Understanding Mild Traumatic Brain Injury and Postconcussion Syndrome

By Frank D. Lewis, PhD, and John Lucas, MA, LPC, LMFT

Despite having a label of "mild," mild traumatic brain injury (MTBI) is a major public health problem in the United States, according to the Centers for Disease Control and Prevention (CDC). It is estimated that up to 1.2 million people sustain a MTBI each year the United States. MTBI accounts for more than 1 million (or 1% of) yearly emergency department visits. Most MTBIs occur to persons under the age of 24 or older than age 74. Fifty percent of MTBIs result from motor vehicle crashes, 25% from falls, 15% from assaults, and 10% from sports injuries. Most fall injuries occur in those younger than 5 and older than 75.[1]

In 2001, the CDC reported the cost of MTBI to be $16.7 billion.[1] Most of this economic cost was the result of lost productivity. These figures do not include the indirect cost of family caregiver expense and those who were treated in the emergency department, released, and then received subsequent care in other hospitals for symptoms related to their injury. A study by Boake and colleagues[2] found that MTBI patients had similar duration of work absences as did general trauma patients. In their study, work absences resulting from MTBI ranged from 1 week to 3 months. Other researchers have reported unemployment rates of 15% 2 years from the onset of the MTBI.[3,4] Perhaps the most important step in ensuring that these patients receive access to proper care is a thorough understanding of the definition and diagnosis of MTBI.

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Definition and Diagnosis

Failure to properly diagnosis a MTBI can readily lead to symptom exacerbation and chronic emotional and physical problems. An accurate understanding of the disorder will help case managers make better decisions regarding the nature, frequency, and duration of treatment necessary to minimize the severity of symptoms. However, defining and diagnosing MTBI has proven to be challenging. Traditionally, diagnosis of MTBI relied heavily on the Glasgow Coma Scale (GCS), a broad measure of neurologic functioning, ranging from nonresponsive-to-external-stimuli to fully oriented and conversant. The GCS emphasis on consciousness level and depth of coma has significant value for directing the emergency care of more severe neurological injuries, yet underestimates the presence of significant symptoms of MTBI. For example, most MTBIs are admitted to emergency departments with the highest GCS score of 15, which could be erroneously interpreted as normal neurological function. In response to these limitations, more comprehensive injury classifications have been developed. In 1993 the America Congress of Rehabilitative Medicine defined a MTBI as one that resulted from a blow to the head causing any alteration of mental status with the following criteria: (1) loss of consciousness not exceeding 30 minutes, (2) after 30 minutes an initial GCS score of 13 to 15, and (3) posttraumatic amnesia (PTA) not lasting longer than 24 hours.[5] Although widely used, this system of classification lacks the clarity and specificity necessary to detect characteristic symptoms of MTBI. The symptoms that should be considered are presented in Table 1.

To address these limitations and improve diagnostic accuracy, the CDC[6] published a detailed operational definition of MTBI in 2003. Its criteria include the following:

- Any period of observed or self-reported transient confusion, disorientation, or impaired consciousness
- Any period of observed or self-reported dysfunction of memory (amnesia) around the time of injury
- Observed signs of other neurological or neuropsychological dysfunction, such as:
  - Seizures acutely following head injury
  - Among infants and very young children: irritability, lethargy, or vomiting following head injury
  - Symptoms among older children and adults such as headache, dizziness, irritability, fatigue, or poor concentration (when identified soon after injury can be used to support the diagnosis of mild TBI, but cannot be used to diagnosis in the absence of loss of consciousness or altered consciousness)

- Any period of observed or self-reported loss of consciousness lasting 30 minutes or less

Since 2003, several organizations have published definitions of MTBI, with considerable overlap of acute injury characteristics. McCrea[7] provides a summary and review of various definitions. Taken together, these definitions forge a consensus among professionals as to the defining characteristics of MTBI. With this understanding, research is now advancing in the area of neuroimaging techniques with the hope of providing precise assessment of the structural and functional changes that are correlated with subtle neurological injury and recovery.

Neuroimaging and MTBI

Traditionally, head CT scans are taken in emergency departments to detect the presence of moderate and severe brain injuries. The CT scan is effective in detecting structural abnormalities and hemorrhagic lesions that require surgical intervention. The CT scans, however, lack the sensitivity necessary to detect small petechial hemorrhages and cellular dysfunction associated with MTBI. While effective in ruling out the need for surgical intervention, reliance on CT can lead to a false assumption of normal brain function.

Although not foolproof, magnetic resonance imaging (MRI) is more sensitive for detecting subtle brain abnormalities caused by MTBI. A review of studies in which patients were given both CT and MRI scans found that the MRI detected brain abnormalities in 30% of the patients that had normal CT scans.[8] Despite the increased capability to reveal brain abnormalities, MRIs have been shown to be only minimally effective in predicting the presence of symptoms long term.

Diffusion tensor imagining (DTI), a relatively recent variation on MRI, can detect small neural abnormalities not seen on the traditional MRIs.[9] Specifically, it is possible to observe microscopic abnormalities of white matter neural tracts using DTI. Also, DTI allows researchers to detect improvement in neural connectivity following brain injury.

When neuroimaging does identify abnormalities after a MTBI, the injury is considered to be a "complicated MTBI." If the scans are normal, the injury is classified as "uncomplicated MTBI." Patients with complicated MTBI—with lesions, sustained metabolic change, or diffuse injury—are at risk for slow or incomplete recovery. Kashluba and colleagues[10] found that MTBI patients with identifiable lesions experience outcomes more similar to patients with moderate brain injury as measured by GCS. These patients experienced significant symptoms 1 year post-injury. Symptoms were severe enough to prevent return to work or normal productive activity.

Mechanism of Injury

The neurological damage caused by a MTBI was once thought to occur in the same fashion as that of moderate and severe injuries. Shearing or rotational forces resulting from a blow to the head cause tearing and misalignment of axons and ultimately death of the cell. The damage, referred to as diffuse axonal injury, is widespread, typically concentrating at the junction between the gray matter of the cerebral cortex and the white matter of ascending and descending neurons. The ability to recover from such injuries relies primarily on adjacent uninjured neurons assuming function of the damaged neurons. Concussions or MTBIs were considered to be a milder form of diffuse axonal injury.

Recent research suggests a more hopeful model of mild injuries, which involves cellular dysfunction instead of death. A blow to the head of sufficient force to cause a MTBI initiates a process of accelerated neurotransmitter release, which increases cellular metabolism and impairs the connectivity of neurons. Giza and Hovda[11] refer to this disruption in function as a "neurologic metabolic cascade." This disruption of cellular function causes posttraumatic amnesia, headache, and the ▶

TABLE 1

Symptoms of Mild Traumatic Brain Injury

Physical
Headache
Seizure acutely following blow to head
Nausea
Vomiting
Dizziness
Fatigue
Hypersensitivity
Sleep disturbance
Numbness/tingling
Cognitive
Decreased attention/concentration
Short term memory deficits
Diminished capacity of working memory
Decreased processing speed
Decreased verbal fluency
Diminished executive functioning : planning, judgment, insight
Emotional/Behavioral
Mood changes or mood swings
Depression
Anxiety
Irritability

other cognitive symptoms of MTBI. In uncomplicated MTBIs, neurological function typically returns to normal after a period of days to weeks. As normal neural function returns, symptoms subside. In rare cases, symptoms persist for an indefinite period at which time the patient may be given a diagnosis of postconcussion syndrome (PCS).

Postconcussion Syndrome

In medical terminology, a syndrome is defined as the presence of clinically recognizable features or characteristics that occur together and in which the presence of one or more features indicates the possibility of the presence of others. In the case of postconcussion syndrome, symptoms are preceded a maximum of 4 weeks by a MTBI with a loss of consciousness. Additionally, diagnosis requires symptoms in at least three or more of the following categories[12]:

- Headache, dizziness, malaise, fatigue, noise tolerance
- Irritability, depression, anxiety, emotional lability
- Subjective concentration, memory, or intellectual difficulties without neuropsychological evidence of marked impairment
- Insomnia
- Reduced alcohol tolerance
- Preoccupation with the above s ymptoms and fear of brain damage with hypochondriacal concern and adoption of a sick role

Other features that may be associated with PCS include changes in personality, apathy, lack of spontaneity, learning difficulties, and worsening academic performance (children) or occupational performance (adults).

As is the case with MTBI, diagnosing PCS can be quite problematic. Diagnosis of the syndrome is plagued with poor reliability.[7] Many of the symptoms are subjective and may have been present, at least in part, prior to the TBI. Also, PCS symptoms are similar to other disorders, such as fibromyalgia, chronic pain, and chronic depression. Iverson13 reported that 90% of persons with depression and no TBI history would likely meet PCS criteria. In addition to symptom overlap, several demographic and psychosocial factors are predictive of PCS after a MTBI including female gender, older age, unstable relationships, lack of social support, preexisting psychiatric problems, chemical dependency, and litigation. Some have hypothesized that depression, anxiety, and stress in the weeks following a head injury contribute to the persistence of symptoms.[7] Therefore, to be effective, treatment models must address the underlying psychological factors maintaining the symptoms.

Model for Clinical Management

Obviously initial treatment is often given in the emergency department, where more serious brain injury is ruled out. As indicated previously, for most cases, initial symptoms are temporary, clearing by 3 to 4 weeks. During this acute phase, the treating physician will likely prescribe medication to alleviate symptoms of pain and anxiety and recommend lifestyle changes. Depending on the individual, these changes might include avoiding contact sports and situations placing one at risk for fall or collision, bright lights, vigorous exercise, and prolonged time in front of computer screens. The patient will likely be counseled to get adequate rest and sleep, while work and school schedules may be modified.

Following the acute phase of injury, if the symptoms have not resolved, clinical management should shift to a comprehensive multidisciplinary approach.[7] An Internet search reveals numerous clinics offering comprehensive assessment and treatment of MTBI and PCS. To be effective, these programs must have rehabilitation professionals with expertise in treating brain injury. Often these teams are led by a neuropsychologist in close association with a physician, typically a physiatrist or neurologist with a brain injury specialty. The management process begins with a thorough patient history to include information concerning previous concussions or head injuries, developmental issues (eg, learning disability or ADHD), psychiatric issues, and social history. The physician may order additional neuroimaging to determine the presence of lesions or other neurological impairment.

Next the neuropsychologist initiates a neuropsychological evaluation to determine performance on a wide array of cognitive functions potentially affected by the brain injury. Key among these functions are working memory, attention and concentration, verbal problem solving, visuo-spatial perception, reasoning, and processing speed. The battery should include tests (ie, MMPI-2 validity profile, Test of Malingering) designed to detect symptom exaggeration and malingering motivated by secondary gain.

Test results are reviewed by the neuropsychologist and the physician. Based on the findings, the appropriate treatment team is assembled and a treatment plan is written to address deficits and monitor progress. Depending on the extent of deficits, the team may include all or some of the following specialists: psychologist/counselor, speech pathologist, physical therapist, and occupational therapist. Often multiple therapists will approach the same functional deficit from a somewhat different treatment approach. Consistent communication on problems and progress is essential for a positive outcome. The psychologist plays a key role in adjustment to the changes initiated by the brain injury. The psychologist is typically the team member who conducts patient and family brain injury education and, along with the physician, discusses the course of treatment and prognosis. The psychologist

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also provides counseling and support, teaches coping and compensatory strategies, and provides family counseling to help loved ones adjust and react to potential changes in behavior. The speech pathologist focuses on improving attention and concentration, while providing strategies to compensate for memory loss. When necessary, physical therapists improve conditioning, strength, and vestibular and balance problems. Occupational therapists typically will address safety and judgment in the home and work setting. Other team members who may be enlisted in the treatment include an educator to facilitate a successful transition to school and a dietitian to promote a brain healthy diet.

When injury initiates an array of both neurologic and non-neurological symptoms, the team approach to treatment offers several advantages over single provider care including:

- Detailed assessment of risk factors in multiple domains of functioning
- Behavioral management to prevent the development of maladaptive behavior and symptom exacerbation
- Treatment from multiple specialists in brain injury
- Support system and education for the patient and family
- Availability of specialized psychological and counseling services
- Consistent observation and monitoring of progress
- Allowance for repeated practice of adaptive behaviors
- A positive treatment context with high expectation for success

While additional empirical studies will help to improve the efficacy of MTBI treatments, the most comprehensive studies to date suggest the team approach with supportive counseling and education given soon after injury offers the best hope for positive functional outcomes and minimized PCS symptoms.[7] The case example that follows illustrates frustrations often experienced by MTBI patients. Frustration stems not only from physical and cognitive symptoms, but also from the impact they have on work performance and personal relationships. Unfortunately, for patients, convincing physicians and payers that the symptoms are real may pose their greatest challenge.

Case Example

Julia, a healthy women in her early 50s, was employed as a claims adjuster earning a 6-figure salary. She had a long and positive employment history and was highly regarded by her peers in the business. While on the job inspecting a roof, she was hit on the top of her head by a large metal extension ladder when it became dislodged from its rails. Although dazed and disoriented, she reported no loss of consciousness. She refused medical treatment at the time and continued to work. Later that day she complained of a severe headache. Three days later she was referred for evaluation and testing. She reported that her tests were unremarkable; however, she was prescribed physical therapy to treat neck pain and other somatic complaints. She attended physical therapy for approximately 1 month, during which time she did not work. Following therapy, she returned to her job. After 2 months her symptoms worsened. As a result, she was referred for a neurosurgery consult. Her appointment with the neurosurgeon occurred approximately 4 months after her injury. At the time of her appointment, she reported daily headaches, intermittent forgetfulness, and heightened emotionality. Other symptoms included posterior neck pain, pain in her upper thoracic region, tingling in her right hand, and weakness in her right leg. Julia reported that her neurosurgeon was skeptical with regard to her report of cognitive symptoms. He believed that because she was so articulate in describing her difficulties, she must have been cognitively intact. According to Julia, he made her feel that her symptoms were just "in her head" at best, or worse, she was faking in order to get disability payments. Nonetheless, his evaluation cited cervical strain and posttraumatic headaches with "elements to suggest postconcussion syndrome." Ultimately, he did recommend continuing physical therapy and rehabilitation to address her PCS. Insurance approval for outpatient treatment of PCS was not obtained until almost 2 months after her neurosurgery appointment, or 6 months postinjury.

Prior to therapy, Julia received a comprehensive neuropsychological evaluation, which she paid for out of pocket. That evaluation showed impaired functioning in working memory, processing speed, and attention/concentration. Tests showed no evidence of malingering or symptom faking. Julia's insurance approved physical and speech therapies each for 1 hour per day 5 days per week. She was also approved to receive counseling 3 days per week. Insurance approval ▶

for treatment was given weekly contingent on progress in the program. Julia attended therapy sessions in the morning and went to work in the afternoon.

Julia's counseling sessions were the focal point of her treatment because of her emotional lability and anxiety about changes in cognitive functioning. Concerns regarding her ability to return to work at pre-injury performance and income levels contributed greatly to her anxiety and emotionality. At work she felt she was falling behind, regardless of how hard she tried.

Julia was married just a few months prior to her injury. She described her marriage as a good one with high expectations for success, but she stated that she did not feel like herself. She worried her husband might think she was not the women he married. She described herself as easily irritated and short tempered. She reported that extreme fatigue greatly curtailed her evening activities; stating she only wanted to "go home and sleep after work." When requested, Julia's husband attended therapy sessions. He was very supportive and demonstrated an understanding that changes in Julia's mood were largely due to her injury.

Julia put forth significant effort in her counseling and other therapy sessions. Each week in therapy was marked by improvements in emotional control and adjustment to her limitations. She reported improvements in her ability to focus and complete assignments. While she still experienced headaches, they were less frequent and less severe. She also reported that her cervical pain was more manageable.

Julia's improvements in therapy, however, only transferred minimally to her job. Compared to her pre-injury performance, she was slow and inefficient. Anxiety made it difficult for her to sustain attention to her duties. As Julia's therapists began to focus on helping her utilize newly learned coping skills while at work, her coverage was abruptly terminated. After only 3 weeks of treatment, Julia was discharged. Against the recommendations of the therapy team, she returned to work full time. For 6 months she struggled. Neck pain and stress escalated, making it difficult for her to concentrate and complete her work. Ultimately, almost 1 year from her injury, Julia lost her job. At home, Julia's marital problems had worsened.

Julia experienced a poor outcome from her brain injury. During therapy she realized some relief from symptoms, but treatment was ended prematurely and she continued to experience difficulty at work and stress in her marriage. Julia's case was poorly managed from the beginning. Without empirical study, one cannot definitively say what her outcome would have been had she received treatment sooner, but comprehensive specialized rehabilitation given shortly after injury, when her symptoms were emerging, would have likely prevented her downward spiral. An early neuropsychological evaluation could have ruled out malingering and facilitated proper coordination of her treatment with experts in treating MTBI and PCS.

Summary

Most of the estimated 1.2 million persons who sustain a MTBI each year will experience an excellent functional outcome in a relatively short period of time. However, for approximately 15% of those cases, cognitive, emotional, and physical symptoms will persist. Symptoms are the result of both neurological and non-neurological factors that left untreated may worsen as maladaptive patterns of coping develop out of a lack of understanding of symptoms and prognosis. The multidisciplinary team model provides an important option in managing complicated cases of MTBI, preventing symptoms of PCS, and achieving meaningful functional outcomes. CEU

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