

T2 MR :

1

2 3 4 5

31 T2

19

30 , 2, 4, 6 , 1, 3, 5, 7, 11, 21 31 T2

T2

31

(<6)

2

4-6 가 T2 6 10 6

(1-11)

가 3

가

T2

1

가

7

(21-31)

,

가

가

31

T2

가

:

T2

가

가

가

가

1

T2

T2

(echo planar imaging)
ted imaging)

(diffusionweigh-

(1, 2).

가

(3, 4).

1

1.8-4.4 kg

19

Ketamine (Ketamine hydrochloride 50 mg/ml,

Rompun(Xylazine hydrochloride, Bayer Korea,) 2.5 mg/kg 0.125 mg/kg

1
2
3
4
5

1997

1998 12 9

1999 4 13

Lidocaine 0.5ml
 5cm
 4.0 black silk (Mersilk)
 24 G IV catheter
 X-
 125 μ m Ivalon (Nycomed, Ingenor)
 2ml catheter
 Ivalon 1ml 10-15 가
 Ivalon X- catheter
 100ml
 MR
 MR 1.5T superconductive Vision (Siemens, Erlangen, Germany)
 small FOV radio-frequency coil (8.5cm)
 가 MR 18-
 19. C 30 (n=19),
 2 (n=17), 4 (n=11), 6 (n=10), 1 (n=5), 3 (n=4), 5 (n=4), 7 (n=4), 11 (n=4), 21 (n=3), 31 (n=3)
 T2 EPI sequence
 가 gradient pulse b 0 1,000 sec/mm²
 130mm, 4mm
 T2 parameter TR/TE 3000/96,
 60-75mm, 4mm, 2
 2 30
 MR
 1mm³ 2.5% glutaraldehyde in phosphate-buffered saline (PBS), PH 7.2 4 °C
 2 0.1M PBS
 propylene oxide Luft
 (5) poly/Bed 812 Resin 37 °C 12
 , 45 °C 48
 1 μ m toluidine blue (Leica REICHT
 Supernova) diatome
 Watson(6) Reynolds(7) Copper grid
 Uranyl acetate lead citrate (model JEM 1200EX-II; JEOL USA, Peabody, Mass, USA)
 MR Magic View workstation (Siemens, Erlangen, Germany)
 (signal intensity) T2

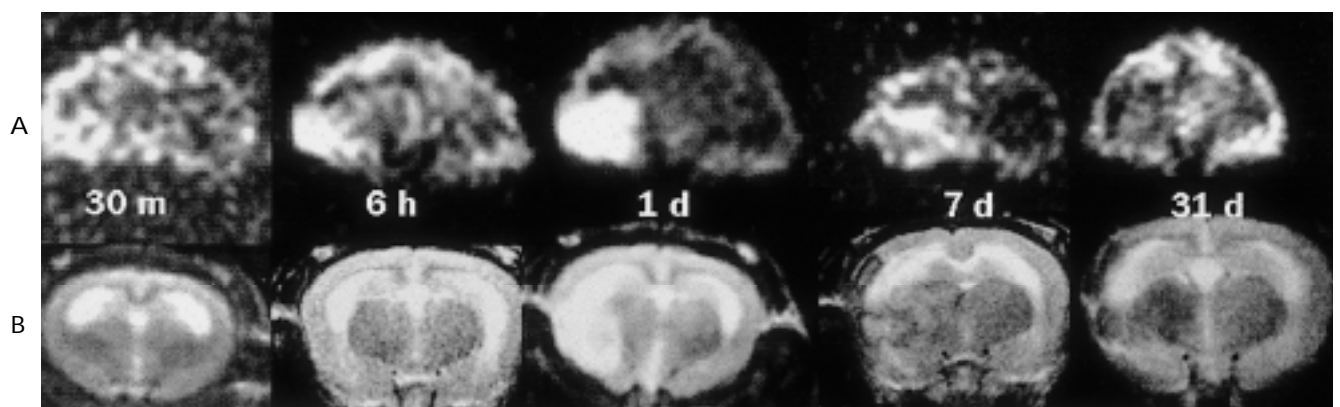


Fig. 1. Serial diffusion-weighted (A) and T2-weighted images (B) after development of infarction.
 A. High signal intensity is detected in the right hemisphere at 30 minutes and its intensity increases till 1 day. Thereafter its intensity decreases gradually.
 B. High signal intensity is detected from 1 day and thereafter its intensity decreases continuously.

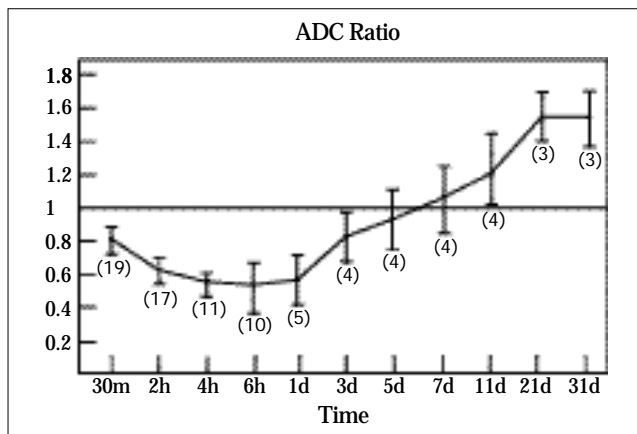


Fig. 2. ADC ratios plotted against time from infarction onset. The ratio is 0.81 at 30 min, is lowest at 4 and 6 hours. Thereafter the ratio continue to increase. number in parenthesis; the number of rabbits.

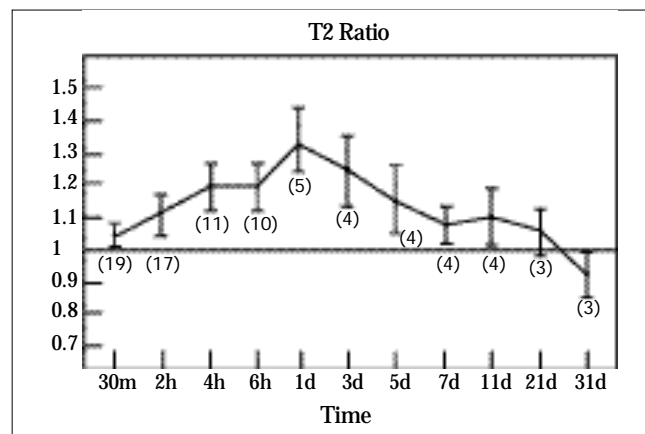


Fig. 3. T2 signal intensity ratios plotted against time from infarction onset. The ratio is higher than 1 from 30 min, is highest at 1 day, and thereafter continue to decrease. The ratio is below 1 after 21 days. number in parenthesis; the number of rabbits.

(ADC ratio) T2
data

(ADC)

ADC ratio = lesion side ADC / normal side ADC,

ADC = $\log_0 (S_0/S_i) / b_1 - b_0$

$(S_0 \quad b_0, S_i \quad b_1)$.

T2

T2

T2 signal intensity ratio = lesion side signal intensity
/ normal side signal intensity .

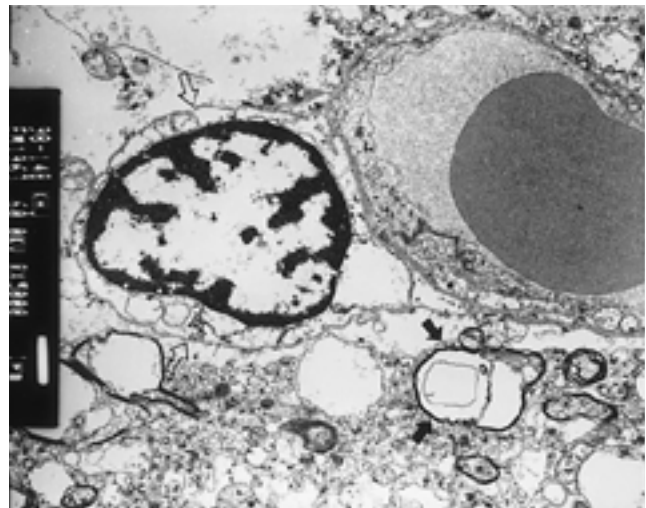


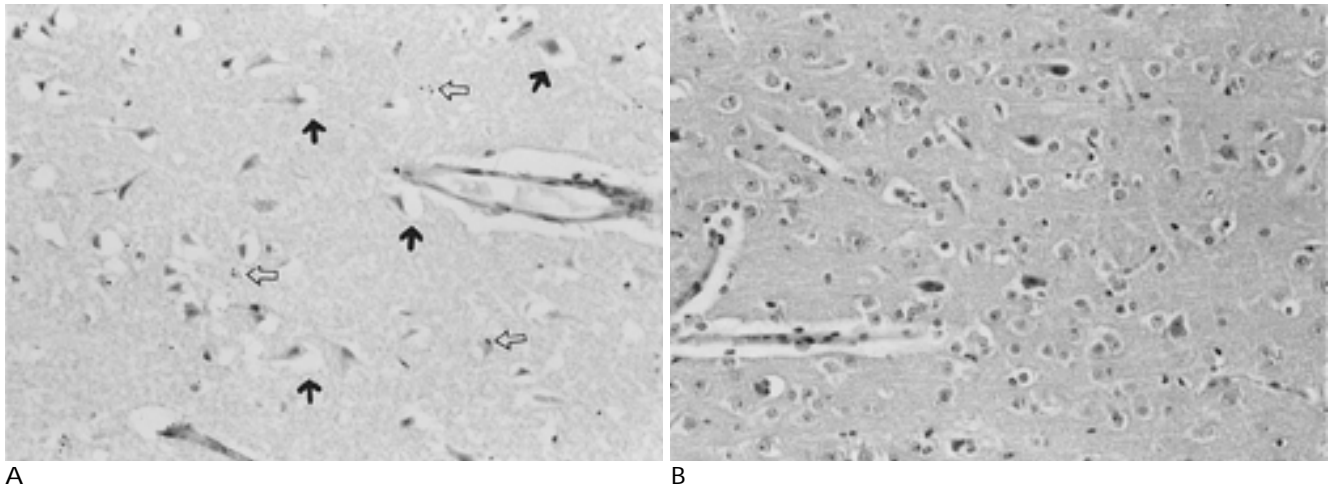
Fig. 4. Electron microscopic finding at 6 hours after infarction onset ($\times 5,000$). Neuropils are swollen and have many vacuoles. Axons (arrows) are degenerated and multiple pores in astrocytic cytoplasm (open arrows) are noted. However, the basement membranes of blood vessel are intact.

MR
T2
30 19 18
가
2 6 1
가 3 가 31
(Fig. 1A).
30 T2
가 가 , 2
17 1 가 . 6
10 6
. 1 5
가 7 가 가
가 31

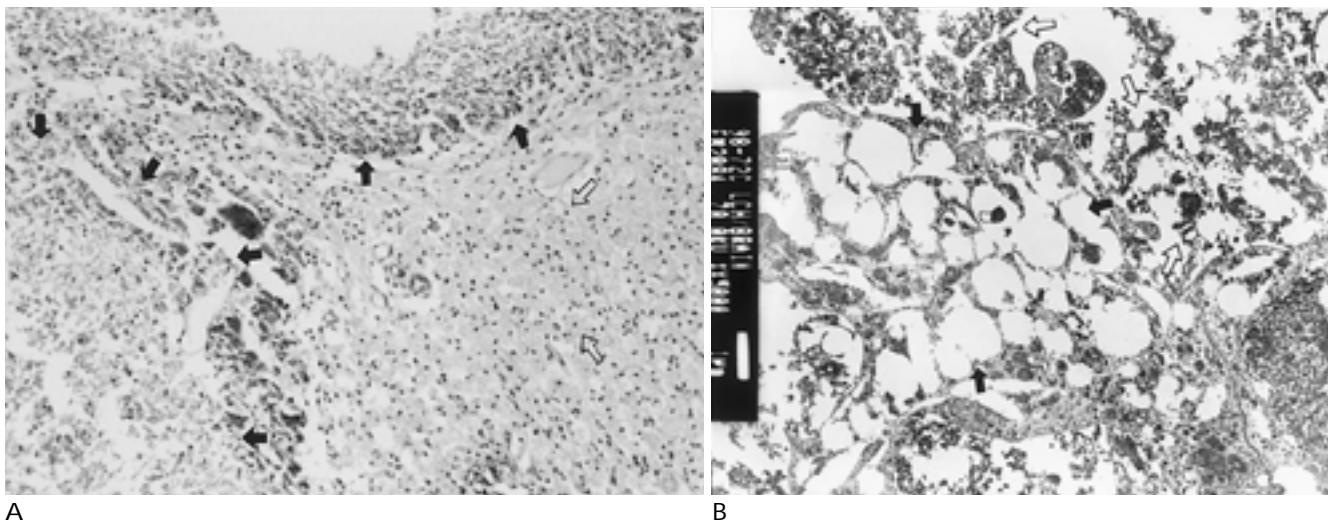
3 (Fig.1B).
4 6
0.56 가 가 5 7
1 31 가 (Fig. 2).
T2
1 , 1 가
(Fig. 3).

6

(neuropil)



A
Fig. 5. Microphotographic finding at 5 days.
A. Dilatation of perineural space, loosening of neuropil matrix and red neurons (arrows) are noted. Karyorrhectic debris (open arrows) implying cell destruction are also revealed (H & E, $\times 200$).
B. Normal area is shown as a comparison (H & E, $\times 200$).



A
Fig. 6. Microphotographic (A) and electron microscopic (B) findings at 31 days.
A. There are noted liquefaction necrosis with dystrophic calcification (arrows) at left and upper sides, and a large sheet of foamy histiocytes (open arrows) at right side (H & E, $\times 100$).
B. Prominent liquefaction necrosis (arrows) and amorphous granular structures (open arrows) derived from necrotic cell destruction are revealed in extracellular space ($\times 5,000$).

(vacuolization), mitochondria (Fig. 4). 가
5 (red (extracellular space) (Fig. 6B).
neurons), (neuropil)
(Fig. 5).
31 가 (6) 6
(Fig. 6A). T2

[illegible]

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Diffusion- and T2-weighted MR Imagings of Cerebral Infarction in Rabbit: Time Course of Imaging Findings and Histologic Correlation¹

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Purpose : To correlate the serial findings obtained by diffusion- and T2-weighted imaging with histologic findings obtained from 30 minutes to 31 days after the development of cerebral infarction in rabbits.

Materials and Methods : Nineteen male New Zealand white rabbits were subjected to intracerebral embolic infarction. Diffusion- and T2-weighted imagings were performed at 30 min, 2, 4 and 6 hours, and 1, 3, 5, 7, 11, 21 and 31 days. Apparent diffusion coefficient (ADC) ratios and T2 signal intensity ratios of infarcted and normal brain were calculated. Microphotographic or electron microscopic (EM) examinations were performed during hyperacute, acute and chronic infarctions.

Results : During hyperacute infarction, diffusion-weighted images showed high signal intensity in the infarcted area, and ADC ratios ranged from 0.81 to 0.56. High signal intensity on diffusion-weighted images continued until day 3, decreasing thereafter. The ADC ratio increased continuously after day 1. High signal intensity on T2-weighted images was noted from 6 hours and continued until day 7, decreasing thereafter. Microphotographic findings at 6 hours were normal, but EM examination revealed cellular swelling with intact basement membrane, suggesting cytotoxic edema. During acute infarction, abnormal dilatation of the perineural space, cell destruction, and loosening of the neuropil matrix were revealed by microphotography. During chronic infarction, microphotographic and EM findings revealed liquefaction necrosis.

Conclusion : These data indicate that in cases of hyperacute infarction, diffusion-weighted images reflect cytotoxic edema more accurately than do T2-weighted images. A gradually increasing ADC ratio during the course of infarction may be associated with vasogenic edema and cell lysis.

Index words : Brain, infarction

Brain, MR

Magnetic resonance (MR), diffusion study

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