of antiepileptics after seizure development is typically delayed 1 to 2 years after the last seizure. Electroencephalography can provide more information and it is reasonable to include neurologists in this decision.

PAROXYSMAL SYMPATHETIC HYPERACTIVITY

Paroxysmal sympathetic hyperactivity is thought to result from uninhibited sympathetic outflow after a central nervous symptom insult.[13] It has been described after stroke, anoxic brain injury, and encephalitis, in addition to TBI. First described in 1954 as an "autonomic seizure,"[14] more than 30 terms for this condition have been found in review of the literature, including sympathetic storming and dysautonomia.[15] Not surprisingly, definitions and diagnostic criteria also vary and recommendations have been made to more clearly define this syndrome.[16] In general, it is agreed that the diagnosis is based on paroxysmal cycling of agitation/dystonia in association with autonomic symptoms including tachycardia, tachypnea, elevation in systolic blood pressure, hyperthermia, and diaphoresis occurring for at least 3 consecutive days, 2 weeks or greater after