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Transient Ischemic Attack Definition, Diagnosis, and Risk Stratification

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Abstract

Transient ischemic attack (TIA) can convey a high imminent risk for the development of a major stroke and is therefore considered to be a medical emergency. Recent evidence indicates that TIA with imaging proof of brain infarction represents an extremely unstable condition with early risk of stroke that is as much as 20 times higher than the risk after TIA without tissue damage. The use of neuroimaging in TIA is therefore critical not only for diagnosis but also for accurate risk-stratification. In this article, we discuss recent advances in diagnostic imaging, categorizations, and risk stratification in TIA.

Keywords

Transient ischemic attack; definition; diffusion-weighted imaging; imaging; risk stratification; risk scores

Introduction

Each year, approximately 200 000 to 500 000 patients are diagnosed by a physician as having experienced a transient ischemic attack (TIA) in the United States [1,2]. An additional 300,000 to 700,000 individuals experience neurological symptoms suggestive of a TIA but never seek medical attention for their symptoms [1,2]. The clinical syndrome of TIA designates that the abnormality in the cardiovascular system leading to compromised blood flow to the brain is unstable and, if not properly treated, may also cause a debilitating ischemic stroke. The most remarkable characteristic of TIA is, perhaps, the temporal information it conveys that relates to the timing of an upcoming stroke. The excess risk after TIA can be *imminent*; the risk is highest within hours of TIA and declines steadily within the

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ensuing days, weeks, and months [3]; nearly half of strokes occurring within the next 30 days occur within the first 24 hours after TIA. It is estimated that 12% to 30% of patients report a history of TIA soon before their stroke and approximately a quarter of them occur during the hours before the stroke [4,5]. TIA constitutes a true medical emergency. Early initiation of preventive treatment for TIA (for instance within 24 hours instead of 20 days) can reduce the 90-day risk of stroke by approximately 80% [6]. While rapid and accurate diagnosis and urgent initiation of treatment are key to the management of TIA, given that nearly one half of population reports a brief episode of focal loss of brain function (either TIA or TIA-mimics) at one point in their lifetime [7], indiscriminate use of diagnostic and therapeutic resources may exhaust the health care system. It is critical to accurately identify patients who are most likely to benefit from further diagnostic investigations and rapid treatment. The purpose of this review is to provide an overview on the traditional TIA concept, introduce the new tissue-based definition, and discuss the potential utility of advanced diagnostic imaging and risk stratification in TIA.

Clinical diagnosis of TIA

According to the World Health Organization criteria proposed in 1988, transient ischemic attack (TIA) is defined as rapidly developed clinical signs of focal or global disturbance of cerebral function, lasting less than 24 hours, with no apparent non-vascular cause [8]. The National Institute of Neurological Disorders and Stroke Report published in 1990 defines TIA as brief episodes of focal loss of brain function of less than 24-hour duration, thought to be due to ischemia, that can usually be localized to that portion of the brain supplied by one vascular system [9]. Both of these traditional definitions are based on the assumption that rapid resolution of symptoms in TIA indicates an ischemic insult that is transient at the tissue level. Traditional definitions also assume that clinical judgment as to whether the pattern of signs and symptoms fit into a specific arterial territory is an accurate means of attributing transient symptoms to ischemia. Recent advances in neuroimaging have substantially changed our understanding of TIA. Today, we know that none of these assumptions are quite correct. TIA is not necessarily transient at the tissue level; approximately one third of traditionally-defined TIAs are associated with permanent ischemic tissue injury [10]. Likewise, focal symptoms localizable to an arterial territory do not a priori indicate an ischemic mechanism; several non-ischemic mechanisms including seizures, subdural hemorrhage, intracerebral hemorrhage, brain tumors, multiple sclerosis and, migraine can cause transient neurological symptoms confined to a vascular territory [11,12].

Transient events characterized by symptoms that are focal but not clearly attributable to a known cause are considered “atypical” or “uncertain” TIA [9,13]. Clinical features that are not considered to be typical of an ischemic attack include gradual built-up of symptoms (more than 5 minutes); march of symptoms from one body part to another (without passing the midline); progression of symptoms from one type to another; isolated disturbance of vision in both eyes characterized by the occurrence of positive phenomena; isolated sensory symptoms with remarkably focal distribution; isolated brainstem symptoms; and the occurrence of identical spells over a period of greater than 1 year [9,14]. Transient focal atypical spells are common, corresponding to approximately one out of every five emergency room visits due to transient neurological symptoms [13]. Approximately 10% of patients with atypical symptom characteristics reveal an acute brain lesion on diffusion-weighted MR images (DWI), suggesting that atypical spells do not exclusively represent a non-ischemic subset and may, therefore, not be as benign as previously considered [unpublished MGH data].

The window of opportunity for objective assessment of clinical deficit in TIA is very narrow. Approximately 60% of TIAs last less than 1 hour and two thirds of those lasting for less than 1 hour last for less than 10 minutes [15,16]. Only less than 10% of patients can be examined by physicians when they are fully symptomatic [17]. The inevitable use of historical information contributes to the variability in TIA diagnosis; reported rates of disagreement in diagnosis of TIA by history vary between 42% and 86% among neurologists [18,19]. This obviously hampers the clinical and research utility of the term TIA. Recent advances in neuroimaging have revolutionized the evaluation of TIA and provided an opportunity to overcome many of the shortcomings of the clinical-based approach by introducing an objective component to its definition. The following section summarizes the current state of knowledge on the utility of neuroimaging in attributing a transient spell to brain ischemia.

Brain imaging findings in TIA

The introduction of first computed tomography (CT) and later magnetic resonance imaging (MRI) to the evaluation of TIA have challenged the conventional view by demonstrating that clinically transient events are not necessarily transient at the tissue level. Approximately one third of patients with the clinical syndrome of TIA develop a clinically relevant brain infarct [10,20,21]. The first report of imaging proof of brain infarct in patients with TIA dates back to 1979 [22]. Perrone and co-workers reported small hypodense areas on CT consistent with infarction in 12 of the 35 TIA patients they studied. Four years later, in 1983, Waxmann and Toole coined the term “cerebral infarction with transient deficit” to describe transient episodes associated with CT evidence of brain infarction in a clinically relevant location [23]. Since then several researchers have studied TIA patients with CT and reported variable rates of brain infarcts ranging from 4% to 34% [22,24]. Subsequent studies with MRI have revealed somewhat higher rates of infarcts changing between 21% and 67% [25,26]. Although infarct rates on CT and MRI are similar, the diagnostic yield of these two imaging methods in identifying the “clinically-relevant” or “clinically-appropriate” infarct is quite different. The ability to distinguish acute infarcts from chronic lesions is critical to be able to tie a brain infarct to the transient clinical event. CT suffers from limited sensitivity in identifying “clinically-related infarcts” because infarcts observed in TIA are often very small, lack edema and mass effect, and demonstrate no or very subtle contrast enhancement. In a study of 149 patients with TIA, only 16% of infarcts suspected to be acute based on CT assessment had lesion characteristics consistent with acute infarct on DWI [27]. In another study of 57 patients with TIA, lesions designated as clinically-appropriate and acute on conventional MRI (FLAIR- and T2-weighted images) overlapped with an acute DWI lesion in only 48% of the patients, indicating that the so called “appropriate” lesion on conventional MRI was not acute and therefore not related to the transient clinical event in more than half of the patients [28] (Figure 1). DWI’s ability to differentiate between acute and chronic infarcts is clearly a strength in conditions associated with small infarcts such as TIA. Another strength of DWI is its high signal to background contrast. DWI can visualize acute small infarcts that are not visible on CT or conventional MRI in approximately one third of patients with TIA [28]. Current TIA guidelines recommend DWI as the preferred method of imaging in patients with TIA [29]. Nevertheless, MRI cannot be tolerated or is contraindicated in ~10% of patients and this limits its widespread applicability [17]. In addition, availability, feasibility, and affordability of MRI for use in emergency management of TIA are limited in many practices. According to a survey based on emergency department visits in the US between 1992 and 2001, MRI was the first line of imaging in only less than 5% of TIA patients [30]. A more recent survey conducted in 2008 indicates that 15% of Canadian neurologists routinely use MRI in their practice for managing TIA [31]. Despite a trend towards more frequent use of MRI in recent years, the vast majority of TIA patients are currently under-evaluated. Training of first-line physicians,

formation of hospital systems that enable rapid access to MRI, and development of newer imaging techniques with higher spatial resolution and lower cost are critical to enhance the diagnostic evaluation of TIA.

The most important characteristic of TIA-related infarcts on DWI is their strikingly small size [32] (Figures 1, 2, and 3). Infarcts as small as 0.07 ml can occur during a TIA. Ninety-six percent of all infarcts in TIA are smaller than 1 ml. We have previously coined the term “footprints of transient ischemia” to describe such punctate lesions on DWI that remain after complete resolution of TIA symptoms [28]. TIA-related infarcts can occur in any part of the brain including clinically important structures such as the brainstem, internal capsule, and motor cortex as well as less important or silent brain regions. The probability of infarct on DWI increases as the symptom duration increases, yet this relationship is not consistent across all studies [28,32,33]. We have observed brain infarcts occurring in patients with symptoms lasting for as short as 30 seconds as well as normal DWI despite symptoms lasting for several hours. Such imaging evidence suggests that the pathophysiology of TIA includes not only tissue damage but also a component of recovery; it suggests the possibility of interplay between a number of factors such as the size of the ischemic insult and the robustness of the affected neuronal circuitry, including perhaps the strength of the axonal connections as well as of the underlying neurovascular substrate. There are clearly a number of fruitful areas for further investigation into how the recovery from documentable tissue damage that is clearly present in TIA patients might be able to be extended to larger stroke insults.

While the presence of infarct on DWI indicates that the mechanism of transient clinical event is ischemic in origin, the opposite is not always true; DWI can be negative when in fact transient symptoms are due to ischemia. DWI has limited sensitivity for very small infarcts, particularly in the brainstem location [34]. In addition, a short lasting episode of ischemia that is not severe enough to cause permanent tissue injury may cause symptoms in the absence of a DWI lesion [34,35]. The combined use of DWI and perfusion-weighted MRI may improve the sensitivity for detection of tissue ischemia (Figure 4). Based on observational case studies, perfusion-weighted MRI appears to provide evidence consistent with ischemia in an additional 3% to 16% of TIA patients on top of DWI [36–38]. The diagnostic utility of perfusion-weighted MRI currently remains to be confirmed in unbiased large datasets. Limited spatial resolution of currently available perfusion-weighted MRI techniques is also of concern. Improvements in perfusion techniques in the future may overcome these concerns by enhancing the reliability of diagnoses for punctate regions of ischemia that typically occur in TIA [39].

Tissue-based definition of TIA

Advances in diagnostic imaging of TIA have led to the proposition of a new tissue-based definition [29]. This new definition classifies TIA as a transient episode of neurologic dysfunction caused by focal brain, spinal cord, or retinal ischemia, without evidence of acute infarction. All remaining neurological events, regardless of whether symptoms are transient or permanent, are called “ischemic stroke,” as long as they are associated with brain infarction. The tissue-based definition provides a safe passage from subjectivity (arbitrary duration criterion) to objectivity (evidence of brain injury) in defining a TIA. It has gained widespread acceptance in a relatively short time and has been endorsed by the American Heart Association, the American Stroke Council, and the American Academy of Neurology [29]. One aspect of the tissue-based definition that may be perceived as a drawback is the dependency of categorizations on the sensitivity and availability of neuroimaging. The punctate nature of ischemic lesions in TIA particularly makes this problem more acute. Imaging techniques with lower sensitivity, such as CT or conventional MRI, would be

expected to reveal a falsely higher prevalence of TIA as compared to diffusion-based techniques which have higher contrast to noise for acute ischemic lesions and can distinguish between nonspecific chronic white matter lesions and acute ischemic lesions.. The current pace of changes in diagnostic technology challenges the utility of the new definition in comparing studies from different time periods or making uniform categorizations in longitudinal studies covering a long period of time. In order to ensure the comparability of studies, it is critical to explicitly acknowledge the method of imaging applied in research studies using the tissue-based definition. Additionally, as the AHA/ASA recommendations also state, the term “acute neurovascular syndrome” may be used as an intermediate term to describe transient events when neuroimaging is not available or insensitive to detect the brain infarct despite a strong clinical suspicion for ischemic stroke.

In contrast to the traditional concept that considers TIA as a medical emergency and a sign of an upcoming stroke, TIA, when using the new tissue-based definition, should be considered as an extremely low-risk condition. According to a recent pooled analysis of 4574 patients with TIA from 12 centers, the 7-day risk of stroke after a “TIA with no infarction” was 0.4% [10]. This is a risk that is approximately 20 times smaller than the corresponding risk after “imaging-positive TIA” [10,17,40–42]. The early risk of stroke after imaging positive TIA is not only higher than the risk after imaging-normal TIA but also substantially greater (5 to 10 times) than the corresponding risk for recurrent stroke after an ischemic stroke [17,32,43,44]. Thus, imaging-positive TIA represents an extremely unstable syndrome. These short-term prognostic estimates suggest that it may be operationally more accurate to stratify acute neurovascular syndromes into three prognostically different categories: “transient ischemic attack with normal imaging or TIA”, “transient symptoms with infarction or TSI”, and “ischemic stroke” [32,45]. Interestingly, there is a clear parallel to the heart: acute cardiovascular syndromes are also classified into three major categories: stable angina pectoris, unstable angina pectoris or non ST elevation myocardial infarction, and ST elevation myocardial infarction [46]. By analogy to the cardiac classification, “ischemic stroke” corresponds to ST elevation myocardial infarction (evidence of major ischemia and infarction by ECG and biochemical markers), “TSI” to unstable angina pectoris or non-ST elevation myocardial infarction (evidence of minor ischemia or infarction by ECG or biochemical markers), and “TIA with no infarction” to stable angina pectoris or the chest pain syndrome (no ECG or biomarker evidence of ischemia or infarction) [32,45]. This syndromic classification not only takes into account the pathophysiology (the presence of tissue-injury) but also incorporates prognostic estimates into definitions, providing the ability to serve as a means to guide treatment strategies. The syndromic classification, however, just like the conventional definition, is time-based, necessitating the use of an arbitrary definition for transiency of symptoms.

Prediction of stroke risk after TIA

While the stroke risk after TIA is high (approximately 10% in 7 days), the majority of TIAs (approximately 90%) do not pose an imminent risk of stroke. Accurate differentiation of the 10% at risk from the remaining 90% is critical for optimal stroke prevention. Individualization of TIA management requires a risk-based approach where high-risk and low-risk individuals follow different paths [6,47]. High-risk patients would be expected to benefit from urgent referral to specialized stroke centers, timely identification of the underlying etiology, and institution of specific preventive treatments such as antiplatelet agents, anticoagulants, and carotid endarterectomy. Admission of high-risk patients to specialized stroke centers may also provide the opportunity to administer acute treatments in a timely manner in the event of a subsequent stroke. In contrast, elective evaluation would offer benefit to low-risk patients by avoiding their exposure to the risks, discomforts, and

costs of hospitalization. The high prevalence of TIA also makes identification of low-risk patients critical, particularly in settings where resources are limited.

Several prognostic scores for prediction of early stroke risk after TIA have been developed during the last decade (Table 1). There are briefly two types of prognostic scores: “clinical scores” and “clinical-plus” scores. The former is based on clinical predictors that are either baseline patient features or TIA characteristics and include the California score [48], ABCD score [49], and ABCD² score [50]. Clinical-plus scores provide risk estimates based on imaging and other diagnostic test findings in addition to clinical predictors and include the Clinical and Imaging-based Predictive Score (CIP) [17], ABCD² plus imaging score (ABCD²-I) [10], ABCD² plus dual TIA and imaging score (ABCD³-I) [21], and Recurrence Risk Estimator Score (RRE) [51]. Clinical and clinical-plus scores serve different purposes. Clinical scores are generally easy to apply as they require simple clinical information such as patient age, history of vascular risk factors, and symptom characteristics that are available to physicians in most clinical settings. They are particularly well-suited for use in primary care and population-based settings to identify high-risk patients who may benefit from referral for assessment on an emergency basis. Clinical-plus scores, on the other hand, require burdensome laboratory testing for risk stratification. Nevertheless, the additional information provided by diagnostic evaluations helps further segregate high-risk referrals based on clinical scores into different risk levels. Thus, the clinical-plus scores are more ideal for use in referral centers for individualizing acute TIA management.

Table 1 summarizes important aspects of validated clinical-based and clinical-plus scores. The ABCD² score is one of the most widely used clinical scores. The ABCD² is a unified clinical score generated from a combined dataset from two prior scores (California score [48] and ABCD score [49]). It provides estimates for 2-day, 7-day, and 90-day risk of stroke across 8 possible scores changing from 0 to 7. Higher scores correlate with the presence of vascular risk factors, longer symptom duration, and certain clinical symptoms such as weakness and speech abnormality. It has been suggested that the ABCD² score predicts stroke risk partly because of its ability to discriminate between a true TIA and a suspected TIA with eventual diagnosis of a nonvascular TIA-mimic [52–54]. The major strength of the ABCD² score is its simplicity. Its major weakness is the limited discriminative ability. The area under the receiver operating characteristics (ROC) curve or AUC for the ABCD² score for prediction of 7-day stroke risk is 0.66 (95% CI, 0.53–0.78) [10]. Based on the most widely used cut-off score of 4, the ABCD² score identifies high and low risk patients with 92% sensitivity and 33% specificity. In other words, nearly two thirds of patients who do not develop a subsequent stroke are falsely classified as high-risk. This obviously hampers its utility in prioritization of resources in referral centers.

Clinical-plus scores have been developed to enhance the discriminative ability of clinical scores (Table 1). The CIP model is a simple, web-based system that combines DWI findings obtained within the first 24 hours of TIA and dichotomized ABCD² score (<4 vs. ≥4) to predict the 7-day risk of subsequent stroke (available at <http://cip.martinos.org>) [17]. Risk estimates are provided for each of the 4 possible combinations of imaging and dichotomous ABCD² score: 0% for normal DWI-low ABCD² score, 2% for normal DWI-high ABCD² score, 5% for positive DWI-low ABCD² score, and 15% for positive DWI-high ABCD² score. The CIP model provides superior predictive performance as compared to the ABCD² score (AUC, 0.81 vs 0.66). It has been estimated that 25% of the patients graded low-risk by the ABCD² score are classified high-risk according to the CIP model. Likewise, 64% of the patients graded moderate- or high-risk per the ABCD² score are classified low-risk by the CIP model. The CIP score does not assess the predictive value of DWI in simultaneous context with individual components of the ABCD² score. Therefore, strictly speaking, it is not a unified score. The ABCD²-I score has been developed to integrate individual

components of the ABCD² score and brain imaging in a pooled dataset of 4574 patients (3206 with DWI and 1368 with CT) [10]. In this score, each component of the ABCD² score is weighted as originally described (Table 1). The imaging component is weighted by 3 points. This leads to an 11 point system in which the scores change from 0 to 10, with 10 indicating the highest risk strata. Regardless of the type of imaging (DWI or CT) used, addition of imaging evidence of infarction to the ABCD² score significantly improves the discriminative ability of predictions (AUC for 7-day risk predictions: 0.66 for ABCD², 0.78 for ABCD²-I with CT, 0.78 for ABCD²-I-DWI). Comparable performance of CT to DWI in the pooled analysis suggests that brain infarcts convey similar predictive value regardless of their age. Acute infarcts bear prognostic information by marking a spell truly ischemic. Chronic infarcts may also imply ischemia as the causative mechanism of transient symptoms by indicating a baseline ischemia prone-state. Caution, however, should be exercised before widespread application of CT for risk prediction in TIA. Only approximately a quarter of patients in the pooled dataset had CT as the initial line of imaging. The high variance in case-mix, setting (population-based, specialized neurovascular unit-based, emergency-based), timing of imaging, and the method of image assessment (blind assessment of one type of imaging to others) in published reports of CT-based datasets necessitates further studies in larger cohorts for conclusive evidence on the prognostic value of CT in TIA.

CIP and ABCD²-I scores do not take into account the etiologic mechanism of TIA in risk predictions. The etiologic TIA mechanism is known to be an important predictor of stroke risk [51]. The ABCD³-I score has been developed to partially fill this void [21]. The score adds two additional components to the ABCD²-I score: history of another TIA within 7 days prior to the index TIA and ipsilateral carotid stenosis causing at least 50% narrowing. The score was generated in a pooled dataset consisting of 2654 patients from 8 studies and validated in a separate dataset of 1232 patients. The ABCD³-I is a 14 point score system where scores change between 0 and 13, 0 being the lowest and 13 being the highest risk category. The overall AUC for prediction of stroke risk in 7 days is 0.92 in the derivation and 0.71 in the validation datasets. The score also provides good discrimination for prediction of 28 and 90 day risks. Although incorporation of TIA etiology (carotid stenosis) into the risk prediction after TIA is a strength, the ABCD³-I score does not take into account the whole spectrum of TIA etiologies (cardiac embolism, small artery occlusion, other uncommon causes, undetermined causes) that might provide additional prognostic information. Its relatively poor performance in the validation dataset is also of concern and suggests a need for further validation.

Since the early risk of stroke after a “TIA with no infarction on DWI” is negligibly low (0% to 0.4% [10,17], a clinically relevant question in the emergency setting is which patient with “TIA with infarction” is at imminent risk of developing a stroke. The recurrence risk estimator or *RRE score* has been developed to provide risk predictions in imaging-positive patients [51]. It was originally generated to predict early risk of recurrent stroke in patients with ischemic stroke. The RRE is a web-based (<http://www.nmr.mgh.harvard.edu/RRE/>), 7-point score composed of 2 clinical and 4 imaging predictors each weighted by one point. Clinical predictors are prior ischemic event within the preceding month of TSI and etiologic stroke mechanism as classified by the Causative Classification System [55]. Imaging predictors include infarct characteristics such as age, location, distribution, and number of infarcts on DWI (Table 1). The RRE provides risk estimates based on information available to the physician immediately after initial stroke evaluation in typical clinical practice. These include findings from clinical history, ECG, and baseline brain and vascular imaging. In a study of 257 patients with imaging-positive TIA, the RRE score applied within 24 hours of TIA provided an AUC of 0.85 for 7-day risk of stroke. The RRE score promises to further stratify high risk patients at risk of developing stroke, but additional validation studies of this score in external settings need to be performed.

Summary

Recent advances in neuroimaging have enhanced the understanding of TIA. Accumulated evidence indicates that clinically transient spells are not transient at the tissue level and leave infarct on the brain in nearly one third of patients. TIA-related acute infarcts are typically extremely small and often are not detected by CT and conventional MRI. Diffusion-weighted imaging (DWI) is currently the preferred method of imaging in TIA. Recognition of acute infarcts in patients with TIA has challenged the long standing conventional definition and led to the proposition of a new tissue-based definition. The tissue based definition reserves the term TIA for transient episodes without evidence of acute infarction. TIA, as defined by the tissue-based definition, is not a medical emergency anymore; it is a very low risk condition (early risk less than 0.4%). In contrast, "TIA with infarction" represents an extremely unstable condition with early risk of stroke that is as much as 20 times higher than the risk after TIA with normal imaging. The high prevalence of TIA necessitates accurate risk stratification to assure efficient use of resources. Several prognostic scores for prediction of early stroke risk after TIA with varying complexity and utility have been developed during the last 5 years. A staged approach to risk stratification in TIA based on availability of diagnostic resources should be advocated. Scores that are based on baseline patient features and TIA characteristics such as the California, ABCD score, and ABCD² scores are generally simple, can be widely applied, and are well-suited for use in primary care and population-based settings for selection of patients for referral to specialized centers. Clinical-plus scores such as the CIP, the ABCD²-I, the ABCD³-I, and the RRE scores, on the other hand, require additional laboratory testing, and are more difficult to apply, but provide risk estimates with higher accuracy as compared to clinical scores. Clinical-plus scores could be used in specialized stroke centers to individualize TIA management and prioritize hospital resources. The RRE score could allow a more refined and targeted approach by further stratifying imaging-positive TIAs (the high-risk subset by other clinical-plus scores) into different risk levels. Future clinical studies must address the utility and cost-benefit of individualized TIA management based on staged risk stratification.

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Figure 1 **Figure 1.**

An 86 year old woman with incoherent speech and left facial droop for 5 minutes. The FLAIR image shows several scattered and confluent periventricular and subcortical white matter hyperintense foci (the left image). Diffusion-weighted images (middle and right images) demonstrate two punctate foci of restricted diffusion representing acute infarcts involving the left precentral gyrus and posterior left parietal lobe (arrows). Notice that lesions observed on FLAIR images are not associated with restricted diffusion on diffusion-weighted images, indicating that they are not acute.

Figure 2 **Figure 2.**

A 53 year old man with a 2 minute episode of tingling and clumsiness of the left hand. The diffusion-weighted images shows a 7 mm focus of restricted diffusion involving the right precentral gyrus (arrow). Notice the punctate nature of the lesion.

Figure 3

Figure 3. A 65 year old man with a 5 minute episode of slurred speech on the day of admission. Diffusion-weighted images show multiple, mostly punctate foci of restricted diffusion (arrows) in both right and left hemispheres suggesting embolism from a proximal source.

Figure 4 **Figure 4.**

An 82 year old man with 2 distinct episodes of aphasia, one lasting for 30 minutes and the other for 15 minutes, within a two-hour period on the day of admission. There is no evidence of acute infarction on the diffusion-weighted image (the left image). The time to peak map (the right image), on the other hand, demonstrates signal changes consistent with hypoperfusion in the entire left MCA territory (arrows), marking ischemia as the cause of his transient episodes.

Table 1

Prognostic scores for prediction of stroke risk after TIA

Score Characteristics	California	ABCD	ABCD ²	CIP	ABCD ² -I	ABCD ³ -I	RRE
Components (points assigned)							
Age > 60 years	1	-	-	-	-	-	-
Age ≥60 years	1	1	1	1	1	1	-
Symptom duration > 10 min	-	-	-	-	-	-	-
Symptom duration > 10–59 min	-	1	1	1	1	1	-
Symptom duration ≥60 min	1	2	2	2	2	2	-
Focal weakness	1	2	2	2	2	2	-
Speech impairment	1	-	-	-	-	-	-
Speech impairment without weakness	-	1	1	1	1	1	-
Diabetes mellitus	-	-	1	1	1	1	-
SBP> 140 mmHg or DBP ≥90 mmHg	-	1	-	-	-	-	-
SBP ≥140 mmHg or DBP ≥90 mmHg	-	-	1	1	1	1	-
DWI evidence of acute infarction	-	-	-	1	3	2	1
TIA within the preceding 7 days	-	-	-	-	-	2	-
Ipsilateral carotid stenosis ≥50%	-	-	-	-	-	2	-
Etiologic TIA mechanism (CCS subtype)	-	-	-	-	-	-	1
Multiple infarcts of different ages on DWI	-	-	-	-	-	-	1
Simultaneous acute infarcts in different circulations	-	-	-	-	-	-	1
Multiple acute infarcts	-	-	-	-	-	-	1
Isolated cortical infarcts	-	-	-	-	-	-	1
TIA or stroke within the preceding 30 days	-	-	-	-	-	-	1
Total possible scores	5	6	7	-	10	13	7
Risk prediction interval							
	90 days	7 days	2,7, and 90 days	2 and 7 days	7 and 90 days	2,7,28,90 days	7,14,90 days

The CIP model provides risk estimates based on 4 possible combinations of DWI findings and dichotomized ABCD² score. No points are assigned in the CIP system. SBP, systolic blood pressure; DBP, diastolic blood pressure. Systolic and diastolic blood pressures are based on measurements on first assessment after TIA. RRE score assigns 1 point to TIA etiology for CCS subtypes of large artery atherosclerosis and other uncommon causes.