Stroke
JOURNAL OF THE AMERICAN HEART ASSOCIATION
American Stroke Association
A Division of American Heart Association

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Stroke 1999, 30:1174-1180
doi: 10.1161/01.STR.30.6.1174
Stroke is published by the American Heart Association. 7272 Greenville Avenue, Dallas, TX 72514
 Print ISSN: 0039-2499. Online ISSN: 1524-4628

Diffusion MRI in Patients With Transient Ischemic Attacks

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Background and Purpose—Diffusion MRI has established value in patients with ischemic stroke but has not been systematically investigated in patients with transient ischemic attack (TIA).

Methods—Clinical, conventional MRI, and diffusion MRI data were collected on 42 consecutive patients with symptoms of cerebral TIA. TIA imaging data were compared with those from a contemporaneous group of 23 completed stroke patients.

Results—Twenty of the 42 TIA patients (48%) demonstrated neuroanatomically relevant focal abnormalities on diffusion-weighted imaging (DWI) and apparent diffusion coefficient (ADC) imaging. When present, DWI/ADC signal changes in TIA patients were less pronounced and smaller in volume than those in completed stroke patients. TIA symptom duration was significantly longer for DWI-positive than for DWI-negative patients, 7.3 versus 3.2 hours. Diffusion MRI information changed the suspected anatomic and vascular TIA localization and the suspected etiologic mechanism in over one third of patients with diffusion MRI abnormalities. Of the 20 TIA patients with identifiable lesions on diffusion MRI, 9 had follow-up imaging studies; of these, 4 did not show a relevant infarct on follow-up imaging.

Conclusions—Diffusion MRI demonstrates ischemic abnormalities in nearly half of clinically defined TIA patients. The percentage of patients with a DWI lesion increases with increasing total symptom duration. In nearly half, the diffusion MRI changes may be fully reversible, while in the remainder the diffusion MRI findings herald the development of a parenchymal infarct despite transient clinical symptoms. Finally, diffusion imaging results have significant clinical utility, frequently changing the presumed localization and etiologic mechanism. **(*Stroke*. 1999;30:1174-1180.)**

Key Words: cerebral ischemia, transient ■ magnetic resonance imaging ■ magnetic resonance imaging, diffusion-weighted

Transient ischemic attacks (TIAs) are defined as neurological symptoms of vascular etiology that resolve within 24 hours.[1] The core elements of this definition were first promulgated in the 1950s, on the basis of sparse data.[2] Since then, controversy has arisen regarding the utility and accuracy of this definition, which is based on clinical manifestations and an arbitrarily assigned time window rather than tissue changes and physiological processes. Large-scale studies have altered our understanding of the typical duration of TIAs, showing that most TIAs resolve within 10 to 60 minutes rather than lasting several hours.[3,4]

Imaging studies have challenged the simplistic assumption that because clinical TIA symptoms resolve, significant ischemic tissue injury must not occur. Several studies have shown that many patients meeting the clinical criteria for TIA demonstrate neuroanatomically relevant infarcts on standard neuroimaging (2% to 48% using CT,[5–10] 31% using MRI).[11] The probability of finding an infarct on imaging appears to increase when the TIA persists longer than 1 hour. Waxman and Toole[12] coined the term "cerebral infarction with transient signs" (CITS) to describe patients who meet clinical criteria for TIA but show a relevant infarct on imaging.

The success of diffusion MRI (DWI) in recent studies of clinical stroke suggests that this advance in MR technology may also improve our understanding of the pathophysiological processes associated with TIA. The initial discovery that brain tissue experiencing ischemia produces a hyperintense DWI signal grew from animal studies.[13] These models have clearly demonstrated that the DWI hyperintensity delineates tissue damaged by ischemia[14] and that abnormal diffusion characteristics can become identifiable within a minute of the complete interruption of blood flow.[15] Subsequent studies of human stroke have demonstrated that DWI scans made early in the infarction process have good potential for predicting clinical outcome.[16]

We hypothesized that diffusion MRI would provide a more sensitive and specific evaluation of ischemic insult in TIA patients compared with prior CT and MRI studies. We studied consecutive TIA patients using diffusion MRI and compared them with a group of contemporaneous completed stroke patients to determine (1) the incidence of DWI and apparent diffusion coefficient (ADC) abnormalities in TIA patients; (2) whether the presence of a diffusion MRI abnor-

Received January 12, 1999; final revision received March 25, 1999; accepted March 25, 1999.

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TABLE 1. Choices for Suspected Anatomic Localization, Vascular Localization, and TIA Mechanism

Anatomic Localization	Vascular Localization	Mechanism
Right cortical	ACA	Small-vessel lacunar disease
Left cortical	Superficial MCA	Large-vessel atherothrombotic disease
Right pure subcortical	Deep MCA	Cardioembolic
Left pure subcortical	PCA	Other
Right brain stem	Basilar artery	
Left brain stem	Vertebral artery	
Right cerebellum	Brain stem perforators	
Left cerebellum		

mality correlates with the duration, location, or mechanism of symptoms; (3) whether the diffusion MRI signature in TIA differs from that in completed stroke; and (4) the impact of diffusion imaging data on clinical diagnosis of TIA localization and mechanism.

Subjects and Methods

Subjects

We collected clinical, conventional MR, and diffusion MR data on consecutive patients who presented to the UCLA Medical Center over a 6-month period with symptoms of a TIA. TIAs were defined as symptoms of presumed ischemic cerebrovascular etiology lasting less than 24 hours. Patients with brain stem and/or hemispheric symptoms were included, whereas patients with isolated amaurosis fugax were excluded. All MRI scans were obtained within 3 days of symptom onset. The following clinical data were compiled on all patients: age, sex, symptom(s) of TIA, date and time of symptom onset, date and time of symptom resolution, history of previous clinical TIA(s) or stroke(s), and presence of vascular risk factors (hypertension, coronary artery disease, diabetes mellitus, hypercholesterolemia, and history of tobacco use).

We also recorded a stroke neurologist's leading neuroanatomic, vascular anatomic, and etiologic diagnoses of the TIA (1) reached without information from DWI and (2) reached after reviewing the results of DWI. The method of rendering the etiologic diagnosis was informed by but not restricted to criteria of standard stroke classification schemes.[17,18] Available choices for each of these categories are shown in Table 1. Both the prediffusion and postdiffusion imaging diagnoses incorporated information available from the conventional T1-weighted, T2-weighted, and proton density (PD)-weighted MR sequences that were obtained on all patients. The diagnoses also incorporated information from other testing performed during the admission, including carotid imaging and echocardiography in all patients and more detailed studies such as catheter cerebral angiography in selected patients, when deemed appropriate by the attending physician.

MRI Methods

MRI was performed on a 1.5-T Siemens Vision MR system equipped with echo planar imaging data acquisition capability designed to obtain rapid diffusion images. MRI studies included sagittal T1-weighted, axial T2-weighted, axial T1-weighted, PD-weighted, and DWI sequences (approximately 30 minutes of scanning time). Most patients also underwent standard intracranial and/or extracranial MR angiography.

To obtain diffusion information rapidly and maintain consistency with several recent clinical trials, diffusion imaging was performed using a slice thickness of 7 mm with no interslice gap and 2 levels of diffusion sensitization (b=0, 1000 s/mm^2). The higher level of diffusion sensitization was replicated in each of the 3 orthoganol, principal gradient directions (read, slice select, phase encode [*X*, *Y*, *Z* planes]), and DWI images were formed from average of these. ADC images were computed by use of the equation

$$ADC_{i,j}=\{\ln[S_{i,j}(0)]-\ln[S_{i,j}^{av}(1000)]\}/1000$$

where the subscript i,j denotes the image voxel; $S_{i,j}(0)$ denotes the image intensity obtained without diffusion sensitization, and $S_{i,j}^{av}(1000)$ denotes the directionally averaged signal intensity measured at the higher level of sensitization. The ADC calculation was not performed in image voxels where the b=1000 signal intensity was <10% of the maximum (extracranial regions). Intracranial regions having ADC values characteristic of cerebrospinal fluid (ie, >2000 μm^2/s) were zeroed to provide visualization of parenchymal ADC with higher visual dynamic range. The calculated ADC images were displayed using a gray scale.

For all patients we recorded date and time of MRI, presence of a DWI abnormality, presence of a DWI lesion correlate on T2, PD, or T1 images, and evidence of prior old stroke(s) and/or ischemic white matter disease on T1, T2, and PD images. Readings were performed jointly by a neuroradiologist and a neurologist who were aware of the patient's history. For patients demonstrating a DWI abnormality, further image processing and data analysis were performed by an independent neuroradiologist who was unfamiliar with the cases. Additional analyses included calculation of DWI lesion volume and mean lesion intensity, ADC lesion volume, and the mean ADC value for each lesion.

DWI lesion volumes were first identified by visual inspection for regions of hyperintensity, then measured by outlining regions of interest by hand with the aid of an image analysis system. Final lesion volume was then calculated by multiplying the area obtained from each section by the slice thickness. DWI signal increase was calculated by comparing the mean signal intensity within the region of DWI abnormality to the mean signal intensity in the contralateral region, using the following formula: 1−(mean DWI signal intensity)/(mean corresponding contralateral region signal intensity). If multiple scattered or noncontiguous abnormalities were noted, volumes were calculated by summing individual volumes of all abnormal lesions. Regions having abnormal ADC were identified using an automated thresholding procedure that identified regions having an ADC of <545 μm^2/s. The area and the mean ADC within the area were then calculated.

To compare the ADC and DWI signatures (lesion volumes and intensities) to those of patients with completed stroke, we collected ADC and DWI data on 23 patients with completed clinical infarcts selected from stroke patients admitted to our institution over the same time period. For each of the 6 months, we used the first 3 to 4 completed stroke patients from each month who underwent acute MRI with a DWI sequence. Consecutive patients were selected and included large-vessel atherthromboembolic, cardioembolic, and small-vessel ischemic stroke subtypes. Lesion volume and intensity were calculated for this group by the same neuroradiologist and same method described for the TIA patients.

Statistical Analyses

Between-group comparisons were performed with both parametric tests for normally distributed data (the Student *t* test and $\div^2$ test) and nonparametric tests for nonnormally distributed data (rank sum test).

Results

Over a 6-month period, 67 patients were admitted to our university hospital with symptoms suggestive of a TIA. Of these 67 patients, 6 had symptoms of amaurosis fugax and were excluded from our analysis. Of the remaining 61 patients, 42 were eligible for MRI scanning, including DWI. Reasons for patients not undergoing MRI were presence of a metal implant (5), refused/claustrophobic (6), MRI technical difficulty (3), and nonstroke attending preference (5).

Of these 42 patients, 20 (48%) demonstrated a DWI abnormality consistent with acute neural bioenergetic com-

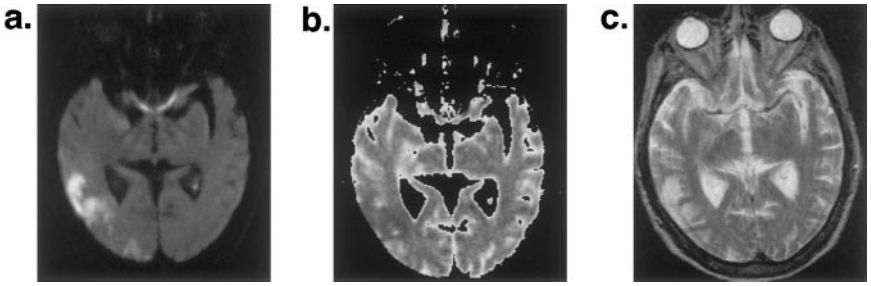

Figure 1. A large region of DWI and ADC imaging abnormality in the right temporal lobe in a patient with a left hemiparesis that rapidly resolved within 5 hours of onset. Other slices (not shown) demonstrated frontal and parietal regions of abnormality. a, DWI image; b, ADC image; and c, baseline T2-weighted image.

promise (examples are shown in Figures 1 through 3). In 5 (25%) of these 20 patients, there was no lesion correlate on initial T2-weighted sequences. The remaining 15 patients did exhibit T2-weighted lesion abnormalities in the same regions as DWI alterations. Two patients with visible but very small DWI abnormalities did not have a measurable ADC volume. There were no significant differences in baseline characteristics and vascular risk factors between patients demonstrating DWI abnormalities versus those who did not (Table 2).

An accurate estimation of TIA duration (all were unequivocally <24 hours) was available for 15 of the 20 patients with DWI abnormalities and 17 of the 22 without DWI abnormalities. Duration of TIA symptoms for patients without a DWI abnormality was a mean of 3.2 hours (±4.7 hours SD) and median of 0.5 hours versus a mean of 7.3 hours (±6 hours SD) and median 4.0 hours for patients with a DWI abnormality (*t* test for difference in means, $P=0.03$). The percentage of patients with a DWI abnormality within various symptom duration intervals increased as the total duration of symptoms increased (Figure 4).

The mean time from symptom onset to MRI study for all TIA patients (with available data) was 17 hours (range, 1.25 to 73 hours) and did not significantly differ between the 2 groups (mean, 15.8 hours for DWI-negative patients and 19.5 hours for DWI-positive patients). One patient in the group without DWI lesions was still symptomatic at the time of the MR imaging, while 2 in the DWI- positive category were still symptomatic at the time of MR imaging. Interval from time of resolution of TIA symptoms to time of MRI for patients with a DWI abnormality was a mean of 12.7 hours (median, 8.8 hours) in patients with a DWI abnormality versus a mean of 12.9 hours (median, 5.1 hours) in patients without a DWI abnormality (rank sum test for difference in medians, $P=0.7$).

In the 20 patients with diffusion MR abnormalities, DWI signal changes were localized to the brain stem in 4 patients, the cerebellum in 2 patients, subcortical hemispheric structures in 7 patients, and cortical regions in 7 patients. Vascular territories affected were superficial middle cerebral artery in 6 patients, deep middle cerebral artery in 6 patients, brain stem perforators in 4 patients, and posterior cerebral arteries in 2 patients. In these 20 patients, the final etiologic mechanism was felt to be small-vessel lacunar in 9 patients, large-vessel atherothrombotic in 4 patients, and cardioembolic in 7 patients.

DWI results altered the attending physician's opinion regarding vascular localization in 7 of 20 patients, anatomic localization in 8 of 20 patients, and probable TIA mechanism in 6 of 20 patients. The types of alterations in diagnosis were quite varied, and no single pattern predominated. For example, among etiologic diagnoses, of 4 patients initially suspected of having large-artery atherothrombotic mechanisms, 1 changed after DWI to likely cardioembolic and 1 changed to likely small vessel; of 7 initial cardioembolic diagnoses, 1 changed to likely large-vessel atherothrombotic and 1 to likely small vessel; and of 9 initial small-vessel diagnoses, 1 changed to likely large-vessel atherothrombotic and 1 to likely cardioembolic.

There were significant differences in the DWI and ADC MR signature between the TIA patients with DWI abnormalities and the patients with completed stroke (Table 3, Figure 5). There was a strong correlation ($r=0.77$) between ADC and DWI volumes.

All 20 TIA patients demonstrating DWI abnormalities were contacted for a follow-up MRI, and 9 of these patients agreed to return for repeat neuroimaging. Three patients were studied with head CT and 6 with brain MRI 2 to 7 months after the event. Of these 9 patients, 5 (3 MRI, 2 CT) demonstrated a subsequent infarct in the region corresponding to the original DWI abnormality (Figure 3) while 4 (3 MRI, 1 CT) did not (Figure 2). Five of the 22 patients without a DWI abnormality underwent follow-up imaging (3 MRI, 2

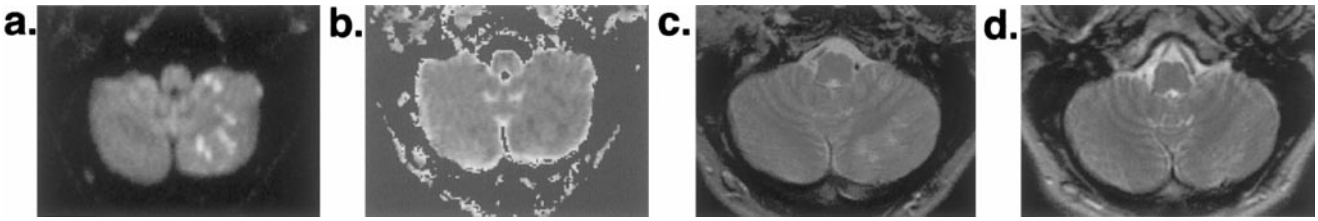

Figure 2. Several foci of left cerebellar DWI and ADC abnormality in a patient presenting with 6 hours of vertigo and left-arm clumsiness. Other slices (not shown) also showed foci of right cerebellar DWI abnormality. Follow-up MRI at 3 months did not show corresponding late ischemic changes on T1- or T2-weighted images. a, DWI image; b, ADC image; c, baseline T2-weighted image; and d, late (follow-up) T2-weighted image.

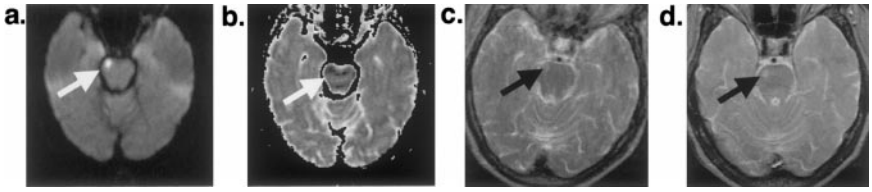

Figure 3. Diffusion MR sequences show an abnormality in the right cerebral peduncle of the midbrain in a patient presenting with 16 hours of transient left hemiparesis. Repeat MRI 5 months later shows an infarct in the same region. a, DWI image; b, ADC image; c, baseline T2-weighted image; and d, late (follow-up) T2-weighted image.

CT) 2 weeks to 15 months after the event. None demonstrated a subsequent relevant infarct.

Discussion

Traditionally, the term "transient ischemic attack" often was taken to imply absence of permanent ischemic injury. Since the 1970s, however, increasing evidence has accumulated from neuroimaging studies using CT and conventional T1- and T2-weighted MRI suggesting that a substantial proportion of patients with TIAs experience some degree of permanent ischemic injury. These studies, however, were limited by the inability of noncontrast and even contrast CT and MRI to accurately differentiate acute lesions from prior, unrelated ischemic events.[9]

Our findings with diffusion MRI confirm and expand on these earlier imaging studies. We found that 48% of cerebral TIA patients demonstrated diffusion MRI evidence of acute bioenergetic compromise. Among TIA patients with early DWI abnormalities who had follow-up imaging, approximately one half exhibited late CT or MRI evidence of established infarction. Together, these data suggest that approximately one quarter to one third of cerebral TIA patients actually have cerebral infarction with transient signs.

We also identified a distinct subset of TIA patients, representing about one fifth of TIA cases, who had early DWI abnormalities but no late evidence of established infarction. This finding suggests that DWI abnormalities may be fully reversible in humans if early restoration of blood flow is obtained. The reversibility of DWI lesions has been consistently demonstrated in animals but only in a few case reports in humans.[13,16,19–21] Hasegawa and colleagues[22] found that there was a critical intensity threshold for reversal of ADC abnormalities that might allow discrimination of potentially salvageable tissue, while Minematsu and colleagues[21] reported that reversible DWI lesions were less intense than irreversible lesions.

TABLE 2. Baseline Patient Characteristics

	No DWI Abnormality n=22	DWI Abnormality n=20
Age, y (mean, range)	72 (41–82)	72 (37–93)
Sex, F/M	11/11	6/14
Hypertension, n	15 (68)	8 (40)
Diabetes mellitus, n	6 (27)	7 (35)
Coronary artery disease, n	9 (41)	10 (50)
Tobacco use, n	11 (50)	9 (45)
Hypercholesterolemia, n	15 (68)	9 (45)
History of prior stroke, n	5 (23)	7 (35)
History of prior TIA, n	6 (27)	4 (20)

Values in parentheses are percent unless otherwise specified.

This study allowed us to compare the diffusion MR signatures of clinical TIA patients with those in patients with clinically completed stroke. In TIA patients, the ADC volume, mean ADC value, DWI volume, and DWI signal intensity were all significantly less abnormal than in acute stroke patients. It is important to point out that the ADC calculations were based on only 2 b values, thus yielding only semiquantitative measurements. Nonetheless, the significant differences between the 2 groups support the concept that the cerebral ischemia experienced by patients with TIAs is smaller in volume and lesser in severity than that experienced by patients with clinically completed stroke syndromes.

Diffusion MRI signatures of brain bioenergetic compromise may be visualized with either DWI or ADC imaging or both. In many cases, these techniques provide complementary data. Among the TIA patients, the DWI sequences allowed immediate and clear visualization of diffusion abnormalities while the ADC maps provided a more precise and semiquantitative characterization of the lesions. Lesion volumes defined by DWI abnormalities generally exceeded lesion volumes defined by ADC abnormalities. This discordance likely reflects the strong influence of transverse relaxation time (T2) on the DWI image intensity, leading to overestimation of the effect directly related to acute versus subacute ischemia. There were only 2 patients with visible but very small DWI abnormalities who did not have measurable ADC volumes. These patients likely had abnormalities that were either beyond the spatial resolution of current ADC detection techniques or beyond our chosen ADC threshold.

Several prior studies,[6,8,10] but not all,[11] have demonstrated a correlation between the duration of TIA symptoms and the subsequent development of an infarct: longer events are more likely to produce permanent alterations on CT and standard MR imaging. We found significant statistical correlation between duration of TIA symptoms and presence of a lesion on DWI. This correlation, however, was not absolute, and several patients with DWI lesions had symptoms lasting <10 minutes, while some patients in the DWI negative group had symptoms lasting hours.

In addition to improving our understanding of the underlying pathophysiological processes that occur with TIAs, our data add to a growing body of evidence demonstrating the clinical utility of DWI.[23,24] A variety of studies have demonstrated that the diagnosis of TIA is often difficult, especially for the nonneurologist.[25,26] Kraaijeveld and colleagues[27] found κ measures of interrater agreement of only 0.65 among 8 experienced neurologists diagnosing 56 TIA patients and only 0.31 for determination of the vascular territory involved. The size, appearance, and location of the lesion(s) may help guide physicians in determining the underlying TIA mechanism and in choosing the optimal therapeutic regimen to

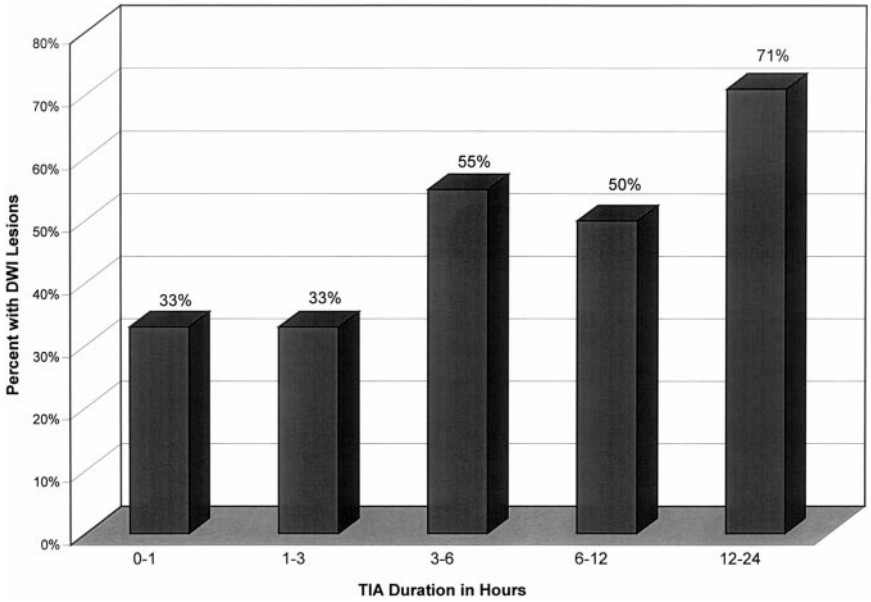

Figure 4. Relationship of TIA symptom duration to presence or absence of DWI abnormality in different duration cohorts.

reduce the probability of recurrent TIAs or completed stroke in the future.

In our study, information obtained from the DWI study led to a change in the suspected anatomic localization, vascular localization, and TIA mechanism in over one third of patients. In addition to clarifying the site and source of ischemia in patients with clinically definite TIAs, diffusion imaging also may be helpful in patients with atypical transient neurological symptoms, when it is unclear whether the event was a TIA versus migraine, hyperventilation, brief seizure, or other TIA mimic. Although DWI abnormalities have rarely been reported in TIA mimics, a visualized diffusion abnormality in these cases generally provides supportive evidence of the diagnosis of TIA.

The observation that DWI alone was positive in 25% of patients while 75% had correlative lesions identified retrospectively on T2 imaging underestimates the diagnostic impact of DWI. Even in the patients with T2 visible lesions, the diffusion imaging provided added clinical utility. Many of the T2-positive patients had multiple foci of increased T2 signal, and determining which, if any, T2 foci were new and related to the recent TIA may not have been possible without the DWI sequences. Standard T2 studies alone are generally incapable of differentiating acute from chronic events.

TABLE 3. Comparison of DWI Signature Between TIA Patients With Diffusion MR Abnormality and Patients With Completed Stroke

	TIA Patients (n=20)	Completed Stroke Patients (n=23)	
	Mean (SEM)	Mean (SEM)	*P*
DWI lesion volume, cm^3	2.9 (8.4)	22.2 (7.8)	0.0002†
ADC volume,* cm^3	0.7 (3.4)	10.5 (3.2)	0.0001†
DWI intensity, %	35 (5)	62 (5)	0.001‡
Mean ADC value, $\mu m^2/s$	442 (6.7)	409 (6.1)	0.009‡

*Includes 2 patients with visible but very small DWI abnormalities who did not have a measurable ADC volume.

†Rank sum test.

‡*t* test.

Identification of which patients have a new infarct on imaging may have important prognostic value.[28] In the Dutch TIA trial,[29,30] evidence of any cerebral infarct on CT was an independent risk factor for subsequent stroke, myocardial infarction, or vascular death. Evans and colleagues[7] reported that in TIA patients, CT-verified infarction increased the risk of death by 109% over a 10-year period following the TIA. However, this study did not correlate the risk with evidence of a new, appropriately located TIA-related infarct. Eliasziw and colleagues[31] did not find an increased risk of ipsilateral stroke in a group of TIA patients with severe carotid stenosis treated medically as part of the North American Symptomatic Carotid Endarterectomy Trial; however, these results cannot be generalized to all TIA patients. Only larger series with long-term follow-up will be able to distinguish whether there is a difference in prognosis in TIA patients without diffusion abnormalities, TIA patients with transient diffusion abnormalities but no eventual T2 lesion, and patients with diffusion abnormalities and a subsequent T2 lesion.

To the best of our knowledge, no prior formal studies have been reported that specifically evaluated DWI abnormalities in TIA patients. In their study of DWI findings in 40 patients presenting with symptoms of acute ischemic stroke, Warach and colleagues[32] reported that 4 patients did not have a DWI correlate and that all 4 patients had resolution of their symptoms (at least 1 within 24 hours). Sorensen et al[20] also reported negative DWI results in two patients whose clinical findings resolved within 24 to 48 hours. However, both subsequently gave a history suggestive of hemiplegic migraine. We are not aware of any prior report of positive DWI or ADC studies in patients with presumed cerebrovascular disease whose clinical symptoms completely resolved within 24 hours. There are, however, several reports[33,34] of reversible DWI abnormalities in patients with transient global amnesia and status epilepticus.

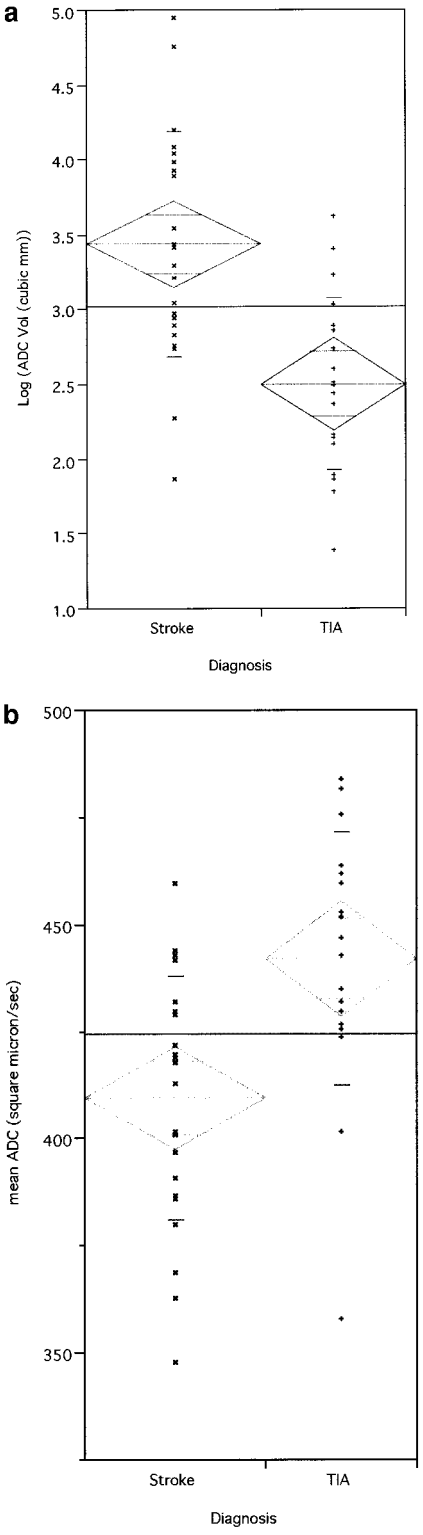

Figure 5. Comparison of ADC measurements between patients with completed stroke and TIA patients with DWI abnormalities: a, ADC lesion volume (logarithmic scale); b, mean ADC.

There are several limitations to this study. Perfusion-weighted imaging, not performed in our patients, would likely have provided additional relevant pathophysiological information. Several functional studies using SPECT or xenon CT have demonstrated that up to one third of TIA patients may have a focal area of hypoperfusion.[35–37] Also, we were not able to obtain follow-up MR imaging studies on all our patients, limiting the strength of our observation regarding long-term imaging outcome. Although it is unlikely that patients without a DWI abnormality acutely would go on to have a relevant infarct on subsequent imaging, this will need to be confirmed in a prospective study.

Several issues require further study. The clinical prognostic significance of finding an associated DWI abnormality in a patient with a TIA remains uncertain. We concur with the general view advanced by Caplan[38] that all TIA patients are at significant risk of subsequent vascular events, and it is the underlying mechanism rather than the duration of symptoms that is most critical to determine. However, it may be that within each mechanism category, longer duration of a TIA or presence of a DWI abnormality identifies a subgroup at increased risk. How often patients with DWI abnormalities are experiencing ongoing ischemia will need to be clarified by concurrent perfusion studies. The pathologic correlates of DWI changes in TIA require investigation including how often signal abnormalities reflect, at the histopathologic level, absence of infarction, incomplete infarction, or complete infarction.

In summary, this study demonstrated that among 42 consecutive TIA patients, 20 (48%) demonstrated acute, relevant DWI abnormalities. The presence of a DWI lesion increased with increasing total symptom duration. This information was clinically useful in determining the anatomic and vascular localization of the event and the etiologic mechanism. In 4 of 9 patients undergoing follow-up imaging, a relevant infarct was not visualized, suggesting that the acute DWI changes can sometimes reflect fully reversible ischemia. Finally, in those patients with DWI lesions, the MR signature (DWI volume, ADC volume, mean ADC value, and DWI intensity) differed significantly from that of patients with completed stroke.

Acknowledgments

Financial support for this project was provided by Grant-in-Aid 9650621N from the American Heart Association (to Dr Alger) and by a fellowship grant from the National Stroke Association (to Dr Kidwell).

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