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TRAUMATIC BRAIN INJURY AND THE RISK OF DEMENTIA

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Abstract

Traumatic brain injury (TBI) is common in the geriatric population, creating risk for both immediate and long-term impacts on health. Falls are the most common cause of TBI in seniors. Symptoms of mild TBIs (concussions) may be overlooked by patients, and depending on the severity of the injury, patients may not present to their doctors until significant cumulative damage has been done. TBI is typically, initially a clinical diagnosis — patients can present with a variety of neurocognitive and psychiatric symptoms. In some cases patients may recover completely, although not always. TBI can present with immediate complications such as seizures and intracranial hemorrhage. It can also present with more insidious complications such as subdural hematoma, and it can have chronic complications such as mood disorders or cognitive and behavioural impairment. It is important to recognize that even a single episode of moderate to severe TBI can increase one's risk for various dementias, including Alzheimer's dementia. When managing patients with TBI, it is important to recognize when they need admission to hospital for observation and to assess their cognition and function. As physicians, we must not forget to address safety for independent living as well as driving fitness with our patients. Lastly, it is important to know how the injury occurred and to prevent it from happening again.

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Key Points

- Falls are the most common cause of Traumatic Brain Injury (TBI) in the elderly.
- It is important to document baseline neurocognitive, psychiatric and functional status around the time of injury and be able to track this over time.
- Moderate to severe TBI, even a single episode, increases one's risk for dementias.
- Patients with TBI need to be observed for a minimum of 24 hours and 1-2 days of complete mental and physical rest is strongly recommended.
- Severe TBI and older age is associated with increased mortality and poor outcome.

Introduction

Traumatic brain injury (TBI) varies widely in severity, from mild transient symptoms to needing admission to the intensive care unit. Falls are the number one cause of TBI in the elderly. Approximately 50% of those 65 years old and above will experience at least one fall-related injury annually, and up to 60% of falls in the elderly result in TBI[1]. Each year 160,000 Canadians are diagnosed with brain injury[2].

When assessing a patient for TBI, it is not enough to address only the current symptoms of TBI. One must also ask why the trauma occurred in the first place. Reversible conditions such as orthostatic hypotension (or see www.posturalhypotension.ca) and medications that contribute to falls should be identified. For more information on falls see www.stopfalls.ca.

TBI is more than an acute neuropsychiatric illness; it also has long-term implications for one's risk for dementias. In recent years, increasing evidence points to TBI being a cumulative and progressive disease: each trauma adds to the previous injury, and each injury continues to contribute to ongoing brain damage[3].

This article will focus on the diagnosis, treatment and prognosis of TBI in the elderly and its association with various dementias. It will not cover chronic traumatic encephalopathy, which is proposed to be the result of repetitive head trauma commonly seen in professional athletes and younger adults.

Case

Mrs. F is a 78-year-old female who is seeing her family physician because of increased confusion following a fall three weeks ago. This was her first fall although she has had ongoing dizziness, especially when getting up from her bed or out of a chair. This time she fell forward while getting up from her seat after a meal. Mrs. F cannot recall how she fell, but denies a loss of consciousness. She noticed bruises over her forehead and knees. The days after her fall her family noticed that she was confused regarding time and date. She also had trouble recalling conversations. In addition, she seemed more irritable than usual. She also complains that sleep has been worse. She did not seek medical attention right away, because other than a few minor headaches she felt fine. Now her family is concerned because the confusion has not resolved.

Her past medical history is significant for hypertension and type II diabetes (A1c 7.5). She is on low dose ASA, an ACE inhibitor, amlodipine and Seroquel for sleep. She uses zopiclone occasionally.

On physical examination, supine blood pressure is 144/72 with HR 82 and regular. Standing BP is 122/64 with HR 90. Mrs. F complains of dizziness when standing. She has resolving bruises over her forehead and knees with no other signs of trauma. Cardiovascular and respiratory examinations are unremarkable. Neurologic exam reveals normal cranial nerves 2 through 12, normal tone and reflexes and Babinski is downward going bilaterally. Sensation is normal except for decreased pinprick and soft touch over her lower extremities. MOCA is 18/30 with deficits in trails, orientation, serial sevens and recall. Mental status exam reveals a woman who is dressed appropriately and is alert but demonstrates occasional lapses of attention. She reports feeling happy. She appears to be euthymic, but her mood is labile. She does not voice any delusions or report any hallucinations.

What is her diagnosis? Is she at increased risk for developing a dementia? What can you do to lessen her risk of another brain injury?

What is TBI?

The definition of TBI continues to evolve as our understanding of the condition evolves, but it can be understood as neuropathic damage and dysfunction of the brain resulting from a direct or indirect force that is transmitted to the head or body[3]. Symptoms of TBI are both physical and neuropsychiatric.

Table 1. Symptoms of TBI[4]

Neurologic/cognitive	Psychiatric
Loss of consciousness	Anxiety
Amnesia, retrograde and anterograde	Depression
Headache	Irritability
Dizziness/decreased balance	Sleep disturbance, insomnia or somnolence
Vertigo with nausea/emesis	Labile mood
Cortical blindness	Fatigue
Global amnesia	
Noise/light sensitivity	
Inattention	
Language disturbance, e.g., slurred speech	
Impaired short-term memory	
Seizure	

TBI is still mostly a clinical diagnosis based on symptoms, signs and the temporal relation between the timing of the trauma and the presenting symptoms, but neuroimaging can help. CT head is the most commonly used modality, but MRI is much more sensitive at picking up changes associated with TBI. CT is often normal in concussion or mild TBI, but MRI can show small contusions, petechial hemorrhages, axonal injury and small extra-axial hematomas[3]. Traditionally TBI can be further graded as mild, moderate or severe according to Glasgow Coma Scale (GCS) of 13-15, 9-12 and 8 or less respectively. Of note, other grading scales such as Full Outline of Unresponsiveness (FOUR) Score and neuroimaging scales also exist. It is relatively easy to diagnose moderate to severe TBI, where there is physical evidence of trauma to the brain accompanied by symptomatic complaints or impaired level of consciousness, but diagnosing mild TBI can be more challenging. Patients themselves often lack insight into their own injuries due to the injury itself, or choose not to seek medical attention because the symptoms are mild and transient.

The symptoms of TBI vary between patients. This is because injuries to different parts of the brain create different effects. For example, focal injury to the frontal lobe may lead to disinhibition, labile mood and executive impairments including difficulties with attention, information processing speed and even awareness, whereas focal injury to the parietal lobe may lead to disturbed visuospatial perception. It is important to recognize common post-TBI syndromes, such as post-concussion syndrome typically following concussion or mild TBI and deficits following moderate to severe TBI including executive dysfunction syndrome, epilepsy, post-traumatic headache[5] and vertigo.

Pathophysiology – How does TBI impact our brain?

Injuries to the brain can be divided into primary and secondary processes[3]. Primary injury is the direct result from a mechanical force applied to the brain. Secondary injuries, such as those resulting from subdural or subarachnoid hemorrhages, are the developing injuries[3]. These include electrolyte imbalance, mitochondrial dysfunction, neuroinflammation, hypoxic-ischemia, apoptosis and hypometabolism, which lead to diminished blood flow. They in turn result in cerebral edema, raised intracranial pressure and cause axonal swelling[3,6,7,8]. These processes can further be categorized as focal if affecting a localized area of the brain or diffuse if affecting multiple compartments of the brain.

Why is the pathophysiology of interest? It is because the above processes result in similar metabolic changes seen in other progressive neurologic diseases. There is evidence to suggest that TBI promotes the accumulation, misfolding and aggregation of proteins, such as phosphorylated-Tau, beta-amyloid and TDP-43 (tar DNA-binding protein)[4]. These changes are not unique to TBI. Beta-amyloid and p-Tau are seen in Alzheimer's dementia[9], TDP-43 and p-Tau are seen in frontotemporal dementia[10] and TDP-43 is seen in amyotrophic lateral sclerosis (ALS) and Parkinsonism[4]. We know that the above processes lead to progressive brain deterioration. It is therefore not surprising that studies show TBI is associated with degenerative neurologic diseases.

Prognosis and Risk of Dementia

Prognosis is dependent on the severity of TBI and age. Severe TBI may require surgery and ICU admission and tends to have poor prognosis. GCS of 8 or less is associated with 30% mortality. Hypotension and fixed and dilated pupils more than 4 mm are also associated with increased mortality. There is also suggestion that mortality risk remains elevated for at least a decade following hospitalization[6,11]. Of those who survive severe TBI, only 25% achieve long-term functional independence[12]. The risk for poor outcome also sharply rises with age over 60 years old[6]. Both moderate and severe TBIs are associated with permanent cognitive and functional impairments. These neuropsychological symptoms tend to stabilize after 3-12 months from the time of the initial injury; the patient may establish a new cognitive and functional baseline at that time[13].

Different patterns of TBI result in different progressive neurologic diseases. Repetitive minor traumas to the brain, including concussions, are suspected to be associated with chronic traumatic encephalopathy (CTE), as seen in athletes and soldiers. In contrast, a single episode of moderate to severe TBI can lead to earlier onset of Alzheimer's dementia by 2-8 years[14], it can increase the risk of Parkinson's disease[4] and it can lead to earlier onset of frontotemporal dementia[10]. TBI is also associated with ALS, Creutzfeldt-Jakob disease and Parkinsonism[4].

Management

When managing acute mild and moderate TBI, it is important to take a history following any head trauma and to obtain collateral history. One needs to identify immediate neurologic emergencies, such as intracranial hemorrhages and hematomas; recognize and manage neurologic sequelae, such as seizures and mood disorders; and prevent future episodes of TBI. Patients should be advised to have both physical and cognitive rest for at least 1-2 days following their injury to promote recovery. Neuroimaging should be obtained in accordance with the Canadian CT head rule. One should be vigilant about post-traumatic seizures even though it is uncommon in mild to moderate TBIs[16]. Subdural hemorrhage is another sequela that needs to be considered. It can present as an acute, subacute or chronic complication and may require surgical intervention.

Cognitive screening and assessment should be part of your management, since we know that symptoms of TBI can evolve with time and TBI itself is a risk factor for dementia. Neuropsychological testing is the gold standard for assessing baseline cognition once TBI has stabilized. However, it is time consuming, costly and access to a specially trained psychologist may be limited. Other shorter standardized tests are available, but their main purpose is to assess if the patient is still in a confused state. One test, which may be reasonable to use in the clinic setting, is the revised Westmead post-traumatic amnesia scale[17]. This version is quick to

administer, is used in the emergency department and has been found to correlate with findings on neuropsychological testing. The downside is that the studies using this scale involve a much younger population and it is not known how it would be interpreted in those with existing cognitive impairment. It contains 10 questions that test orientation and recall. Any one incorrect answer is considered a positive test[17,18]. Of note, one study excluded those with known neurologic diseases and the other study had a mean age of 28 years old, and it is unlikely that either study had a significant proportion of subjects with underlying cognitive impairment. Therefore, the revised Westmead post-traumatic amnesia scale may not be suitable for the geriatric population with cognitive impairment. There is also evidence, although limited, to suggest that MOCA may be a reasonable alternative test[19]. Regardless of the test of choice, the important point is that there should be a cognitive assessment around the time of injury, so that as symptoms evolve with time, whether they improve or worsen, there is an objective documentation of baseline cognition for comparison. At this time no medication has been proven to be effective in the treatment of chronic cognitive impairment secondary to TBI[20]. However, there is some evidence to support cognitive rehabilitation. A meta-analysis in 2009 by Rohling et al. confirmed that there is a small but significant improvement in attention following cognitive training in those with TBI. Improvements in language and visuospatial domains were also observed, but these were mainly in stroke patients[21]. Whether this evidence can be extrapolated to the geriatric population is questionable, since these studies did not include those above the age of 65.

Patients with mild TBI should be observed for a minimum of 24 hours. It is recommended to admit to hospital for observation those patients with a GCS less than 15, an abnormal CT scan, seizures or a high risk of bleeding (those with bleeding diathesis and those taking anticoagulants)[22]. In the geriatric population, it is important to identify whether TBI is the result of a fall. Falls are often multifactorial (see www.stopfalls.ca). Although oftentimes the etiology or risk factors for the fall cannot be completely resolved, one can still minimize future risks. Medications that can contribute to falls should be decreased in dose or discontinued if possible (see Interventions to Reduce Medication-Related Falls and 4D-AID: A Practical Approach to the Assessment of Orthostatic Hypotension in Older Patients). Furthermore, physicians should determine if the patient is safe to return to independent living both from a physical and cognitive standpoint. Neurologists, rehabilitation medicine specialists, psychiatrists and geriatricians can all aid in the management of TBI.

Should your patient with TBI be driving?

Fitness to drive should be assessed following TBI, since many of the symptoms can impact one's ability to drive. Unfortunately, discussion about relicensing occurs less than 50% of the time following a diagnosis of TBI[23]. One reason may be that there is no one simple test for assessment. A recent European retrospective study by Aslaksen and colleagues showed that a combination of three neuropsychological tests had 82% accuracy in predicting driving fitness[24]. It is not clear, however, whether this result can be extrapolated to a non-European population. Screening tools are being developed in Canada but are not yet available (www.candrive.ca). For now, a complete medical exam, including cognitive assessment and a discussion with the patient, is the best approach. According to the Canadian Medical Association Driver's Guide 9th ed. (PDF free to Canadian Medical Association members), a formal driving evaluation needs to be done if there is any uncertainty in one's ability to drive. Patients can be referred to their local licensing authority for further assessment or be evaluated by an experienced occupational therapist at a rehabilitation facility. Physicians should be clear with patients that they should not drive until further evaluation.

Conclusions

TBI ranges from mild to severe. It may have both physical and cognitive effects on the patient. While it is important to assess for acute management decisions, it is also important to consider disposition of the patient – is the patient safe to be on his/her own, what is the new baseline of ADL and IADL and should the patient be driving. Lastly, TBI is not a condition without long-term sequelae. Survivors of moderate to severe TBI are at risk for dementia and therefore should be followed longitudinally for new cognitive decline.

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