

PREVENTION IN ACTION

Watch Out for Psychiatric Illness After Mild TBI

PERSPECTIVE

Until recently, mild traumatic brain injury was presumed to be not very important for generating long-term symptoms or problems. In fact, this consideration was a huge source of contention among those who granted disability status of patients with mild TBI.

Also, disagreement prevailed within legal circles about various injury-related lawsuits, as most companies did not want to pay for the post-mTBI headaches, symptoms of depression, insomnia, and so forth.

Similarly, mTBI has been underconsidered as a source of psychiatric symptoms among mental health clinicians. Few psychiatrists routinely ask patients about mTBI.



BY CARL C. BELL, M.D.

This mindset might be exacerbated by the fact that when

there is no loss of consciousness associated with a head injury, individuals often don't seek medical care, and by the measures used to gauge the severity of head trauma and the nomenclature used to describe it.

The term "mild" with respect to traumatic brain injury does not reflect the severity of the injury, but rather the length of time the individual experiences postinjury confusion or disorientation.

On the Glasgow Coma Scale, an injury causing less than 30 minutes of altered consciousness is deemed mild. To patients, families, and even clinicians, that connotation might minimize the awareness of the potential for long-term symptoms.

Since neuroimaging has become more readily available and the science has become more specific, mTBI and the possibility of postinjury symptoms have recently gained more traction. But there is still no way to show cause and effect between mTBI and the broad range of neuropsychiatric symptoms that have been attributed to it.

Until research catches up with reality, the best way to manage psychiatric symptoms in mTBI patients is to first identify such patients through routine history and, educate the patient and family, validate the patient's symptoms, and treat with therapy and medication.

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Mild traumatic brain injury is a "silent epidemic," according to the Centers for Disease Control and Prevention. But the condition has been generating a lot of noise recently.

Reports of studies showing a link between mild traumatic brain injury, or concussion, and lingering alterations in cognitive and motor function in high-profile populations, such as U.S. soldiers returning from Iraq and Afghanistan, college athletes, and professional football players, have begun to give voice to the potential public health burden imposed by such injuries.

Generally defined as a head injury resulting from contact or acceleration or deceleration forces that induce an alteration in mental status (with or without a loss of consciousness) and a Glasgow Coma Scale score of 13-15, mild traumatic brain injury (mTBI) accounts for as many as 90% of all cases of head injury, World Health Organization estimates show.

The acute, outward signs and symptoms of this type of injury appear to be short lived, and patients, families, and even clinicians historically have minimized the potential relationship between the injury and subsequent symptoms of cognitive or other impairment. There is considerable literature reporting the strong association between TBI and psychiatric disorders. Major depression is the most prevalent psychiatric disorder after TBI, with estimated rates ranging from 14% to 77%, according to Theresa A. Ashman, Ph.D., of the Mount Sinai School of Medicine, New York.

In one often-cited study, Dr. Jesse R. Fann of the University of Washington, Seattle, and colleagues investigated the risk of psychiatric illness after TBI among patients in an adult health maintenance organization. Of 939 patients diagnosed with TBI in 1993, the prevalence of any psychiatric illness in the first year after mild TBI was 34%, compared with 18% in the non-TBI control group (Arch. Gen. Psychiatry 2004;61:53-61).

More recently, a study of 2,552 retired professional football players showed a significant association between recurrent concussion and a diagnosis of clinical depression. Compared with retired players without a history of concussion, those who experienced three or more previous concussions were three times more likely to be diagnosed with depression, and those with a history of one or two previous concussions were 1.5 more likely to be diagnosed with depression, reported lead investigator Kevin M. Guskiewicz, Ph.D. (Med. Sci. Sports Exerc. 2007;39:903-9).

"Traditionally, it was thought that depression following a mild head injury was a reaction to the fact that the person had an accident or, for an athlete,

he or she could not return to play," said neuropsychologist Alain Ptito, Ph.D., of the Montreal Neurological Institute and Hospital. Dr. Ptito and his colleagues recently used magnetic resonance imaging to identify neural substrates of depression symptoms related to mTBI in 56 male athletes. "Our study clearly demonstrates that the story is not that simple—that depression [in this population] seems to originate from a cerebral dysfunction.

"The athletes with concussion and depression symptoms showed reduced activation in the dorsolateral prefrontal cortex and striatum and attenuated deactivation in medial frontal and temporal regions," the authors wrote. "The severity of symptoms of depression correlated with neural responses in brain areas that are implicated in major depression" (Arch. Gen. Psychiatry 2008;65:81-9).

Screening for depression in post-mTBI patients can take as little as 5 minutes and can be achieved by telephone, according to Harvey S. Levin, Ph.D., of Baylor College of Medicine in Houston. Dr. Levin and his colleagues developed a prediction model using a brief screening measure for depression to identify patients with mTBI at high risk for a major depressive episode by 3 months post injury.

The investigators recruited a prospective cohort of 129 consecutive adults with mTBI who were evaluated at a large, metropolitan Level I trauma center. All of the patients underwent CT scans within 24 hours of their injury. They also completed the self-report Center for Epidemiologic Studies Depression scale (CES-D) at 1 week post injury and the current major depressive episode module of the Structured Clinical Interview for the DSM-IV at 3 months post injury.

Logistic regression was used to generate a prediction model of a major depressive episode at 3 months post injury using the CES-D score as an independent variable. Major depressive episode was present in 15 subjects at 3 months post injury (Arch. Gen. Psychiatry 2005;62:523-8).

The findings support the feasibility of the early detection of patients with mTBI who are at high risk for developing major depression, the authors wrote.

The findings also raise the possibility that coordinating outpatient psychiatric services with trauma centers could improve outcomes associated with mTBI by mitigating secondary conditions, they said.

In addition to screening for depression among mTBI patients, consistent screening for mTBI among patients with psychiatric symptoms should be mandated, Dr. Ashman said. Such screening is especially important in practices involving populations where "hidden TBI" is known or suspected

to be common such as athletes and the elderly.

The treatment of depression secondary to mTBI should be approached from a multidisciplinary perspective. "When possible, individuals with TBI should have a neuropsychological evaluation to determine the nature and extent of the cognitive impairments and plan treatment," Dr. Ashman and colleagues wrote in a review article of the neurobehavioral consequences of traumatic brain injury.

"Remediation, which may be coupled with psychotherapy, can then be provided by rehabilitation psychologists or neuropsychologists, in conjunction with speech therapists, occupational therapists and other rehabilitation professionals" (Mt. Sinai J. Med. 2006;73:999-1005).

No medication has received approval from the Food and Drug Administration for the treatment of any neuropsychiatric consequence of mTBI, but antidepressant therapy has been shown to improve mood and, potentially, cognitive performance in these patients.

In a 2001 study, for example, Dr. Fann and colleagues conducted an 8-week, nonrandomized, single-blind, placebo run-in trial of sertraline in a group of 15 patients with mTBI and depression.

The investigators conducted neuropsychological testing before and after the treatment trial. Compared with baseline, depression scores improved significantly, as did measures of psychomotor speed, recent verbal memory, recent visual memory, and general cognitive efficacy improved with treatment, the authors wrote.

When pharmacotherapy is considered in this population, it is essential to start medications at low doses and to titrate slowly because of the potential susceptibility to adverse cognitive effects, Dr. Ashman said.

It is best to avoid medications that are highly sedative and those that have deleterious effects on the central nervous system, she noted.

Finally, education is a key component of depression management in mTBI. Education after mTBI for the patient as well as family, friends, employers, and others, should begin early and include an explanation of the range of possible symptoms, the usual time course for resolution, and the potential for long-term problems, according to Dr. David B. Arciniegas of the University of Colorado, Denver.

Also, "the clinician should offer validation of the person's experience of symptoms," and couple the validation with the development of realistic goals aimed at returning to normal activities, he said. ■

By Diana Mahoney. Share your thoughts and suggestions at cpnews@elsevier.com.