ORIGINAL ARTICLE

How important is susceptibility-weighted imaging in mild traumatic brain injury?

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ABSTRACT

BACKGROUND: Mild traumatic brain injury (mTBI) is a public health problem that is recognized as a "silent epidemic" in its late stages due to undiagnosed axonal damage rated 13 and above on the Glasgow Coma Scale (GCS). Injury-related microhemorrhages often cannot be detected on computed tomography (CT) scans and conventional magnetic resonance imaging (MRI). This study aims to investigate whether susceptibility-weighted imaging is feasible in mTBI patients.

METHODS: Fifty-eight patients with GCS scores of 14 and 15 and with symptoms of brief mental fogs, impairment of concentration, memory loss, headache, dizziness, or imbalance after brain injury were examined at the emergency service. A brain CT scan and MRI containing diffusion-weighted and susceptibility-weighted imaging (SWI) sequences were performed on the patients whose symptoms did not seem to alleviate after the sixth hour. Thirteen patients were excluded from this study because of advanced age, diabetes, a history of hypertension or its chronic sequelae, or acute cerebrovascular disease; 45 patients were included in this study.

RESULTS: The patients' CT results were normal, and no diffusion restrictions were observed. The SWI revealed microhemorrhages in seven patients (15.6%). Five of these patients had hyperintense areas in conventional sequences corresponding to the hemorrhages spotted in the SWI. In three of the five patients, these pockets of hemorrhages were higher in number and size in comparison with conventional in the SWI sequence.

CONCLUSION: Susceptibility-weighted imaging, which can be used to assess the presence and severity of microhemorrhages due to diffuse axonal injury, is recommended for determining the cause of symptoms in patients with mTBI, to continue targeted treatment and prevent complications that may develop.

Keywords: Axonal injury; microhemorrhage; mild traumatic brain injury; susceptibility-weighted.

INTRODUCTION

Mild traumatic brain injury (mTBI) that arises from diffuse axonal injury has become a public health problem recognized as a "silent epidemic" owing to its parenchymal changes being difficult to diagnose.[1] Mild traumatic brain injury includes patients with a Glasgow Coma Scale score (GCS) of 13 and above and accounts for 75% of traumatic brain injuries (TBI).[2] Although brain computed tomography (CT) images of these patients may appear normal, somatic, cognitive, or sensorial symptoms may be present. Findings that could corroborate that the symptoms are due to diffuse axonal damage may not be observed on conventional magnetic resonance imaging (MRI). Most of these symptoms, such as headaches, dizziness, short-term memory loss of fewer than 30 seconds, impaired concentration, insomnia, and depression, may subside within a few weeks. However, the symptoms of 10–20% of these patients may last for three months or more. This clinical entity is known as a persistent post-concussive syndrome (PPCS).[3–5] PPCS decreases

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the quality of life and may lead to anxiety, depression, and social dysfunction.[6] After cranial trauma, a CT can be used to assess parenchymal, subdural, and epidural hematomas. The non-invasiveness of brain CT, which also offers quick results, is more advantageous compared with other methods of diagnosis concerning defining acute traumatic lesions and indicating anatomic localization. However, the sensitivity of brain CT for the detection of non-hemorrhagic trauma is lower than that of conventional MRI.[7] Conventional MRI imaging is 30% more sensitive for the detection of diffuse axonal injuries (DAI) in mTBIs.[8] Diffuse axonal injury (DAI) is diagnosed by the presence of pockets of microhemorrhages in subcortical and periventricular white matter areas. FLAIR, one of the conventional MRI sequences, is better at detecting axonal injuries than the T2-weighted sequence (T2W).[9] However, hyperintense foci in FLAIR and T2W sequences cannot distinguish lesions due to aging or microvascular diseases from axonal damage.[10] In contrast, diffusion-weighted imaging (DWI) can differentiate between vasogenic edema and cytotoxic edema. Focal cytotoxic (diffusion restriction) edema regions can also be observed in DAI patients.[11] In serious cases of TBI, DWI can reveal the extent and size of abnormalities (diffusion restrictions in DAI patients) more precisely compared with T2W and FLAIR images.[12] However, to our knowledge, there are no published data to date, suggesting that DWI is useful in patients with mTBI.

Susceptibility-weighted imaging (SWI) is a technique that makes use of the magnetic sensitivity difference among the tissues. SWI can also detect DAI related microhemorrhage sites six times more sensitively than conventional MRI sequences.[13] For mTBIs, there is a need for sequences that support conventional sequences and help confirm the diagnosis.

This study investigates the effectiveness of the SWI sequence in detecting axonal injury in mTBI.

MATERIALS AND METHODS

Approval for this study was obtained from Karadeniz Technical University's Faculty of Medicine Ethics Committee (Approval No.: 2016/18). Patients admitted to our hospital's emergency service over two years due to traumatic incidents, such as traffic accidents, falls or blows, were examined. Patients noted to have a Glasgow Coma Scale score of 13 and higher and a normal brain CT after the neurological examination were considered to have mTBI according to the framework[2] defined by Geurts et al. brain CT was requested if the patients had a loss of consciousness for more than five minutes, difficulty remembering pre-traumatic events after 30 minutes had elapsed, or a dangerous traumatic incident (motor vehicle accident, being thrown out of a vehicle, falling from a height of three feet or more, falling from five or more ladder steps). Fifty-eight patients with a normal CT scan but with post-traumatic headache, dizziness, memory loss of more than five minutes, impaired concentration, insomnia and depression, or ataxia and mild drowsiness during the neurological examination were included in this study.

The symptomatic treatment of patients was started after admission; neurological examinations were then repeated on the 3rd and 6th hour from admission. Brain MRI was performed for patients who did not respond to the symptomatic treatment after the 6th hour from admission had elapsed. DWI and SWI sequences were also added to the conventional sequences.

Brain CT scans were performed using the Toshiba Alexion™ Advance (Toshiba Medical Systems Corporation, Nashua, Japan) computerized tomography device with 16 slices (field of view of 22 cm, 170 mA, 100 kV, thickness 5 mm, images reconstructed to a size of 1 mm).

MRI scans were conducted with a Magnetom Aera (Siemens Healthcare, Germany) MRI device with 1.5 Tesla magnet power and 32-channel head coils. Brain MRI scans were performed with axial T1W (TR:417ms; TE:8.9ms, slice thickness 3 mm), T2W (TR:5480ms; TE:100ms, slice thickness 3 mm), FLAIR (TR:6000ms; TE:86ms; TI:2026ms, slice thickness 3 mm) SWI (TR:49ms; TE:40ms; flip angle 15°, slice thickness 2.5 mm) with maximum intensity projection (MIP) and DWI (EPI diffusion TE:75ms; TR:5100ms; slice thickness of 5), coronal T2W sequences.

Radiological examinations were reviewed together by two radiologists who had at least 10 years of neuroradiology experience, and the results were accepted unanimously.

Thirteen of the patients were excluded due to diffusion restrictions related to microvascular diseases such as hypertension, acute strokes, or a history of ischemic attacks in DWI, T2W and FLAIR sequences. The remaining 45 patients were included in the study. These patients had no other trauma history. No diffusion restrictions were observed on these patients' brains; however, T2W and FLAIR images showed subcortical and periventricular white matter lesions. If the SWI showed no low signals of bleeding in the lesions, this was interpreted as a "secondary microvascular event or gliosis" and subsequently labeled "nonspecific;" if a low signal was noted, this was interpreted as a microhemorrhage secondary to axonal injury. The patients with microhemorrhage were given supportive treatments within a hospital environment until their symptoms showed improvement. Patients were assessed with neurological examinations for ongoing or new symptoms during follow-up every six months over two years.

The Kolmogorov-Smirnov test was used to determine the appropriateness of the data to normal distribution. The test later revealed that the data did not align with the normal distribution. The trauma patterns, Glasgow Coma Scale values, admission symptoms, and SWI findings were categorized. The Chi-Square test was used to compare categorical data. De-

Table 1. Type of symptoms and findings symptoms

Symptoms and findings	n	%	p
Nausea	25	55.6	0.52
Headache	16	35.6	0.31
Vertigo	18	40	0.39
Amnesia	7	15.6	0.54
Vomiting	5	11.1	0.90
Stupor	1	2.2	0.06

mographic data, such as information on age and gender, were retrieved. The median value (min.-max.) was obtained with descriptive statistics, as the data did not align with the normal distribution. If the P-value was less than 0.05, the results were considered significant.

RESULTS

Twenty-three of the 45 patients were male (51.1%), 22 were female (48.9%), and their mean age was 47.93±21.49. There was no difference between the mean age of male patients (45, min. 18, max. 86) and female patients (51, min. 18, max. 99) (p=0.19). The most common form of trauma suffered by the patients was falls (62.2%). The most common symptom was nausea (55.6%) (Table 1). At the time of admission, the GCS was 15 in 44 patients and 14 in one patient.

Table 2. Comparison of age, number of lesions on MRI, and lesion of the patients with microhemorrhage

Patients	Age (years)	DWI*	T2*	FLAIR*	SWI*	Boyut artışı
1	21	–	4	4	6	+
2	70	–	2	2	2	–
3	59	–	–	–	1	
4	78	–	4	4	6	+
5	67	–	2	2	2	+
6	82	–	2	2	2	–
7	67	–	–	–	5	

*Number of lesions. MRI: Magnetic resonance imaging; DWI: Diffusion-weighted imaging; FLAIR: Fluid-attenuated inversion recovery; SWI: Susceptibility-weighted imaging.

The SWI showed hypo-intense, microhemorrhage areas in seven of the 45 patients (15.6%; Table 2). There was no restricted diffusion in any focus. In five of the seven patients, these areas were hyper-intense in T2W and FLAIR. In three of these patients, the microhemorrhage fields shown by the SWI were significantly larger in size and number than the hyper-intense areas in FLAIR (Figs. 1 and 2). In the other two patients, the SWI showed hypo-intense microhemorrhage areas, while there were no intensity changes in FLAIR and T2W (Fig. 2 and 3). The neurological examination performed at

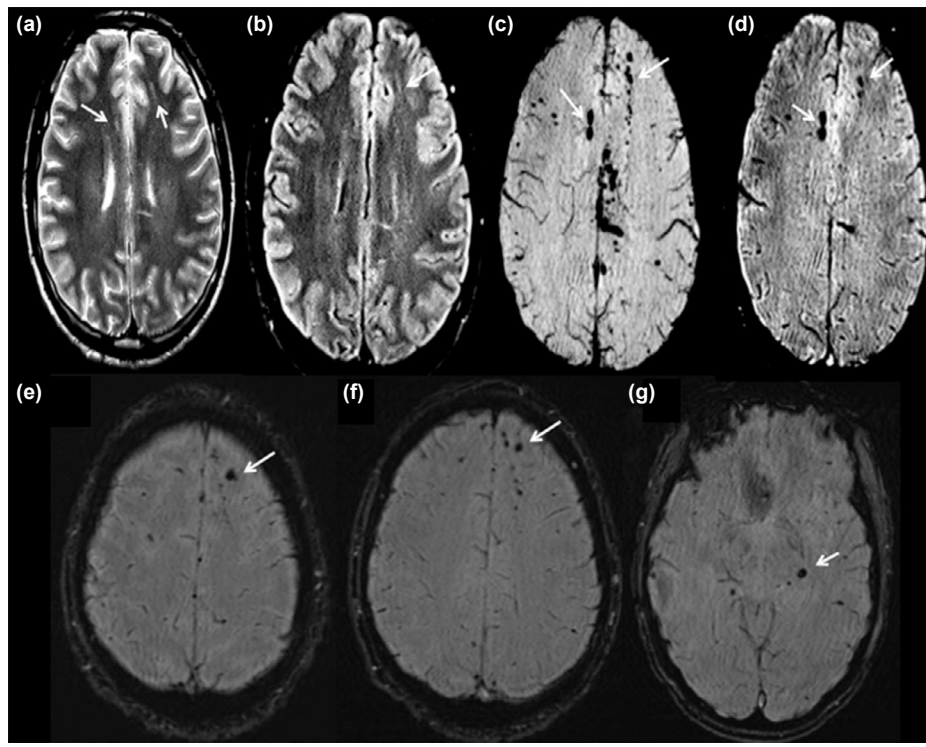

Figure 1. A male case with mTBI. Pathology was not observed in CT. In MRI: **(a)** T2W and **(b)** FLAIR; hyperintense nonspecific were observed in the corpus callosum genus and left centrum semiovale (arrows), while in the **(c)** SWI-MIP and **(d-g)** SWI; there are increases of number and size of the lesions: bilateral frontal white matters and left para hippocampal area (arrows).

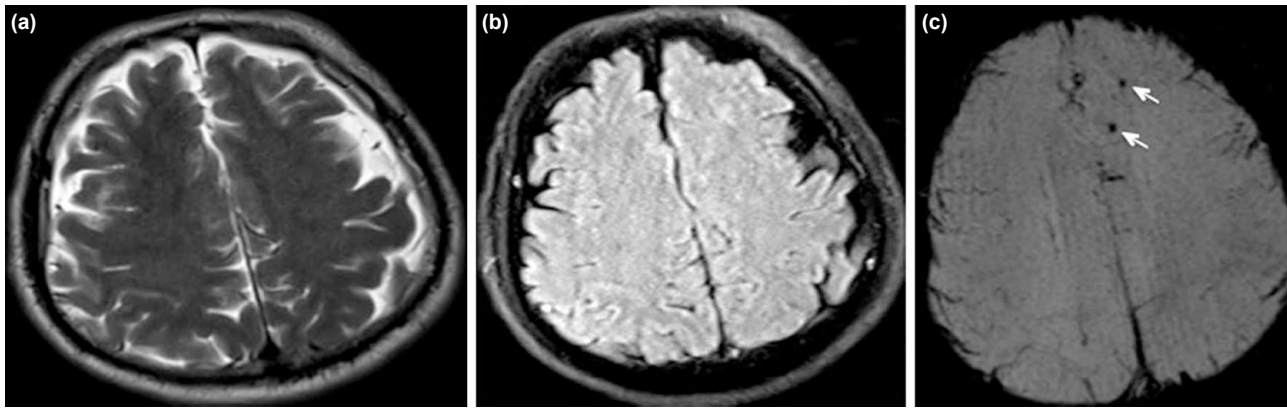

Figure 2. A female case with mTBI. Pathology was not observed in T2W and FLAIR **(a, b)**. SWI MIP images show a lesion at the level of in the left centrium semiovale **(c)** (arrows).

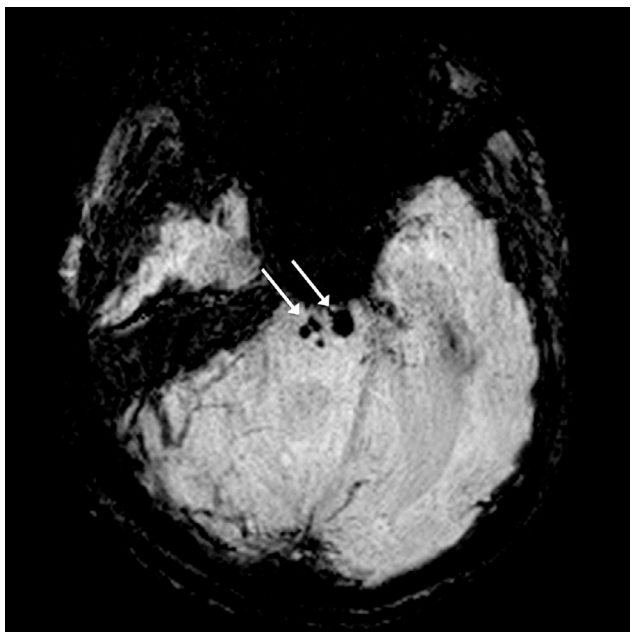

Figure 3. A male case with mTBI. SWI MIP image shows microhemorrhage lesions at the bulb level (arrows).

six-month intervals for two years revealed the development of no new finding in any of the patients.

DISCUSSION

In the present study, there were no specific findings in the brain CT and conventional MRI examinations of patients with posttraumatic mTBI. However, there were heterogeneous symptoms. In these patients, microhemorrhage was detected in the SWI sequence in localizations consistent with nonspecific changes in T2W and FLAIR. As opposed to conventional sequences, lesions were more common and larger in SWI. Thus, it is possible to say that symptoms had developed due to DAI.

With the diagnosis of posttraumatic DAI, the patients' stays in the hospital were extended, and they were kept under surveillance for 24 hours. They received conservative treatment within this period. We recommended that they avoid life-threatening risks that could result in recurrent trauma after their discharge. Neurological examinations were repeated over a period of six months for two years; there were no long-term complications noted.

DAI is suspected in the presence of heterogeneous symptoms in patients with mTBI.[14] The DAI can be concentrated in a small area or may extend over a wide area along the diffuse axon. The use of advanced MRI techniques has recently provided important benefits because the absence of symptomatic findings in CT or conventional MRI cannot exclude the possibility that the symptoms do not have an organic background in mTBI patients. With the SWI sequence, the size of the microhemorrhage and the presence of additional foci can be shown more clearly, and it is also useful in the identification of traumatic microhemorrhages and hemorrhagic DAI lesions in chronic stage mTBI patients.[15] Furthermore, microhemorrhages have been the subject of research in many other diseases, such as strokes, amyloid beta-related angiitis, and Binswanger's disease.[16–18]

The presence and prevalence of DAI in mTBI is an important indicator of long-term cognitive and neuropsychiatric disorders.[19,20] Athletes, such as boxers and football players, have been diagnosed with the progressive neurodegenerative syndrome, also called chronic traumatic encephalopathy, after postmortem histochemical and immunochemical tissue analyses of the brain following repeated blunt head traumas were performed. Suicides were also reported in some cases of the disorder.[21] Kanayama et al.[22] found changes in the cortical and hippocampal cytoskeletal proteins in recurrent mTBIs not occurring with a single trauma.

The number of studies confirming that the sensitivity of MRI in showing microhemorrhages is higher in T2* and SWI sequences is increasing. Although the T2* sequence is sensitive in the detection of microhemorrhages, microhemorrhages cannot be detected with this sequence in 25% of patients with cerebral amyloid angiopathy.[23] The SWI sequence is

a new sequence that creates contrasts using the magnetic susceptibility differences of the tissues and is 25% better than the T2* sequence for detecting hemorrhages and 37.5% better than the conventional sequences.[24] In our patients, intense nonspecific changes were observed in conventional sequences, and the SWI detected microhemorrhage areas in only two of the patients.

According to the American College of Radiology (ACR) Appropriateness Criteria, among imaging modalities, MRI is 'usually not appropriate' for imaging minor and mild head traumas with a GCS of >13.[25] Similarly, Tavender et al.[26] did not consider MRI as an appropriate initial imaging modality in this patient group. In the present study, the patients with mTBI had a GCS of ≥14. The patients had subjective complaints, and some (15.6%) patients also presented with bleeding foci in the white matter. Thus, we consider that performing MRI and especially obtaining the SWI sequence might be beneficial in mTBI cases with subjective complaints.

The current study has few limitations, such as its single-centered design and relatively small sample size. Furthermore, including a control group comprising individuals with similar symptoms to neuropsychiatric-cognitive disorder but no history of trauma would have provided more interesting results. However, we were not able to achieve this due to the difficulty of recruiting volunteers. Nevertheless, it seems clear that these individuals would not have as much hemosiderin accumulation as mTBI patients.

To conclude, the SWI sequence is one of the new MRI sequences that may be helpful in determining the diagnostic and treatment modalities in patients with mTBI due to the additional data it provides. It can also aid in patient follow-up. Routine use of this sequence in patients with mTBI can help achieve short-term stabilization of patients during the healing process and, in the long term, prevent complications through protection from recurrent trauma if DAI is present.

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Conflict of Interest: None declared.

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ORİJİNAL ÇALIŞMA - *ÖZET*

Hafif travmatik beyin hasarında duyarlılık ağırlıklı görüntüleme ne kadar önemli?

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AMAÇ: Hafif travmatik beyin hasarı (mTBI), Glascow Koma Skalası (GCS) 13 ve üzerinde olan, tanı alamayan aksonal hasar nedeniyle geç dönemde 'sessiz epidemik' olarak adlandırılan bir halk sağlığı sorunudur. Bilgisayarlı tomografi (BT) ve konvansiyonel manyetik rezonans görüntülemede (MRG) hasara bağlı mikrohemorajiler çoğunlukla görüntülenememektedir. Bu çalışmada, MRG sekanslarından duyarlılık ağırlıklı görüntülemenin (SWI) mTBI olgularında uygulanabilir olup olmadığının araştırılması amaçlanmıştır.
GEREÇ VE YÖNTEM: Beyin hasarı sonrası GCS'si 14 ve 15 olan, kısa süreli bilinç bulanıklığı, konsantrasyon zorluğu, hafıza kaybı, baş ağrısı, baş dönmesi, dengesizlik gibi semptomları bulunan 58 hasta acil serviste değerlendirildi. Altıncı saatten sonra semptomları gerilemeyen hastalara beyin BT ve difüzyon ağırlıklı ve SWI sekanslarını içeren MRG çekildi. On üç hasta, ileri yaş, diyabet, hipertansiyon öyküsü ve kronik sekel değişiklikler, akut serebrovasküler hastalık bulgusu bulunması nedeniyle dışlandı.
BULGULAR: Olguların BT bulguları normaldi, difüzyon ağırlıklı sekanslarda difüzyon kısıtlanması izlenmedi. Yedi hastada (%15.6) SWI'da mikrohemoraji odakları saptandı. Bunların beşinde SWI'daki mikrohemoraji alanları konvansiyonel sekanslarda hiperdens odaklar olarak izlendi. Bu olguların üçünde ise mikrohemoraji odakları sayı ve boyut açısından SWI sekansında T2A ve FLAIR'a göre artmıştı.
TARTIŞMA: Diffüz aksonal hasara bağlı mikrohemoraji varlığını ve ciddiyetini değerlendirebilen SWI, hafif beyin hasarlı (mTBI) hastalarda semptomların nedenini açıklamak, nedene yönelik tedavisini sürdürmek ve gelişebilecek komplikasyonları önlemek için tavsiye edilir.
Anahtar sözcükler: Aksonal hasar; duyarlılık ağırlıklı; hafif travmatik beyin hasarı; mikrohemoraji.

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