Diffusion-weighted MRI in the acute stage of transient ischemic attacks

H. Shimizu, T. Inoue*, T. Yoshimoto*, H. Kabasawa#, T. Tsukamoto#
Departments of Neurosurgery, Kohnan Hospital and *Tohoku University School of Medicine, Sendai, and #R&D, GE Yokogawa Medical Systems, Hino, Tokyo, Japan

INTRODUCTION
Diffusion-weighted magnetic resonance imaging (DWI) has been an important clinical tool to investigate patients with cerebral ischemia. DWI visualizes ischemic cerebral lesions within an hour. The capability of DWI to demonstrate responsible lesions as early as patient's arrival potentially influences decision making for the individual diagnostic and therapeutic strategies. For example, a DWI finding of cortical ischemic lesions may indicate a more emergent need for evaluation of cardiac disease or steno-occlusive disease of major cerebral arteries than that of common lacunas. The transient ischemic attack (TIA) is a term of symptomatology and includes several different etiologies such as atherosclerotic thrombosis or embolism, cardioembolism, and lacunas. Therefore, prompt determination of the etiology is important for prevention of further TIAs. The primary purpose of this study was to investigate the sensitivity of DWI for detecting ischemic lesions in patients with TIAs within six hours after the onset. In a part of subjects, we compared DWI with single-photon emission computed tomography (SPECT) to clarify the role and limitation of DWI in the emergency care of TIA.

METHODS AND SUBJECTS
This study included 23 patients and 27 TIAs. Two patients had two occasions of TIA, respectively. There were 16 males and 9 females, ages from 38 to 84. All patients underwent the DWI study within 6 hours after the onset of the last TIA. Regardless of the symptom at the DWI study, all patients were clinically followed for 24 hours to confirm the diagnosis of TIA.

All imaging were performed using GE Signa 1.5 T and a standard head coil. The DWI sequence was a navigated spin echo (first 11 TIAs) or a single shot spin echo type (other 16 TIAs) DWI. Fast spin echo T2-weighted imaging (T2WI) was also performed at all DWI studies. Time-of-flight three dimensional magnetic resonance angiography (MRA) of the circle of Willis was obtained in 26 TIAs. 99Tc-HMPAO or -ECD SPECT was performed immediately after the DWI study in 10 TIAs. Emergency digital subtraction angiography (DSA) was performed in 12 TIAs in which steno-occlusive major artery lesions were suspected.

Navigated spin echo DWI used motion-probing gradient (frequency direction, b value of 600 sec/mm2) which was incorporated into the conventional SE sequence. Both peripheral pulse gating and our navigator echo sequence compatible with multislice DWI were used to reduce motion artifacts. The details of this navigator echo have been published elsewhere (1). Imaging parameters were TR/TE/slice thickness/matrix: 2000-2500msec / 120msec / 8mm / 256x128. Parameters for single shot spin echo type DWI were 4000msec / 110msec / 7mm / 128x128, and b value was 1000 sec/mm2.

Follow-up T2WI or CT was obtained 24 hours after the DWI study or later to evaluate final ischemic lesions.

RESULTS AND DISCUSSION
In 15 TIAs, DWI demonstrated positive hyperintensity lesions. DWI was negative in 12 TIAs. Suspected etiology of TIAs was atherosclerotic in 4 (DWI positive: DWI negative = 2:2), lacuna or perforator area in 10 (5:5), and cardioembolic in 13 (6:5).

In all 27 TIAs, mean time interval between the onset of the last TIA and DWI was 2.7±1.8 hours (mean ± standard deviation). The interval was 3.5±2.0 and 1.8±0.8 hours in DWI positive and negative TIAs, respectively. Duration of TIA was 5.4±4.6, 7.6±8.4 and 2.7±5.6 hours in all, DWI positive and DWI negative TIAs, respectively.

There were 9 TIAs which were still symptomatic during DWI study. Of those, DWI was positive in 7 and negative in 2 TIAs. In the other 18 TIAs, the interval between the end of TIA and DWI was 1.1±1.6 hours (n=8) in DWI positive TIAs and 0.9±0.8 hours (n=10) in DWI negative TIAs.

Eight of 10 SPECT studies demonstrated focal flow reduction compatible with TIA symptoms. When DWI was positive (n=6), SPECT also showed focal abnormality which included, but was much larger than, DWI hyperintensity. When DWI was negative (n=4), SPECT appeared normal in 2, however, focal flow reduction was demonstrated in the remaining 2 TIAs.

In 15 DWI positive TIAs, there were final corresponding ischemic lesions on T2WI or CT in 14 TIAs. In these 14 TIAs, the final lesion was as same as the DWI hyperintensity lesion in 11, smaller in 1 and larger in 2. The remaining one TIA with DWI positive finding did not show the final lesion. In 12 DWI negative TIAs, there were final lesions in 2 TIAs and no lesion detected in 10 TIAs.

It is important to discriminate cortical TIAs and lacunar TIAs for the first step of clinical decision making. When DWI lesion was cortical (n=8), the final clinical diagnosis was also cortical in all TIAs. When DWI lesion was lacunar (n=8), the final clinical diagnosis was also lacunar in 5 TIAs but was cortical in 2 TIAs. In 12 DWI negative TIAs, the final diagnosis were cortical in 7 and lacunar in 5.

DISCUSSION
Our results are summarized as follows;
1) DWI in the acute stage of TIA was positive in 56% (15/27).
2) Positive DWI correlated with symptom presence at DWI study and symptom duration, but not with other factors.
3) Final infarctions were mostly as same as DWI lesions, however, smaller in 2 and larger in 4.
4) Differentiation of cortical and lacunar TIAs was possible in 13 of 15 DWI positive TIAs.

These results were compatible with a recent report (2), which investigated TIA patients within 3 days of onset. This suggests that even in TIA, most of DWI hyperintensities represent clinically irreversible lesions. However, in a few patients, DWI in the acute stage of TIA may demonstrate responsible lesions that are otherwise undetectable.

CONCLUSIONS
More than half of TIAs demonstrated DWI lesions which were useful to differentiate cortical and lacunar TIAs. This implies that DWI in the acute stage of TIA may contribute to etiologic understanding and decision making in therapeutic strategies.

REFERENCES