Article • Optometric Evaluation and Management of Neuromyelitis Optica (NMO)

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ABSTRACT

Background: Neuromyelitis optica, a debilitating condition now regarded as a distinct disease entity, can be distinguished from other demyelinating conditions through optometric testing, including visual evoked potential, optical coherence tomography, and Goldmann visual field.

Case Report: A 19-year-old African American female was referred by a local emergency department after presenting with asymmetric bilateral vision loss associated with numbness and tingling. After an episode of optic neuritis and myelitis, a brain MRI not meeting the diagnostic criteria for multiple sclerosis at disease onset, and detectable AQP4 antibodies, the patient was diagnosed with neuromyelitis optica. This report follows the patient's visual findings and miraculous recovery over seven visits. Several findings are tracked, including visual acuity, color vision, visual fields, macular and optic nerve thickness, and visual evoked potential.

Conclusion: This case report demonstrates the corresponding and confirmatory roles of specialty optometric testing and may help identify future research treatment options.

Keywords: ColorDx, Goldmann visual field, neuromyelitis optica, optical coherence tomography, visual evoked potential

Introduction

Neuromyelitis optica (NMO) is an immune-mediated chronic inflammatory disease of the central nervous system.[1] The condition is characterized by optic neuritis, myelitis, and two out of three of the following supporting criteria: spinal cord magnetic resonance imaging (MRI) lesion extending over three or more vertebral segments, brain MRI not meeting the diagnostic criteria for multiple sclerosis (MS) at disease onset, and NMO-IgG seropositive status, which indicates detectable aquaporin-4 antibodies (AQP4-Ab) in serum studies.[1,2] The role of antibody testing is important to help distinguish optic neuritis in NMO from that in MS and should be performed to initiate treatment as soon as possible.[3] The prevalence of the condition is estimated to range from 1 to 4.4 per 100,000, with a ratio of 9:1 female to male. Although the median age of onset is between 30 and 50 years, cases of childhood and elderly onset have been reported.[2,4] The condition may be monophasic, but it is more commonly found to be relapsing, with two to six attacks separated by periods of months to years of remission.[5] Autoimmune associations have been found with myasthenia gravis, systemic lupus erythematosus, Sjögrens syndrome, celiac disease, and sarcoidosis.[4] NMO can be assessed by an eye care professional taking a detailed patient history, conducting a thorough clinical evaluation, looking for characteristic physical findings, and investigating using a variety of specialized tests such as visual evoked potential (VEP), optical coherence tomography (OCT), and/or visual field.

Case Report

A 19-year-old African American female presented to the emergency room with bilateral vision loss that began in her left eye and progressed to her right eye within one week. Three months prior to any visual symptoms, she experienced occasional numbness and tingling down her legs, as well as difficulty making a tight fist with both hands. She described experiencing white spots that began peripherally and grew bigger to eventually become blurry vision with missing pieces centrally. Her visual acuity was measured to be 20/800 in her right eye and hand motion in her left. After six

days of diagnostic testing, the patient was diagnosed with NMO. This diagnosis was supported by having an episode of optic neuritis, an episode of myelitis, a brain MRI not meeting the diagnostic criteria for MS at disease onset, and detectable AQP4 antibodies, all of which were diagnosed after being admitted to the hospital.

The patient's MRI from her hospital visit revealed a faint abnormal enhancement of the posterior intraorbital portion of the left optic nerve extending posteriorly to the pre-chiasmatic portion of the nerve. There was a possible abnormal enhancement of the prechiasmatic portion of the right optic nerve, and there was also believed to be a very faint abnormal enhancement of the prechiasmatic portion of the right optic nerve. Both optic nerves were reported to be within normal limits in size, and the intraorbital contents were otherwise unremarkable. The abnormal enhancement of the posterior left optic nerve and prechiasmatic portion of both optic nerves is most consistent with the demyelinating process seen in optic neuritis. These findings can be seen in Figure 1.

Prior to being discharged from the hospital, the patient was prescribed prednisone 10mg daily for inflammation, pantoprazole 40mg QAM for gastroesophageal reflux, gabapentin 300mg TID for peripheral neuropathy, and diphenhydramine 25mg Q6H and acetaminophen 325mg Q4H for mild pain. She was referred to The Eye Center (TEC) at Southern College of Optometry for a complete vision evaluation.

At her first visit to TEC (Visit 2 in Table 1), about three weeks after being admitted to the hospital, she reported that her vision had slowly started to improve, starting in her right eye and followed by her left. Although her leg numbness had dissipated, she was experiencing intermittent joint pain and was regularly using ice for relief. She denied any history of headaches, double vision, alcohol abuse, tobacco abuse, or any other medical or ocular history prior to being admitted to the hospital.

This patient was followed closely due to her rapidly changing symptoms and unstable findings. She was seen every three weeks between Visits 1 and 4, ten weeks between Visits 5 and 6, and twelve weeks between Visits 6 and 7. Over the period of six weeks, her acuity improved in her right eye from 20/80 to 20/20. In her left eye, over a period of nineteen weeks, her vision improved from 20/150 to 20/20.

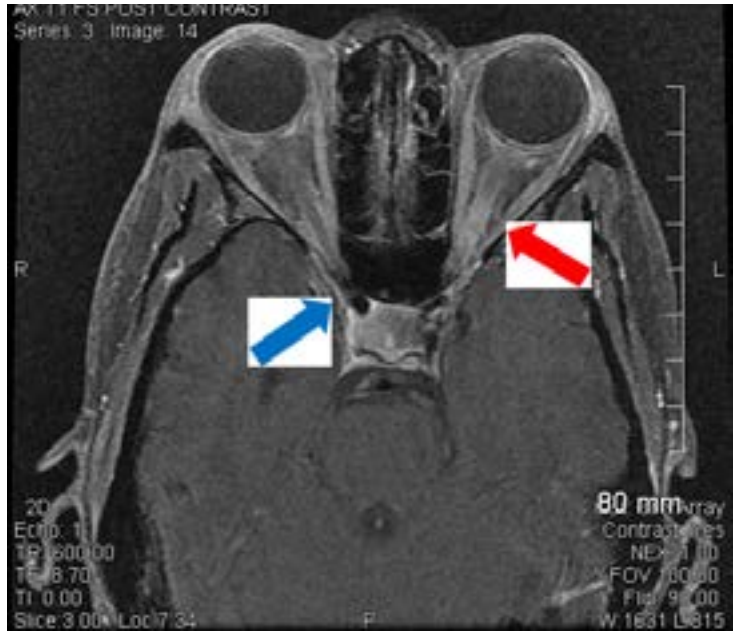

Figure 1. *MRI of the orbits obtained at the patient's hospital visit (Visit 1). Red arrow: faint abnormal enhancement involving the posterior intraorbital portion of the left optic nerve extending posteriorly to involve the pre-chiasm. Blue arrow: very faint abnormal enhancement of the prechiasmatic portion of the right optic nerve.*

At Visit 7, she was able to see 20/15 in all conditions. It is important to note that at each visit, there was no improvement in visual acuity upon refraction. At Visit 2, her contrast sensitivity was reduced but equal in both eyes at 40%. Over time, her contrast sensitivities improved, more so in her right eye compared to her left eye, but they still remain abnormal at 25% in her right eye and 32% in her left eye; normal contrast sensitivity being 3%. Pupils were always noted to be equal, round, and reactive to light, with no relative afferent pupillary defect. Eye movements were found to be smooth, accurate, full, and extensive (SAFE). A decrease in color vision was noted in both eyes, worse in the left eye compared to the right. A summary of

Table 1. Entrance Test Findings over Seven Consecutive Visits (Visit 1: hospital, Visits 2-7: TEC)

Visit		1	2	3	4	5	6	7
Visual acuity	OD	20/800	20/80	20/40	20/20	$20/20^{+}$	$20/20^{+}$	$20/15^{+}$
	OS	HM	20/150	$20/40^{+}$	$20/40^{+}$	$20/25^{-}$	$20/20^{-}$	$20/15^{-}$
Contrast sensitivity	OD		40%		25%		25%	
	OS		40%		32%		32%	
Pupils	PERRL (-) APD							
EOMs	SAFE							
Color vision	OD			2/6		*		^
	OS			1/6		*		^

HM: Hand motion
SAFE: Smooth accurate full extensive
*See Figure 2 ^See Figure 3

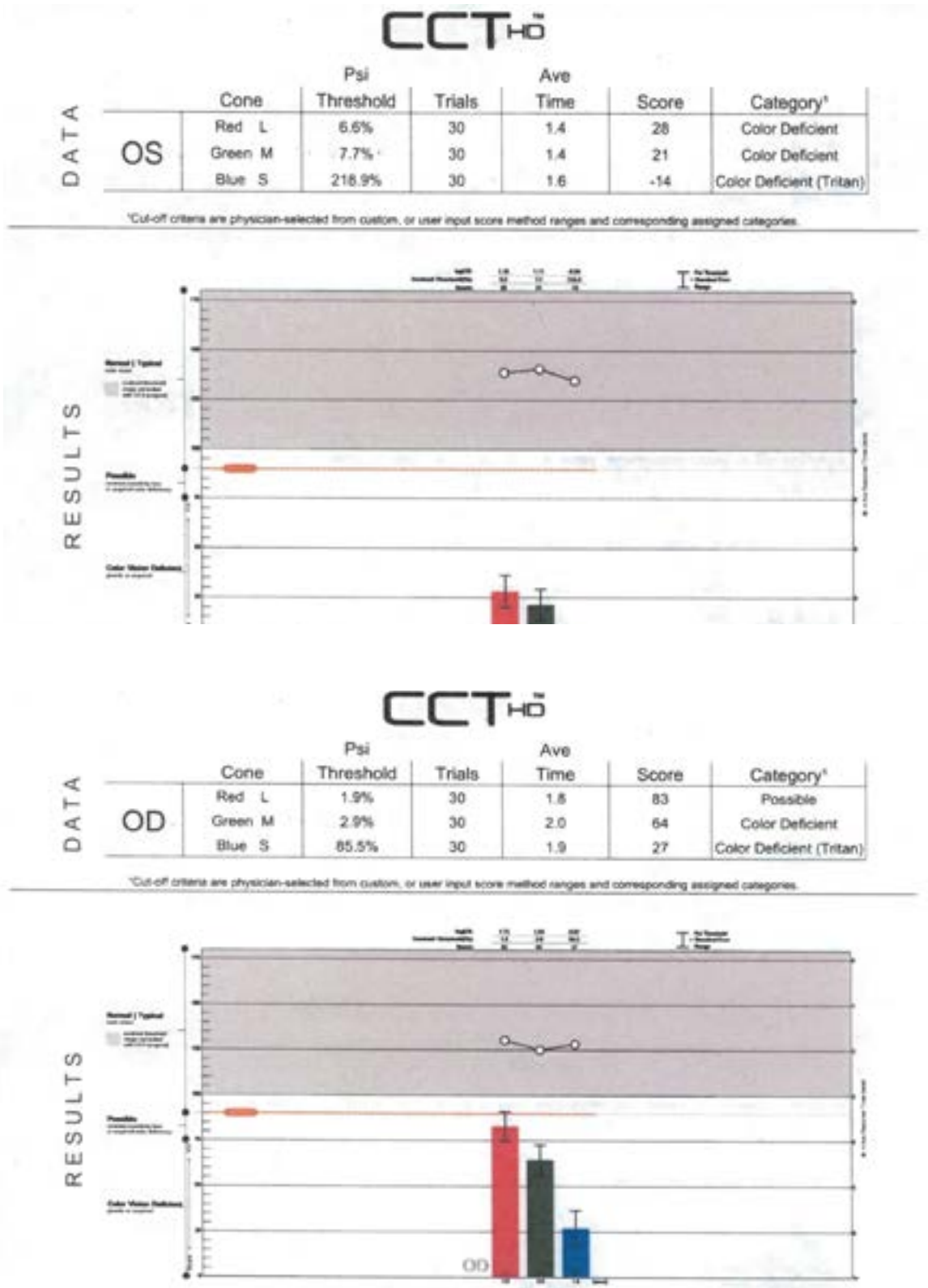

Figure 2. *ColorDx obtained at Visit 5, OD (top), OS (bottom)*

CCT HD

DATA

	Cone	Psi Threshold	Trials	Ave Time	Score	Category*
OD	Red L	1.3%	30	2.0	98	Normal
	Green M	1.7%	30	1.6	87	Possible (Deutan)
	Blue S	16.2%	30	1.4	99	Normal
OS	Red L	2.3%	30	1.6	73	Color Deficient (Protan)
	Green M	1.8%	30	1.6	84	Possible
	Blue S	21.5%	30	1.4	87	Possible (Tritan)

*Cut-off criteria are physician-selected from USAF, or user input score method ranges and corresponding assigned categories.

RESULTS

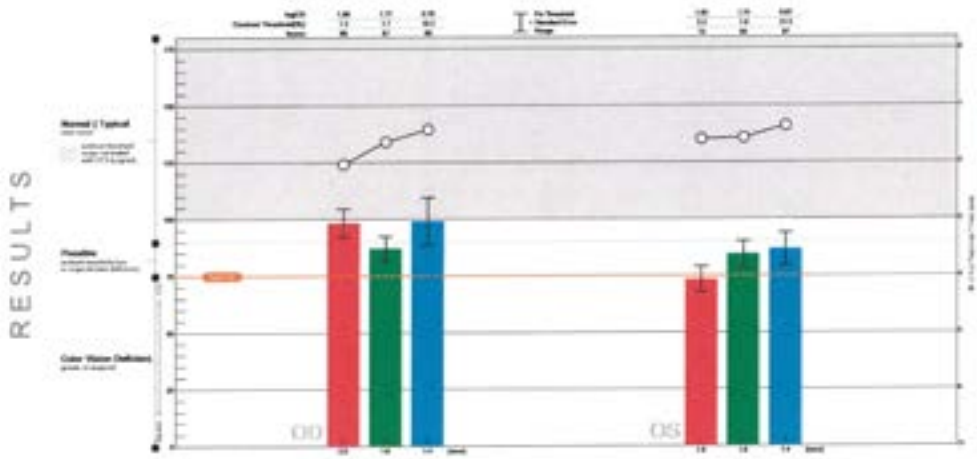

Figure 3. *ColorDx obtained at Visit 7*

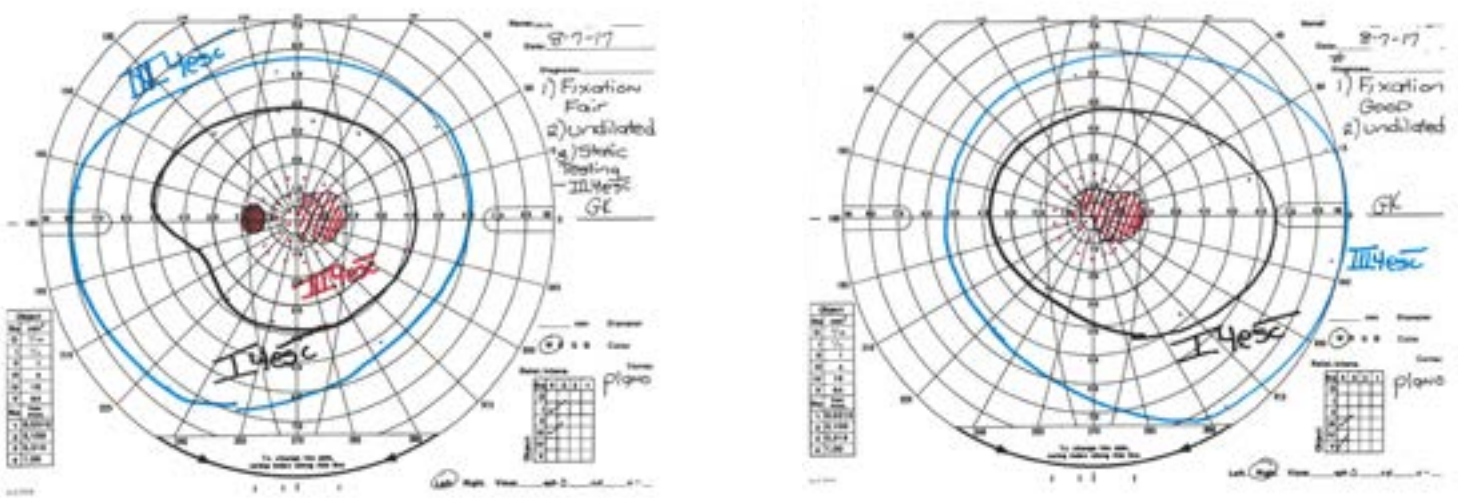

Figure 4. *Goldmann visual field obtained at Visit 2*

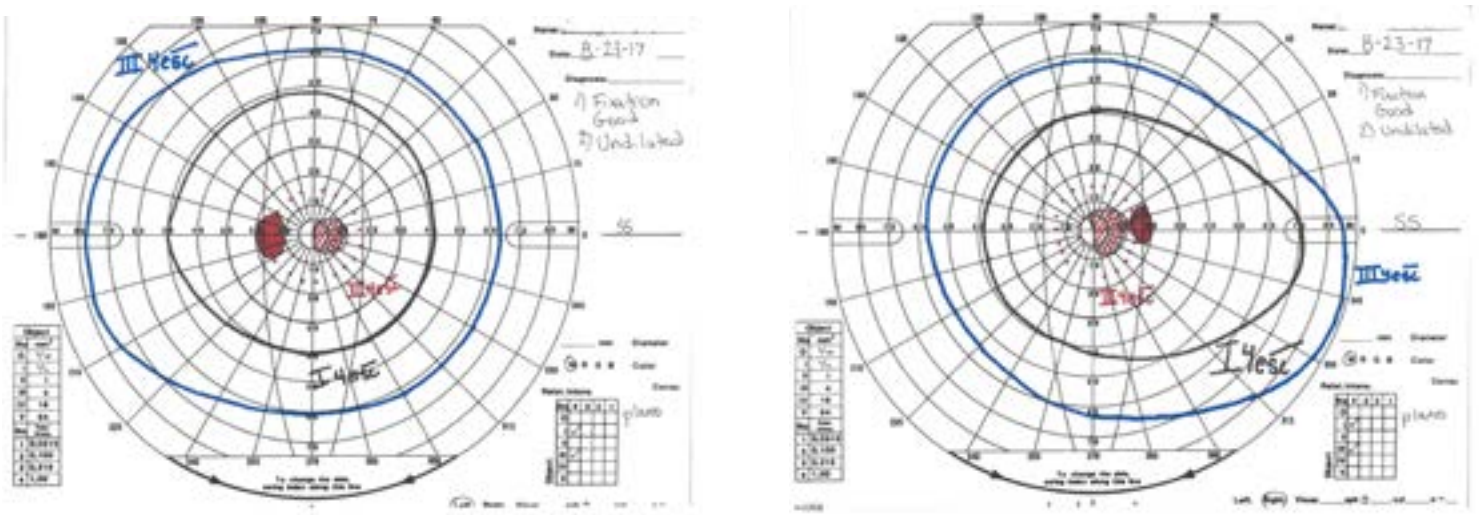

Figure 5. *Goldmann visual field obtained at Visit 3*

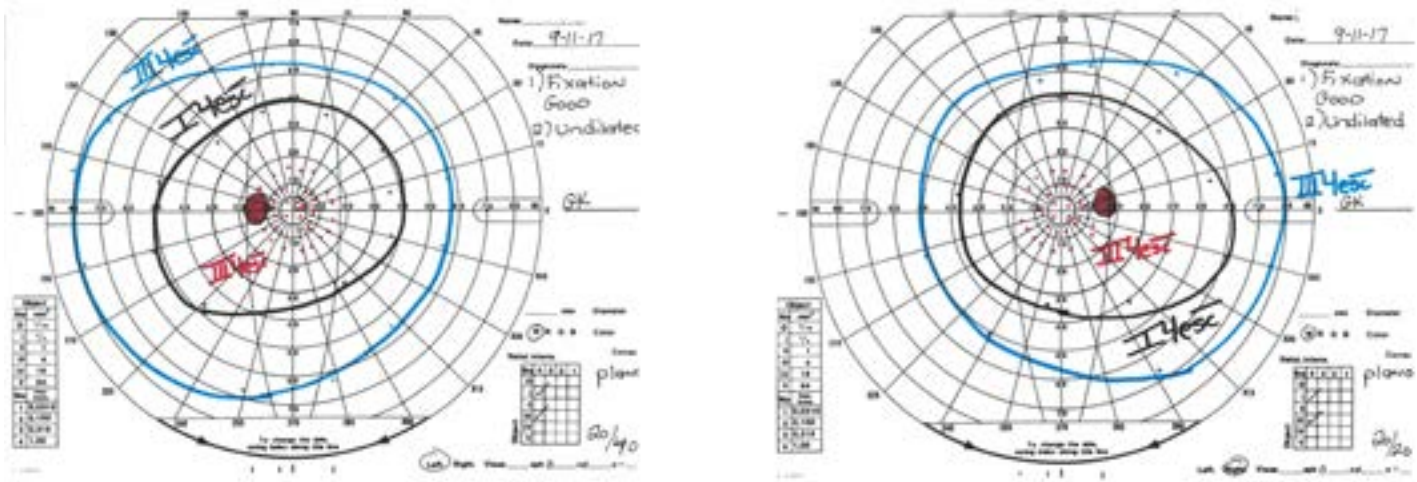

Figure 6. *Goldmann visual field obtained at Visit 4*

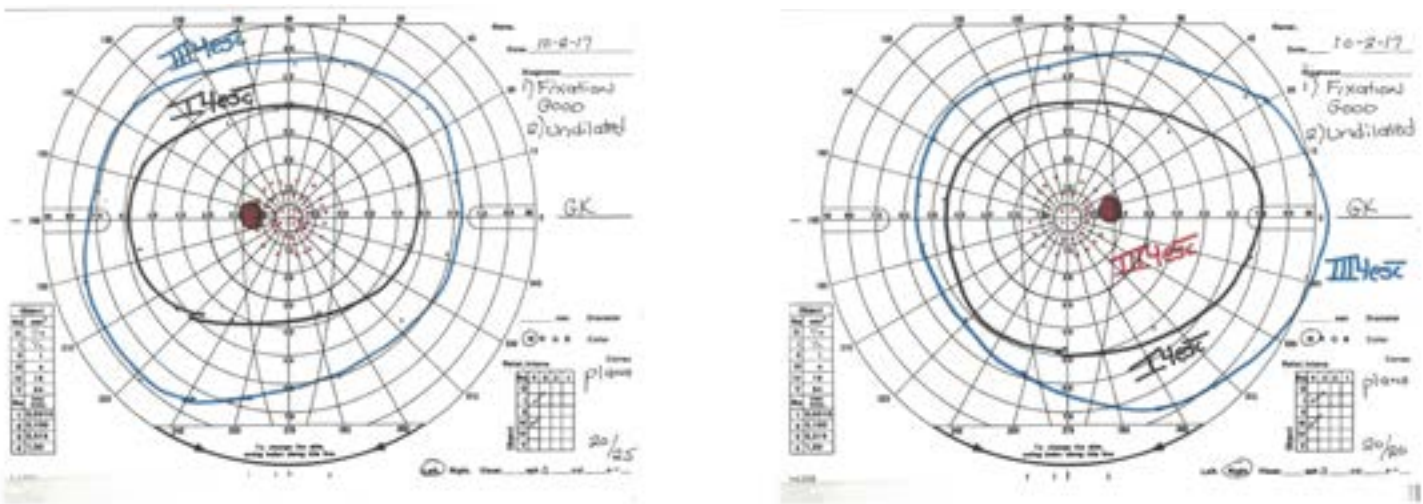

Figure 7. *Goldmann visual field obtained at Visit 5*

the entrance test findings from the patient's seven visits is found in Table 1.

After an abnormal but inconclusive result on color vision testing using the Hardy-Rand-Rittler (HRR) test, a computerized color vision test was administered. The ColorDx uses the Landolt C to isolate each type of cone (L-cone, M-cone, S-cone) photoreceptor. This test was established to look specifically at acquired color vision loss.[6] Due to the high prevalence of decreased S-cone sensitivity often found with acquired optic nerve head disease, the test is superior to tests like the HRR test or the Ishihara color test.[2] When this test was administered at the patient's fifth visit, there was a marked decrease in each eye individually in M- and S-cones. L-cones were found to be deficient in the left eye and possibly deficient in the right eye. This test was repeated at her last visit. At this visit, the results improved significantly for all cone photoreceptors in both eyes individually (Figures 2 and 3).

After unreliable, abnormal confrontation field and facial Amsler test findings, a Goldmann visual field was conducted and repeated at Visits 2-5. This test was repeated until findings were stable in order to have a functional representation of structural damage and ultimately to aid the patient in using her functional vision.

At the second visit, there was no noted peripheral constriction in either eye, but there was an incongruous right homonymous hemi-macular hemianopia, as indicated by the red hash marks (Figure 4). At her third visit, there was no noted constriction in either eye, but there was still an incongruous right homonymous hemi-macular hemianopia. At this point, the size of the scotomas had already decreased by about 50% in each eye. In the patient's right eye, there was now a distinct differentiation between the physiologic blind spot and the acquired scotoma (Figure 5). At her fourth visit, which was less than eight weeks after she initially presented to the hospital, the scotoma in the patient's right eye had completely resolved and in her left eye was significantly reduced, by about 85% from the initial presentation (Figure 6). At her fifth visit, the test was repeated for a final time. There was functional stability in the patient's right eye, demonstrating normal

findings in both the peripheral and central vision. In the patient's left eye, the scotoma had completely resolved, with no noted peripheral constriction. At this point, the patient reported that the parts of her vision that were once missing were no longer absent (Figure 7). This visual representation provides useful information on how to help the patient best utilize her vision.

OCT imaging was obtained at visits 3, 4, and 6. The test was repeated several times in order to correlate any changes in structure with noteworthy changes in visual function. This imaging allows for a structural representation of functional damage, and it is non-invasive and objective, requires little cooperation from the patient, and is lower in cost compared to an MRI.[7] Additionally, an OCT is an appropriate test to monitor lesions anterior to the lateral geniculate nucleus (LGN). Fairly symmetric paramacular thinning was found at all three visits. There was no significant difference in findings at the 3rd visit compared to the 4th visit, demonstrating a period of structural stability. There was further paramacular thinning noted at visit 6, more in the left eye compared to the right (Figure 8).

A retinal nerve fiber layer (RNFL) OCT was obtained in order to understand better the permanent damage to the patient's optic nerve. There was a reduction in thickness of RNFL found temporally, worse in the left eye compared to the right eye, that decreased over time. This slightly asymmetric thinning in RNFL thickness may indicate an asymmetric retrograde degeneration (Figure 9).[8]

A VEP using the Diopsys NOVA system was used to measure the function of the entire visual system without requiring a response from the patient. The test was conducted at five different spatial frequencies (8x8 check size: 0.6 cycles/degree, 16x16 check size: 1.2 c/deg, 32x32 check size: 2.4 c/deg, 64x64 check size: 4.8 c/deg, 128x128 check size: 9.6 c/deg) while the patient wore three electrodes and watched the checkerboard alternate checks from white to black to white for twenty seconds. Each eye was tested individually, and both eyes were tested together.

The patient's relative amplitudes within each testing session and over the five visits were variable. At the first few visits, the patient's amplitudes were questionably abnormal. Over time, the relative amplitudes became somewhat normal in each eye individually but remained abnormal with both eyes together due to lack of binocular summation. Binocular summation is achieved when binocular amplitudes are at least 10% greater than the eye with the highest amplitude.[9] This case demonstrates a period of destructive interference in the primary visual cortex, where the brain may now decide to find a way to "shut down" an eye because each eye is no longer constructively assisting the other.

Latency is an absolute measure that represents the time it takes for a signal to travel from the retina to the primary visual cortex. In a normal subject, the measure is typically 100msec.[10] In this patient's case, latency was found to increase over time, meaning visual information was taking longer to get to the primary visual cortex, under all conditions. These results show that over time, there was an improvement in relative amplitude with no binocular summation and an increase in absolute latency at all five spatial frequencies in the right eye, left eye, and with both eyes together. The results from this test, over five visits at five different spatial frequencies, can be found in Table 2.

Lastly, a thorough ocular health assessment was conducted at the patient's hospital visit. There were no noted abnormalities in either the anterior or posterior segment of either eye. Due to poor fixation, tactile pressures were obtained and found to be soft and equal in both eyes. The patient was dilated with one drop of 1% tropicamide in both eyes. The optic nerve heads were pink, distinct, flat, and had a normal cup-to-disc ratio, with no evidence of nerve pallor or edema in either eye. An undilated assessment was conducted at all follow-up visits. Findings were noted to be stable and unremarkable at each visit. Posterior pole findings can be seen in Figure 10.

The differential diagnoses considered in this case included:

- Optic neuritis secondary to MS
  This condition typically occurs in patients 18-45 years old secondary to demyelination of the optic nerve. Although symptoms such as loss of vision, dyschromatopsia, variable visual field defects, and optic disc edema may be present in both optic neuritis secondary to MS and NMO, the conditions are easily differentiated with an MRI due to specific white matter lesions presenting in optic neuritis secondary to MS.[2,4,11]
- Ischemic optic neuropathy (arteritic)
  Ischemic optic neuropathy is an ophthalmic condition that typically presents secondary to giant cell arteritis (GCA) in patients older than 55. Associated signs of the condition, such as jaw claudication, scalp tenderness, fever, and weight loss, help to differentiate the condition from NMO. Due to the elevated sedimentation rate

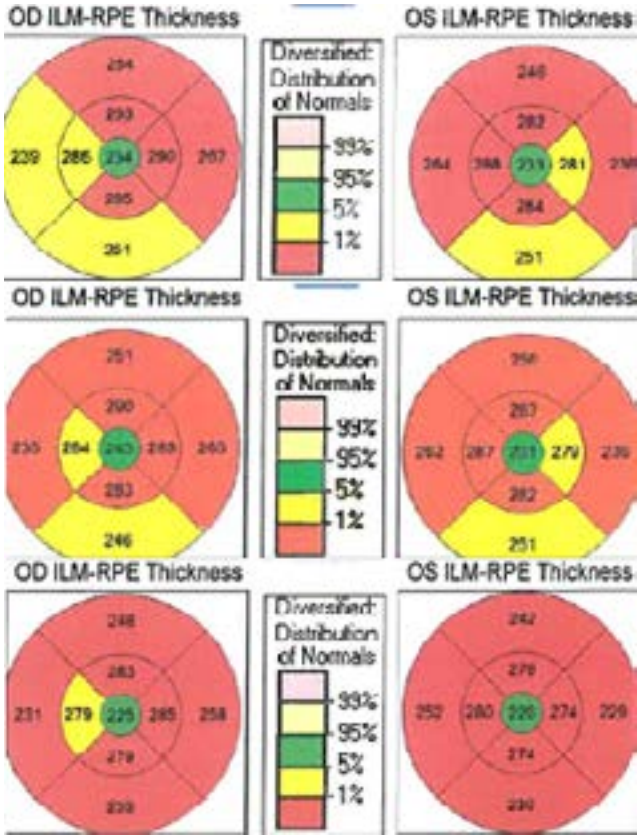

Figure 8. *Macular OCTs obtained at Visit 3, Visit 4, and Visit 6*

(ESR), C-reactive protein, and platelet count, the conditions can also be differentiated by obtaining specific blood work.[2,4]

- Acute papilledema

  Optic disc swelling secondary to increased intracranial pressure can be differentiated from NMO due to the signs and symptoms of each condition or via lumbar puncture. In acute papilledema, there tends to be a minimal decrease in visual acuity and a visual field defect corresponding to an enlarged physiologic blind spot.[2] If a lumbar puncture is conducted, elevated opening pressure would be indicative of papilledema.[4]

- Leber hereditary optic neuropathy (LHON)

  This condition is caused by a gene mutation of mitochondrial DNA; therefore, the patient may have a family history of the condition. LHON also typically occurs in males between 15 and 30 years of age.[2,4]

- Toxic or metabolic optic neuropathy

  Toxic or metabolic optic neuropathy can be differentiated from NMO via a thorough case history. The etiology of the condition includes substance abuse, such as alcohol or tobacco; severe malnutrition; severe malabsorption, often seen in conditions such as pernicious anemia; or a toxic medication.[2,4] Due to this patient having no prior significant medical history and adamantly denying any alcohol or tobacco use, this condition was easily ruled out.

- Neuromyelitis optica

Treatment and Management

When admitted to the hospital, this patient was treated with intravenous steroids to help decrease inflammation of the spinal cord and optic nerve.[12] Once released from the hospital, she was put on an oral taper and was referred to a neurologist and an optometrist. Her neurologist monitored the condition closely without medication due to the patient becoming pregnant in the early stages of disease onset. Medications to consider for decreasing the relapse rate of NMO include azathioprine, rituximab, mycophenolate, mycophenolate mofetil, mitoxantrone, and plasma exchange.[1] A curative treatment for NMO does not exist, but treatment goals include long-term stabilization and symptomatic therapy for residual findings. As an eye care professional, the treatment goals for this case are structured around helping the patient manage their visual symptoms.

Visit 2: Multiple hand-held and stand magnifiers were trialed secondary to the patient's incongruous right homonymous hemi-macular hemianopia. All devices left the patient feeling limited and outdated. As a nursing student, her near demand far exceeded

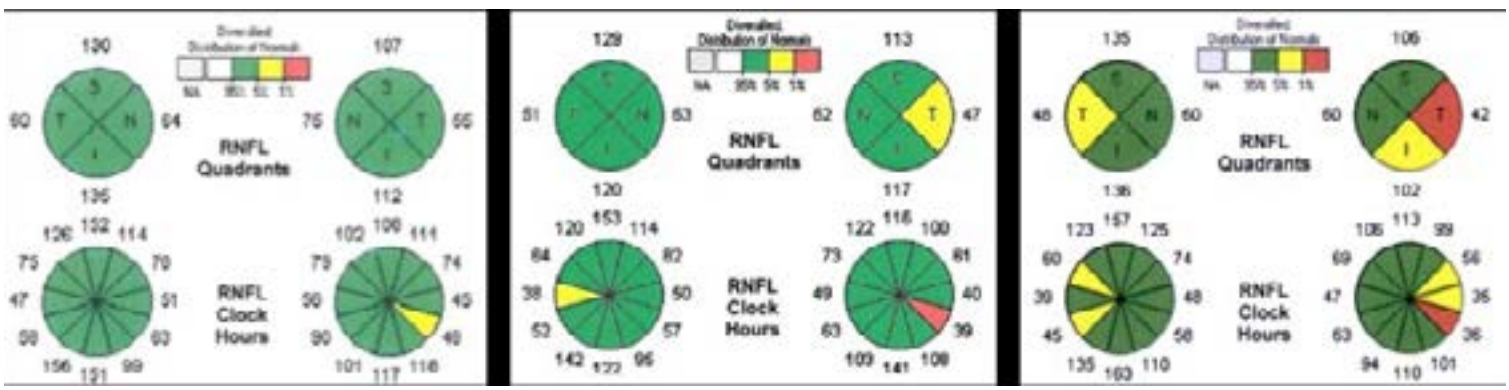

Figure 8. *RNFL OCTs obtained at Visit 3, Visit 4, and Visit 6*

Table 2. VEP Results over Five Visits at Five Different Spatial Frequencies
(8x8 check size= 0.6 cycles/degree, 16x16= 1.2 c/deg, 32x32= 2.4 c/deg, 64x64= 4.8 c/deg, 128x128= 9.6 c/deg)

		Visit 2		Visit 3		Visit 4		Visit 6		Visit 7	
Check Size	Eye	Amplitude (μV)	Latency (msec)	Amplitude (μV)	Latency (msec)	Amplitude (μV)	Latency (msec)	Amplitude (μV)	Latency (msec)	Amplitude (μV)	Latency (msec)
8x8	OD	5.0	100.6	11.2	124.0	10.4	135.7	21.3	133.8	11.0	134.6
8x8	OS	18.5	124.0	12.9	158.2	21.0	144.5	7.3	67.4	9.4	132.1
8x8	OU	9.4	133.8	11.5	165.0	14.6	88.9	11.2	101.6	9.8	128.9
16x16	OD	5.4	115.2	13.6	126.0	12.2	132.8	23.6	131.8	21.0	128.4
16x16	OS	13.2	123.0	7.6	104.5	12.9	126.9	9.7	66.4	8.4	132.3
16x16	OU	7.5	136.7	6.5	134.8	10.1	88.9	5.0	101.6	6.0	124.7
32x32	OD	8.3	113.3	14.2	115.2	10.1	141.6	17.7	140.6	7.4	118.0
32x32	OS	11.1	96.7	11.9	147.5	13.3	134.8	19.6	138.7	9.7	117.2
32x32	OU	5.4	124.0	13.3	118.2	11.8	149.4	10.0	79.1	8.6	121.1
64x64	OD	4.2	96.7	9.9	121.1	10.6	144.5	19.0	137.7	12.0	121.9
64x64	OS	7.9	82.0	15.2	90.8	11.7	154.3	18.5	149.4	11.7	126.5
64x64	OU	5.3	101.6	8.8	123.0	9.5	103.5	25.8	130.9	12.8	120.1
128x128	OD	12.1	104.5	4.6	119.1	17.3	147.5	17.8	158.2	20.1	142.3
128x128	OS	13.6	158.2	4.9	178.7	13.1	143.5	26.0	162.1	12.2	140.4

her need for clear vision at distance. An Eschenbach Smartlux Digital Portable Video was then trialed with success. This device, which allows users to enlarge and enhance small text while remaining binocular, was dispensed to assist with school work. Additionally, the device can be put on a video or camera setting to refer back to an image at a later time.

Visit 3: After two weeks of successfully using the Eschenbach Smartlux, the patient felt that she was able to keep up with her homework demand, but she was struggling to see the board in class. Several low vision distance devices were trialed. At first, multiple monocular telescopes were attempted, but all left the patient feeling as though her peripheral vision was limited. Since the patient's peripheral vision was intact, the next device trialed was a Max TV. This device met the patient's needs secondary to her rapidly changing distance acuity and desire to remain binocular. This device can be manually adjusted with the knobs on the side of the lenses, allowing the patient to make changes based on her current visual status.

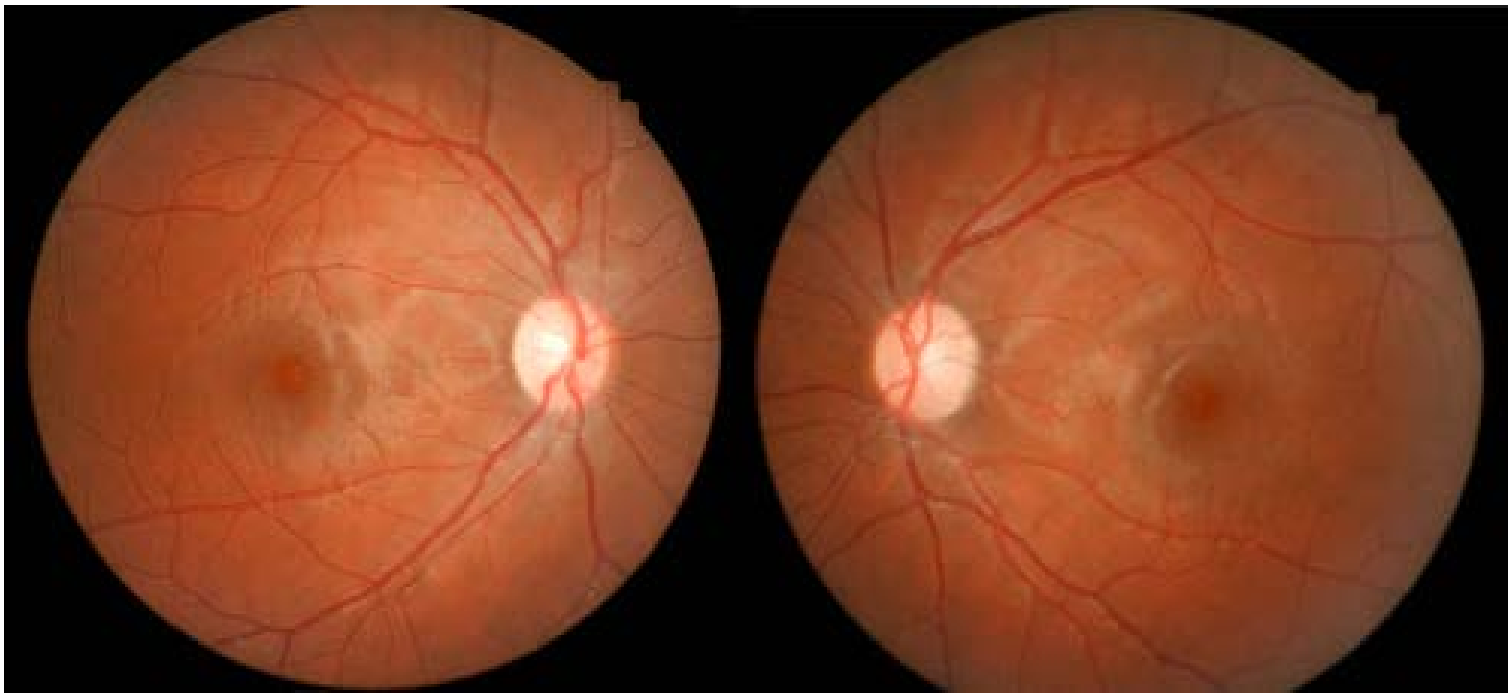

Figure 10. *Retinal photos obtained at Visit 5*

Visit 4: Devices were reassessed, and no changes were made to the patient's visual aids.

Visit 5: Several tinted sunglasses were trialed after the patient reported difficulty transitioning from dark to light environments. After subjectively rejecting various shades of brown, grey, and green tints, the patient had a positive response to a yellow filter. This tinted lens allowed for faster recovery of vision when changing environments and a decrease in symptoms. Yellow tinted glasses, Night Drivers ML 3H2W606RSND, were ordered, and the patient was instructed to continue using the Max TV and Eschenbach Smartlux as needed.

Visit 6: The yellow-tinted glasses were dispensed, and a reassessment of the patient's current devices was conducted. Continued success was noted with the Eschenbach Smartlux, but due to improvement in distance acuity, the patient no longer felt a need for the Max TV.

Visit 7: Devices were reassessed, and no changes were made to the patient's visual aids.

The optometric management of patients with NMO will vary based on the patient's symptoms and findings. In this case, the patient was monitored every three weeks until her findings stabilized, and then every few months thereafter. Frequent follow-up exams are recommended until the patient's findings have stopped changing. A visual field can be obtained in order to document remarkable changes in the patient's functional vision. This test helps eye care providers better explain a patient's visual deficit and assist the patient in utilizing his or her remaining field. A Goldmann visual field was administered, rather than a Humphrey visual field, due to concerns with slower response secondary to intermittent joint pain. A macular and RNFL OCT can be used to document any noteworthy changes in structure within the retina and optic nerve head. This objective measure can allow for close monitoring of the condition. Lastly, a VEP was obtained at five of the patient's visits due to a rapid change in findings. This test can help eye care providers better understand the function of the visual system beyond the optic nerve head.[13] These tests allow for a comprehensive assessment and understanding of the patient's visual system.

Discussion

NMO, also referred to as Devic's disease, is a devastating condition affecting both vision and mobility.[5] The condition should be assessed by a neurologist and co-managed with an eye care provider. Although the condition's treatment and management may vary based on the severity and presentation of the case, all cases should be monitored closely for changes in signs and symptoms.[1] Due to technological advances in optometric equipment, the role played by eye care providers has changed over time. As demonstrated in this case, non-invasive optometric testing can be used to monitor and to manage this condition.

There was a remarkable recovery in visual acuity, visual field, and color vision in this case. Although visual acuity tends to improve over months in patients with NMO, the final acuity depends on the severity of vision loss and the number of attacks.[4] Typically, when the condition is monophasic, the initial vision loss is devastating, but recovery can be dramatic. This allows patients to maintain some degree of independence. The relapsing form of the condition tends to present with less initial vision loss and with marked recovery, but recurrent episodes diminish recovery gains.[5] At this point, it is still unknown whether the patient will relapse or remain in the monophasic form of the condition.

Although contrast sensitivity is not an important diagnostic indicator of this condition, the information from this test can help track a patient's recovery. In this case, the patient had reduced contrast sensitivity, worse in the left eye compared to the right eye, that slowly improved over time. Even though an improvement was noted in these findings, the results still remain abnormal.

Visual field patterns in NMO are variable and include non-central scotomas and altitudinal hemianopsias; these can be visually devastating compared to field defects seen in optic neuritis secondary to MS.[16,17] Although there are various instruments to measure a patient's visual field, in this case, a Goldmann visual field is far superior to other instruments. As mentioned earlier, it is a manual test that can be conducted at a slower speed to accommodate the patient's response time. In addition, the test can easily be paused to accommodate a break should the patient need. Ultimately, visual field testing helps display a functional representation of structural damage found on OCT imaging and VEP testing.

Macular and RNFL OCTs provide an objective measure of the patient's retinal structures. Chronic autoimmune inflammation seen in NMO seems to target Müller cells and/or retinal astrocytes, which are enriched at the fovea.[15] This pathophysiology likely causes microstructural changes, leading to foveal

thinning. It is likely that NMO is much more visually devastating than optic neuritis secondary to MS due to this chronic autoimmune inflammation.[7] Further studies are needed to better understand how the autoimmune inflammatory process targets Müller cells and/or retinal astrocytes ultimately leading to a devastating visual outcome.[15] There is a noteworthy correlation between RNFL average thickness and visual field function. In patients with NMO, there is a significant reduction in thickness of retinal peripapillary nerve fibers found temporally, where the papillomacular fibers are located.[8] In this case, there was noted temporal RNFL thinning, worse in the left eye compared to the right. This was reflected in the patient's incongruous right homonymous hemi-macular hemianopia.

There are several advantages to running an OCT in cases such as NMO. Similar to an MRI, the instrument provides an objective measure. In contrast to an MRI, an OCT is easily repeated at a low cost, is non-invasive, and requires little cooperation from the patient.[11] OCT imaging also helps relate structural damage to functional changes. It is possible that the diagnostic criteria for NMO may change once more OCT data is collected and analyzed in patients with this condition.

Although research on the role of VEP testing for patients with NMO is currently being conducted and analyzed, it is evident from this case that findings rapidly change. When this test was performed close in time to disease onset, the results indicated variable amplitude findings in each eye individually, with normal latency findings. As the test was rerun at the follow-up visits, the amplitudes became less variable in each eye individually but not with both eyes together. As discussed earlier, this indicated that binocular summation was not present.[9] Although normal latency has been noted to be at 100msec, characterizing normal vs. abnormal latency is based on age, gender, visual acuity, and visual field. With time in this case, there was an increase in latency, likely indicating that further disruption in myelination may have continued to occur months after the attack.[14] This may be an indication that more frequent follow-up testing should be conducted in order to better understand what is happening during each stage of the disease process.

VEP patterns may be used to help differentiate NMO from other conditions, such as optic neuritis secondary to MS.[10] In NMO, research indicates that there is an abnormal amplitude with a majority of patients having a normal latency.[13] This is exactly what was found when the test was first conducted in this case. Over time, there is a change in findings, and therefore, it is evident that future studies should conduct more frequent follow-ups of the test. In optic neuritis secondary to MS, evidence indicates variable amplitude findings with latencies getting longer and longer over time.[14] More research and data is needed to better understand how VEP testing can assist eye care providers in diagnosing and co-managing NMO. Future diagnostic criteria for the conditions may involve using a VEP, in addition to an MRI, to help differentiate various conditions.

After a few months of closely following this case, the patient reported that she was pregnant. The ramifications of pregnancy in conjunction with NMO have recently been researched. The risk of relapse is measured and recorded as the annualized relapse rate (ARR). Studies indicate that the ARR increases during pregnancy and the postpartum period, with the highest ARR occurring three months postpartum.[18] Why this occurs has not yet been researched, but it is hypothesized that the change in hormone levels may be a contributing factor. For this reason, the patient was monitored monthly until she was six months postpartum, and every three to six months thereafter.

An eye doctor's role encompasses recognizing the psychological effects that vision loss has on their patients. Patients experiencing an initial attack often have difficulty expressing their visual symptoms. Providing literature for patients to find solidarity will minimize the negative psychological effects. *Patient H69: The Story of my Second Sight* is a book written by Vanessa Potter. Potter is an award-winning broadcast producer working within the London advertising industry who was diagnosed with NMO in 2012. She wrote a memoir narrating her first-hand experience of waking up to find herself blind and paralyzed. Potter states, "It was the experience of dramatically losing, then regaining my sight that led me to adopt the pseudonym of Patient H69 in order to tell my story via immerse art and storytelling."[19] This resource helped this patient cope and better express her symptoms.

Conclusion

NMO can be co-managed by an eye care professional who takes a detailed patient history, conducts a thorough clinical evaluation, and looks for characteristic physical findings by investigating with a variety of specialized tests, such as VEP, OCT, and Goldmann visual field. Although this case report demonstrates the corresponding and confirmatory roles of specialty optometric testing, more studies

are needed to better understand how these tests can help identify future research and treatment options. A curative treatment does not exist, but current treatment options aim to reduce the ARR. Once the autoimmune inflammation targeting Müller cells and retinal astrocytes is better understood, other treatment options should be explored.

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