Evaluation and management of mild traumatic brain injury: An Eastern Association for the Surgery of Trauma practice management guideline

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BACKGROUND: An estimated 1.1 million people sustain a mild traumatic brain injury (MTBI) annually in the United States. The natural history of MTBI remains poorly characterized, and its optimal clinical management is unclear. The Eastern Association for the Surgery of Trauma had previously published a set of practice management guidelines for MTBI in 2001. The purpose of this review was to update these guidelines to reflect the literature published since that time.

METHODS: The PubMed and Cochrane Library databases were searched for articles related to MTBI published between 1998 and 2011. Selected older references were also examined.

RESULTS: A total of 112 articles were reviewed and used to construct a series of recommendations.

CONCLUSION: The previous recommendation that brain computed tomographic (CT) should be performed on patients that present acutely with suspected brain trauma remains unchanged. A number of additional recommendations were added. Standardized criteria that may be used to determine which patients receive a brain CT in resource-limited environments are described. Patients with an MTBI and negative brain CT result may be discharged from the emergency department if they have no other injuries or issues requiring admission. Patients taking warfarin who present with an MTBI should have their international normalized ratio (INR) level determined, and those with supratherapeutic INR values should be admitted for observation. Deficits in cognition and memory usually resolve within 1 month but may persist for longer periods in 20% to 40% of cases. Routine use of magnetic resonance imaging, positron emission tomography, nuclear magnetic resonance, or biochemical markers for the clinical management of MTBI is not supported at the present time. (*J Trauma Acute Care Surg*. 2012;73: S307–S314. Copyright © 2012 by Lippincott Williams & Wilkins)

KEY WORDS: Mild traumatic brain injury; concussion; guidelines.

STATEMENT OF THE PROBLEM

It is estimated that 1.1 million people sustain a mild traumatic brain injury (MTBI) each year in the United States.[1] MTBI is a common cause for presentation to the emergency department (ED) and for admission to trauma centers. Despite this, the optimal management of MTBI is unclear, and its natural history remains poorly characterized. The acquisition of high-quality data has been impaired by a variety of factors. Patients may present in an acute or delayed fashion and are seen in widely varying practice settings, such as trauma centers, nontrauma hospital EDs, urgent care clinics, and primary care offices. Patients often present with a constellation of symptoms that are difficult to quantify and that are not specific to MTBI. Follow-up rates may be low, especially for patients with minimal symptoms. This in turn may cause studies to include a nonrepresentative sample of patients with MTBI.

One of the main problems with the literature on this topic is the lack of a consistent definition of MTBI. Several national organizations have published definitions,[1–4] but they have not been consistently applied in the medical literature or in individual patient records. Published guidelines also differ on whether the term *concussion* should[2] or should not[5] be used interchangeably with *MTBI*. Three commonly used guidelines mandate that any brain computed tomographic (CT) scan result must be negative for the TBI to be considered mild,[2,4,5] but another one suggests that some patients with positive findings can be included,[1] and another only states that the CT scan result "may be normal."[3]

The natural history of MTBI is poorly understood in part because the studies conducted thus far vary widely in their inclusion criteria, methodology, and outcome variables measured. The studies tend to be scattered across a wide variety of journals in a number of disciplines and originate in many different countries. As a result, there has been an accumulation of a large number of studies in which each uses a different

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measurement tool to describe a set of different outcome variables in its own unique study population.

Recommendations on the management of patients with MTBI were published by a Practice Management Guidelines (PMG) work group of the Eastern Association for the Surgery of Trauma (EAST) in 2001.[4] This document included literature from 1975 to 1998. The purpose of this update was to revise and expand on these recommendations using literature published up to 2011.

PROCESS

A search of Pubmed and Cochrane databases was performed. Key words included *closed head injury*, *concussion*, and *traumatic brain injury* and included descriptors such as mild and minor. Additional references were obtained in the reference sections of retrieved articles, from review articles, and from Web resources. English-language references from 1980 to 2011 were examined, and articles published after 1999 were emphasized. A significant number of studies that were noncontributory were excluded. There was a notable lack of randomized, controlled trials, and it was not possible to restrict our review of any MTBI subtopic to such trials. In total, 112 articles were determined to be relevant for consideration for this PMG update. Recommendations were characterized as Level 1, 2, or 3 in the same fashion as in other EAST guidelines.

DEFINITION

MTBI is defined as an acute alteration in brain function caused by a blunt external force and is characterized by a Glasgow Coma Scale (GCS) score of 13 to 15, loss of consciousness for 30 minutes or less, and duration of posttraumatic amnesia of 24 hours or less. If a brain CT scan has been performed, its result must be normal. The terms *mild traumatic brain injury* and *concussion* may be used interchangeably.

RECOMMENDATIONS

Level 1

There are no level 1 recommendations

Level 2

1. Clinicians should perform brain CT scan on patients that present with suspected brain injury in the acute setting if it is available.
2. If CT resources are limited, consideration may be given to the use of a set of standardized criteria (e.g., the Canadian CT Head Rule [CCHR], New Orleans Criteria [NOC]) to determine which patients with MTBI receive a brain CT scan. Clinicians should be aware that this practice is associated with a nonzero missed injury rate.

Level 3

1. Clinicians should not routinely use magnetic resonance imaging (MRI), positron emission tomography, or nuclear magnetic resonance in the clinical management of patients with MTBI at the present time (Level 3).
2. Patients with an isolated MTBI and a negative brain CT scan result may be discharged from the ED if they have no other injuries or issues requiring hospital admission (Level 2).
3. Patients taking warfarin who present in the acute setting with an MTBI should have their international normalized ratio (INR) level determined. (Level 3).
4. Anticoagulated patients with supratherapeutic INR values and a normal initial brain CT scan result remain at significant risk for interval development of intracranial hemorrhage and should be admitted for a period of observation (Level 3).
5. Patients may be advised that measurable deficits in cognition and memory usually resolve at 1 month but that in 20% to 40% of cases, postconcussive symptoms may persist for 3 months or longer (level 3).
6. The ability to safely operate a motor vehicle may be impaired for a variable length of time in patients with MTBI. The timing of resumption of driving should be individualized (Level 3).
7. The timing of returning to work for patients with MTBI should be individualized. Formal neuropsychologic testing can be considered in some cases (Level 3).
8. Biochemical markers such as S-100, neuron-specific enolase, and serum tau should not be routinely used in the clinical management of patients with MTBI except in the context of a research protocol (Level 3).

SCIENTIFIC FOUNDATIONS

Definition and Epidemiology

Several national organizations have published guidelines describing a definition of MTBI. These include the Centers for Disease Control and Prevention (CDC, 2003),[1] the Veterans Affairs/Department of Defense (VA/DoD) Clinical Practice Guidelines (2009),[2] the American College of Rehabilitation Medicine definition (1993),[3] and the 2001 EAST PMG guidelines.[4] Each definition is in agreement that the mechanism must involve a blunt external force with a resulting physiologic alternation in brain function.[1–4] Although the language describing the nature of the alteration in brain function varies, most agree that the presenting GCS score should be 13 to 15,[1–4] that any loss of consciousness should be less than 30 minutes,[1–3] and that the duration of posttraumatic amnesia should be less than 24 hours.[1–3]

Other aspects of the definition of MTBI vary between these four guidelines. Patients having seizures after injury can still be considered to have an MTBI according to one definition[1] but would be excluded by another,[4] and the issue was not addressed in the other two guidelines.[2,3] The presence of a focal neurologic deficit is allowed by one definition[3] but not the other three.[1,2,4] The CDC guidelines state that some patients with positive findings on brain CT scan can still be considered to have an MTBI,[1] but in the VA/DoD[2] and 2001 EAST[4] guidelines, the CT scan finding must be negative, and the ACRM definition only says that the CT scan "may be normal."[3]

Disagreement also exists on whether the terms *concussion* and *MTBI* are synonymous. The VA/DoD guidelines recommend that the terms be used interchangeably in the medical record and when speaking to patients.[2] In contrast, the

2009 Consensus Statement on Concussion in Sport, a highly referenced sports medicine consensus paper, states that MTBI is a distinct process from concussion and that the terms should not be used interchangeably.[5] However, the panel did not propose a definition of MTBI or how it might be distinguished from concussion. Generally speaking, the emergency medicine and trauma literature tends to use the term *MTBI*, and the sports medicine literature tends to use the term *concussion*.

The epidemiology of MTBI remains poorly understood in part owing to inconsistent definitions and terminology. National, regional, and hospital data registries typically quantify TBI cases using International Classification of Diseases —9th Rev.—Clinical Modification (ICD-9-CM) codes. Concussion is currently listed as ICD-9-CM code 850. However, only some of the 850 codes (850.0, 850.1, 850.11) would be characterized as an MTBI using existing definitions. Codes 850.12 and 850.4 would be characterized as "moderate" TBI under any of the commonly used definitions,[1–4] yet the term *concussion* is still used by the ICD-9-CM system. Furthermore, clinical documentation often does not contain enough detail for patients with MTBI to be properly coded as 850.0 to 850.11. These factors make it difficult to determine exactly what percentage of all patients with TBI have an MTBI. A recent CDC report indicated that approximately 1.7 million patients with a TBI seek medical attention annually in the United States,[6] and an earlier CDC report estimated that 75% of all patients with TBI have an MTBI.[1]

Role of Imaging

Noncontrast brain CT scans have been used to determine the presence of intracerebral lesions after trauma since the mid-1970s. The 2001 EAST guidelines recommend obtaining a brain CT scan for essentially all patients presenting with an MTBI.[4] In contrast, the 2003 CDC[1] and the 2009 VA/DoD[2] guidelines do not list criteria for obtaining a brain CT scan. It has become commonplace at many US medical centers to perform a brain CT scan for any patient who presents acutely with any loss of consciousness or other clinical sign of MTBI.

The practice of obtaining a brain CT scan for every patient that could conceivably have positive findings leads to a significant number of negative study findings and may also be burdensome from a financial and resource standpoint. As a result, there has been considerable effort directed toward identifying patients that present clinically with an MTBI but are unlikely to have an intracranial lesion on CT scan.[7–13] One of the most important of these efforts was the study introducing the so-called *Canadian CT Head Rule*, published by Stiell et al.[8] in 2001. The authors enrolled 3,121 patients at 10 Canadian medical centers that presented within 24 hours of injury with a history of blunt head trauma, an initial GCS score of 13 to 15, and amnesia or disorientation. The primary outcome was need for neurologic intervention, and the secondary outcome was "clinically important" injury seen on CT scan. A set of five "high-risk" and two "medium-risk" history and examination findings were generated, which predicted the primary and secondary outcomes. The presence of a high-risk factor (failure to reach GCS score of 15 in 2 hours, suspected open or depressed skull fracture, vomiting $\geq$ 2 episodes, sign of basal skull fracture, and age $\geq$ 65 years) was 100% sensitive for predicting the need for neurologic intervention and would yield a 32% CT rate. The presence of a medium-risk factor (amnesia for >30 minutes and dangerous mechanism of injury) was 98.4% sensitive for clinically important brain injury and would result in a 54% CT rate. These seven risk factors were termed the *Canadian CT Head Rule* (CCHR).[8] One important feature of the study is that it makes the assumption that certain brain lesions are not clinically important. These were defined by the authors as a solitary contusion of less than 5 mm, localized subarachnoid blood less than 1 mm thick, subdural hematomas less than 4 mm, pneumocephalus, and closed depressed skull fracture not through the inner table. The CCHR performed favorably in a subsequent external validation study,[11] but a follow-up study by the authors revealed that the brain CT rates actually increased in the Canadian medical centers in which it was implemented.[10] Although acceptance of the CCHR has not yet been widespread, its use has been recommended in the eighth edition of Advanced Trauma Life Support.[14]

In 2000, Haydel et al.[7] published a study identifying seven clinical findings, later referred to as the NOC, which predicted the presence of lesions on brain CT scan for patients presenting with a TBI and an initial GCS score of 15. These included intoxication, age greater than 60 years, headache, vomiting, deficits in short-term memory, physical evidence of trauma above the clavicles, and seizure. All patients with lesions on brain CT scan had one or more of these seven findings. One study comparing the CCHR and the NOC showed that the NOC were more sensitive for the detection of positive CT findings; however, the potential decrease in CT use was only 3% for the NOC versus 37% for the CCHR.[11]

Given the wide variation in practice patterns and resource availability throughout the world, it is unlikely that any one set of criteria will be accepted universally. Applying a set of criteria derived in one location may have different results in other populations. For example, in the CHALICE study[15] (conducted in the United Kingdom), use of the listed criteria led to a CT scan rate of 14%, whereas in a subsequent validation study in Australia, use of the same criteria led to a 46% CT scan rate.[16] It has been pointed out that published CT scan guidelines all demonstrate a tradeoff between sensitivity and specificity.[12] Efforts to achieve an overall reduction on CT use will inevitably lead to a higher missed injury rate, although whether these injuries are clinically significant is debatable.[8,9] These differences have significant implications that may influence the degree to which an algorithm is adopted in different areas. It may not be realistic to expect that a set of criteria that is generated in a given location with its own unique medicolegal environment can be easily transferred to another area with the same degree of acceptance.

Other structural and functional imaging modalities have been used clinically and in research for patients with MTBI. Among these, MRI is the most readily available in the clinical setting. Its sensitivity for the detection of contusions, shear injury, and extra-axial hematomas is higher than that of CT scan, although for fractures, it is lower.[17] It may detect white matter lesions consistent with shear injury in patients presenting with normal CT scan findings.[18] However, data to support the notion that the presence of these lesions correlates

with worsened neuropsychologic outcome or the development of postconcussive symptoms are limited.[19] A variety of functional imaging modalities have also been explored, including functional MRI,[20] diffusion tensor MRI,[21] positron emission tomography,[22,23] and proton nuclear magnetic resonance.[24] Use of a noninvasive device designed to assess cerebral perfusion known as the brain acoustic monitor has also been described.[25] These modalities are expected to remain an active area of research. However, at the present time, there are insufficient data to support the routine use of any of these modalities in the clinical setting.

Indications for Discharge

In 2011, Holmes et al.[26] published results from a prospective multicenter observational study that described the clinical outcome of children presenting with a blunt head injury, GCS score of 14 to 15, and a negative brain CT scan finding. A total of 13,543 patients with a mean age of 8.9 years were included, and the original decision to obtain a CT scan was left to the discretion of the treating physicians. Charts were reviewed for the hospitalized patients (17%), and telephone follow-up was done for the patients discharged home (83%). The outcome measures were subsequent positive findings on CT or MRI scans and need for neurosurgical interventions. Overall, 0.16% of patients with a normal brain CT scan results were found to have positive findings on a subsequent CT scan. The percentage was slightly higher for patients with GCS score of 14 (0.6%) than it was for those with GCS score of 15 (0.12%). None of the patients required neurosurgical intervention. The authors concluded that children with GCS score of 14 to 15 and a negative brain CT scan finding could safely be discharged home unless another indication for admission was present.[26]

The largest study on this subject in adults was published by Livingston et al.[27] in 2000. The study included 1,788 patients with GCS score of 14 to 15 and an initial brain CT scan interpreted as negative. Overall, 1.1% of patients later had their brain CT interpretations changed from negative to positive, and 0.3% of patients required neurosurgical intervention such as intensive care unit monitoring or antiedema medications. Only one patient with an initially negative brain CT scan finding required a craniotomy, and this was for elevation of a complex craniofacial fracture rather than for hemorrhage. The negative predictive value of a negative brain CT scan result for the need for subsequent neurosurgical intervention was 99.7%.

In 2004 af Geijerstam et al.[28] reviewed the MTBI literature from 1966 to 2004 to determine the frequency of clinical deterioration for patients presenting with a GCS score of 15 and a negative brain CT scan result. The study included more than 62,000 patients of all ages divided among 93 publications. Adverse outcomes within the first 2 days after injury were examined. Only three patients were reported to have had a significant clinical deterioration within the first 2 days after injury. The authors concluded that brain CT scan could be safely used to decide which patients should be admitted to the hospital.

Patients who are therapeutically anticoagulated (e.g., with warfarin, clopidogrel, or other agents) may warrant special consideration. All of the larger studies listed previously either excluded patients taking these agents or included a small or unspecified number of them.[7–13,15,16,26–28] To date, there has been no sizable study outlining a set of clinical criteria that defines a subset of therapeutically anticoagulated patients for which a brain CT scan can be safely withheld after MTBI. It also remains unclear how long patients taking warfarin that have a negative brain CT scan result should be observed in a hospital setting. One study by Kaen et al.[29] showed that 1.4% of patients taking therapeutic warfarin or heparin had the interval development of intracranial hemorrhage in the first 24 hours after an initially negative brain CT scan result.

A study by Cohen et al.[30] in 2006 showed that anticoagulated patients with supratherapeutic INR values are at significant risk for clinical deterioration, morbidity, and mortality after any TBI, including MTBI. The study included patients from a prospective TBI database as well as charts reviewed during the ACS verification process at several trauma centers. Within this group, 77 patients had a GCS score of 13 to 15 and a supratherapeutic INR value (mean 4.4). Twenty patients were discharged home, and 35% of these had negative CT scan results. Eighteen patients (90%) returned to the ED with significant intracranial hemorrhage. Among the patients admitted for observation (n = 45) most (70%) had a brain CT scan; the CT scan result was normal 88% of the time. Despite this, 80% of the admitted patients had a significant clinical deterioration and development of new hemorrhages on repeated CT scan. The authors recommended that all patients taking warfarin who present with an MTBI have their INR levels determined, that all patients with supratherapeutic INR levels be admitted for observation even if the initial brain CT scan result is normal, and that INR levels should be reversed at least to therapeutic levels.

Outcome

The existing literature regarding the prognosis of patients with MTBI was reviewed by the WHO Collaborating Centre Task Force in 2004.[31] The review included 120 studies conducted in a variety of disciplines in children and adults. Most studies agreed that most of the patients showed clinical recovery within 3 months to 12 months. Studies involving cognition and memory tended to show return to baseline by 3 months in children and in most adults. A meta-analysis published in 2003 also showed that cognitive deficits tend to resolve by 3 months.[32] However, other authors have reported that cognitive and memory deficits often persist longer than 3 months.[33,34] The heterogeneity of the current literature on memory and cognitive changes after MTBI makes it difficult to make more specific conclusions. The studies are of widely varying quality, and the inclusion criteria and definition of MTBI are rarely consistent between them. In addition, a large number of different outcome assessment tools have been used. These have included computerized[33] and manual[35] evaluations of memory, reaction time, and decision time. Standardized tools such as the Functional Independence Measure,[36] Wechsler Adult Intelligence Scale,[37,38] and the Immediate Postconcussion Assessment and Cognitive Testing Battery (ImPACT)[34] are commonly used, as are a variety of other tools.[38–40]

A variety of other symptoms are often present after MTBI. These may include symptoms such as headache,

dizziness, fatigue, anxiety, depression, irritability, and personality changes. The presence of one or more of these symptoms is often referred to as the postconcussive syndrome (PCS). Specific criteria for PCS have been described,[41,42] but in routine practice, the term is often used for patients with any residual symptoms. In one study by Ingebrigsten et al.,[43] 62% of patients had one or more symptoms at 3 months after injury, with 40% meeting ICD-9 criteria for PCS. In another study, 42% of patients reported the presence of four or more residual symptoms at 3 months.[44] Faux et al.[45] reported the presence of PCS at 3 months after injury in 35% and 25% of Canadian and Australian patients with MTBI, respectively. In contrast, a study by Yang et al.[46] found that only 13% of patients with MTBI had one or more residual symptoms at 8 weeks. This degree of variation is not surprising given the different patient populations and methods for determining the presence of symptoms in each study. However, a consistent finding in most PCS studies is that headache, dizziness, and fatigue are among the most common symptoms.[43,44,46]

The natural history of PCS in patients with MTBI remains poorly understood. Most studies have been hampered by the lack of a control group of injured patients without TBI. Trauma patients in general have a significant incidence of individual symptoms that overlap with PCS, especially those with posttraumatic stress disorder. It has been noted that the high incidence of posttraumatic stress disorder in combat veterans is a significant confounding factor in terms of determining if reported symptoms are truly caused by the blunt head injury itself.[47,48] The biologic basis of PCS is also poorly characterized, and the evidence that structural damage is the cause is weak.[49] Maruta et al.[50] described some functional brain abnormalities in patients with PCS in a highly technical study involving video-oculography, but the clinical correlation is uncertain.

No specific therapy has been shown to be consistently effective for PCS. Analgesics and antidepressants are commonly used. Patients are often referred for psychological evaluation or counseling. One prospective randomized trial by Bell et al.[51] evaluated the effect of a structured series of telephone counseling sessions on patients with PCS. The counseling group did have a reduction in chronic PCS symptoms. To date, this technique has not been described elsewhere.

Return to Activity

Clinicians that deal with patients with MTBI are often asked to speculate when the optimal time of returning to driving should be. There are obvious potential negative consequences of driving a motor vehicle in the presence of impaired memory, attention, or cognitive function. Research in this area has been limited. Preece et al.[52] used a computerized driving simulation to demonstrate that patients with MTBI were slower to anticipate and react to traffic hazards. The same authors also found that patients with MTBI were slower to respond to road hazards than a group of control patients with minor orthopedic injuries.[53] Impaired driving ability in patients with MTBI may not be identified by routine cognitive evaluations.[54] Patients are often unaware of their deficits[54] and may return to driving despite the presence of residual impairment.[55] Most do not undergo a formal evaluation of driving ability after injury.[56] A variety of commercial driving simulation products exist but are not available in most settings, are rarely covered by third party payers, and are not well supported by clinical data. However, structured driving evaluation programs for patients with MTBI have been shown to be logistically feasible.[57] Driving remediation for patients with more significant deficits is also possible but may be labor intensive.[58] At the present time, no specific method of driving evaluation for patients after MTBI is supported by adequate data.

Determining the optimal and safe time to return to work for patients after MTBI is also challenging. Specific guidelines exist for only a few occupations, such as civilian and military aviation.[59] For US military personnel, DoD guidelines recommend a return to work at maximal capacity soon after an initial period of rest.[2] Virtually no guidelines exist for the civilian population. Typically, clinicians can only offer common-sense advice for patients, who in turn are often left to judge for themselves when is the optimal time to return to work. At the present time, there is no evidence to support the routine use of any one specific measurement tool for determining the optimal return to work time after MTBI in the general population.

The EAST MTBI PMG committee also attempted to include guidelines regarding the management of concussion in athletes. This body of literature was diverse and extensive enough that a comprehensive review was thought to be beyond the scope of this PMG. The committee did review a number of well-known studies conducted in athletes. In the 2003 NCAA Concussion Study, athletes with a history of concussion were found to be at higher risk for the development of subsequent concussions, which were in turn found to be associated with a longer recovery time.[60] The same group noted that postconcussive symptoms were nearly resolved within 7 days in most athletes.[61] It has been suggested that postconcussive symptoms may tend to last longer in female athletes.[62] Athletes may be reluctant to report concussions owing to concerns over loss of playing time.[63] A great deal of effort has been made in a variety of different sports to determine the proper time for return to play after a concussion. Player evaluations are typically done by a team's coaching or medical staff. These are normally kept confidential, and these data have not been systematically reported in peer-reviewed journals or databases. As such, the PMG did not attempt to generate specific guidelines for return to play in athletes.

Neuropsychological Testing and Cognitive Rehabilitation

The role of neuropsychological testing for MTBI remains unclear. Formal neuropsychological evaluation may identify a variety of cognitive,[64] behavioral, or other deficits.[65] Limited data exist to guide the clinician on which patients to refer for such an evaluation. Studies on this topic tend to suffer from a variety of weaknesses as outlined by Sherer et al.[66] The impact on patient outcome is also uncertain. It has been speculated that this therapy may be more useful for MTBI than for moderate and severe TBI.[67] However, in another study of patients with significant PCS, neuropsychological therapy did not lead to a decrease in symptoms.[68]

Patients with MTBI are often referred for other types of rehabilitation. These include interventions aimed at improving

memory, attention, and other types of executive function. They may be conducted by practitioners in a variety of disciplines, including speech therapy, occupational therapy, physical therapy, and others and are collectively referred to in this PMG as *cognitive rehabilitation*. The recent literature on this subject has been systematically reviewed by Cicerone et al.[69,70] and subject to a meta-analysis by Rohling et al.[71] Although there is evidence to support the use of cognitive rehabilitation to improve memory, communication, and executive function,[69,70,72] most of the studies were not specific to MTBI. Patients with other neurologic disorders and with all types of TBI are often grouped together in these studies so that patients with MTBI often constitute only a small percentage of patients in a given study. Other methodologic problems with these studies are also common.[70,71] Currently, no specific set of indications for referral for cognitive rehabilitation after MTBI have been defined, and its impact on patient outcome is unknown.

Biochemical Markers

An increasing amount of research in recent years has been done on the use of biochemical markers for patients with TBI. These have included S-100B,[73–76] serum tau,[76–78] neuron-specific enolase,[79] and others.[75,80] These molecules are present in the brain and may be detected in the serum after brain injury. For patients with MTBI, possible uses for biochemical markers include screening to determine which patients should receive a brain CT scan[75,77] and for determining prognosis.[74] One prospective study showed that patients with elevated S-100B levels were more likely to have intracranial hemorrhage on brain CT scan.[75] However, there was a degree of overlap in S-100B levels for patients with and without brain injury. Therefore, the sensitivity and specificity of S-100B levels for the presence of intracranial hemorrhage will vary depending on which cutoff level is used.[75] S-100B levels may also be affected by alcohol intoxication.[73] Serum tau has also been examined but was not useful for determining which patients had an intracranial hemorrhage in one study[75] and was not useful in predicting outcome in another.[76] Neuron-specific enolase has examined in one study, but the outcome variable measured (Glasgow Outcome Score) was not well suited to evaluate patients with MTBI.[77] Molecular markers may ultimately be found to be more useful for patients with severe TBI.[75,80] At present, there is insufficient evidence to support the use of these biochemical markers in the clinical management of individual patients with MTBI except in the context of a research protocol.

CONCLUSION

Some recommendations from the 2001 EAST MTBI guidelines are essentially unchanged in this update. However, a number of alterations and additions have been made. The previous admonition to obtain a brain CT scan in all patients with suspected brain injury has been modified to reflect the use of standardized criteria (such as the CCHR) in some centers to identify patients that require a CT scan. The indications for ED discharge of patients with MTBI were examined in a number of high-volume studies since 2001, and our recommendation was changed to Level II to reflect this. Two specific recommendations on anticoagulated patients with MTBI were added.

MTBI will remain a significant public health problem for the foreseeable future given the significant socioeconomic costs associated with it. The high incidence of MTBI makes it theoretically amenable to high-quality clinical trials. These would be facilitated by the use of a consistent definition and terminology among different disciplines. Newer imaging modalities and more sophisticated outcome measurement tools may also give more insight into the optimal management for MTBI.

AUTHORSHIP

The study was designed by the EAST Practice Management Guidelines Committee. R.R.B, S.E.R., R.J. and J.M.W. conducted the literature searches. All authors participated in the analysis of articles and in the formation of guidelines. R.R.B. and S.E.R. wrote the manuscript. All authors participated in critical revision and approved the final version.

DISCLOSURE

The authors declare no conflicts of interest.

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