Stroke

AHA SCIENTIFIC STATEMENT

Diagnosis, Workup, Risk Reduction of Transient Ischemic Attack in the Emergency Department Setting: A Scientific Statement From the American Heart Association

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ABSTRACT: At least 240 000 individuals experience a transient ischemic attack each year in the United States. Transient ischemic attack is a strong predictor of subsequent stroke. The 90-day stroke risk after transient ischemic attack can be as high as 17.8%, with almost half occurring within 2 days of the index event. Diagnosing transient ischemic attack can also be challenging given the transitory nature of symptoms, often reassuring neurological examination at the time of evaluation, and lack of confirmatory testing. Limited resources, such as imaging availability and access to specialists, can further exacerbate this challenge. This scientific statement focuses on the correct clinical diagnosis, risk assessment, and management decisions of patients with suspected transient ischemic attack. Identification of high-risk patients can be achieved through use of comprehensive protocols incorporating acute phase imaging of both the brain and cerebral vasculature, thoughtful use of risk stratification scales, and ancillary testing with the ultimate goal of determining who can be safely discharged home from the emergency department versus admitted to the hospital. We discuss various methods for rapid yet comprehensive evaluations, keeping resource-limited sites in mind. In addition, we discuss strategies for secondary prevention of future cerebrovascular events using maximal medical therapy and patient education.

Key Words: AHA Scientific Statements ■ atrial fibrillation ■ cerebral angiography ■ diffusion magnetic resonance imaging ■ emergency service, hospital ■ ischemic attack, transient ■ stroke

Transient ischemic attack (TIA) is clinically described as an acute onset of focal neurological symptoms followed by complete resolution. TIA has been recognized as a risk factor for future stroke since the 1950s. Use of a time-based versus tissue-based definition for TIA has been debated since the 1980s and intensified with the widespread availability of magnetic resonance imaging (MRI).[1,2] In 2009, the American Heart Association redefined TIA using a tissue-based approach (ie, symptom resolution plus absence of infarction on brain imaging) rather than the time-based approach (ie, symptom resolution within 24 hours alone).[3] TIA is now widely understood to be an acute neurovascular syndrome attributable to a vascular territory that rapidly resolves, leaving no evidence of tissue infarction on diffusion-weighted imaging (DWI) MRI. A patient with resolved symptoms and MRI demonstrating infarct should be diagnosed with an ischemic stroke.

EPIDEMIOLOGY

The true incidence of TIA in the United States is difficult to determine given its transitory nature and the lack of standardized national surveillance systems. In addition, lack of symptom recognition by the public suggests that many TIAs go undetected. Estimates of 90-day stroke risk after TIA range from 10% to 18% and highlight the importance of rapid evaluation and initiation of secondary prevention strategies in the emergency department (ED).[4–6] In a large and nationally representative

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Table 1. Factors Suggestive of TIA Versus TIA Mimic

Factors	TIA	TIA mimic
Demographics	Older age	Younger patient without vascular risk factors
Medical history	Presence of vascular risk factors (hypertension, diabetes, coronary artery disease, peripheral artery disease, smoking, obesity, hyperlipidemia, atrial fibrillation, previous stroke, obstructive sleep apnea)	History of epilepsy, migraines, brain tumor
Symptomatology	Abrupt onset Maximal symptoms at onset Duration typically <60 min Preserved mentation Localizing/focal neurological symptoms corresponding to a vascular territory: dysarthria/aphasia, facial droop, hemiparesis, hemibody numbness Dizziness paired with cranial neuropathies, vision loss/diplopia, difficulty with coordination or gait/truncal ataxia, severe nausea/vomiting may suggest posterior circulation process Hypertensive on presentation Headache with ptosis and miosis might indicate dissection	Symptoms that spread/march from site of onset might suggest seizure Altered mentation Migraine headache Presence of signs or symptoms suggesting an alternative diagnosis (ie, positive visual phenomena, seizure-like activity, positional vertigo without localizing/focal symptoms)

This table is meant as a guide to approaching a patient with neurological symptoms and should not be the sole determinant of ultimate diagnosis. Patient-specific factors must also be considered.

TIA indicates transient ischemic attack.

population-based study, Kleindorfer et al[6] estimated an incidence of 240 000 TIAs in the United States in 2002.[6] Similar to cardiovascular disease and stroke, TIA incidence increases with age. Significant race-based disparities have been reported with Black Americans having a 1.4 times greater risk of TIA compared with White Americans,[6] although recent data on these disparities are lacking.

Population data have demonstrated an overall reduction in both TIA incidence and TIA admission rates over time.[7,8] These findings have been attributed to improved vascular risk reduction and stroke care,[8] changing trends in hospital admissions, and implementation of ED TIA protocols emphasizing short-term follow-up in dedicated TIA clinics.[9] Some evidence suggests that TIA incidence is decreasing more substantially in men than in women.[7]

CLINICAL EVALUATION

Acute onset of focal neurological symptoms followed by complete resolution is suggestive of a TIA. Therefore, patients for whom TIA is being considered must have a neurological examination consistent with their baseline status. Specific details of the history and presentation can help differentiate TIA from alternate diagnoses. Nonspecific symptoms or examination findings (eg, isolated dizziness, confusion/lethargy/encephalopathy), focal symptoms with other features (eg, headache, seizure), or new radiological findings (eg, mass lesion) may suggest an alternate diagnosis or "mimic" (Table 1). In cases of diagnostic uncertainty, conventional wisdom suggests performing a neurovascular workup for suspected TIA to reduce the risk of a recurrent event, ideally with expedited neurological consultation.

DIAGNOSTIC EVALUATION

Brain Imaging

In the ED, the role of acute phase imaging is to rule out alternative diagnoses, aid in risk stratification, and identify potentially symptomatic lesions. An initial noncontrast head computed tomography (NCCT) is part of many stroke/TIA protocols given its accessibility in the ED setting,[10] and is a useful initial test to evaluate for subacute ischemia, hemorrhage, or mass lesion. NCCT alone, however, has limited utility in patients whose symptoms have resolved.[3] Although its sensitivity to detect an acute infarct is low, it does have utility in ruling out TIA mimics.

Multimodal brain MRI is the preferred method to evaluate for acute ischemic infarct and ideally should be obtained within 24 hours of symptom onset, and in most centers will follow a NCCT.[11] Some centers might have rapid access to MRI in the ED. In cases where MRI with DWI can be obtained without delay, NCCT can be safely avoided in a stable patient with completely resolved symptoms.

TIAs typically last minutes with a linear increase in the likelihood of infarction with increasing symptom duration.[12] MRI with DWI demonstrates lesions in ≈40% of patients presenting with TIA symptoms, and DWI positivity is associated with a >6-fold increased risk of recurrent stroke at 1 year.[13] If a DWI-positive lesion is identified, a diagnosis of ischemic stroke is typically made, followed by hospital admission. The distribution of DWI lesions can help with identification of stroke pathogenesis (eg, a single lacune in a deep structure suggests small vessel disease; scattered emboli in multiple territories might point to cardioembolic mechanism such as atrial fibrillation [AF]; watershed distribution of lesions suggests large vessel disease) and guides further workup and secondary prevention strategies.

In the clinical scenario in which MRI cannot be obtained acutely to definitively distinguish TIA from stroke, it remains reasonable to make a clinical diagnosis of TIA in the ED on the basis of a negative NCCT and symptom resolution within 24 hours.[14] A potential next step would be hospital admission for MRI, comprehensive workup, and neurology consultation. Other options might include transferring patients to a facility with advanced imaging and vascular neurology expertise or arranging a timely (ideally <24 hours) outpatient MRI. Early MRI can identify higher-risk patients and avoids MRI degradation

 Stroke. 2023;54:e109–e121. DOI: 10.1161/STR.0000000000000418

Table 2. Advantages and Limitations of Noninvasive Techniques Available to Assess Patients With TIA[22,23]

Noninvasive techniques	Sensitivity for 50%–69% carotid stenosis, %	Specificity for 50%–69% carotid stenosis, %	Sensitivity for 70%–99% carotid stenosis, %	Specificity for 70%–99% carotid stenosis, %	Advantages	Limitations	Considerations
Doppler ultrasonography	36	91	89	84	Low cost No intravenous contrast	Operator variability Results might be affected by patient body habitus or vessel anatomy Only assesses cervical vessels Insensitive for dissection Not available in the emergency department, but potentially available in observation units	
Computed tomography angiography	67	79	87	95	Widely available as a STAT scan in emergency departments Can be performed simultaneously with noncontrast head computed tomography Can assess cervical and intracranial vessels Sensitive for cervical and intracranial atherosclerosis, dissection, other vasculopathies	Requires intravenous contrast Radiation exposure Limitations in interpretation in the setting of significant calcification	Should be ordered as computed tomography angiography head and neck
Magnetic resonance angiography	77	97	88	84	Can be performed simultaneously with magnetic resonance imaging of brain Can assess cervical and intracranial vessels Sensitive for dissection No radiation Can be performed without contrast	Contraindicated in some patients with implants and devices Costly compared with computed tomography and Doppler ultrasonography Longer time to schedule, perform, can rarely perform acutely May overestimate stenosis	Should be ordered as head and neck Contrast-enhanced magnetic resonance angiography allows for higher-quality images and less motion artifact Food and Drug Administration warning on gadolinium because of retention in the body and brain, especially in the case of repeated injections

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that might occur with delayed imaging capture.[15] A TIA evaluation without MRI might miss higher-risk patients who require closer monitoring.

Vascular Imaging

Evaluation of TIA requires adequate vascular imaging. Noninvasive imaging to screen for carotid stenosis (or vertebral artery stenosis for patients with posterior circulation symptoms) should be a routine component of the acute phase imaging for patients with TIAs.[11,16] A primary goal of vascular imaging is to identify patients with high-grade cervical carotid stenosis who might be candidates for revascularization.[11,16–18] Nearly half of patients with transient neurological symptoms and DWI lesions have stenosis or occlusion of either extracranial or intracranial large arteries.[19] Symptomatic intracranial stenosis is associated with higher risk of recurrent stroke, therefore many centers include intracranial in addition to cervical vascular imaging in the workup of patients with TIA.[20,21] Identification of symptomatic intracranial stenosis might sway toward admission for centers with low risk tolerance, and potentially caution clinicians against overly aggressive blood pressure reduction that might lead to the recurrence of symptoms.

Multiple noninvasive techniques are available to assess cervical carotid and intracranial vasculature from the ED (Table 2).[22,23] Computed tomography angiography (CTA) is the most widely accessible modality in EDs and can be obtained rapidly in conjunction with NCCT. CTA has a higher sensitivity and positive predictive value than magnetic resonance angiography (MRA) for detection of intracranial stenosis and occlusion, and is recommended over time-of-flight (without contrast) MRA.[24,25] Further, CTA is considered safe in patients with known chronic kidney disease and is not associated with significant risk of acute kidney injury.[26] Centers may still obtain a serum creatinine before obtaining CTA, however, to establish baseline renal function. For patients in whom the administration

of iodinated contrast is a concern, expedited MRI with MRA is an alternative. Time-of-flight MRA is of lesser quality and may overestimate the degree of cervical carotid stenosis compared with gadolinium-enhanced MRA.[27] Although time-of-flight is suitable for screening purposes, gadolinium-enhanced MRA neck is preferred in patients who can safely receive gadolinium contrast.

Duplex carotid ultrasound and transcranial Doppler are noncontrast options to evaluate cervical and intracranial vessels, respectively, but may not be available in the ED. Admission to a 24-hour observation or an inpatient unit are typically required to obtain these studies. Digital subtraction angiography, although considered the gold standard to assess cerebral vasculature, is invasive and should not be used to screen for carotid stenosis.

Laboratory and Cardiac Testing, Neurology Consultation

Point-of-care blood glucose testing should be performed for all patients with suspected TIA to rule out hypoglycemia, a known stroke mimic. A complete blood count, chemistry panel, hemoglobin A1c, and lipid profile can help identify potential risk factors. A nonfasting lipid profile is acceptable to identify hyperlipidemia as a risk factor.[28] In addition, patients >50 years of age with visual complaints may benefit from screening with erythrocyte sedimentation rate and C-reactive protein to assess for temporal arteritis. Other workups, including for infectious or toxic/metabolic processes, could be performed if such diagnoses are suspected.

Telemetry, troponin assays, and electrocardiography are warranted on all patients with TIA given the shared risk factors for myocardial infarction and ischemic stroke, and to screen for AF. Initial electrocardiography detects AF in up to 7% of patients with ischemic stroke and TIA, but longer cardiac monitoring results in higher detection rates, especially among patients with palpitations or structural heart disease.[29] Focal neurological symptoms in the absence of vasculopathy may suggest a cardioembolic process. Detection of AF is important to guide medical management and prevent future events.[30] In patients with TIA/stroke in whom a cardioembolic source is suspected, the American Heart Association/American Stroke Association recommends prolonged rhythm monitoring (30 days) within 6 months of the event.[17] This can be coordinated through cardiology, vascular neurology, primary care, or, if possible, the ED. The role of routine transthoracic echocardiogram for patients with TIA has not been well established but is often performed to identify a source of cardiac embolism and structural abnormalities associated with arrhythmia (eg, left atrial dilation).[16] If suspicion for a cardioembolic process is low and the patient is otherwise safe for discharge, a transthoracic echocardiogram could be arranged as an expedited outpatient study (ideally within 1 week).

When available, a neurology (preferably vascular neurology) consultation, either in person or through telemedicine, is a central part of the evaluation of patients with suspected TIA. The involvement of early neurology consultation has been associated with lower 90-day and 1-year mortality rates.[31] Multiple studies have demonstrated noninferiority of telemedicine neurology evaluations compared with traditional in-person evaluations in terms of patient and caregiver satisfaction.[32] If early neurology consultation is not possible, consider establishing a mechanism by which an appointment with a neurologist, ideally within 48 hours but no later than a week after TIA, can be scheduled given the high risk of stroke in the days after TIA.[33]

Considerations for Clinical Practice

- NCCT is insensitive to rule out small acute ischemic strokes but can help rule out TIA mimics.
- MRI with DWI is the preferred imaging modality to rule out acute infarct. If MRI with DWI can be obtained without delay for patients with TIA, NCCT can be safely avoided.[34]
- NCCT and CTA can be performed together to evaluate for hemorrhage and symptomatic stenosis.
- CTA is safe in patients with chronic kidney disease, and the risk of acute kidney injury related to contrast administration is low.
- Extended cardiac monitoring in selected patients is helpful to evaluate for potential sources of cardiac embolism.
- Patients benefit from early neurology consultation; preferably in the ED or rapid follow-up within 1-week after the TIA.

RISK STRATIFICATION

The use of validated risk scores as risk stratification tools for suspected TIA have gained wider acceptance.[4,5] An ideal scale for stroke risk prediction after TIA is one that is easy to calculate, has high predictive value, can categorize patients into clinically distinct risk groups, has been validated, and has broad generalizability. Several TIA risk stratification instruments are available to help predict the short-term stroke risk for individual patients and to guide disposition (Table 3). It is critical, however, for physicians to be aware of the limitations of the various TIA risk-scoring systems, including accuracy (ie, not incorporating high-risk features such as carotid stenosis, recurrent TIA, or AF) and external validity.

The most widely used risk stratification tool is Age, Blood Pressure, Clinical Features, Duration, and Diabetes ($ABCD^2$) score. It can be used to stratify patients into low-, moderate-, or high-risk groups on the basis of

 Stroke. 2023;54:e109–e121. DOI: 10.1161/STR.0000000000000418

Table 3. Comparison of ABCD², ABCD³, and ABCD³-I Scores

Components	ABCD² score	ABCD³ score	ABCD³-I score	ABCD³-I (d, c/i) score
Risk factor				
Age ≥60 y	1	1	1	1
Blood pressure ≥140/90 mm Hg	1	1	1	1
Diabetes	1	1	1	1
Clinical features				
Unilateral weakness	2	2	2	2
Language disturbance without weakness	1	1	1	1
Symptom duration, min				
≥60	2	2	2	2
10–59	1	1	1	1
<10	0	0	0	0
>10	N/A	N/A	0	0
Dual transient ischemic attack	N/A	2	2	2
Imaging				
Ipsilateral ≥50% stenosis of internal carotid artery	N/A	N/A	2	N/A
Ipsilateral ≥50% stenosis of internal carotid artery and major cerebral artery	N/A	N/A	N/A	2
Acute diffusion-weighted imaging hyperintensity	N/A	N/A	2	2
Total points	0–7	0–9	0–13	0–13
ABCD² score	2-d risk (%)	7-d risk (%)	90-d risk (%)	
Low (0–3)	1.0	1.2	3.1	
Moderate (4–5)	4.1	5.9	9.8	
High (6–7)	8.1	11.7	17.8	

ABCD² indicates age/blood pressure/clinical features of transient ischemic attack/duration/diabetes score; ABCD³, ABCD² plus Dual TIA; c, carotid stenosis; d, diffusion-weighted image; I, imaging; i, intracranial stenosis; and N/A, not applicable.

clinical features and medical history (Table 3).[5,32] ABCD² has also been used for clinical trial enrollment[35,36] and may be helpful in identifying patients with true TIA versus mimics.[37] A meta-analysis of 33 studies and just over 16 000 patients found that the ABCD² had high sensitivity but low specificity when stratifying high risk (score of 6–7) versus low risk (score of 0–3) patients; at 7 days after TIA, the high-risk group had a recurrent stroke risk of 7% versus 2.1% in the low-risk group.[38]

It is important, however, to note the limitations of the ABCD² score. First, it does not include symptoms that might suggest a "posterior circulation" process, such as dysmetria, ataxia, or homonymous hemianopia. It also does not account for TIA mechanism or the presence of ipsilateral large artery stenosis on imaging, and therefore should be part of a more comprehensive assessment.[38] Large artery disease (eg, unstable or severe carotid atherosclerosis, intracranial atherosclerosis, cervical artery dissection, or carotid web) or AF may further increase the risk of recurrent stroke or neurological worsening beyond that of a traditional risk-scoring score.[39]

Performing acute phase vessel imaging in the ED is important regardless of ABCD² score because it can guide immediate management.[10,40,41] For example, in patients with presumed symptomatic cervical or intracranial stenosis, more frequent neurological checks, stricter blood pressure parameters to avoid hypotension, dual antiplatelet therapy, and early consultation of surgical specialties could all be considered, potentially preventing early recurrence of symptoms.

TIA scores have evolved to incorporate radiographic data and a history of dual (≥2 episodes of TIA symptoms within the past 7 days) TIA to better identify high-risk patients. In the ABCD³ system, new or old infarction on MRI or CT imaging was independently associated with recurrent stroke risk and increased predictive value.[42] ABCD³-I incorporates dual TIA, MRI findings, and carotid stenosis, and ABCD³-I (d, c/i) incorporates intracranial stenosis.[41–43] Dual TIA and crescendo TIA[44] (recurrent, transient, events characterized by increasing duration, frequency, or severity) are high-risk clinical features that may warrant admission. Crescendo TIAs specifically may suggest the presence of ipsilateral carotid stenosis.

Considerations for Clinical Practice

- TIA risk stratification scales aid in the identification of high-risk patients and help guide disposition.

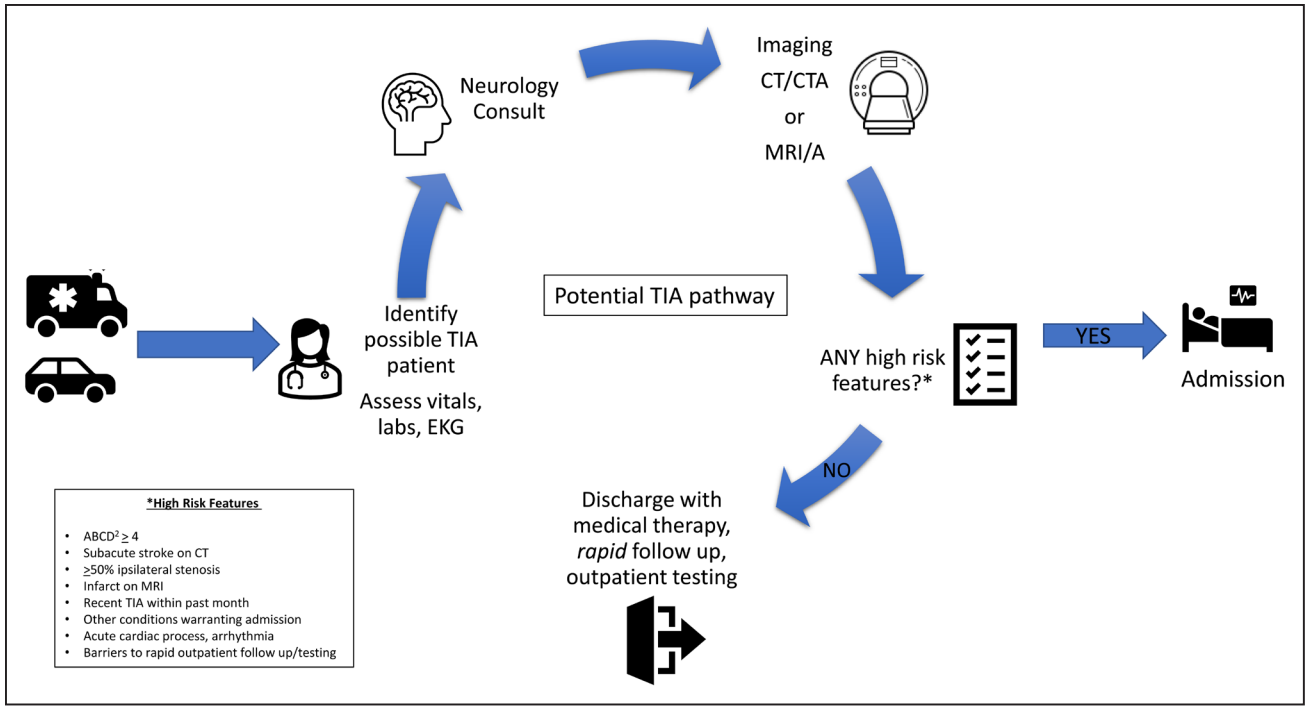

Figure. A potential TIA pathway that incorporates clinical evaluation, imaging, and risk stratification to guide disposition decisions.

Modifications are expected when rapid neurology consultation or MRI are not available. CT indicates computed tomography; CTA, computed tomography angiography; MRA, magnetic resonance angiography; MRI, magnetic resonance imaging; and TIA, transient ischemic attack.

- Given their limitations, TIA risk stratification scales should be part of a more comprehensive evaluation.
- Vessel imaging in the ED is warranted regardless of ABCD^2 score or likelihood of admission.

PATIENT DISPOSITION

Factors affecting the capability of EDs or medical centers to care for patients with suspected TIA include clinician experience, risk tolerance for neurovascular conditions, availability of CT and MRI, and access to neurovascular expertise. Acute stroke–ready hospitals, primary stroke centers, and comprehensive stroke centers have staff education, neuro-imaging resources, and acute neurovascular consultation availability. Where designated stroke centers are not present, protocols for the care of neurovascular patients may need to be modified to reflect available resources.

Expedited TIA management pathways and diagnostic protocols have been evaluated in nonrandomized trials in a variety of clinical environments and are supported by earlier clinical policy statements.[45] Implementation of these pathways has been associated with increased use of evidence-based strategies, reductions in treatment delays (ie, time to MRI), reduction in ED length of stay, reduction in admission rates, and cost savings without evidence of an increase in short-term stroke risk or mortality.[10,17,40] Several studies have reported that the cost of an inpatient TIA workup ranges from $1547 to as high as $10 876 for a 24-hour admission, compared with a range of $890 to $1600 for expedited outpatient workup.[10,46,47] Prolonged ED and hospital stays may therefore be more costly, add to the current national ED boarding crisis, and may not be justified for patients with TIA at low risk of subsequent stroke if reasonable alternatives are available.[48] Many of these investigations, however, were performed at certified stroke centers, so such protocols may be difficult to deploy in more resource-limited settings.

Successful ED TIA protocols include several components that may account for their effect: a clinical protocol enabling rapid identification and diagnosis, rapid access to diagnostic testing and advanced imaging, risk stratification criteria (eg, ABCD^2 score), access to neurovascular expertise, implementation of appropriate secondary prevention interventions, and access to a short-term follow-up clinic (Figure 1). To successfully facilitate an expedited TIA pathway, partnership across departments and service lines is necessary (ie, having an agreement with radiology to provide reserved MRI time slots for patients with TIA in the ED or in short-term observation areas, time-sensitive outpatient access to echocardiograms for suitable patients, and rapid access to neurology/TIA clinics). Different thresholds for ABCD^2 score have been reported in the TIA pathway literature to guide the decision to admit to hospital (eg, ABCD^2 >6, ABCD^2 >4) versus a 24-hour observation unit, or discharge to a TIA clinic for lower ABCD^2 scores.[10,40,41,49] The presence

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Table 4. Secondary Prevention Checklist for Patients With Suspected Transient Ischemic Attack[17]

Care component	ABCD^2 <4 (low risk)	ABCD^2 ≥4* (high risk)	ABCD^2 ≥6 and symptomatic ipsilateral intracranial stenosis*
Antiplatelet (should be started within 12–24 h of symptom onset)	Aspirin 50–325 mg daily OR Clopidogrel 75 mg daily OR Aspirin 25 mg/extended release dipyridamole 200 mg twice daily	Aspirin 81 mg plus clopidogrel 75 mg daily for 21–90 d† THEN transition to single therapy	Aspirin 81 mg plus clopidogrel 75 mg daily for 21–90 d OR Ticagrelor 180 mg load followed by 90 mg twice daily plus aspirin 75–100 mg daily for 30 d‡ THEN transition to single therapy
Antihypertensives (long-term goal blood pressure <130/80 mm Hg)	Angiotensin-converting enzyme inhibitor, angiotensin II receptor blockers, thiazide diuretic. Calcium channel blockers can be considered for patients who need additional options.		
Anticoagulation (for patients with atrial fibrillation or other indications§)	Apixaban, dabigatran, edoxaban, rivaroxaban, warfarin		
Lipid lowering (goal low-density lipoprotein cholesterol <70 mg/dL)	3-Hydroxy-3-methylglutaryl-coenzyme A reductase inhibitors (first line), and ezetimibe then PCSK9 (proprotein convertase subtilisin/kexin type 9) inhibitor (if needed for very-high-risk patients)		
Nutritionist consult	Encourage a low sodium or Mediterranean diet. For patients with diabetes, start conversation and consider referral to a nutrition specialist.		
Counsel regarding modification of lifestyle factors in an individualized, culturally sensitive manner	Smoking cessation Physical activity Alcohol moderation		
Follow-up appointment	Expedited transient ischemic attack/neurology and primary care clinics		

*When possible, strongly consider hospital admission.

†Based on CHANCE trial (Clopidogrel in High-Risk Patients With Acute Non-Disabling Cerebrovascular Events) and POINT trial (Platelet-Oriented Inhibition in New TIA and Minor Ischemic Stroke) protocols.[33,34]

‡Based on THALES trial (Acute Stroke or Transient Ischaemic Attack Treated With Ticagrelor and ASA for Prevention of Stroke and Death) protocol.[50]

§In patients with moderate to severe mitral stenosis or mechanical heart valve, warfarin is preferred.

ABCD^2 indicates age/blood pressure/clinical features of transient ischemic attack/duration/diabetes score; and TIA, transient ischemic attack.

of a high ABCD^2 score, infarction on MRI, or presumed symptomatic (>50%) extracranial or intracranial stenosis resulted in hospital admission in these studies. In the absence of a high-risk ABCD^2 score or presumed symptomatic stenosis, other high-risk features such as MRI evidence of infarction, severe hypertension, dual TIA, severe metabolic derangements, or abnormal electrocardiography findings, including a new diagnosis of AF, may also warrant hospital admission. Patients in underserved areas or who face barriers such as lack of transportation to follow-up appointments or timely completion of outpatient studies may also benefit from admission.[50] Admission to the hospital for high-risk patients may also allow for faster administration of acute stroke treatments in the case of a recurrent event.

Although completion of the evaluation may be faster in the ED and hospital in some institutions, other institutions have shown that dedicated outpatient clinics performing an expedited workup after the initial ED visit can also complete the evaluation safely and rapidly.[51] Discharging a patient with a TIA from the ED can be safe if proper protocols are in place before discharge, followed by outpatient completion of the workup and rapid follow-up in a dedicated TIA clinic, preferably within 48 hours. Lack of such a clinic might warrant either ED observation or admission, even for the low-risk patient. Ultimately, the appropriate disposition for patients with TIA is based on both clinical/patient characteristics, and on available resources locally, as well. The individual patient and institutional factors are critical in making the appropriate decision about where patients with TIA should be managed.

Considerations for Clinical Practice

- Presumed symptomatic (>50%) extracranial or intracranial stenosis warrant hospital admission.
- Acceptable disposition options include rapid ED TIA protocols with expedited referral to specialized cerebrovascular or TIA-specific clinics, admission to a 24-hour ED observation unit, or standard hospital admission.
- To determine disposition of patients with TIA, consider short-term stroke risk on the basis of presentation and vessel imaging, timeliness of a reliable workup, availability of rapid outpatient follow-up, and the patient's ability to return for rapid workup in a clinic setting.
- Institutional and regional factors should guide protocols for the decision-making about disposition for patients with TIA.

RISK REDUCTION AFTER TIA

Patients with TIA require prompt initiation of evidence-based interventions. The ABCD^2 score can help to guide appropriate treatment, with patients with higher scores likely benefiting from dual antiplatelet therapy.[35,36] We provide a brief overview of evidence-based medical therapy that might be considered (Table 4)[17,35,36,52]; however, this topic is covered more extensively in recent guidelines.[17]

Antithrombotics

Antithrombotic therapy is warranted in all patients with suspected TIA who have no known contraindications. Antiplatelet therapy is recommended for stroke prevention in patients who do not have an indication for anticoagulation.[17] Short-term dual antiplatelet therapy with aspirin plus either clopidogrel or ticagrelor has been shown to reduce risk of recurrent events in selected high-risk patients with TIA who present within 24 hours of symptom onset.[35,36,52,53] Consider consulting vascular neurology to determine the appropriateness and duration of dual antiplatelet therapy, to arrange follow-up, and to ensure transition back to monotherapy to reduce risk of future bleeding complications. For patients with TIA already on single-agent therapy, it is not well established whether increasing antiplatelet doses or switching to another agent confers any benefit.[17]

Therapeutic anticoagulation is effective at lowering the risk of stroke in patients with AF, and evidence has shown it can safely be prescribed from the ED.[30,54] For patients who have TIA with known AF not previously receiving anticoagulation, contacting the primary care physician or cardiologist to discuss risks versus benefits of anticoagulation, or other options where anticoagulation may be contraindicated, can be helpful to ensure appropriate secondary prevention. Even for patients with history of falls, evidence suggests that therapeutic anticoagulation remains the optimal therapy and outweighs bleeding risks in most cases.[55] MRI can also detect DWI hyperintensities and cortical microhemorrhages that might sway decision-making, therefore, waiting to start anticoagulation until MRI is complete is reasonable.

Statins

High-intensity statin therapy has been shown to reduce stroke recurrence in patients with ischemic stroke by 16% and has been well-established as a key aspect of secondary prevention after TIA or stroke.[17,56] Beyond reduction in low-density lipoprotein levels, statins are also beneficial for plaque stabilization, improvement in endothelial dysfunction, and inflammatory responses.[57]

Hypertension

Initiation of antihypertensive agents, preferably in partnership with outpatient clinicians, is critical for lowering risk of future events. Reducing blood pressure with an ultimate outpatient target of <130/80 mm Hg has demonstrated a reduction in recurrent stroke risk by 22%.[17,56]

Diabetes

Among patients with TIA or ischemic stroke, diabetes and hyperglycemia are associated with early neurological worsening and stroke recurrence.[58,59] A screening hemoglobin A1c is suggested in patients with suspected TIA. Severe hyperglycemia at the time of presentation may warrant hospital admission for glycemic management. First-line treatments for those with type 2 diabetes include metformin and lifestyle optimization.[17] Additional agents to consider include sodium-glucose linked transporter inhibitors and the glucagon-like peptide-1 receptor agonists, although these agents would typically be prescribed by primary care physicians or endocrinologists.[60]

Behavioral and Lifestyle Counseling

The American Heart Association/American Stroke Association recommends implementing behavior change interventions to improve stroke literacy, lifestyle, and medication adherence to reduce the risk of recurrent stroke.[17] Although lifestyle and behavioral interventions that improve vascular risk factor control can be difficult to implement after stroke/TIA,[61–63] several evidence-based interventions (eg, electronic decision support for primary care physicians) have successfully improved counseling or risk factor control.[17,64] Whereas extensive counseling may be considered out of the scope of acute care in the ED, incorporating behavioral health counseling as part of an ED observation pathway should be considered.

Increased physical activity is another cornerstone of stroke prevention.[17] Among patients with TIA or nondisabling stroke caused by intracranial stenosis in the medical therapy group of the SAMMPRIS trial (Stenting vs. Aggressive Medical Management for Preventing Recurrent Stroke in Intracranial Stenosis), greater physical activity was associated with a decreased likelihood of a recurrent stroke.[65] Various interventions have improved physical activity after stroke/TIA (eg, text-messaging,[66] cardiac rehabilitation)[67]; however, the effect is strongest among studies that implement interventions early after the event.[17,68] Likewise, discussions about a healthy diet, specifically Mediterranean-type or DASH (Dietary Approaches to Stop Hypertension) diets that have demonstrated reductions in stroke risk, can be implemented successfully.[69]

The American Heart Association/American Stroke Association recommends that patients with TIA receive counseling to limit excessive alcohol intake and referral for specialized services for patients with substance use disorders.[17] A systematic review of smoking-cessation interventions for patients with stroke and TIA found that the combined use of smoking-cessation medications with behavioral interventions may increase smoking cessation, but the quality of the evidence was low.[70]

Underserved and Rural Communities

Despite improved emphasis on stroke systems of care in recent years, disparities in treatment rates, mortality, and 30-day readmissions have remained constant or widened in rural populations.[71] Disparities in underresourced communities may stem from patient (mistrust,

literacy), clinician (unconscious bias, lack of underrepresented clinicians), system (access to and quality of care), or policy (health insurance, resource allocation) sources.[50] Factors that influence disparities may include a focus on individual risk factors without addressing social determinants of health such as access to transportation, fewer primary care physicians in both rural and high-risk areas, and high costs of care. Culturally tailored behavioral interventions should address both the individual's risk factors and their social determinants of health.[61]

Development of Telestroke networks provide rural and under-resourced communities greater access to vascular neurologists, which is especially relevant for time-sensitive treatments such as thrombolytic therapy for acute ischemic stroke in patients with ongoing symptoms. Telemedicine, similarly, might also be a useful way for physicians to facilitate rapid TIA diagnosis, identify underlying causes, implement individualized risk reduction plans, and provide access to stroke prevention clinics attended by vascular neurologists.

Education Required: Signs and Symptoms of Stroke

A patient's knowledge retention of stroke signs and symptoms or stroke risk factors is often poor after discharge with TIA or stroke.[72,73] Furthermore, patients with TIA or mild symptoms are less likely to activate emergency services on a subsequent visit.[74] It is therefore critical to develop consistent, innovative approaches to provide education for patients with TIA and their families in the ED. Multilingual education materials on stroke signs and symptoms, when to return for emergency care, time sensitivity of treatment options, and understanding the importance of secondary prevention treatments should be provided. Further studies are needed to develop effective approaches to stroke and TIA education in the ED setting.

Considerations for Clinical Practice

- Maximal medical therapy includes antithrombotic therapy paired with blood pressure, glucose and lipid control with targets adherent to established guidelines.[17]
- The ABCD^2 score can be used to guide antithrombotic regimens.
- Counseling paired with medical management may be helpful to patients with substance use disorders.
- Attention to social determinants of health can aid in addressing health disparities.

FOLLOW-UP APPOINTMENTS

Primary care providers are essential in the long-term management of cardiovascular risk factors and should be notified when a patient presents to the ED with TIA. Given the subsequent risk for recurrent stroke after TIA,[75] rapid follow-up with both a neurologist and primary care physician is warranted. One study found a significant reduction in 90-day stroke risk when time from TIA to stroke specialist review changed from 3 days to 1 day (10.3% to 2.1%, respectively).[74]

Telehealth or other electronic modalities of communication are potential options for clinicians to address risk factor management if resources are limited. Nurse-led, telephone-based interventions and other integrated mobile health technology programs have led to improved blood-pressure control and lipid management in rural populations by increasing the proportion of patients reached through these interventions.[76,77]

CONCLUSIONS

TIA is a strong predictor of future stroke and requires careful evaluation to properly identify high-risk patients. Several tools are available to aid in the evaluation of patients with suspected TIA, including risk stratification scales, acute phase imaging, and neurological consultation. Incorporating these steps into a clinical pathway can facilitate appropriate disposition of patients with TIA, such as hospital admission for those at highest risk while reducing unnecessary hospitalizations for lower-risk patients. Implementation of patient-specific secondary prevention strategies is critical to prevent future events. It is up to each center to use the resources available and create a pathway to ensure successful management and disposition of patients with TIA, with the ultimate goal of reducing the risk of future stroke.

ARTICLE INFORMATION

The American Heart Association makes every effort to avoid any actual or potential conflicts of interest that may arise as a result of an outside relationship or a personal, professional, or business interest of a member of the writing panel. Specifically, all members of the writing group are required to complete and submit a Disclosure Questionnaire showing all such relationships that might be perceived as real or potential conflicts of interest.

This statement was approved by the American Heart Association Science Advisory and Coordinating Committee on September 12, 2022, and the American Heart Association Executive Committee on October 26, 2022. A copy of the document is available at https://professional.heart.org/statements by using either "Search for Guidelines & Statements" or the "Browse by Topic" area. To purchase additional reprints, call 215-356-2721 or email Meredith.Edelman@wolterskluwer.com.

The American Heart Association requests that this document be cited as follows: Amin HP, Madsen TE, Bravata DM, Wira CR, Johnston SC, Ashcraft S, Burrus TM, Panagos PD, Wintermark M, Esenwa C; on behalf of the American Heart Association Emergency Neurovascular Care Committee of the Stroke Council and Council on Peripheral Vascular Disease. Diagnosis, workup, risk reduction of transient ischemic attack in the emergency department setting: a scientific statement from the American Heart Association. *Stroke.* 2023;54:e109–e121. doi: 10.1161/STR.0000000000000418

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Disclosures

Writing Group Disclosures

Writing group member	Employment	Research grant	Other research support	Speakers' bureau/ honoraria	Expert witness	Ownership interest	Consultant/ advisory board	Other
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This table represents the relationships of writing group members that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all members of the writing group are required to complete and submit. A relationship is considered to be "significant" if (a) the person receives $5000 or more during any 12-month period, or 5% or more of the person's gross income; or (b) the person owns 5% or more of the voting stock or share of the entity, or owns $5000 or more of the fair market value of the entity. A relationship is considered to be "modest" if it is less than "significant" under the preceding definition.

†Significant.

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This table represents the relationships of reviewers that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all reviewers are required to complete and submit. A relationship is considered to be "significant" if (a) the person receives $5000 or more during any 12-month period, or 5% or more of the person's gross income; or (b) the person owns 5% or more of the voting stock or share of the entity, or owns $5000 or more of the fair market value of the entity. A relationship is considered to be "modest" if it is less than "significant" under the preceding definition.

*Modest.

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