

Clinical & Refractive Optometry is pleased to present this continuing education (CE) article by Dr. Leonid Skorin Jr., and Alicia M. Yantes entitled **Unusual Visual Field Defect Secondary to Posterior Cerebrovascular Accident**. In order to obtain a 1-hour Council of Optometric Practitioner Education (COPE) approved CE credit, please refer to page 23 for complete instructions.

Unusual Visual Field Defect Secondary to Posterior Cerebrovascular Accident

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ABSTRACT

Cerebrovascular accident (CVA), also known as a stroke, is the third leading cause of death and the leading cause of long-term disability in the industrialized world.¹ Posterior cerebral strokes are less common than anterior or middle cerebral strokes but are more often associated with visual manifestations. This article includes a Case Report on a patient with an unusual visual field defect associated with a posterior CVA. Stroke classifications, posterior cerebral circulation anatomy, visual field defects, brain imaging, and treatment strategies are also discussed.

INTRODUCTION

According to the World Health Organization (WHO) a stroke is defined as “rapidly developing clinical signs of focal (or global) loss of cerebral function, with symptoms lasting more than 24 hours or leading to death, with no apparent cause other than that of vascular origin.”^{1,2} Patients experiencing stroke like-symptoms lasting less than 24 hours are classified as having a transient ischemic attack (TIA); however, symptoms lasting longer than an hour may cause permanent infarction.^{2,3}

Strokes can be divided into two main categories: ischemic and hemorrhagic. Ischemic strokes are caused by artery, or rarely vein, occlusion which disrupts the circulation of blood flow to the brain tissue. Ischemia accounts for 80% of all strokes and usually presents as a sudden onset.⁴ The most common etiology is atherosclerosis, followed by diseases of small intracranial

arteries, arterial dissection, embolism from the heart, vasculitis, and hematological diseases.^{2,4,5} Hematologic disease causing pathological clot formation is found in less than 8% of ischemic stroke patients.²

Hemorrhagic strokes result from a ruptured blood vessel which decreases blood supply to the brain tissue. Hemorrhagic strokes are either subarachnoid or intracerebral. A subarachnoid hemorrhage is most often caused by rupture of an aneurysm with profuse bleeding into the subarachnoid space. An intracerebral hemorrhage is bleeding within the brain tissue usually caused by high blood pressure. Hemorrhagic strokes have a high mortality rate and account for 20% of all strokes.^{2,4}

A TIA is sometimes referred to as a mini-stroke and is an episode of focal neurological symptoms with abrupt onset and rapid resolution. Transient ischemic attacks last less than 24 hours and are due to altered circulation to a limited region of the brain. A visual TIA is known as amaurosis fugax or transient monocular blindness and is attributed to transient ischemia of the retina in one eye. Amaurosis fugax is most commonly associated with an atherothrombotic embolism from the internal carotid artery and increases the risk of stroke by 3% to 5% per year.⁷ Transient ischemic attacks are important risk factors for strokes and patients need appropriate workup and preventative stroke treatment.^{6,7}

Other risk factors for a CVA include increased age, hypertension, carotid artery disease, smoking, hyperlipidemia, diabetes mellitus, atrial fibrillation, atherosclerosis of the aortic arch, connective tissue diseases, and migraine.^{4,8,9} The most common risk factors are hypertension and advanced age, followed by diabetes mellitus.⁹ Symptoms of a CVA include sudden weakness or numbness of the face, arm, or leg; sudden loss of vision or dimming of vision; sudden difficulty speaking or understanding speech; sudden severe headache; and sudden falling, gait disturbance, or dizziness.⁴

CASE REPORT

A 56-year-old African American male presented at our clinic for vision changes in both eyes. His vision started getting blurry that morning and he noticed a black curtain at the top of his vision. He also stated that his peripheral

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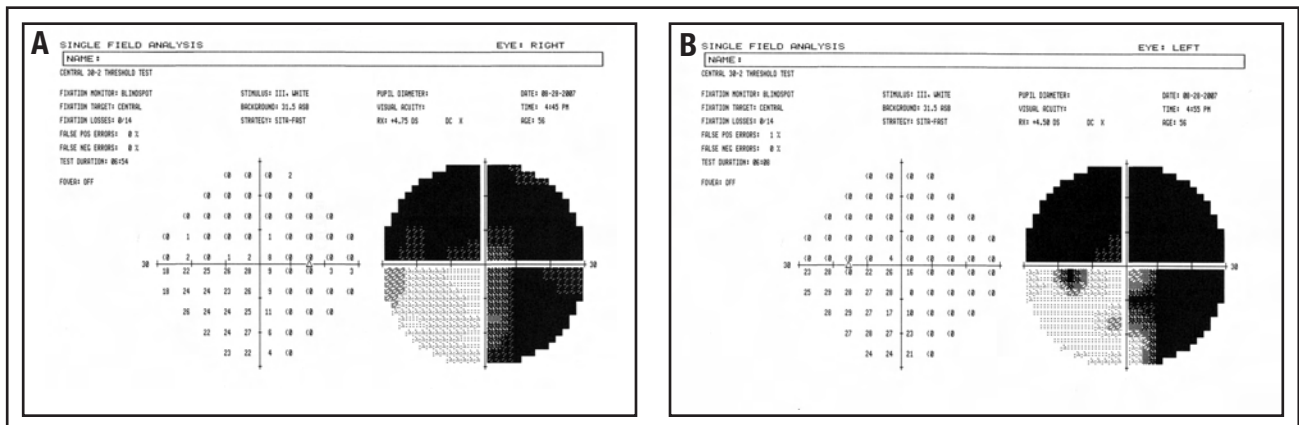


Fig. 1 (A) Visual field of the right eye showing a right-sided hemianopic defect and superior altitudinal defect. **(B)** Visual field of the left eye showing a right-sided hemianopic defect and superior altitudinal defect.

vision was missing on the right side. Other ocular symptoms included eye strain, tearing, headache, and diplopia. Two days prior he was seen in the emergency room for complaints of dizziness, headache, and difficulty walking. His medical history included systemic hypertension but he was not taking his anti-hypertensive medication at this time. The patient was also a smoker. An electrocardiogram, complete blood count, and computed tomography (CT) were done by the emergency room physician. The CT revealed a right parietal-occipital lesion. All other tests were consistent with an acute hypertensive episode and the patient was treated with clonidine and instructed to restart hydrochlorothiazide for his systemic hypertension. The patient was advised to get magnetic resonance imaging (MRI) of the brain and to remain in the hospital for observation but he refused medical advice and was released that day in stable condition. Personal ocular history included primary open angle glaucoma and laser peripheral iridotomy in both eyes. He was using timolol 0.5% twice a day in both eyes for his glaucoma.

Distant visual acuities at this exam were 6/12 (20/40) in both the right and left eyes. Extraocular movements were full with no restrictions and pupils were equal, round, and reactive to light with no afferent pupillary defect. Intraocular pressures were 20 mmHg in the right eye and 22 mmHg in the left eye. Confrontation visual fields showed right-sided and superior visual field defects in both eyes.

External exam revealed no palpable pain over the temporal arteries and a slight palpable strain over the occipital region. Biomicroscopy was unremarkable with clear corneas and deep and quiet anterior chambers. A patent iridotomy was present in each eye.

The dilated fundus exam revealed a cup to disc ratio of 0.60 in the right eye and 0.65 in the left eye. The peripheral retina appeared normal in both eyes with no retinal detachments, holes, or tears. An optical coherence tomography scan of the optic discs was performed and showed no nerve fiber layer depression in the right eye and moderate fiber layer depression in the left eye. This corresponded to the patient's history of glaucoma.

A 30-2 Humphery visual field was performed at this exam (Fig. 1). The results were reliable and revealed a congruent homonymous right hemianopsia and bilateral superior altitudinal defects. Macular sparing was not exhibited on the visual field results.

Due to the extent of the visual field defect, an occipital/parietal CVA secondary to an acute hypertensive episode was suspected. An MRI was ordered with and without gadolinium enhancement. The MRI revealed one old infarct in the right posterior parietal region and three new infarcts including one in the right cerebellum (Fig. 2), one in the right medial occipital lobe, and one in the left medial occipital lobe (Fig. 3). The left medial occipital lobe lesion was larger and more extensive than the right medial occipital lobe lesion. The new infarcts were all supplied by the posterior circulation. No intracranial hemorrhages or masses were found. The MRI confirmed a posterior CVA which corresponded to the visual field changes of a congruent homonymous right hemianopsia and bilateral superior altitudinal defects. The patient was referred to neurology for a complete work up.

DISCUSSION

Posterior cerebral artery (PCA) strokes account for approximately 26% of ischemic strokes.^{9,10} The posterior cerebral circulation begins in the aortic arch and branches

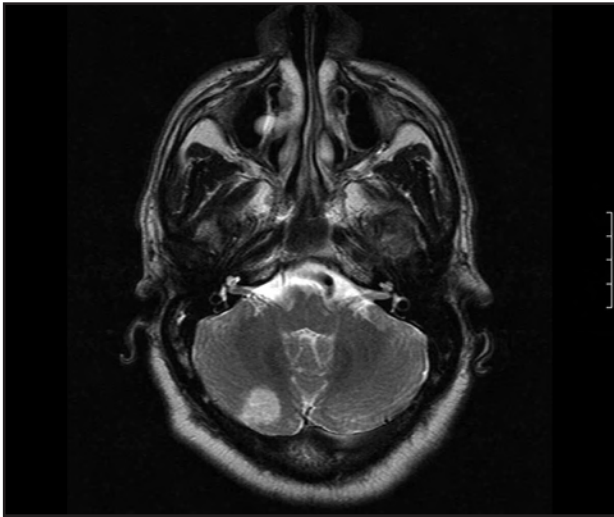


Fig. 2 MRI scan showing ischemia in the right cerebellum.

directly into the left subclavian artery and into the right subclavian artery by way of the brachiocephalic artery. The subclavian arteries give rise to the right and left vertebral arteries which enter the skull through the foramen magnum and meet at the base of the medulla oblongata to form the basilar artery. The basilar artery supplies branches to the cerebellum and brainstem before dividing into the right and left PCA delivering blood to the occipital lobe.¹¹ The PCA is further subdivided into proximal (P1) and distal (P2) segments defined by the posterior communicating artery. The P1 segment splits into the thalamoperforating arteries which lead to the thalamus, the posterior choroidal arteries which supply the ventricles and choroid plexus, and the short and long circumflex arteries which supply the brainstem. The P2 segment divides into the temporal arteries and the internal occipital arteries. The temporal arteries include the common, anterior, middle, and posterior branches which supply the fusiform gyrus and inferior temporal gyri. The internal occipital arteries include the parieto-occipital artery extending to the precuneus and cuneus areas of the occipital lobe and the calcarine artery providing the major blood supply to the striate cortex. Branches from the posterior temporal or parieto-occipital arteries can also supply blood to the striate cortex along with the occipital branch from the middle cerebral artery.^{10,11,12}

According to Marinkovic et al, the region of the brain supplied by an artery varies in individuals and depends on the size and territory of ramifications of that artery, as well as the size of surrounding cortical branches.¹² Brain tissue ischemia depends on the following factors: the size of the region supplied by the affected artery, the cause of the occlusion, the efficiency of the arterial anastomoses, and the characteristics of the

general and local brain vasculature and blood flow. Individual variations can lead to discrepancies between the expected and real size and shape of ischemic areas in some patients.¹²

Visual field defects are the most common clinical sign of a posterior ischemic stroke.⁸ Visual field testing is important in locating lesions or infarctions along the visual pathway. Lesions anterior to the chiasm will present as a visual field defect in only one eye, lesions within the chiasm will most likely show bitemporal or binasal defects, and lesions posterior to the chiasm will present as homonymous defects. Homonymous visual field defects are defined as defects that affect the nasal field of one eye and the temporal field of the other eye and are considered congruent if the two defects are similarly shaped. The most common visual field defect associated with an occipital lobe lesion is a congruent homonymous hemianopsia.¹¹

Automated static perimetry is the visual field technique used most often to identify visual field defects in neurologic diseases.¹³ Static perimetry is comparable to manual kinetic perimetry in detecting visual field loss and occipital lobe lesions; however, it lacks the accuracy that the tangent screen and Goldmann perimetry provide in the localization of the lesion. One study by Wong et al, found that the tangent screen and Goldmann perimetry corresponded well with the MRI in patients with occipital lobe infarcts while central 30-2 Humphrey visual fields did not correspond with the tangent screen, Goldmann perimetry, or MRI in half the patients.¹³ Even though kinetic perimetry is more reliable in localizing occipital lobe lesions, the Humphrey Field Analyzer still remains adequate in detecting them.¹³

The primary visual cortex, or striate cortex, is located in the medial aspect of the occipital lobe straddling the calcarine fissure. Superior to the calcarine fissure is the cuneus gyrus corresponding to the inferior visual field. Inferior to the calcarine fissure is the lingual gyrus corresponding to the superior visual field. The left striate cortex is represented in the right visual field and the right striate cortex is represented in the left visual field. The macular area is located in the most posterior aspect of the occipital pole while the peripheral visual field is found more anterior.^{11,14,15}

Macular sparing occurs when the posterior pole is uninvolved in the occipital lobe lesion and at least three degrees of central vision are unaffected on perimetry testing. Macular splitting occurs when a lesion involves the posterior pole or less than three degrees of central vision is spared. The strongest influence on macular sparing is the blood supply to the posterior pole including the extent of the calcarine artery and other contributing branches.¹⁴

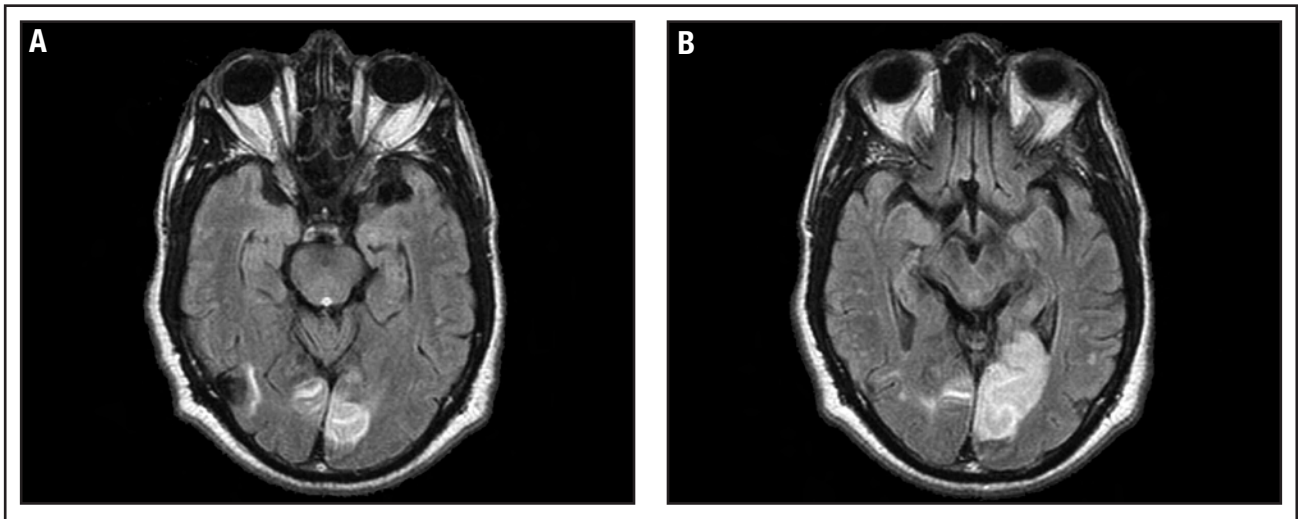


Fig. 3 Axial MRI scans showing (A) right and left medial occipital lobe lesions and (B) a more rostral view of the extensive left medial occipital lobe lesion.

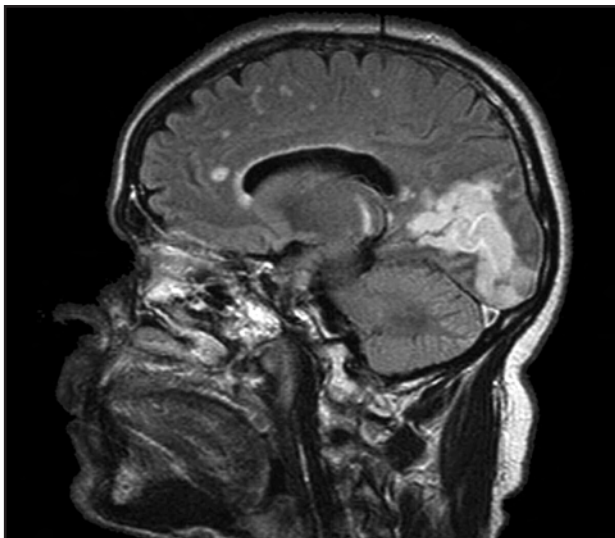


Fig. 4 Sagittal MRI scan showing a left medial occipital lobe lesion involving the striate cortex from the parieto-occipital sulcus to the posterior pole including both the cuneus and lingual gyri.

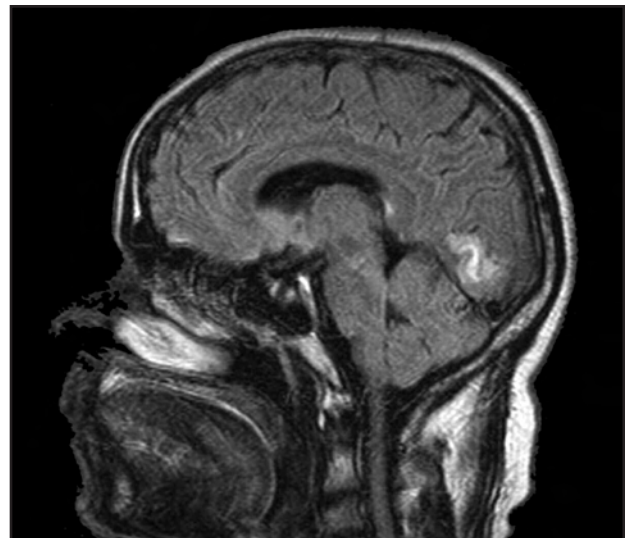


Fig. 5 Sagittal MRI scan revealing a right medial occipital lobe lesion involving only the lingual gyrus.

Our patient had a unique visual field defect assessed by the Humphrey Field Analyzer indicating a congruent homonymous right hemianopsia and bilateral superior altitudinal defects with macular splitting. This implies a left medial occipital lobe infarct affecting both the cuneus and lingual gyrus along with left posterior pole involvement, and a right medial occipital lobe infarct affecting only the lingual gyrus and right inferior posterior pole. This is unique because most occipital lobe lesions are unilateral and cause only a right or left homonymous hemianopsia. The extent of our patient's cerebral vasculature most likely contributed

to this abnormal presentation. The MRI confirmed infarcts in these areas (Fig. 4 and Fig. 5).

Computed tomography and MRI both provide important information in the diagnosis of a CVA. Computed tomography of the head is the most frequently used scan during the initial workup of a stroke to distinguish between ischemia and subarachnoid or intracerebral hemorrhage. Computed tomography is quick, accurate, and cost-effective in ruling out a hemorrhagic stroke; however, the presence of ischemia may not be identified until a minimum of two hours after stroke onset and

usually much later.^{5,16} Magnetic resonance imaging has a higher sensitivity than conventional CT and is better at diagnosing early ischemia, lesions in the posterior fossa, identification of small lesions, and documentation of vessel occlusion and brain edema.^{5,16} New cerebral ischemia did not show up on the CT of our patient during initial workup most likely because the stroke onset was too recent or there was too much artifact from the bones in the skull in the posterior fossa which interfered with the scan.

TREATMENT

Distinguishing between ischemic and hemorrhagic stroke is important for determining appropriate treatment. Current treatment for ischemic strokes includes tissue plasminogen activator (tPA) and antiplatelet or anticoagulation therapy. Intravenous tPA was approved in the United States in 1996 and in Canada in 1999 for the use in acute ischemic strokes.^{17,18} Tissue plasminogen activator is a secreted serine protease which converts the proenzyme plasminogen to plasmin, a fibrinolytic enzyme that dissolves blood clots. Tissue plasminogen activator is effective within the first three hours of stroke onset when administered intravenously, and up to six hours after onset when administered through an arterial catheter directly to the site of occlusion.^{16,17} Only 1% to 2% of patients qualify for this treatment due to specific time requirements and inherent risk of bleeding complications.^{2,17}

Besides tPA, antiplatelet and anticoagulation therapy is important in treating acute ischemic strokes and in preventing strokes in patients with significant risk factors. The most commonly used antiplatelet medications include aspirin (75-325 mg/day), clopidogrel (Plavix, Sanofi Aventis/Bristol-Myers Squibb), dipyridamole (Persantine, Boehringer Ingelheim), and a combination of aspirin and extended-release dipyridamole (Aggrenox, Boehringer Ingelheim).^{2,16,17,19,20,21} According to a study done by Halkes et al, the combination of aspirin and extended-release dipyridamole is significantly more effective than aspirin alone and does not cause excessive bleeding that may occur with other antiplatelet combinations.²¹ Anticoagulation therapy includes warfarin (Coumadin, Bristol-Myers Squibb), which is most often used with cardiogenic embolism.²⁰

Treatment for hemorrhagic stroke includes stabilizing the patient's vital signs, treating the cause of the bleeding, and surgically removing blood in moderate to severe cases. Tissue plasminogen activator, antiplatelet, and anticoagulation therapy is contraindicated and potentially fatal in hemorrhagic strokes due to increased bleeding with these treatments. Both ischemic and hemorrhagic stroke patients with permanent disability can also benefit from rehabilitation training.

CONCLUSION

Stroke is a common cause of death and disability worldwide. It is important to understand the types, risk factors,

symptoms, and diagnostic tools to rule in or rule out a stroke in patients with visual complaints. Visual fields are important and useful in detecting lesions along the visual pathway. The most common visual field defect associated with an occipital lobe lesion is a congruent homonymous hemianopsia, but other patterns can also present. Treatment of an acute stroke, prevention of a secondary stroke, and control of significant risk factors are important for a good prognosis and increased life expectancy. □

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