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CE Credit - Case Report(s) & Topic Review

A Stroke of Bad Luck: Bilateral Visual Field Loss in a Young Male

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ABSTRACT

Introduction: Lacunar infarcts are small-vessel ischemic strokes that frequently cause lasting neurologic defects, including homonymous visual field loss.

Case Presentation: A 38-year-old male presented with a history of sudden visual distortion followed by a headache. Best-corrected visual acuity was 20/20 in each eye, and ocular health appeared unremarkable. However, confrontation fields and automated visual field testing revealed a highly congruent right inferior homonymous defect. Neuroimaging subsequently confirmed a 7-mm lacunar infarct in the left corpus callosum, along with multiple chronic cerebellar lacunar infarcts. The patient was co-managed by neuro-ophthalmology, neurology, cardiology, and primary care, with treatment initiated for hypertension, hypercholesterolemia, and hypertriglyceridemia.

Discussion: Lacunar strokes account for approximately 20-30% of ischemic events and may present with subtle visual symptoms. This case demonstrates the value of visual field testing in identifying neurologic pathology and localizing lesions affecting the visual pathway. As primary eye care providers, optometrists are often among the first healthcare professionals to detect stroke-related visual complaints. Recognition of ocular manifestations and timely referral can facilitate earlier diagnosis and intervention.

Conclusion: Long-term management of stroke survivors requires close interprofessional collaboration to manage systemic vascular risk factors, reduce the risk of recurrence, and optimize patient quality of life.

INTRODUCTION

Approximately one-third to one-half of patients who experience a stroke develop residual visual field loss[1,2]. Ischemic strokes account for nearly three-quarters of all strokes in the United States[3]. Lacunar infarcts, also known as lacunar strokes, occur when small arteries measuring less than 1 centimeter (cm) in diameter are obstructed within the cerebral hemispheres of the brain[4,5,6]. These lesions most commonly occur within the visual cortex of the occipital lobe and along the optic radiations[1,7]. Damage to these areas may result in homonymous visual field defects, in which the same side of the visual field is affected in both eyes[7]. The purpose of this case is to highlight the importance of interprofessional healthcare, review relevant in-office diagnostic testing, and discuss the significance of timely diagnosis and long-term management in stroke patients.

CASE REPORT

Initial Visit

A 38-year-old Caucasian male presented to the clinic for a new patient comprehensive exam. The patient reported that approximately one month earlier, he awoke and, while stretching, experienced sudden visual distortion. He described the episode as resembling “an old TV with images moving on the screen”, lasting less than one minute, with a missing portion of vision in the right visual field. He subsequently developed a headache that lasted approximately three days and resolved spontaneously. Since this episode, the patient reported that there was a persistent bright spot in his right visual field, but denied recurrent headaches, nausea, additional vision loss, blurred vision, or loss of balance.

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Following this initial visual event, the patient was seen by his primary care provider (PCP), where he was diagnosed with migraine headaches. The patient was prescribed 50mg sumatriptan for headaches as needed and referred for optometric evaluation and neurologic imaging of the head and neck. At the PCP visit, he was also diagnosed with hypertension and treated with 10mg Lisinopril once daily. Case history revealed that he underwent magnetic resonance imaging (MRI) of the brain and orbits one day prior to his eye examination, and the results were still pending.

Upon examination, uncorrected visual acuity was 20/20 in both eyes. Pupils were equal, round, and reactive to light without an afferent pupillary defect (APD). Extraocular motilities were full and smooth in all directions of gaze in both eyes. Confrontation fields revealed an outer inferior temporal restriction in the right eye and an outer inferior nasal restriction in the left eye. Ishihara color vision testing was normal at 12/12 in each eye.

Anterior segment slit lamp examination revealed normal findings in both eyes. His intraocular pressure (IOP) was 15mmHg in each eye. Dilated fundus examination revealed pink and distinct optic nerves with a cup-to-disc ratio of 0.20 horizontally and vertically in both eyes. A half-disc-diameter flat nevus was noted superior temporal in the right eye and superior nasal in the left eye. Otherwise, posterior segment findings were unremarkable.

Additional testing at this visit included a 30-2 SITA FAST Humphrey Visual Field (HVF) (Figures 1-2), which was reliable in both eyes with minimal fixation losses and no significant false positives or negatives. Automated visual field testing revealed an absolute right inferior homonymous defect with respect to the vertical meridian.

The patient was diagnosed with a congruent right homonymous visual field defect and urgently referred to neuro-ophthalmology. Although this type of visual field defect typically warrants immediate emergency evaluation, the subacute nature of symptoms and ongoing PCP management supported direct referral to neuro-ophthalmology.

Neuro-Ophthalmology Visit

The patient was evaluated three weeks later by neuro-ophthalmology. He reported no changes in visual symptoms. Ocular findings were consistent with the initial optometric examination. Repeat 30-2 SITA FAST HVF demonstrated a stable, highly congruous partial homonymous right-sided inferior visual field defect. An optical coherence tomography (OCT) was taken of the optic nerves; and the retinal nerve fiber layer were normal in both eyes.

MRI demonstrated a 7-mm non-enhancing white matter lesion in the left corpus callosum with associated T1 hypointensity. Several chronic lacunar infarcts were noted in the cerebellum. There was no evidence of optic neuritis or an orbital mass. Given the sudden onset of symptoms, lack of improvement over time, cardiovascular risk factors and chronic cerebellar infarcts, the visual field defect was determined to most likely be ischemic in etiology. The lesion was classified as a suspected lacunar infarct involving the splenium of the left corpus callosum.

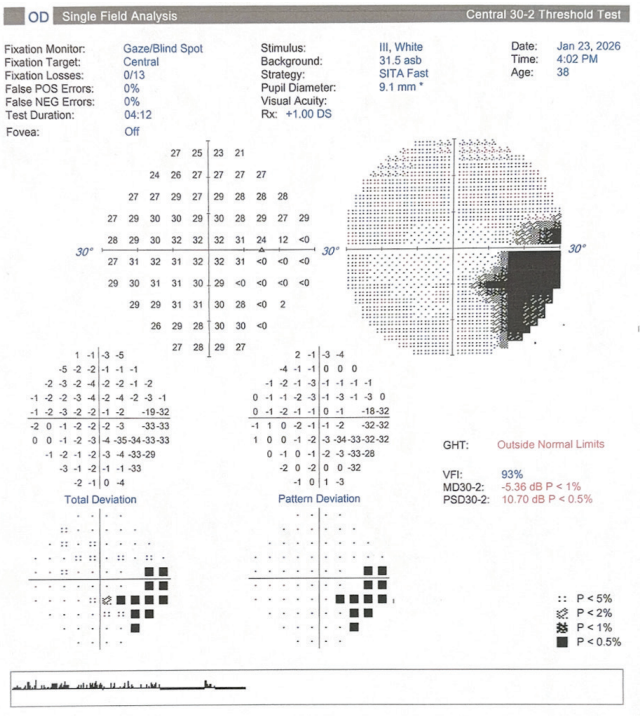

Figure 1: 30-2 SITA FAST HVF of the right eye

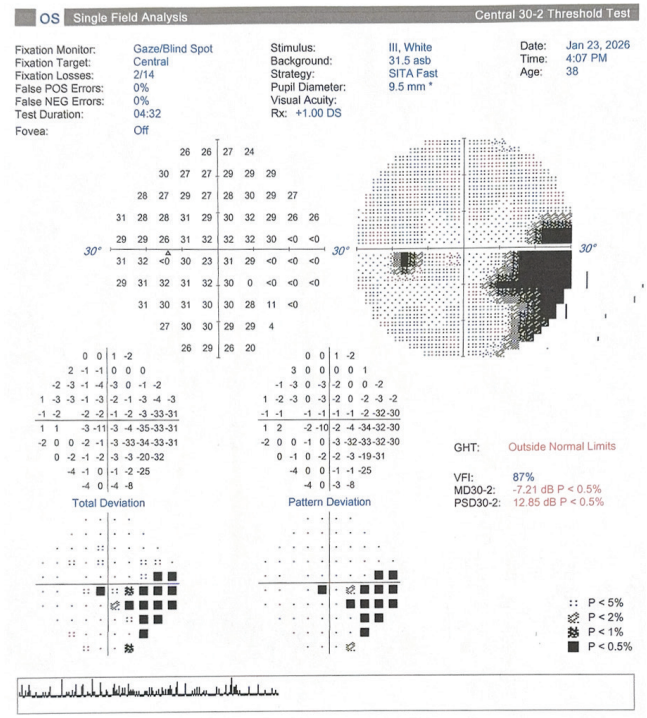

Figure 2: 30-2 SITA FAST HVF of the left eye

The neuro-ophthalmologist referred the patient to stroke neurology and recommended additional stroke work-up, including a computed tomography angiography (CTA) of the brain and neck, repeat lipid testing, and inhalation of 81mg aspirin daily.

Neurology Visit
CTA of the head and neck revealed small hypodensities in the left splenium of the corpus callosum and left occipital lobe around the calcarine fissure. No significant carotid stenosis or other abnormalities were identified. Findings were consistent with the previous MRI results. The patient was instructed to return to clinic in three months for repeat MRI and was referred to cardiology for echocardiogram and electrocardiogram.

Primary Care Follow-up
Lipid panel revealed elevated cholesterol (229 mg/dL), triglycerides (178 mg/dL) and low-density lipoproteins (153 mg/dL). The patient was started on 80mg Atorvastatin daily and counseled regarding dietary modifications. He also reported a persistent cough likely secondary to Lisinopril. The patient was switched to 25mg Losartan daily.

DISCUSSION

Lacunar strokes comprise approximately 20-30% of ischemic strokes[4,8]. Due to the small caliber of the affected vessels in the brain, several etiologies are considered, with lipohyalinosis being among the most common[4]. Other important causes to consider include atherosclerotic disease and cardioembolism[4,5,6]. Chronic hypertension remains major risk factor[5]. Additional risk factors include but are not limited to male sex, age over 55, hyperlipidemia, diabetes mellitus and smoking[4,8,9].

Clinical presentation varies depending on lesion location and may range from asymptomatic disease to significant neurologic impairment. Asymptomatic progression is common in early stages of small vessel disease[4]. Common lacunar stroke syndromes include pure and/or ataxic hemiparesis, dysarthria-clumsy hand syndrome, pure sensory strokes and mixed sensorimotor strokes[5]. Although visual field defects are not classically emphasized in lacunar stroke syndromes, post-chiasmal lesions may produce homonymous visual field loss[6].

Visual field defects occur in approximately one-third to two-thirds of stroke patients[2,10]. Homonymous hemianopia is among the most common visual sequalae and typically results from post-chiasmal lesions affecting the optic radiations or visual cortex[2,11]. Patients may also present with other eye-related symptoms including diplopia, cranial nerve palsies, strabismus, vergence abnormalities and nystagmus[5,11,12]. Transient visual loss may represent a transient ischemic attack (TIA) and should be treated as a warning sign for future stroke[5].

MRI remains the preferred imaging modality for identifying ischemia, edema, and chronic infarcts[13]. CT and CTA provide additional evaluation of vascular anatomy and ischemic burden[14]. In optometric practice, OCT, fundus photography, and automated visual field testing are essential tools for evaluating visual sequelae of stroke. Among these, visual field testing is particularly valuable because characteristic patterns of visual loss can help localize lesions within the visual pathway.

Differential diagnoses in this case included cytotoxic lesions of the corpus callosum (CLOCC), neuromyelitis optica (NO) and multiple sclerosis (MS). However, the patient's stable visual field defect, absence of optic nerve abnormalities, lack of fluctuating neurologic symptoms, and neuroimaging findings favored an ischemic etiology[15,16,17,18].

Acute treatment options for ischemic stroke include intravenous thrombolysis and endovascular mechanical thrombectomy, both of which are highly time dependent[6,19]. Because many patients present outside these treatment windows, management often focuses on antiplatelet therapy and aggressive vascular risk reduction[6]. Once ischemic neuronal damage occurs, it is generally irreversible; therefore, long-term care emphasizes adaptation, prevention of recurrence, and optimization of quality of life[20].

Approximately 50-70% of stroke survivors experience visual dysfunction[1,11]. Optometrists play a critical role in identifying stroke-related ocular manifestations, including retinal artery occlusions, hypertensive retinopathy, visual field defects and TIAs. Early recognition and prompt referral are essential for timely diagnosis and intervention, as it can be life threatening.

CONCLUSION

Lacunar strokes may present with subtle yet clinically significant visual manifestations. This case highlights the importance of objective visual field testing in detecting functional vision loss and localizing underlying cerebral pathology. As accessible frontline providers, optometrist play a vital role in recognizing stroke-related ocular findings and facilitating timely referral. Long-term management requires interprofessional collaboration to reduce systemic vascular risk factors, prevent recurrence, and optimize patient quality of life.

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