Can the ABCD\textsuperscript{2} Risk Score Predict Positive Diagnostic Testing for Emergency Department Patients Admitted for Transient Ischemic Attack?

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Background and Purpose—We sought to determine if the ABCD\textsuperscript{2} score, typically used for risk stratification, could predict having a positive diagnostic test in patients evaluated acutely for transient ischemic attack.

Methods—We performed a retrospective cohort study for patients admitted from our emergency department with a new diagnosis of transient ischemic attack confirmed by a neurologist. ABCD\textsuperscript{2} scores were calculated and patients with a score of \( \geq 4 \) were placed in the high-risk cohort. Tests evaluated included electrocardiogram, CT, MRI, MR angiography, carotid ultrasonography, and echocardiography. Specific test findings considered to signify positive diagnostic tests were created a priori.

Results—We identified 256 patients with transient ischemic attack for inclusion; 167 (61%) were female, the median age was 60 years (interquartile range, 50 to 72), and 162 (63%) patients had an ABCD\textsuperscript{2} score of \( \geq 4 \). Rates of completion of diagnostic testing were electrocardiogram, 270 (100%); CT, 224 (88%); MRI, 89 (35%); MR angiography, 68 (27%); carotid ultrasonography, 125 (49%); and echocardiography, 135 (53%). Univariate analysis found a significant association only with elevated ABCD\textsuperscript{2} score and carotid duplex testing (\( P < 0.05 \)).

Conclusion—An elevated ABCD\textsuperscript{2} score may help predict patients with severe carotid occlusive disease but does not predict positive outcome in other commonly ordered tests for patients being evaluated for transient ischemic attack. An elevated ABCD\textsuperscript{2} score cannot be recommended as a tool to guide diagnostic testing in patients presenting acutely with transient ischemic attack. (Stroke. 2009;40:3202-3205.)

Key Words: ABCD\textsuperscript{2} score ■ carotid stenosis ■ stroke ■ transient ischemic attack
with an ED diagnosis of TIA were screened. Patients who had a subsequent neurologist diagnosis of TIA were included for analysis. Patients who had an ED diagnosis of TIA and returned to baseline while in the ED but later in the hospitalization either developed a new focal neurological deficit or were diagnosed with cerebral infarction based on radiological imaging were included for analysis. Patients subsequently diagnosed with cerebral infarction were included because this diagnosis may be made on the basis of diagnostic testing. For example, patients with an initial diagnosis of TIA but with a positive diffusion-weighted imaging MRI or subacute infarct on CT may receive a discharge diagnosis of cerebral infarction. Patients presenting with new focal neurological deficits in the ED which were still present at the time of neurology evaluation were felt to be an acute cerebral infarction and not a TIA. These patients were excluded from final analysis. These patients were considered a misdiagnosis during the ED evaluation.

If the diagnosis of TIA was uncertain, the staff neurologist who evaluated that patient was queried for clarification. Patients who left the hospital against medical advice or were transferred to another hospital facility were excluded. Patients were enrolled only for their hospital stay and were not included if they were placed on anticoagulation. On discharge, an additional 3 (1.1%) patients were placed on anticoagulation. Of those, 146 (33%) were given an alternative diagnosis not related to TIA or cerebral vascular disease. Of the ED patients diagnosed with TIA, 58 (13%) were given a neurology diagnosis of acute cerebral infarction. Of the patients diagnosed with a neurology diagnosis of cerebral infarction, 24 (9%) had no new neurological deficit at the time of admission and developed a cerebral infarction at some point during hospitalization. This allowed 256 patients for analysis. The length of stay for the patients diagnosed with TIA and cerebral infarction was 1 day (interquartile range, 1 to 3 days) and 2.5 days (interquartile range, 1 to 5 days), respectively.

### Results

During the study period, 436 patients received an ED diagnosis of TIA. Of those, 146 (33%) were given an alternative diagnosis not related to TIA or cerebral vascular disease. Of the ED patients diagnosed with TIA, 58 (13%) were given a neurology diagnosis of acute cerebral infarction. Of the patients diagnosed with a neurology diagnosis of cerebral infarction, 24 (9%) had no new neurological deficit at the time of admission and developed a cerebral infarction at some point during hospitalization. This allowed 256 patients for analysis. The length of stay for the patients diagnosed with TIA and cerebral infarction was 1 day (interquartile range, 1 to 3 days) and 2.5 days (interquartile range, 1 to 5 days), respectively.

### Discussion

TIA is estimated to be diagnosed at a rate of 240 000 cases each year. The use of diagnostic testing for patients with TIA...
could predict a noncerebrovascular diagnosis in patients referred to a neurology clinic for TIA. They used a lower ABCD\textsuperscript{2} score (0 to 1) to define low risk. Their population included inpatients and outpatients and did not define the timing of the diagnostic brain imaging. Similar to our results, they found a high score was significantly associated with carotid vascular disease. Unlike our results, they found a high score was significantly associated with vascular lesions on brain imaging. The different rates of brain imaging among populations, 46.4% and 87.5% for the Quinn study and ours, respectively, suggest that they were more selective on who received brain imaging. The differences in study objectives, in rate of testing, and the different cutoffs used to determine low versus high risk make comparisons of the results of the 2 studies problematic.

It would be helpful to have an instrument that could direct diagnostic testing of patients with TIA. Currently, such a tool does not exist. Physicians will need to continue to use their judgment in directing diagnostic testing for these patients.

Our study was conducted at a single center so our findings may be different than other centers with significantly different populations. Not every patient received every test. This would not be practical and is not possible with the retrospective nature of the study. Our rates of testing were much higher than a published Canadian study on patients with TIA. Despite our higher rates of testing, it is possible that limited testing could introduce selection bias. We did not study how this diagnostic testing affected patient outcomes, which would require a prospective analysis. We did not use imaging criteria as part of the diagnostic criteria for TIA because it would confound the results. We did not evaluate outpatient workups for TIA. This would be difficult because very few patients with the acute presentation of TIA were discharged (4%).

**Summary**

We found a high ABCD\textsuperscript{2} score, of \( \geq 4 \), was associated with a significantly increased likelihood of carotid stenosis in patients presenting with TIA. If the score was used as a means of determining which patient gets testing, 15% of patients with significant carotid stenosis would have been missed. Although a high ABCD\textsuperscript{2} score is suggestive of significant carotid disease, we cannot recommend it as a screening tool for deciding in which patients to obtain carotid imaging. A high score was not associated with positive diagnostic testing for patients undergoing brain MRI, MR angiography, CT, echocardiography, or electrocardiography and the ABCD\textsuperscript{2} score should not be used to influence diagnostic testing for these modalities.

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**Disclosures**

None.
References


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