

Perfusion MRI Abnormalities in Speech or Motor Transient Ischemic Attack Patients

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Background and Purpose—Transient ischemic attack (TIA) patients may deteriorate rapidly. MRI is being increasingly used to assess such patients. One possible mechanism of neurological worsening is the presence of perfusion abnormalities. We sought to identify what proportion of TIA patients had evidence of perfusion abnormalities on MRI.

Methods—TIA patients were prospectively enrolled and had a MRI completed as soon as possible. The images were assessed for the presence of perfusion abnormalities.

Results—Sixty-nine TIA patients were enrolled, and 62 had perfusion imaging. In 56 patients (81%), the symptoms had resolved before imaging. In 21 patients (33.9%), there was evidence of a perfusion abnormality defined by relative mean transit time delay. In 12 patients (19.4%), the perfusion abnormality was present despite having complete resolution of neurological symptoms. We found no relationship between the presence of a perfusion abnormality and the clinical outcome.

Conclusions—A proportion of TIA patients have perfusion abnormalities evident on MRI. (*Stroke*. 2005;36:2487-2489.)

Key Words: acute stroke ■ MRI ■ transient ischemic attack

Diffusion weighted imaging (DWI) is important in the diagnosis of ischemic stroke patients. Currently, the definition of transient ischemic attack (TIA) is a sudden onset of neurological symptoms that are of vascular etiology and resolve within 24 hours. However, many patients with TIA have injury observed on DWI imaging.¹⁻⁴ The assumption that TIAs are associated with complete resolution of brain ischemia leaving no permanent brain injury^{5,6} is false. There is growing evidence that TIA is not a benign condition and that the risk of a subsequent stroke is high within the first 48 hours⁷ of symptom onset.

Perfusion weighted imaging (PWI) using gadolinium-based dynamic-susceptibility contrast provides information on ischemia. In this prospective study, we sought to understand whether MRI perfusion abnormalities exist among TIA patients despite the rapid resolution of symptoms.

Methods

Patients who were prospectively enrolled with hemiparesis or aphasia that resolved within 24 hours were examined within 12 hours of onset, were independent on the modified Rankin scale, and were ≥ 18 years of age. Images were obtained using a 3-T scanner. The PWI was acquired using dynamic susceptibility contrast imaging. Imaging was assessed by a neuroradiologist blind to clinical information except the symptom side. Images were examined for the presence of an acute DWI lesion, for an occlusion on intracranial

magnetic resonance angiography (MRA), and for a perfusion delay on the mean transit time (MTT) map.

Patients were assessed with the National Institutes of Health Stroke Scale at 24 hours and with the modified Rankin scale at 3 months. The potential mechanism assigned using the Trial of Org 10172 in Acute Treatment classification and any recurrent strokes were recorded.

The association between the presence or absence of a relative MTT delay (PWI lesion) and baseline characteristics was assessed. Logistic regression modeling using backward elimination was used to identify the predictors of a perfusion abnormality.

Results

Sixty-nine TIA patients were enrolled. The median duration of symptoms was 90 minutes (range, 5 to 1380 minutes). Twenty-seven patients were women (39%). In 56 of the patients (81%), the symptoms had resolved by the beginning of the MRI. Thirty-two patients (46.4%) had evidence of a DWI hyperintensity.

In 7 patients (10.1%), the PWI sequence was not completed or was not interpretable because of technical difficulty. These patients were excluded from additional analysis. In 21 patients (33.9%), there was evidence of a delay in the MTT map (Tables 1 and 2). The mean time to MRI scan was 9.2 hours in patients with a PWI lesion and was 13.6 hours in patients without a PWI lesion ($P=0.04$). At the time of MRI,

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TABLE 1. Comparison of Clinical Factors in Patients with PWI Lesions and Those Who Do Not Have Evidence of PWI Lesion (Univariable Analysis)

Variables	PWI Lesion (n=21)	No PWI Lesion (n=41)	P Value
Age (mean, SD)	69 (12)	66 (12)	0.25
Male gender	52%	61%	0.59
NIHSS (median)	0 (0–1)	0 (0–0)	0.07*
Onset-to-MRI time (median, interquartile range)	356 (184–811)	725 (342–1285)	0.03*
Internal carotid artery stenosis >50%	14%	2%	0.11
Symptoms resolved before beginning of MRI	57%	95%	0.001
Any intracranial occlusion	48%	10%	0.003

Variables (blood pressure, serum glucose, diabetes mellitus, etc) that were not significant at $P < 0.05$ are not shown.

*Mann–Whitney U test.

12 patients (19.4%) had complete resolution of neurological symptoms, yet still had perfusion abnormalities (Figure).

In a multivariable model adjusting for age and time from onset to MRI, occlusion was the most important predictor of PWI lesion ($P = 0.001$). However, the time to MRI was a predictor of the presence of PWI lesion ($P = 0.039$). Patients with shorter onset to MRI times were more likely to show PWI abnormalities.

Nearly all of the patients (66 of 69, 95%) had a modified Rankin score of 0 or 1 at 3 months. Of the 3 patients who had a modified Rankin score > 1 , all had resolved by the time of the MRI scan, but 2 had a persistent PWI abnormality. Two of these patients had recurrent strokes (1 who had a PWI abnormality and 1 who did not).

Discussion

We found that, despite many patients having transient symptoms, a proportion of motor or speech TIA patients have perfusion abnormalities evident on MRI. Patients with shorter onset to MRI times were also more likely to show PWI abnormalities. We found no relationship of perfusion abnormalities to clinical outcome. The only factor predictive of a perfusion abnormality in a multivariable analysis was the presence of a vessel occlusion on MRA.

Similar to our results, a recent study⁸ found that 32% of TIA patients had evidence of an MRI perfusion abnormality. We found that nearly all of the patients had an excellent

TABLE 2. No. of Patients with DWI Positive/Negative and PWI Positive/Negative Respectively, Grouped by Time to Magnetic Resonance Scanning

Variables	<6 h (n=23)	6–12 h (n=12)	12–24 h (n=16)	>24 h (n=11)
DWI+, PWI+	10	3	2	3
DWI+, PWI–	3	2	4	3
DWI–, PWI+	1	0	1	0
DWI–, PWI–	9	7	9	5

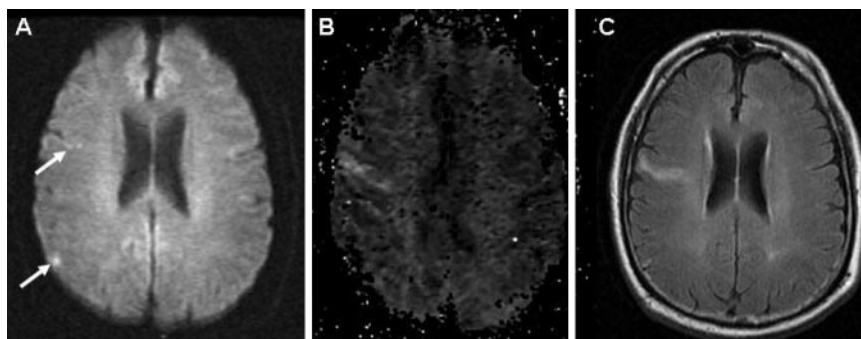
outcome. A possible reason for good outcomes in this study is that all of the patients were admitted to the hospital. If there was a clinical deterioration before 24 hours, then these patients would not be classified as a TIA. Importantly, a discord between the clinical resolution and the presence of a PWI abnormality was observed. Additional study using quantitative perfusion techniques is required to determine whether symptom resolution is associated with benign oligemia.⁹

The 3-T MRI may be a limitation, because it may identify more areas of perfusion abnormality than a 1.5T system; also, these results are only applicable to speech and motor TIAs, because sensory TIAs were excluded. Patients with severe carotid stenosis can have chronic MTT delays,¹⁰ and follow-up perfusion imaging would have been helpful to see if any perfusion lesions were chronic, especially in patients with no evidence of diffusion lesions. The delay to MRI was longer in patients without a PWI lesion, suggesting that our results may underestimate the proportion of TIA patients with perfusion abnormalities. Cerebral blood volume and blood flow maps were also not examined because of technical difficulties. The reader did know symptom side, which may have biased detection of perfusion abnormality. Blinded review may have resulted in fewer perfusion lesions seen.

A proportion of TIA patients have evidence of perfusion abnormalities on MRI. We did not find any relationship with clinical outcome or recurrent events.

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(A) DWI lesions shown by arrows at baseline in a TIA patient whose symptoms of left-sided weakness resolved within 30 minutes. (B) MTT abnormality at baseline. (C) Asymptomatic, hyperintense region in right middle cerebral artery territory on the fluid-attenuated inversion recovery at 30 days.

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References

1. Kidwell CS, Alger JR, Di Salle F, Starkman S, Villablanca P, Bentson J, Saver JL. Diffusion MRI in patients with transient ischemic attacks. *Stroke*. 1999;6:1174–1180.
2. Crisostomo RA, Garcia MM, Tong DC. Detection of diffusion-weighted MRI abnormalities in patients with transient ischemic attack: correlation with clinical characteristics. *Stroke*. 2003;34:932–937.
3. Ay H, Oliveira-Filho J, Buonanno FS, Schaefer PW, Furie KL, Chang YC, Rordorf G, Schwamm LH, Gonzalez RG, Koroshetz WJ. 'Footprints' of transient ischemic attacks: a diffusion-weighted MRI study. *Cerebrovasc Dis*. 2002;14:177–186.
4. Inatomi Y, Kimura K, Yonehara T, Fujioka S, Uchino M. DWI abnormalities and clinical characteristics in TIA patients. *Neurology*. 2004;62:376–380.
5. Albers GW, Caplan LR, Easton JD, Fayad PB, Mohr JP, Saver JL, Sherman DG. TIA Working Group. Transient ischemic attack—proposal for a new definition. *N Engl J Med*. 2002;347:1713–1716.
6. Warach S, Kidwell CS. The redefinition of TIA: the uses and limitations of DWI in acute ischemic cerebrovascular syndromes. *Neurology*. 2004;62:359–360.
7. Johnston SC, Gress DR, Browner WS, Sidney S. Short-term prognosis after emergency department diagnosis of TIA. *JAMA*. 2000;284:2901–2906.
8. Restrepo L, Jacobs MA, Barker PB, Wityk RJ. Assessment of transient ischemic attack with diffusion- and perfusion-weighted imaging. *AJNR Am J Neuroradiol*. 2004;25:1645–1652.
9. Kidwell CS, Alger JR, Saver JL. Beyond mismatch: evolving paradigms in imaging the ischemic penumbra with multimodal magnetic resonance imaging. *Stroke*. 2003;34:2729–2735.
10. Chaves CJ, Staroselskaya I, Linfante I, Llinas R, Caplan LR, Warach S. Patterns of perfusion-weighted imaging in patients with carotid artery occlusive disease. *Arch Neurol*. 2003;60:237–242.

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