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Stroke in the Emergency Department

Stroke is the most common neurological emergency, and, because effective treatments are available that must be started within minutes, most acute neurological presentations should be assumed to be a stroke until proven otherwise by history, exam, or radiographic testing. Unfortunately, there is not a quick and easy laboratory or clinical test to determine for sure that the patient lying in front of you is having a stroke, so an accurate history and exam are essential.

Is This a Stroke?

DEFINITION

The term "stroke" usually refers either to a cerebral infarction or to a non-traumatic cerebral hemorrhage. Although it will vary depending on the population you are seeing (ethnicity, age, comorbidities), the ratio of infarcts to hemorrhages is about 4 to 1. As will be described in more detail in Chapter 3, cerebral infarcts can be caused by a number of pathological processes, but all end with an occlusion of a cerebral artery or vein. If the arterial occlusion results in a reduction of blood flow insufficient to cause death of tissue (infarction), it is termed "ischemia."

As will be described in more detail in Chapter 12, non-traumatic cerebral hemorrhages are caused by a number of pathological processes which all lead to bleeding into the brain parenchyma and ventricles. Bleeding into the subarachnoid space (Chapter 13) is usually caused by a ruptured aneurysm or vascular malformation. Other types of brain bleeding, for example into the subdural or epidural space, are usually traumatic and are not considered in this book.

PRESENTATION

When taking the history, the most characteristic aspect of a cerebral infarct or hemorrhage is the abrupt onset, so be sure to get the exact flavor of the onset. It is also imperative to determine as precisely as possible the time of onset. The symptoms most often stay the same or improve somewhat over the next few hours, but may worsen in a smooth or stuttering course. Ischemic strokes (but not hemorrhages) may rapidly resolve, but even if they resolve completely, they may recur after minutes to hours.
The second characteristic historical aspect of cerebral infarcts is that the symptoms will usually fit the distribution of a single vascular territory. This is also the most important characteristic of the neurological exam in a patient with an infarct. Therefore, patients with an infarct will present with symptoms and signs in the middle, anterior, or posterior cerebral arteries, a penetrating artery (producing a "lacunar" syndrome), or the vertebral or basilar artery (see below).

Parenchymal hemorrhages also occur in characteristic locations, and usually show the same symptom complex and signs as cerebral infarcts except that early decrease in level of consciousness, nausea and vomiting, headache, and accelerated hypertension are more common with hemorrhages.

Subarachnoid hemorrhages classically present as a bursting, very severe headache ("the worst headache of my life"), and are often accompanied by stiff neck, decreased consciousness, nausea, and

vomiting. Focal neurological signs are often absent; if present, they usually signify associated bleeding into the parenchyma.

Signs and symptoms characteristic of the various arterial territories are:

- **Middle cerebral** – contralateral loss of strength and sensation in the face, arm, and to a lesser extent leg. Aphasia if dominant hemisphere, neglect if non-dominant.
- **Anterior cerebral** – contralateral loss of strength and sensation in the leg and to a lesser extent arm.
- **Posterior cerebral** – contralateral visual-field deficit. Possibly confusion and aphasia if dominant hemisphere.
- **Penetrating** (lacunar syndrome) – contralateral weakness or sensory loss (usually not both) in face, arm, and leg. No aphasia, neglect, or visual loss. Possibly ataxia, dysarthria.
- **Vertebral** (or posterior inferior cerebellar) – ataxia, dysarthria, dysphagia, ipsilateral sensory loss on the face, and contralateral sensory loss below the neck.
- **Basilar** – various combinations of limb ataxia, dysarthria, dysphagia, facial and limb weakness and sensory loss (may be bilateral), pupillary asymmetry, disconjugate gaze, visual-field loss, decreased responsiveness.

DIAGNOSIS

There is currently no 100% sensitive and specific test for cerebral infarction in the emergency department, so the diagnosis is usually made on the basis of a characteristic history, exam, presence of comorbidities, and the absence of seizures or other stroke mimics. CT scanning is usually negative in the first 3 hours, or shows only subtle signs that have low interobserver reliability. If available, MRI, or detection of an occluded artery by transcranial Doppler or arteriography (by CT, MRI,

or intra-arterial catheterization), can be confirmatory. Parenchymal or subarachnoid hemorrhage, on the other hand, can be reliably detected by emergency CT scanning.

STROKE MIMICS

All of the following may present similarly to a stroke. In all cases, the distinction can be made by an emergent MRI scan, which will show an abnormal diffusion-weighted signal in most stroke cases, but not in mimics.

- **Seizures** – If a seizure has a focal onset in the brain, the patient may be left with weakness, numbness, or speech or vision problems for a period of time (usually less than 24 hours) after the seizure. Unlike the typical cerebral infarct, focal deficits after a seizure are often accompanied by lethargy and have a resolving course, but if the patient has had a seizure accompanying a stroke it is impossible to know for sure how much of the deficit the patient displays is due to each. This is why patients with seizures at onset are usually excluded from clinical trials of new stroke therapies.
- **Migraine** – Patients may have unilateral weakness or numbness, visual changes, or speech disturbances associated with a migraine headache (migraine with aura, previously called "complicated migraine"). Also, patients with migraine with aura are at higher risk for stroke. In trying to make the distinction between complicated migraine and stroke, it is important to remember that because of the high prevalence of both migraine and stroke in the general population, it is dangerous to attribute the patient's deficit to migraine just because the patient has a migraine history. The best rule of thumb is not to make the diagnosis of migraine with aura or migrainous stroke unless the patient has a history of previous migraine events similar to the deficit displayed in the emergency department.

- **Syncope** – This is usually due to hypotension or a cardiac arrhythmia. Stroke rarely presents with syncope alone. Patients with vertebrobasilar insufficiency may have syncope, but there are usually other brainstem or cerebellar findings if syncope is part of the stroke presentation.
- **Hypoglycemia** – Patients with low blood sugar may have symptoms that exactly mimic a stroke. The important thing is to check the blood sugar and, if it is low, correct it. If the symptoms do not resolve with correction of the hypoglycemia, the symptoms are probably from a stroke.
- **Metabolic encephalopathy** – Patients may have confusion, slurred speech, or rarely aphasia with this condition. They usually do not have other prominent focal findings.
- **Drug overdose** – Similar to metabolic encephalopathy.
- **Central nervous system tumor** – The location of the tumor would determine the type of signs and symptoms seen. A tumor, unlike a stroke, usually does not present with sudden focal findings, unless accompanied by a seizure (see above).
- **Herpes simplex encephalitis (HSE)** – This infection tends predominantly to affect the temporal lobes, so patients may have signs of aphasia, hemiparesis, or visual-field cuts. Onset can be rapid, and in its early stages it may mimic a stroke, but fever, CSF pleocytosis, seizures, and decreased level of consciousness are more prominent with HSE.
- **Subdural hematoma** – Depending on the location, this may cause contralateral weakness or numbness that may mimic a stroke. A CT scan can make this diagnosis, but the subdural hematoma, if small, may be subtle.
- **Peripheral compression neuropathy** – This may cause weakness or numbness in a particular peripheral nerve distribution, and it is usually not sudden in onset.
- **Bell's palsy (peripheral seventh nerve palsy)** – The important point here is that the forehead and eye closure are weak on the same side. One can have a stroke involving the pons and produce a peripheral seventh

nerve palsy, but usually there are other signs and symptoms such as weakness, a gaze palsy, or ipsilateral sixth nerve palsy.

- **Benign paroxysmal positional vertigo (BPPV)** – This may cause vertigo, nausea, vomiting, and a sense of imbalance, usually with turning of the head in one direction. This characteristic syndrome is due to labyrinthine dysfunction and not stroke. However, as with syncope, the presence of any brainstem or cerebellar signs should alert one to the possibility of a stroke.
- **Conversion disorder** – Patients may develop neurological signs or symptoms of weakness, numbness, or trouble talking that are manifestations of stress or a psychiatric illness. Always assume that your patient has a true neurological illness first.
- **Stroke recrudescence** – Worsening of pre-existing neurological deficit, usually due to an intercurrent toxic, metabolic, or infectious process (see Chapter 7).

What Type of Stroke?

As discussed previously, there are two main types of stroke, ischemic and hemorrhagic. The majority of this book describes the approach to either type of stroke, but there are specific chapters on ischemic stroke (Chapter 3), TIA (Chapter 9), ICH (Chapter 12), and SAH (Chapter 13).