## Posterior Cerebral Artery Ischemic Stroke in a Patient with Hypoplastic P1 Segment of the Posterior Cerebral Artery

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#### **Abstract**

Posterior circulation stroke usually presents with clinical challenges, and symptoms may include blindness. Hypoplastic or atretic cerebral artery may be a risk factor for future ischemic stroke. We present a 75-year-old male patient with posterior cerebral artery ischemic stroke who presented with sudden visual deterioration, and brain magnetic resonance angiography showed hypoplastic right P1.

Keywords: Hypoplastic artery, ischemic stroke, posterior circulation, visual impairment

#### **INTRODUCTION**

Visual impairment is a common symptom associated with stroke, though it may rarely be the only presentation in the posterior circulation stroke. About 20%-30% of stroke affects the posterior circulation, with infarction involving the posterior cerebral artery (PCA) territory accounting for 6.8%-9.6%.[1] The clinical presentation of posterior circulation stroke differs considerably when compared to anterior circulation strokes, due to the vast array of supra- and infratentorial structures supplied by the posterior circulation.[1,2] The incidence of cortical blindness is as high as 70% in a patient with occipital strokes. The most common cause of cortical infarction in the distribution of the PCA is emboli from the heart or vertebrobasilar circulation.[1,3] Other known causes include prolonged hypotension or hypoxia usually resulting in bilateral cortical blindness.

Hypoplasia of the pre-communicating segment of the PCA is usually associated with a large posterior communicating artery which may provide an easy route for cardiac embolus and thus predispose to ischemic stroke in the territory supplied. PCA ischemic stroke in our patient with hypoplastic P1 segment of PCA suggests that hypoplastic PCA is a predisposing factor for future ischemic stroke.<sup>[1,2]</sup>

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#### CASE REPORT

Our patient is a 75-year-old man with a background history of glaucoma who presented with a history of sudden visual deterioration as he woke up early in the morning, seven days before presentation. There was no history of headache, vomiting, or altered sensorium. There was no history of any focal body weakness. He is a known hypertensive, controlled on medication. There was no history suggestive of diabetes mellitus.

Examination revealed an aged man with normal mental status. Pupils were 3 mm and brisk. The visual acuity was 6/12 OS and 6/60 OD, and clinical visual field assessment revealed a left homonymous hemianopia with macular sparing. The long tract examination was normal. The examination of the other systems were normal.

A clinical diagnosis of visual deterioration secondary to posterior circulation stroke with background glaucoma was formed. The

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lipid profile was within normal limits, and echocardiography showed no thrombus. Cranial computerized tomography scan obtained three days post ictus [Figure 1] showed a right medial occipital lobe infarct. The carotid Doppler ultrasound obtained on 13 days post ictus showed both common carotid arteries with a normal course, caliber, color flow, and spectral waveform. The carotid arteries with a intima-media thickness is increased bilaterally measuring 1.0 mm and 1.1 mm on the right and left, respectively. The right common carotid artery resistivity index (RI) was 0.77 (increased), and the left common carotid artery RI was 0.70 (normal), normal up to 0.75. A hypoechoic plaque was seen at the right side with a punctate calcification within it. There was no associated significant narrowing. The vertebral arteries showed cephalad flow bilaterally having RI of 0.77 and 0.56 on the right and left, respectively, normal up to 0.75. The sonographic impression was bilateral carotid arteriosclerosis, worse on the right. Brain magnetic resonance imaging obtained 20 days post ictus [Figure 2] showed a right medial occipital lobe infarct, and brain magnetic resonance angiography obtained 20 days post ictus [Figure 3] showed hypoplastic right P1. He was managed conservatively, and he is being followed up at an outpatient clinic.

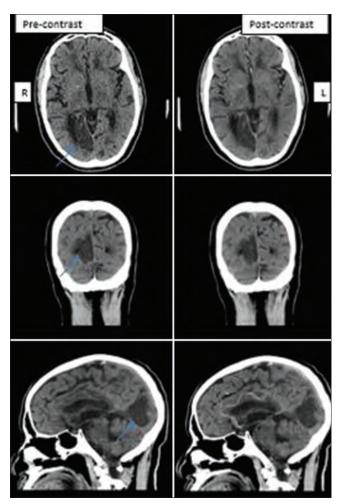


Figure 1: Cranial computerized tomography scan with arrows pointing to the infarction

#### DISCUSSION

The posterior circulation involves the arterial system which supplies a wide variety of eloquent brain regions which include the brain stem, cerebellum, medial occipital cortex, and the thalamus. The occipital lobe receives a dual blood supply from both the middle cerebral and posterior cerebral arteries making it less susceptible to vascular insult. [4] A stroke affecting these brain regions may present in a complex and unusual manner, and a high index of suspicion is essential for prompt and proper diagnosis. [1,2,5]

Cortical blindness is common in posterior circulation stroke, most especially when the PCA territory is involved as in the index patient. Posterior circulation stroke is common in elderly hypertensive patients, and cardiac embolus is usually the common cause followed by intrinsic vascular disease. The echocardiography showed no embolus in the cardiac chambers, but the carotid Doppler ultrasound showed an atheromatous plaque on the right carotid bulb which could be the source. [3,6,7] There has been a significant correlation between the presence of hypoplasia or atresia of an artery in the posterior circulation and the occurrence of ischemic stroke. [1,2]

Prolonged hypotension due to the use of antihypertensive drugs may precipitate ischemic stroke in the territory supplied by the hypoplastic artery. The index elderly patient on antihypertensive has hypoplasia of right P1 and developed ischemic stroke in the right visual cortex. There is a tendency for cardiac or internal carotid artery-derived embolus to go through a dominant posterior communicating artery in cases of hypoplastic P1, thus predisposing a patient to ischemic stroke of the visual cortex.<sup>[4,8]</sup> The use of antihypertensive in this patient may also predispose to hypotension, which may further compromise blood supply through the hypoplastic right P1. The presence of an atheromatous plaque and hypoplastic P1 on the right, may explain the ipsilateral involvement of the patient's pathology. A hypoplastic basilar artery is a risk factor for bilateral cortical blindness because it usually compromises arterial supply through both posterior cerebral arteries.<sup>[9]</sup> Management is usually conservative, and revascularization is rarely necessary.

Visual impairment may be the only manifestation of the ischemic stroke in the region supplied by the PCA, because it usually affects a small territory due to shared blood supply with anterior cerebral artery through anastomosis with distal branches of the pericallosal arteries.<sup>[10]</sup> Visual impairment is a common but often neglected complication of stroke with a significant impact on the patients' quality of life.<sup>[11,12]</sup> The incidence of visual impairment in PCA stroke is as high as 70% and neuro-ophthalmologists should take an active role in the rehabilitation protocol.<sup>[11,13]</sup>

#### Conclusion

Posterior circulation stroke may present to clinicians in a variety of ways including blindness. The presence of carotid

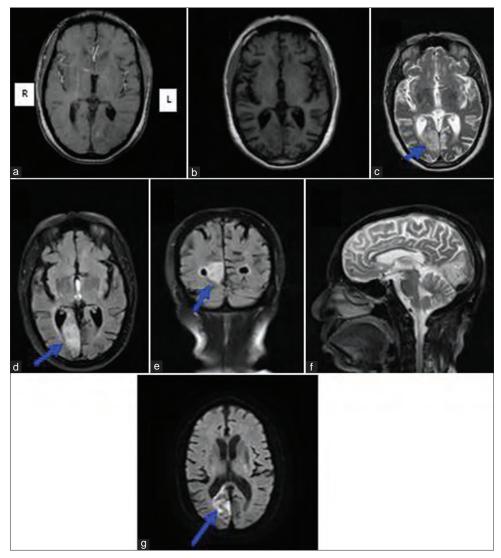


Figure 2: Brain magnetic resonance images: (a) Contrast T1W sequence, (b) T1W sequence, (c) T2W sequence, (d) axial fluid-attenuated inversion recovery, (e) coronal fluid-attenuated inversion recovery, (f) sagittal T2W sequence, (g) axial diffusion-weighted imaging sequence with blue arrow pointing to the area of ischemia

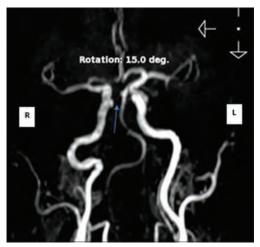


Figure 3: Brain magnetic resonance angiography with arrow pointing to the right hypoplastic P1

atheromatous plaque and hypoplastic cerebral artery are risk factors for future ischemic stroke. The role of routine brain angiography and management protocol to prevent future ischemic stroke in hypertensive patients should be examined with a large study.

#### Consent

We obtained the consent of the patient for this publication.

#### **Declaration of patient consent**

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his consent for his images and other clinical information to be reported in the journal. The patient understands that name and initials will not be published and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.

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Nil.

#### **Conflicts of interest**

There are no conflicts of interest.

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