This commentary discusses traumatic brain injury (TBI) related to falls among elderly individuals, as well as common TBI sequelae and their treatment. It also discusses the current understanding of TBI-related dementia and chronic traumatic encephalopathy.

Falls are responsible for more than one-third of all traumatic brain injuries (TBIs) within the general population and more than 60% of all TBIs among people older than 65 years [1]. Fall-related TBIs among elderly individuals are responsible for an estimated 141,998 emergency department visits, 81,500 hospitalizations, and 14,347 deaths in the United States each year. Many more people are treated in the primary care setting or do not seek treatment. Patients over age 75 years have the highest rates of both TBI-related hospitalization and death [1]. This is a significant public health concern as the nation’s elderly population continues to increase. It also represents a challenge for clinicians across the spectrum of care, as patients with TBI are frequently encountered in the emergency department and by providers in primary care, surgical specialties, nonsurgical specialties, allied health, and neuropsychology. Thus nearly all medical professionals need to have a robust understanding of fall-related TBI care.

The evaluation and diagnosis of mild TBI, in particular, is challenging in older persons, especially those with pre-existing dementia or cognitive disorders. Because mild TBI includes any loss or alteration of consciousness, a pre-existing alteration in consciousness may obfuscate diagnosis. TBI should be high on the differential diagnosis when there is a significant mechanism or evidence of head injury, such as bruising, lacerations, or facial bone fractures. Conversely, common symptoms of brain injury—such as balance impairment, depression, and cognitive deficits—may be misattributed to other causes, especially when elderly patients experience a fall that was not witnessed by anyone [2]. The frequent use of anticoagulants for comorbid conditions in this population leads to an increased risk of hemorrhage, even with low-velocity head trauma. A high index of suspicion for intracranial hemorrhage is thus warranted, as these patients may present with vague neurological complaints and a normal physical examination. Some patients may present days or weeks after trauma, such as may occur with subdural hemorrhage.

Initial Assessment of Brain Injury and Indications for Imaging

The initial assessment of brain injury must focus on patient stabilization and the prevention of secondary complications. Increased intracranial pressure due to bleeding or edema can lead to cerebral anoxia or infarct. These complications can often be prevented with careful clinical examination, imaging, and the appropriate use of neurosurgical interventions.

Computed tomography (CT) remains the most useful initial imaging tool due to its sensitivity for acute hemorrhage and fractures. While CT lags behind magnetic resonance imaging (MRI) in sensitivity for some findings, such as the microhemorrhages seen in diffuse axonal injury, CT’s speed and ability to detect lesions requiring neurosurgical intervention make it the preferred initial imaging modality. It is well accepted that patients with a Glasgow Coma Scale (GCS) score of 13 or less require a CT scan after known head trauma. For patients with mild TBI and a GCS score of 14 or 15 on arrival to the emergency department, the 2008 guidelines from the American College of Emergency Physicians and the Centers for Disease Control and Prevention are often used to determine the need for imaging [3]. According to these guidelines, any patient over 60 years of age should receive a head CT scan if they experienced loss of consciousness or post-traumatic amnesia. Imaging should also be considered for patients older than 65 years of age, even in the absence of loss of consciousness. Clinicians also need to consider the presence of neurological deficit, vomiting, severe headache, GCS score less than 15, physical signs of basilar skull fracture, coagulopathy, or a dangerous mechanism of injury (including ejection from motor vehicle, struck pedestrian, and fall from a height greater than 3 feet or...
5 stairs). In an Australian study of hospitalizations related to TBI, the most likely initial imaging finding was subdural hemorrhage (42.9%). Traumatic subarachnoid hemorrhage was seen in 12.7% of patients, and concussive injury was noted in 24.1% [4].

It should be noted that a normal CT scan cannot completely rule out extra-axial hemorrhage or a structural brain injury. MRI with susceptibility weighted images and diffusion tensor imaging are more sensitive for hemorrhagic and non-hemorrhagic axonal injury, respectively [5]. Furthermore, ongoing research is needed to establish the role of functional neuroimaging as well as that of serum biomarkers.

**Rehabilitation of Elderly Patients with TBI**

Common symptoms following TBI include cognitive impairment, fatigue, vestibular disequilibrium, sleep-wake
cycle impairment, psychiatric and behavioral disturbances, headaches, and vision and hearing changes. More severe TBI may also be associated with weakness, muscle spasticity, dysphagia, seizures, speech impairment, and hormone and electrolyte changes. In addition, it is common for patients with severe TBI to experience confusion, agitation, and combativeness—particularly when emerging from a comatose state. Secondary complications can include cerebral infarct due to increased intracranial pressure, hydrocephalus, and injuries from additional falls.

Management of symptoms following TBI presents unique challenges in elderly patients. For example, tricyclic antidepressants—which are commonly used in the treatment of post-traumatic headache—may lead to complications in elderly patients due to anticholinergic side effects. Likewise, medications commonly used to treat muscle spasticity may lead to sedation. In fact, elderly patients with TBI should be presumed to be more sensitive to any centrally acting medication. In particular, benzodiazepines and typical antipsychotics such as haloperidol should be avoided due to evidence that they impair recovery from TBI [6].

As budgetary constraints sometimes preclude one-on-one care in hospitals and nursing facilities, there is danger that potentially harmful sedatives will be used as a means of behavioral control. The use of enclosure beds and patient restraints are appropriate in some cases, but they are wrought with ethical concerns. An alternative is hospital sitters, who stay with patients and can help to keep them calm, but we lack evidence-based guidelines to support the use of such personnel. Several studies have failed to show that hospital sitters prevent falls or are cost-effective, and assigning hospital sitters to elderly patients may contribute to staffing shortages in other areas of the unit [7]. However, the question of whether hospital sitters are beneficial needs to be addressed in the brain injury population, especially because symptoms of confusion, restlessness, and poor balance are often present in patients with minimal strength impairment. Clinicians should not underestimate the value of a trusted family member’s presence in the redirection of an agitated patient, and facility visitation policies should allow spouses and other visitors to fulfill this role.

Fall prevention is of paramount importance in the recovery from TBI. This is of even greater consequence among patients with intracranial hemorrhage or recent cranectomy, in whom a repeat fall can be disastrous. In addition to reducing polypharmacy, it is important to address any physical weakness, coordination impairment, or vestibular dysfunction with an appropriately tailored physical therapy program. Symptoms of orthostasis and the presence of comorbidities such as polyneuropathy, vision impairment, or benign paroxysmal positional vertigo must also be considered and addressed. Home assessment and modifications may be indicated near the time of discharge.

Given the complex medical, behavioral, physical, and cognitive sequelae of TBI, a multidisciplinary approach to treatment is often indicated. Acute inpatient rehabilitation facilities offer physical therapy, occupational therapy, speech therapy, rehabilitation nursing, and neuropsychological services. However, changes in Medicare reimbursement have resulted in stricter admission standards for acute inpatient rehabilitation facilities and have increasingly emphasized reducing length of stay and discharging patients home from the acute inpatient rehabilitation setting.

In comparing 2 studies from the TBI Model Systems Project over the 10-year period 1996–2006, average length of stay in an inpatient rehabilitation facility for older patients with TBI (mean age of 67 years) plunged by nearly 70%, from 56 days to 19 days. Patients in the earlier study were admitted with lower overall functional status as measured by the Functional Independence Measure (FIM) scale, and they were discharged with higher functional status (see Table 1). Presumably, many of these lower-functioning patients are now discharged directly to nursing facilities, where they receive less therapy, nursing care, and physician oversight [8, 9]. The impact of these changes on patient outcomes is unknown, and more research is needed to better guide these practices and policies.

**Dementia, Chronic Traumatic Encephalopathy, and Chronic Sequelae of TBI**

A growing body of evidence shows that there is a correlation between prior brain injury and subsequent development of dementia, but interpretation of these data is often difficult due to the presence of covariates such as drug and alcohol abuse or underlying medical conditions. Nonetheless, it appears that prior brain injury is a risk factor for dementia, with evidence of both higher frequency and younger onset. A recent cohort study of 825,816 men completing mandatory Swedish military service showed that a history of TBI increased risk of young-onset dementia at an average follow-up time of 33 years; this correlation was present even after controlling for covariates such as premorbid cognitive function, alcohol intoxication, blood pressure, depression, and socioeconomic status [10].

Another recent study in California compared TBI patients age 55 years and older versus non-TBI trauma patients who presented to emergency departments. They found that

| TABLE 1. Changing Trends in Inpatient Rehabilitation for Older Adults With Traumatic Brain Injury |
|---------------------------------------------|----------|----------|
| Mean age, in years (SD)                    | 1996* (n = 50) | 2006* (n = 267) |
| Rehabilitation length of stay, in days     | 66.8 (9.2)    | 66.6 (9.0)    |
| Admission FIM                              | 56.0       | 18.8       |
| Discharge FIM                              | 52.2       | 53.6       |
| Discharge FIM                              | 90.6       | 87.8       |
| Percent of patients discharged to community| 82%        | 81%        |

*Data are from Cifu et al [8].
*Data are from Frankel et al [9].
the TBI patients were more likely than the non-TBI control group to have developed dementia after an average follow-up period of 6 years (8.4% versus 5.9%, respectively). This suggests that elderly individuals may be more susceptible to the sequelae of mild brain injury [11].

The mechanism of chronic cognitive decline has yet to be proven, and it is unclear whether brain injury initiates a neurodegenerative process or if neuronal loss from the initial injury diminishes cognitive reserve. Pathology studies of TBI-related dementia and chronic traumatic encephalopathy (CTE) may offer some hints as to the etiology of these changes. However, specific criteria for the pathological diagnosis of CTE have not yet been defined. Brain atrophy is often reported, but it has not been possible to distinguish between acute brain injury sequelae and progressive neuron loss. Deposition of tau protein neurofibrillary tangles, amyloid beta, and TAR DNA-binding protein 43 has been observed [12]. These neuropathological features are present in a variety of dementias and neurodegenerative disorders, but it remains to be seen whether this reflects shared pathogenesis. Clinical diagnosis of CTE is even more ambiguous. However, various neuropsychiatric and behavioral symptoms have been reported, including depression, aggression, emotional lability, poor judgment, and suicidal ideation [12].

Neuropsychological evaluation is important for detection and classification of cognitive and behavioral symptoms in elderly patients. The diagnostic utility of neuropsychological testing is well documented in cases of TBI, but its use for diagnosis of CTE is controversial, as research findings have been inconsistent [13]. Still, neuropsychological testing can be useful for determining the likelihood of other causes of cognitive and behavioral disturbance, such as primary psychiatric illness. Also, neuropsychological testing is useful for clinical decision making, planning, and monitoring of treatment effects. Evaluation of a patient’s cognitive abilities relates to their real-world functioning, and there is evidence that neuropsychological assessment is effective in predicting occupational and psychosocial outcomes following a concussion [14]. In elderly patients, neuropsychological assessment can help clinicians make recommendations regarding driving ability and activities of daily living, and such assessment is recommended when there is concern about cognitive or behavioral disturbances affecting function. If the results of testing suggest a possible diagnosis of CTE or other neurodegenerative disease, then repeat testing will usually be recommended after 6–12 months to monitor for further decline or to check for treatment effects. In addition to identifying cognitive impairments, neuropsychological testing can be useful for identifying psychiatric disturbances such as depression and anxiety, which are common after TBI and are known to affect recovery. Psychotherapy, counseling, and/or psychopharmacological treatments may be recommended.

Conclusions

Falls are the most common cause of TBI among elderly individuals, and they therefore represent a significant public health concern. The treatment of fall-related TBI is complex and requires a multidisciplinary treatment approach. However, appropriate care can result in continued functional independence and discharge back to the community. Ongoing research is needed to elucidate the pathogenesis of TBI-related dementia as well as age-related differences in the sequelae of TBI.

Acknowledgments

Potential conflicts of interest. W.F. and M.H. have no relevant conflicts of interest.

References

1. Faul M, Xu L, Wald MM, Coronado VG. Traumatic Brain Injury in the United States: Emergency Department Visits, Hospitalizations and Deaths 2002–2006. Atlanta, GA: Centers for Disease Control and Prevention, National Center for Injury Prevention and Control; 2010.
2. Levine J, Flanagan SR. Traumatic brain injury in the elderly. In: Zasler ND, Katz DI, Zafonte RD, eds. Brain Injury Medicine: Principles and Practice. 2nd ed. New York, NY: Demos Medical Publishing; 2013:420-433.
3. Jagoda AS, Bazarian JJ, Bruns JJ Jr, et al. Clinical policy: neuroimaging and decision making in adult mild traumatic brain injury in the acute setting. Ann Emerg Med. 2008;52(6):714-748.
4. Harvey LA, Close JC. Traumatic brain injury in older adults: characteristics, causes and consequences. Injury. 2012;43(11):1821-1826.
5. Yuh EL, Gean AD. Structural neuroimaging. In: Zasler ND, Katz DI, Zafonte RD, eds. Brain Injury Medicine: Principles and Practice. 2nd ed. New York, NY: Demos Medical Publishing; 2013:194-217.
6. Flanagan SR, Hibbard MR, Riordan B, Gordon WA. Traumatic brain injury in the elderly: diagnostic and treatment challenges. Clin Geriatr Med. 2006;22(2):449-468.
7. Harding AD. Observation assistants: sitter effectiveness and industry measures. Nurs Econ. 2010;28(5):330-336.
8. Cifu DX, Kreutzer JS, Marwitz JH, Rosenthal M, Englander J, High W. Functional outcomes of older adults with traumatic brain injury: a prospective, multicenter analysis. Arch Phys Med Rehabil. 1996;77(9):883-888.
9. Frankel JE, Marwitz JH, Cifu DX, Kreutzer JS, Englander J, Rosenthal M. Follow-up study of older adults with traumatic brain injury: taking into account decreasing length of stay. Arch Phys Med Rehabil. 2006;87(1):57-62.
10. Nordström P, Michaëlsson K, Gustafson Y, Nordström Å. Traumatic brain injury and young onset dementia: a nationwide cohort study. Ann Neurol. 2014;75(3):374-381.
11. Gardner RC, Burke JF, Nettiksimmons J, Kaup A, Barnes DE, Yaffe K. Dementia risk after traumatic brain injury vs nonbrain trauma: the role of age and severity. JAMA Neurol. 2014;71(12):1490-1497.
12. Smith DH, Johnson VE, Stewart W. Chronic neuropathologies of single and repetitive TBI: substrates of dementia? Nat Rev Neuro. 2013;9(4):211-221.
13. Saulle M, Greenwald BD. Chronic traumatic encephalopathy: a review. Rehabil Res Pract. 2012;2012:816069.
14. Boake C, Mills SR, High WM Jr, et al. Using early neuropsychologic testing to predict long-term productivity outcome from traumatic brain injury. Arch Phys Med Rehabil. 2001;82(6):761-768.