Turkish Neurosurgery 2008, Vol: 18, No: 1, 1-9

Magnetic Resonance Imaging Finding in Severe Head Injury Patients with Normal Computerized Tomography

Normal Kranial Tomografi Bulguları Olan Ağır Kafa Travmalı Olgularda Manyetik Rezonans Görüntüleme Sonuçları

Ayhan KARA[1]
Suat Erol ÇELİK[2]
Sedat DALBAYRAK[3]
Mesut YILMAZ[4]
Gür AKANSEL[5]
Gürcan TİRELİ[6]

[1,2] Beyoğlu Göz Eğitim ve Araştırma Hastanesi, Neurosurgery Department, İstanbul, Turkey
[3,4] Kartal Eğitim ve Araştırma Hastanesi, Neurosurgery Department, İstanbul, Turkey
[5,6] Kartal Eğitim ve Araştırma Hastanesi, Neuroradiology Department, İstanbul, Turkey

Received: 28.09..2007
Accepted: 20.10.2007

Correspondence address:
Suat Erol ÇELİK
E-mail : suaterolcelik@yahoo.com
Phone : +90 212 2516910
Fax : +90 212 2455463

ABSTRACT

AIM: Computerized tomography (CT) has been the primary diagnostic instrument of neurosurgical centers for head injury until now. However, there is a subgroup of patients who have persisting neurological deficits without visible pathology on CT.

MATERIAL and METHODS: Between 2000 and 2002, 3000 patients were enrolled in this prospective study in emergency unit. There was a total of 124 patients (4.1%) who had persisting severe neurological deficits with normal findings on repeated CT scans. These patients underwent 1.5 Tesla cranial MRI study between posttraumatic days 2 and 10 (5.2±4.5). Data on epidemiological, clinical, radiological factors and final outcome were collected.

RESULTS: 76 (61.2%) of the patients were male and 48 (38.7%) were female. 113 (91.1%) of the 124 patients showed significant primary pathology on MRI study. The most common pathology was shear injuries in 75 (60.4%) followed by cortical contusion in 45 (36.2%) cases. 27 (21.7%) patients showed primary brain stem lesions and 8 (0.6%) patients showed subcortical gray matter lesions. 32 patients showed mixed lesions. There was one traumatic aneurysm detected.

CONCLUSION: The MRI examination in head injury is informative in detecting subtle lesions when the neurological condition cannot be explained by CT.

KEY WORDS: Computed tomography, Magnetic resonance imaging, Severe head injury, Shear injury

ÖZ

AMAÇ: Kranial bilgisayarlı tomografi bulguları normal olan ağır kafa travması olgularında, kranial manyetik rezonans görüntülemenin kullanılabilirliği araştırılmıştır.

YÖNTEM ve GEREÇ: Kartal Eğitim ve Araştırma Hastanesi Acil Servisine 2000-2002 tarihleri arasında başvuran 3000 olgu incelendi. Bunların 124 (%4.1)ünde tekrarlanan tomografi sonuçları normaldi ve bu olgular posttravmatik 2-10 günler arasında 1.5 tesla MRI ile incelendiler. MRI bulguları ve klinik sonuçları değerlendirildi.

BULGULAR: En sık gözlenen bulgu 75 (%60.4)olguda aksonal travmalardı. İkinci sıklıkta 45 (%36.2) olguda kortikal kontüzyonlar izlendi.27 (%21.7) olguda primer beyin sapı lezyonları ve 8 (%0.6) olguda subkortikal gri cevher lezyonları,1 olguda da travmatik anevrizma izlendi. 32 olguda mikst lezyonlar izlendi.

SONUÇ: MRI görüntüleme tekrar edilen tomografi bulguları normal olan ağır kafa travmalı olgularda, nörolojik durumla uyumlu patolojiyi göstermede önem taşımaktadır

ANAHTAR SÖZCÜKLER: : Bilgisayarlı tomografi, Manyetik resonans görüntüleme, Ağır kafa travması, Diffüz aksonal travma

INTRODUCTION

The diagnosis and management of head injury was revolutionized after the introduction of CT into the clinical practice. The early diagnosis of intracranial major hemorrhages and calvarial changes allowed the clinician to initiate rapid surgical intervention. Parenchymal abnormalities on the CT such as edema, ventricular compression, hydrocephalus or shifting effects of midline structures dictates the therapeutic options to the surgeon and predicts the prognosis (6). As a relatively low-priced diagnostic tool, CT has high availability in most trauma centers. However, there is a subgroup of patients who have continuation of neurological deficits with normal CT findings (20). Specifically, CT has relatively poor diagnostic yield in the posterior fossa and temporal tip lesions due to bony artifacts; also small hemorrhages, contusions and axonal injuries may be overlooked due to the low resolution (25).

MRI is a new diagnostic tool for imaging craniocerebral trauma but it has some particular restrictive specifications. MRI is more expensive. It requires compatible ventilator equipment in patients in a deep coma and MR imaging is somewhat cumbersome to perform in patients requiring ventilation. It also has limited capability to screen acute phase hemorrhages and calvarial fractures (25). In addition to all the negative factors, there is also no cumulative neurosurgical experience on MRI investigation in craniocerebral trauma so far. To the author's opinion, MRI has significant sensitivity for the majority of intracerebral lesions after the third posttraumatic day (21), especially in patients with persisting neurological deficit and initially normal CT findings. In the concept of the study, the authors tried to elucidate the diagnostic capability and therapeutic implications of MRI after severe head injury in one of the foremost national trauma centers.

CLINICAL MATERIAL AND METHODS

The study was prospectively prepared and planned at the Kartal Educational State Hospital, one of the biggest trauma centers of Istanbul. The study was reviewed and approved by the ethics committee of the hospital. Three thousand patients admitted within six hours after the trauma were enrolled between January 1999 and September 2002. Minor/mild head injuries and penetrating injuries were excluded. The patients with severe head injury or GCS equal to or less than 10 were further investigated. The patients with explanatory CT findings on their neurological status, such as epidural, intracerebral hemorrhage or depressed cranial fracture were excluded. 221 patients who had persisting neurological deficits underwent secondary CT scanning within 24 hours after admission. The operated patients and those who died in this period were also excluded. The remaining 124 patients who had normal CT findings underwent both CT and MRI investigation between the 2nd and 10th posttraumatic days. The demographic characteristics of these 124 patients are shown in (Table I). MRI investigations were performed as early as possible for all patients when vital functions were considered stable.

CT was performed using a third generation General Electric CT SYTEC 3000 scanner (General Electric Co. Medical Systems Division, Milwaukee, Wisconsin/USA). The CT images were obtained using one centimeter slices (cm) on supratentorial and 0.5 cm thickness slices on infratentorial cranial space with contrast enhancement. MRI was performed on a Philips NT 15 (1.5 Tesla) magnetic field (Philips Medical Division, The Netherlands). Two spin echo techniques or sequences were utilized. T1-weighted images obtained with either an inversion-recovery sequence with a repetition time (TR) of 2000-2300 msec and inversion time (TI) of 500-600 msec or a spin-echo sequence with TR of 400-1000 msec and echo time of (TE) 25-40 msec. T2-weighted images were obtained by using a spin-echo sequence with TR of 2300-2900 msec and TE of 80-120msec. Both T1- and T2-weighted images were obtained in coronal, axial and sagittal planes for all patients.

Table I: The Demographic Data of 124 Patients

Sex	Age	Admission GCS	Type of Trauma
76 (Male)	61 (15-40)	65 cases (4-7)	108 (Traffic accident)
48 (Female)	63 (41-70)	59 cases (8-10)	16 (Fall)

After completing the MRI examination, an attempt was made to investigate any intracranial traumatic lesion or abnormality. The authors (neurosurgeons and two neuroradiologists) tried to differentiate secondary lesions (such as cerebral infarction, hypoxic injury, shift effect and edema) from the primary lesion. The primary lesions were classified as follows: 1: Shear injuries with or without an affect on the corpus callosum and basal ganglia, 2: Contusions of cerebral cortex. 3: Subcortical gray-mater injuries, 4: Primary brainstem injuries. These primary lesions were further divided into nonhemorrhagic or hemorrhagic subsets because of treatment differences. We followed two different criteria to define a hemorrhagic lesion on MRI. The hemorrhage presented as hyperintense areas relative to the normal brain tissue on T1-weighted images as the paramagnetic properties of methemoglobin shortens the relaxation time. Secondary hemorrhage displayed as a central hypointensity on T2-weighted images (10). A hyperintense appearance on T1-weighted images with more than 95% sensitivity is predictable for hemorrhagic axonal injuries. When a similar hyperintense signal was present in T2-weighted images, it will show a nonhemorrhagic lesion with 93% sensitivity (25). The observed pathologies in the slices were compared, classified and the total number of lesions for each category was matched.

STATISTICAL ANALYSIS

Data are expressed as means ± standard error of the mean. A one-way analysis of variance test was used to compare the data. Statistical significance was assumed at an error probability of $p<0.05$. Statistical analyses were performed using the Sigma Stat software (Jandel Scientific, SPSS, Erkrath, Germany).

RESULTS

The demographic characteristics of the patients are shown in Table 1. 113 of 124 patients had significant primary lesions on MRI. After the classification of these lesions; the most common pathology was shear injuries (SI) (Table II) (Figure 1). SI lesions were generally less than 1cm diameter and typically present in the white matter or gray-white matter boundary (corticomedullary junction) in the cranial space. The characteristic findings of SI are multiple, small and focal signal abnormalities of white matter. SI was present in 75 (60.4%) patients in this series. 61.3% of this subgroup had multicenter lesions. The majority of SI lesions were nonhemorrhagic (82.3%) and hemorrhage was observed only in a small percentage of patients (17.7%). The nonhemorrhagic lesions could be identified easily in T2-weighted slices with 100% and 72% sensitivity on T1-weighted slices. CT has no capability to demonstrate SI if there is no significant hemorrhage. The majority of nonhemorrhagic SI lesions were primarily observed in frontal (25%) and temporal (14.6%) localizations (Table III). Lesions were also observed in the internal capsule, corona radiata and corpus callosum. Corpus callosum was another common site for SI, making up 23.1% of all lesions. The lesions were placed primarily on the splenium (60.5%), and also on the body (28.9%) and genu (10.5%) of corpus callosum. Hemorrhagic lesions of SI have a tendency to be located in internal capsule and corona radiata of the parietal lobes. 8.3%

Table II: Classification and Frequency of Primary Traumatic Brain Lesions in 113 patients

Type of lesion	No of patients	Multiplicity	Ob Tot Les	Hem/ Non
SI	75	56	164	29/135*
Cortical contusions	45	16	131	117/14
Pr. brain stem lesions	27	6*	37	13/24*
Subcortical gray matter	8	6	15	8/7
Traumatic aneurysm	1	0	1	1

* Significantly different than other groups ($p<0.01$). No of patients: number of the patients, Ob Tot Les: observed total lesions, Hem/Non: hemorrhagic/ non hemorrhagic.

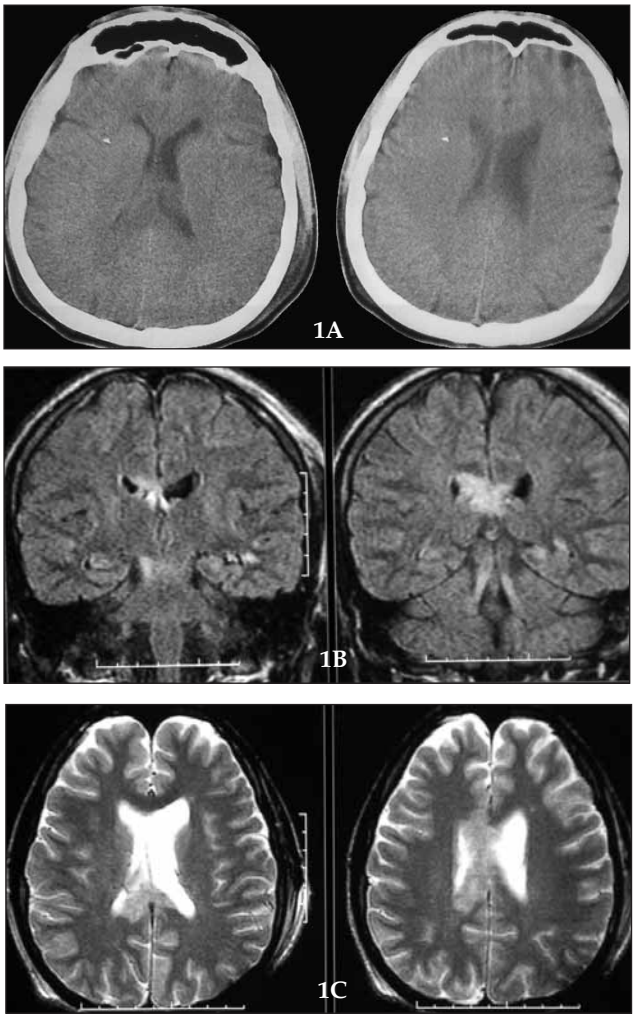

Figure 1: *(**A**) Slices of tomography show only subgaleal edema on the left side but no parenchymal pathology. T1 (**B**) and T2 (**C**) slices of MRI of the same patient show diffuse axonal injury settled of corpus callosum without parenchymal edema.*

of SI lesions were on the corona radiata and nearly half of these lesions (47%) were hemorrhagic. 14.6% of the SI lesions were in the capsula interna and the majority of them were hemorrhagic. GCS of the patients was significantly lower when the patients had multiple lesions. The initial GCS was 7.2±3.1 for single lesion and 5.5±2.6 for multiple lesion patients (p> 0.05).

The second relevant pathology was cortical contusion (CC) (Figure 2). These lesions were traumatic abrasions of cortical surface and generally larger than diffuse axonal injury, often being 2-4cm in the maximum dimension. There were 45 patients (35%) with cortical contusions and all were located in or near the cortex. MRI is highly sensitive for

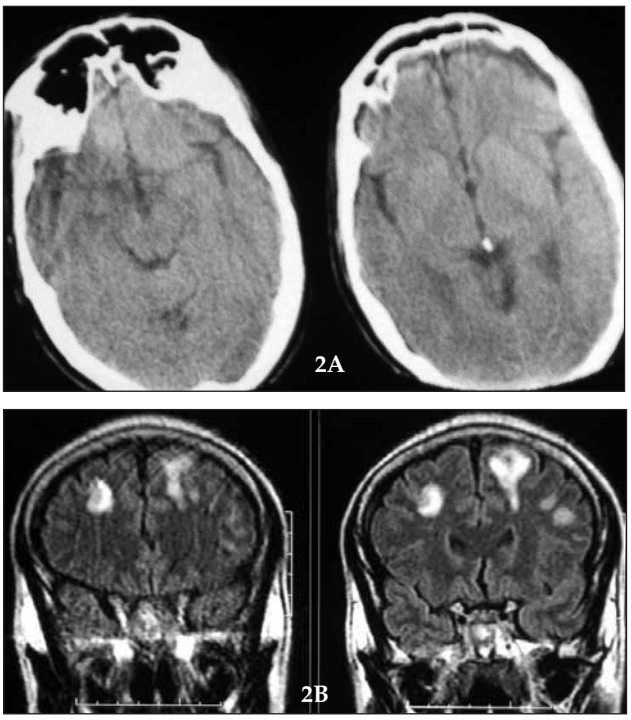

Figure 2: *(**A**) Posttraumatic computed tomography slices show normal appearance of basal sisterns and periventricular area. (**B**) T1 slices of MRI show multiple settlements of cortical contusions in the same patient.*

detection of contusional lesions on both T1- (92.6%) and T2- (96.3%) weighted slices. Most contusional lesions was hemorrhagic (89.3%) in our series. The lesions were generally settled on the superficial cortex, showing some cortical or dural base. The observed lesions of CC have no effect on white matter and show a completely different nature than ischemic lesions. The majority of CC's were located in the temporal region (50.3%), followed by the frontal (29%) and parietooccipital (14.5%) areas (Table IV). 36.1% of all cortical contusions were multiple and these patients had worse GCS levels than single lesion cases. The initial GCS was 6.6±2.5 for single lesion and 5.1±2.8 for multiple lesion patients (p> 0.05). The majority (63.9%) of the cases with cortical contusions had a calvarial fracture but there was no accompanying lesion observed in parenchymal CT slices.

Twenty-seven patients showed primary brainstem lesions (23.8%) (Figure 3). Nine of these were hemorrhagic (33.3%). Such type of lesions is supposed to occur by initial force (6, 7, 12). The GCS of these patients had decreased according to the placement of lesion/lesions. The mean initial GCS

Table III: Shear injuries/ Distribution of Lesions

Location	Number of lesions		
	Hemorrhagic	Nonhemorhagic	Total
White matter (85)			
Frontal	3	38	41
Temporal	1	23	24
Parietal	5	11	16
Cerebellum	0	4	4
Capsule interna (24)	4	20	24
Corpus callosum (38)			
Genu	0	4	4
Body	2	9	11
Splenium	6	17	23
Other (17)	8	9	17
Total	29	135	164

Table IV: Cortical Contusions/ Distribution of Lesions

Location	Number of lesions		
	Hemorrhagic	Nonhemorrhagic	Total
Parietooccipital (19)			
Medial	4	0	4
Lateral	8	1	9
Superior	4	2	6
Frontal (38)			
Superior	4	0	4
Inferior	18	2	20
Medial	6	1	7
Lateral	6	0	6
Pole	1	0	1
Temporal (66)			
Inferior	16	3	19
Medial	17	1	18
Lateral	13	3	16
Pole	13	0	13
Cerebellum (8)			
Hemispheric	4	1	5
Vermian	3	0	3
TOTAL	117	14	131

was 6.2±2.1 in this group of patients. Sixteen patients in this group had lower cranial nerve dysfunction. The majority of the brainstem lesions were located in the midbrain and dorsal pons. The lesions were mainly nonhemorrhagic and multiple. Rotational shearing forces are believed to cause the primary brainstem lesions (3, 7, 27).

Eight patients showed subcortical gray matter lesions, making up a small percentage of primary lesions (7.07%) (Figure 4). More than half of these

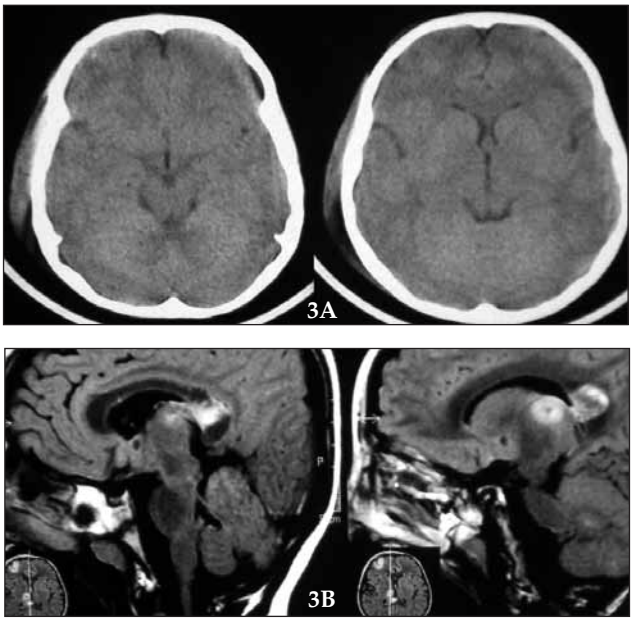

*Figure 3: (**A**) Slices of computed tomography show a normal appearance of brain parenchyma with right side subgaleal traumatic edema. (**B**) T1 slices of MRI show severe frontal contusion with midbrain and brainstem punctate lesions.*

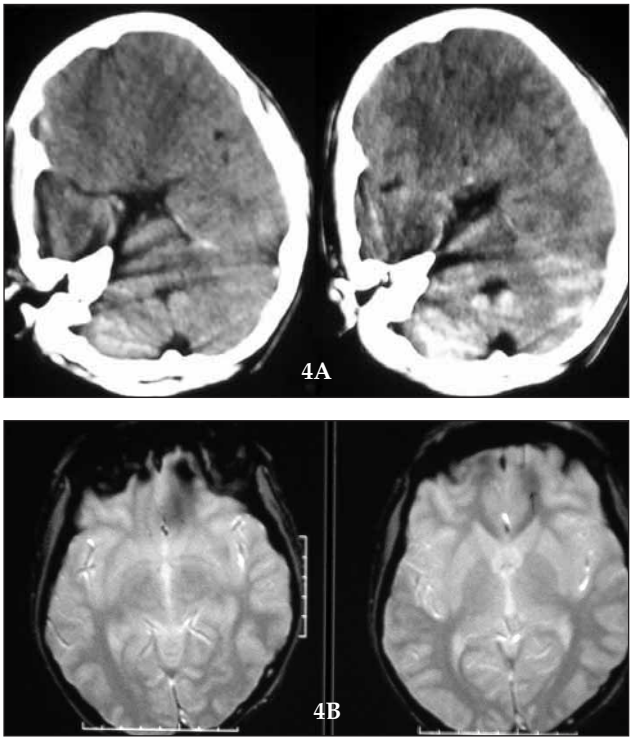

*Figure 4: (**A**) Posttraumatic computed tomography show a normal appearance of frontobasal region. (**B**) A left side hypointense posttraumatic gray mater lesion observed in proton density slices.*

lesions were hemorrhagic in nature (58.6%). This condition may be explained by the high vascularity of subcortical areas.

These subcortical areas have a rich network of perforating arteries anatomically. The majority of these lesions were settled on the thalamus and basal ganglia. The neurological condition of these patients was worse than the other patients. The mean GCS was 6.3±2.1. The reason for the profound neurological deterioration might be the deep and intersection settlement of hemorrhagic lesions. Such lesions are probably the prominent reason for the high mortality of severe head injury. The subcortical lesions were highly multiple (83.2%) and there was no hemispherical difference.

One patient showed both hemorrhagic contusion and one saccular aneurysm on the left carotid bifurcation that was detected during the MRI study to be confirmed by digital subtraction angiography.

The Glasgow Outcome Scale and patient scores at the end of third and sixth months are presented in (Tables V and VI) (16). The patients harboring mixed and multiple lesions had a significantly worse prognosis.

DISCUSSION

Complex anatomical and pathophysiological phenomena are initiated in the human brain after severe craniocerebral injury (CCI). From the point of view of the neurosurgeon, the identification of these lesions is imperative to navigate different therapeutic approaches. Secondly; identification also helps the clinician to distinguish primarily traumatic lesions from secondary insults and even to predict the prognosis of the patient (26). CT still remains the most essential diagnostic device in emergency units, and is an inexpensive, reliable, repetitive, swift and easy accessible facility providing soft tissue and bony details. Ventilation of CCI patients can also be easily provided at the time of CT screening (23, 25). Nevertheless CT has some limitations in CCI. It may overshadow minor lacunas, small contusions, diffuse axonal injury and some posterior cranial fossa pathologies due to bony artifacts. Moreover CT may mask low-lying pathologies in the presence of significant hemorrhage or edema. On the other hand MRI also has some unfavorable points in comparison to CT. It is expensive and requires special ventilator equipment when breathing support is needed. MRI is also time-consuming and has a limited ability to

Table V: Glasgow Outcome Scale.

Score	Meaning
5	Good recovery, resumption of normal life despite minor deficits.
4	Moderate disability (disabled but independent)- travel by public transportation, can work in sheltered setting (exceeds mere ability to perform activities of daily living).
3	Severe disability (conscious but disabled), dependent for daily support
2	Persistent vegetative state, unresponsive & speechless; after 2-3 weeks may open eyes and have sleep/wake cycles.
1	Death

Table VI: Glasgow Outcome Scale according to the lesion types at 3 and 6 months

Type of lesion	No. of patients	GOS at 3rd month	GOS at 6th month
SI multiple*	56	2.3±1.3	2.1±1.2
SI single	19	3.2±1.6	3.2±1.3
CC single	29	3.5±1.2	3.8±1.6
CC multiple*	16	2.4±1.4	2.4±1.4
Brain stem lesions*	27	1.3±0.4	1.3±0.4
Sc. Gray matter	8	3.6±1.4	3.8±1.8
Mixed lesions*	32	2.1±1.3	2.4±1.6

GOS: Glasgow outcome scale/ No. of Patients: number of patients
SI: Shear injury, CC: Cortical contusion, Sc: Subcortical

show early hemorrhagic lesions within the first three days (17, 19, 21). Another controversy is that CT has been used to diagnose mainly surgical lesions in CCI by clinicians until now. In other words; if the patient has no surgical lesion on CT, medical therapy and prediction of recovery has been mainly based on the neurological condition (GCS) or vital signs (ICP, blood pressure, cerebral oxygenation etc.) of the patients (18, 22, 26).

Early experiences of MRI examinations in severe CCI suggested that it may be more sensitive and significantly specific than CT in identifying some types of traumatic lesions (1, 2, 3, 6, 7, 19, 21, 25). The present study has emphasized that MRI is more sensitive than CT in detecting traumatic lesions especially after three days. MRI can predict poor prognosis much better than CT especially in nonsurgical and severely injured patients. 113 of the 124 patients (91%) had significant pathological lesions on MRI screening which were sufficiently severe to explain the neurological condition of the patients.

The first author to systematically analyze and describe patterns of traumatic stress mechanisms in CCI was Holbourn. After his postmortem studies; he also detailed traumatic brain lesions into two categories as lesions secondary to skull fractures and lesions resulting from shearing forces secondary to rotational accelerations (12). These mechanisms have been better understood after MRI studies. The primary traumatic intraaxial lesions may be classified in four categories. 1; shear injuries, 2; contusions of cortical areas, 3; subcortical gray matter injuries and 4; brainstem lesions (6, 7). We

believe that traumatic subarachnoidal hemorrhage may also be added to this scheme as a final category.

The most commonly diagnosed lesions in our series were shear injuries, cortical contusions, lesions of corpus callosum and the pontobulbar contusions. The importance of these lesions was that all are difficult to diagnose by CT but straightforwardly identified by MRI. Depending on different imaging parameters; MRI has increased sensitivity to differentiate normal and injured tissues (13). The contrast of the lesion is increased by relative differences in the T1 and T2 relaxation times of the soft tissues as well as the proton density. The bright imaging sensitivity of MRI for nonhemorrhagic lesions probably depends on its great ability to detect local water changes in traumatized tissues (1, 2, 3, 7, 11). Local water changes may be secondary to intracellular edema that may be induced hypoxic alterations of cellular metabolism. Otherwise, extracellular edema may be created by damage to the blood-brain barrier (4, 20, 22). MRI has also greater sensitivity to detect hemorrhagic lesions after CCI, but this sensitivity is time-dependent. Since hemorrhagic lesions can be visualized after methemoglobin formation in 72 hours, MRI has considerable superiority over CT after three days from CCI. The small bone neighboring or diffuse hemorrhages can easily be detected by MRI (21, 23, 24, 25). The first three days are critically important times after CCI especially in hemorrhagic lesions; however CT is the first diagnostic tool for most trauma centers. The initial diagnosis is generally made and operative interventions or navigation of therapeutic approaches are selected depending on the initial CT. MRI is applicable only after the patient is considered stable. Nevertheless the majority of SI and primary brain stem lesions are mainly nonhemorrhagic. The diagnosis of such lesions largely depends on MRI. Nevertheless; there are some studies showed the value of diffusion-weighted MRI for the evaluation of diffuse axonal injury in CCI. The diffusion-weighted images can differentiate between lesions with decreased and increased diffusion in patients with DAI. Further; diffusion-weighted images are capable of identifying additional shearing injuries not visible on T2/FLAIR or T2 sequences (11, 14).

The present report indicates that the majority of severe CCI cases without pathology in CT may have positive MRI findings. MRI is not only useful in identifying low-lying pathologies in severe trauma cases but also provides some prognostic information to the clinician. Most previous studies were retrospective and included patients with mild head injury. Our study investigated MRI findings in CT-negative patients with severe CCI. This study supports the importance of MRI in detecting acute and subacute hemorrhagic and nonhemorrhagic lesions, infarcts and brainstem injuries in severe CCI. Detailed detection and delineation of parenchymal traumatic lesions with MRI may allow more precise diagnosis and prediction of prognosis thus guiding medical therapy in the future.

REFERENCES

1. Cecil KM, Hills EC, Sandel ME, Smith DH, McIntosh TK, Mannon LJ, Sinson GP: Proton magnetic resonance spectroscopy for detection of axonal injury in the splenium of the corpus callosum of brain-injured patients. J Neurosurg 88:795-801, 1998
2. Doezema D, King JN, Tandberg D, Espinosa MC, Orrison WW: Magnetic resonance imaging in minor head injury. Ann Emerg Med 20:1281-1285, 1991
3. Firsching R, Woisceneck D, Diedrich M, Klein S, Ruckert A, Wittig H, Dohring W: Early magnetic resonance imaging of brainstem lesions after severe head injury. J Neurosurg 89:707-712,1998
4. Garnett MR, Blamire AM, Corkill RG, Rajagopalan B, Young JD, Cadaux-Hudson TA, Styles P: Abnormal cerebral blood volume in regions of contused and normal appearing brain following traumatic brain following traumatic brain injury using perfusion magnetic resonance imaging. J Neurotrauma 18:585-593,2001
5. Gennarelli TA, Thibault LE, Adams JH, Graham DI, Thompson CJ, Marcincin RP: Diffuse axonal injury and traumatic coma in the primate. In : Dacey RG Jr, Winn HR, Rimel RW, Jane JA, eds. Trauma of the central nervous system. New York: Raven, 1985: 169-193
6. Gentry LR: Imaging of closed head injury. Radiology 191:1-17, 1994
7. Gentry LR, Godersky JC, Thompson B: MR imaging of head trauma, review of the distribution and radiopathologic features of traumatic lesions. AJNR 9:101-110, 1988
8. Gentry LR, Godersky JC, Thompson B, Dunn V: Prospective comparative study of intermediate-field MR and CT in the evaluation of closed head trauma. AJNR 9:91-100, 1988
9. Gentry LR, Thompson B, Godersky JC: Trauma to the corpus callosum, MR features. AJNR 9: 1129-1138, 1988
10. Gomori JM, Grossman RI, Goldberg HI, Zimmerman RA, Bilanuk LT: Intracranial hematomas; imaging by high field MR. Radiology 157:87-93, 1985
11. Hergan K, Schaefer PW, Soransen AG, Gonzales RG, Huisman TA: Diffusion-weighted MRI in diffuse axonal injury of the brain. Eur Radiol 12(10):2536-2541, 2002

12. Holbourn AHS: The mechanics of head injuries. Br Med Bull 3:147-149, 1945
13. Hughes DG, Jackson A, Mason DL, Berry E, Hollis S, Yates DW: Abnormalities on magnetic resonance imaging seen acutely following mild traumatic brain injury: Correlation with neuropsychological tests and delayed recovery. Neuroradiology 46:550-558,2004
14. Huisman TA, Sorensen AG, Hergan K, Gonzales RG, Schaefer PW: Diffusion-weighted imaging for the evaluation of diffuse axonal injury in closed head injury. J Comput Assist Tomogr. 27(1):5-11,2003
15. Jane JA, Steward O, Gennarelli T: Axonal degeneration induced by experimental noninvasive head injury. J Neurosurg 62:96-100, 1985
16. Jennett B, Bond M: Assessment of outcome after severe brain damage. A practical scale. Lancet 1, 1(7905): 480-484,1975
17. Komatsu S, Sato T, Kagawa S, Mori T, Namiki T: Traumatic lesions of the corpus callosum. Neurosurgery 32-35,1979
18. Lee TT, Galarza M, Villanueva PA: Diffuse axonal injury is not associated with elevated intracranial pressure. Acta Neurochir (Wien) 140:41-46,1998
19. Levin HS, Amparo E, Eisenberg HM, Williams DH, High WM, Mcardle CB, Weiner RL: Magnetic resonance imaging and computerized tomography in relation to the neurobehavioral sequelae of mild and moderate head injuries. J Neurosurg 66: 706-713, 1987
20. Meyer CA, Mirvis SE, Wolf AL, Thompson RK, Gutierrez MA: Acute traumatic midbrain hemorrhage, experimental and clinical observations with CT. Radiology 179: 813-818, 1991
21. Mitti RL, Grossman RI, Hiehle JF, Hurst RW, Kauder DR, Gennarelli TA, Alburger GW: Prevalence of MR evidence of diffuse axonal injury in patients with mild head injury and normal head CT findings. Am J Neuroradiol 15:1583-1589, 1994
22. Prat R, Calatayud-Maldanano V: Prognostic factors in posttraumatic severe diffuse brain injury. Acta Neurochir (Wien) 140:1257-1261, 1998
23. Sahuquillo-Barris J, Laarca-Ciuro J, Vilalta-Castan J, Rubio-Garcia E, Rodriguez-Pazos M: Acute subdural hematoma and diffuse axonal injury after severe head trauma. J Neurosurg 68:894-900, 1988
24. Scheid R, Preul C, Gruber O, Wiggins C, von Cramon DY: Diffuse axonal injury associated with chronic traumatic brain injury: Evidence from T2-weighted gradient echo imaging at 3T. AJNR 24:1049-1056,2003
25. Snow RB, Zimmerman RD, Gandy SE, Deck MDF: Comparison of magnetic resonance imaging and computed tomography in the evaluation of head injury. Neurosurgery 18:45-52, 1986
26. Takanashi Y, Shinonaga M: Magnetic resonance imaging for surgical consideration of acute head injury. J clin Neurosci. 8(3):240-244,2001
27. Tong KA, Ashwal S, Holshouser BA, Shutter LA, Herigault G, Haacke EM, Kido DK: Hemorrhagic shearing lesions in children and adeloscents with posttraumatic diffuse axonal injury; improved detection and initial results. Radiology 227(2):332-339, 2002