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Transient ischemic attack

Heed the warning

By Vince Vacca, MSN, RN, CCRN, SCRN

MR. R, 60, EXPERIENCES a sudden onset of left upper extremity paresis and right-sided visual loss. He reports an episode of similar symptoms a few days ago that resolved, so he didn't seek medical attention. He's 68 in (173 cm) tall and weighs 210 lb (95 kg) with a body mass index of 31.9. His health history includes type 2 diabetes, hypertension, dyslipidemia, and myocardial infarction. His medications include a calcium channel blocker, an oral antihyperglycemic agent, and a statin. He utilizes diet and exercise to try to control his weight. He has no history of smoking.

Mr. R arrives at the nearest ED within 30 minutes of symptom onset, where he's rapidly triaged and assessed. A stat noncontrast head computed tomography (CT) scan, performed within 25 minutes of arrival, doesn't reveal any pathology, such as cerebral hemorrhage, to explain the sudden onset of stroke-like signs and symptoms, which are rapidly resolving without treatment. Is Mr. R experiencing a transient ischemic attack (TIA)?

The annual incidence of TIA in the United States is estimated to be 200,000 to 500,000. About half of those who experience a TIA don't report it, representing lost opportunities for both intervention and stroke prevention. The actual incidence isn't known due to underreporting.[1]

In the past, TIA was diagnosed based on abrupt onset and duration of characteristic neurologic signs and symptoms that resolved spontaneously within 24 hours.[2] However, this definition is now considered inadequate because even episodes of relatively brief ischemia can cause permanent brain injury.

Today, the definition is tissue-based rather than time-based. A TIA is now defined as a transient episode of neurologic dysfunction caused by focal brain, spinal cord, or retinal ischemia without acute infarction.[3,4,5] Diagnosis is based on findings of an abrupt onset of focal neurologic signs or symptoms without evidence of brain tissue infarction on imaging.[4] The current definition offers a significant opportunity for timely interventions to prevent a TIA from evolving into a stroke.[3]

Because a TIA results from cerebrovascular disturbances that exist on the same continuum as stroke, it's a medical emergency requiring prompt assessment and intervention.[4] This

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article discusses the nurse's responsibility to recognize signs and symptoms of TIA and to respond quickly and appropriately with current, evidence-based interventions.

Ominous warning

TIA can be a precursor to stroke because both are caused by the same cerebrovascular disease processes. TIA is associated with a 9.9% risk of stroke at 2 days postevent, 13.4% at 30 days, and 10% to 17% at 90 days.[1,6-8] The post-TIA stroke risk is different for each patient based on individual modifiable risk factors (hypertension, diabetes, abnormal blood lipid profile, smoking status, sedentary lifestyle, and obesity) and nonmodifiable risk factors (age, gender).[9] TIA affects more males than females and more Blacks than Whites, but the rate increases for all population groups with advancing age.[4] (See *Risk factors for TIA and stroke*.) Although many TIAs resolve spontaneously, others progress to stroke with permanent neurologic deficits in less than 24 hours.[1,10]

Given the association with vascular disease, it should be no surprise that a TIA also raises the risk of other vascular events, such as unstable angina, myocardial infarction, peripheral artery disease, and renal artery disease.[11]

Typically three clinical features suggest a TIA: symptom onset within seconds of the event, no history of similar episodes, and absence of nonspecific symptoms such as gastrointestinal distress, chest pain, or shortness of breath.[1] No one can predict which TIAs are likely to progress, so they're considered medical emergencies and evaluated as potential strokes. Rapid recognition and response is essential to reduce the risk of disability and death.[4,8,10]

As the American Heart Association/American Stroke Association (AHA/ASA) points out, time lost is brain lost.[12] Rapid, accurate history taking, clinical evaluation, and diagnosis are essential to differentiate TIA from acute ischemic or hemorrhagic stroke or other central nervous system pathology.

Variable signs and symptoms

A thorough baseline neurologic assessment includes evaluation of mental status, cranial nerve function, motor function, sensory function, and reflexes. For details, see "Performing a Focused Neurologic Assessment" in the December issue of *Nursing2013*.

Signs and symptoms of TIA vary depending on the cerebral vascular territory affected. (See *Mapping the brain's blood supply*.) For example, a TIA affecting anterior cerebral artery circulation, which includes cerebral blood vessels originating from the carotid arteries, can manifest as hemiparesis, hemianesthesia, contralateral motor or sensory deficits of the face or extremities, amaurosis fugax (temporary monocular blindness), a "shade" descending over the line of vision, or transient graying, fogging, or blurred vision.[13]

Signs and symptoms of a TIA affecting the posterior cerebral artery circulation, which includes cerebral blood vessels originating from the vertebral and basilar arteries, can include dysarthria, dysphagia, diplopia, bilateral blindness, unilateral or bilateral motor and sensory weakness, quadriparesis, ataxia, vertigo, and dizziness.[13]

The risk of progression to stroke from TIA is greatest with carotid artery stenosis rather than lacunar pathology (occlusion of smaller, distal arteries) or cardioembolic sources.[4,14] Because TIA and minor strokes are often caused by unstable plaque affecting large arteries that supply extensive brain tissue territories, strokes that follow TIA may be severely disabling or fatal.[14]

In many cases, TIA signs and symptoms resolve before the patient can be evaluated, making a thorough patient history essential for diagnosis and management.

Assessment tools

The National Institutes of Health Stroke Scale (NIHSS) is a simple, validated, and reliable tool for assessing initial stroke severity. It's been shown to predict mortality in acute ischemic stroke in several studies.[15]

Every patient with stroke-like signs and symptoms, including those suggesting a TIA, should undergo an NIHSS evaluation on presentation. The 15-item systematic assessment tool provides a quantitative measure of stroke-related neurologic deficits by helping clinicians evaluate the effect of acute ischemic infarction on level of consciousness, language, extinction and inattention (formerly neglect), visual-field loss, extraocular movements, motor strength, ataxia, dysarthria, and sensory loss. Unlike patients with established strokes, patients experiencing a TIA will have either normal scores (if signs and symptoms have resolved) or scores rapidly improving to normal. For details about scoring, visit http://www.nihstrokescale.org.

Another assessment tool used in addition to the NIHSS is the ABCD^2 score,

Risk factors for TIA and stroke[34]

Nonmodifiable

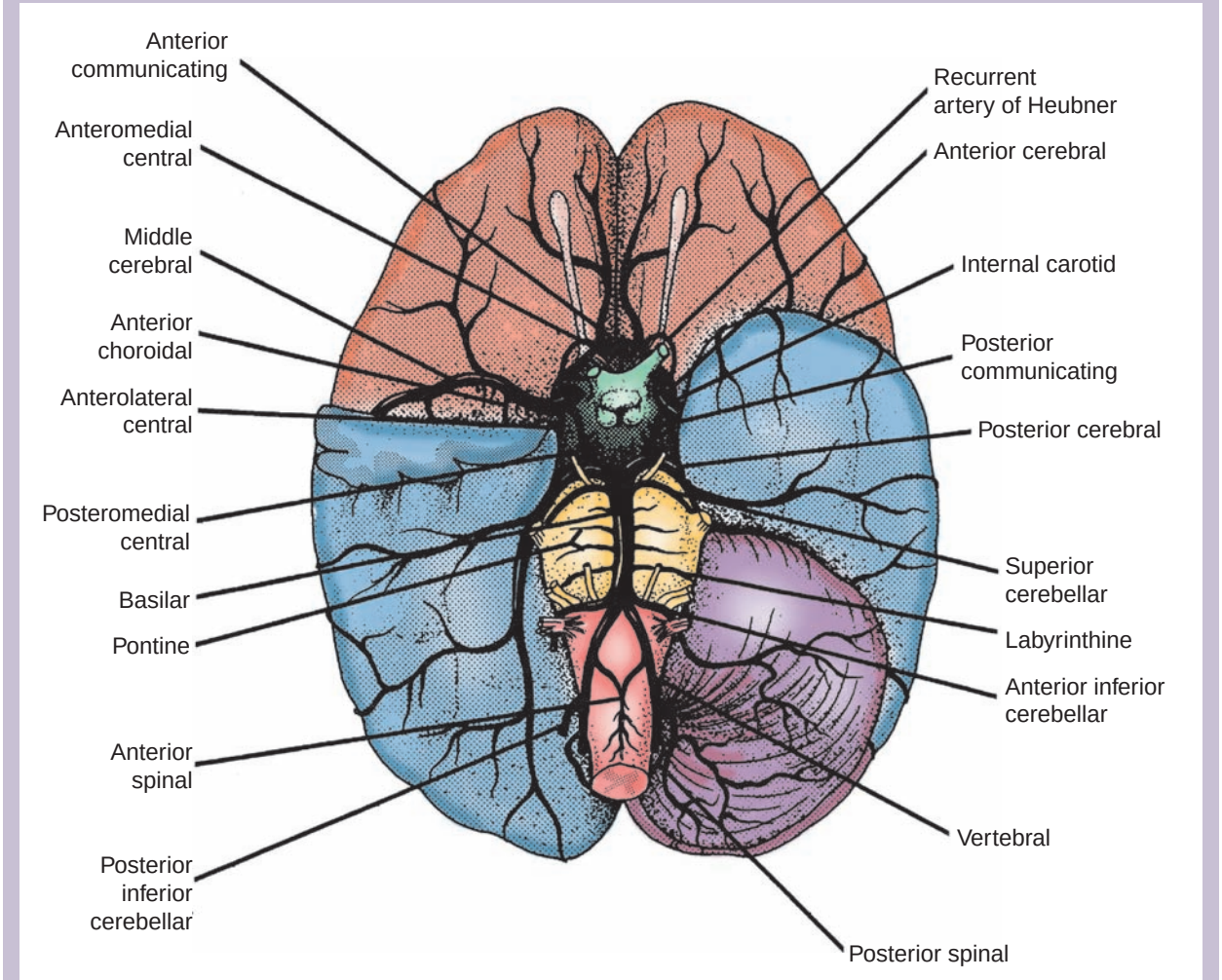
- age
- gender
- race/ethnicity
- heredity

Modifiable

- hypertension
- cardiac disease (coronary artery disease, AF, valvular disorders)
- diabetes mellitus
- hypercholesterolemia
- smoking
- excessive alcohol use
- physical inactivity
- obesity.

Mapping the brain's blood supply[33]

This is a view of the brain's ventral surface with the right cerebellar hemisphere and the tip of the right temporal lobe removed.



which helps clinicians determine short-term stroke risk after a TIA. Ranging from 0 to 7, the ABCD^2 score is calculated by adding points for five factors: **A**ge, **B**P, **C**linical features of TIA, **D**uration, and **D**iabetes.[16] The patient's score provides decision support for hospitalization of higher-risk patients or discharge of lower-risk patients from the ED to complete their TIA evaluation in an outpatient setting.[2,11] Higher ABCD^2 scores on initial evaluation indicate stroke risk at 2, 7, 30, and 90 days. For example, an ABCD^2 score of 6 confers a 2-day stroke risk of 8%, which is considered high-risk.[1]

Hospitalization for observation is generally not indicated for patients with ABCD^2 scores of 0 to 3 (2-day stroke risk, 1%), unless there's another indication such as new-onset atrial fibrillation (AF).[1,6,17] Some facilities have an outpatient TIA clinic to which these patients are referred for further evaluation, usually within 1 to 2 days.

For most patients with an ABCD^2 score of 4 or 5 (2-day stroke risk, 4.1%), hospital observation is indicated. Head and neck imaging should take place during the ED admission. If stenosis of greater than 50% is identified in either carotid artery, hospital admission is recommended.

For patients with an ABCD^2 score of 6 or 7 (2-day stroke risk, 8.1%), hospital admission is recommended.[1,6,11] If a patient already hospitalized for any reason develops acute onset of neurologic symptoms suggesting TIA, a rapid response team leading to neurologic expertise or a code stroke is indicated.

Diagnostic studies

Lab testing can help rule out metabolic and hematologic causes of

neurologic signs and symptoms and may include a comprehensive metabolic panel, complete blood cell count, coagulation profile, cardiac biomarkers, erythrocyte sedimentation rate, and lipid profile based on individual presentation, assessment findings, and risk factors.

Neuroimaging studies include brain CT or magnetic resonance imaging (MRI). Estimates are that one-third of patients meeting traditional TIA criteria (such as signs and symptoms lasting less than 24 hours) have evidence of infarction on brain imaging. These lesions, detectable by MRI, confirm that the episode was caused by cerebral ischemia.[1] By current standards, if a patient presents with transient signs and symptoms that have rapidly reversed but imaging reveals evidence of infarction, he or she has an established stroke, not a TIA.

AHA Class I recommendations for TIA workup include neuroimaging evaluation within 24 hours of symptom onset, consisting of brain tissue evaluation by CT or MRI, and noninvasive imaging of the craniocervical vessels, which can be accomplished with ultrasound, magnetic resonance angiography (MRA), or computed tomography angiography (CTA).

The AHA guidelines recommend a stat noncontrast CT scan within 25 minutes of arrival in the ED for patients presenting with stroke symptoms and inpatients with suspected stroke. If this isn't possible, the AHA recommends neuroimaging within 24 hours of symptom onset.[2,8]

Results of the noncontrast CT must be interpreted within 45 minutes by an expert to guide further treatment.[18] If the noncontrast CT is negative for the presence of blood or other pathology that can explain the patient's signs and symptoms, the clinician may consider other neuroimaging studies that can support the diagnosis of TIA.[2]

Diffusion-weighted imaging by MRI (DWI) can reliably show infarcted brain tissue. The AHA and the American Association of Neurology recommend performing this study within 12 to 24 hours of TIA symptom onset.[1,4] It's the most sensitive imaging/diagnostic technique for identifying areas of infarcted brain tissue in small or perforator cerebral arterial territories, identifying the event as a stroke rather than a TIA.[6,19] Because DWI is superior to noncontrast CT as an imaging modality to identify infarcted brain tissue, it's used to identify patients who are tissue-damage positive (stroke) or tissue-damage negative (TIA). Other options include perfusion-weighted MRI, which assesses capillary perfusion.[20]

DWI is recommended as the most sensitive diagnostic imaging study following TIA. Although parenchymal (brain tissue) imaging with MRI best shows the presence of stroke, an additional important consideration is cervical imaging to assess for carotid artery stenosis, which is a known cause of 15% to 20% of ischemic strokes.[21,22]

Additional imaging studies include carotid duplex ultrasonography, transcranial Doppler (TCD), and cerebral angiography.

An ECG can identify AF or another cardiac abnormality that could contribute to the TIA diagnosis. In some cases, a transthoracic or transesophageal echocardiogram may be indicated to identify cardiac sources for thrombi and emboli.[4]

Management strategies

For many patients with ischemic stroke and precursor events such as TIA, the cause of signs and symptoms is unclear (either because none is found or because several mechanisms are coexisting); however, platelet aggregation with thrombus formation and subsequent embolization are thought to be the most common causative mechanisms.[11] For this reason, patients are started or continued on antiplatelet agents. Aspirin, clopidogrel, and dipyridamole as monotherapy or as combined therapies are recommended by the AHA/ASA and the American College of Chest Physicians as acceptable first-line drug interventions for prevention of secondary ischemic events.[11,23] Oral antiplatelet agents are effective for reducing future atherothrombotic events in patients with stroke and TIA, as demonstrated and reported from the Early Use of Existing Preventive Strategies for Stroke (EXPRESS) trial.[11]

Whether preexisting or new-onset, AF can lead to thrombus formation and stroke because of the prothrombotic state, endothelial dysfunction, and blood stasis associated with this dysrhythmia.[1,24] Anticoagulants are indicated for these patients with careful follow-up monitoring.[24]

The $CHADS_2$ Risk Assessment is a validated clinical tool used to guide treatment and prevent TIA and ischemic stroke in patients with nonvalvular AF. The acronym stands for these risk criteria: **C**ongestive heart failure, **H**ypertension, **A**ge 75 or older, **D**iabetes mellitus, and **S**troke or TIA in the past. Each criterion is assigned one point except for stroke/TIA in the past, which gets two points. The $CHADS_2$ score is used to help clinicians decide whether patients with nonvalvular AF should be started on antiplatelet monotherapy or combination therapy.[25]

Finally, risk factor management should be aggressively pursued. Hypertension is the most important modifiable risk factor. Dyslipidemia, as a risk factor for cerebral and cardiovascular disease leading to myocardial infarction, TIA, or stroke, should be assessed and treated as indicated. Dual antiplatelet agents, including aspirin and clopidogrel, as well as aggressive risk factor modification primarily targeting BP < 140/90 mm Hg (less than 130/80 in patients with diabetes) and LDL < 70 mg/dL, were reported as

effective at reducing risk for stroke or death associated with stenosis of greater than 70% in a major intracranial artery.[26]

Recommendations from the AHA include use of antihypertensives for all patients with a history of hypertension who've had an ischemic stroke or TIA. The AHA suggests that the target BP be individualized, but benefit has been associated with a 10/5 mm Hg reduction in systolic and diastolic values, respectively. Although no specific antihypertensive regimen is recommended due to individual patient characteristics, the AHA does suggest that a combination of a diuretic and an angiotensin-converting enzyme inhibitor is useful.

Statin therapy has been shown to stabilize atherosclerotic plaque, decrease intimal-medial thickness of the carotid arteries, and promote antioxidant, anti-inflammatory, and antiplatelet effects. Results from the Stroke Prevention by Aggressive Reduction in Cholesterol Levels (SPARCL) trial reported that 80 mg/day of atorvastatin in patients with a recent TIA or stroke reduced the incidence of fatal or nonfatal stroke by 16%.[27]

The AHA also recommends several other lifestyle modifications to reduce the risk of TIA, ischemic stroke, and recurrent stroke. These include sodium restriction, weight loss if indicated, low-fat dairy products, a diet rich in consumption of fruits and vegetables, regular physical exercise, and limited alcohol consumption.[8]

Invasive procedures

Identification of critical lesions such as atherosclerotic plaque and thrombosis presents the opportunity for surgical or endovascular interventions before progression to stroke.[28] Carotid endarterectomy (CEA) has been shown to reduce the risk of stroke by 18% in symptomatic patients with a history of TIA or stroke who have 70% to 99% carotid artery stenosis. CEA reduces the stroke risk by 8% in patients with carotid artery stenosis of 50% to 69%, as reported in both the North American Symptomatic Carotid Endarterectomy Trial and the European Carotid Surgery Trial.[29,30]

For patients who are at high surgical risk and younger than age 70, carotid artery stenting has been shown to be an effective alternative to CEA if performed by experienced providers.[31,32] The decision is based on degree of surgical risk for endarterectomy.

Assessing Mr. R

Mr. R's signs and symptoms completely resolve within an hour of onset, consistent with TIA. However, his $ABCD_2$ score is 6, indicating that he's at high risk for stroke within 2 days.

To further evaluate Mr. R's brain tissue for evidence of ischemia or infarction, he undergoes an MRA, which reveals an 85% stenosis of his proximal cervical right internal carotid artery (ICA).

Because Mr. R's signs and symptoms have resolved with no evidence of infarction, his transient neurologic deficits are attributable to a TIA. However, he's at significant risk for a potentially severe and disabling or even fatal stroke.

Because of his significant carotid artery stenosis, Mr. R is admitted to the hospital for further evaluation and management. The nurse keeps the head of the bed flat, as tolerated, to increase cerebral perfusion to ischemic brain tissue, and administers isotonic I.V. fluids as prescribed to maintain euvolemia. One goal is to normalize his BP with antihypertensive medications while preserving cerebral perfusion. He's also started on antiplatelet therapy with clopidogrel. His target goal for low-density lipoprotein (LDL) is less than 70 mg/dL, so the healthcare provider increases his statin dose. Two days later, Mr. R undergoes successful and uncomplicated CEA of his right ICA.

Before discharge, Mr. R meets with a dietitian and a certified diabetes educator to review positive lifestyle choices. Patient education includes a review of the signs and symptoms of TIA/stroke and instruction in what to do if he experiences any of these signs and symptoms after discharge. CEA, drug therapy, and lifestyle modifications, along with regular follow-up by his healthcare provider, have given Mr. R the treatment and support necessary to reduce his risk of experiencing future neurologic events. ■

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