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FINANCIAL DISCLOSURE

Dr. Schneider (editor), Dr. Stapczynski (editor), Ms. Light (nurse planner), Dr. Nakayama (author), Dr. Andrews (peer reviewer), Ms. Mark (executive editor), Ms. Coplin (executive editor), and Ms. Hatcher (editorial group manager) report no financial relationships with companies related to the field of study covered by this CME activity.

RELIAS MEDIA

Transient Ischemic Attack

Introduction

Recent research on transient ischemic attacks (TIA) has changed how emergency medicine providers evaluate and manage this sometimes difficult diagnosis. This article provides readers with current information and relevant studies pertaining to TIAs. It will review recent changes in the way a TIA may be considered, as well as decision-making about workup, diagnosis, and disposition. Current trends also will be discussed. These changes have come about with the wider use of expanded computed tomography (CT) and magnetic resonance imaging (MRI) protocols, particularly diffusion-weighted imaging (DWI), in addition to rigorous studies with decision-making tools.

Epidemiology

According to the American Heart Association/American Stroke Association (AHA/ASA), about 240,000 U.S. adults experience a TIA each year. By definition, TIA represents a transient and spontaneously resolving set of symptoms; however, there is a significantly increased risk for future ischemic events in the next 24 hours and coming weeks.[1] Within one year after a TIA, 5.1% of patients experienced a recurrent cerebrovascular event and 6.2% experienced an ischemic cardiovascular event or death.[2] Other studies have noted a 5% risk of ischemic stroke within the first 24 hours and as high as 12% in the first 30 days following a TIA.[3] With 690,000 adults experiencing an ischemic stroke annually in the United States, prevention of future ischemic attacks is essential.[1] There are large financial implications from TIA and ischemic stroke, with an estimated direct medical cost that may increase from $28 billion to $96 billion between 2010 and 2030.[4]

Two definitions of TIA are used clinically. The modern definition incorporates tissue-based information to aid the clinician with the diagnosis, while the conventional definition is time-based. Under the time-based clinical model, TIA is defined as signs or symptoms of a focal neurological deficit lasting < 24 hours. Once symptoms last ≥ 24 hours or there are imaging findings consistent with the exam, it is considered a stroke.[6] Limitations on the availability of DWI for the tissue-based definition in many facilities make it difficult to implement this model consistently. In one review, researchers compared the long-term outcomes of patients with TIA tissue-based diagnosis vs. those diagnosed only on symptom duration. These authors noted that the early risk of stroke after a TIA was vastly different depending on the presence of brain infarction detected by DWI; the risk was between 0% and 0.4% without infarction and 5% to 10% with infarction.[7] Interestingly, in one study with the time-based decision, the prevalence of infarcts was 21-67% using DWI.[8]

EXECUTIVE SUMMARY

- Transient ischemic attack (TIA) is a clinical syndrome; the key features are the sudden onset of the loss of a neurologic function, such as weakness, balance, speech, or vision.
- If the initial computed tomography scan does not show any intracranial hemorrhage, most patients who have experienced a TIA should be started on antiplatelet therapy.
- Diffusion-weighted imaging by magnetic resonance imaging can detect a small infarct in a significant number of patients who have experienced a TIA.
- There is no consensus regarding the use of clinical decision tools that estimate the risk of subsequent stroke in patients with TIA.
- The development of an institution-specific protocol for the emergency department assessment and disposition of patients with TIA is recommended.

Etiology

There are several mechanisms that result in the clinical manifestation of a TIA. Each mechanism causes a temporary symptomatic disruption in blood flow to a specific part of the brain, brainstem, or retina, with eventual return of perfusion and function. The TOAST classification for stroke subtypes developed for a randomized, controlled trial of a low-molecular weight heparinoid for treatment of acute stroke (Trial of Org 10172 in Acute Stroke Treatment) also can be used to categorize TIAs: large-artery atherosclerosis, cardioembolism, small-vessel occlusion, stroke of other determined etiology, and stroke of undetermined etiology.[9,10,11]

Large-artery atherosclerosis is implicated when stenosis of more than 50% or occlusion of a major artery is found on imaging without possible cardiogenic embolism. Most lesions are associated with atherosclerosis. (*See Figure 1.*) Cardioembolic events are due to embolism of thrombi present in the heart. Definitive diagnosis requires identification of the thrombotic source (e.g., detection of left atrial thrombus on transesophageal echocardiogram). Other indications may be evident with previous TIA or stroke in multiple vascular territories or systemic embolization.

Small-artery occlusions (lacune) are considered with a small (< 1.5 cm) lesion on CT/MRI without a cardiac source or more than 50% stenosis of an ipsilateral artery. Risk factors for small vessel disease, such as smoking, hypertension, and diabetes, support this etiology.

Acute stroke of other determined etiology is a classification arising from rare cases of stroke, for example: hypercoagulable states, hematologic disorders, or vasculopathies. Similar to lacunar strokes, cardiac embolism and large-artery atherosclerosis should be ruled out.

The last classification is stroke of undetermined etiology. As the name implies, one specific cause cannot be determined. Patients may have no cause found on evaluation or have two or more likely causes.[11] Determining a stroke or TIA subtype is more important than classification alone, as the subtype determines future treatment (i.e., cardioembolic = anticoagulation).

With cerebrovascular thrombus formation, the native vessel is affected. For example, in patients with diabetes, vascular injury may arise from endothelial dysfunction, inflammation, or decreased pliability. Over time, atherosclerosis develops and causes ischemia via plaque rupture with occlusion or decreased flow with a watershed type of TIA.[5,9,10,12] Large vessel disease, such as in the carotid arteries, can cause cerebral hypoperfusion and lead to watershed ischemia with decreased flow as well as to artery embolization.[12]

Risk Factors

The AHA/ASA published guidelines on stroke risk reduction in 2014. Following these recommendations would allow for greatly reduced future risk of stroke and TIA. Risk factors from these guidelines and other sources are noted in Table 1. While it is beyond the scope of this topic to cover each risk factor, it is important to identify modifiable risk factors that a clinician may target for potential treatment.

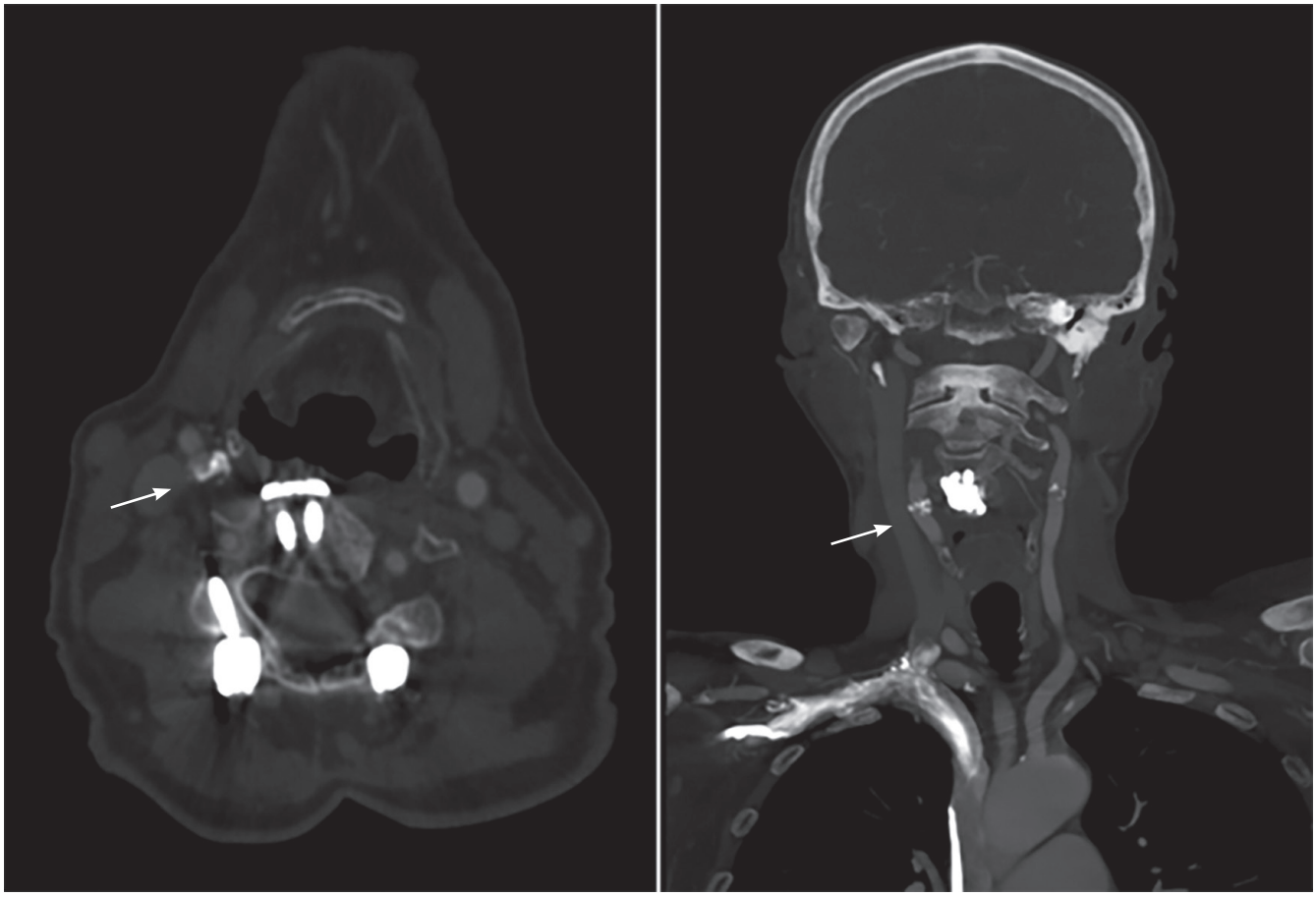
Clinical Presentation

TIAs present in many different forms, but often are similar to the presentation of patients with stroke. The most common presentations of a TIA include: sudden weakness or sensory loss in the face, arm, or leg, especially if it occurs on one side of the body; sudden difficulty talking or understanding; sudden difficulty with vision; or sudden trouble walking due to dizziness or loss of balance and coordination.[13,14] Patients typically present with sudden onset of symptom(s), since a gradual crescendo of symptoms is not typical. It may take up to 24 hours for the symptoms to resolve, with about 10 minutes of symptoms being the most common duration for TIA. Based on presenting symptoms, there should be a correlating neuroanatomical pattern.

Lacunar ischemia typically is associated with predominantly motor and sensory deficits. TIAs due to ischemia in the region of the middle cerebral artery may have weakness and/or decreased sensation on the face and extremities of the contralateral side, dysarthria, aphasia, neglect, gaze preference, or homonymous hemianopsia (loss of peripheral vision on the contralateral side of the TIA or stroke). TIAs affecting the posterior circulation cause symptoms from disruption of the brainstem, cerebellum, or occipital cortex. Loss of consciousness may occur as a result of ischemia to the brainstem. Other brainstem findings include dysarthria, diplopia, and cranial nerve deficits (ocular palsies), and also may include motor weakness (corticospinal tract).

Cerebellar TIAs often are difficult to detect, since the symptoms are

Figure 1. Right Internal Carotid Artery Stenosis (Arrows) of About 60% in a Patient Diagnosed With TIA



nonspecific and physical findings require specific testing. Nausea, vomiting, and dizziness often are presenting complaints that may be attributed to other causes when patients with TIA or stroke initially are evaluated. Nystagmus, ataxia, and dysmetria also may be present if a focused exam is completed. Dizziness alone may represent a stroke or TIA and may be expressed as vertigo or disequilibrium. Vertigo can be due to disorders of the balance glands in the inner ear (termed peripheral vertigo) or may arise from conditions in the brainstem (termed central vertigo); in about 80% of patients with vertigo, it is of peripheral origin. Traditionally, the differentiation of peripheral from central vertigo has been done using historical factors and associated symptoms. The accuracy of this approach was not great and techniques using provocative testing have proven better. The HINTS (Head-impulse-Nystagmus-Test-of-Skew) has a high sensitivity and specificity, 100% and 96%, respectively, for identifying central causes.[15]

The HINTS exam is performed with three different maneuvers: head impulse, nystagmus, and test of skew. In the head impulse test, the patient is asked to look at a fixed object. The examiner slowly turns the head to one side and then rapidly back to the middle position. A peripheral cause is likely when the patient is not able to maintain eye fixation on the visual target during the slow turning (occasionally seen) or after the rapid return to the neutral position (most sensitive finding). A central cause is suspected when the patient is able to maintain focus.

Transient nystagmus on lateral gaze may be seen in normal individuals, but sustained nystagmus is abnormal. If it is bidirectional, rotatory, or vertical, a central cause should be considered likely.

Lastly, the test of skew is performed by having the patient visually fix on a non-moving object. The examiner covers one eye at a time, then uncovers it, and covers the other eye. When the eye is uncovered and is not fixed on the target, it is considered abnormal.[15]

Peripheral vertigo is diagnosed when the provider finds agreement in all three portions of the HINTS exam: a positive head impulse test (not able to maintain eye fixation), unidirectional and horizontal nystagmus, and a negative skew test (able to maintain fixation on target). Mixed findings cannot be used to differentiate peripheral from central causes. Other findings common in patients with central vertigo may be helpful: headache or neck pain, truncal instability, or other neurological exam finding.

Patients with symptoms involving the posterior cerebral artery likely will present with vision loss. They also may have confusion, headache, memory

Table 1. Risk Factors for Transient Ischemic Attack or Stroke[1,5]

- Prior TIA or stroke
- Hypertension
- Diabetes mellitus
- Hypercholesterolemia
- Atrial fibrillation
- Congestive heart failure
- Smoking
- Patent foramen ovale
- Aortic atheroma
- Substance abuse
- Antiphospholipid antibodies
- Sickle cell disease
- Mechanical or other heart valve disease
- Hypercoagulation
- Sleep apnea
- Obesity
- Physical inactivity
- Pregnancy
- Advanced age
- Family history

impairments, problems with repetition, and sometimes motor weakness (from perforators of PCA and midbrain). Ipsilateral cranial findings and contralateral motor or sensory findings should alert providers to brainstem involvement. Headaches also may occur in TIAs. One study found 13.3% of patients diagnosed with a TIA had a new type of headache at the time of onset.[16]

Anterior cerebral artery (ACA) TIAs and strokes are uncommon and account for less than 2% of all strokes. Compared with other stroke territories, ACA strokes have a high percentage of crural monoplegia (weakness in one leg), which is the result of blood supplied to the neurons associated with control of the lower extremities. More than 80-90% of patients with ACA strokes present with this symptom.[17]

When examining patients with any symptoms concerning for stroke or TIA, providers should refer to the National Institutes of Health Stroke Scale (NIHSS) as well as conduct a focused exam on the area of concern. Providers should attempt to localize complaints to vascular territories described above when feasible. Providers also must pay careful attention to the history of what occurred and the complaints, because frequently they may be resolved by the time of presentation.[16]

Differential Diagnosis

There are many mimics to strokes and TIAs. Different findings on exam or history may suggest a mimic vs. a true TIA or stroke. Seizures are a common mimic and usually can be distinguished by certain findings: postictal state, loss of consciousness, seizure-like activity, or history of epilepsy. Somatoform and conversion disorder separate themselves with changes in the clinical picture and fluctuations of symptoms, non-neuroanatomic exam and history findings, or a history of mental illness. Migraine headaches usually have a history of similar events, possible aura, and headache. However, about 14% of patients with ischemic stroke had headaches that were described as different from their usual migraines if they had a history of headaches.[14] Toxic or metabolic abnormalities typically have nonfocal neurologic exams.[18]

Workup and Diagnosis

A full neurologic and cardiac examination should be performed on suspected TIA patients. Vital signs, point-of-care blood glucose, CT scan of the head, and electrocardiogram (ECG) should be obtained. A noncontrast CT scan of the head may differentiate the patient from a subdural hematoma, tumor, and intracerebral hemorrhage. Hypoglycemia also may mimic a TIA and can be identified quickly with a finger stick glucose test. ECG is an essential test, as it can easily identify atrial fibrillation and other rhythm abnormalities. Other tests should include a complete blood count, coagulation studies (prothrombin time/INR and activated partial thromboplastin time), markers for cardiac ischemia, and serum electrolytes/renal function tests.[18,19,20]

Additional imaging may include CT angiography (CTA) of the head and neck, ultrasound of the carotids, as well as MRI. CTA of the head and neck can be completed along with a head CT, is quick to perform, and delivers high-quality vascular imaging. CTA may be superior to carotid ultrasound in that the extracranial and intracranial vessels can be evaluated. Vascular imaging may help to identify significant stenosis from large-artery atherosclerosis or even chronic occlusions in some patients. Having 50% or greater stenosis on diagnostic angiogram or occlusion in a congruent vessel puts the patient at high risk for recurrent stroke. (*See Figure 1.*) The North American Symptomatic Carotid Endarterectomy Trial (NASCET) demonstrated that patients with stroke or TIA benefitted from carotid endarterectomy (CEA) compared with medical management when carotid artery stenosis was greater than 70%.[21] The current AHA/ASA guidelines recommend consideration of CEA in patients with stenosis greater than 50%.

DWI is able to show abnormal diffusion of water molecules in the setting of brain ischemia.[19] (*See Figure 2.*) As noted earlier, a high percentage of patients with TIAs diagnosed using the time-based approach have infarcts identified on DWI.[7] An important feature of these TIA-related infarcts seen on DWI is their small size (more than 90% are less than 1 mL), which explains their low detection rate with noncontrast CT. As previously stated, MRI is not rapidly available at all institutions and, thus, the clinical and historical exam should determine further workup. When diagnosis is questionable, MRI may help to confirm the diagnosis of a stroke mimic or to rule out cerebral ischemia.[22]

Treatment

Prompt use of secondary stroke intervention has been shown to reduce the risk of future strokes by as much as 80%. Revascularization should be considered with symptomatic carotid artery stenosis in patients who have had a cerebrovascular event. Endarterectomy for symptomatic carotid stenosis of 50% or greater on diagnostic angiogram (or 70% or greater on noninvasive imaging

Figure 2. MRI DWI of an Acute Infarct of the Left Corona Radiata (Arrow)

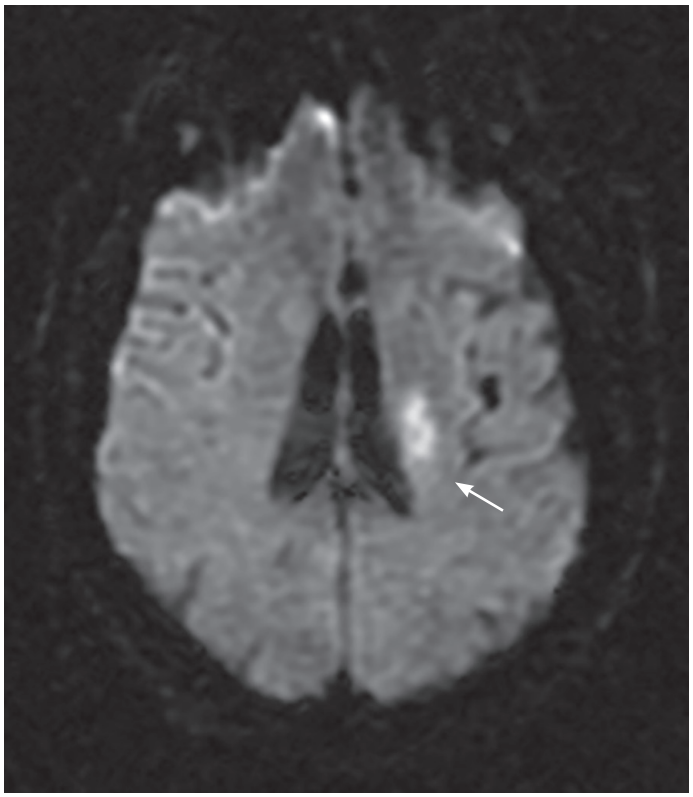


Table 2. Summary of Antiplatelet Treatment[19,22]

- For noncardioembolic TIA, use of antiplatelet agents rather than oral anticoagulation is recommended.
- Aspirin 81-325 mg/day monotherapy or combination aspirin 25 mg/extended-release dipyridamole 200 mg twice daily for initial therapy
- Clopidogrel 75 mg alone is reasonable in place of aspirin or aspirin/dipyridamole when aspirin is contraindicated
- May consider clopidogrel for 90 days plus aspirin within 24 hours of a TIA
- No significant evidence to support increasing dose of aspirin for those already on aspirin therapy. May consider adding another agent; however, no agent(s) have been studied sufficiently

with CTA, MR angiogram, or carotid ultrasound) has been shown to be an effective form of prevention if done within the first two weeks of an event. Therefore, diagnosing carotid stenosis is important in TIA because it causes 50% of early recurrences yet only makes up about 10% of all TIAs. When a clear cardioembolic cause is found (i.e., atrial fibrillation), anticoagulation should be initiated. With all other causes, treatment is centered around initiation of antiplatelet agents, statins, and risk-modification strategies. Common risk modification involves treatment of hypertension, smoking cessation, or weight loss.[19]

All patients with TIAs should be on antiplatelet agents unless anticoagulation is required. Aspirin 81-325 mg daily usually is first-line therapy. (*See Table 2.*) Intracranial atherosclerosis should be treated with medical management. Stenting of intracranial arterial stenosis was not found to reduce the occurrence of stroke and is not recommended when compared to medical management in the Stenting vs. Aggressive Medical Management for Preventing Recurrent Stroke in Intracranial Stenosis trial.[22] Specifically for significant stenosis (70-99%), medical management included 325 mg aspirin and 75 mg clopidogrel for 90 days.[19,23]

The benefits of dual antiplatelet therapy were noted in the Clopidogrel in High-risk Patients with Acute Non-disabling Cerebrovascular Events (CHANCE) trial that compared the effects of clopidogrel plus aspirin vs. aspirin alone. Patients were given a loading dose with 300 mg of clopidogrel followed by 75 mg/d for 90 days plus aspirin at a dose of 75 mg/d. The aspirin-alone group received 75 mg/d plus placebo for 90 days. Recurrent stroke occurred in 8.3% of the clopidogrel-aspirin arm compared with 11.8% in the aspirin-only group ($P < 0.001$). The risk of bleeding was not significantly different between the two groups.[24] Limitations included the population studied was Chinese and included a high proportion of males, and the percentage of patients treated with antihypertensive(s) and lipid-lowering medication was lower than normally seen in North America.[19]

Disposition

A variety of risk-stratification tools have been developed to determine the risk of subsequent stroke after a TIA. (*See Table 3.*) The two most commonly used are the ABCD and the ABCD2 scores, developed as clinical decision rules for assessing the risk of stroke patients in a nonspecialist setting. The addition of diabetes to the original ABCD score created the ABCD2 score:

- Age ≥ 60 years (1 point);
- Blood pressure ≥ 140/90 mmHg on first evaluation (1 point);
- Clinical symptoms of focal weakness (2 points) or speech impairment without weakness (1 point);
- Duration ≥ 60 minutes (2 points) or 10-59 minutes (1 point);
- History of diabetes (1 point).

The initial studies of the ABCD2 determined that a score ≥ 4 was deemed higher risk with a two- to seven-day chance of stroke occurring in 4.1-11.7%

Table 3. Risk Stratification Tools for Occurrence of Stroke After TIA

Tools (points assigned)	ABCD	ABCD2	CIP	ABCD2-I	ABCD3-I
Clinical Features					
Age ≥ 60 years	1	1	1	1	1
Symptom duration 10-59 minutes	1	1	1	1	1
Symptom duration ≥ 60 minutes	2	2	2	2	2
Focal weakness	2	2	2	2	2
Speech impairment without weakness	1	1	1	1	1
Diabetes mellitus		1	1	1	1
Systolic blood pressure > 140 mmHg or Diastolic blood pressure > 90 mmHg		1	1	1	1
TIA within preceding seven days					2
Imaging Features					
Acute infarction on diffusion-weighted imaging			1	3	2
Ipsilateral carotid stenosis ≥ 50%					2
Total possible score	6	7	Four categories: ABCD2 < 4 or ≥ 4 and/or infarct seen on DWI	10	13
Risk prediction interval	Seven days	2, 7, and 90 days	2 and 7 days	7 and 90 days	2, 7, 28, and 90 days

Abbreviations: ABCD = Age, Blood pressure, Clinical features, Duration score; ABCD2 = Age, Blood pressure, Clinical features, Duration, Diabetes score; CIP = Clinical and Imaging-based Predictive score; ABCD2-I = ABCD2 plus diffusion-weighted imaging; ABCD3-I – ABCD2 plus TIA within seven days of index event and diffusion-weighted imaging

of patients and < 4 was low risk with a two- to seven-day chance of stroke between 1.0-1.2%.

Newer studies show there is large proportion of patients with low risk who would benefit from more prudent evaluation and treatment because of high rates of future stroke in seven days. Subsequent studies reported recurrent strokes in seven days were from 25% of patients with scores in the low or intermediate risk scores ≤ 4.[25,26] Furthermore, in 2016, the American College of Emergency Physicians made a moderate recommendation against the use of the ABCD2 score to identify patients who can be discharged safely from the emergency department.[27] For example, a significant number of high-risk patients with symptomatic carotid stenosis may be missed.

Supplementing the ABCD2 assessment with DWI is one way to improve the predictive power seen with the Clinical and Imaging-based Predictive score (CIP) and ABCD2 plus diffusion-weighted imaging (ABCD2-I) tools.[27] Adding two additional criteria — TIA within seven days preceding the index visit and carotid stenosis — creates the ABCD3-I tool:[28] (*see Table 3*)

- TIA prompting medical attention plus at least one other TIA in the preceding seven days (2 points);
- Ipsilateral ≥ 50% ICA stenosis (2 points);
- Acute diffusion-weighted imaging hyperintensity (2 points).

The TIA risk prediction tools are a work in progress, and additional validation studies need to be done before the addition of DWI and carotid imaging to clinical scoring tools can be recommended for routine use.

When patients are admitted, they are likely to have further imaging studies done compared to patients

not admitted. Researchers using a retrospective cohort study on national Veterans Administration data from 2008 found patients were more likely to have any neuroimaging of the brain (91% vs. 78%), MRI (59% vs. 16%), carotid artery imaging (52% vs. 38%), or echocardiogram (46% vs. 22%). They also found 26% of patients who were admitted with a diagnosis of TIA from the emergency department had the discharge diagnosis of stroke.[30] Similar rates of alternative diagnosis were found in 36% of patients in another study.[31]

In a study performed in Canada, researchers noted that patients discharged from the hospital were found to have an 8% return rate within one week of the initial event.[30] With both studies (VA and civilian), the long-term outcome was similar for those admitted and those discharged.[30,31] With a high return rate of stroke, a clinical pathway may help with deciding on a patient's disposition. One study implemented a TIA protocol to standardize and streamline evaluation of those patients.[32] The authors found a decrease in overall time in the hospital with their protocol. Under the protocol, patients either were discharged, admitted, or observed in an emergency department observation unit. Standardized workup included brain and vascular imaging. MRI with DWI was recommended. In regard to vascular imaging, MRA head/neck was first line, followed by CTA head/neck, and carotid duplex. Neurology was consulted. Admission criteria included the following:

- ≥ 50% stenosis of a blood vessel that could explain symptoms;
- ischemic lesion on MRI or CT scan;
- more than one TIA in the past month;
- ABCD2 score of 6-7;
- medical instability (e.g., new-onset atrial fibrillation, uncontrolled hypertension);
- outpatient workup cannot be easily or effectively arranged.

With a negative workup, patients had arranged prompt follow-up in a neurology clinic unless another diagnosis was favored, such as migraine or low probability, in which case they were referred for non-urgent neurology clinic or primary care follow-up. Patients with suspicion of TIA were started on 81 mg unless already on antithrombotic therapy or contraindicated. No difference was found with respect to recurrent TIA or stroke in 90 days either before or after implementation of the protocol.

Ultimately, disposition from the emergency department is decided based on multiple factors. In one study, researchers recommended that providers consider patients' presentation, workup, history, access to follow-up, and overall risk.[32] In this study, patients were in the hospital for less time than in other studies. However, many were sent to an ED observation unit, which might not be present at all facilities. Additionally, access to MRI and in-house neurology consult may not be available at smaller tertiary facilities. Providers also should consider local availability of neurology follow-up when deciding on outpatient management.

Summary

- Diagnosing a TIA clinically is still acceptable, but additional imaging can enhance risk prediction.
- Treating TIAs with etiology-specific interventions can significantly reduce the chance of a future stroke.
- There is no consensus regarding risk-stratification tools, and recent studies question the validity of the ABCD2 score for patients presenting to the emergency department with a TIA.
- Models incorporating imaging and/or recent TIA in future scoring systems may be useful in the future.
- Decisions for disposition from the emergency department are multifactorial and influenced by resources available in the emergency department, institution, and community for rapid follow-up. Imaging findings, medical instability, or access to care should guide whether a patient should be admitted or can be worked up as an outpatient.

References

1. Kernan WN, Ovbiagele B, Black HR, et al. Guidelines for the prevention of stroke in patients with stroke and transient ischemic attack: A guideline for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke* 2014;45:2160-2236.
2. Amarenco P, Lavallée PC, Labreuche J, et al; TIAregistry.org Investigators. One-year risk of stroke after transient ischemic attack or minor stroke. *N Engl J Med* 2016;374:1533-1542.
3. Chen R, Ovbiagele B, Feng W. Diabetes and stroke: Epidemiology, pathophysiology, pharmaceuticals and outcomes. *Am J Med Sci* 2016;351:380-386.
4. Heidenreich PA, Trogdon JG, Khavjou OA, et al. Forecasting the future of cardiovascular disease in the United States. *Circulation* 2011;123:933-944.
5. Sacco RL, Kasner SE, Broderick JP, et al; on behalf of the American Heart Association Stroke Council,

Council on Cardiovascular Surgery and Anesthesia, Council on Cardiovascular Radiology and Intervention, Council on Cardiovascular and Stroke Nursing, Council on Epidemiology and Prevention, Council on Peripheral Vascular Disease, and Council on Nutrition, Physical Activity and Metabolism. An updated definition of stroke for the 21st century: A statement for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke* 2013;44:2064-2089.

6. Altavilla R, Anticoli S, Venti MP, et al. clinical skills or high-tech MR in TIA patients: What makes the difference? *Neurol Sci* 2018;39:2091-2096.
7. Sorensen AG, Ay H. Transient ischemic attack: Definition, diagnosis, and risk stratification. *Neuroimaging Clin N Am* 2011;21:303.
8. Kim J, Park JE, Nahrendorf M, Kim DE. Direct thrombus imaging in stroke. *J Stroke* 2016;18:286-296.
9. Markl M, Semaan E, Stromberg L, et al. Importance of variants in cerebrovascular anatomy for potential retrograde embolization in cryptogenic stroke. *Eur Radiol* 2017;27:4145-4152.
10. Liberman AL, Zandieh A, Loomis C, et al. Symptomatic carotid occlusion is frequently associated with microembolization. *Stroke* 2017;48:394-399.
11. Adams HP Jr, Bendixen BH, Kappelle LI, et al. Classification of subtype of acute ischemic stroke. Definitions for use in a multicenter clinical trial. TOAST. Trial of Org 10172 in Acute Stroke Treatment. *Stroke* 1993;24:35-41.
12. Blasberg TF, Wolf L, Henke C, Lorenz MW. Isolated transient vertigo: Posterior circulation ischemia or benign origin? *BMC Neurol* 2017;17:111.

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CME/CE Objectives

Upon completion of this educational activity, participants should be able to:

- recognize specific conditions in patients presenting to the emergency department;
- apply state-of-the-art diagnostic and therapeutic techniques to patients with the particular medical problems discussed in the publication;
- discuss the differential diagnosis of the particular medical problems discussed in the publication;
- explain both the likely and rare complications that may be associated with the particular medical problems discussed in the publication.

13. Abbatemarco JR, Rae-Grant AD. Transient neurologic syndromes: A diagnostic approach. *Cleve Clin J Med* 2018;85:155-163.
14. Yew KS, Cheng EM. Diagnosis of acute stroke. *Am Fam Physician* 2015;91:528-536.
15. Bangergee G, Stone SP, Werring DJ. Posterior circulation ischaemic stroke. *BMJ* 2018;361:k1185.
16. Lebedeva ER, Gurary NM, Olesen J. Headache in transient ischemic attacks. *J Headache Pain* 2018;19:60.
17. Arboix A, García-Eroles L, Sellarés N, et al. Infarction in the territory of the anterior cerebral artery: Clinical study of 51 patients. *BMC Neurol* 2009;9:30.
18. Coutts SB. Diagnosis and management of transient ischemic attack. *Continuum* (Minneap Minn) 2017;23(1, Cerebrovascular Disease):82-92.
19. Jauch EC, Saver JL, Adams HP Jr, et al; American Heart Association Stroke Council; Council on Cardiovascular Nursing; Council on Peripheral Vascular Disease; Council on Clinical Cardiology. Guidelines for the early management of patients with acute ischemic stroke: A guideline for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke* 2013;44:870-947.
20. Brazzelli M, Chappell FM, Miranda H, et al. Diffusion-weighted imaging and diagnosis of transient ischemic attack. *Ann Neurol* 2014;75:67-76.
21. Ferguson GG, Eliasziw M, Barr HW, et al. The North American Symptomatic Carotid Endarterectomy Trial: Surgical results in 1415 patients. *Stroke* 1999;30:1751-1758.
22. Chimowitz MI, Lynn MJ, Derdeyn CP, et al; SAMMPRIS Trial Investigators. Stenting versus aggressive medical therapy for intracranial arterial stenosis. *N Engl J Med* 2011;365:993-1003.
23. Wang X, Zhao X, Johnston SC, et al; CHANCE investigators. Effect of clopidogrel with aspirin on functional outcome in TIA or minor stroke: CHANCE substudy. *Neurology* 2015;85:573-579.
24. Wardlaw JM, Brazzelli M, Chappell FM, et al. ABCD2 score and secondary stroke prevention: Meta-analysis and effect per 1,000 patients triaged. *Neurology* 2015;85:373-380.
25. Fothergill A, Christianson TJ, Brown RD Jr, Rabinstein AA. Validation and refinement of the ABCD2 score: A population-based analysis. *Stroke* 2009;40:2669-2673.

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26. American College of Emergency Physicians. Suspected Transient Ischemic Attack. Critical issues in the evaluation of adult patients with suspected transient ischemic attack in the emergency department. 2016; Available at: https://www.acep.org/patient-care/clinical-policies/suspected-transient-ischemic-attack/#sm.0000g2j3p13ykcvvzg71f5uvlmqo8. Accessed Nov. 1, 2018.
27. Giles MF, Albers GW, Amarenco P, et al. Early stroke risk and ABCD2 score performance in tissue- vs time-defined TIA: A multicenter study. *Neurology* 2011;77:1222-1228.
28. Mayer L, Ferrari J, Krebs S, et al. ABCD3-I score and the risk of early or 3-month stroke recurrence in tissue- and time-based definitions of TIA and minor stroke. *J Neurol* 2018;265:530-534.
29. Cheng EM, Myers LJ, Vassar S, Bravata DM. Impact of hospital admission for patients with transient ischemic attack. *J Stroke Cerebrovasc Dis* 2017;26:1831-1840.
30. Kapral MK, Hall R, Fang J, et al. Association between hospitalization and care after transient ischemic attack or minor stroke. *Neurology* 2016;86:1582-1589.
31. Schrock JW, Glasenapp M, Victor A, et al. Variables associated with discordance between emergency physician and neurologist diagnoses of transient ischemic attacks in the emergency department. *Ann Emerg Med* 2012;59:19-26.
32. Jarhult SJ, Howell ML, Barnaure-Nachbar I, et al. Implementation of a rapid, protocol-based TIA management pathway. *West J Emerg Med* 2018;19:216-223.

CME/CE Questions

1. What is the modern definition of a transient ischemic attack?
   a. Focal neurologic deficit lasting > 24 hours without acute findings noted on CT or MRI
   b. Focal neurologic deficit lasting < 24 hours without acute findings on MRI or CT scan
   c. Focal neurologic deficit lasting > 24 hours with acute findings on MRI
   d. Focal neurologic deficit lasting < 24 hours with acute findings on CT and MRI
2. A 55-year-old male presents with a history of right-sided weakness lasting only 20 minutes. It is completely resolved at the time of evaluation. Vital signs at triage are pulse 88, respiratory rate (RR) 12, temperature 98.6° F, blood pressure 130/75 mmHg, and oxygen saturation 99% on room air. He has history of smoking and high cholesterol, and states that this has never happened before. What can be said about the patient's ABCD2 score based on the reading?
   a. The patient has a score of 5 and is at moderate risk for stroke in the next several days.
   b. The patient has a score of 3 and is at high risk for stroke in the next several days.
   c. The patient has a score of 3 and can be discharged safely with a low risk of stroke in the next several days.
   d. The patient has a score of 3; however, this reading should not be used alone to risk stratify TIA patients.
3. The patient from question 2 has a CT head scan without contrast and CT angiography of the head and neck. What finding on his imaging

is the most worrisome for an increased risk of stroke?
a. Left-sided carotid stenosis of 60% on CT angiography with a normal head CT
b. Right-sided carotid stenosis of 60% on CT angiography with a normal head CT
c. Normal imaging studies
d. Right-sided carotid stenosis of 20% on CT angiography with normal head CT

4. Which of the following would be the most appropriate disposition for this patient?
   a. Discharge and recommend follow-up with his primary care provider in one week.
   b. Admit for further evaluation and management of the symptomatic carotid stenosis.
   c. Discharge and recommend follow-up on an as-needed basis.
   d. Provide outpatient referral to psychiatry for somatoform disorder.
5. Under the TOAST classification of stroke, what are the five categories for the etiology of a stroke?
   a. Large-artery atherosclerosis, cardioembolism, small-vessel occlusion, stroke of other determined etiology, and stroke of undetermined etiology
   b. Hemorrhagic, ischemic, small vessels, other, and undetermined
   c. Large-artery atherosclerosis, hypertension, smoking, diabetes, and hyperlipidemia
   d. Large-artery atherosclerosis, hemorrhagic, ischemic, cardiogenic, and vaso-occlusive
6. Which imaging modality has a very high sensitivity for detecting brain ischemia?
   a. A noncontrast head CT with CT angiography of the head and neck
   b. Carotid ultrasound with head CT without contrast
   c. Carotid ultrasound, head CT without contrast, and CT angiography of the head and neck
   d. Diffusion-weighted MRI
7. A patient presents with weakness on the right side that is rapidly improving. TIA is suspected. What should be included in the patient's workup?
   a. ECG and point-of-care glucose only
   b. ECG, point-of-care glucose, cardiac enzymes, coagulation studies, complete blood count, electrolyte panel including renal function studies, and noncontrast head CT
   c. Noncontrast head CT, ECG, and point-of-care glucose
   d. ECG, point-of-care glucose, complete blood count, and cardiac enzymes
8. Which of the following maneuvers is performed when administering the HINTS exam?
   a. Head impulse test, nystagmus, and test of skew
   b. Head induction test, neck movement, and test of skew
   c. Head impulse test, nystagmus, and test of vision
   d. Head impulse test, neuroimaging, and test of skew
9. Patients diagnosed with TIA and new-onset atrial fibrillation should be considered candidates for what therapy?
   a. Antiplatelet therapy alone
   b. Dual antiplatelet therapy
   c. Oral anticoagulation
   d. Oral anticoagulation and antiplatelet therapy

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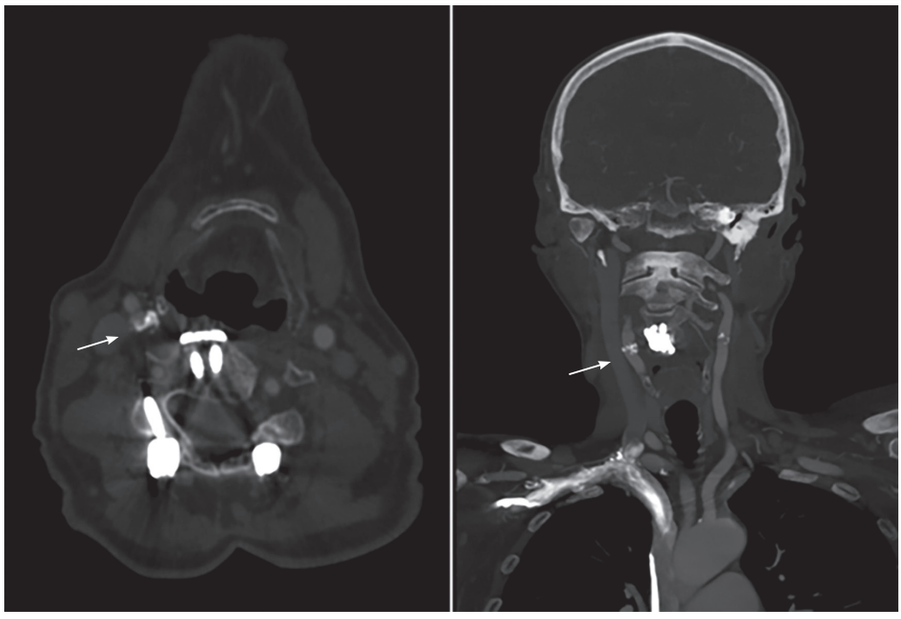
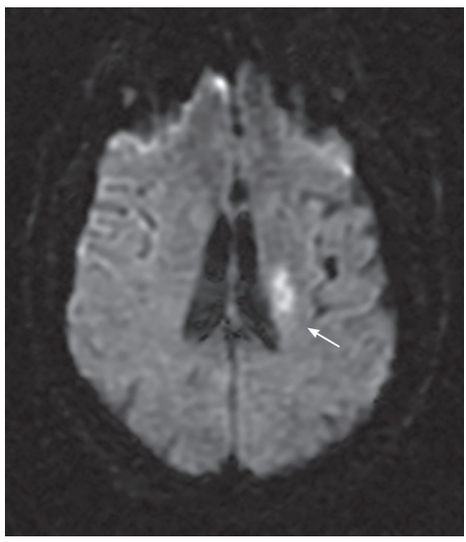
EMERGENCY MEDICINE **REPORTS**

Transient Ischemic Attack

Right Internal Carotid Artery Stenosis (Arrows) of About 60% in a Patient Diagnosed With TIA



MRI DWI of an Acute Infarct of the Left Corona Radiata (Arrow)



Risk Factors for Transient Ischemic Attack or Stroke[1,5]

- Prior TIA or stroke
- Hypertension
- Diabetes mellitus
- Hypercholesterolemia
- Atrial fibrillation
- Congestive heart failure
- Smoking
- Patent foramen ovale
- Aortic atheroma
- Substance abuse
- Antiphospholipid antibodies
- Sickle cell disease
- Mechanical or other heart valve disease
- Hypercoagulation
- Sleep apnea
- Obesity
- Physical inactivity
- Pregnancy
- Advanced age
- Family history

Risk Stratification Tools for Occurrence of Stroke After TIA

Tools (points assigned)	ABCD	ABCD2	CIP	ABCD2-I	ABCD3-I
Clinical Features					
Age ≥ 60 years	1	1	1	1	1
Symptom duration 10-59 minutes	1	1	1	1	1
Symptom duration ≥ 60 minutes	2	2	2	2	2
Focal weakness	2	2	2	2	2
Speech impairment without weakness	1	1	1	1	1
Diabetes mellitus		1	1	1	1
Systolic blood pressure > 140 mmHg or Diastolic blood pressure > 90 mmHg		1	1	1	1
TIA within preceding seven days					2
Imaging Features					
Acute infarction on diffusion-weighted imaging			1	3	2
Ipsilateral carotid stenosis ≥ 50%					2
Total possible score	6	7	Four categories: ABCD2 < 4 or ≥ 4 and/or infarct seen on DWI	10	13
Risk prediction interval	Seven days	2, 7, and 90 days	2 and 7 days	7 and 90 days	2, 7, 28, and 90 days

Abbreviations: ABCD = Age, Blood pressure, Clinical features, Duration score; ABCD2 = Age, Blood pressure, Clinical features, Duration, Diabetes score; CIP = Clinical and Imaging-based Predictive score; ABCD2-I = ABCD2 plus diffusion-weighted imaging; ABCD3-I – ABCD2 plus TIA within seven days of index event and diffusion-weighted imaging

Summary of Antiplatelet Treatment[19,22]

- For noncardioembolic TIA, use of antiplatelet agents rather than oral anticoagulation is recommended.
- Aspirin 81-325 mg/day monotherapy or combination aspirin 25 mg/extended-release dipyridamole 200 mg twice daily for initial therapy
- Clopidogrel 75 mg alone is reasonable in place of aspirin or aspirin/dipyridamole when aspirin is contraindicated
- May consider clopidogrel for 90 days plus aspirin within 24 hours of a TIA
- No significant evidence to support increasing dose of aspirin for those already on aspirin therapy. May consider adding another agent; however, no agent(s) have been studied sufficiently

Supplement to *Emergency Medicine Reports*, February 15, 2019: "Transient Ischemic Attack." Author: Derek Nakayama, MD, Emergency Medicine, Arlington, WA.