DIAGNOSIS OF STROKE AND STROKE MIMICS IN THE EMERGENCY SETTING

Kevin M. Barrett, Joshua M. Levine, Karen C. Johnston

ABSTRACT

Patients with suspected stroke require urgent evaluation in order to identify those who may be eligible for time-sensitive therapies. A focused and systematic approach to diagnosis improves the likelihood of identifying patients with probable ischemic stroke and minimizes the chances of exposing patients with alternate diagnoses to potentially harmful treatment. This chapter emphasizes the historical, examination, and neuroimaging findings useful in the rapid evaluation and diagnosis of patients with suspected ischemic stroke. Other entities that may present with strokelike symptoms will also be discussed.


INTRODUCTION

Stroke typically presents with the sudden onset of focal neurologic deficits. Appropriate delivery of acute stroke therapies depends on accurately establishing the time of symptom onset, performing a focused bedside assessment, and rapidly interpreting ancillary tests. This chapter emphasizes a systematic diagnostic approach that will facilitate expeditious identification of patients eligible for acute therapies. By necessity, the discussion that follows is presented sequentially. However, it is important to recognize that many elements of the stroke evaluation occur in parallel, depending on resource availability or clinical circumstances. Specific evidence-based stroke therapies are covered in a subsequent chapter.

BEDSIDE ASSESSMENT

History

The ability to treat acute ischemic stroke patients with IV thrombolysis or endovascular therapies depends on accurately establishing the time of symptom onset. Under ideal circumstances, the patient is able to provide a

Note: Text referenced in the Quintessentials Preferred Responses, which appear later in this issue, is indicated in yellow shading throughout this chapter.

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detailed account of symptom onset. The history should focus on eliciting information that will establish eligibility for thrombolytic therapy or identify potential exclusionary conditions. Situations may arise in which the precise time of symptom onset may be difficult to establish. The use of cues (eg, “before or after lunch?” or “before or after the evening news?”) may be helpful in generating an estimated time of onset. If family or friends are present, it is wise to corroborate the patient’s report of symptom onset with eyewitnesses. In some circumstances, a precise time of symptom onset will prove impossible to determine, and the line of questioning should then shift to identifying when the patient was last neurologically normal. For those patients who awaken with symptoms, the time of onset becomes the time at which they went to bed (assuming they were normal at that time). For patients with heralding symptoms or TIA, it is necessary to ensure complete resolution before the clock can be “reset.”

The nature of symptom onset should be obtained when possible. Symptoms that begin abruptly suggest a vascular etiology, whereas symptoms that begin in one region and gradually spread to involve other areas may support an alternate etiology (ie, migraine). Inquiry should be made regarding risk factors for vascular disease, as well as any history of seizures, migraine, insulin use, or drug abuse that may support another cause for the patient’s symptoms. Information necessary for decisions regarding thrombolysis must be obtained in a structured fashion to minimize the possibility of overlooking critical information (Table 1-1). Accompanying symptoms, particularly headache, warrant further exploration. Ictal, or so-called thunderclap headache, should alert the clinician to the possibility of subarachnoid hemorrhage. Small bleeds or sentinel leaks from unruptured intracranial aneurysms may not be evident on CT scan. Further evaluation with lumbar puncture to exclude the presence of blood in the subarachnoid space may be warranted (Edlow and Caplan, 2000).

The initial evaluation of the potential stroke patient often occurs in a high-acuity area. Medical personnel responsible for establishing IV access, initiating cardiorespiratory monitoring, performing blood draws, and performing electrocardiography compete for the patient’s attention. Additionally, the presence of aphasia or neglect may limit the patient’s ability to provide accurate

**TABLE 1-1 Important Historical Information in the Suspected Stroke Patient**

- **History of the Present Illness**
  - Time of symptom onset
  - Evolution of symptoms
  - Convulsion or loss of consciousness at onset
  - Headache
  - Chest pain at onset

- **Medical History**
  - Prior intracerebral hemorrhage
  - Recent stroke
  - Recent head trauma or loss of consciousness
  - Recent myocardial infarction

- **Surgical History**
  - Recent surgical procedures
  - Arterial puncture

- **Review of Systems**
  - Gastrointestinal or genitourinary bleeding

- **Medications**
  - Anticoagulant therapy
information. To the physician performing the initial assessment, these activities pose significant challenges.

Despite such barriers, critical elements of the history may be obtained indirectly. Emergency medical personnel provide important information regarding vital signs and blood glucose levels obtained in the field. Observations regarding level of consciousness, initial severity of deficits, and the presence of bowel or bladder incontinence at the scene provide useful clues to the etiology of the presenting symptoms. Family members provide important observations and additional history. Reaching a family member by telephone may be necessary if no one is immediately available. In certain circumstances, attempting to reach the patient’s primary care provider may prove useful. Documentation of prior stroke, TIA, or other neurologic morbidity in medical records is valuable. Chronic or previously resolved deficits may potentially confound interpretation of neurologic examination findings in the acute setting. The presence of advanced dementia or other neurodegenerative process may influence the decision to pursue further aggressive interventions. Examination of medication bottles, if they accompany the patient, may provide clues to coexisting medical conditions or anticoagulant use.

**Neurologic Examination**

The examination should focus on identifying signs of lateralized hemispheric or brainstem dysfunction consistent with focal cerebral ischemia. Commonly encountered ischemic stroke syndromes are outlined in **Table 1-2**. The location of vascular occlusion and

<table>
<thead>
<tr>
<th>Vascular Territory</th>
<th>Signs and Symptoms</th>
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<tbody>
<tr>
<td>Left middle cerebral artery distribution</td>
<td>Aphasia, right hemiparesis/hemisensory disturbance, right homonymous hemianopia, left head and gaze preference</td>
</tr>
<tr>
<td>Right middle cerebral artery distribution</td>
<td>Left hemispatial neglect, left hemiparesis/hemisensory disturbance, left homonymous hemianopia, right head and gaze preference, anosognosia</td>
</tr>
<tr>
<td>Left posterior cerebral artery distribution</td>
<td>Right visual field defect, impaired reading with intact writing (alexia without agraphia), poor color naming, right hemisensory disturbance</td>
</tr>
<tr>
<td>Right posterior cerebral artery distribution</td>
<td>Left visual field defect, visual neglect, left hemisensory disturbance</td>
</tr>
<tr>
<td>Vertebrobasilar distribution</td>
<td>Dizziness, vertigo, nausea, diplopia, quadripareisis, crossed motor or sensory findings (ipsilateral face, contralateral body), truncal or limb ataxia, visual loss/dimming, impaired consciousness</td>
</tr>
<tr>
<td>Penetrating artery distribution (ie, lacunar syndromes)</td>
<td>(A, B) Contralateral hemiparesis alone (pure motor stroke) OR contralateral hemiparesis + ataxia out of proportion to weakness (ataxic-hemiparesis); no cortical signs</td>
</tr>
<tr>
<td>(A) Internal capsule/corona radiata</td>
<td>(C) Contralateral sensory loss alone (pure sensory stroke); no cortical signs</td>
</tr>
<tr>
<td>(B) Ventral pons</td>
<td></td>
</tr>
<tr>
<td>(C) Thalamus</td>
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</tbody>
</table>

**TABLE 1-2** Common Ischemic Stroke Syndromes

CORTICAL SIGNS VS NO CORTICAL SIGNS = KEY!
The extent of collateral flow dictate whether the complete or partial syndrome is present. Frequently, important examination findings are observed while obtaining the history. Thus, level of consciousness and the presence of a gaze deviation, aphasia, neglect, or hemiparesis, may be established within minutes of the initial encounter. The NIH Stroke Scale (NIHSS) is a validated 15-item scale that is used to assess key components of the standard neurologic examination and measure stroke severity (Lyden et al, 1999). Although initially designed to measure clinical differences in experimental stroke therapy trials (Brott et al, 1989), the NIHSS has gained widespread acceptance as a standard clinical assessment tool. The scale assesses level of consciousness, ocular motility, facial and limb strength, sensory function, coordination, language, speech, and attention. Scores range from 0 (normal) to 42 (maximal score) (Table 1-3). The NIHSS may be performed rapidly and predicts short-term and long-term neurologic outcomes (Adams et al, 1999).

Ancillary Testing
Laboratory and cardiac evaluation supplement the clinical impression derived from the bedside assessment. Some conditions that may present with stroke-like symptoms may be identified based on laboratory results (eg,
hypoglycemia). In addition, abnormal laboratory values may exclude patients from receiving thrombolytic therapy. Recently published guidelines recommend routine laboratory testing of blood glucose, electrolytes, complete blood count, prothrombin time, activated partial thromboplastin time, international normalized ratio, and renal function (Adams et al, 2007). Testing for stool guaiac is not routinely recommended unless an indication exists (eg, melena or hematochezia). Initiating treatment with IV recombinant tissue-type plasminogen activator (rt-PA) prior to obtaining results of coagulation studies may be safe and feasible (Sattin et al, 2006). In patients otherwise eligible for thrombolytic therapy, the American Heart Association/American Stroke Association (AHA/ASA) guidelines support the decision to initiate treatment prior to results of platelet or coagulation studies, unless a bleeding disorder or thrombocytopenia is suspected.

Cardiac abnormalities are common in patients with stroke. Cardiac enzymes and a 12-lead EKG are recommended for all stroke patients. Myocardial infarction and atrial fibrillation are common causes of cardioembolism and are readily identified in the acute setting. The utility of routine chest radiography as part of the acute stroke evaluation is limited (Sagar et al, 1996) and currently not routinely recommended. As discussed previously, unless warranted by the presence of sudden, severe headache, there is no role for routine CSF examination. Urine toxicology screen, blood alcohol level, arterial blood gas, or pregnancy tests may be indicated when the clinical history is limited or uncertain.

**NEUROIMAGING**

Brain imaging is the only reliable means to differentiate between ischemic and
hemorrhagic stroke and is therefore mandatory prior to thrombolytic therapy (Besson et al, 1995; Mader and Mandel, 1998). This chapter will cover the basics of standard CT and MRI used in the emergency setting. More advanced vascular and physiologic imaging will be discussed in another chapter.

CT

Noncontrast head CT is the study most readily available in most stroke centers. CT is sensitive to intracranial blood and may be rapidly performed as part of the acute stroke evaluation. CT is also inexpensive and less susceptible than MRI to artifact introduced by patient movement. In the acute setting, early ischemic change (EIC) may be apparent on CT. Loss of differentiation of the gray-white matter interface, particularly in the region of the insular cortex or the lentiform nucleus, may be indicative of early cerebral ischemia. Sulcal effacement, representing focal tissue edema, may be appreciated in areas of relative hypoperfusion and may be another early indicator of ischemia. Whether these changes are present in the minutes to hours after symptom onset is probably related to the severity and extent of ischemia, collateral circulation, and presence of large vessel occlusion. Detection of EIC is variable (Grotta et al, 1999) and is likely related to reader experience. One study identified EICs in 75% of patients presenting within 3 hours of symptom onset (Barber et al, 2000), and an even higher prevalence was observed within 6 hours in patients with hemispheric strokes (von Kummer et al, 1996). The presence of EICs involving greater than one-third of the middle cerebral artery (MCA) territory was used as an exclusion criterion in early clinical trials of thrombolytics in an effort to minimize the risk of hemorrhagic complications (Hacke et al, 1995).

The presence of EICs, however, was not independently associated with adverse outcome after rt-PA treatment in the National Institute of Neurological Disorders and Stroke (NINDS) trial and therefore should not preclude thrombolytic therapy in otherwise eligible patients (Patel et al, 2001).

Increased CT attenuation within an arterial segment, the “hyperdense” artery sign, is an occasional finding. It is observed most commonly in the MCA and is associated with occlusive thrombus within the vessel lumen (Tomsick et al, 1989) (Figure 1-1). Although the hyperdense MCA sign is fairly specific for vascular occlusion (Bastianello et al, 1991), the sensitivity

Table 1-3 Continued

<table>
<thead>
<tr>
<th>Category</th>
<th>Scale Definition</th>
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<tbody>
<tr>
<td>8. Sensory</td>
<td>0 = Normal</td>
</tr>
<tr>
<td></td>
<td>1 = Mild loss</td>
</tr>
<tr>
<td></td>
<td>2 = Severe loss</td>
</tr>
<tr>
<td>9. Language</td>
<td>0 = Normal</td>
</tr>
<tr>
<td></td>
<td>1 = Mild aphasia</td>
</tr>
<tr>
<td></td>
<td>2 = Severe aphasia</td>
</tr>
<tr>
<td></td>
<td>3 = Mute or global aphasia</td>
</tr>
<tr>
<td>10. Dysarthria</td>
<td>0 = Normal</td>
</tr>
<tr>
<td></td>
<td>1 = Mild</td>
</tr>
<tr>
<td></td>
<td>2 = Severe</td>
</tr>
<tr>
<td>11. Extinction/inattention</td>
<td>0 = Normal</td>
</tr>
<tr>
<td></td>
<td>1 = Mild</td>
</tr>
<tr>
<td></td>
<td>2 = Severe</td>
</tr>
</tbody>
</table>


of this finding is low. The MCA “dot sign” seen in the sylvian fissure with occlusion of distal MCA branches (Barber et al, 2001) and the hyperdense basilar artery sign in patients with basilar artery thrombosis (Ehsan et al, 1994) have also been described. A recent systematic review of studies reporting on early CT signs in acute ischemic stroke found interobserver agreement to be moderate to poor and increased likelihood of poor functional outcome when early infarction signs were present (Wardlaw and Meilke, 2005). An example of early ischemic changes is demonstrated in Case 1-1.

The appearance of ischemic changes on CT evolves over time. Within 12 to 24 hours, an indistinct area of low density becomes apparent in the affected vascular distribution. After 24 hours, the ischemic region becomes increasingly hypodense and better circumscribed. Mass effect develops and results in sulcal asymmetry or ventricular distortion. The presence of a clearly delineated area of hypodensity with associated mass effect should, therefore, prompt reassessment of the time of symptom onset in patients thought to be eligible for thrombolytic therapy, as distinct hypodensity is inconsistent with focal cerebral ischemia of less than 3 hours’ duration.

Neurologists involved in the evaluation of patients with acute stroke must be sufficiently skilled to identify radiographic contraindications to thrombolytic therapy. Generally speaking, a “central to peripheral” approach to visual inspection of CT images may help to rapidly identify nonischemic causes of strokelike symptoms and to identify subtle EIC. First, inspection of the midline structures (ie, ventricles and basal cisterns) for shift may help rapidly identify space-occupying mass lesions, which may require urgent surgical intervention. The basal cisterns,

![Noncontrast head CT from a 74-year-old woman with the abrupt onset of dysarthria and left-sided weakness. The hyperdense right middle cerebral artery (arrows) is visible on these axial images at two sequential levels (A, B). Follow-up cerebral angiography confirmed occlusion of the proximal right middle cerebral artery.](image)

**FIGURE 1-1**

**KEY POINTS**

- Brain imaging is the only reliable means to differentiate between ischemic and hemorrhagic stroke and is therefore mandatory prior to instituting thrombolytic therapy.
- CT is sensitive to intracranial blood, is widely available, and may be rapidly performed as part of the acute stroke evaluation.
- Loss of differentiation of the gray-white matter interface, particularly in the region of the insular cortex or the lentiform nucleus, may be indicative of early cerebral ischemia.
- Sulcal effacement, representing focal tissue edema, may be appreciated in areas of relative hypoperfusion and may be another early indicator of ischemia.
interhemispheric fissure, and sylvian fissures should be scrutinized for subarachnoid blood. The fourth ventricle should be identified, particularly in patients with posterior circulation symptoms. Although CT evaluation of the posterior fossa may be challenging because of technical limitations, distortion of the fourth ventricle may result from mass effect related to cerebellar infarction and often heralds obstructive hydrocephalus due to evolving edema. Examination of the periphery of the brain parenchyma for extraaxial collections should follow.

The brain parenchyma should approximate the inner table of the skull in a fairly symmetric fashion, acknowledging that atrophy may occur in a regional distribution. As with ischemic changes, the appearance of subdural collections evolves with time and these may appear isodense to brain parenchyma. Finally, careful inspection for early ischemic changes (see above) may be guided by localizing information from the clinical examination. Effective and timely administration of acute stroke therapy relies on accurate bedside assessment and exclusion of intracranial

Case 1-1

A 48-year-old man was seen by the acute stroke intervention team after he had developed left-sided weakness. He was last known to be normal 2 hours earlier. The patient reported mild headache but otherwise denied any problems.

Neurologic examination demonstrated a right head and gaze preference, left homonymous hemianopsia, left lower facial weakness, dysarthria, and dense left hemiparesis with absent sensation in the left arm and leg. The patient did not acknowledge the presence of his left-sided weakness. His NIHSS score was 14. Noncontrast head CT demonstrated a large area of subtle hypodensity with loss of the gray-white interface in the insular ribbon (white arrow) consistent with right MCA distribution ischemia. Sulcal effacement and blurring of the deep nuclei (arrowhead) are also seen (Figure 1-2).

Comment. This case exemplifies early ischemic changes that may be seen with CT in the early hours after stroke onset. The clinical syndrome fit well with a nondominant hemisphere ischemic event, and the early ischemic changes appreciable on noncontrast CT helped confirm the clinical assessment. The patient underwent emergent vascular imaging and was found to have a right internal carotid artery occlusion.

FIGURE 1-2

Increased CT attenuation within an arterial segment, the “hyperdense” artery sign, is an occasional finding in acute ischemic stroke.

The appearance of ischemic changes on CT evolves over time.
hemorrhage. Subtle changes of early ischemia, while helpful in confirming clinical suspicion, are often identified with the assistance of a radiologist and should not directly influence therapeutic decisions.

**MRI**

Evaluation of patients with acute stroke with MRI has clear advantages. Compared with CT, MRI with diffusion-weighted imaging (DWI) sequences is more sensitive for acute cerebral ischemia and improves diagnostic accuracy (Fiebach et al, 2002). DWI may detect abnormalities within minutes after onset of cerebral ischemia (Hjort et al, 2005) and delineates the location, size, and extent of hyperacute ischemia. MRI better evaluates the posterior fossa and improves visualization of small cortical infarctions (Figure 1-3).

Historical concerns about the ability of MRI to identify acute intracerebral hemorrhage have been addressed by several studies. Conventional T1-weighted and T2-weighted MRI pulse sequences are able to identify subacute and chronic blood, but they are less sensitive for parenchymal hemorrhage during the first 6 hours after symptom onset. Susceptibility-weighted MRI, or gradient-recalled echo (GRE), sequences have improved sensitivity for recently extravasated blood products (Patel et al, 1996). A prospective study of MRI and CT performed within 6 hours of stroke symptom onset demonstrated that the accuracy of GRE sequences for acute hemorrhage is equal to that of CT (Kidwell et al, 2004). A smaller multicenter study found a similarly high accuracy of GRE for identification of acute intracerebral hemorrhage (Fiebach et al, 2004).

Some centers have developed extensive experience with MRI in acute stroke and have adopted the use of MRI protocols for routine evaluation of patients with stroke. A recent prospective study performed at such a center demonstrated the superiority of MRI for detection of acute stroke in the full spectrum of patients who presented for emergency assessment of stroke-like symptoms (Chalela et al, 2007). Based on these results, the authors have advocated the use of MRI as the sole modality in the evaluation of patients with suspected stroke. The utility of MRI in detecting subarachnoid hemorrhage has not been rigorously evaluated, and only limited data are available (Wiesmann et al, 2002). The previous studies of MRI for the acute evaluation of stroke were performed using 1.5 tesla (T) magnets. Further studies will be needed to clarify the role of newer generation 3T MRI scanners for hyperacute stroke diagnosis.

**KEY POINTS**

- Diffusion-weighted imaging is more sensitive for detection of acute cerebral ischemia and improves diagnostic accuracy.
- A prospective study of MRI and CT performed within 6 hours of stroke symptom onset demonstrated that the accuracy of gradient-recalled echo sequences for acute hemorrhage is equal to that of CT.

**Figure 1-3** CT and MRI images from a 39-year-old man who developed sudden speech difficulty. The CT images (top row) demonstrate hypodensity in the posteroinferior left frontal (top left, white arrow) lobe and adjacent superior left temporal lobe as well as loss of gray-white junction and sulcal effacement in the posterior parietal region (top right, white arrow). MR diffusion-weighted images (bottom row) demonstrate the abnormalities clearly (bright signal).
The vast majority of emergency departments lack the resources necessary to perform emergent MRI. The costs of the technology, including around-the-clock technician support, are prohibitive for many centers and have limited widespread implementation. Abbreviated stroke MRI protocols have been developed to address concerns about additional time needed to acquire MRI images compared with CT. This issue is important given the association between time to initiation of thrombolytic therapy and likelihood of an excellent neurologic outcome (Marler et al, 2000). MRI is contraindicated in patients with pacemakers or other metallic hardware and is further limited by its susceptibility to motion artifact in agitated patients.

STROKE MIMICS

When acute ischemic stroke is suspected, it is crucial to consider and to exclude alternative diagnoses, especially intracranial hemorrhage. Many conditions, including systemic abnormalities and other nervous system diseases, present with focal neurologic deficits that “mimic” acute ischemic stroke. A recent prospective study of more than 300 patients who presented to an urban teaching hospital with suspected stroke found mimics in 31% at the time of final diagnosis. Early studies found the frequency of stroke mimics range from 1% (O’Brien et al, 1987) to 19% (Libman et al, 1995) in patients with suspected or initially diagnosed stroke. The time of patient assessment relative to symptom onset, examiner experience, and availability of imaging results at the time of initial diagnosis were variable in these studies. A recent prospective study of more than 300 patients who presented to an urban teaching hospital with suspected stroke found mimics in 31% at the time of final diagnosis (Hand et al, 2006). The most frequent mimics were postictal deficits (21%), sepsis (13%), and toxic-metabolic disturbances (11%). Seventy-five percent of mimics in the study were neurologic disorders, and 42% of patients with a mimic had experienced a previous stroke. Eight variables independently associated with a correct diagnosis were identified. The most powerful predictors of an accurate stroke diagnosis were “definite history of focal neurologic symptoms” (odds ratio [OR] 7.21; 95% confidence interval [CI], 2.48–20.93) and an NIHSS score greater than 10 (OR 7.23; 95% CI, 2.18–24.05). In patients with known cognitive impairment, the likelihood of having a stroke was markedly reduced (OR 0.53; 95% CI, 0.14–0.76). While studies such as these improve our general understanding of the
Case 1-2

An 82-year-old woman was seen by the acute stroke intervention team for the sudden onset of speech difficulty 90 minutes earlier. She had been working with a physical therapist at home when she became unable to speak. There was no associated weakness, alteration of consciousness, or headache. Per report, she had experienced a minor “stroke” approximately 2 weeks earlier but had made some improvement.

The woman was afebrile, her initial blood pressure was 142/72 mm Hg, and the finger-stick glucose level was 188 mg/dL. She was awake, alert, and appropriate. Language examination was remarkable for impaired fluency with the ability to say only fragments of words. She was able to follow simple midline commands but was unable to follow complex commands. She was unable to repeat, read, or name objects. There was no limb weakness or sensory disturbance. Her NIHSS score was 6.

Noncontrast head CT demonstrated a subtle hyperdense lesion with mass effect involving the left temporoparietal region (Figure 1-4, top, arrows). Given the radiographic findings suggestive of an underlying structural lesion, the patient did not receive thrombolytic therapy. Follow-up MRI demonstrated an ill-defined enhancing lesion involving the white matter and cortex of the left parietal lobe suggestive of a low-grade neoplasm (Figure 1-4, bottom, arrows).

Comment. This case is an example of a stroke mimic. The abrupt onset of symptoms might not prompt initial consideration of an underlying structural lesion as a potential etiology. However, one study found that 6% of patients with brain tumors presenting to an emergency department had symptoms of less than 1 day’s duration (Snyder et al, 1993). Sudden onset of focal symptoms in patients with either diagnosed or undiagnosed tumors may result from seizures, hemorrhage into the tumor, or obstructive hydrocephalus caused by increasing mass effect.
frequency and nature of stroke mimics, the results are less applicable to individual patients. As emphasized previously, the diagnosis of stroke is based on a composite of information obtained from the history and the pattern of findings on physical examination. A single symptom or sign cannot be used to rule in or rule out the diagnosis (Goldstein, 2006).

**STROKE CHAMELEONS**

The clinician should also be aware of common atypical stroke presentations. Recognition of these entities is important so as not to miss an opportunity to offer treatment to an otherwise eligible patient with stroke. These patients may not be triaged into acute stroke pathways and, therefore, may be at higher risk of misdiagnosis. The term *stroke chameleon* has been aptly used to describe an atypical stroke presentation that appears to mimic another disease process (Huff, 2002). The clinician should suspect such problems when symptom onset is abrupt or occurs in patients with risk factors for cerebrovascular disease.

A small proportion of patients with stroke may present with symptoms suggestive of an acute confusional state (eg, delirium). While encephalopathy typically reflects diffuse hemispheric dysfunction, a “pseudo-encephalopathy” may occur with focal cerebral ischemia involving the limbic cortex or orbitofrontal regions. “Confusion” may also be reported in patients with fluent aphasia or neglect syndromes without accompanying motor deficits. Systematic neurologic examination should identify these focal features and increase the clinical suspicion of stroke. Likewise, examination of visual fields will avoid overlooking patients with

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**TABLE 1-5  Characteristics of Common Stroke Mimics**

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Comments</th>
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<tbody>
<tr>
<td>Seizure (postictal)</td>
<td>Focal deficits likely are caused by seizure-induced neuronal dysfunction (reversible). May occur with simple partial or generalized seizures. Clinical seizure is often un witnessed or unrecognized. Spontaneous resolution occurs over hours (may last up to 48 hours).</td>
</tr>
<tr>
<td>Hypoglycemia</td>
<td>Aphasia or hemiplegia may be present. Variable drowsiness or obtundation. Blood glucose usually &lt;45 mg/dL. Resolution of symptoms (immediate—hours) with IV glucose.</td>
</tr>
<tr>
<td>Metabolic encephalopathy</td>
<td>Etiologies include hyperosmolar hyperglycemia, hyponatremia, and hepatic encephalopathy. May be associated with altered level of consciousness, poor attention, or disorientation (eg, delirium) asterixis.</td>
</tr>
<tr>
<td>Conversion reaction</td>
<td>Diagnosis of exclusion. Conversion disorder is the most common psychiatric diagnosis. Comorbid psychiatric problems are common. Paresis, paralysis, and movement disorders are common.</td>
</tr>
<tr>
<td>Reactivation of prior deficits</td>
<td>Imaging evidence or history of remote stroke is often apparent. Previous deficit may have resolved completely.</td>
</tr>
</tbody>
</table>
Cortical blindness or visual neglect syndromes.

Chest pain or discomfort mimicking myocardial ischemia has been reported in patients with infarction of the thalamus, corona radiata, or lateral medulla (Gorson et al., 1996). In some of these patients, the sensory symptoms were part of a more extensive stroke syndrome, but the clinician should be aware of this possibility. Distal arm paresis with patterns of weakness conforming to peripheral nerve distributions may result from focal cerebral ischemia. Radial or ulnar involvement has been reported with small cortical infarction of the motor cortex (Gass et al., 2001). Again, abrupt onset and the presence of vascular risk factors should alert the astute clinician.

CONCLUSION

Timely and accurate diagnosis of acute ischemic stroke in the emergency setting relies on eliciting a focused history, performing an efficient, thorough, neurologic examination, and interpreting the results of laboratory and neuroimaging studies. The sudden onset of focal neurologic symptoms in a recognizable arterial distribution is the hallmark of stroke. Accurately determining time of symptom onset, measuring the severity of neurologic deficit, and excluding nonischemic causes of strokelike symptoms may lead to identification of patients eligible for acute stroke therapy. An efficient and systematic approach to stroke diagnosis facilitates evidence-based treatment.

REFERENCES


