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JOURNAL OF THE AMERICAN HEART ASSOCIATION

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Stroke 2006;37;1710-1714; originally published online Jun 8, 2006;

DOI: 10.1161/01.STR.0000227195.46336.93

Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75214
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ISSN: 1524-4628

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Is the ABCD Score Useful for Risk Stratification of Patients With Acute Transient Ischemic Attack?

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Background and Purpose—A 6-point scoring system (ABCD) was described recently for stratifying risk after transient ischemic attack (TIA). This score incorporates age (A), blood pressure (B), clinical features (C), and duration (D) of TIA. A score <4 reportedly indicates minimal short-term stroke risk. We evaluated this scoring system in an independent population.

Methods—This was a prospective study of TIA patients (diagnosed by a neurologist using the classic <24-hour definition) hospitalized <48 hours from symptom onset. The primary outcome assessment consisted of dichotomization of patients into 2 groups. The high-risk group included patients with stroke or death within 90 days, $\geq 50\%$ stenosis in a relevant artery, or a cardioembolic source warranting anticoagulation. All others were classified as low risk. Findings on diffusion-weighted MRI (DWI) were also evaluated when performed and patients classified as DWI+ or DWI-.

Results—Over 3 years, 117 patients were enrolled. Median time from symptom onset to enrollment was 25.2 hours (interquartile range 19.8 to 30.2). Overall, 26 patients (22%) were classified as high risk, including 2 strokes, 2 deaths, 15 with $\geq 50\%$ stenosis, and 10 with cardioembolic source. The frequency of high-risk patients increased with ABCD score (0 to 1 13%; 2 8%; 3 17%; 4 27%; 5 26%; 6 30%; P for trend=0.11). ABCD scores in the 2 patients with stroke were 3 and 6. Of those who underwent MRI, 15 of 61 (25%) were DWI+, but this correlated poorly with ABCD score (0 to 1 17%; 2 10%; 3 36%; 4 24%; 5 13%; 6 60%; P for trend=0.24).

Conclusions—Although the ABCD score has some predictive value, patients with a score <4 still have a substantial probability of having a high-risk cause of cerebral ischemia or radiographic evidence of acute infarction despite transient symptoms. (*Stroke*. 2006;37:1710-1714.)

Key Words: prognosis ■ transient ischemic attack

There is an evolving consensus that patients experiencing a transient ischemic attack (TIA) are at high short-term risk of stroke. In a large observational study, 10% of patients with TIA experienced a stroke within 3 months, and half of these strokes occurred within 48 hours of the initial TIA.¹ Similar early stroke risk has been demonstrated in multiple other distinct populations.²⁻⁵ However, the optimal clinical approach to these patients remains poorly defined, and practice varies widely.⁶ American Heart Association guidelines note the absence of any prospective data on when, if ever, hospitalization is indicated for patients with TIA.⁷ Nonetheless, given the potentially devastating outcome of stroke after TIA, many authorities recommend emergent inpatient evaluation of most patients with recent TIA in an attempt to identify high-risk patients in need of specific preventative therapy.^{8,9} Like clinicians caring for patients with acute chest

pain, those caring for TIA patients “constantly maneuver between unnecessary admissions and premature discharges.”¹⁰

Concerns about the considerable expense associated with urgent diagnostic evaluation and treatment of patients with TIA suggest that strategies to identify those patients at highest risk would be useful. To this end, several studies have evaluated clinical predictors of short-term stroke risk.^{1,11} One such study suggested a simple 6-point risk score incorporating age (A), blood pressure (B), clinical features (C), and duration (D) of TIA (ABCD).¹² A score <4 was associated with minimal stroke risk within the 7 days after TIA. This ABCD score was derived from a population-based cohort of TIA patients and then validated by the same group in a separate population. We sought to evaluate this scoring system in an independent, prospectively observed population of patients with TIA.

Received April 11, 2006; accepted April 19, 2006.

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Stroke is available at <http://www.strokeaha.org>

DOI: 10.1161/01.STR.0000227195.46336.93

Materials and Methods

We are conducting a prospective observational study of patients with suspected TIA evaluated within 48 hours of symptom onset. TIA was defined as acute onset of focal cerebral or monocular symptoms lasting <24 hours and thought to be attributable to a vascular cause in the opinion of the neurologist evaluating the patient. All patients for whom there was sufficient clinical suspicion to justify diagnostic testing for a neurovascular cause were eligible for inclusion in this study. It is our practice to hospitalize all patients with acute TIA to expedite their evaluation and implement immediate treatment; accordingly, all patients included in this study were hospitalized.

On enrollment, a standardized case report form was completed collecting data on clinical features of the TIA, medical history, and examination findings. Subsequently, all relevant diagnostic testing was recorded, and an assessment of the presumed cause of the TIA was determined at hospital discharge and at the 90-day follow-up. Recurrent events, including stroke, recurrent TIA, myocardial infarction, or death, and therapeutic interventions were also determined at hospital discharge and at 90-day follow-up. Diagnosis of stroke was based on the standard clinical definition and required independent confirmation by 2 neurologists.¹³

The primary outcome measure was the dichotomization of subjects into high-risk and low-risk categories. The high-risk group included patients with stroke or death within 90 days, $\geq 50\%$ stenosis in a vessel referable to symptoms, or a cardioembolic source warranting anticoagulation. All other patients were classified as low risk. This dichotomization scheme was chosen to reflect the reality that observational studies occur on a background of variable diagnostic evaluation and therapeutic intervention that likely modify patient outcome. Ideally, risk stratification schemes should identify both patients at short-term risk of stroke and those who have a high-risk cause for which specific early therapeutic intervention (such as carotid endarterectomy or initiation of anticoagulant therapy) might be warranted. Diagnosis of a $\geq 50\%$ stenosis required demonstration on magnetic resonance angiography, computed tomography angiography, or catheter angiography interpreted by an independent neuroradiologist, except in the case of extracranial carotid artery disease, in which case duplex ultrasonography alone was considered an acceptable alternative. A cardioembolic source was considered present if long-term anticoagulation was felt to be warranted based on findings on echocardiography or ECG.

Findings on early MRI (when performed) were also evaluated, although MRI evaluation was not a requirement or focus of the study, and patient selection for MRI was not systematic but based on individual clinician practice and resource availability. Patients with acute infarction on diffusion-weighted imaging (DWI) were classified as DWI+ and those without infarction as DWI-. All suspected DWI+ lesions were confirmed by independent neuro-radiologist and neurologist review; disagreement was resolved through discussion.

Determination of ABCD risk score was performed in a manner identical to that reported by the originators of this score.¹² This 6-point score incorporates age (≥ 60 years=1 point), blood pressure (systolic blood pressure ≥ 140 mm Hg or diastolic blood pressure ≥ 90 mm Hg=1 point), clinical features (unilateral weakness=2 points; speech disturbance without weakness=1 point; other symptoms=0 points), and duration of symptoms (≥ 60 minutes=2 points; 10 to 59 minutes=1 point; <10 minutes=0 points). Although the actual ABCD score was computed retrospectively, the components of the score were prospectively collected as part of the described data set. Classification of patients into high- and low-risk groups was done blinded to ABCD score.

Informed consent was obtained from all subjects, and the protocol was approved by the Office of Human Research at our institution.

Statistical Analysis

Groups of patients were compared with χ^2 tests, Wilcoxon ranked sum tests, or logistic regression, as indicated. Odds ratios (ORs) and

95% CIs are reported for all comparisons. Linear regression was used to test for a trend in the relationship between ABCD score and risk. All tests were 2 sided. An association was considered significant if $P < 0.05$. All statistical analyses were performed using STATA version 8.0 (Stata Corporation).

Results

Over a 3-year period, 117 patients were enrolled. One patient, diagnosed with carotid occlusion at presentation and thus included in the high-risk group, could not be reached for 90-day follow-up. Characteristics of enrolled patients are shown in Table 1. Time from symptom onset to enrollment was a median of 25.2 hours (interquartile range 19.8 to 30.2). Clinical events occurred in 4 patients (2 strokes, 2 deaths); 3 of these patients also had a high-risk cause of TIA. A $\geq 50\%$ stenosis in a vessel referable to the patients' symptoms was found in 15 patients (14%), and a cardioembolic source warranting anticoagulation was found in 10 patients (9%). Identified cardioembolic sources were congestive heart failure with ejection fraction $\leq 30\%$ (n=4), atrial fibrillation (n=2), bioprosthetic aortic valve (n=1), akinetic apical segment postmyocardial infarction (n=1), patent foramen ovale (PFO) with atrial septal aneurysm (ASA) and Valsalva at symptom onset (n=1), and PFO+ASA with a familial hypercoagulable state (n=1). Both patients with PFO/ASA had DWI abnormalities. Overall, 26 patients (22%) were classified as high risk.

ABCD Score and Outcome

The utility of the ABCD scoring system for assessing risk is shown in Table 2. Increasing ABCD scores were marginally associated with increasing risk (P for trend=0.11) but were not associated with abnormalities on DWI (P for trend=0.24). ABCD scores in the 2 patients with stroke were

TABLE 1. Patient Characteristics

Age (mean [SD])	63.0 \pm 14.0
Systolic BP (mean [SD])	154 \pm 28
Diastolic BP (mean [SD])	83 \pm 16
Hypertension	76 (65%)
Clinical features	
Unilateral weakness	42 (36%)
Speech disturbance without weakness	28 (24%)
Other	47 (40%)
Duration of symptoms	
≥ 60 minutes	69 (59%)
10–59 minutes	27 (23%)
<10 minutes	21 (18%)
Diabetes	18 (15%)
Male sex	51 (44%)
CAD/myocardial infarction	16 (14%)
Peripheral vascular disease	7 (6%)
Current smoker	15 (13%)
Migraine	13 (11%)
Time onset to enrollment, hours (median [interquartile range])	25.2 (19.8–30.2)

CAD indicates coronary artery disease.

TABLE 2. ABCD Score and Risk Classification (*P* for trend=0.11) and MRI Results (*P* for trend=0.24)

ABCD Score	Patients	High Risk	% Risk (95% CI)	Early MRI	DWI+ (%)
<1	8	1	13 (0–53)	6	1 (17)
2	13	1	8 (0–36)	10	1 (10)
3	23	4	17 (5–39)	11	4 (36)
4	44	12	27 (15–43)	21	5 (24)
5	19	5	26 (9–51)	8	1 (13)
6	10	3	30 (7–65)	5	3 (60)
Total	117	26	22 (15–31)	61	15 (25)

3 and 6; these occurred 26 hours and 39 hours after TIA onset, respectively. Both patients who died had an ABCD score of 5. Table 3 shows the association between individual potential risk factors and patient outcome.

Notably, patients without weakness or speech disturbance still had significant probability of being high risk (15%) or DWI+ (8%).

Early MRI

Of the subgroup of patients who underwent early MRI, DWI abnormalities were found in 25% of patients; 60% of DWI+ patients were high risk compared with 8.7% of DWI– patients (unadjusted OR, 15.8; 95% CI, 3.7 to 67.5). After adjustment for ABCD score, the presence of a DWI+ lesion remained a strong predictor of high-risk status (OR, 14.9; 95% CI, 3.4 to 64.8). Table 3 shows the association between individual potential risk factors and MRI results. Notably, patients with unilateral weakness or speech disturbance were significantly more likely to be

DWI+ than those with other clinical symptoms. Compared with those who did not undergo early MRI, those who did were younger (58.5 versus 67.4 years; $P=0.0006$), less likely to have a history of hypertension (54% versus 77%; $P=0.01$), and less likely to present with systolic blood pressure ≥ 140 or diastolic blood pressure ≥ 90 (59% versus 84%; $P=0.003$). There was no difference in clinical symptoms, TIA duration, sex, other vascular risk factors, or composite “high-risk” outcome frequency between those who did and did not undergo early MRI.

Discussion

Although we found some predictive value to the ABCD risk score, its discriminatory ability is not optimal. Patients with a score of 0 to 3 still had a clinically significant probability (roughly in the 10% to 20% range) of having stroke within 90 days or a high-risk cause of cerebral ischemia warranting specific intervention, and a similar percentage had evidence of infarction on early MRI.

The size of our study was insufficient to fully evaluate the independent predictive value of the various factors used in deriving the ABCD score. However, the point estimates of the ORs did support the use of age and symptom duration and type as predictors of subsequent risk. Hypertension (defined either by history or measured blood pressure at presentation) was less clearly of value.

A previous study of 120 patients with TIA or minor stroke demonstrated that DWI was a useful tool for risk stratification, with a 90-day stroke rate of 10.8% in patients with a DWI lesion compared with 4.3% in those without a DWI lesion.¹⁴ Similarly, our study showed that MRI was a powerful tool for risk stratification, with 60% of those with a DWI

TABLE 3. Association Between Individual Risk Factors and Risk Classification and MRI Results

	High-Risk/ Total Patients	OR (95% CI)	<i>P</i>	DWI+/ Total Patients	OR (95% CI)	<i>P</i>
Risk Factors (present vs absent)						
Age >60 years	18/70 vs 8/47	1.69 (0.67–4.3)	0.27	7/28 vs 8/33	1.04 (0.32–3.35)	0.95
SBP >140 or DBP >90 mm Hg	17/83 vs 9/34	0.72 (0.28–1.81)	0.48	7/36 vs 8/25	0.51 (0.16–1.67)	0.27
Hypertension	18/76 vs 8/41	1.28 (0.50–3.26)	0.61	6/33 vs 9/28	0.47 (0.14–1.53)	0.21
Clinical features						
Unilateral weakness	12/42	2.29 (0.80–6.50)	0.12	8/22	6.29 (1.16–34.0)	0.03
Speech disturbance without weakness	7/28	1.90 (0.59–6.16)	0.28	5/15	5.5 (0.91–33.3)	0.06
Other	7/47	1.0		2/24	1.0	
Duration of symptoms						
>60 minutes	16/69	1.81 (0.47–6.95)	0.39	9/38	1.55 (0.29–8.43)	0.61
10–59 minutes	7/27	2.10 (0.47–9.36)	0.33	4/11	2.86 (0.41–20.1)	0.29
<10 minutes	3/21	1.0		2/12	1.0	
Diabetes	2/18 vs 24/99	0.39 (0.08–1.82)	0.23	1/9 vs 14/52	0.34 (0.04–2.96)	0.33
Male sex	11/51 vs 15/66	0.94 (0.39–2.26)	0.88	7/27 vs 8/34	1.14 (0.35–3.66)	0.83
CAD/myocardial infarction	6/16 vs 20/101	2.43 (0.79–7.48)	0.12	2/8 vs 13/53	1.03 (0.18–5.72)	0.98
Peripheral vascular disease	2/7 vs 24/110	1.43 (0.26–7.85)	0.68	0/4 vs 15/57	...	
Current smoker	3/15 vs 23/102	0.86 (0.22–3.30)	0.83	3/7 vs 12/54	2.60 (0.52–13.40)	0.25
Migraine	5/13 vs 19/101	2.70 (0.79–9.17)	0.11	4/9 vs 11/51	2.91 (0.67–12.71)	0.16

CAD indicates coronary artery disease.

lesion being high risk compared with 8.7% of those without a DWI lesion. Indeed, the predictive value of a DWI lesion, based on the identified OR, was substantially higher than any other predictors examined, even after adjustment for ABCD score. This suggests a role for MRI in the evaluation of TIA analogous to the use of cardiac enzymes in patients with chest pain; in both cases, confirmation of tissue injury serves as a powerful predictor of subsequent risk.

The rate of stroke after TIA (1.7% at 90 days) in this study was substantially lower than that in previous reports. There are likely several important reasons for this disparity.

First, the median time to enrollment in this study was just >24 hours. Given that data from previous studies suggest that the initial 48 hours after TIA are the highest-risk period, it is likely that some strokes occurring within the first 24 hours after TIA were not captured.

Second, given the lack of a consensus “gold standard” for TIA diagnosis, all studies of this type may experience misclassification bias both with respect to the initial TIA diagnosis and final outcome. This is particularly true in large databases but might be ameliorated by detailed assessments on a smaller scale. We used broad inclusion criteria to identify patients with suspected TIA in this study, which may have resulted in inclusion of relatively more patients with symptoms deriving from nonvascular causes who would be expected to be at low risk of subsequent stroke. Conversely, compared with other studies wherein emergency physicians made the initial diagnosis of TIA, it might have been expected that neurologists would more readily exclude the nonvascular causes and enrich this study with a higher-risk population. To address this limitation, some studies have used clinical criteria to create categories of definite/probable/possible TIA, but these criteria have not undergone rigorous assessment to verify their validity.^{12,15} Further, some have excluded patients in whom comprehensive diagnostic testing points to a diagnosis other than TIA. This strategy is inappropriate for a prospective study and is inconsistent with the likely use of a risk stratification scheme in clinical practice. In our study, all patients were assessed by a neurologist who felt sufficient clinical suspicion of a vascular cause of their symptoms to warrant hospital admission and diagnostic testing.

Finally, at our institution, we have an aggressive approach to the evaluation and treatment of patients with suspected TIA. We routinely hospitalize most patients with acute TIA, implement measures to optimize cerebral perfusion as done with stroke patients, start immediate antithrombotic therapy, use statin therapy in most patients, and perform urgent diagnostic testing to determine TIA cause. Patients with relevant extracranial carotid stenosis generally undergo revascularization during initial hospitalization, and patients with an appropriate cardioembolic source are started on anticoagulants before discharge. In contrast, in a large study of TIA prognosis, only 14% of patients were hospitalized, and only 30% were discussed with or seen by a neurologist.¹ Another study reported carotid ultrasonography performed in only 44% of patients and antithrombotic therapy prescribed in less than two thirds of patients after acute TIA.³

A major difference between our study and the initial report of the ABCD score is the outcome measure described. We used a composite measure of stroke, death, or a high-risk

cause of cerebral ischemia, as opposed to the 7-day stroke risk after TIA. We feel this is appropriate because a risk stratification scheme should ideally be able to identify those patients with a high-risk cause of cerebral ischemia warranting specific early therapy, such as carotid endarterectomy. Further, studies using only stroke risk as an outcome measure will be biased by the large variation in practice patterns that likely modifies event rates. The low rate of clinical events (2 strokes and 2 deaths) in our population precludes separate analysis of the predictive value of the ABCD score for clinical outcomes alone.

Other studies of the prognosis after acute TIA, although larger than ours, have important limitations. For instance, in the landmark report by Johnston et al, prospective identification of patients with TIA was based on the diagnosis recorded by emergency department physicians without explicit defining criteria. Clinical features were determined based on retrospective chart review, assessment of diagnostic evaluation and treatment was limited, and determination of outcome events was based on identification in computerized databases supplemented by medical record review, as opposed to explicit individual patient follow-up.^{1,11} In contrast, all patients in our cohort underwent detailed evaluation by a neurologist in the acute period and were followed prospectively for 90 days. Extensive clinical data, including diagnostic evaluation and treatment, were collected prospectively and systematically.

In summary, we found the ABCD score to have limited clinical utility in risk stratification of patients with acute TIA. We also found a much lower short-term risk of recurrent stroke than has been reported previously. On the other hand, we did identify high-risk causes of cerebral ischemia warranting specific intervention in a substantial proportion of patients.

Source of Funding

B.L.C. is supported by an American Heart Association fellow-to-faculty transition award.

Disclosures

None.

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