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# CEREBROVASCULAR DISORDERS: ISCHEMIC STROKE IS CAUSED BY CERDIOEMBOLIC SOURCE AND ATRIAL FIBRILLATION

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**Annotation**

Ischemic stroke is the fifth leading cause of death and a major condition feared by older adults. Clinical identification of patients with cerebral ischemia is important to provide appropriate, immediate treatment and initiate stroke preventive strategies. Ischemic stroke is also called brain ischemia and cerebral ischemia. Ischemia is the medical term for "lack of blood supply." The blockage is often caused by blood clots or fatty deposits inside the blood vessel. According to the American Stroke Association, ischemic is the most common type of stroke, accounting for about 87% of all stroke cases.

**Keywords**: ischemic stroke, transient ischemic attack (TIA), cerebral ischemia, hypoperfusion, vessel stenosis, thrombosis, atrial fibrillation.

Ischemic stroke has been classically defined as a fixed focal neurologic deficit attributable to an arterial or venous territory and lasting longer than 24 hours. Transient ischemic attack (TIA) has been classically defined as a transient focal neurologic deficit attributable to an arterial territory lasting less than 24 hours. However, diffusion-weighted magnetic resonance imaging (DWI) has shown that many TIAs are associated with tissue damage. Therefore, a tissue-based diagnosis of stroke and TIA has been proposed. The proposed definition of TIA is a transient episode of neurologic dysfunction caused by focal brain, spinal cord, or retinal ischemia without acute infarction. A patient who has symptoms lasting less than 24 hours but who has an abnormality on DWI would be considered to have had an ischemic stroke.

Cerebral ischemia may result from several mechanisms, including hypoperfusion, thrombosis, and embolism. Hypoperfusion may cause either global deficits or focal deficits. Watershed or border zone infarctions often result from hypoperfusion,

often in the clinical setting of an arterial stenosis. Thrombosis often occurs at the site of a vessel stenosis or plaque rupture. Embolism may result from a cardiac embolus (thrombus, myxomatous emboli, or vegetation), from artery-to-artery emboli, or, rarely, from air, fat, or amniotic fluid.

Causes of arterial cerebral ischemia can be classified broadly into 6 categories: cardioembolic disease; large vessel extracranial disease (aorta, carotid, and vertebral arteries); large-vessel intracranial disease (intracranial internal carotid, middle cerebral, anterior cerebral, vertebrobasilar, and posterior cerebral arteries); small-vessel disease; abnormalities intrinsic to the blood (eg, prothrombotic disorders); and other diseases. Despite patients undergoing thorough evaluations, the cause of 20% to 30% of ischemic stroke is cryptogenic (ie, without a defined cause). Cryptogenic stroke has been hypothesized to be due to nonstenotic plaque rupture, paroxysmal atrial fibrillation, underlying malignancies, or undefined coagulation disorders. Embolic stroke of undetermined source, a subset of cryptogenic stroke, has a defined minimum evaluation required before one concludes there is not an identifiable cause. Cardioembolic disease is most prevalent (30%-40%), followed by lacunar disease (20%-30%), cryptogenic disease (20%-30%), large-vessel disease (10%-15%), and coagulation disorders (<5%). It is important to keep in mind that certain conditions may mimic cerebral ischemia. The proposed definition of TIA is a transient episode of neurologic dysfunction caused by focal brain, spinal cord, or retinal ischemia without acute infarction. A patient who has symptoms lasting <24 hours but who has an abnormality on DWI would be considered to have had an ischemic stroke. Common mimics of TIA include focal seizures, migraine equivalents, and metabolic disorders. The prevalent etiologic factor of stroke is cardioembolic; however, up to 30% of strokes are cryptogenic.

The term embolic stroke of undetermined source helps further define subsets of patients with cryptogenic stroke and defines the minimum workup to diagnose this type of stroke.

Evaluation of a patient with suspected arterial cerebral ischemia should begin with an evaluation to identify whether the patient is a candidate for intervention (intravenous tissue plasminogen activator, intra-arterial thrombolytics, or

mechanical thrombectomy). The National Institutes of Health Stroke Scale score (often calculated at hospital admission) helps determine the risk of hemorrhage with intravenous tissue plasminogen activator therapy and predicts the outcome after cerebral ischemia.

After acute decisions are made, the patient is often hospitalized. Thereafter, goals include 1) identify the cause of cerebral ischemia and select the appropriate antithrombotic (antiplatelet agent vs anticoagulant), 2) identify and treat modifiable risk factors, 3) monitor for stroke-related complications, and 4) initiate rehabilitation. The diagnostic workup is aimed at identification of the potential cause of cerebral ischemia. Hence, the appropriate antithrombotic (antiplatelet agent vs anticoagulant) for secondary prevention can be selected. In addition, identification of the cause—for example, a symptomatic high-grade carotid stenosis—may identify patients who require surgical or endovascular treatment. The second goal prompts modification of the contributing risk factors. The third goal is to identify and prevent complications related to cerebral ischemia (aspiration pneumonia, urinary tract infections, deep vein thrombosis or pulmonary embolism, myocardial infarction, arrhythmias, and depression).

A general approach to stroke evaluation is based on the pretest probability (ie, prevalence) of causes in addition to the identification of causes that change management. The approach varies depending on other factors that influence the pretest probability of individual causes collected in the initial history, physical examination, laboratory studies, and imaging. Additional recommendations for diagnostic evaluation are noted in individual sections below. Computed tomography (CT) and magnetic resonance imaging (MRI) are common tools used in the evaluation of cerebral ischemia. CT of the head should be performed for all patients who present with suspected stroke. This CT aids in the distinction between hemorrhage and ischemia. Although it may be negative for stroke in the first 24 hours, the early signs of ischemia include sulcal effacement, loss of the gray-white junction, and a hyperdense artery (eg, basilar or middle cerebral artery). The most useful brain MRI sequences for cerebral ischemia are fluid-attenuated inversion recovery (FLAIR), DWI, and the apparent diffusion coefficient (ADC) map. DWI measures the random diffusion of water molecules. Random diffusion of water is

relatively free in the extracellular space and more restricted in the intracellular space. When cytotoxic edema forms early in cerebral ischemia, water shifts from the extracellular space to the intracellular space, and diffusion is restricted. In general, the DWI signal is bright and the ADC map is dark in acute cerebral ischemia. These sequences both normalize over 2 to 3 weeks; thus, DWI is useful in determining the lesion acuity, confirming clinical localization, and confirming the diagnosis and tissue damage. The DWI signal must be evaluated with the ADC map and the clinical history, since a DWI signal abnormality can also be present with acute demyelinating lesions, neoplasms, infection, Creutzfeldt-Jakob disease, and other disease process.

Approximately 30% to 40% of ischemic strokes or TIAs are due to a cardioembolic source; thus, the pretest probability for any patient is high. Major cardioembolic sources are those for which the medical literature clearly shows a causal relationship with cerebral ischemia and for which clinical trials of treatment exist. Minor cardioembolic sources are those for which the medical literature presents controversial or inconclusive findings related to the relationship to cerebral ischemia. Cardiac examination, chest radiography, and electrocardiography are recommended for all patients with cerebral ischemia. Selected patients may benefit from echocardiography, extended cardiac monitoring (Holter, event recorder, mobile cardiac outpatient telemetry, or insertable cardiac monitoring device), or blood cultures. Suspicions of cardiac disease that would warrant such tests include multiple arterial territory ischemic strokes, no evidence of large- or small-vessel disease as a cause, palpitations, large left atrium, mitral valve disease, obstructive sleep apnea, and history of cardiac disease. Cardiac CT and MRI are evolving and may be alternative tests to echocardiography.

Atrial Fibrillation. Nonvalvular atrial fibrillation is one of the most common causes of cerebral ischemia. Atrial fibrillation affects 1% of the general US population but 10% of persons older than 75 years. Both paroxysmal fibrillation and chronic atrial fibrillation are risk factors for ischemia, but paroxysmal atrial fibrillation is often difficult to prove. Factors that increase the risk of thromboembolism in the presence of atrial fibrillation include age older than 75 years, hypertension, diabetes mellitus, history of thromboembolism, and congestive heart failure. The CHADS2

(congestive heart failure, hypertension, age ≥75 years, diabetes mellitus, and prior stroke or TIA) score and the CHA2 DS2 -VASc (congestive heart failure, hypertension, age 65-74 years, age ≥75 years, diabetes mellitus, prior stroke or TIA, female sex, and vascular disease) score can be used to predict the risk of recurrent thromboembolism. The risk may be as high as 10% to 12% per year for patients with multiple risk factors. Paroxysmal atrial fibrillation can be missed with 24- hour telemetry or Holter monitoring. Up to 15% of patients with cryptogenic stroke may have paroxysmal atrial fibrillation as a cause of their stroke. Extended cardiac monitoring (surface monitoring or implantable device) for at least 30 days should be considered for patients with cryptogenic stroke. Ongoing research is evaluating echocardiographic, serum, and electrocardiographic biomarkers that would predict atrial fibrillation or atrial cardiopathy without extended monitoring. Warfarin (international normalized ratio goal, 2-3) is superior to aspirin and superior to clopidogrel plus aspirin for prevention of thromboembolism due to atrial fibrillation. Four novel anticoagulants—dabigatran (a direct thrombin inhibitor), rivaroxaban, apixaban, and edoxaban (factor Xa inhibitors)—have been compared with warfarin and are alternatives to warfarin for nonvalvular atrial fibrillation. The Watchman Left Atrial Appendage Closure Device (Boston Scientific Corp), is an alternative to long-term anticoagulation for patients with atrial fibrillation who are at high risk for intracranial or systemic bleeding when receiving anticoagulation. The Watchman occludes the left atrial appendage and thus prevents stroke.

All in all, an ischemic stroke results from a blockage in one of the arteries supplying blood to the brain. Early signs include a drooping face or mouth, speech slurring, mental confusion, and muscular weakness. A stroke is a medical emergency and getting care within 3 hours of the first symptoms improves the outcomes.

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