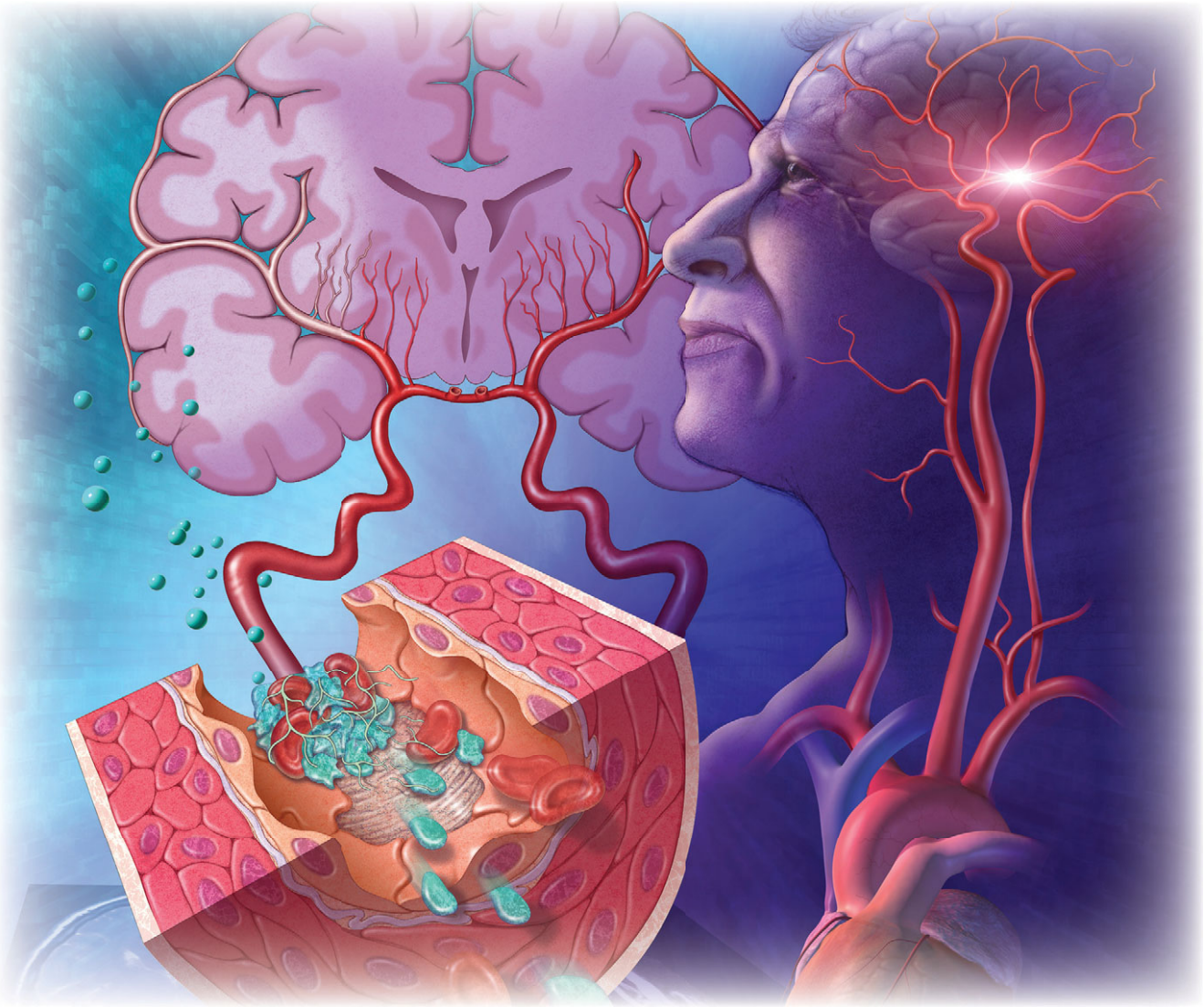

# Transient Ischemic Attack: What You Need to Know

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In a recent study, almost a third of primary care presentations consistent with transient ischemic attack were not followed up for at least a month—even though a TIA precedes roughly one in four strokes. The authors explore the relationship between TIA and stroke and outline an appropriately aggressive approach to diagnosis and management of suspected TIA.

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Just as angina is often the first sign of an impending acute myocardial infarction, a transient ischemic attack (TIA) is often the first sign of an imminent stroke—the leading cause of disability and the third leading cause of death in the United States.[1] Approximately 795,000 Americans experience a new or recurrent stroke each year.[2] The management of ischemic stroke is primarily supportive and death or the degree of disability is largely predetermined, although tissue plasminogen activator can sometimes reverse the course of the event. That means the opportunity to recognize and successfully manage transient ischemic attack may be the best chance physicians have to spare a given patient irreversible harm or loss of life from stroke.

Historically, physicians have not been aggressive in the workup of TIA. A recent study reported that 31% of patients presenting to primary care with symptoms consistent with TIA had no further workup within the first month.[3] This article will discuss how to ensure that patients presenting to urgent care get the thorough and timely workup necessary to reduce the risk of recurrent TIA and permanent and debilitating stroke.

## DRAWING THE LINE

Ischemic stroke and transient ischemic attack share the same pathophysiology. Risk factors for both include hypertension, smoking, obesity, and diabetes. Either may be due to areas of stenosis in major vessels such as the internal carotid artery or the anterior, middle, or posterior cerebral artery, or in the vertebral or basilar artery. If the stenosis is severe enough and collateral flow is impaired, TIA or stroke may occur. Stenosis of the tiny penetrating arteries, usually from longstanding hypertension or diabetes, can similarly cause TIA or small strokes, called *lacunes*. Embolic TIAs and strokes generally arise from cardiac pathology but may also result from primary vascular disease such as atherosclerosis.

The definition of the term *transient ischemic attack* is still a work in progress. The original or classic definition states that a TIA is a focal neurologic deficit caused by focal brain ischemia that completely resolves in less than 24 hours.[4] This time limit, adopted in 1975 by the National Institutes of Health, is arbitrary and originated in the 1950s and 1960s, when advanced imaging was not available. Simply put, it was thought that symptoms lasting less than 24 hours were unlikely to cause infarction and permanent impairment. In fact, most TIAs resolve within one hour and if they do not, there is less than a 15% chance that they will resolve within 24 hours.[4]

The notion of a 24-hour time frame has also undergone scrutiny with the advent of new imaging technology such as CT and MRI. Multiple studies have shown that significant numbers of patients who had been diagnosed with TIA actually had permanent ischemic brain injuries. Therefore, a newer, tissue-based definition has been proposed that emphasizes the distinction between ischemia (TIA) and infarction (stroke). This definition, proposed in the *New England Journal of Medicine* in 2002, states that "a TIA is a brief episode of neurologic dysfunction caused by focal brain or retinal ischemia, with clinical symptoms typically lasting less than one hour, and without evidence of acute infarction."[4] Ideally, then, each patient would undergo advanced imaging such as CT or MRI to better assess for any signs of infarction.

Diffusion-weighted MRI measures the diffusion of water and has been shown to differentiate TIA from stroke within 6 hours of symptom onset. This same technology, however, has also served to cloud the definition of TIA. In a recent study, 87 patients with a clinical diagnosis of TIA but negative CT underwent diffusion-weighted MRI. Thirty-six (41%) had evidence of infarction.[5] This has led some authorities to classify this subgroup of patients as having "transient symptoms associated with infarction."[5] Currently, the presence of new brain infarction does not influence management of patients with transient symptoms, so the distinction may be more academic than clinical.

**>>FAST TRACK<<**

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## ASSESSING THE PATIENT

Clinical symptoms of TIA, which vary widely, can be sorted into three groups according to whether

there is ischemia in the anterior, posterior, or deep circulation.

The *anterior circulation* involves the right and left internal carotid arteries and their branches (ophthalmic, anterior cerebral, and middle cerebral) and supplies blood to the retinas and most of the cerebral hemispheres. The classic ocular manifestation of TIA is amaurosis fugax, temporary monocular blindness caused by occlusion of the ophthalmic artery. It should be noted, though, that patients can simply have a visual field deficit rather than complete blindness. When the anterior cerebral artery is involved, common symptoms include unilateral weakness, paralysis, or numbness in the lower extremities. When the problem is in the middle cerebral artery, these same symptoms involve the contralateral face and upper extremity. Other symptoms include aphasia (when the dominant hemisphere is affected), neglect, and homonymous hemianopia.

The *posterior circulation* supplies blood to the occipital lobes, brain stem, and cerebellum through the basilar and vertebral arteries as well as the posterior cerebral artery. In TIAs involving the posterior system, common symptoms include ataxia, vertigo, nausea, and vomiting. Disruptions in the occipital lobe can cause gaze disturbances, leading to diplopia. Other symptoms include dysarthria and dysphagia. It should be noted that when these symptoms are a result of TIA or stroke, they appear in clusters and are rarely found in isolation.

Lacunar TIAs are caused by ischemia in the *deep circulation* of the brain and result in pure motor or sensory deficits.

Patients with suspected TIA must undergo a standardized systematic evaluation to determine the extent of impairment, rule out potential mimics of TIA, and elucidate possible causes. Complete history and physical exam are required; the latter includes a review of vital signs, pulse oximetry, ECG, complete blood count, basic metabolic panel, cardiac enzymes, and head CT. Derangements in glucose and other electrolyte levels need to be identified because they can cause altered neurologic function. ECG is most useful in detecting atrial fibrillation, which is a risk factor for TIA because it causes embolic events. CT is most useful for detecting other problems that mimic TIA, such as a brain tumor, as well as for identifying a subdural, epidural, or subarachnoid hemorrhage. Diffusion-weighted MRI, as noted earlier, can detect ischemic lesions within minutes of onset. The National Stroke Association also recommends carotid artery imaging using CT or MR angiography or Doppler ultrasound, if available, as well as transthoracic or transesophageal echocardiography with testing for right to left shunting if a cardioembolic source is suspected.[6]

**>>FAST TRACK<<**

**CT is most useful for detecting other problems that mimic TIA.**

## PREDICTIVE RELATIONSHIP

In general, about 25% of strokes are preceded by TIA. Several studies analyzing this relationship underscore the importance of an expedited workup and treatment regimen. Shah and colleagues recently published a critically appraised topic on the short-term risk of stroke following TIA.[7] Eight studies—five prospective, three retrospective—were selected. Two of the five prospective studies were based on patients presenting solely to emergency departments, while the other studies included large numbers of patients who were seen in primary care settings. The combined average 2-day risk of stroke was 1.5% to 10%, the 7-day risk was 4% to 13%, and the 30-day risk was 5% to 18%. One of the studies included data showing that 64% of those who suffered a subsequent stroke were significantly disabled and 21% died.[7]

The $ABCD^2$ prediction rule is a new tool that calculates 2-day stroke risk to help clinicians determine whether to admit a patient presenting with TIA.[8] It is based on data from two clinical prediction rules: the ABCD score and the California score. Points are given based on (A) age, (B) blood pressure, (C) clinical features, (D) duration of symptoms, and (D) diagnosis of diabetes mellitus. Age over 60 is given 1 point. For blood pressure, a systolic reading greater than 140 or a diastolic reading greater than 90 is given 1 point. Under clinical features, unilateral weakness is given 2 points, speech disturbance 1 point, and all other clinical presentations are given 0 points. For duration of symptoms, a neurologic deficit lasting more than 60 minutes is given 2

points; between 10 and 59 minutes, 1 point; and if less than 10 minutes, no points are given. An additional point is assigned if the patient has diabetes. A total score of 3 or less indicates there is a 1% chance of stroke within the next 2 days. Scores of 4-5 and 6-7 predict a 2-day stroke risk of 4.1% and 8.1%, respectively.[8]

Certainly, a score of 5 or more should prompt the physician to admit for expedited care. It is important to remember, though, that the $ABCD^2$ score is simply one part of the evaluation and must not be the sole determinant of admission or discharge. Certain high-risk groups must be admitted regardless of their score; these include patients with recurrent TIA during any type of antiplatelet or anticoagulant therapy, those with TIA of presumed cardiac origin (such as atrial fibrillation), and those with crescendo TIAs.

The National Stroke Association's recent guideline update for the management of TIA places increased emphasis on speed in the workup.[6] Admission should be considered for all patients with their first presumed TIA if they present within 24 to 48 hours—the so-called vulnerable period. This is to facilitate definitive secondary prevention and the use of lytic therapy if symptoms recur. Patients who are discharged must be fully informed of the need to return if symptoms recur. Other reasons to admit based specifically on these guidelines include crescendo TIAs, duration of symptoms greater than 1 hour, known carotid stenosis of 50% or worse, atrial fibrillation or other cardioembolic potential, or known hypercoagulable state.

## POSTDIAGNOSTIC MANAGEMENT

Interestingly, a recent trial suggests that a special TIA observation unit within an emergency department may do as well or better for these patients compared with hospital admission, a concept that may merit consideration by urgent care facilities as well.[9] In this study, patients with TIA who had normal CT, ECG, and lab test results and no known embolic source were either

admitted or placed in an accelerated diagnostic protocol. Each group underwent serial clinical exams, neurologic consult, carotid Doppler ultrasound, echocardiography, and cardiac monitoring. Of the patients in the special diagnostic protocol, 15% had a positive finding that triggered admission. Length of stay was significantly less for the emergency department observation unit patients, with a median length of stay of 25.6 hours versus 61.2 hours for inpatients.

Having a TIA observation unit seems to save money as well. Median cost per patient was $890 for observed patients versus $1547 for admitted patients. It should also be noted that carotid imaging and echocardiography were completed more frequently in the observation unit group. Three-month follow-up revealed similar rates of return visits, subsequent strokes, and other major clinical events.

Clearly, observation is the safest management for TIA patients. If, however, a patient will not stay or is otherwise discharged, several guidelines should be followed.[6] Close follow-up should be ensured. Unless there is a specific contraindication, antiplatelet therapy should be initiated. This can be in the form of aspirin 50 to 325 mg daily, clopidogrel 75 mg daily, or a 25/200 mg aspirin/dipyridamole combination twice daily.

In rural practice or where follow-up cannot be assured, other steps need to be taken. For patients with atrial fibrillation, warfarin should be prescribed with a target international normalized ratio of 2.5. Enoxaparin should also be considered initially due to the hypothetical risk of hypercoagulability within the first few days of warfarin initiation. Other interventions should include blood pressure control and use of a statin. After a brief waiting period of 24 to 36 hours, blood pressure should be reduced to a goal of less than 120/80 mm Hg. This can be done with first-line agents such as thiazides and ACE inhibitors or angiotensin receptor blockers. Second-line agents include beta-blockers for those with heart disease and calcium channel blockers for African Americans. Atorvastatin 80 mg daily has been shown to reduce recurrent stroke risk.

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Efforts should also be made toward smoking and alcohol cessation, glucose control for diabetic patients, weight loss, and referral for sleep studies in those who may have sleep apnea. Patients should be cautioned against the use of any sympathomimetic drugs or drugs that increase the risk of blood clotting, such as birth control pills or hormone therapy.[6]

We suggest the liberal admission of patients diagnosed with TIA. Although discharge with close follow-up may be appropriate in a certain subgroup, an observation unit or hospital admission offers the benefits of expedited diagnostics and interventions that can prevent the devastating consequences of stroke. □

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