Imaging of Traumatic Brain Injury

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KEYWORDS

• Trauma • Brain injury • Computed tomography • MR imaging

KEY POINTS

- Most patients with traumatic brain injury (TBI) have mild TBI (mTBI) and typically have no abnormalities on computed tomography (CT) and conventional MR imaging.
- Advanced MR imaging techniques including diffusion weighting, functional imaging, and spectroscopy are potential biomarkers for mTBI.
- CT is the initial diagnostic test in TBI. Conventional MR imaging in the acute phase is used as a problem solver when CT does not explain the neurologic deficit.

INTRODUCTION

Traumatic brain injury (TBI) is a major health and socioeconomic concern throughout the world[1,2] and is the leading cause of mortality and morbidity among young people.[3] The incidence of TBI is increasing in the developing countries, because of the increase in the number of motor vehicle accidents.[3] In advanced nations, the incidence of TBI caused by falls among the aging population is increasing[4] and is changing the occurrence of different forms of TBI, specifically increasing the incidence of focal brain injuries in the form of contusions. Meanwhile the incidence of diffuse axonal injury (DAI) caused by high-velocity traffic accidents is decreasing in developed nations.[4]

In the United States, an estimated 1.1 million emergency department visits and 235,000 hospital admissions occur yearly because of TBI.[5] Although most of these injuries are categorized as mild TBI (mTBI), a considerable number of these patients nevertheless experience permanent deficits.[6] Approximately 52,000 deaths are attributed to TBI per year in the United States.[7–9] TBI principally affects young men, resulting in lost productivity because of disability and lost years because of death. The financial burden to society is estimated to be more than $60 billion per year in the United States alone.[10] This article discusses the role of imaging in diagnosis and the spectrum of findings seen in patients with mild, moderate, and severe TBI.

CLASSIFICATION

TBI is usually classified by clinical severity using the Glasgow Coma Scale (GCS).[11] The mortality from TBI is related to the severity of injury as determined by GCS score.[12,13] GCS (range, 3–15) consists of the sum of the 3 component scores (eye, motor, and verbal scales): mTBI, greater than 12 to less than or equal to 15; moderate TBI, greater than 8 to less than or equal to 12; severe TBI, less than or equal to 8. TBI has also been classified according to the severity of structural damage based on neuroimaging.[14] However, the classification systems based on neuroimaging have limitations, because of severe underestimation of the extent of DAI by imaging modalities.

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Also, the systems broadly place injuries into diffuse and focal categories and fail to account for the specific type of mass lesions (eg, epidural vs subdural). The lack of specification fails to correctly classify patients with combined DAI and focal injuries.[4,15]

TYPES OF BRAIN INJURY

TBI is divided into primary and secondary injuries.[4,5,16] Primary injuries occur as a direct result of traumatic impact. Secondary injuries result from a complex biochemical cascade of events that exacerbates the primary injury by resulting in cerebral edema and herniation.[17] The primary parenchymal lesions contain an epicenter with axons, glial cells, and vascular structures that sustain irreversible damage.[16] Primary injury can result in either focal or diffuse lesions. Primary injuries at the macroscopic level are calvarial fractures, extra-axial hemorrhage (epidural hematoma [EDH], subdural hematoma [SDH], subarachnoid hemorrhage [SAH], and intraventricular hemorrhage [IVH]), and intra-axial injuries (contusions, DAI, and brain stem injury).[16] At the cellular level, the initial events include microporation of membranes, leaky ion channels, and changes in the intracellular proteins that occur minutes to hours after initial injury.[4]

Surrounding the epicenter is traumatic penumbra with cells that have sustained reversible damage.[16] Penumbra is the site where most of the deleterious secondary biochemical changes occur. The extent of various physiologic changes that occur during the early or late posttraumatic period, such as hypoxia, hypotension, pyrexia, and coagulopathy, may exacerbate the secondary events and determine the evolution of penumbra either into irreversible lesions or complete resolution.[4] The evolutionary changes in penumbra explain the appearance of new lesions not apparent on initial scans.[18]

Secondary injury is initiated by various pathophysiologic cascades of events that follow the initial injury at both cellular and macroscopic levels to manifest as secondary lesions on neuroimaging. The cellular reactions that develop over hours and days include neurotransmitter release, free-radical generation, calcium-mediated damage, gene activation, mitochondrial dysfunction, and inflammatory responses.[4] The release of neurotransmitters exacerbates the already leaky ion channels that cause primary injury and results in astrocytic swelling and cerebral edema. Cell necrosis is mainly caused by free-radical generation and calcium-mediated injury. Gene activation and expression of proapoptotic protein factors cause apoptotic astrocytic and oligodendrocytic cell death. Mitochondrial dysfunction can decrease adenosine triphosphate production and oxygen consumption, which can further lead to axonal necrosis and apoptosis.[19]

The differentiation of TBI into primary and secondary injuries is important, because secondary injuries are often preventable, whereas primary injuries are not.[16]

MILD TRAUMATIC BRAIN INJURY

Most patients with TBI (up to 75%) are considered to have mTBI.[20,21] Computed tomography (CT) and conventional MR imaging examinations are typically normal and advanced neuroimaging techniques are required. Diffusion-weighted imaging (DWI), functional MR (fMR), arterial spin labeling (ASL), and spectroscopy show structural and functional abnormalities (**Fig. 1**).[22–29] Studies also indicate that these techniques can be used as biomarkers to diagnose and monitor recovery.

Blunt force from contact sports and blast injuries is a common mechanism for mTBI. The angular or rotational force that produces this injury results in widespread, diffuse effect on the entire brain parenchyma. Early diagnosis of mTBI and careful follow-up imaging to monitor healing holds the potential to prevent long-term neurodegenerative processes, such as chronic traumatic encephalopathy, that occur as a long-term complication of repetitive mTBI.[22,30]

The criteria to diagnose the 2 types of mTBI are shown in **Table 1**.[31] Unlike mTBI, patients with complicated mTBI have minor structural abnormality on CT or conventional MR imaging.

POSTCONCUSSIVE SYMPTOMS

Although most patients with mTBI are discharged quickly from the trauma center, a significant portion (~40%) of patients with mTBI remain impaired for at least 3 months, and a substantial number of these patients show deficits up to 1 year after injury.[20,21,32] One year following injury, 82% of patients with mTBI reported the presence of at least 1 postconcussive symptom.[33] The resulting lost productivity has socioeconomic consequences. Postconcussive symptoms include neuropsychological (difficulty with socializing, depression, anxiety), cognitive (attention, executive function, working memory, reduced information processing speed), and somatic symptoms (headaches, chronic pain, sensory perception disorders, language difficulty).[34–38] These symptoms may also be present in patients with mTBI who lack evidence of intracranial injury on conventional