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Bilateral Parieto-Occipital Cortex Infarcts and their Effects on the Visual Field

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Abstract

Cerebrovascular accidents are a leading cause of serious long-term disability. Accurate diagnosis of a cerebrovascular accident is crucial to prevent morbidity, mortality and functional loss. A case report involving a visual field defect secondary to a bilateral parieto-occipital cortex infarct is discussed. Reviews of the blood supply to the brain and the visual field pathway are presented to highlight the importance of understanding the anatomy. A discussion of ancillary testing as well as interdisciplinary care is also provided to educate on proper patient management.

Key Words: *occipital lobe, visual field defect, cerebrovascular accident, stroke, vascular anatomy*

Background

Cerebrovascular accidents (CVAs), also known as strokes, are the leading preventable cause of disability in nearly 130,000 people in the United States per year. Strokes lead to approximately one in 20 deaths.[1,2] Due to their high prevalence, it is important to become familiar with strokes and their various consequences. The following case report involves a 62-year-old Indian male with non-insulin-dependent diabetes mellitus, hypertension, hypercholesterolemia and coronary artery disease status post coronary artery bypass grafting (CABG). He presented to the emergency department with complaints of bilateral blurred near vision in conjunction with a headache for a duration of one month. His case demonstrates the crucial nature of understanding the anatomy and blood flow of the brain. The appropriate use of ancillary diagnostic testing to capture the entire clinical picture is discussed. Further, this case highlights the optometrist's role in working with an interdisciplinary team to maximize patient care. The intended audiences are students, residents and clinicians in the optometric field.

Case Description

Initial visit: consult from emergency department

A 62-year-old Indian male presented to the emergency department complaining of bilateral blurred near vision in conjunction with a headache for a duration of one month. He described the headache as having a gradual onset and affecting the frontal and temporal lobes bilaterally. He also reported experiencing dizziness, intermittent tongue numbness and intermittent slurred speech. The patient denied symptoms of nausea, vomiting or disorientation. His blood pressure in the emergency room was 140/90 mmHg.

Ocular history included refractive error in both eyes and bilateral senile cataracts more advanced in the left eye. The patient had a medical history of non-insulin-dependent diabetes mellitus, hypertension, hypercholesterolemia and coronary artery disease after a CABG. His medications included atorvastatin 80 mg daily, aspirin 325-mg delayed release tablet daily, ezetimibe 10 mg daily, glipizide XL 5-g oral tablet (extended release) daily, metformin 500-mg oral tablet (extended release) daily and metoprolol

succinate 25 mg daily. He had no known allergies and denied any use of smoking, drugs or alcohol. Family history included his mother being diagnosed with diabetes and hypertension.

During the ocular examination, the patient had difficulty reading the distance visual acuity chart due to uncorrected refractive error; therefore, only near visual acuity was measured. Near visual acuity without correction was 20/100, but improved to 20/40 in the right and left eye with a +2.00D spherical lens with pinhole and illumination. A manifest refraction was not performed because this was an emergency room consult. Ishihara color testing was performed with +2.00D spherical lens and the result was 14/14, right and left eye separately.

The pupils were equal, round and reactive to light and revealed (-) afferent pupillary defect in either eye. Confrontation visual fields were full to finger counting in both eyes and the extraocular muscles were full in both eyes. Goldmann tonometry intraocular pressure measurements were 15 mmHg in the right and left eye.

In the right eye, anterior segment examination revealed 360-degree arcus, two round stromal paracentral scars, nuclear sclerotic cataract 2+ with central vacuoles and posterior subcapsular cataract grade 1+. In the left eye, anterior segment examination revealed 360-degree arcus, nuclear sclerotic cataract 2+ and posterior subcapsular cataract grade 1+. Optic nerve head evaluation revealed 0.3 round, pink and distinct optic nerves in both eyes. Mild retinal pigment epithelium macular changes without any holes or tears were observed in both eyes.

Considering the patient's age and symptoms, giant cell arteritis (GCA) needed to be ruled out. Erythrocyte sedimentation rate blood work was normal. A computed tomography (CT) scan of the head was performed and revealed chronic lacunar infarcts with mild chronic microvascular ischemic changes, but no acute complications.

Ancillary testing such as optical coherence tomography (OCT) or a visual field were not done during this visit because the patient was examined after clinic hours. An OCT of the macula and optic nerve head would reveal any occult or subtle changes that could be producing the reduced vision.

The clinical exam findings of the initial consult suggested the reduced vision was secondary to cataracts, and the headaches were deemed to be non-ocular in nature. The patient was given Tylenol for his headaches and advised to return to the eye clinic the next day for a refraction. If the refraction did not improve his vision, an OCT of the macula and optic nerve head would be performed.

Follow-up visit #1: eye clinic

Manifest refraction produced inconsistent findings with varying best distance visual acuities of 20/40- in the right eye and 20/50 in the left eye and no improvement with pinhole. The pupils were equal, round and reactive to light and revealed (-) afferent pupillary defect. Intraocular pressure and slit lamp examination findings were unchanged in either eye since the initial visit.

Macular imaging with Spectralis® OCT (Heidelberg, Germany) was unremarkable with normal foveal contour in both eyes. OCT optic nerve evaluation showed normal retinal nerve fiber layer thickness in both eyes. All other findings were unchanged from the consult the previous day. It was concluded at this visit that the patient's reduction in visual acuity was secondary to cataracts. A cataract extraction evaluation was scheduled for the next available appointment.

Figure 1. (A) MRI of the head without contrast revealed bilateral occipital infarcts. **(B)** CTA of the head and neck showed asymmetry of vertebral arteries.

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Subsequent visits with neurology, neurosurgery and vascular surgery specialists

The patient continued to have headaches, blurred vision, dizziness, intermittent tongue numbness and intermittent slurred speech. Due to the atypical nature of this patient's headaches, the neurologist ordered the following imaging:

- magnetic resonance imaging (MRI) of the brain, which revealed evolving late subacute infarcts involving the bilateral parieto-occipital cortices and no acute intracranial hemorrhages **(Figure 1A)**
- magnetic resonance angiography (MRA) of the brain, which revealed possible arterial dissection involving bilateral distal vertebral and proximal basilar arteries. Based on the MRA findings, the patient was started on anticoagulation therapy with heparin drip and computed tomography angiography (CTA) of the head/neck was ordered emergently.
- CTA of head/neck revealed severe stenosis of vertebral artery segments with no distinct dissection flap identified, no evidence for dissection or hemodynamically significant stenosis in the extracranial segments of the bilateral vertebral arteries, and basilar artery focal flow-limiting stenosis. **(Figure 1B)**

A vascular surgery specialist was consulted on the findings and confirmed intracranial attenuation of bilateral vertebral arteries. The patient was referred to neurosurgery. Neurosurgery decided that no neurosurgical intervention was indicated at that time. Heparin was discontinued and the patient was started on aspirin 81 mg and Plavix 75 mg for three months. The patient's symptoms were stable and a neuro-ophthalmology consult was recommended.

Follow-up visit #2: neuro-ophthalmology clinic

Figure 2. Distinct bilateral inferior quadrantanopsia on visual field testing correlated with the patient's bilateral parieto-occipital infarcts with macular sparing.
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Ocular examination was stable from the first follow-up exam; however, visual field testing was performed. Results revealed bilateral inferior quadrantanopsia with macular sparing. **(Figure 2)** It was explained to the patient that his visual field defect secondary to his stroke was contributing to his blurry vision, especially while doing near work. Inferior field defects affect function in down gaze, which is the natural reading position. The patient was advised to continue care with the neurologist and to call immediately if symptoms worsened, as this could be a sign of changes in his neurological condition. Separate glasses were prescribed for distance and near to provide a greater reading area for the patient. In addition, prisms were discussed as a future option if the patient did not feel comfortable with the new glasses. Prisms in the glasses could potentially bring the inferior view higher in the patient's line of sight. The patient was asked to return for a repeat visual field exam in two months.

At the two-month follow-up examination, clinical findings were stable. Although best-corrected distance visual acuity remained unchanged, the patient felt his vision was better. He was scheduled for repeat dilation and visual field exam in four months and instructed to return to the clinic sooner if he noticed any changes.

Education Guidelines

Learning objectives

1. Understand stroke pathology
2. Understand vascular anatomy along the visual pathway and its correlation with visual field defects
3. How to use different types of imaging to aid in diagnosing strokes
4. How to manage a patient who has experienced a stroke

Key concepts

1. Occipital lobe infarcts and corresponding visual field deficits
2. Assembling the clinical picture with diagnostic tools
3. Recognizing the importance of interdisciplinary care for successful patient management

Discussion points

1. What are risk factors for a stroke?
2. What causes a stroke?
3. Which cerebral artery supplies the occipital lobe?
4. What are the arteries that give the macula a dual blood supply?
5. Should optometric clinicians perform visual field testing on all stroke patients?
6. How can OCT be useful for this patient?
7. What are some indications for neuroimaging in optometry?
8. How can optometrists maximize care for stroke patients?
9. What should be discussed with patients who are at risk for a stroke?

Literature Review

Cerebrovascular accidents have varying neurological effects depending on the location of the lesion and vascular supply involved. It has been reported that the most common visual symptoms of stroke patients are visual field loss, blurred vision, reading difficulty and diplopia. Less common symptoms include oscillopsia, visual hallucinations, depth impairment, photophobia and color disturbances.[3] Spontaneous recovery can occur in up to 40% of patients within the first few weeks and up to six months after injury.[4] The most common visual field defect secondary to CVA is homonymous hemianopsia respecting the vertical meridian. Other defects include homonymous quadrantanopsia or altitudinal defects. This patient presented with bilateral inferior quadrantanopsia, which ultimately formed bilateral altitudinal defect.

Approximately 75% of occipital lobe lesions are from infarctions of the middle cerebral or posterior cerebral arteries.[5] Occipital lobe lesions normally generate contralateral homonymous scotomas that are particularly congruous. This characteristic of congruity is important because it helps differentiate occipital lobe lesions from other lesions in the visual system that produce incongruous visual field loss such as damage to the optic radiations or optic tracts.

J. Lawton Smith‘s review of 100 cases of homonymous hemianopic visual field defects secondary to strokes revealed that the majority of defects were due to occipital lobe lesions.[6] Furthermore, CVAs are the most common cause of homonymous hemianopic visual field defects from the occipital lobe.[7-9] The etiologies of infarctions in the occipital lobe are primarily emboli from the heart or vertebrobasilar artery system.[7]

Discussion

To facilitate the learning experience, the audience for this teaching case report should have basic knowledge of ocular anatomy, visual field deficits and their corresponding anatomical locations, and stroke pathology. The focus of this case presentation is to relate structure to function and vice versa. A review of ancillary testing options available to an optometrist, such as bloodwork, visual fields, OCT and radiological testing, would also be beneficial. To present this case in a teaching manner, a PowerPoint

lecture should include a brief background of cerebrovascular accidents, the case presentation, learning objectives, key concepts, literature review and discussion points. A handout of images and tables should be given to the audience for visual reinforcement.

The learning objectives should be discussed by breaking down the key clinical points of the patient's exam and correlating those findings with a review of anatomy and radiologic imaging. Discussion points should be presented as stand-alone slides. The audience can be broken into smaller groups so learners have the opportunity to deliberate amongst themselves prior to coming to a consensus. Tables and images should be presented throughout the PowerPoint lecture to tie all of the information together. The audience can be given a handout of the patient's chief complaint, history and ocular examination findings from the initial emergency department consult. Other management and treatment options should be discussed from the initial visit, and the urgency of providing appropriate care should be highlighted. Concluding the presentation with the optometrist's role in managing stroke patients and patients at risk for stroke should help the audience to become better clinicians.

The patient's initial complaints of headaches with blurred vision prompted the emergency department to gather an interdisciplinary team to provide the best care for the patient. Not only was the optometrist involved, but also the neuro-ophthalmologist, neurologist, neurosurgeon and vascular surgeon.

It is important to be aware that in addition to headaches and blurred vision, the patient's chief complaints included dizziness, intermittent tongue numbness and intermittent slurred speech. These symptoms can be categorized as precursors to stroke, which is also known as transient ischemic attack (TIA). TIA symptoms cause sudden neurologic impairments that can last minutes or less than 24 hours and are considered "warning strokes." An estimated 15-30% of patients with an ischemic stroke report experiencing a preceding TIA.[10]

Patients are at highest risk of a subsequent stroke within the first week after a TIA (5% within 7 days), but the risk of subsequent stroke at one-year follow-up varies from 4.4% to 21%.[11] Common symptoms of TIA include complete paralysis of one side of the body, sudden loss or blurring of vision, dizziness, confusion, difficulty with comprehension, disorientation and dysphagia. Urgent evaluation, management and treatment can significantly reduce the risk of subsequent stroke.

Table 1. Click to enlarge

What are risk factors for a stroke?

Many factors, including age, diabetes, hypertension, hypercholesterolemia and coronary artery disease, put this patient at high risk for a stroke.[1,12,13] **(Table 1)**

What causes a stroke?

To manage and treat a patient in whom a CVA is suspected, it is important to understand the various types and causes of CVA. Although hemorrhagic strokes are considered to have higher mortality risk, the majority of strokes are secondary to ischemia (10 times more frequent).[14] An ischemic stroke occurs when blood is obstructed (clot) within the blood vessel. Clots can form a cerebral thrombosis or a cerebral embolism. Hemorrhagic strokes occur when the blood vessel is weakened to the extent that it ruptures and bleeds, compressing areas of the brain. Aneurysms or arteriovenous malformations are types of anomalous blood vessels that can cause intracerebral or subarachnoid hemorrhages.

Which cerebral artery supplies the occipital lobe?

The posterior cerebral artery (PCA) supplies blood not only to the occipital lobe but also to the temporal lobe, thalamus, corpus callosum and internal capsule. Branches of the PCA include the parieto-occipital artery, calcarine artery and the anterior, middle and posterior temporal arteries.[15] Branches of the inferior calcarine artery supply the inferior cortex, whereas branches of the superior calcarine artery and parieto-occipital artery supply the superior cortex.[8]

What are the arteries that give the macula a dual blood supply?

The posterior cerebral artery and the middle cerebral artery both supply circulation to the visual cortex, which corresponds to the macular fibers responsible for central vision. Thus, an infarct of the middle cerebral artery can spare the macula because it can still receive nourishment from the posterior cerebral artery. In fact, the macular representation is disproportionately larger than its location in the posterior pole of the occipital lobe. It is estimated that 50-60% of the visual cortex represents only 10-30 degrees of central vision.[16]

Radiologic imaging in this case revealed an occlusion of the vertebrobasilar arteries. Understanding vascular anatomy is crucial for localizing the lesion and managing patients who have had stroke. The circle of Willis is an important vascular structure that sits at the base of the brain. The anterior circulation of the circle of Willis starts with the internal carotid artery (ICA), enters the cranial cavity bilaterally, and divides into the anterior cerebral artery and middle cerebral artery. The anterior communicating artery connects the anterior cerebral arteries from both sides. The posterior circulation includes a single basilar artery that anastomoses the right and left vertebral arteries. The basilar artery also leads to the PCA bilaterally and is connected to the ICA by the posterior communicating arteries.[15] In this case, the patient's vertebral and basilar arteries were impaired, thus the occipital lobe was affected. His visual field loss correlated to an infarct from vertebrobasilar insufficiency. Based on anatomy, this would manifest bilaterally as was the case with this patient. Occlusion of the vertebrobasilar system can manifest not only as homonymous hemianopsia but also as tunnel vision or an altitudinal visual defect. For the patient in this case study, a bilateral inferior quadrantanopsia formed an altitudinal defect with macular sparing in both eyes.

The tip of the occipital lobe lies within the visual cortex in the region of the calcarine fissure. Inferior visual field defects localize lesions to the contralateral upper calcarine cortex and vice versa because the representation of the horizontal meridian is along the base of the calcarine fissure.[17,18] It has been proposed that the visual cortex in the occipital lobe is arranged topographically as V1, V2 and V3 with V1 in the deeper layer and V3 in the superficial layer.[18] A lesion in any of these areas would induce a quadrantanopsia visual field defect. As such, the occlusion of the parieto-occipital artery involving V1, V2 and V3 above the calcarine fissure resembles what was seen in this case: bilateral inferior quadrantanopsia visual field loss as the parieto-occipital artery feeds the superior part of the striate cortex.

Should optometrists perform visual field testing on all stroke patients?

Knowledge of common visual field patterns is helpful when considering differential diagnoses and it also gives optometrists an outlook on how patients see their world. Visual field defects can affect activities of daily living such as driving, walking, reading, etc. Stroke is the most common cause of homonymous hemianopsia, and it is commonly overlooked. In a study by Rowe and the Vision in Stroke group UK, 16% of stroke patients reported no visual symptoms, yet 85% of those patients were objectively shown to have visual impairment.[3] It is important to properly evaluate patients post-stroke whether or not they have symptoms of visual impairment. Homonymous hemianopsia can impede rehabilitation and is related to worse functional outcomes in stroke patients. Therefore, visual field testing should be recommended for all patients who have suffered a stroke involving the cerebral hemispheres.[9]

How can OCT be useful for this patient?

OCT is a useful tool for detecting subtle changes in macular volume, the peripapillary retinal nerve fiber layer, macular ganglion cell layer and optic nerve head.[19] In contrast, seeing retinal nerve fiber loss/atrophy on clinical examination requires at least 50% atrophy in the affected area.[20] GCA was considered a possible diagnosis in this case, but OCT showed normal findings in each eye, enabling any optic nerve head or macular pathology to be ruled out as a contributor to the patient's symptoms. Because the patient's infarcts were in the occipital lobe at the tip of the visual pathway, no atrophy was seen at the optic nerve head.

What are the indications for neuroimaging in optometry?

Without proper imaging, the patient in this case report could have been misdiagnosed, potentially significantly altering management and treatment options. Imaging is crucial in determining the diagnosis, extent and etiology of a stroke. Indications for neuroimaging are listed in **Table 2**. Initially, this patient underwent a CT scan that revealed chronic lacunar infarcts with mild chronic microvascular ischemic changes. Non-contrast head CT scans are the imaging modality of choice for evaluating acute stroke patients because they can identify early stroke signs and testing is relatively quick.[21] The contrast agents used for CT scans are iodinated; therefore, allergies to iodine and renal failure are contraindications.[22] MRI and CT scans are commonly ordered in cases with suspicion for infarcts, but MRI is usually preferred for imaging vertebrobasilar infarcts because MRI is less prone to artifacts and exhibits better contrast discrimination in the posterior fossa.[23]

Table 2. Click to enlarge

MRI is superior to CT for evaluation of soft tissue. The most common MRI pulse sequences are T1 and T2 weighting, which examine how fast the tissue can become magnetized and how fast it loses magnetization. As a general rule, T1-weighted images are better for viewing anatomy and T2-weighted images are better for viewing pathology. With MRI, clinicians also have the option of fat suppression to enhance pathology recognition. Gadolinium is a contrast material commonly used in MRI to show areas of the blood-brain barrier that have been compromised. Diffusion-weighted sequences (DWI) aid better recognition of acute cerebral infarctions within the first hours of a stroke, revealing a bright lesion on the scan. Gradient echo sequences (GRE) have the ability to reveal hemorrhages in patients with underlying vascular malformations, intracerebral hemorrhages or traumatic brain injury.[22] With superior resolution, MRI is commonly the modality of choice in neuro-radiological imaging.[7,23] MRI is contraindicated in the presence of metallic devices such as pacemakers or prosthetics.

CT, CTA and MRA are non-invasive techniques to help doctors visualize the neurovascular anatomy. Blood flow restrictions, intracranial aneurysms or arteriovascular malformations, and patients who are symptomatic for carotid artery disease with transient vision loss (amaurosis fugax) can be tested using these modalities.[7] The advantages of CTA over MRA include better image resolution and faster results. Further, CTA is indicated for patients who have aneurysm clips or pacemakers and patients who are claustrophobic.[22] The advantage of MRA is that it can be performed with or without contrast. Also, MRA can identify high-grade atherosclerotic injuries in the head and neck, carotid and vertebral artery dissection, fibromuscular dysplasia and venous thrombosis.[21] CTA and MRA are associated with lower morbidity than cerebral arteriography, which is the gold standard for detecting aneurysmal compression. However, if the suspicion of aneurysm is significant and CTA and MRA are negative, cerebral arteriography may be warranted.[24]

How can optometrists maximize care for stroke patients?

Co-managing the patient with the neuro-ophthalmologist, neurologist, neurosurgeon and vascular surgeon contributed to the accurate diagnosis in this case. It also enabled successful treatment and management for this patient. Without the proper referrals and the efforts of the involved physicians, the patient could have been misdiagnosed or lost to follow-up.

Optometrists play a crucial role in detecting, treating and managing stroke patients as well as patients who are at risk for stroke. Although patients can be asymptomatic post-stroke, it is important to assess them for any visual impairment. Stroke patients can have visual field defects, ocular motility defects, low vision issues or perceptual defects that can be addressed with various treatment options that optometrists can provide. These options include refraction, prisms, occlusion, orthoptic exercises and low vision aids.[3] The goal is to enhance patients' independence in conducting their activities of daily living. Thus, proper evaluation by a low vision specialist or neuro-rehabilitation optometrist can be advantageous.

What should be discussed with patients who are at risk for stroke?

The ideal treatment for stroke is prevention, which makes patient education crucial. Patients should be educated about risk factors, importance of compliance with follow-ups, and healthy lifestyle recommendations. It has been reported that more than 45% of acute stroke patients were able to regain functional independence in six months.[25] However, depending on the severity of the stroke and the patient's overall health, stroke can result in long-term disability or death.

The patient described in this report is now in the phases of recovery and rehabilitation with the goal of regaining independence. Stroke survivors in rehabilitation programs work with specialists in rehabilitation nursing, physical therapy, occupational therapy, speech language pathology, audiology, recreational therapy, nutritional care, rehabilitation counseling, social work, psychiatry/psychology, chaplaincy and patient/family education.[26]

Conclusion

Cerebrovascular accidents are a leading cause of serious long-term disability. Comprehension of the vascular anatomy of the head is crucial in recognizing the potential for ischemic injuries to the visual system. It is critical that optometrists are aware of the mechanism of strokes and the potential visual and systemic consequences. Educating patients about stroke signs and symptoms gives them an idea of when to seek care and helps to save lives. As primary eyecare providers, optometrists play a key role in identifying stroke patients and patients at risk for stroke. Because patients may present with only ocular manifestations, these encounters need prompt referrals in conjunction with using diagnostic tools to help capture the entire clinical picture.

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