CLINICAL VIGNETTE

Subtle Visual Deficits Presenting as a Posterior Cerebral Artery Stroke

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History

A 60-year-old male with multiple medical problems including hypertension, DM2, coronary artery disease and COPD presented to the emergency department (ED) with sudden onset of blurry vision in the right eye that began the prior night. He reported having associated eye pain and a pulsatile left-sided headache. The morning of his presentation, he experienced blurry vision and pain in his left eye that was worse than the right eye symptoms that started the previous night. He noted a variety of associated symptoms including photophobia, lightheadedness, nausea, slurred speech, and an aura of objects moving around in his visual fields. He had difficulty describing his symptoms, however, consistently reported bilateral eye blurriness as inability to see objects in his peripheral visual fields.

Physical Exam

On exam, he was afebrile with blood pressure of 163/68, heart rate of 88, respiratory rate of 14, and with normal oxygen saturation by pulse oximetry on room air. His triage eye exam recorded a visual acuity of 20/100 on his right eye and 20/70 on his left eye. When assessment was repeated, visual acuity was noted to be 20/200 in the right eye and 20/200 in his left eye. His pupillary response was symmetric. Intraocular pressures were unremarkable and extraocular muscles were intact but eye movement elicited pain. Confrontational visual fields revealed a bilateral right homonymous hemianopsia. The remainder of his eye exam was unremarkable. His neurologic exam revealed alexia and mild right upper extremity decreased sensation to light touch. His National Institute of Health Stroke Scale (NIHSS) score was 3, on the basis of complete hemianopia and right upper extremity sensory deficit.

Labs and Imaging

His laboratory studies were unremarkable. MRI brain revealed a large, early subacute infarct in the left posterior cerebral artery (PCA) region, including both the left occipital lobe and the left posterior mesial temporal lobe. MRA study of the Circle of Willis shows cutoff of the proximal left PCA at the P1-P2 junction. The final diagnosis was left PCA ischemic stroke of cardioembolic, thromboembolic, or atheroembolic etiology.

Discussion

Posterior circulation strokes (PCS) account for about 25% of all strokes.[1] The National Institutes of Health Stroke Scale (NIHSS) is heavily weighted towards identification of hemispheric disease resulting in patients with PCS receiving lower NIHSS scores despite the potential of having more disabling deficits.[2] While there is some overlap between anterior and posterior circulation strokes, the following findings have a 100% specificity for PCS: Horner's syndrome, oculomotor nerve palsy, quadrantanopia, and crossed sensory deficits.[1,3] Nausea and vomiting (33.8%) and ataxia (31.5%) are significantly more common in posterior circulation strokes.[4] A case series from the New England Medical Center Posterior Circulation registry of 407 patients reported the most frequent PCS symptoms are dizziness (47%), unilateral limb weakness (41%), dysarthria (31%), headache (28%), and nausea or vomiting (27%).[1] The vague nature of presenting signs and symptoms of PCS may impede the early recognition and activation of a stroke protocol.[2] This report will review the posterior circulation anatomy, demonstrate the importance of maintaining a working knowledge of the key differences between anterior and posterior circulation strokes as well as raise awareness of the challenges in recognizing PCS.

The posterior cerebral circulation is supplied by the vertebral, basilar, posterior cerebral, and posterior communicating arteries. A stroke involving the extracranial vertebral artery most commonly manifests as dizziness, however, strokes of the intracranial vertebral artery present with a classic clinical picture.[3] For example, Wallenberg, or lateral medullary, syndrome occurs with occlusion of the posterior inferior cerebellar artery (PICA). The clinical findings include: reduced pain and temperature sensation to the ipsilateral face and contralateral body; dysphagia, hoarseness, hiccups, and a reduced gag reflex; vertigo and nystagmus; and ipsilateral Horner's syndrome, ataxia, and dysmetria. The most frequently clinical signs and symptoms are hemibody sensory deficits (94%), vertigo and dizziness (91%), gait ataxia (88%), facial sensory deficits (85%), nausea and vomiting (73%), Horner's syndrome (73%), nystagmus (67%), dysphagia (61%), and hoarseness (55%)[5]. Prominent oculomotor changes include the ocular tilt reaction, positional nystagmus, gaze holding impairment, and saccadic abnormalities.[5] Further extension of the stroke to involve the ventral medulla may result in cardiac parasympathetic dysfunction.[6]

Anterior spinal artery strokes manifest as medial medullary syndrome. Features include contralateral hemiparesis, contralateral proprioception deficits and ipsilateral hypoglossal nerve palsy. The most common clinical findings are motor dysfunction (91%) commonly manifesting as hemiparesis (68%), as well as sensory dysfunction (73%) which may manifest as paresthesia (55%) or impaired perception of vibration (48%), position (41%), touch (32%), temperature (22%), and pinprick (17%).[7] Additional findings include dysarthria (63%), vertigo and dizziness (59%), nystagmus (44%), and dysphagia (29%).[6] A study of 86 patients found the majority (76%) endured rostral medullary lesions while the rest had ventro-dorsal medullary lesions.[7] Of note, motor deficits, sensory deficits, and vertigo/dizziness are closely involved in ventral, middle, and dorsal medullary dysfunction, respectively.[7] While ipsilateral hypoglossal nerve palsy is a fundamental component of the medial medullary syndrome triad, this study only found 3% with this and 10% presenting with contralateral tongue deviation.[7]

The basilar artery is most commonly occluded in the medial third portion (62.1%), followed by the proximal (39.1%) and distal (23%) thirds.[8] The intracranial vertebral artery is concurrently diseased in 75% of patients with basilar artery occlusion.[8] A study of 85 patients found 62% of individuals experienced prodromal symptoms prior to the basilar artery occlusion.[8] Forty-nine percent of these patients experienced vertigo and nausea while 33.9% experienced headache or neckache.[9] Impairment of the basilar artery manifests most commonly with bulbar and pseudobulbar signs (73.6%) and may also present with vertigo and dizziness (54%), hemiplegia (50.6%), cerebellar signs (49.4%), oculomotor abnormalities (44.8%), headache (41.4%), sensory deficits (39.1%), and nausea (34.5%).[7] The most common ocular abnormality is horizontal gaze paresis.[9] Locked-in syndrome, a feared complication of basilar artery occlusion resulting in pontine infarction is found in 1-10% of cases.[8,9] Occlusion of the distal basilar artery causes infarction of the midbrain, thalamus, temporal lobe, and occipital lobes, manifesting as a distinct clinical picture without any motor changes.[10] Individuals experience visual abnormalities, including impaired vertical gaze, hyperconvergence, skew deviation, and Collier sign, as well as behavioral changes such as somnolence, abulia, and hallucinations.[3]

PCA infarctions result in contralateral hemianopia with macular sparing as the macula is perfused by the middle cerebral artery. The Lausanne Stroke Registry include 117 patients with PCA strokes. The majority experienced visual abnormalities such as hemianopsia (67%), quadrantanopsia (22%), visual neglect (9.5%), visual agnosia (8.5%), bilateral visual field defects (7%), and prosopagnosia (6%).[11] Other findings included memory impairment (17.5%), dysphasia (14.5%), motor deficits (12%), hallucinations (10%), dyslexia without dysgraphia (8.5%), sensory deficits (6.8%), color dysnomia (5%), and dyslexia with dyspgraphia (4%).[12] Some individuals may only present with behavioral changes such as hallucinations, confusion, agitation, and delirium.[13] Sensory deficits that result from PCA stroke are attributed to hypoperfusion of the thalamogeniculate and lateral posterior choroidal arteries that supply the ventrolateral thalamus.[4]

Clinical Course and Correlation of Imaging and Clinical Findings

Our patient experienced a sudden, painful loss of peripheral vision in his right eye with a pulsatile, left-sided headache. The following morning, he presented to the ED with a painful loss of peripheral vision in his left eye. This acute presentation of right homonymous hemianopsia is consistent with a PCA stroke involving the left optic tracts, optic radiations, or occipital lobe.

On neurologic exam, our patient was also found to have alexia without agraphia, dysarthria, and decreased sensation to light touch on his right upper extremity. These findings are consistent with infarction of the left PCA affecting both the left occipital and temporal lobes. Alexia without agraphia occurs when PCA strokes impair blood supply from the dominant occipital lobe to the splenium of the corpus callosum.[11] Our patient is left-handed, which may lead to the assumption that he has right-brain dominance as handedness is contralaterally associated with brain dominance.[14] However, the majority of left-handed individuals, still exhibit left-brain dominance, explaining the alexia without agraphia in our patient with a left PCA stroke.[15] The New England Medical Center and the State of Qatar posterior stroke registries found 31% and 64% of posterior circulation infarcts present with dysarthria, which supports our patient's clinical finding.[16]

Diagnostic imaging further elucidated the extent of the PCA stroke. MRI demonstrates a large, early subacute infarct in the left PCA region, including both the left occipital lobe and the left posterior mesial temporal lobe. Fifty-three percent of homonymous hemianopsia cases are attributed to occipital lobe lesions, 35% to optic radiation lesions, and 7% to optic tract lesions.[17] Our patient's presentation is consistent with prior case reports as imaging demonstrates impaired perfusion of the left occipital lobe and the left, lower optic radiations that travel through the left temporal lobe, resulting in right homonymous hemianopsia.

The patient's MRA revealed occlusion of the proximal left PCA near the P1 and P2 segments. This hypoperfusion of the territories supplied by the P1 and P2 segments explains the patient's dysarthria and decreased sensation to light touch on the right upper extremity. Because the P1 segment supplies the midbrain, interruption of blood flow can impact the corticobulbar tracts, manifesting as dysarthria.[11] The P2 segment branches into the posterior choroidal and thalamogeniculate arteries, which supply the ventrolateral thalamus.[4] A retrospective study found that 25% of patients with PCA infarcts had sensory deficits, with 73% of these deficits attributed to infarcts of the ventrolateral thalamus.[4]

Our patient's past medical history is significant for poorly controlled type 2 diabetes, hyperlipidemia, coronary artery

disease, COPD and a wheelchair-bound state. The patient also reported significant weight loss, early satiety, and melena in the last 6 months that raises suspicion for cancer, which potentially creates a hypercoagulable state. Our patient has numerous risk factors for atherosclerosis. A meta-analysis of seven trials with a total of 8097 patients reported 35% of PCA strokes were due to atherosclerosis, 18% due to cardioembolism, 13% due to small vessel disease, and 15% to undetermined etiology.[15]

The patient was hospitalized for 6 days for evaluation and management of a left PCA ischemic stroke. His right homonymous hemianopsia persisted and had associated visual agnosia, acalculia, finger agnosia, anterograde amnesia, right-left confusion, and alexia. He had no anomia. His alexia was believed to be due to hypoperfusion of splenium of corpus callosum. The etiology of his stroke was thought to be atheroembolic (bilateral carotid plaques on carotid ultrasound) versus thromboembolic due to potential hypercoagulable state from underlying undiagnosed malignancy. He was discharged to a skilled nursing facility for continued care and rehabilitation.

Given our patient's history of migraines and presentation of a pulsatile, unilateral headache, photophobia, nausea, and aura of objects moving his visual field, recognition of his stroke would have been difficult. The subtle presentation of posterior circulation strokes mandates that we closely scrutinize visual changes to optimize stroke management.

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