CLINICAL VIGNETTE

Homonymous Hemianopia in Primary Care Setting

Amy S. Wang

Introduction

Homonymous Hemianopia (HH) is when a visual field deficit appears on the same side for both eyes. The deficit can be on the outer field of one eye and the inner field of the other eye. HH can present as complete or incomplete visual field deficit depending on the area of injury along the neural ophthalmic pathway from post chiasm to the visual center in the occipital lobe. The visual change can occur suddenly, or it can be a gradual process with patient unawareness. The visual field impairment can increase fall risk as well as adversely affect a patient's daily activities and driving safety. More severe visual field deficits can lead to greater loss of functional ability and increase psychological stress.[1] There is a higher prevalence of depression in the elderly with poor eyesight.[2] Stroke patient with motor deficit in addition to hemianopia have 20% decreases in achieving independence in ambulation and self-care.[2] Often, the visual field defect goes unrecognized in post stroke patients. On the other hand, infarct within the occipital lobe can presents with only visual field changes.[3] The recovery potential within the first 3-5 months is good; therefore, early detection is critical.[2,4] A simple confrontation test in high-risk patients in clinic can lead to early detection and intervention. The following two cases illustrate the different presentations of HH in outpatient primary care.

Case 1

An 88-year-old African American female with hypertension, presented to urgent care with sinus discomfort for 3 days and sudden visual change in her left eye since that morning. Patient was unable to see out of the left side of her left eye unless she turned her body. She attributed her eye symptom to her left sinus congestion with increase nasal drainage and worsening sinus headache. She tried decongestants without improvement. She denied fever or other upper respiratory tract symptoms and was free of dizziness and other focal neurologic changes. She had no blurry vision, no double vision, or eye pain. She normally wears glasses. She does not smoke, drink alcohol, or use recreational drugs. On exam, her blood pressure was 132/86, pulse of 86. Patient was well-developed, well-nourished African American who appeared younger than stated age in no acute distress. Exam was significant for left sided maxillary sinus tenderness, nasal passage swelling, and nasal discharge. Visual acuity showed right eye 20/60, left eye 20/80, and both eyes 20/60 with glasses. Her pupils were equally round and reactive to light bilaterally, extraocular motor intact with normal conjunctiva and sclera. Confrontation testing revealed defects of the left temporal superior and inferior visual fields. In addition, there was also a defect of the right nasal superior visual field. Cardiac and pulmonary exam were normal, and a complete neurologic exam was normal except the visual field change. After consulting with the Ophthalmologist on call, she was transferred to ER for stroke work up. Head CT scan revealed a subacute right sided non-hemorrhagic occipital lobe infarct. She was admitted for observation and eventually discharged with evaluation for optical rehabilitation. Her potential for recovery is good given the early discovery.

Case 2

An 84-year-old Spanish speaking female with hypertension, diabetes, and rheumatoid arthritis was brought to clinic by her friend complaining of generalized body ache for 5 days. Per her friend, patient used to be very active and able to ambulate well. She noted that for past 5 days, patient appeared more tired and weak with complaints of pain in her neck and back. She also had decreased appetite. She denied fever, chill, and upper respiratory tract symptoms. She also denied chest pain, shortness of breath, abdominal pain, dysuria, headache, and acute neurologic changes. Her main complaint was fatigue and back pain. Her medications include methotrexate, metformin, lisinopril, and aspirin. On exam, blood pressure was 102/78, pulse 74, respiration rate of 12, O2 sat of 96%. Patient was a thin, frail appearing lady in a wheelchair in mild discomfort but in no acute distress. She had normal cardiac, pulmonary, and abdominal exams. There was mild tenderness to palpation along the vertebral column from neck to the lumbar region. She also exhibits paraspinal muscle tenderness. Cranial nerves were grossly intact. She did have decreased motor strength in bilateral lower extremities 4/5. Sensory exam was grossly intact. Visual field evaluation with the confrontation test revealed deficits of the right temporal field and a left nasal superior quadrant. Until the examination, the patient was unaware of the visual deficit. She was transferred to ER for stroke evaluation. Stat CT of head subsequently revealed left

sided parieto-occipital infarct with hemorrhagic conversion. It was unclear when her symptom started. She was admitted to the hospital for further management.

Discussion

The visual field of each eye is divided into temporal and nasal fields. The temporal field is enervated by the nasal retinal fiber, while the nasal field is enervated by the temporal retinal fiber of the eye. The neural ophthalmic pathway starts from the optic nerve and travels through optic chiasm. In the optic chiasm, the temporal nerve fiber travel ipsilaterally into the optic tract, while the nasal nerve fiber crosses the chiasm and into the contralateral optic tract. Combined nerve fibers emerge from optic chiasm as optic track on both sides. The optic tract contains both the ipsilateral temporal retinal fiber and the contralateral nasal retinal fiber. From there, the optic tracks pass through the lateral geniculate nucleus becoming optic radiations through the temporal and parietal lobes before terminating at the occipital lobe of the respective side. Any injury or interference along the pathway will result in visual field defect with a specific pattern. A lesion in right optic tract would lead to a nasal visual field deficit of the right eyes and temporal visual field deficit in the left eye, hence the term homonymous hemianopia.

Homonymous hemianopia can be a fixed defect due to tumor, infarct, or head trauma. It can also be a transient change due to migraine, transient ischemic attack, or seizure. Stroke is the most common cause with 58% due to ischemic cerebral infarct and 10% from hemorrhagic infarct with predominant location at the occipital lobe.[5] Multiple sclerosis, CNS infection, degenerative dementia, Creutzfeldt Jakob disease, and severe hyperglycemia are uncommon causes. Complete homonymous hemianopia is the most common form of HH due to the middle cerebral or posterior cerebral artery stroke affecting either the optic radiation or visual cortex of the occipital lobe. Incomplete homonymous hemianopia is when the visual field deficit is not the same for both eyes. The visual field is subdivided into four quadrants. When the damage is at the optic radiation that traveled inferiorly through the temporal or parietal lobe, a quadrant defect will occur. Superior quadrantanopia has its lesion at optic radiation inferiorly in the temporal lobe while inferior quadrantanopia has damage at the parietal lobe. In an incomplete HH, patient can have a quadrant deficit in one eye while the other eye can have deficit in half of the visual field.

The visual field exam with confrontation test should be performed with high-risk elderly or post-stroke patients. Although limited in the primary care setting, visual field testing can be effective to screen for subtle ocular stroke. Taking a good history is key to accurate diagnosis. It is important to note the timing of onset, laterality, pain, redness, and associated ocular symptoms. In addition, it is also important to note comorbid conditions increasing stroke risk and medications that can affect visual symptoms. Exam should include visual acuity with glasses and general inspection of eyes, including extraocular movement and pupil response. Office ophthalmoscopy can detect intra ocular abnormalities. Confrontation testing can be used to screen for neurologic visual field deficit. It has its limitation, but when done properly, it is still a good screening exam. To perform the test, the clinician should be one meter away from the patient. Holding out fingers on the left and right superior quadrant of patient's visual field simultaneously, the clinician can ask how many fingers are being held up on each side. Different number of finger should be used on each hand. The process is repeated for inferior quadrants. This allows for detection of homonymous defect and also determines if it is a hemianopia or quadrantanopia. Subsequently, test each eye individually with finger counting in all four quadrants, both nasal and temporal regions. The nasal quadrant defect can be easily missed and can lead to failure to detect complete or incomplete homonymous hemianopia. Confrontation test has limitations and may underestimate the degree of visual field loss. When a defect is noted, it is important to refer for formal visual field testing using automated perimetry.[4]

Once the visual field deficit is identified, it is important to address and treat the underlying cause. Early initiation of optical rehabilitation with optical, compensatory, or restitution therapy is critical in preserving patient's vision and functional independence. The optical therapy aims to shift the incident ray of light to noninvolved area of the visual field using optical device. A prism is most commonly used. It changes the light path from blind area toward seeing area.[2] Restorative therapies attempt to restore visual function in area of VF defects using specifically designed computer-based software. The theory is that repeated stimuli could reactivate neurons, thus restoring visual function even with only 10-15 % surviving neurons.[6] The compensatory therapies or visual search training is an eye movement therapy. Patient with HH compensate by increasing disorganized saccadic eye movement toward the defect. The therapy improves visual search by training patient to practice organizing large saccades into the hemianopic field.[2,4] It is the preferred approach to rehabilitate hemianopic dyslexia.[2] Systematic scanning with increased head movements can help patient avoid obstacles. All these methods have limited success; however, until further study and new understanding, it is still important to start therapy early.

Conclusion

Vision change with visual field defects always indicates CNS abnormality and should not be ignored. Some patients will have spontaneous recovery or at least improvement of vision within the first few months of stroke with rehabilitation. This makes early detection very critical. Quick confrontation testing can be an effective screening tool in the outpatient setting. It is important for primary care physicians to detect and evaluate for visual field deficit in post-stroke or high-risk patients as early intervention will affect the outcome of preserving patient's vision and quality of life.

REFERENCES

1. **Qiu M, Wang SY, Singh K, Lin SC**. Association between visual field defects and quality of life in the United States. *Ophthalmology*. 2014 Mar;121(3):733-40. doi: 10.1016/j.ophtha.2013.09.043. Epub 2013 Dec 15. PubMed PMID: 24342021; PubMed Central PMCID: PMC3943627.

2. **Agarwal A, Kedar S**. Prognosis and Treatment of Visual Field Defects. *Semin Neurol*. 2015 Oct;35(5):549-56. doi: 10.1055/s-0035-1563573. Epub 2015 Oct 6. PubMed PMID: 26444400.
3. **Sand KM, Thomassen L, Næss H, Rødahl E, Hoff JM**. Diagnosis and rehabilitation of visual field defects in stroke patients: a retrospective audit. *Cerebrovasc Dis Extra*. 2012 Jan;2(1):17-23. doi: 10.1159/000337016. Epub 2012 Mar 22. Erratum in: Cerebrovasc Dis Extra. 2015 Sep-Dec;5(3):114. PubMed PMID: 23060894; PubMed Central PMCID: PMC3468812.
4. **Luu S, Lee AW, Daly A, Chen CS**. Visual field defects after stroke--a practical guide for GPs. *Aust Fam Physician*. 2010 Jul;39(7):499-503. Review. PubMed PMID:20628665.
5. **Zhang X, Kedar S, Lynn MJ, Newman NJ, Biousse V**. Homonymous hemianopias: clinical-anatomic correlations in 904 cases. *Neurology*. 2006 Mar 28;66(6):906-10. PubMed PMID: 16567710.
6. **Pouget MC, Lévy-Bencheton D, Prost M, Tilikete C, Husain M, Jacquin-Courtois S**. Acquired visual field defects rehabilitation: critical review and perspectives. *Ann Phys Rehabil Med*. 2012 Feb;55(1):53-74. doi: 10.1016/j.rehab.2011.05.006. Epub 2011 Dec 7. English, French. PubMed PMID: 22209077.

Submitted July 12, 2016