To do or not to do? Magnetic resonance imaging in mild traumatic brain injury

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(Received 29 August 1999; accepted 12 June 2000)

Clinical quantification of mild traumatic brain injury (MTBI) patients should be based on Glasgow coma scale (GCS) score, duration of loss of consciousness (LOC) and post-traumatic amnesia (PTA). In addition, a short practicable neuropsychological test might be useful in detecting minor memory and attentional deficits. MRI appears to be the most sensitive imaging method for assessing MTBI so far, but information regarding a visualized lesion is not usually utilized in the classification of MTBI. Magnetic resonance imaging (MRI) should, therefore, play a major role in any MTBI classification scheme. An appropriate MRI protocol has to be chosen using at least $T_1$ weighted, $T_2$ weighted, proton density and gradient-echo (GRE) sequence images, all in at least two planes, in order to detect and classify all lesions precisely. Owing to the fact that acute lesions may be missed, it is advisable to perform MRI in the first 2 weeks following trauma. Further research is necessary to clarify the relationship between chronic symptoms after MTBI and MRI abnormalities. It may, thus, be possible to provide optimal strategies for emergency department management, to define a group of patients with a need for acute and rehabilitative intervention after MTBI, and to predict their outcome.

Introduction

Mild traumatic brain injury leads to acute and possibly chronic symptoms. The former include headaches, dizziness, nausea, vomiting, irritability, and sensitivity to light and noise. Attentional and memory deficits, lack of insight, rapid fatigue and a need for extended sleep compatible with an early deficit in arousal, have been reported [1–3].

After 3 months or more, a group of patients still complained of chronic symptoms such as impairment of cognitive function resulting in failure to cope with work, increasing fatigue after periods of increased activity, behavioural changes including bad temper, irritability and poor insight, symptoms such as sensitivity to light and noise, paradoxical insomnia, headaches due to head and neck injury or to stress, and reactive symptoms in the form of depression or anger [2].

Many attempts have been made to classify MTBI on the basis of early or late clinical signs [4]. Definitions vary widely, due to the complexity of cerebral injuries. The severity of MTBI is defined by acute injury characteristics as the initial GCS score, duration of LOC and PTA [1]. Focal neurological signs should be absent.
Neuroimaging studies should be negative, but this defining characteristic may be more complex [1].

A definition of MTBI was developed recently by the Head Injury Interdisciplinary Special Interest Group of the American Congress of Rehabilitation Medicine with respect to the wide spectrum of injury severity: (a) any period of LOC of <30 minutes and a GCS score of 13–15 30 minutes later, (b) any loss of memory for events immediately before or after an accident, with PTA of <24 hours, (c) any alteration in mental state at the time of the accident, and (d) focal neurological deficit that may or may not be transient [5]. Mechanisms of trauma were also discussed. CT, MRI, EEG or routine neurological evaluations may be normal. After varying lengths of time, patients can exhibit persistent physical, emotional, cognitive and behavioural symptoms, which may result in functional disability.

Various authors have tried to break down MTBI further, particularly with the aim of finding additional prognostic factors or of determining fitness in order that concussed athletes may return to play [6, 7].

Common clinical parameters such as the GCS score, duration of LOC and PTA do not correlate strongly with the presence of structural lesions visualized by neuroimaging techniques, even in patients with very mild TBI [8]. By mutual clinical agreement, it is questionable as to whether a patient may be defined as MTBI if structural lesions exist [1]. However, with the increasing sensitivity of imaging methods, more and more structural lesions are being found, even in MTBI patients lacking both loss of consciousness and very short or post-traumatic amnesia [9, 10]. Furthermore, neuropsychological dysfunction does not correlate with specific lesion patterns.

A neuroimaging revolution started with the era of CT. Advantages of CT scanning in TBI patients are the wide availability, the short examination time, and lower costs. Limitations, however, are the relatively low contrast resolution, low sensitivity for non-haemorrhagic lesions, and the difficulty of obtaining multiplanar images. Small lesions may be indistinct on CT scans. In contrast to CT investigation, MRI offers multiple imaging planes for classifying the type of traumatic lesion by means of visualization of size, shape, distribution and location of the lesions. The superiority of MRI in detecting intracranial lesions and diffuse axonal injury in mildly head injured patients has been demonstrated in various studies [9, 11–14].

The prevalence of MRI abnormalities in MTBI is still unclear. There are only a few MRI studies which have prospectively investigated only MTBI patients. Doezema et al. [15] showed traumatic intracranial abnormalities in six out of 58 patients discharged from the emergency room after minor head injuries. Three patients had cortical contusions and three small subdural haematomas. Mittl et al. [14] suggested that diffuse axonal injury (DAI) lesions can be identified in ~30% of MTBI patients. Even in very mild TBI, three out of 12 patients had contusions (figures 1(a) and (b)), DAI lesions and an epidural haematoma [8].

Pathophysiology of MTBI

Studies dealing with closed head injury have particularly included subjects with more severe injury. Although the extent of the lesions varies according to the degree of closed head injury, it has been shown that the type of lesion in MTBI
is similar [16, 17]. While DAI caused by acceleration/deceleration trauma appears to be the major neuropathological substrate of all traumatic brain injuries, there is merely less damage in MTBI [1, 17, 18]. Whiplash injuries and assault have been reported as more common causes of MTBI in the US [19]. Both in histopathologic studies on animal models and in closed head injury patients, as well as in neuroimaging studies, asymmetrically distributed lesions have been found to be mainly located in the subcortical white matter, corpus callosum, and upper brain stem [20–23]. The lesions were ovoid, with the long axis parallel to the fibre bundles, ranging in size from 1–15 mm. Due to the substantial effect of shearing forces, the common locations of DAI lesions are especially adjacent tissues varying in density and rigidity, e.g. cerebral gray and white matter. Two forms of axonal injury are suggested: (a) primary axotomy at the time of impact, and (b) secondary axotomy, whereby cytoskeletal dissolution proceeds to axonal swelling after ~12–18 hours, followed by axonal retraction [18, 24, 25]. Small vessel injury can lead to haemorrhagic DAI lesions, predominantly in the gray matter, owing to better vascularization.

Cortical contusions, rarely described in MTBI, are found particularly in the orbitofrontal and temporal regions (figures 1(a) and (b) and 2(a) and (b)) and tend to be more superficial and more likely to contain areas of haemorrhage [26]. Injury to larger blood vessels results in intracerebral haemorrhages. Furthermore, small subdural and epidural haematomas have been described in MTBI patients [8, 15].
A number of factors influence the visibility of traumatic brain lesions. The use of high-field-strength systems involving gradient echo imaging (1.5 Tesla) has improved sensitivity, e.g. for susceptibility effects in acute and chronic haemorrhage [14, 27]. Important influences on a particular pulse sequence are the interval of time since injury, size and anatomic location of the lesion and the presence of haemorrhage [28]. With regard to the dynamics of DAI and haemoglobin degradation products, acute or subacute lesions can only be detected precisely on MRI between 24 hours to ~2 weeks after injury [29]. Smaller lesions, especially, will be more difficult to detect over the ensuing weeks, as oedema subsides. Fairly stable lesions can be found later in the chronic phase.

In general, it is essential to obtain $T_1$ and $T_2$ weighted images in at least two different imaging planes for MR scanning of contusions and haemorrhagic lesions. $T_1$ weighted images are useful for anatomical localization, while $T_2$ weighted images for detection and both types of imaging are necessary for accurate detection and temporal staging of haemorrhagic lesions [28, 29]. Additionally obtained GRE sequences are helpful in identifying haemorrhagic lesions because of the higher sensitivity to susceptibility effects of blood products.

In the acute and subacute phases of DAI, oedema and haemorrhagic lesions are easily detected with high sensitivity by MRI on both sagittal or axial $T_1$ and axial $T_2$ weighted MR images [28, 29]. $T_1$ weighted images are highly sensitive for haemorrhagic lesions, particularly at least 3 days after injury. Small, non-
haemorrhagic lesions are often missed with CT but may be better detected with $T_2$ weighted images on MRI scans. Visualization of oedema and axoplasmatic leakage resulting from axonal disruption will be maximal within the first 2 weeks. Early investigation is, therefore, recommended [28, 29]. Although high-signal-intensity foci in the white matter depicted on conventional $T_2$ weighted spin-echo MR images are non-specific, they should arouse suspicion of shear injury foci in MTBI patients, especially in the younger age group [14]. GRE sequences will provide further information of haemorrhagic lesions. Use of Gadolinium-DTPA will not reveal any further information after MTBI, although rapid, transient blood–brain barrier opening is to be considered in experimental closed head injury, particularly in the first few minutes after the impact [8, 30].

Diffusion is a physical process resulting from the random movement of microscopic particles. The use of the proper pulse sequence permits the acquisition of diffusion imaging, i.e. images in which areas of rapid proton diffusion can be distinguished from areas with slow diffusion. Diffusion imaging is of considerable clinical interest, because it identifies areas of ischemic brain damage at a very early stage, both in animal models and in stroke patients [31]. Many commercial MRI systems have the facility of diffusion spin-echo planar imaging, thus allowing a brain diffusion study within seconds. Nevertheless, the role of diffusion imaging in patients with traumatic brain disease has not yet been studied. Recent MRI techniques, such as diffusion imaging and fluid attenuation inversion recovery sequences (FLAIR), further improve the sensitivity of MRI in head trauma [32, 33].

In the chronic phase of DAI in the detection of non-haemorrhagic DAI lesions, e.g. gliotic scars, proton-density and $T_2$ weighted images, are useful in addition to FLAIR [28]. Due to the susceptibility effects of haemoglobin degradation products such as haemosiderin, haemorrhagic lesions at the chronic stage are better visualized with strongly $T_2^*$ weighted GRE sequences (fast low-angle shot = FLASH) by using a longer TE interval [14, 28, 29]. In the infratentorial region, moderately $T_2^*$ weighted images should be preferred, due to the susceptibility artifacts of the air containing mastoid cells and sinuses adjoining the skull base [28].

**MRI-findings in MTBI**

Small, non-haemorrhagic lesions and surrounding oedema often remain undetected in CT, and there is often a discrepancy between the patient’s clinical condition and the CT findings. The outstanding role of MRI in detecting DAI in mildly head injured patients has been demonstrated [12, 14]. In MRI investigation, DAI is characterized by multiple, small elliptical lesions situated deep under the cortex. According to Gentry [29], ~80% of DAI lesions are non-haemorrhagic, but the proportion of haemorrhagic DAI lesions is much greater than suspected [28]. The majority of DAI lesions are located in the white matter, followed by the most commonly involved area of the corpus callosum, especially in the posterior body and splenium. Lesions in the latter are detected in conjunction with DAI in the lobar white matter. Brainstem lesions are predominantly found in the dorsolateral parts of the midbrain and upper pons, especially in the superior peduncles. They are usually very small and non-haemorrhagic and typically associated with similar lesions in the corpus callosum and the white matter. Patients with brainstem lesions usually present with a longer period of LOC and more clinical symptoms, so this
pattern will probably not be a feature of MTBI [29, 34]. There is, however, some evidence of brain stem dysfunction after MTBI [35].

The prevalence of non-haemorrhagic and haemorrhagic contusions, intracerebral, subdural and epidural haemorrhages is low in MTBI. The neuro-radiological features have been described extensively elsewhere [27–29].

**Functional imaging in MTBI**

SPECT Tc-99m HMPAO imaging appears to be the most clinically useful and available method for cerebrovascular haemodynamic assessment in trauma patients [29]. Especially in mild-to-moderate TBI, there may be some advantages over the morphologic modality of MRI, owing to the higher sensitivity of SPECT brain perfusion imaging [36]. The most common sites of abnormal perfusion tend to be the temporal lobe, frontal lobe and, interestingly, the basal ganglia, even in patients without LOC. A SPECT study of four MTBI patients found minimal correlation compared with neuropsychological findings [37].

**Discussion**

An accurate and consistent definition of MTBI is important in the initial phase, but also in neurorehabilitation management and research [4]. The need for a classification system based on severity of injury is, therefore, strongly recommended. Duration of coma, PTA, history of the mechanism of injury, and neurological examination (including neuropsychological tests) will contribute to clinical definitions. In contrast to Gentry [29] and Parizel et al. [28], it is suggested that only patients with a GCS score of 15 and a LOC of 20 minutes or less can be classified as MTBI [1]. However, even in a clinically homogeneous group of very mild TBI patients, traumatic MRI abnormalities and longstanding neuropsychological dysfunction have been observed [8]. The focal changes demonstrated in MRI may represent the pathologic substrate that underlies the post-concussive syndrome [14]. DAI should, therefore, be suspected in any MTBI patient. The clinical value of DAI for the outcome is rather unclear. It is presumed that the presence of DAI depicted by MRI is not necessarily associated with poor outcome, as reported previously [27–29]. Additional neuropsychological assessment and other outcome measures have not been performed so far in patients with or without the presence of DAI.

Neuropsychological sequelae after MTBI, which are most commonly reported as deficits in attention, learning and memory, reaction time and information processing, are probably due to the pattern of neural injury [38, 39]. If there are no complicating factors, most patients recover in ~1–3 months [40]. A few studies have investigated the relationship between lesions detected by MRI and neuropsychological function in mild-to-moderate TBI patients [41, 42]. Patients were admitted because of their unclear clinical conditions, and patient groups were very inhomogeneous, e.g. GCS score of 15 to 9 and lower. They were, thus, not representative of the range of MTBI, but several observations regarding it were made. The significance of the lesions detected for cognitive function has not yet been established.

MRI tends to be restricted to patients with chronic symptoms both in clinical practice and forensically. However, in the chronic stage, discrete lesions may be
missed and unspecific abnormalities detected cannot be related to trauma in any case. Neuroradiologists, therefore, insist on MRI investigation in the first 2 weeks after any TBI, owing to the course following primary or secondary axonal injury [28, 29].

SPECT studies in mild-to-moderate TBI are problematic in several ways: non-specificity of the findings, lack of correlation between abnormal scans and neuropsychological findings, lack of follow-up studies, absence of a blind comparison with healthy volunteers, and the fact that only retrospective studies have been available in the field of MTBI. Other techniques assessing cerebral metabolism, biochemistry, and haemodynamics (e.g. PET, MR spectroscopy, MR perfusion imaging) are associated with various problems or deficiencies that greatly limit their current clinical usefulness. Primarily, these are high cost, prohibitive expense, limited availability, lack of information or insufficient anatomic information. Because of the experimental status of these investigations, and the assumed low sensitivity for MTBI, more severe trauma patients are studied as individual cases.

References


