PEER REVIEWED

Hemi-Spatial Neglect as a Consequence of Acute Cerebrovascular Accident

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Abstract

Hemi-spatial neglect is a neurologic manifestation of a pathological defect along the post-chiasmal visual pathway. This case outlines a discussion of the clinical presentation and diagnosis of this visual dysfunction.

Key Words*: hemi-spatial neglect, cerebrovascular accident, magnetic resonance imaging, computed tomography scan*

Background

Hemi-spatial neglect is a failure of awareness on the side and space opposite the site of brain injury.[1] The most common etiology of hemi-spatial neglect is hemorrhagic or ischemic cerebrovascular accident (CVA), commonly referred to as a stroke.[1] Patients with hemi-spatial neglect fail to recognize or attend to objects on the left or right side of the visual field and are often unaware of these deficits.[2] Optometrists can perform paper- and pencil-testing such as clock drawing, scene copying and line bisection to support the diagnosis of hemi-spatial neglect in-office.[3] The sudden onset of this finding warrants urgent imaging studies such as a computed tomography (CT) or magnetic resonance imaging (MRI) to elucidate the etiology of the deficit.[4] Failure to diagnose acute CVA may occur if the CT scan is negative and the patient is unable to recognize the visual dysfunction.

Purpose

The following case report outlines the outcome of a consult that was placed from the emergency room to the on-call optometrist after a negative CT scan. The role of optometry in a hospital setting is explored, and the pathophysiology, clinical manifestations and treatment of hemi-spatial neglect is discussed. This case would benefit third- and fourth-year students to reinforce clinical competence in neuro-ophthalmic disease as well as optometry residents who take emergency call or work in a hospital setting.

Case Description

An 88-year-old male presented to the emergency department for severe ocular pain, which began 6 hours prior. The pain was described as sharp with 10/10 severity and began in his right eye before traveling to the back of his head. He also experienced mild confusion and disorientation at the onset of ocular symptoms according to his daughter, who was with the patient at the time his symptoms began. The patient denied any other neurologic symptoms, specifically: no paresthesia, unilateral weakness or slurred speech. The patient's medical history was positive for essential hypertension for which he took lisinopril. He did not take any other medication. Social history was negative for tobacco, alcohol or recreational drug abuse. He had no known drug or seasonal allergies. He was oriented to time, place and person and his mood was appropriate.

At the time of admission, the patient's blood pressure measured 105/72 mmHg and his oxygen saturation was normal on room air. The emergency room physician expressed concern for stroke because of the report of confusion and disorientation, and a CT scan was ordered. This scan was read as negative for intracranial hemorrhage and neoplasm 1 hour later. After the CT scan came back negative, the emergency room physician recommended that the patient undergo an eye examination and expressed concern that the patient may have been experiencing acute angle closure glaucoma due to his initial complaint of ocular pain. An emergent consult was placed to the on-call optometrist, who evaluated the patient half an hour later. Before case history could be obtained by the optometrist, the patient experienced a hypotensive crisis, became disoriented and experienced an episode of emesis. The patient's blood pressure measured 88/62 mmHg at this time. The emergency room physician reclined the patient and dispensed IV fluids, and the blood pressure stabilized to 112/75 mmHg. At this time, the optometrist decided to perform a bedside eye examination in the emergency room instead of transporting the patient upstairs to the eye clinic to mitigate the risk of inducing another hypotensive crisis.

Upon examination, the patient denied any visual complaints at distance without correction and had no visual complaints at near through his most recent pair of reading glasses. His last eye exam was more than 1 year ago, and he denied a diagnosis of any ocular condition other than cataracts, which were removed many years prior by a general ophthalmologist. Family ocular history was positive for age-related macular degeneration (mother).

Visual acuity testing was attempted with a Feinbloom chart held at the end of the bed slightly toward the left of the patient, but the patient was unable to see the largest letter with either eye. When the chart was brought closer and held to the right of the patient, he was able to read the lowest line from 5 feet away. Unaided distance visual acuity was recorded as 5/10 OD and 5/10 OS. Pupils were equal, round and reactive to light; no afferent pupillary defect was noted. Confrontation visual field test revealed a restriction of the entire nasal field OD and a restriction of the entire temporal field OS; both field defects respected the vertical midline. Extraocular motility (EOM) evaluation manifested a restriction of movement in left gaze with no voluntary movement to the left OU. The doll's head maneuver was performed, which increased the patient's range of motion to the left in both eyes while the patient attempted to maintain his gaze straight ahead toward the examiner. Cover test was orthophoric at distance and near. Perkins applanation tonometry measured 11 mmHg OD and 11 mmHg OS at 12:39 a.m. Slit lamp biomicroscopy revealed normal adnexae, lids, lashes, puncta and palpebral and bulbar conjunctivae in both eyes. The right and left cornea were clear. Both anterior chambers were quiescent without evidence of cells or flare; estimations of the temporal and nasal angle were >1:1 by Van Herick technique OU. Both irides were flat and blue. Pupils were dilated using one drop of 1% tropicamide and one drop of 2.5% phenylephrine OU. Evaluation of the posterior segment revealed posterior chamber intraocular lenses without posterior cortical opacification OU. Fundus assessment revealed optic nerve cup-to-disc ratios of 0.40/0.40 OD and 0.45/0.45 OS. Both cups were deep with robust rim tissue 360 degrees and no evidence of pallor or edema. Both maculae were flat and clear. The vitreous was optically clear OU. The right eye had a Hollenhorst plaque at the superior temporal arterial bifurcation OD; the vasculature was normal OS. The periphery was flat without breaks or tears 360 degrees OU.

The restriction of the nasal field in the right eye and restriction of the temporal field in the left eye upon confrontation fields in addition to the lack of voluntary eye movement to the left suggested a diagnosis of left hemi-spatial neglect secondary to an acute post-chiasmal CVA. Cognitive testing was not performed at this time as priority was given to expedited neurological imaging. An MRI of the brain and orbits with and without contrast was recommended to the emergency department as well as admittance to the inpatient neurology service. Carotid duplex imaging was also recommended to explore the etiology of the Hollenhorst plaque observed on fundus exam. The patient and his daughter were educated regarding the exam findings and the suspicion for stroke as well as the need to obtain an MRI to rule out other etiologies of his visual field loss. A follow-up was scheduled for the next day with the in-patient optometry

service.

Follow-up visit #1

The exam was performed bedside at the in-patient ward because the patient continued to have mobility restrictions. He continued to report no visual complaints at distance without correction or near with his reading glasses and had no complaints of visual field loss in either eye. Visual acuity remained stable with uncorrected distance visual acuity recorded as 10/10 OD and OS with the Feinbloom chart held at the end of the bed while ensuring that the chart was held only toward the right of the patient's visual field. Entrance testing was unchanged from the previous examination with no improvement of the left visual field restriction with confrontation field testing OU. EOM continued to show no voluntary leftward eye movement. Hand-held slit lamp biomicroscopy revealed normal lids, lashes, conjunctivae, cornea, anterior chambers and irides. The patient was not dilated at this time, and the posterior health examination was performed with an ophthalmoscope. Fundus assessment revealed optic cup-to-disc ratios of 0.40/0.40 OD and 0.45/0.45 OS. Both cups were deep with robust rim tissue 360 degrees and no evidence of pallor or edema. Both maculae were flat and clear. The vitreous was optically clear in both eyes. The Hollenhorst plaque in the right eye was no longer present at the superior temporal bifurcation OD, and the vessels appeared normal in caliber without occlusion OU.

Following the ocular health examination, the patient was asked to describe the entire room around him and name objects that he could see. He pointed out several objects on the right side of the room including a clock, hospital bed, doctor and window. He did not voluntarily name or describe any objects on the left side of the room. He was then given a blank clock-drawing template and was asked to fill in all of the numbers around the clock and to draw the long and short hands. The patient completed the clock as shown in **Figure 1**.

He was then given a line bisection test to complete. The patient was asked to draw a vertical line through the middle of all of the horizontal lines on the paper in front of him. He completed the test as shown in **Figure 2**.

The patient was then given a scene copying test to perform. He was asked to redraw the pictures he saw below the original version. The scene was placed on a clipboard, and the patient completed the test as shown in **Figure 3**.

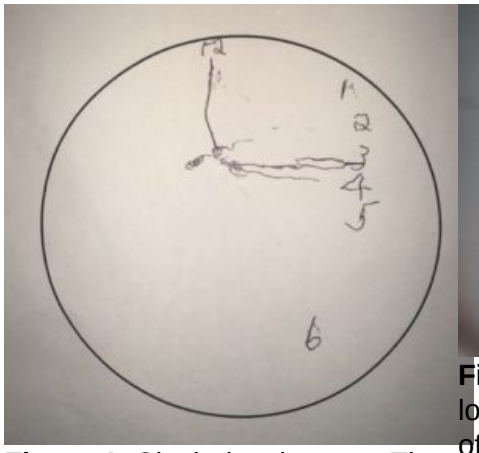

Figure 1. Clock drawing test. The patient filled in numbers 12 through 6 and then drew the long and short hands to represent 3 p.m. When asked to fill in the rest of the numbers, he responded, “I think that’s all there is.” Click to enlarge

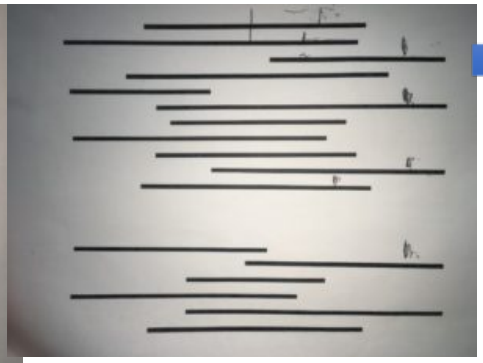

Figure 2. Line bisection test. The long thin vertical line in the middle of the top bar was completed as a reference for the patient to indicate how to complete the test. The remaining vertical lines represent the patient’s attempt to bisect the bars with a vertical line. The vertical lines were drawn far to the right of the center of each horizontal bar, and most of the horizontal bars on the left side of the paper were ignored completely. Click to enlarge

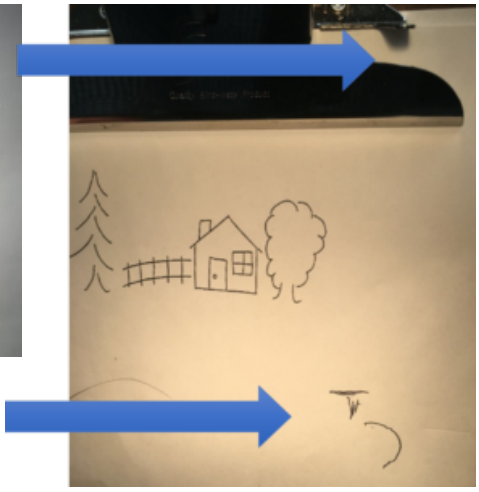

Figure 3. Scene copying test. The patient drew very little and said, “I think that is it.” It was initially assumed that the patient had a poor understanding of the procedure, and the test was discontinued. Later, upon closer inspection, it was observed that the patient had in fact drawn the upper right aspect of the clipboard. The top arrow points to the upper right aspect of the board’s metal clip, and the bottom arrow points to the patient’s drawing of the metal clip. The entire scene of the trees, house and fence is likely on the left side of the patient’s visual field within the area he cannot attend to as a result of hemi-spatial neglect. Click to enlarge

A diagnosis of left hemi-spatial neglect was made at this visit, supported by the following observations: the repeatability of left field restriction on confrontation fields, a lack of voluntary eye movement to the left side on EOM testing, failure to draw shapes or numbers on the left side of paper neglect testing, and the denial of any vision complaints. The patient was scheduled to return to the eye clinic in 1 week for Humphrey visual field testing if he was able to be transported safely from the inpatient ward.

Follow up visit #2

The patient was seen at the outpatient optometry clinic 1 week later for visual field testing. The radiology report of the MRI of the brain had been completed by this point and was read as a “gyriform T2 hyper intensity in the right occipital cortex and subcortical white matter with minimal extension into the parietal and temporal lobe.” The patient was placed on intravenous unfractionated heparin by the in-patient neurology team to mitigate the risk of further ischemia. An axial section of the patient’s MRI is shown in **Figure 4**.

After the MRI results were reviewed with the patient, he was asked to perform a Humphrey visual field 24-2 SITA-FAST test. The patient completed the test as show in **Figure 5**.

The loss of visual sensitivity of the left half of the visual field confirmed the repeatable results of previous

confrontation field testing and also correlated to the cortical infarction observed on the right side of the occipital lobe on the MRI of the brain. The findings were explained to the patient and family, who were interested in the prognosis of visual recovery. The patient and family were informed of the guarded prognosis of any recovery in vision, but potential visual rehabilitation options to improve function were reviewed. The patient and family chose to consider visual rehabilitation options after the patient had stabilized medically, as he had experienced multiple hypotensive crises since he was admitted to the neurology ward. The prognosis for the patient's neurological and systemic condition mirrored the guarded prognosis of visual recovery, and management of the patient's CVA and systemic hypotension was deferred to the in-patient neurology team. A follow-up visit was scheduled for 1 month.

Follow up visit #3

The patient returned 1 month later for dilated fundus exam and repeat neglect testing. He reported no new visual symptoms but indicated an awareness of the visual field defects for the first time. He was given a blank clock drawing template and asked to fill out the numbers and to draw hands to indicate 3:45 p.m. The result is shown in **Figure 6**.

The anterior and posterior segment health was unchanged in both eyes upon examination. Consideration was made for visual rehabilitation strategies at this visit, but the patient continued to experience other systemic complications related to his CVA, which limited his ability to participate in rehabilitative therapy. Follow-up was scheduled for 1 month to explore visual rehabilitation options in the event that he was stabilized medically at that point. However, the patient passed away soon after this exam as a result of an infection from a catheter that was placed in the medical ward.

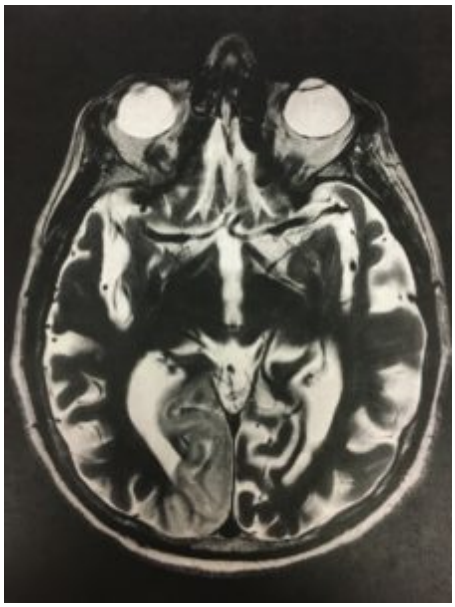

Figure 4. Axial section of the patient's MRI of the brain with contrast. An infarction of the right occipital cortex can be seen as hyperintensity of the gyri in that region.
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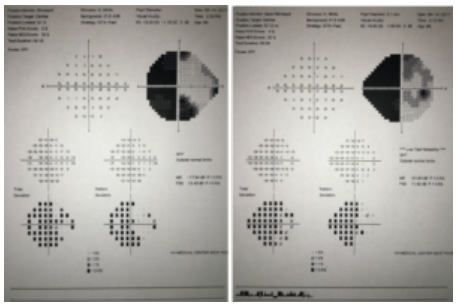

Figure 5. Humphrey visual field 24-2 SITA-Fast test results in the left eye (left) and right eye (right) revealing decreased sensitivity of the left visual field respecting the vertical midline OU. The high rate of false positives lowers the overall reliability of the results, which may explain some of the defects that appear to the right of the midline OU. Click to enlarge

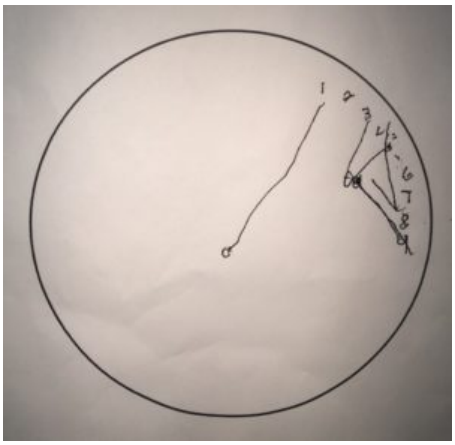

Figure 6. Repeat clock drawing test. The patient included more numbers than he did in his initial clock drawing test but confined the numbers to the right side of the clock face. Hands from the number 3 to 9 are drawn with other hands drawn in error. The patient reported, "this does not look right" regarding the placement of his numbers but did not want to repeat the test.
Click to enlarge

Education Guidelines

Key concepts

1. In-depth understanding of the visual pathway is key to localizing potential intracranial lesions that result in visual field dysfunction

2. Optometrists should know the visual manifestations of acute stroke so the patient can be diagnosed and treated quickly
3. A full differential diagnosis for hemi-spatial neglect should be considered in patients with acute presentation
4. Visual neglect can be detected with careful analysis of ocular and cognitive testing
5. Follow-up with patients after diagnosis of hemi-spatial neglect is key to tracking any partial recovery that takes place

Learning objectives

At the conclusion of this case report, readers should be able to:

1. Identify the potential visual manifestations of acute stroke
2. List the differential diagnoses in patients with acute one-sided field loss
3. Differentiate homonymous hemianopia from hemi-spatial neglect
4. Describe expected results of paper- and pencil-testing in patients with hemi-spatial neglect
5. Explain the connection between the location of a lesion along the visual pathway and the location of the visual field defect
6. Understand the importance of CT scan and MRI in the diagnosis of acute CVA

Discussions questions

1. What are potential etiologies of hemi-spatial neglect?
2. How does the location of the lesion along the visual pathway impact the way in which the visual field can be affected?
3. What are neurological symptoms other than vision loss that are associated with intra-cranial lesions that develop posterior to the chiasm along the visual pathway?
4. How does the location of a lesion along the visual pathway impact the way in which vision loss manifests?
5. What regions of the brain are implicated in the pathophysiology of hemi-spatial neglect?
6. What theory explains the reason why left-sided neglect is more common than right-sided neglect?
7. How does the prognosis for recovery from hemi-spatial neglect differ in ischemic vs. hemorrhagic stroke?
8. Why is CT scan indicated before initiation of intravenous tissue plasminogen activator (tPA) after diagnosis of acute CVA?
9. What are potential visual rehabilitation options for patients with homonymous hemianopia and neglect?

Learning assessment

- Review images of normal intracranial MRI to orient students to the left and right side and identify important structures along the visual pathway
- Break students up into small groups and task them with researching the conditions that are considered as part of the differential diagnosis for acute hemi-spatial neglect; then have students compare and contrast the signs and symptoms of these conditions
- Assign a student as doctor and another as patient and have them practice patient education for acute stroke and visual field loss in a role-playing scenario
- Give students blank clock chart diagrams and have them draw out the results they are likely to see in patients with hemi-spatial neglect

Discussion

Hemi-spatial neglect is a failure of awareness on the side and space opposite the site of brain injury.[1]

Typically, this defect is a result of right-sided brain injury.[1] The most common etiology of hemi-spatial neglect is acute CVA but it may also be caused by traumatic brain injury (TBI) or neoplasm.[5,6,7] Other etiologies such as focal inflammation and infection can occur but are uncommon.

Hemi-spatial neglect should be differentiated from field loss such as homonymous hemianopia. While both of these dysfunctions represent loss of visual sensitivity of one half of the visual field,[8] homonymous hemianopia alone does not manifest the loss of attention or awareness to the side of the field that patients can no longer visualize.[8] In other words, patients with homonymous hemianopia who do not exhibit neglect are almost always aware that half of their visual field is missing in both eyes and can consciously draw their attention towards the side of their visual field that has diminished sensitivity.[9]

Neurologic visual field defects manifest differently depending on their location along the visual pathway. Loss of visual sensitivity in the right or left visual field in both eyes is indicative of a lesion along the visual pathway posterior to the optic chiasm.[8] This field loss is described according to how similar the defect is between the two eyes, with more similar visual field defects being more congruous. The more posterior the lesion along the pathway, the more congruous the defect appears.[10]

There are often other neurologic symptoms that patients experience along with visual field loss, depending on the size and extent of the lesion. These symptoms include slurred speech, unilateral limb weakness and paresthesia but may include other processing deficits such as simultanagnosia, anosognosia and anosodiaphoria.[11,12] The neurologic presentation of hemi-spatial neglect is likely due to the proximity of spatial awareness processing to the occipital lobe.[13,14] Hemi-spatial neglect can manifest as the loss of visual attention to one half of the visual field, but patients may also experience verbal, motor or tactile neglect.[15] These other manifestations of neglect were not observed in this patient. It is interesting that this patient experienced acute ocular pain before his diagnosis of CVA, as ocular pain is not a typical presenting symptom of stroke. However, acute headache has been reported in up to 35% of patients with acute ischemic CVA,[16] which the patient may have erroneously localized to his eye.

The pathophysiology of hemi-spatial neglect is not fully established, but several theories have described potential mechanisms.[17] One theory implicates the “peri-sylvian neural network,” which represents the cortical connections between the inferior parietal, superior/middle temporal, and ventral parietal lobes.[18] The interconnections between these regions of the brain may be involved with determining the patient’s ego-centric space.[17,18] Another unique feature of hemi-spatial neglect is that patients exhibit left hemi-spatial neglect much more often than right-sided neglect. Approximately 20% of patients with acute brain damage exhibit some form of hemi-spatial neglect, while approximately 50% of patients with right-sided brain damage exhibit signs of neglect.[19] The theory behind this discrepancy is a postulated redundancy in cortical processing of the right visual field. According to this “coding theory,” the left visual field likely receives processing only from the right side of the brain, while the right visual field is likely processed by both the right and left brain.[20] Therefore, if the right visual field is processed by both sides of the brain, the right side of the brain may still be able to process the right visual field in the presence of left-sided brain injury. This disparity in processing accounts for the disproportionally high number of cases of left hemi-spatial neglect because in the presence of right-sided brain injury, the left side of the brain would not be able to properly process the left visual field.[17]

The anterior and posterior segment health evaluations are often unremarkable in cases of intracranial pathology such as stroke. However, it is important to conduct a complete ocular health examination to rule out ocular pathology that may result in visual field loss. Specifically, the optic nerve should be carefully examined to rule out conditions such as arteritic ischemic optic neuropathy, optic neuritis and glaucoma. Peripheral retinal examination should also be conducted to rule out peripheral pathology such as retinal detachment. The non-occluding Hollenhorst plaque that was observed in the patient’s right eye upon initial examination was of no visual consequence but may have been part of the larger plaque that

ultimately occluded the posterior cranial vasculature.

The differential diagnosis of hemi-spatial neglect should include any intracranial pathology that may result in compression or dysfunction of cortical matter along the post-chiasmal visual pathway. These etiologies include intracranial neoplasm, focal inflammation, infection and hemorrhage.[6,7,21] The differential diagnoses considered in this patient after the initial exam included:

- **TBI.** TBI results from trauma and can cause visual field defects due to compression of the visual pathway by secondary hemorrhaging.[5]
- **Intracranial neoplasm.** Intracranial neoplasm can cause similar visual field defects to CVA or TBI due to mass effect along the post-chiasmal visual pathway.[6]
- **Migraine with aura.** Visual aura associated with a migraine may transiently obstruct vision, which may mimic visual field defects such as homonymous hemianopia.[22]
- **Multiple sclerosis.** Multiple sclerosis is a demyelinating disease of the central nervous system characterized by inflammatory attacks, which can cause visual field defects if the inflammatory lesion develops along the visual pathway.[23]

Most of these conditions can be differentiated with an MRI of the brain and orbits with and without contrast.[4,6] For this reason, an MRI should be considered in all patients with new onset hemi-field dysfunction.

A myriad of cognitive testing methods have been developed to aid in the diagnosis of hemi-spatial neglect. Paper- and-pencil testing is a widely used tool for diagnosing hemi-spatial neglect and conceptualizing the extent of the dysfunction. Clock drawing, scene copying and line bisection tests are all examples of paper- and pencil-testing that were utilized in this case. Clock drawing requires placement of analog clock numbers within a pre-drawn circle,[24] scene copying entails having a patient redraw a series of pictures below the originals,[25] and the line bisection test has the patient draw a vertical line in the middle of a series of horizontal lines.[3,26] Multiple tests should be performed to confirm diagnosis, as the sensitivity of paper- and pencil-testing is significantly higher when multiple tests, rather than one, are performed.[3] In this case, paper- and pencil-testing was useful in confirming the diagnosis of hemi-spatial neglect and educating the patient's family members on the nature and severity of his condition.

Automated visual field testing is also important in order to document the location, depth and size of the sensitivity loss.[27] Many parameters may be employed when testing visual field loss with Humphrey visual fields. A larger testing parameter such as 30-2 may detect more peripheral defects but takes longer to perform.[28] In patients who fatigue easily or have a poor attention span as a result of stroke, a parameter such as a 24-2 SITA-FAST may be employed to decrease errors in reliability such as fixation loss, false positives and false negatives.[28] Visual field testing is also a useful way to educate the patient and family on what the patient is experiencing as well as monitor for any improvement, stability or decline in visual sensitivity.

Prognosis for visual recovery from visual field defects resulting from stroke is guarded. However, up to 50% of patients may experience spontaneous visual recovery within the first 3-6 months.[29] Visual field defects following hemorrhagic CVA may improve somewhat as the blood reabsorbs, but visual field defects as a result of ischemic infarction have a poorer prognosis because the tissue is no longer viable.[30]

The initial management of neglect depends on the time of symptom onset as well as the etiology. Intravenous tPA is indicated in cases of ischemic stroke.[31,32] The Food and Drug Administration currently only approves the use of tPA within 3 hours of symptom onset, though there is evidence to support its use up to 4.5 hours.[31,33] However, a CT scan must be obtained first to ensure that the etiology is not

hemorrhagic, as tPA may exacerbate intracranial infarction if bleeding is observed. This presents a unique challenge to acute medical providers, as approximately one-third of patients with acute stroke do not present with detectable signs on CT scan within 6 hours of symptom onset.[34] The emergency room attending physician in this case made the decision not to administer tPA based on the negative CT scan as well as the absence of any neurologic symptoms. By the time the eye examination had been initiated, the window for tPA administration had already expired.

After the etiology and extent of the CVA has been established, management focuses on mitigating the risk of subsequent strokes and treating any secondary neurological complications. Patients may be placed on anticoagulants such as aspirin or heparin to keep emboli from forming and traveling to the heart, lungs or brain.[35] Alternative methods such as lowering blood pressure, hemostatic therapy, minimally invasive surgery, anti-inflammation therapy and neuroprotection are also being explored.[36]

Visual rehabilitation may also be considered in patients who have stabilized medically after their initial stroke. The goal of therapy is typically compensatory in nature. While there is no standard of care regarding management of hemi-spatial neglect, many strategies have been proposed to help patients improve their function. The use of prism has been reported to help patients detect visual stimuli in the missing visual field,[37-39] but more research is needed to determine the long-term efficacy of this method. Other techniques such as teaching the patient how to scan more extensively with exploratory eye movements have also been suggested, though the patient's restricted attention span is often a limiting factor.[39] Unfortunately, none of these strategies was implemented in this case, as the patient never entirely stabilized and passed away before visual rehabilitation options could be fully explored.

Conclusion

Acute CVA should be included in the differential diagnosis of patients presenting with acute hemi-spatial neglect. This case demonstrates the importance of careful entrance testing analysis, as the decision to obtain an MRI was based upon clinical evidence of stroke, even in the absence of pathology on CT scan. It is important for clinicians to be aware that acute stroke may not manifest on a CT scan, and an MRI is indicated if there is still suspicion for CVA. Although the prognosis for recovery in hemi-spatial neglect is generally guarded, spontaneous improvement may occur. Patients should be extensively educated and followed closely to monitor for improvement, stability or decline in their visual field and mental status.

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