DVI Lesions and TIA Etiology Improve the Prediction of Stroke After TIA

David Calvet, MD; Emmanuel Touzé, MD, PhD; Catherine Oppenheim, MD, PhD; Guillaume Turc, MD; Jean-François Meder, MD, PhD; Jean-Louis Mas, MD

Background and Purpose—The ABCD² score has been shown to predict the early risk of stroke after transient ischemic attack (TIA). The additional predictive value of diffusion-weighted imaging (DWI) and TIA etiology is not well known.

Methods—From January 2003 to June 2007, 343 consecutive patients (mean±SD age, 62.4±15.4 years) with TIA were admitted to our stroke unit. Most (339) patients underwent DWI and all had an etiologic work-up and were followed up for 3 months. The predictive value of the ABCD² score, positive DWI findings, large-artery atherosclerosis (LAA), and atrial fibrillation (AF) with respect to occurrence of ischemic stroke at 1 week and 3 months was assessed.

Results—DVI was positive in 136 (40%) patients. Sixty (17%) patients had LAA and 27 (8%) had AF. Patients with positive DWI findings were more likely to have unilateral weakness (odds ratio [OR]=2.2; 95% CI, 1.3 to 3.7), TIA duration ≥60 minutes (OR=2.6; 95% CI, 1.3 to 5.2), ABCD² >5 (OR=4.7; 95% CI, 2.0 to 11.0), LAA (OR=1.8; 95% CI, 1.0 to 3.1), and AF (OR=3.5; 95% CI, 1.5 to 8.0). During follow-up, 5 patients had a stroke within 7 days (absolute risk=1.5%, 95% CI, 0.3% to 2.7%), and 10 had a stroke within 3 months (absolute risk=2.9%; 95% CI, 1.1% to 4.7%). All early strokes but 1 occurred in patients with positive DWI findings. ABCD² score and positive DWI findings were associated with an increased 7-day and 3-month risk of stroke. At 3 months, ABCD² score >5 (hazard ratio=10.1; 95% CI, 1.1 to 93.4), positive DVI result (hazard ratio=8.7; 95% CI, 1.1 to 71.0), and LAA (hazard ratio=3.4; 95% CI, 1.0 to 11.8) were independently associated with an increased risk of stroke. There was no association with AF.

Conclusions—Taking DVI and TIA etiology into account in addition to the ABCD² score improves the prediction of the early risk of stroke after TIA. (Stroke. 2009;40:000-000.)

Key Words: atherosclerosis ■ diffusion-weighted imaging ■ prognosis ■ transient ischemic attack

The early risk of stroke after transient ischemic attack (TIA) is higher than previously thought and is predictable from simple clinical features.1–3 The ABCD² clinical score has been shown to accurately predict that risk in population- and emergency department–based studies.4 It has also been suggested that the presence of ischemic lesions on diffusion-weighted imaging (DWI) and TIA etiology, such as large-artery atherosclerosis (LAA)4 and atrial fibrillation (AF), could improve stroke risk prediction after TIA.4–7 However, studies that assessed the prognostic value of positive DWI findings and TIA etiology either were underpowered or used composite outcomes that included cardiovascular events or recurrent TIA.5–10 Moreover, because positive DWI findings and TIA etiology may be correlated with several components of the ABCD² score,11,12 it remains uncertain whether those potential factors can predict the early risk of stroke independently of the ABCD² score. We therefore assessed the additional value of positive DWI findings and TIA etiology to predict the early risk of stroke in a cohort of 343 consecutive TIA patients admitted to a stroke unit.

Methods

Among 3022 consecutive patients admitted to our stroke unit for ischemic stroke or TIA from January 2003 to June 2007, 343 patients with probable or possible TIA according to National Institute of Neurological Disorders and Stroke criteria13 and who were admitted within 48 hours of symptom onset were enrolled. Patients are referred to our stroke unit by emergency departments from different hospitals located in our geographic area, general practitioners, and emergency ambulance services. Patients are admitted as soon as the neurologist is contacted by telephone; no selection criteria were applied for TIA and they were treated as inpatients. Data on management and DWI–magnetic resonance imaging (MRI) findings of the first patients enrolled in this cohort have been previously reported.8,14,15 Demographic data, vascular risk factors, and past medical history were collected at the time of admission on a standardized case report form. The first blood pressure recorded after TIA was collected from general practitioner notes, emergency department notes, or at admission to the stroke unit, depending on where the patient was first examined after the TIA. In patients with several recent TIs, the most recent event was considered.

Among the 343 enrolled patients, all but 4 (pacemaker in 2 and prosthetic valve in 2) underwent brain MRI (1.5-T MRI unit equipped with echoplanar capability; Signa, General Electric Medical Systems, Milwaukie, Wis) within the day after admission. Our

Received January 29, 2008; final revision received May 7, 2008; accepted May 30, 2008.

Correspondence and reprint requests to Pr Jean-Louis Mas, Service de Neurologie, Hôpital Sainte-Anne, 1 rue Cabanis, 75014 Paris, France. E-mail jl.mas@ch-sainte-anne.fr
© 2008 American Heart Association, Inc.

Stroke is available at http://stroke.ahajournals.org

DOI: 10.1161/STROKEAHA.108.515817
diagnosed at admission or during hospitalization. Patients were
tined, according to TOAST classification, as the presence of a
(78%) underwent transesophageal echocardiography. LAA was de-
(340, 99%) underwent transthoracic echocardiography, and 269
normal Doppler results or contraindications to MRI. Most patients
re produce a good result of a variable. Ideal prediction
routine MRI stroke protocol included the following sequences
acquired in the axial plane: spin-echo DWI (b=0 to 1000 s/mm²),
fast fluid-attenuated inversion recovery, gradient-echo T2-weighted
and 3D time-of-flight angiography of the circle of Willis. The
presence of acute ischemic lesions was defined by areas of high
signal intensity on DWI. All films and reports were retrospectively
reviewed without knowledge of baseline clinical and follow-up data.
All patients underwent a standardized etiologic work-up, includ-
ing standard blood tests, 12-lead ECG, prolonged 3-lead cardiac
monitoring, and Doppler ultrasound. All Doppler ultrasound scans
were performed with a Phillips ATL 5000 echograph, and standard-
ized diagnostic criteria were used for artery stenosis.16 Cervical
gadolinium-enhanced MR angiography was performed in 307 (90%)
routine MRI stroke protocol included the following sequences
acquired in the axial plane: spin-echo DWI (b=0 to 1000 s/mm²),
fast fluid-attenuated inversion recovery, gradient-echo T2-weighted
and 3D time-of-flight angiography of the circle of Willis. The
presence of acute ischemic lesions was defined by areas of high
signal intensity on DWI. All films and reports were retrospectively
reviewed without knowledge of baseline clinical and follow-up data.
All patients underwent a standardized etiologic work-up, includ-
ing standard blood tests, 12-lead ECG, prolonged 3-lead cardiac
monitoring, and Doppler ultrasound. All Doppler ultrasound scans
were performed with a Phillips ATL 5000 echograph, and standard-
ized diagnostic criteria were used for artery stenosis.16 Cervical
gadolinium-enhanced MR angiography was performed in 307 (90%)
routine MRI stroke protocol included the following sequences
acquired in the axial plane: spin-echo DWI (b=0 to 1000 s/mm²),
fast fluid-attenuated inversion recovery, gradient-echo T2-weighted
and 3D time-of-flight angiography of the circle of Willis. The
presence of acute ischemic lesions was defined by areas of high
signal intensity on DWI. All films and reports were retrospectively
reviewed without knowledge of baseline clinical and follow-up data.
All patients underwent a standardized etiologic work-up, includ-
ing standard blood tests, 12-lead ECG, prolonged 3-lead cardiac
monitoring, and Doppler ultrasound. All Doppler ultrasound scans
were performed with a Phillips ATL 5000 echograph, and standard-
ized diagnostic criteria were used for artery stenosis.16 Cervical
gadolinium-enhanced MR angiography was performed in 307 (90%)
routine MRI stroke protocol included the following sequences
acquired in the axial plane: spin-echo DWI (b=0 to 1000 s/mm²),
fast fluid-attenuated inversion recovery, gradient-echo T2-weighted
and 3D time-of-flight angiography of the circle of Willis. The
presence of acute ischemic lesions was defined by areas of high
signal intensity on DWI. All films and reports were retrospectively
reviewed without knowledge of baseline clinical and follow-up data.
All patients underwent a standardized etiologic work-up, includ-
ing standard blood tests, 12-lead ECG, prolonged 3-lead cardiac
monitoring, and Doppler ultrasound. All Doppler ultrasound scans
were performed with a Phillips ATL 5000 echograph, and standard-
ized diagnostic criteria were used for artery stenosis.16 Cervical
gadolinium-enhanced MR angiography was performed in 307 (90%)
routine MRI stroke protocol included the following sequences
acquired in the axial plane: spin-echo DWI (b=0 to 1000 s/mm²),
fast fluid-attenuated inversion recovery, gradient-echo T2-weighted
and 3D time-of-flight angiography of the circle of Willis. The
presence of acute ischemic lesions was defined by areas of high
signal intensity on DWI. All films and reports were retrospectively
reviewed without knowledge of baseline clinical and follow-up data.
All patients underwent a standardized etiologic work-up, includ-
ing standard blood tests, 12-lead ECG, prolonged 3-lead cardiac
monitoring, and Doppler ultrasound. All Doppler ultrasound scans
were performed with a Phillips ATL 5000 echograph, and standard-
ized diagnostic criteria were used for artery stenosis.16 Cervical
gadolinium-enhanced MR angiography was performed in 307 (90%)
routine MRI stroke protocol included the following sequences
acquired in the axial plane: spin-echo DWI (b=0 to 1000 s/mm²),
fast fluid-attenuated inversion recovery, gradient-echo T2-weighted
and 3D time-of-flight angiography of the circle of Willis. The
presence of acute ischemic lesions was defined by areas of high
signal intensity on DWI. All films and reports were retrospectively
reviewed without knowledge of baseline clinical and follow-up data.
All patients underwent a standardized etiologic work-up, includ-
ing standard blood tests, 12-lead ECG, prolonged 3-lead cardiac
monitoring, and Doppler ultrasound. All Doppler ultrasound scans
were performed with a Phillips ATL 5000 echograph, and standard-
ized diagnostic criteria were used for artery stenosis.16 Cervical
gadolinium-enhanced MR angiography was performed in 307 (90%)
routine MRI stroke protocol included the following sequences
acquired in the axial plane: spin-echo DWI (b=0 to 1000 s/mm²),
fast fluid-attenuated inversion recovery, gradient-echo T2-weighted
and 3D time-of-flight angiography of the circle of Willis. The
presence of acute ischemic lesions was defined by areas of high
signal intensity on DWI. All films and reports were retrospectively
reviewed without knowledge of baseline clinical and follow-up data.
All patients underwent a standardized etiologic work-up, includ-
ing standard blood tests, 12-lead ECG, prolonged 3-lead cardiac
monitoring, and Doppler ultrasound. All Doppler ultrasound scans
were performed with a Phillips ATL 5000 echograph, and standard-
ized diagnostic criteria were used for artery stenosis.16 Cervical
gadolinium-enhanced MR angiography was performed in 307 (90%)
routine MRI stroke protocol included the following sequences
acquired in the axial plane: spin-echo DWI (b=0 to 1000 s/mm²),
fast fluid-attenuated inversion recovery, gradient-echo T2-weighted
and 3D time-of-flight angiography of the circle of Willis. The
presence of acute ischemic lesions was defined by areas of high
signal intensity on DWI. All films and reports were retrospectively
reviewed without knowledge of baseline clinical and follow-up data.
All patients underwent a standardized etiologic work-up, includ-
ing standard blood tests, 12-lead ECG, prolonged 3-lead cardiac
monitoring, and Doppler ultrasound. All Doppler ultrasound scans
were performed with a Phillips ATL 5000 echograph, and standard-
ized diagnostic criteria were used for artery stenosis.16 Cervical
gadolinium-enhanced MR angiography was performed in 307 (90%)
routine MRI stroke protocol included the following sequences
acquired in the axial plane: spin-echo DWI (b=0 to 1000 s/mm²),
fast fluid-attenuated inversion recovery, gradient-echo T2-weighted
and 3D time-of-flight angiography of the circle of Willis. The
presence of acute ischemic lesions was defined by areas of high
signal intensity on DWI. All films and reports were retrospectively
reviewed without knowledge of baseline clinical and follow-up data.
All patients underwent a standardized etiologic work-up, includ-
ing standard blood tests, 12-lead ECG, prolonged 3-lead cardiac
monitoring, and Doppler ultrasound. All Doppler ultrasound scans
were performed with a Phillips ATL 5000 echograph, and standard-
ized diagnostic criteria were used for artery stenosis.16 Cervical
gadolinium-enhanced MR angiography was performed in 307 (90%)
routine MRI stroke protocol included the following sequences
acquired in the axial plane: spin-echo DWI (b=0 to 1000 s/mm²),
fast fluid-attenuated inversion recovery, gradient-echo T2-weighted
and 3D time-of-flight angiography of the circle of Willis. The
presence of acute ischemic lesions was defined by areas of high
signal intensity on DWI. All films and reports were retrospectively
reviewed without knowledge of baseline clinical and follow-up data.
All patients underwent a standardized etiologic work-up, includ-
ing standard blood tests, 12-lead ECG, prolonged 3-lead cardiac
monitoring, and Doppler ultrasound. All Doppler ultrasound scans
were performed with a Phillips ATL 5000 echograph, and standard-
ized diagnostic criteria were used for artery stenosis.16 Cervical
gadolinium-enhanced MR angiography was performed in 307 (90%)
routine MRI stroke protocol included the following sequences
acquired in the axial plane: spin-echo DWI (b=0 to 1000 s/mm²),
fast fluid-attenuated inversion recovery, gradient-echo T2-weighted
and 3D time-of-flight angiography of the circle of Willis. The
presence of acute ischemic lesions was defined by areas of high
signal intensity on DWI. All films and reports were retrospectively
reviewed without knowledge of baseline clinical and follow-up data.
All patients underwent a standardized etiologic work-up, includ-
ing standard blood tests, 12-lead ECG, prolonged 3-lead cardiac
monitoring, and Doppler ultrasound. All Doppler ultrasound scans
were performed with a Phillips ATL 5000 echograph, and standard-
ized diagnostic criteria were used for artery stenosis.16 Cervical
gadolinium-enhanced MR angiography was performed in 307 (90%)
routine MRI stroke protocol included the following sequences
acquired in the axial plane: spin-echo DWI (b=0 to 1000 s/mm²),
fast fluid-attenuated inversion recovery, gradient-echo T2-weighted
and 3D time-of-flight angiography of the circle of Willis. The
presence of acute ischemic lesions was defined by areas of high
signal intensity on DWI. All films and reports were retrospectively
reviewed without knowledge of baseline clinical and follow-up data.
All patients underwent a standardized etiologic work-up, includ-

Calvet et al DWI and Stroke Risk After TIA 3

Table 2. Clinical and MRI Characteristics of Patients With Stroke During Follow-Up

<table>
<thead>
<tr>
<th>Sex, Age, y</th>
<th>Cause of Initial TIA</th>
<th>Initial DWI Results</th>
<th>Time From TIA to Stroke</th>
<th>DWI at the Time of Ischemic Stroke</th>
</tr>
</thead>
<tbody>
<tr>
<td>M, 68</td>
<td>LAA (basilar artery)</td>
<td>Left superficial PCA infarct</td>
<td>10 h</td>
<td>Right superficial PCA infarct</td>
</tr>
<tr>
<td>M, 78</td>
<td>LAA (basilar artery)</td>
<td>Right superficial PCA infarct and right paramedian pontine infarct</td>
<td>6 d</td>
<td>Growth of the right paramedian pontine infarct</td>
</tr>
<tr>
<td>M, 77</td>
<td>Small-vessel disease</td>
<td>Small deep infarct (left internal capsule)</td>
<td>88 d</td>
<td>Small deep infarction (right internal capsule)</td>
</tr>
<tr>
<td>M, 62</td>
<td>LAA (basilar artery)</td>
<td>Both middle cerebellar artery infarcts</td>
<td>12 h</td>
<td>Extension of the right middle cerebellar artery infarct</td>
</tr>
<tr>
<td>F, 38</td>
<td>Isolated angitis</td>
<td>Left superficial ACA infarct</td>
<td>1 d</td>
<td>Small deep infarction (left corona radiate)</td>
</tr>
<tr>
<td>F, 75</td>
<td>Cardioembolism (AF)</td>
<td>Bilateral paramedian pontine infarction</td>
<td>9 h</td>
<td>Bilateral PCA infarcts</td>
</tr>
<tr>
<td>F, 77</td>
<td>LAA (carotid artery*)</td>
<td>Normal</td>
<td>7 d</td>
<td>Left superficial MCA infarct</td>
</tr>
<tr>
<td>M, 74</td>
<td>LAA (carotid artery*)</td>
<td>Left superficial MCA territory</td>
<td>8 d</td>
<td>Recurrent left superficial MCA territory</td>
</tr>
<tr>
<td>M, 92</td>
<td>Small-vessel disease</td>
<td>Small deep infarction (left corona radiate)</td>
<td>45 d</td>
<td>Small deep infarction (left internal capsule)</td>
</tr>
<tr>
<td>M, 76</td>
<td>Cardioembolism†</td>
<td>Multiple left MCA infarcts</td>
<td>12 d</td>
<td>Recurrent left MCA infarct</td>
</tr>
</tbody>
</table>

PCA indicates posterior cerebral artery; ACA, anterior cerebral artery; MCA, middle cerebral artery. All strokes that occurred during follow-up were confirmed by brain imaging.

*Carotid artery occlusion.
†Left atrium thrombus.

Results

Among the 343 patients enrolled in the study, 301 (88%) had a probable TIA (including 21 patients with amaurosis fugax) and 42 had a possible TIA. The median (IQR) time from TIA onset to admission to the stroke unit was 11.0 (5.0 to 22.5) hours. The characteristics of the patients are shown in Table 1. The carotid territory was involved in 198 (58%) patients; the vertebrobasilar territory in 85 (25%), and undetermined territory in 59 (17%). Sixty patients had LAA extracranial carotid artery, n = 38; basilar artery, n = 7; other extra- or intracranial arteries, n = 15) and 27 had AF (previously known, n = 19; diagnosed at admission, n = 5; diagnosed during hospitalization, n = 3). Among the 38 patients with LAA carotid artery TIA, endarterectomy was performed in 12 patients and angioplasty/stenting in 3 between 4 and 22 days after TIA, with no complications. The 23 remaining patients had either 50% to 69% nonsurgical carotid artery stenosis (n = 11) or carotid artery occlusion (n = 12). Regarding patients with AF, anticoagulation was started or adjusted at admission in 24 patients and started at the time of diagnosis in 3.

The median (IQR) time from TIA onset to DWI was 19.5 (8.0 to 28.3) hours. Acute ischemic lesions were demonstrated by DWI in 136 patients (40%). As shown in Table 1, patients with positive DWI findings were more likely to have unilateral weakness as a symptom of TIA (OR = 2.2; 95% CI, 1.3 to 3.7), TIA duration ≥60 minutes (OR = 2.6; 95% CI, 1.3 to 5.2), ABCD² > 5 (OR = 4.7; 95% CI, 2.0 to 11.0), LAA (OR = 1.8; 95% CI, 1.0 to 3.1), and AF (OR = 3.5; 95% CI, 1.5 to 8.1). Conversely, they were less likely to have a history of diabetes mellitus (OR = 0.4; 95% CI, 0.2 to 1.0), although the difference was not statistically significant (P = 0.06). There was an association between ABCD² score and AF (ABCD² 4 to 5, OR = 1.5; 95% CI, 0.6 to 4.0; ABCD² > 5, OR = 5.5; 95% CI, 1.8 to 17.1) but no significant association with LAA (ABCD² 4 to 5, OR = 1.4; 95% CI, 0.8 to 2.6; ABCD² > 5, OR = 1.7; 95% CI, 0.7 to 4.6).

During follow-up, 10 patients had ischemic stroke, 14 had recurrent TIA, and 2 died of cancer. Five ischemic strokes occurred within the first week after the qualifying event, of which 4 were within 48 hours. All strokes but 1 occurred in the same arterial territory as that of the TIA, and we did not identify any new etiologies (Table 2). The absolute risk of ischemic stroke was 1.2% (95% CI, 0.0% to 2.4%) at 48 hours, 1.5% (95% CI, 0.3% to 2.7%) at 7 days, and 2.9% (95% CI, 1.1% to 4.7%) at 3 months.

As shown in Table 3, the 5 strokes that occurred within 7 days and 9 of the 10 strokes that occurred within 3 months were in patients with baseline ABCD² scores ≥ 4. In patients with ABCD² scores > 5, all strokes occurred in patients with positive DWI findings (n = 21, 70%). LAA accounted for 2 of the 3 strokes that occurred within 7 days and 2 of the 4 strokes that occurred within 3 months. In patients with ABCD² scores of 4 or 5, all strokes occurred in those with positive DWI findings (n = 69, 40%). LAA accounted for none of the 2 strokes that occurred within 7 days and 2 of the 5 strokes that occurred within 3 months. One stroke that occurred within 3 months was in a patient with an ABCD² score < 4 and normal DWI results. That patient had LAA.

In univariate analysis, ABCD² score and positive DWI were significantly associated with an increased risk of stroke at 7 days and 3 months (Table 4). LAA was associated with an increased risk of stroke at 3 months. By contrast, there was no significant association with AF. At 3 months, in multivariate Cox analysis including positive DWI (hazard ratio [HR] = 8.7; 95% CI, 1.1 to 71.0), ABCD² score (score > 5, HR = 10.2; 95% CI, 1.1 to 93.4; score 4 to 5, HR = 3.3; 95% CI, 0.4 to 28.7) and LAA (HR = 3.4; 95% CI, 1.0 to 11.8) remained associated with an increased risk of stroke, although
the result was not statistically significant for an ABCD² score of 4 or 5.

The absolute risk of stroke in patients with ABCD² scores ≥4 was 2.5% (95% CI, 0.4% to 4.7%) at 7 days and 4.4% (95% CI, 1.7% to 7.1%) at 3 months. The absolute risk in patients with ABCD² scores ≥4 and positive DWI findings was 5.4% (95% CI, 0.9% to 9.9%) at 7 days and 9.7% (95% CI, 3.6% to 15.8%) at 3 months. The absolute risk in patients with an ABCD² score ≤4, positive DWI, and LAA was 9.3% (95% CI, 0% to 21.6%) at 7 days and 18.2% (95% CI, 2.1% to 34.1%) at 3 months. The 3-month prognostic value of ABCD² score, assessed by \( c \) statistics, was 0.75 (95% CI, 0.61 to 0.89). The \( c \) statistic rose to 0.84 (95% CI, 0.70 to 0.97) when DWI was added and to 0.87 (95% CI, 0.78 to 0.96) when LAA was added.

### Discussion

This study, which enrolled a large number of TIA patients who systematically underwent DWI-MRI and etiologic investigations, shows that in addition to the ABCD² score, positive DWI and, to a lesser extent, LAA are independently associated with an increased early risk of stroke after TIA.

The absolute risks of ischemic stroke after TIA were 1.5% at 7 days and 2.9% at 3 months, which is consistent with the rates reported in previous specialist stroke service–based studies. Interestingly, despite a low early risk of stroke in our population, the ABCD² score remained useful to predict the risk of stroke, with 90% (n=9) of strokes occurring in 59% of patients (n=201) with an ABCD² score ≥4. The \( c \) statistic (0.75) was similar to that found in the populations used to validate the ABCD² score.

We found that 40% of TIA patients had positive DWI results. This prevalence is in keeping with those observed in previous studies, ranging from 21% to 67%. A positive DWI result was associated with some components of the ABCD² score but remained significantly associated with an increased early risk of stroke after adjustment for ABCD² score. Interestingly, all strokes in patients with ABCD² scores ≥4 (n=201) occurred in those with DWI lesions (n=90, 45%; Table 2), which emphasizes the ability of DWI to further identify patients at high early risk of stroke after TIA.

The predictive value of ischemic lesions on computed tomography and DWI in TIA patients has been reported in previous studies, but none of them took both DWI and ABCD² score into account in prognostic models. In the largest of those studies, TIA patients with a positive DWI result were 10 times more likely to have a TIA or stroke than those with negative DWI results, a relative risk very close to that

### Table 3. Ischemic Strokes at 7 Days and 3 Months According to the ABCD² Score, Positive DWI Result, and LAA Etiology

<table>
<thead>
<tr>
<th>ABCD² Score, n (%)</th>
<th>DWI, n (%)</th>
<th>7 Days</th>
<th>3 Months</th>
<th>LAA, n (%)</th>
<th>7 Days</th>
<th>3 Months</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt;5, 30 (9)</td>
<td>Positive, 21 (70)</td>
<td>3</td>
<td>4</td>
<td>Yes, 7 (23)</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Negative, 9 (30)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>No, 23 (77)</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>4–5, 171 (50)</td>
<td>Positive, 69 (40)</td>
<td>2</td>
<td>5</td>
<td>Yes, 33 (19)</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Negative, 102 (60)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>No, 138 (81)</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>&lt;4, 138 (41)</td>
<td>Positive, 46 (33)</td>
<td>0</td>
<td>0</td>
<td>Yes, 20 (14)</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Negative, 93 (77)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>No, 118 (86)</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

### Table 4. Risk of Ischemic Stroke at 7 Days and 3 Months: Predictive Value of the ABCD² Score, DWI, LAA, and AF

<table>
<thead>
<tr>
<th>No. at Risk</th>
<th>No. of Strokes</th>
<th>7 Days</th>
<th>3 Months</th>
<th>7 Days</th>
<th>3 Months</th>
</tr>
</thead>
<tbody>
<tr>
<td>ABCD² score</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;4</td>
<td>139</td>
<td>0</td>
<td>0</td>
<td>&lt;0.0001</td>
<td>1</td>
</tr>
<tr>
<td>4–5</td>
<td>173</td>
<td>2</td>
<td>1.2 (0–2.8)</td>
<td>2.9 (0.4–5.5)</td>
<td>4.1 (0.5–34.8)</td>
</tr>
<tr>
<td>&gt;5</td>
<td>31</td>
<td>3</td>
<td>9.7 (0–20.1)</td>
<td>13.0 (1.0–25.0)</td>
<td>19.1 (2.1–171.1)</td>
</tr>
<tr>
<td>DWI</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Negative</td>
<td>203</td>
<td>0</td>
<td>0</td>
<td>0.006</td>
<td>1</td>
</tr>
<tr>
<td>Positive</td>
<td>136</td>
<td>5</td>
<td>3.7 (0.6–5.8)</td>
<td>6.6 (2.5–10.7)</td>
<td>13.7 (1.7–105.4)</td>
</tr>
<tr>
<td>LAA</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>283</td>
<td>3</td>
<td>1.1 (0–2.3)</td>
<td>1.8 (0.2–3.4)</td>
<td>1.4 (1.4–16.8)</td>
</tr>
<tr>
<td>Yes</td>
<td>60</td>
<td>2</td>
<td>3.4 (0–8.1)</td>
<td>8.4 (1.4–15.5)</td>
<td>9.7 (0.2–10.4)</td>
</tr>
<tr>
<td>AF</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>316</td>
<td>4</td>
<td>1.3 (0.1–2.5)</td>
<td>2.9 (1.1–4.7)</td>
<td>0.790</td>
</tr>
<tr>
<td>Yes</td>
<td>27</td>
<td>1</td>
<td>3.7 (0–10.8)</td>
<td>3.7 (0–10.8)</td>
<td>1.3 (0.2–10.4)</td>
</tr>
</tbody>
</table>

*Log-rank test.*
observed in our study. The fact that some TIA patients with normal DWI findings had nonischemic events such as epileptic, migrainous, or somatoform disease might explain our finding that DWI is a predictor of early stroke in TIA patients. However, this proportion is likely to be small in specialized settings and would probably not entirely explain the finding.

We found that LAA was also an independent predictor of early risk of stroke, which is consistent with a recent study showing that TIA patients with LAA had the highest risk of stroke at 3 months (20% vs 5.7% in other etiologies) and with previous studies conducted in population-based cohorts showing an increased risk of recurrent stroke in patients with LAA. In our population, LAA (n=60 patients, 17%) accounted for 50% of strokes occurring within 3 months. Patients with an ABCD² score ≥4, DWI lesions, and LAA had the highest early risk of stroke (18% at 3 months), although they represented only 6.5% (n=22) of our population. By contrast, we did not find a significant association between AF and risk of stroke. This finding could be explained by a beneficial effect of early anticoagulation in those patients. However, the lack of association needs to be interpreted cautiously because of the small number of patients with AF, which might partly result from the relatively young age of our population.

Several potential limitations need to be addressed. First, our study was conducted in a specialist stroke service, and although we did not apply any criteria for admission of TIA patients, selection biases may have occurred. The mean age of our population was lower than that of population-based patients, selection biases may have occurred. The mean age of our study was conducted in a specialist stroke service, and with AF, which might partly result from the relatively young age of our population.

Therefore, the low risks observed in our study are likely to be related to early management rather than to selection biases. Second, it is unlikely that the relative effects of DWI and LAA in predicting early risk of stroke would have been very different in a higher-risk population. Third, because we used a single-center study design, our findings would need to be confirmed in other settings to be generalized. However, all of the predictors that we found have been suggested in previous studies. Finally, the small number of outcome events in our study may have affected the accuracy and precision of regression coefficients in multivariate analysis. However, considering the strength of the associations in univariate analysis (HR >3 for each predictive factor) and the absence of effect of adjustment in multivariate models, we think that the predictors that we have identified are likely to be independent.

This study has several practical implications. First, the ABCD² score has the same predictive ability in a specialist stroke service, characterized by a low early risk of stroke, as in large population- or emergency department–based cohorts of patients with TIA. Second, MRI improves the prediction of stroke after TIA, which underlines the need to perform DWI-MR quickly after TIA. The predictive value of positive DWI findings may be of particular interest in patients with a moderate risk according to the ABCD² score to select the subset of patients at highest risk of stroke. Third, considering the high risk in patients with LAA and the increased benefit resulting from early endarterectomy in patients with symptomatic carotid stenosis, our study reinforces the need for an urgent arterial work-up to look for LAA in TIA patients. Although we were unable to prevent all recurrent events in patients with LAA, the rate of stroke recurrence was lower than that observed in previous studies in the subgroup of patients with TIA and/or minor stroke related to LAA. Taken together, all of these factors could help to organize emergency triage of TIA patients and identify those who require admission to specialist stroke services.

Disclosures

None.

References


DWI Lesions and TIA Etiology Improve the Prediction of Stroke After TIA
David Calvet, Emmanuel Touzé, Catherine Oppenheim, Guillaume Turc, Jean-François Meder and Jean-Louis Mas

Stroke. published online November 6, 2008;
Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2008 American Heart Association, Inc. All rights reserved.
Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/early/2008/11/06/STROKEAHA.108.515817.citation

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Stroke can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Stroke is online at:
http://stroke.ahajournals.org/subscriptions/