CHAPTER 36

EXAMINATION OF THE PATIENT WITH CEREBROVASCULAR DISEASE

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The goal of the examination of a patient suspected of having a stroke is to gain immediate information about the probable size, location, and etiology of the stroke. Successful treatment depends on starting within a few hours after the onset. Brain imaging has advanced to allow detection of ischemia within minutes to hours after symptoms begin; imaging is necessary to identify hemorrhage before treatment is considered. Nevertheless, the examining physician has the responsibility to identify the symptoms and signs that guide subsequent therapy. For patients who arrive too late, beyond the time window for acute treatment, the neurologic examination is the first step in the diagnostic workup to establish stroke etiology and to start proper treatment aimed at preventing recurrence of stroke.

GENERAL EXAMINATION

Evaluation of the patient with a suspected stroke of large size must first address the level of consciousness and cardiopulmonary status. Irregular or labored breathing and a decreased level of consciousness, particularly if accompanied by gaze deviation, hemiparesis, or unequal pupils, may indicate the need for immediate intubation to treat impending herniation from massive infarction. Reduced alertness is a sign of either extensive hemispheral injury or involvement of the brainstem reticular activating system, which could result from brainstem infarction or from compression on the brainstem by the herniating uncus of the temporal lobe.

The terms “lethargic” and “stuporous” are often used to describe levels of decreasing consciousness, but it is most useful to describe alertness in terms of the minimal stimulus required for a given response (e.g., “opens eyes to voice” or “semipurposeful withdrawal to moderate noxious stimulus”). Subtler impairment of attention and concentration is tested by asking the patient to count backward from 20 to 1 or say the months of the year backward. The level of alertness may fluctuate after injury to the thalamus, often a hemorrhage. Coexisting metabolic derangement such as drug toxicity or hyperglycemia must be ruled out with appropriate laboratory tests. Papilledema is an additional sign of increased intracranial pressure. Cheyne-Stokes respirations with normal level of consciousness may be associated with a smaller territory infarction that involves the insula. Cardiac conduction defects, arrhythmias, subendothelial myocardial infarction, and neurogenic pulmonary edema may occur as a consequence of subarachnoid hemorrhage or large territory infarction, presumably from centrally mediated increase in sympathetic neurotransmitter release. The blood pressure rises acutely in 70% to 80% of stroke patients as a consequence of the infarction or hemorrhage and then returns to baseline spontaneously over the course of a few days. Except for malignant hypertension with encephalopathy or

hypertensive cerebral hematoma identified on brain computed tomography, blood pressure is not treated acutely. Nuchal rigidity is often present in subarachnoid hemorrhage. Fever may rarely be caused by brainstem infarction or subarachnoid hemorrhage. A systemic etiology must be sought and treated, however, because fever can exacerbate ischemic brain injury.

Beyond the examination required for emergency management of acute stroke, the general examination should focus on the cardiovascular system to seek a likely stroke etiology. Examination of the neck includes auscultation for carotid bruits, which result from turbulent flow in an artery narrowed by an atherosclerotic plaque. Auscultation of bruits may be misleading if the sound arises from stenosis of the unimportant external carotid artery or if the degree of stenosis in the internal carotid artery is great enough to dampen flow velocity below that which produces an audible bruit. Doppler ultrasound or magnetic resonance angiogram of the neck is needed if carotid stenosis is suspected. Fundoscopy can show retinal arterial narrowing, subhyloid hemorrhages, or cotton wool spots—systemic signs of hypertension or diabetes.

The presence of murmurs or arrhythmia on cardiac examination suggests valvular disease or atrial fibrillation, both independent risk factors and indications for anticoagulation therapy to prevent recurrence of stroke. The presence of fever with a cardiac murmur requires blood culture to rule out bacterial endocarditis. Auscultation of the lungs is important as a means of identifying signs of aspiration pneumonia or pulmonary edema caused by congestive heart failure.

NEUROLOGIC EVALUATION

The neurologic examination can provide valuable clues to the size, location, and etiology of the stroke. A few syndromes are predictive of specific stroke etiologies. For example, Wernicke aphasia, homonymous hemianopia, and the “top-of-the-basilar” syndrome of cortical blindness, agitation, and amnesia are nearly always caused by embolism from a proximal arterial or cardiac source. The Wallenberg syndrome, due to infarction of the dorsolateral medulla, is typically caused by thrombosis of the vertebral artery. The “lacunar syndromes” (see below) nearly always result from lipohyalinosis and fibrinoid necrosis of small penetrating arterioles arising from the middle cerebral artery stems, the basilar artery, or the first portion of the posterior cerebral arteries. The severity of the clinical syndrome at onset is highly correlated with the ultimate functional outcome. Level of alertness, ocular motility, motor power, and higher cerebral function are the keys to initial assessment. The most common sign of large stroke is the combination of gaze deviation, hemiparesis, and altered mentation. Hyperhydrosis, or excessive sweating, sometimes unilateral, may also occur in brainstem hemorrhage or large hemisphere stroke. In a comatose patient, asymmetry of tendon reflexes supports a diagnosis of unilateral brain injury when motor and sensory testing are not possible. Hypotonia may occur early after stroke, whereas tone often increases only after several days.

Even if fully alert, patients with gaze deviation and hemiparesis who are within the first several hours after stroke onset are at high risk for profound clinical worsening because of an edema-related mass effect that may peak as late as 3 to 5 days after stroke. Gaze palsies often occur with infarction involving the dorsolateral frontal lobes, producing gaze

deviation to the opposite side of the hemiparesis. Infarction of the lateral pons, on the other hand, produces hemiparesis on the same side as the direction of forced gaze. Other ocular dysmotility syndromes, including internuclear ophthalmoplegia, vertical gaze palsies, and nystagmus, may also occur with smaller infarcts in the brainstem. Visual fields should be tested by asking the patient to count fingers or identify a moving finger in each of four visual quadrants. Homonymous hemianopia may be the only sign of a large posterior cerebral artery territory infarction. An upper or lower homonymous quadrantanopia is produced by infarction involving the optic radiations hugging the lateral ventricular wall in the temporal or parietal lobe, respectively. A sectoranopia may be produced by injury to the lateral geniculate body of the thalamus due to anterior choroidal artery embolism.

Cortical involvement is suggested when aphasia or hemineglect are present. As a sign of dominant hemisphere injury, aphasia may involve abnormal naming, fluency, comprehension, repetition, reading, or writing. Dysfluency predominates with frontal lobe injury, whereas comprehension deficits predominate with posterior temporal and parietal injury. Severe Wernicke aphasia is characterized by poor auditory and reading comprehension and fluent speech littered with paraphasic errors; the syndrome may be misdiagnosed as delirium in the emergency room. Hemineglect, produced most often by nondominant parietal or frontal lobe injury, can be identified by unilateral extinction of visual or tactile stimuli when bilateral stimuli are presented. Rightward deviation on a line bisection test and failure to identify stimuli on the left side of an array of stimuli are also reliable signs of left hemineglect. Short-term verbal memory may be acutely affected by stroke when one or both anteromedial thalami or medial temporal lobes are involved. Short-term memory is easily tested by asking the patient to recall three objects after a

5-minute delay. Long-term memory impairment may appear as disorientation or dementia in a patient with multiple prior strokes in both hemispheres.

Unilateral weakness is typical of stroke. A complete characterization of the motor loss is important because the distribution and time course of weakness will differ by stroke location and etiology. Proximal and distal upper and lower limbs should be assessed independently on a five-point scale of power. Subtle weakness may be apparent only by the presence of a flattened nasolabial fold or widened palpebral fissure on one side of the face or a unilateral pronator drift when the patient is asked to hold the arms outstretched, palms up. Slowed or clumsy fine movements of one hand or ataxic dysmetria on finger-nose-finger may follow a stroke involving either the contralateral corticospinal tract or the ipsilateral cerebellum or cerebellar connections to the brainstem.

Weakness affecting the face, arm, and leg equally in a fully awake patient implies a small infarct in a deep brain region such as the posterior limb of the internal capsule, where the fibers of the corticospinal tract converge into a small anatomic area. This “pure motor hemiparesis” is one of four classic lacunar syndromes caused by small deep infarcts in the capsule, basal ganglia, thalamus, or pons. Hemicorporeal sensory loss is the syndrome associated with a thalamic lacune. “Clumsy hand dysarthria” or “ataxic hemiparesis” may result from lacunes in the corticospinal tract at any level from the corona radiata to the pons. “Fractionated hemiparesis,” for example facio-linguo-brachial paresis with little or no leg weakness, suggests cortical involvement of the perisylvian region due to embolic

occlusion of a branch of the middle cerebral artery. An even smaller middle cerebral artery branch occlusion may produce isolated hand weakness, mimicking an ulnar or median neuropathy. Weakness that affects the leg suggests a paramedian infarction due to embolic occlusion of the anterior cerebral artery. Arm and face weakness from anterior cerebral artery infarction may be due to motor neglect as a consequence of injury to the paramedian supplementary motor area and cingulate gyrus. Weakness of the proximal arm and leg sparing facial and lingual function is the most common result of “borderzone” ischemia due to hemodynamic failure from high-grade internal carotid artery stenosis. In patients with severe carotid occlusive disease, an attack of unilateral tremor or limb shaking may rarely be precipitated by standing, a sign that can be mistaken for focal seizure. Weakness due to hemodynamic failure from large vessel atherostenosis may fluctuate before becoming a fixed hemiparesis. Stroke syndromes due to an embolic arterial occlusion are usually maximal at onset. Weakness caused by lacunar disease may sometimes have a stuttering stepwise worsening course over several days.

SUGGESTED READINGS

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