Impact of Single-Photon Emission Computed Tomography/Computed Tomography (SPECT/CT) and Positron Emission Tomography/Computed Tomography (PET/CT) in the Diagnosis of Traumatic Brain Injury (TBI): Case Report

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Traumatic brain injury (TBI) is defined as damage to the brain resulting from an external force. TBI, a global leading cause of death and disability, is associated with serious social, economic, and health problems. In cases of mild-to-moderate brain damage, conventional anatomical imaging modalities may or may not detect the cascade of metabolic changes that have occurred or are occurring at the intracellular level. Functional nuclear medicine imaging and neurophysiological parameters can be used to characterize brain damage, as the former provides direct visualization of brain function, even in the absence of overt behavioral manifestations or anatomical findings. We report the case of a 30-year-old Hispanic male veteran who, after 2 traumatic brain injury events, developed cognitive and neuropsychological problems with no clear etiology in the presence of negative computed tomography (CT) findings. [*P R Health Sci J 2016;35:170-172*]

Key words: Traumatic Brain Injury (TBI), Single-Photon Emission Computed Tomography (SPECT), Positron Emission Tomography (PET), Somatosensory Evoked Potentials (SSEP)

Traumatic brain injury (TBI) is an insult to the brain caused by an external mechanical force (angular, rotational, shear, or translational) and can cause the permanent or temporary dysfunction of brain cells (1, 2). It is a leading cause of death and disability worldwide and presents serious social, economic, and health problems (2). TBI is classified according to the severity, anatomical features, and mechanism of the injury (3, 4). In cases of mild-to-moderate brain damage, conventional imaging tests such as computed tomography or magnetic resonance imaging may or may not detect the cascade of metabolic changes at the intracellular level, that have occurred or are occurring, that will induce detectable anatomical damage (5). Such imaging limitations, as can occur, are likely to reduce the accuracy of the medical diagnosis and impede the design of an efficacious rehabilitation strategy. We present the case of a 30-year-old Hispanic male veteran who, after sustaining 2 traumatic brain injuries, developed headaches, cognitive impairment, and behavioral problems, none of which could be explained by routine evaluations or anatomical imaging modalities.

Case Report

A thirty-year-old Hispanic male veteran presented to the VA Caribbean Healthcare System complaining of a left frontotemporal headache accompanied by blurry vision, vertigo, and nausea. While on active military duty from 2005 to 2008, he suffered 2 traumatic brain injuries (caused by small explosive missiles) that led to a loss of consciousness for more than 45 minutes. The first episode of traumatic brain injury occurred in 2007 and was complicated by a left eye laceration, a flank hematoma, and a left shoulder injured by metallic fragments. After the second episode in 2008, the patient developed attention and memory problems and began to experience behavioral disturbances (irritability and explosive behavior) that were managed with combat stress courses and antidepressants. His premorbid state was characterized by depression, tobacco use, and alcohol use.

Upon his arrival at the VA System's medical center in 2010, he was referred to the polytrauma clinics for neuropsychological evaluation and cognitive interdisciplinary rehabilitation. A computed tomography (CT) scan was obtained to evaluate intracranial pathology but failed to show any evidence

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of structural brain damage. After obtaining a negative CT, Fluorine-18 Fluorodeoxyglucose positron emission tomography/computed tomography (F-18 FDG PET/CT) and Technetium-99m ethyl cysteinate dimer single-photon emission computed tomography/computed tomography (Tc-99m ECD SPECT/CT) scan were performed. The Tc-99m ECD SPECT/CT revealed evidence of a large area of moderately decreased radiotracer distribution involving the superior, medial, and inferior frontal cortices of the right cerebral hemisphere. In addition, there was moderately decreased radiotracer distribution involving the superior and inferior parietal cortices of the right cerebral hemisphere, plus severely decreased tracer distribution involving the right basal ganglia. The F-18 FDG PET/CT did not show any metabolic or physiologic pathology. After the completion of the imaging procedures, the patient was referred for a somatosensory evoked potential (SSEP) test.

Discussion

Traumatic brain injury might be underdiagnosed in patients who, nevertheless, develop temporary or permanent functional disability or psychosocial impairment in the presence of a negative CT scan. The mechanisms of brain injury and recovery are being widely studied; however, it is still unclear whether functional brain injury can be linked to psychosocial experiences. Brain function could be partially or totally impaired after structural damage that may or may not be detectable with current anatomical imaging technology. When such damage remains undiscovered, rehabilitation strategies will lose their effectivity.

Functional nuclear medicine imaging and neurophysiological parameters can be used to characterize brain damage, as the former provides direct visualization of brain function, even in the absence of overt behavioral manifestations or anatomical findings (6, 7, 8). A Tc-99m ECD SPECT/CT performed on this patient revealed evidence of hypoperfusion involving the superior, medial, and inferior frontal cortices of the right cerebral hemisphere. There was also hypoperfusion involving the right basal ganglia. At the molecular imaging level, the F-18 FDG PET/CT did not show any metabolic abnormalities (figures 1, 2, and 3). This observed mismatch between perfusion defects and metabolic findings and metabolic defects suggests the possibility of up-regulation of the glucose receptors to compensate for diminished perfusion. Additionally, the increased severity and number of perfusion defects compared to metabolic defects suggests that the etiology of the TBI may be in part related to an impaired vasomotor response or endothelial dysfunction and not solely due to the trauma itself.

SPECT/CT studies may show focal areas of hypoperfusion that are inconsistent with the findings of a CT scan. These focal lesions revealed by SPECT/CT offer evidence of organic injury in patients whose conventional neuroimaging studies do not reveal any anatomic abnormalities (9). Furthermore, SPECT/CT findings associated to TBI are usually related to diminished perfusion to the cerebral parenchyma, which differs from the findings of hyperperfusion often seen in patients with Post Traumatic Stress Disorder (PTSD). Usually, when a patient has anxiety, irritability, or PTSD, patterns of increased perfusion to the cerebral parenchyma involving the anterior cingulate gyrus, basal ganglia, temporal lobes, and thalamus can be detected by SPECT/CT.

In this case, SSEP tests results were not available, though they usually are performed in patients as a noninvasive measure to evaluate the integrity or disruption of peripheral and central nerve impulse transmission (10). Studies in patients with anoxic brain injury suggest that evoked potentials can be used to measure the prognosis for recovery. Although the predictive value of evoked potentials in the assessment of TBI recovery strategies has not been fully established, patients who do not respond to cortical stimulation at the onset of anoxic injury may have a risk of poor functional recovery after the brain insult. To study the added value of SSEP in the assessment of TBI patients might be of interest to the clinical and research community.

In conclusion, SPECT/CT findings will impact the

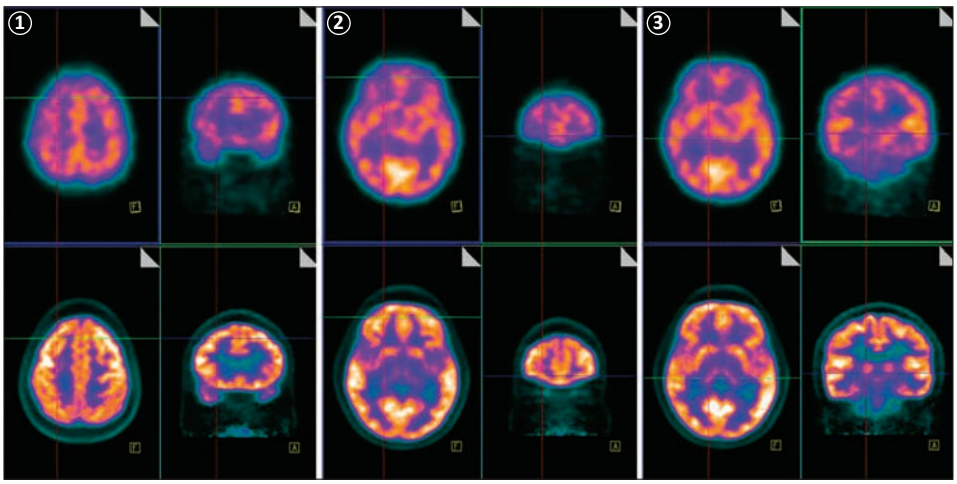

Figure 1. The upper panel is the Tc-99m ECD SPECT/CT imaging showing moderate hypoperfusion at the right superior frontal cortex. The lower panel is the F-18 FDG PET/CT imaging showing normal metabolism in the areas of hypoperfusion. **Figure 2**. The upper panel is the Tc-99m ECD SPECT/CT imaging showing moderate hypoperfusion at the right inferior/orbitofrontal cortex. The lower panel is the F-18 FDG PET/CT imaging showing normal metabolism in the areas of hypoperfusion. **Figure 3**. The upper panel is the Tc-99m ECD SPECT/CT imaging showing severe hypoperfusion at the right thalamus and basal ganglia. The lower panel is the F-18 FDG PET/CT imaging showing normal metabolism in the areas of hypoperfusion.

diagnosis of and further rehabilitation strategies for patients with TBI. In the near future, functional nuclear medicine imaging will be a cornerstone in the development of predictive models and algorithms to target rehabilitation interventions. This case demonstrates how functional nuclear medicine imaging could add value in terms of the diagnosis and localization of cerebral lesions in patients with mild-to-moderate traumatic brain injury.

Resumen

Una lesión cerebral traumática (LCT) se define como un daño al cerebro secundario a una fuerza externa. La LCT es una causa principal de muerte e incapacidad a nivel mundial, presentando serios problemas sociales, económicos y de salud. Los estudios de imágenes con modalidades anatómicas pudieran ó no detectar los cambios metabólicos que ocurrieron o que están ocurriendo a nivel celular en aquellos casos de daño cerebral con severidad de leve-a-moderada. Estudios de imágenes de medicina nuclear y parámetros neurofisiológicos pueden ser usados para caracterizar daños cerebrales, ya que el primero puede proveer una visualización directa de las funciones cerebrales, aún en ausencia de manifestaciones de comportamiento o hallazgos anatómicos. Este es un reporte de caso de un veterano hispano de 30 años de edad que, luego de 2 eventos de lesiones traumáticas cerebrales, desarrolló problemas cognitivos y neurofisiológicos sin etiología clara en presencia de una tomografía computarizada (TC) con hallazgos negativos.

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