

1

:

: 14

17 MR

8

3

3

T2

(ADC: apparent diffusion coefficient)

MR

: T2

14

, 3

ADC 1.21 ±

1.28 ± 0.22

0.34,

1.08 ± 0.28,

0.77 ± 0.25, 0.71 ± 0.22, 0.68 ± 0.27

mm²/ms

ADC

가

mm²/ms

ADC

ADC

(p < 0.05).

(p > 0.05). 3

ADC

(cytotoxic)

(vasogenic)

(Hypertensive encephalopathy)

(cytotoxic edema)

(overregulation)

(preeclampsia - eclampsia),

(breakthrough of autoregulation)

(1, 6, 7, 10).

(extravasation)

(1 - 7).

가

가

가

(1,

Tomography) MRI(Magnetic Resonance Image)

6, 9).

(Diffusion - weighted MR image)

(proton (diffusion)

(micro-

(3, 8 - 10).

scopic mobility)

(6). ,

가

가

(vasogenic

edema)

(interstitial edema)

가가

(1).

mm/2.5 mm, 1 NEX, 24 × 18, 128 × 128, x, y, z

(ADC; Apparent diffusion coefficient)

b factor가 SI0, b factor 가

(apparent diffusion SIb log (SI0 - SIb)/SI0

coefficient; ADC) T2 가

3

(11). mm²/ms

T2 ADC

67, 44.5, 14, 14, 6:8, 14

Wilcoxon rank sum Duncan multiple

range test p 0.05

가

(Table 1). 72 MRI

(n=2) 1

3 MRI

14, 8, 3, 3, 14, T2, 9

가 3

EPI가 1.5T MR (GE (n=8), (n=5), (n=3)

medical system, Milwaukee, Wisconsin, U.S.A.)

T2 (TR 4000 msec, TE 102msec, / , 7

mm/0 mm, 2 NEX, ETL(Echo train length) 12,

20 × 20 cm, 256 × 256) T1 (TR

484 msec, TE 8 msec)

single shot EPI 180(

b - factor 1000 s/mm

TR 10000 msec, TE 12.5 msec, / 5

Table 2. Comparison of ADC Values of Normal and Abnormal White Matter in Three Groups with Hypertension

	Abnormal White Matter (Mean SD)(mm ² /ms)	Normal White Matter
Idiopathic hypertension	1.21 ± 0.34	0.77 ± 0.25
(Pre)Eclampsia	1.08 ± 0.28	0.71 ± 0.22
Cyclosporin-induced HT	1.28 ± 0.22	0.68 ± 0.27

Table 1. Patient Information

No	Diagnosis	Age/Sex	Symptom	MBP	DWI	ADC (mm ² /ms)
1	Pituitary adenoma	61/F	Visual disturbance	155/100	Iso	1.37
2	Hypertension	65/M	Hemifacial spasm	140/80	Iso	0.75
3	Hypertension	45/F	Headache	140/100	Iso/sl. high	1.48
4	Hypertension	33/M	Visual disturbance	210/150	Iso/sl. high	1.33
5	CHF	59/M	Drowsy mental status	140/100	Iso	0.92
6	Old CVA	67/M	Vertigo	170/70	Iso	1.55
7	Cerebellar infarction	83/F	Dizziness, dysarthria	190/130	Iso/sl. high	0.77
8	Hypertension	66/F	Memory disturbance	170/90	Iso	1.51
9	Eclampsia	26/F	Seizure	190/100	Iso	1.37
10	Eclampsia	31/F	Seizure, headache	160/110	Iso	1.07
11	Pre-eclampsia	26/F	Blur vision	140/100	Iso	0.81
12	ADEM/cyclosporin	10/M	Febrile seizure	150/100	Iso	1.28
13	MDS/cyclosporin	37/M	Seizure, headache	160/100	Iso	1.49
14	AA/cyclosporin	14/F	Seizure, dizziness	170/110	Iso	1.06

Note.- MBP, maximal blood pressure; DWI, diffusion-weighted image; ADC, average apparent diffusion coefficient in abnormal white matter; Iso, iso-signal to gray matter; sl. High, slightly high signal to gray matter; CHF, congestive heart failure; CVA, cerebrovascular accident; ADEM, acute disseminated encephalomyelitis; MDS, myelodysplastic syndrome; AA, aplastic anemia

5 , - 가 ($p < 0.05$).

ADC
($p > 0.05$).

ADC

3

ADC

3

1.21 ± 0.34,

1.08 ± 0.28,

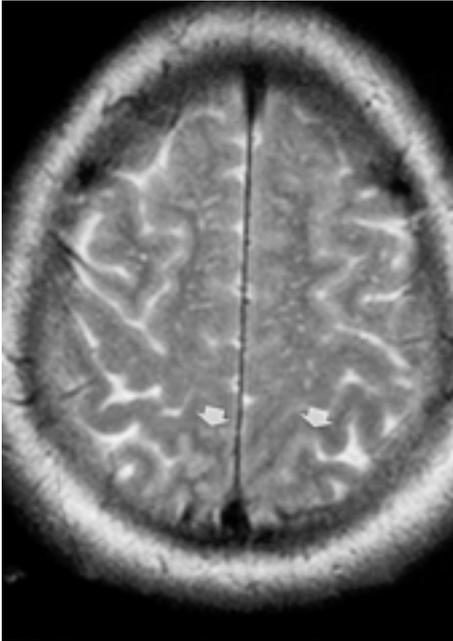
1.28 ± 0.22

ADC

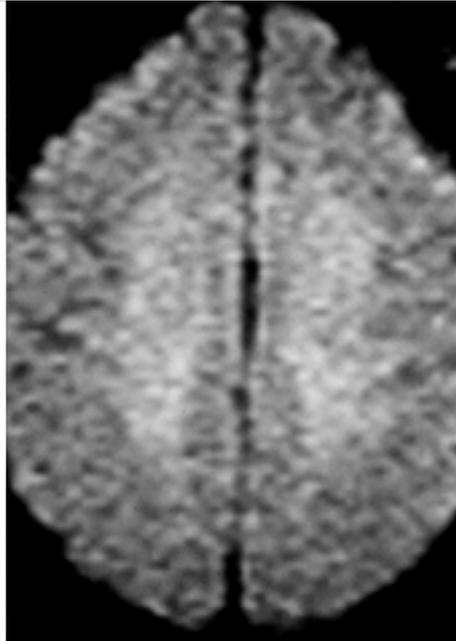
ADC

0.77 ± 0.25, 0.71 ± 0.22, 0.68 ± 0.27

ADC



A



B

Fig. 1. A 33-year-old male with hypertension developed visual disturbance and maximal blood pressure are up to 210/150.

A. T2-weighted image shows slight increased signal intensity in the both peripheral subcortical white matter in the both parietal lobe (arrows).

B. At the same level, high b-factor diffusion weighted image (*b* factor = 1000 m/s) does not demonstrate abnormal signal intensity.



A



B

Fig. 2. A 26-year-old female with pregnancy complains blurred vision and dyspnea.

A. T2-weighted image shows increased signal intensity at the both parietal and occipital lobe (arrows).

B. At the same level of A high-b factor diffusion weighted image (*b* factor = 1000 m/s) show no abnormal signal intensity.

1928 Oppenheimer Fishberg
가
(1, 2, 7). reversible
posterior leukoencephalopathy syndrome
(1, 2).

가
(2). 가
가
가

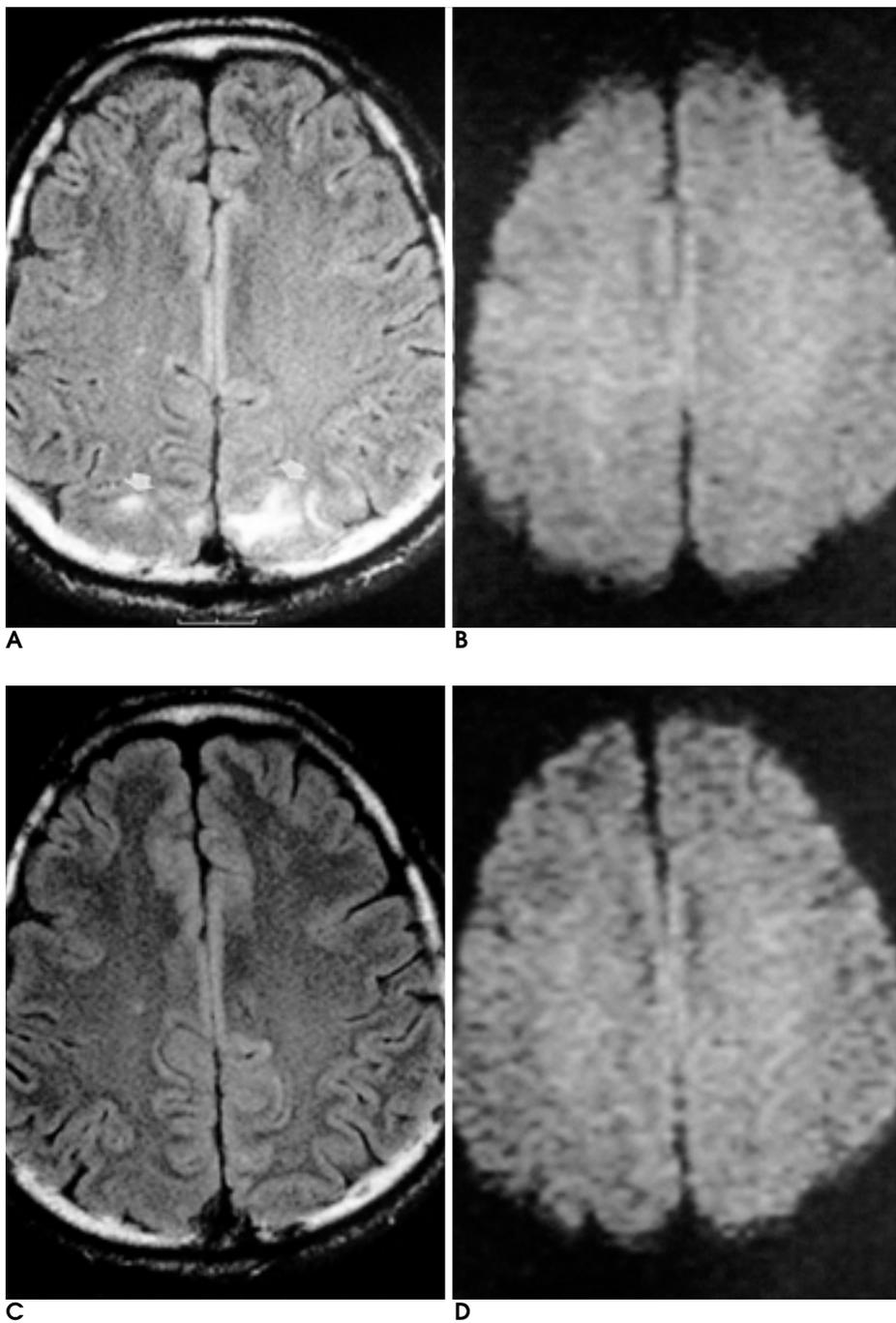


Fig. 3. A 37-year-old male with myelodysplastic syndrome, developed headache, which were follow by a seizure during cyclosporine treatment for bone marrow transplantation.
A. FLAIR(fluid attenuatd inversion recovery) image also demonstrate focal high signal intensity at the both parietal lobe (arrows).
B. High b-factor T2-weighted image (*b* factor = 1000 m/s) does not show abnormal signal intensity.
C, D. Two weeks later, FLAIR image (C), high b-factor T2-weight image (*b* factor = 1000 m/s) (D) demonstrate no abnormal signal intensity.

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The Analysis of Pathogenesis in the Hypertensive Encephalopathy using Diffusion-Weighted MR Imaging¹

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Purpose: To investigate the nature of edematous lesions seen on MR images during acute episodes of hypertensive encephalopathy (HTE) with particular attention to the findings of diffusion-weighted imaging (DWI).

Materials and Methods: A total of 17 MR examinations in fourteen patients with hypertensive encephalopathy were performed. The diagnoses were idiopathic HTE in eight cases, eclampsia in three, and cyclosporin-induced HTE in three. The apparent diffusion coefficients (ADCs) of edematous lesions and normal white matter revealed by DWI were assessed and compared, and the changes observed at follow-up MR imaging were analysed.

Results: DWI obtained within one week of the appearance of acute neurological symptoms revealed the edema as iso-intense in all patients with eclampsia and cyclosporin-induced HTE, and in five of eight patients with idiopathic HTE. In the other three patients with idiopathic HTE, DWI demonstrated slightly hyperintense edema. The ADCs of edematous lesion in patients with idiopathic HTE, eclampsia and cyclosporin-induced HTE were 1.21 ± 0.34 , 1.08 ± 0.28 , and 1.28 ± 0.22 mm²/ms, respectively, while for normal white matter the corresponding figures were 0.77 ± 0.25 , 0.71 ± 0.22 , and 0.68 ± 0.27 mm²/ms. The differences in ADCs between edema and normal white matter were thus significantly different between the three patient groups ($p < 0.05$), while the ADCs of edematous lesions showed no significant variation between these groups ($p < 0.05$). Follow-up MRI revealed that in three cases, edematous lesions were reversible and there were no residual signal changes.

Conclusion: Vasogenic rather than cytotoxic edema is present during the acute stage of HTE.

Index words : Brain, disease

Magnetic resonance (MR), diffusion study

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