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Avoiding Misdiagnosis in Patients With Posterior Circulation Ischemia: A Narrative Review

Kiersten L. Gurley, MD1,2,3, and Jonathan A. Edlow, MD1,2

ABSTRACT
Posterior circulation strokes represent 20% of all acute ischemic strokes. Posterior circulation stroke patients are misdiagnosed twice as often compared to those with anterior events. Misdiagnosed patients likely have worse outcomes than correctly diagnosed patients because they are at risk for complications of the initial stroke as well as recurrent events due to lack of secondary stroke prevention and failure to treat the underlying vascular pathology.

Understanding important anatomic variants, the clinical presentations, relevant physical examination findings, and the limitations of acute brain imaging may help reduce misdiagnosis. We present a symptom-based review of posterior circulation ischemia focusing on the subtler presentations with a brief discussion of basilar stroke, both of which can be missed by the emergency physician. Strategies to avoid misdiagnosis include establishing an abrupt onset of symptoms, awareness of the nonspecific presentations, consideration of basilar stroke in altered patients and using a modern approach to diagnosis of the acutely dizzy patient.

Posterior circulation strokes represent 20% of all ischemic strokes.1–4 Common posterior circulation stroke symptoms, including dizziness, clumsiness or imbalance, visual symptoms (diplopia, field cuts, or blurred vision), anisocoria, confusion and altered mental status, vomiting, headache and neck pain, problems with speech and swallowing, and decreased hearing,5 are less specific than typical anterior circulation stroke symptoms.6 The physical examination to detect them is more nuanced; the National Institutes of Health Stroke Scale (NIHSS) is weighted toward the anterior circulation7 and patients with posterior circulation strokes can have a NIHSS of zero with disabling deficits.8

These patients are misdiagnosed twice as often as those with anterior events. Misdiagnosed patients are at higher risk for complications of the initial stroke as well as recurrent events due to lack of secondary stroke prevention and lack of specific treatment of the underlying vascular pathology. Computed tomography (CT) of the brain, often the default brain imaging test in the emergency department (ED), has low sensitivity for posterior circulation infarction.9–11 Even magnetic resonance imaging (MRI) with diffusion-weighted imaging (DWI) is imperfect, especially in acute posterior circulation strokes.12

Strategies to avoid misdiagnosis include establishing an abrupt onset of symptoms, awareness of the nonspecific presentations, consideration of basilar stroke in altered patients, and using a modern approach to diagnosis of the acutely dizzy patient. We will emphasize the subtler, more easily missed presentations rather than the clinically overt ones.
DISCUSSION/OBSERVATIONS

Relevant Anatomy and Vascular Pathology

Classically, paired vertebral arteries arise from the subclavian arteries, ascend in the neck through the transverse foramina of the cervical vertebrae, and then fuse intracranially to form the basilar artery whose branches supply the posterior circulation and join the anterior circulation via the posterior communicating arteries (Figure 1 and Table 1). This classic anatomy is present in only 50% of individuals. Most variations are clinically insignificant, and often posterior strokes do not strictly follow a specific vascular territory. Some variations including the fetal posterior cerebral artery (f-PCA), the artery of Percheron, and vertebral artery hypoplasia (Figures 1A–1C) are frequently mentioned in radiology reports and may be clinically significant. The f-PCA arises from the anterior circulation so that ipsilateral carotid disease usually associated with anterior circulation symptoms can cause a posterior circulation stroke, affecting the interpretation of vascular imaging (Figures 2A–2C). The artery of Percheron is a single unilateral vessel that supplies both sides of the medial thalamus, ischemia of which results in a bilateral thalamic stroke with significant mental status, memory, psychiatric, speech, and ocular symptoms (Figure 2D). Vertebral artery hypoplasia is overrepresented in patients with posterior inferior cerebellar artery infarctions, which are usually ipsilateral to the hypoplastic vessel. The vascular pathology of posterior and anterior circulation strokes is the same. The proximal vertebral artery is a common location for atherosclerotic occlusive disease and a frequent source of artery-to-artery embolism. Cardioembolism is overrepresented as a cause of posterior circulation stroke compared to anterior circulation events making telemetry a useful adjunct (Figure 2E).

Although uncommon, vertebral artery dissection (VAD) is an important mechanism in posterior circulation strokes accounting for up to 25% of strokes in younger patients (Figures 2F and 2G). Trauma associated with VAD can be trivial or absent. The headache characteristics are variable; thunderclap headache occurs in only 9% of VAD patients; however, the pain is usually unique for the patient and different from prior headaches. In a consecutive series of 186 patients with first-ever VAD, 89% presented with cerebral ischemia and 11% presented with pain only. These proportions may be skewed (toward more ischemia) by referral bias; a systematic review of VAD reported that stroke occurred in 63% of cases. Importantly, this review reported that 25% of patients with VAD had no pain at any point in their course. Intracranial dissections are associated with subarachnoid hemorrhage (SAH). Horner’s syndrome is more common in carotid dissections (38%, from injury to the sympathetic fibers than run along the vessel) than in VAD (13% from a lateral medullary infarct). Unusual etiologies in posterior circulation ischemia are rotational vertebral artery compression (usually associated with a contralateral hypoplastic vertebral artery), vertebrobasilar dolichoectasia (a dilative arteriopathy associated with elongated, tortuous vessels), migraine-related stroke, reversible cerebral vasospasm syndrome, central nervous system vasculitis, and in elderly patients, giant cell arteritis.

Misdiagnosis

Diagnosis of posterior circulation strokes is often missed or delayed. Between 28 and 59% of cerebellar strokes are initially misdiagnosed. In one study of 465 stroke patients, those with posterior strokes were 2.5 times more likely to be missed compared to anterior circulation strokes (38% vs. 16%).

Clinical characteristics are often nonspecific and overlap with those of anterior stroke and stroke mimics. Patients endorse dizziness, altered cognition including psychiatric and memory-related symptoms, nausea and vomiting, headache, blurred vision, visual field abnormalities, dysphagia, hearing loss, clumsiness, and ataxia. In a study of 611 stroke patients, 61 (10%) were initially misdiagnosed. Factors associated with misdiagnosis included visual or gait disturbances, presentation of dizziness, sensory symptoms, and nausea. A recent meta-analysis of stroke misdiagnosis found that mild, nonspecific, and transient symptoms were all associated with misdiagnosis. A diagnostic trap is to not consider stroke in patients without lateralizing symptoms or signs, especially in younger patients. To avoid that trap, consider stroke as the likeliest explanation for the abrupt onset of any neurologic symptoms.

Of a consecutive series of 240 cerebellar stroke patients, 25 (10%) presented with isolated dizziness. Risk factors for misdiagnosis included young age, VAD as a cause, and a presentation of dizziness. Misdiagnosis of posterior circulation stroke is a needle-and-haystack phenomenon; the presenting symptoms are common in ED patients, most of whom are...
not having strokes. However, because these symptoms are so common, even the small fraction of misdiagnosed patients translates into a large absolute number. For patients presenting with dizziness alone, the number potentially harmed has been estimated at between 45,000 and 75,000 patients per year in the United States.

Although no prospective data exist, some findings suggest worse clinical outcomes in misdiagnosed patients compared to correctly diagnosed ones.

Figure 1 Posterior cerebral circulation anatomy. In the classic situation, paired vertebral arteries ascend in the neck and then fuse into the basilar artery. Prior to fusing, the vertebral arteries give off the PICA, which supply flow to the inferior cerebellum and lateral medulla. Dissection of the vertebral often cause ischemia to the PICA, causing cerebellar or lateral medullary strokes. The basilar artery gives off multiple small unnamed penetrating arteries that supply areas of the brain stem. The basilar also gives off the anterior inferior cerebellar artery and the superior cerebellar artery before terminating into the paired PCA, which supply parts of the thalamus and the visual cortex of the occipital lobes. Important variations on this classical anatomy are common. (A) Fetal PCA—The fetal PCA can be unilateral or bilateral, complete or partial. In its more complete form, it is present in roughly 10% of the population. A fetal PCA originates from the internal carotid artery, not the basilar. Data as to whether or not this increases stroke risk are conflicting. The important clinical point is that a carotid lesion can cause a posterior circulation stroke—for example, normally a left internal carotid stenosis would be unrelated to a left occipital stroke; however, with a fetal PCA, the same carotid lesion might be responsible for the same stroke, because it supplies the ipsilateral PCA. (B) Artery of Percheron—The artery of Percheron is present in approximately 5% to 10% of individuals. Normally, the first segment of each PCA supplies each side of the thalamus; with an artery of Percheron, a single unilateral branch supplies both sides of the thalamus. A stroke involving the artery of Percheron can present with abrupt onset of severe problems of alertness including coma. (C) Vertebral artery hypoplasia—Vertebral artery hypoplasia (usually defined as arterial diameter < 2.0 mm) is unilaterally present in approximately 15% of the population but in patients with PICA strokes, it is usually ipsilateral to the smaller artery. PCA = posterior cerebral arteries; PICA = posterior inferior cerebellar arteries.
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Adapted from Nouh et al.\(^2\)

BA = basilar artery; PCA = posterior cerebellar artery.
Reasons for clinical deterioration include extension of the stroke, brain stem compression from posterior fossa edema, and recurrent stroke.43

Another misconception likely resulting in misdiagnosis is that isolated transient dizziness is never due to a transient ischemic attack (TIA), a false notion promulgated by a National Institutes of Health (NIH) consensus conference in 1975.44 In a study of 1,141 stroke patients, of 59 transient neurologic events preceding posterior circulation stroke, only five (8%) fulfilled the NIH criteria for TIA.45 The other 54 cases were isolated vertigo (n = 23); binocular visual disturbance (n = 9); vertigo with other nonfocal symptoms (n = 10); isolated slurred speech, hemisensory tingling,
or diplopia ($n = 8$); and nonfocal events ($n = 4$). Of the posterior strokes, over 8% had transient episodes of isolated vertigo in the 48 hours preceding the stroke, almost certainly, due to transient ischemia. Finally, the limitation of imaging, including DWI-MRI, likely contributes to misdiagnosis. In this group of patients, we recommend a very brief diagnostic “stop” to reduce misdiagnosis. As part of this stop, first make sure there are no suspicious neurovestibular signs (nystagmus, limb ataxia, or gait/truncal ataxia). If a general medical cause still seems likely, evaluate and treat for the presumed diagnosis or diagnoses. For patients with a positive stop or whose history does not suggest a general medical cause, ask questions aimed at timing and triggers to place the patient into one of three categories. For patients in the AVS and t-EVS, physical examination will often allow a specific diagnosis to be made. For patients with the s-EVS, use history to try to distinguish vestibular migraine from TIA or other causes since, by definition, these patients will no longer have symptoms and their dizziness cannot be triggered at the bedside. For each vestibular syndrome, only the most common benign and dangerous diagnosis is listed. AVS = acute vestibular syndrome; BPPV = benign paroxysmal positional vertigo; CPPV = central paroxysmal positional vertigo; s-EVS = spontaneous episodic vestibular syndrome; t-EVS = triggered episodic vestibular syndrome; TIA = transient ischemic attack.

Can these diagnostic traps be mitigated? Neurologists try to match symptoms and signs with the culprit artery. For nonneurologists, distinguishing a lateral medullary from a lateral pontine infarct is less important than distinguishing a stroke from a nonstroke. The presence of robust collateral circulation and vascular anatomic variations can alter the classic posterior circulation syndromes. Therefore, we will focus the discussion on a symptoms-based approach.5

**Dizziness/Vertigo/Lightheadedness**

New evidence suggests that the time-honored “symptom quality” approach to dizziness (i.e., “what do you mean, dizzy?”) is flawed.46 Despite a weak evidence base, this approach has been the predominant paradigm for decades; an approach based on “timing and triggers” is more evidence-based.47,48 The descriptive word that patients use to describe their dizziness is diagnostically meaningless.49 Thus, we use the generic term “dizziness” to mean vertigo, unsteadiness, lightheadedness, and imbalance. Of note, the “timing and triggers” method is no different than taking a history from any other patient: How did the symptoms start? How have they evolved? Are other symptoms present? What is the medical and epidemiologic context?48

Acutely dizzy patients have one of three syndromes (Figure 3). For two of them, the acute vestibular syndrome (AVS—acute onset of persistent dizziness) and the triggered episodic vestibular syndrome (t-EVS—brief episodes of dizziness triggered by something), physical examination often supports a confident and specific diagnosis.50 The third, the spontaneous episodic vestibular syndrome (episodes of dizziness that are not triggered by anything), is most commonly caused by vestibular migraine but the most worrisome cause is posterior circulation TIA. By definition, patients are

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**Figure 3** Diagnostic approach to the acutely dizzy patient. ATTEST = A, associated symptoms; TT, timing and triggers; ES, examination signs; and T, additional testing as needed. The first step is to take a history focused on associated symptoms, timing and triggers of the dizziness, and the overall context. Many patients’ histories will suggest a general medical cause (various toxic, metabolic, infectious, or cardiovascular causes). In this group of patients, we recommend a very brief diagnostic “stop” to reduce misdiagnosis. As part of this stop, first make sure there are no suspicious neurovestibular signs (nystagmus, limb ataxia, or gait/truncal ataxia). If a general medical cause still seems likely, evaluate and treat for the presumed diagnosis or diagnoses. For patients with a positive stop or whose history does not suggest a general medical cause, ask questions aimed at timing and triggers to place the patient into one of three categories. For patients in the AVS and t-EVS, physical examination will often allow a specific diagnosis to be made. For patients with the s-EVS, use history to try to distinguish vestibular migraine from TIA or other causes since, by definition, these patients will no longer have symptoms and their dizziness cannot be triggered at the bedside. For each vestibular syndrome, only the most common benign and dangerous diagnosis is listed. AVS = acute vestibular syndrome; BPPV = benign paroxysmal positional vertigo; CPPV = central paroxysmal positional vertigo; s-EVS = spontaneous episodic vestibular syndrome; t-EVS = triggered episodic vestibular syndrome; TIA = transient ischemic attack.
asymptomatic (otherwise they would have an AVS) and the dizziness is not triggerable so the decisions are based entirely on history; physical examination is unhelpful.

Patients with dizziness from posterior circulation stroke usually present with an AVS. Although other conditions can cause an AVS, three causes predominate—vestibular neuritis, posterior circulation stroke, and multiple sclerosis.\(^49,51\) In one observational study, a new diagnosis of multiple sclerosis accounted for less than 2% (3/170) of cases.\(^52\) Therefore, the major differential is neuritis versus stroke.

In 2009, a study showed that a specialized three component ocular motor examination (head impulse test, nystagmus and test of skew—HINTS) was 100% sensitive in differentiating neuritis from stroke, outperforming MRI (88% sensitive when done within the first 48 hours).\(^53\) The examinations were performed by neurootologists. Studies have shown that stroke neurologists\(^54\) and emergency physicians (EPs) with special training and using Frenzel lenses\(^55\) can also accurately perform the HINTS examination. Our experience is that EPs can learn to perform and interpret these tests without specialized equipment, but prospective data are lacking. Therefore, we recommend two additional components of the examination be routinely performed—a targeted neurologic examination of visual fields, cranial nerves, and the cerebellum as well as evaluation for truncal ataxia and gait.\(^49\)

Occasional patients with benign paroxysmal positional vertigo will endorse constant dizziness (usually with acute intermittent worsening) superficially mimicking an AVS, when in fact they have a t-EVS. In this situation, performing a Dix-Hallpike (or other provocative) maneuvers may disclose the true nature of the problem. Confidently diagnosing a common peripheral vestibular process is one way to rule out a worrisome central one. One key misconception is that dizziness exacerbated by movement means that the process is peripheral. One must distinguish dizziness that is triggered by movement, no dizziness at baseline but dizziness starts with some obligate movement (suggestive a peripheral etiology) from dizziness that is exacerbated by movement, dizzy at baseline but made worse by movement (which can be due to a central process).

**Headache and Neck Pain**

Headaches are common and mostly caused by migraine and tension-type headache. Although more common in hemorrhagic strokes, headache still occurs in between 8 and 27% of cases of ischemic stroke\(^56\) and is even more common in patients with posterior circulation strokes, especially cerebellar. Headache and neck pain may also indicate VAD or SAH requiring emergency evaluation and treatment. Questions aimed at distinguishing the current episode of pain from prior ones may help to distinguish migraine from another process. Systematically performing a neurologic examination that targets the visual fields, cranial nerves, and cerebellar function including gait is one strategy to reduce misdiagnosis in these patients. Absence of new neurologic deficits would make a stroke very unlikely although it is important to recall that VAD can present with isolated pain.\(^56\)

**Sensory Symptoms**

Because sensory symptoms are inherently subjective, patients often have difficulty describing them. A history of sudden onset of lateralized numbness suggest an ischemic etiology. Distinguishing positive symptoms from negative ones can provide additional clues (Table 2). Positive sensory symptoms (tingling, pain, dysesthesias, or an aura) are more often due to migraines, seizure disorders, and peripheral neuropathy, whereas negative sensory symptoms (numbness or loss of sensation, sight or hearing) are more often associated with ischemia.\(^57\)

An exception to this “rule,” thalamic strokes can present with pain (a positive sensory symptom) or hemiballismus (a positive motor symptom; Figure 2D). Pure thalamic infarcts were found in up to 11% of posterior strokes, with some thalamic involvement in up to 27% of cases.\(^58,59\) Although thalamic stroke can present with isolated sensory symptoms, associated motor symptoms, aphasia, psychiatric complaints, pain

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syndromes, and decreased level of consciousness also occur. Sensory findings from PCA territory stroke are usually due to thalamic infarction related to small vessel disease, but are also found in superficial cortical PCA strokes.

**Altered Mental Status**

Altered mental status in the setting of an acute posterior stroke encompasses a broad spectrum—subtle or even transient neuropsychiatric disturbances to coma. Artery of Percheron strokes are rare and cause variable symptoms usually including decreased mental alertness, confusion, and psychiatric symptoms such as confabulation, perseveration, apathy or aggression, and hallucinations. An important clinical clue is a limitation of vertical gaze. Basilar stroke patients are obviously ill and are generally admitted; however, because timely endovascular interventions can be life-saving, early diagnosis is important. Prodromal headache and dizziness and presence of anisocoria and gaze palsies are common. Some basilar stroke patients will have involuntary movements that mimic seizure, leading to misdiagnosis of status epilepticus. Abrupt onset of altered mental status and any of these findings should prompt CT angiogram.

**Nausea and Vomiting**

Nausea or vomiting occurred in 27% of the 407 patients in the New England posterior circulation stroke registry. Vomiting, an independent risk factor for misdiagnosis, may be so severe as to divert attention from other symptoms such as headache or dizziness and rarely occurs in isolation. In a case series of 18 patients with strokes involving the lower brainstem (nucleus prepositus hypoglossi) presenting with dizziness and prominent vomiting, all had some other finding, most commonly truncal ataxia, nystagmus, or facial palsy.

**Visual Symptoms**

Examination of visual fields, pupils, and eye movements may help reduce misdiagnosis. Pertinent physical findings include a Horner’s syndrome, nystagmus, diplopia, and decreased vision (field cut or blurred vision). The ptosis from a Horner’s syndrome is mild and can mimic enophthalmos. The small pupil may be overlooked and is easier seen in a dark room (accentuating the difference in size). The ptosis is ipsilateral to the small pupil (where the ptosis is more apparent and ipsilateral to the large pupil). Patients with a Horner’s syndrome from a lateral medullary stroke usually have other findings as well such as ataxia, diplopia, and sensory changes and often are due to VAD.

Although nystagmus is a physical finding, some patients will spontaneously complain that their eyes or that their visual world are moving (oscillopsia). The details of nystagmus, not just the presence or absence, are diagnostically useful. Persistent nystagmus that is direction-changing, vertical, or torsional should be considered central. A sudden-onset internuclear ophthalmoplegia, caused by a lesion in the medical longitudinal fasciculus characterized by an impairment of adduction of the affected eye on conjugate lateral gaze and nystagmus, is also often due to stroke. Diplopia is another symptom/sign of posterior circulation stroke.

Unilateral PCA infarcts can cause a contralateral homonymous hemianopsia or quadrantanopsia. Patients may be unaware of the field cut and often describe decreased or poor vision in one eye or simply bumping into things on the side of the field cut. In the Lausanne stroke registry of the nearly 3,400 patients, 117 were found to have a pure superficial PCA territory stroke. Of the 117, a total of 78 (67%) had a hemianopia, 26 (22%) had a quadrantanopia, and eight (7%) had bilateral field abnormalities. Assessment of visual fields by bedside confrontation methods is not as accurate as formal perimetry but useful when positive.

Hallucinations can occur in thalamic and superficial PCA infarcts, another exception to the general rule about positive (hallucinations) versus negative (lack of vision) symptoms. In superficial PCA strokes, hallucinations, both simple and complex, were found in 10% of 117 patients. This is similar to the Charles Bonnet syndrome, which follows an acute visual loss.

**Language and Speech Deficits**

Dysarthria, clumsy or slurred speech, occurs with strokes that interfere with the process of forming words and are more common with cortical PCA infarcts. Patients describe “thick” or “heavy” speech. Of 62 consecutive patients with dysarthria from stroke, 61% were due to posterior circulation strokes, mostly in the brain stem and cerebellum. Overall, dysarthria occurs in 29% of cerebellar strokes (as a symptom) and 46% (as a physical finding). Dysarthria was found in nearly half of patients with thalamic
Having the patient say the word “Pawtucket” which tests the “pa,” “ta,” and “ka” sounds (which are made using three different parts of the mouth and tongue) is a quick way to test for dysarthria.

Various types of vascular aphasias also occur. Having a patient say, write, and then read a phrase or short sentence is a simple bedside test of these functions.

Cranial Neuropathy-related Symptoms
Small brain stem strokes can present with prominent cranial nerve–related symptoms, due to damage of the nuclei themselves or to ischemia of the postnuclear fibers as they traverse the brain stem. These patients can present with abnormal eye movements, facial hypesthesia or weakness, acute dizziness or hearing loss, and difficulty phonating and swallowing. In lateral medullary infarct, the common sensory deficit is loss of pain and temperature, leaving light touch intact. Careful cranial nerve testing is important in all patients in whom posterior stroke is a consideration.

Neuroimaging
Some form of brain imaging is required in patients with acute stroke; however, physicians must understand the limitations of early imaging. Brain CT is generally the first test performed; however, its sensitivity for posterior circulation ischemic stroke ranges from 7% to 42%. For patients presenting with acute dizziness, the sensitivity is even lower. More than one-third of patients with acute PCA strokes will show a hyperdense PCA sign on noncontrast CT. DWI-MRI also has important limitations in early-presenting posterior circulation ischemic strokes, especially for those presenting with isolated dizziness. In this setting, the false-negative rate of DWI-MRI is after onset of symptoms ranges from 12% to 18%. For those with small strokes, the false-negative rate is 53%. Physical examination using HINTS is more accurate in these patients than MRI, at least when done by specialists and is well within the scope of practice for EPs.

Overall, for patients with acute ischemic stroke, the evidence shows that the false-negative rate of DWI-MRI is 6.8% with an odds ratio (OR) for having a falsely negative DWI-MRI strongly associated with posterior circulation location (OR = 5.1, 95% confidence interval = 2.3–11.6, p < 0.001). If there is still significant diagnostic ambiguity after physical examination and any imaging that might have been done, it is safest to admit the patient to the hospital for further testing and urgent neurologic consultation.

If VAD or basilar stroke are being considered, vascular imaging is indicated. Although catheter digital subtraction angiography (DSA) remains the criterion standard, magnetic resonance angiography (MRA) and CT angiography (CTA) are typically performed. A CTA has advantages over MRA and is often readily available in the ED. Discussing the differential diagnosis with the radiologist may help to focus their attention to the relevant anatomy and, if clinical suspicion is high and the first imaging study is negative, an alternate study, or occasionally, DSA may be diagnostic.

CONCLUSIONS
Patients with posterior circulation cerebrovascular events are often misdiagnosed. Strategies that may decrease misdiagnosis include:

- With any neurologic symptoms, always establish if the onset is abrupt or not because abrupt onset suggests ischemia.
- Be aware of the nonspecific symptoms of posterior circulation ischemia and that variant vascular anatomy can cause nonclassic findings.
- For dizzy patients with an AVS, use a timing and triggers diagnostic approach taking a history as one would with any other chief complaint instead of focusing the history on the response to the question, “What do you mean by dizzy?”
- Perform a brief posterior circulation physical examination that targets the brain stem, cerebellum, and occipital lobes.
- Be aware of the limitations of brain imaging and adopt a nuanced approach that recognizes that initial brain imaging is frequently nondiagnostic.

References
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