Acute Stroke and Transient Ischemic Attack in the Outpatient Clinic

Salvador Cruz-Flores, MD, MPH

KEYWORDS

• Stroke • TIA • Transient ischemic attack • Emergent evaluation • Risk stratification • Treatment

KEY POINTS

- Stroke and transient ischemic attack are time-critical, treatable, and preventable medical emergencies.
- Decisions on hospital admission and acute management require the establishment of accurate time of onset and/or time when the patient was last seen normal.
- A thorough workup aimed at establishing the cause is required to guide secondary prevention.
- Stroke patients must be cared for at centers with stroke expertise.
- Secondary stroke prevention targets the management of vascular risk factors, appropriate antithrombotic therapy including anticoagulation for those with absolute indication for anticoagulation (ie, atrial fibrillation), and carotid endarterectomy or carotid artery stenting for symptomatic significant carotid artery stenosis.

INTRODUCTION

Cerebrovascular disease is fourth leading cause of death and the leading cause of disability in the United States. In recent years, the incidence and mortality have declined. Stroke is categorized as ischemic (87%) and hemorrhagic (13%).[1,2] Ischemic stroke and transient ischemic attack (TIA) are 2 clinical ends of a common pathophysiologic mechanism, the occlusion of a cerebral artery. As in cardiovascular disease in general, vascular risk factors, such as diabetes mellitus, hypertension, smoking, and hyperlipidemia, play an important role. Compared with acute coronary syndromes in which the vascular occlusion in most cases is local atherothrombosis, ischemic stroke and TIA have a heterogeneous cause, with 4 main subtypes explaining most cases, namely, large vessel atherothrombosis, cardioembolic, lacuna, and cryptogenic.[1,2] In addition, there is a smaller group of uncommon causes such as

Department of Neurology, Paul L. Foster School of Medicine, Texas Tech University Health Sciences Center El Paso, 4800 Alberta Avenue, Room 108, El Paso, TX 79905, USA
E-mail address: Salvador.cruz-flores@ttuhsc.edu

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arterial dissection or prothrombotic states that should be considered in specific circumstances such as in stroke in young patients or after trauma. The causal heterogeneity is an important consideration in deciding diagnostic workup, and therapy for vascular studies is necessary to find carotid stenosis amenable to revascularization; in contrast, a patient with atrial fibrillation may need anticoagulation. Two or more potential causes of stroke may coexist in a patient and all will require treatment.[3]

In recent years, the definitions of ischemic stroke and TIA have changed from a time-based diagnosis (symptom duration >24 vs <24 hours duration for stroke vs TIA) to a diagnosis based on the sine qua non criterion: the presence of an infarct pathologically or by imaging in stroke and its absence in a TIA, regardless of the duration of the symptoms.[4,5] Recent decades have seen an accumulation of knowledge on the natural history and treatment of ischemic stroke and TIA enough to impact the diagnostic and therapeutic paradigm, making these time-critical conditions highly preventable and treatable, and therefore, true medical emergencies that require an expedited approach. This article reviews current concepts on pathophysiology, clinical presentation, diagnosis, and treatment of TIA and ischemic stroke that present in the office.

CLINICAL HISTORY AND EXAMINATION

The patient history of stroke or TIA will include symptoms that indicate a focal neurologic deficit of sudden onset. Focal deficit implies the dysfunction of a discrete area of the brain leading to symptoms and signs that can be located to the area affected (**Box 1**).

Typical symptoms include speech impairment referred by patients as either slurred speech and/or word finding impairment, visual loss in one side, double vision, facial weakness or facial droop, altered mental status, limb weakness, sensory symptoms, and incoordination. Seizures and headache may occur in less than 10% to 15% of patients.[6]

Tips for the bedside evaluation:

- Dysarthria is a frequent sign of stroke, although it has poor localization value.
- Dizziness in isolation is not a common symptom of stroke.
- The combination of headache, dizziness, nausea/vomiting, and difficulty walking is a common presentation of cerebellar infarct even in absence of focal neurologic findings.
- Aphasia and neglect/inattention indicate a cortical lesion in the dominant versus nondominant hemisphere, respectively
- Gaze deviation away from the hemiparesis indicates a large hemispheric infarct in the side toward where the eyes are looking to.
- Gaze deviation toward the hemiparesis indicates a brainstem lesion.
- In trying to localize the lesion, consider that cortical hemispheric lesions will have a gradient in the weakness depending on the vascular distribution affected such that:
  - Middle cerebral artery territory infarcts result in greater weakness in the face and arm compared with the leg.
  - Anterior cerebral artery territory infarcts result in greater weakness in the leg.
- Weakness with no gradient (similar degree in arm and leg) indicates a subcortical lesion (ie, internal capsule as in lacunar infarcts).
- The more posterior the lesion is in the hemisphere, the more sensory and visual symptoms.
- Changes in the level of consciousness, gaze deviation, aphasia, neglect, and weakness (hemiparesis or quadriparesis) indicate the presence of large vessel