Posterior Circulation Ischemic Stroke

by Steven Go, MD

The emergency physician needs to have a high level of suspicion for posterior circulation stroke, recognize the salient risk factors and clinical signs/symptoms, and be fastidious with the history and neurological exam.

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Abstract

Approximately 20-25% of all acute strokes occur in the posterior circulation. These strokes can be rather difficult to diagnose because they present in such diverse ways, and can easily be mistaken for more benign entities. A fastidious history, physical exam, high clinical suspicion, and appropriate use of imaging are essential for the emergency physician to properly diagnose and treat these patients. Expert stroke neurologist consultation should be utilized liberally.

Introduction and Epidemiology

In the United States, approximately 800,000 people suffer a stroke every year.[1] Although the majority suffers an ischemic stroke in the regions of the brain supplied by the anterior circulation (AC), approximately 20-25% of ischemic strokes occur in the territory of the posterior circulation (PC).[2] Mortality in posterior circulation stroke (PCS) (about 3.6% to 18.6%, depending on the stroke registry surveyed[3]) is classically thought to usually be more favorable in anterior circulation stroke (ACS), although that notion has been challenged in the literature.[4] However, what makes PCS such a vexing entity for clinicians is the myriad of ways it can present, ranging from the obvious to the mundane, and is probably more common than generally appreciated. For example, the frequent symptom of vertigo has been estimated to have a cerebrovascular origin 3-7% of the time.[5] In addition, PCS can rapidly progress to a dire condition, despite a sometimes very non-specific first presentation. Therefore, it behooves the emergency physician to be familiar with which patients are at risk for PCS, how to recognize the various symptom complexes, and how to treat these patients in an expeditious fashion. For the purposes of this article, the discussion will be confined to ischemic PCS.

Anatomy and Pathophysiology

The posterior circulation of the brain is pictured in Figure 1. The major arteries of the posterior circulation are the vertebral, posterior inferior cerebellar, basilar, and the posterior cerebral arteries. The posterior circulation is generally thought of as split in three divisions: proximal, middle, and distal. The most common areas of the posterior circulation affected in ischemic stroke are: distal (41%) multiple territories

(25%), proximal (18%) and middle (16%).[6] The major areas of the brain supplied include the brainstem, cerebellum, thalamus, occipital visual cortex, medial temporal lobe, and auditory/vestibular structures. The diversity of function of these structures helps explain how posterior circulation deficits can present in many different ways.

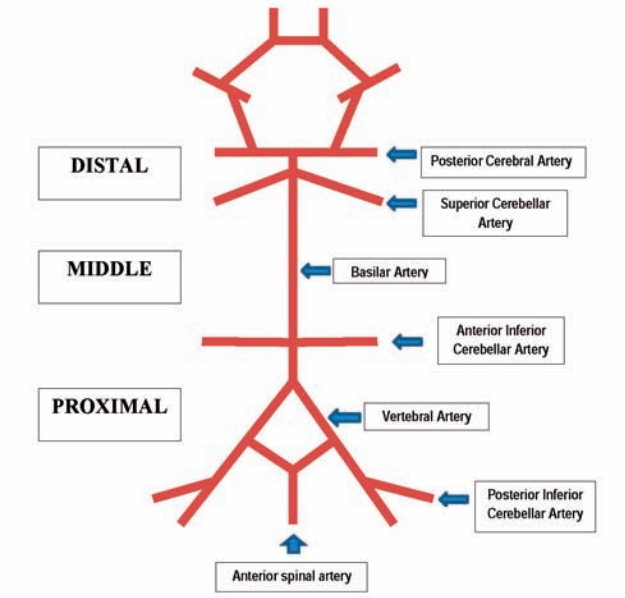
Clinical Presentation

According to a 2012 analysis of the New England Medical Center Posterior Circulation Registry (n = 207)), the most common presenting symptoms of PCS are dizziness (47%), unilateral limb weakness (41%), dysarthria (31%), headache (28%), nausea or vomiting (27%) and blurry vision (20%).[6] The most common clinical signs are unilateral limb weakness (38%), gait ataxia (31%), unilateral limb ataxia (30%), dysarthria (28%), nystagmus (24%), and Babinski's sign (24%).[6]

The vast majority of patients typically have more than one symptom or sign. Since the posterior circulation supplies the brain stem, classic dogma has claimed that the sine qua non of PCS is the presence of "crossed deficits" (ipsilateral cranial nerve deficits coupled with contralateral extremity deficits. These deficits may be either motor or sensory). However, in reality, the presence of crossed deficits is not sensitive, occurring in only 3%-4% of PCS.[7] Other specific, but non-sensitive, signs/symptoms with a high positive predictive value (PPV) are nystagmus (83.7% PPV), Horner's syndrome (100%), oculomotor nerve palsy (100%), and quadrantanopia [defective vision in a quarter of the visual field] (100%).[7] Interestingly, contrary to conventional teaching, disturbed consciousness was not found to be very sensitive (10.3%) or specific (81.4%) for PCS.[7] As shown by these data, although some clinical features make the presence of a neurological emergency obvious, most often, the signs/symptoms of PCS are rather non-specific. Some experts feel that the key to diagnosis is looking for the focality of symptoms that localize to the regions supplied by the posterior circulation; however, clinical features that allow easy discrimination of PCS from more benign entities remain elusive.[8]

Three classic syndromes involving the posterior circulation are worth noting. Basilar artery occlusion affects the middle portion of the circulation and is characterized by unilateral limb weakness, dizziness, dysarthria, diplopia, headache, cranial nerve VII findings, Babinski's sign, and oculomotor findings.[6] When the ventral pons is involved, basilar artery occlusion can also cause "locked in" syndrome in which the patient is fully conscious, yet completely paralyzed, except for blinking and sometimes facial movements.[6] A second syndrome, lateral medullary infarction (Wallenberg's syndrome) is caused by occlusion of the posterior inferior cerebellar artery or the vertebral artery. The classic findings are the crossed findings of decreased pain and temperature on the ipsilateral face and the contralateral side of the trunk and limbs. Additional findings include severe vertigo, unilateral arm weakness, dysphagia, dysarthria, dysphonia, nystagmus (horizontal and rotary), and ataxia. Respiratory function and cardiovascular abnormalities can also occur. Finally, vertebral basilar syndrome is caused by a disruption of the proximal posterior circulation. It is sometimes known as "beauty parlor syndrome" since it is associated with head turning and resultant occlusion of the vertebral artery. It presents with dizziness, nausea and vomiting, unilateral limb weakness and/or ataxia, cranial nerve V findings, nystagmus, and gait ataxia.[6] Despite these general patterns it should be noted that it is rare that the above syndromes present in their purely classic forms.

Figure 1
Major Arteries and Divisions of the Posterior Circulation



History

A rapid, focused history should be taken, including a chronological retelling of the patient's symptoms. Special attention should be paid to the presence of the most common symptoms listed above. A history of head or neck trauma should be ruled out, including cervical manipulation as this has been historically linked to PCS via

cervical arterial dissections.[9] Stroke risk factors[1] should be assessed and documented including advancing age, gender (female > male), ethnicity (African Americans > whites), hypertension, diabetes mellitus, previous TIA/stroke, atrial fibrillation, dyslipidemia, smoking, vascular disease (including coronary heart disease), CHF, smoking, physical inactivity, soda drinking,[10] family history, and chronic kidney disease (Cr ≥ 1.5 mg/dL), [11] and sleep apnea. Stroke risk factors of particular relevance to women include: use of estrogen, migraine with aura, pregnancy and preeclampsia.[1] The possibility of PCS should not be dismissed out of hand on the basis of young age, as cervical artery dissections occur in this group.[12] As with any potential stroke patient, it is imperative to ascertain the exact time the patient's symptoms began, as this has implications in terms of treatments that may be available to that particular patient. During the initial history, the National Institute of Neurological Disorders and Stroke (NINDS) indications and contraindications for systemic thrombolytic therapy should be documented.[13]

If the patient presents with "dizziness", this common symptom must be explored very carefully, as the history may have subtle clues to a need to suspect PCS. First, true vertigo (spinning, whirling, or tilting sensation) must be separated from lightheadedness. Second, the presence of "red flag" associated history or symptoms should be asked about. For example, h/o head or neck trauma (including cervical manipulation), head and/or neck pain, focal neurological symptoms (including cranial nerve findings, focal weakness, and speech difficulties), and visual complaints.

It is also important to realize that symptoms can be transient in the case of a posterior circulation TIA, which makes the diagnosis even more challenging.

Physical Exam

Once PCS is suspected on the basis of the history, a focused exam must be done. Airway should be assessed if altered mental status is present. The head and neck should be examined for trauma. HEENT exam should be done, focused on the ocular exam and carefully inspecting for nystagmus (particularly non-horizontal or gaze-evoked [nystagmus that changes direction as the direction of the gaze shifts]), and visual disturbances, including visual field cuts. Cranial nerves should be examined carefully, as multiple deficits indicate a central process. Cardiac exam for irregularly heartbeat and murmurs should be done. If the patient presents with vertigo, a Dix-Hallpike maneuver may be useful to detect a peripheral lesion, but this exam should not be done if neck trauma is present or if a cervical artery dissection is suspected because they involve rapid rotation of the head. Further specialized neurological tests may help differentiate between peripheral vertigo and stroke.

HINTS testing consists of the Head Impulse test (evaluation of the vestibular-ocular reflex), examination to detect the presence of Nystagmus that changes direction with gaze direction, and Test of Skew (vertical ocular misalignment during the cover-uncover eye test). The presence of one or more findings consistent with central vertigo has been reported to be sensitive and specific for stroke in expert hands; however, multicenter, external validation of HINTS testing with untrained physicians has not yet been published.[14] Finally, the National Institutes of Health Stroke Scale (NIHSS) should be used to perform a rapid (five minutes or less), yet thorough, neurological exam. The NIHSS is readily available on-line in pdf form,[15] and on-line score calculators exist as well.[16] Bedside NIHSS calculators are also available in smartphone apps for various platforms.[17-18] If safe, gait should be assessed and the severity of disturbance (if any) noted. Although use of the NIHSS remains standard of care in the US, it is very important to realize that it is heavily weighted towards anterior circulation findings. That is, a patient can have a clinically significant PCS, and score relatively low on the NIHSS. For example, a motivational speaker may present with isolated aphasia and only have an NIHSS score of 1; yet, for this patient, this deficit is clinically important. Nevertheless, the use of the NIHSS is indicated in all patients suspected of having a stroke.

Differential Diagnosis

A listing of selected conditions that can mimic a PCS can be found in Table 1.[2,19]

Table 1
Conditions that can Mimic a Posterior Circulation Stroke

- Benigh Postitional Vertigo
- Brain Tumor
- Conversion Disorder
- Hypoglycemia
- Intracranial Hemorrhage
- Meniere Disease
- Multiple Sclerosis
- Neuro-Bechet's Disease
- Posterior Reversible Encephalopathy Syndrome
- Sarcoidosis
- Seizure
- Sepsis
- Vestibular Migraine
- Vestibular Neuronitis
- West Nile Encephalopathy

Initial stabilization

As always, airway, breathing, and circulation need to be assessed and stabilized first. Intravenous access should be obtained and cardiac monitor should be applied to detect dysrhythmia. Oxygen should be given if O_2 Saturation on room air is < 95%. Once the patient is stable, a rapid history and physical exam are completed as above. A bedside glucose determination should be done to r/o hypoglycemia as a stroke mimic. This is the only laboratory test whose results must be known prior to imaging. At this point, the priority is to image the patient as quickly as feasible as that will direct any further therapy. There are currently no validated, evidence-based decision rules on whom to image to rule out posterior stroke. Therefore, if PCS is strongly suspected on the basis of the bedside history and exam, radiological imaging is warranted.

Imaging

The first study typically done in these patients is a non-contrasted head computed tomography (CT) to rule out an intracranial stroke mimic, and as a prerequisite to thrombolytic therapy. If PCS is suspected, it is imperative that this study be accomplished within 25 minutes of presentation to the ED, and the study interpreted by an expert radiologist (preferably a neuroradiologist) within 45 minutes of presentation.[13] Although current generation CT is very sensitive to hemorrhage, it is expected that it will be negative in acute ischemic stroke, including PCS. Magnetic resonance imaging (MRI) is more sensitive than computer tomography (CT) for PCS;[20] however lack of availability, cost, and patient tolerance are barriers to ED use. Therefore, if PCS is strongly suspected, vascular imaging of the posterior circulation is appropriate. Typically, this is done as a CT angiogram (CTA) of the head and neck. If available, MR angiogram (MRA) can be done in place of CTA to visualize the cervical and cerebral vasculature. There is a paucity of published data on comparative sensitivity rates between CTA and MRA. However, in one series, the sensitivity of CT Angiogram and MR Angiogram for vertebral artery stenosis ≥ 50% were both good to excellent, up to 68% and 89%, respectively.[21] In another series, CT/CTA was subjectively favored over MR/MRA for vertebral dissection.[22] The other utility of these vascular studies is that they also provide anatomic information with regards to the degree of blockage of the carotid arteries and their branches as well as detecting arterial dissections, which may have implications for further treatment decisions in these patients. In some centers, a non-contrasted head CT is included in the CTA order and the studies may be ordered concurrently. In this circumstance, it is our practice to go with the patient to the CT scanner and review the head CT in real time. If the head CT demonstrates an alternative cause for the patient's symptoms, then the CTA is canceled.

Treatment

Like any other acute stroke, systemic thrombolytic therapy should be considered in a patient with acute PCS.[23] It is beyond the scope of this article to discuss thrombolytic therapy for stroke in detail; however, a few salient points should be kept in mind. Thrombolytic therapy should be given according to the inclusion/exclusion criteria as defined in the literature, including the appropriate time windows. The previously mentioned anterior circulation bias of the NIHSS should be considered when the NIHSS is calculated, and thrombolysis should still be considered even when the NIHSS score is low if the PCS symptoms are clinically significant. PCS patients with a contraindication to systemic thrombolysis may still be candidates for endovascular therapy, such as intra-arterial thrombolysis or mechanical thrombectomy.

Patients with stroke secondary to cervical artery dissection are usually treated with anticoagulation or antiplatelet therapy. However, the optimal treatment strategy is yet to be determined, as recent data regarding the efficacy of systemic thrombolysis and endovascular therapies have been reported.

Because the stroke treatment literature evolves rapidly, treatment decisions can sometimes be difficult. Therefore, liberal consultation with an experienced neurologist with stroke expertise can be enormously helpful both in diagnostic strategies and treatment decisions. In the absence of an onsite consultation, a teleneurologist can suffice.

Disposition and Follow Up

All PCS patients should be admitted to a facility that has expertise in stroke care, preferably to a primary or comprehensive stroke center so that the patient can be managed in a dedicated stroke unit. If the facility

that the patient initially presents to does not have these capabilities, the patient should be transferred to the nearest, most appropriate stroke center from the ED once they are stable for transport. If the patient is to be transferred, consultation with the accepting neurologist should be obtained prior to the administration of thrombolytics in most cases, as the best treatment modality for individual patients, their particular lesions, and their particular clinical presentations may vary.

If symptoms are transient and consistent with TIA, risk stratification scores such as the ABCD^2 score[24] can be used; however, authors have recently questioned the utility of this scale in posterior strokes.[25] Therefore, it may be prudent to admit these patients as well. As with diagnostic and treatment decisions, consultation with a neurologist can be very helpful with difficult disposition decisions.

Conclusion

PCS is an important, but difficult diagnosis to make, given its wide range of presentations. The emergency physician needs to have a high level of suspicion for PCS, recognize the salient risk factors and clinical signs/symptoms, and be fastidious with the history and neurological exam. Finally, they should make frequent and liberal use of stroke neurologists and stroke centers in caring for these challenging patients.

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Disclosure

None reported. **MM**