

Article ▶ The Complexity of Co-existing Functional Vision Loss and Organic Diagnoses

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ABSTRACT

Background: Parkinson's patients commonly experience a visual midline shift that causes postural and gait changes, increasing the risk of falls. If the optometrist can improve the visual midline shift with the use of yoked prism prescriptions, the risk of falls will decline and ultimately decrease the overall potential healthcare cost.

Case Report: A 77-year-old white male diagnosed with Parkinson's presented for an evaluation. The patient complained of double vision and difficulty judging space perception that was affecting his balance when walking. He was treated with a lens prescription that included a combination of yoked and base-in prisms.

Conclusion: After 1 month of wearing the new prism prescription, he no longer experienced double vision and showed improved balance when walking. This case emphasizes the importance of evaluating visual midline shifts and prescribing yoked prisms in this patient population to improve overall balance and to lower the risk of falls.

Keywords: balance, Parkinson's disease, visual midline shift, yoked prisms

Introduction

Non-organic vision loss (NOVL), also known as functional vision loss, is a poorly understood condition that occurs in the absence of identifiable organic pathology. It presents in the form of a visual disturbance, either as a loss of visual acuity and/or a loss of visual field. Although descriptions and categories may vary depending on the literature source, NOVL can be divided into 3 groups: psychogenic, malingering, and factitious.¹ Psychogenic NOVL occurs in a patient who experiences vision loss but is unable to control their symptoms.¹ NOVL from malingering refers to patients feigning symptoms for secondary gain.¹ In factitious

NOVL, the patient intentionally produces symptoms in order to assume the sick role.¹ Diagnosis of functional vision loss has several different characteristics, with the common theme being the absence of organic disease on clinical examination and the presence of inconsistent exam findings. To further complicate the diagnosis of NOVL, organic vision loss can be present concurrently. This is known as functional overlay. The occurrence of functional overlay ranges from 6% to 53%, depending on the source.¹⁻³ In addition to organic disease, NOVL has been found to be associated with psychiatric disorders or psychosocial events in approximately 30% and 36% of cases,

respectively.⁴ Again, these percentages vary significantly across the literature.

Diagnosis bias towards a pre-existing condition may mask the detection of organic disease overlay in NOVL. It is easy to fall back on medical parsimony and to assign new or worsening symptoms to an already established diagnosis. Furthermore, inheriting new patients with an incoming NOVL diagnosis can prove even more challenging to manage when prior history and exam records are not accessible. This case study uncovers organic macular disease at the initial exam in a patient with a previously documented diagnosis of NOVL. Without consistent eye exams and ancillary testing throughout the years, it is difficult to determine the onset of this pathology. With the detection of new organic pathology, questions were raised concerning the validity of the prior NOVL diagnosis. This triggered a meticulous review of past records and set off a flurry of diagnostic testing to aid with differentials. A diagnosis was required to initiate further treatment and counseling, if warranted.

This case had three distinct but interrelated elements. The first was the early diagnosis of functional vision loss and how it was first established. The second was the new diagnosis of organic pathology nearly 25 years after the patient's initial loss of vision and the difficulties inherent in identifying obscure pathology. The final aspect was the important message to remain vigilant in cases when a longstanding diagnosis is present.

Case Report

A 51-year-old African American male, MJ, presented on 06/21/2016 to the West Haven Veteran Affairs (VA) Optometry Eye Clinic for a comprehensive eye exam. He reported longstanding bilateral vision loss that had been present for over 25 years, but he felt that it had progressed within the past few years.

MJ's medical history was remarkable for obstructive sleep apnea, post-traumatic stress

disorder, depression, tinnitus, and plantar fasciitis. His medications at the time of the exam were naproxen 375 milligrams (mg) twice a day, sertraline 100mg once a day, and tamsulosin 0.40mg once a day.

MJ reported that while deployed during Operation Desert Storm in Iraq, he fell off a vehicle and experienced a head trauma. He reported rapid vision loss in the 1990s following his return stateside. After returning, he was seen by a multitude of eye specialists, including retinal specialists and neuro-ophthalmologists. He did not recall ever being given a diagnosis or specific cause for the loss of vision. The most recent eye records from 2014 were from an outside provider, who indicated macular degeneration as the source of the vision loss.

History

A chart review revealed that MJ had best-corrected vision of 20/20 OD and OS from 1984 through 1987 during army physicals. Significant vision loss was first reported in 1991, with distance best-corrected visual acuity (BCVA) measured at 20/200 OD and OS. The refraction at that time was -1.00-1.00x175 OD and -1.00-0.75x150 OS. With repeated coaxing and encouragement, the patient was able to read the 20/50 line at distance with the same prescription through the phoropter OD and OS. Near vision was measured to be 20/40 OD and OS. All other entrance testing was normal OU. The anterior segment was unremarkable OU. Cup-to-disc measurements were 0.50 round, with full and healthy rim tissue OU. Stereoacuity was measured to be 8/9 OU. Color vision testing with the Farnsworth D-15 was normal OU. Amsler grid testing was normal at that time, without scotomas or metamorphopsia OU. A 30-2 threshold visual field (VF) exam revealed small, bilateral central scotomas. A 10-2 VF was repeated twice and revealed variable, inconsistent, and non-repeatable central defects. The patient was referred to a retina specialist and neuro-ophthalmologist

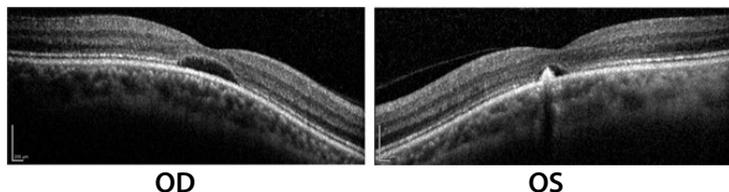


Figure 1. Macular OCT of the right eye (OD) and left eye (OS). A neurosensory detachment can be seen centrally OD with the RPE intact. A neurosensory detachment can also be seen centrally OS with associated subretinal fibrotic tissue within the cavity created.

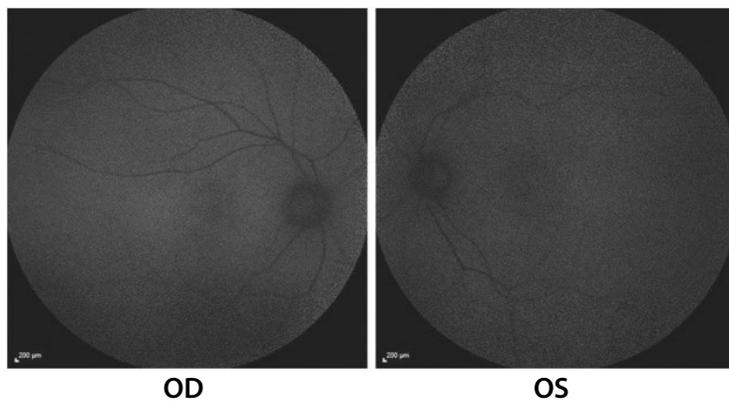


Figure 2. Fundus autofluorescence of the right eye (OD) and left eye (OS). Imaging was of poor quality but was unremarkable for hyper- or hypofluorescence OU.

for further evaluation. During subsequent visits with specialists, a fluorescein angiogram (FA), an electroretinogram (ERG), and a visually evoked potential (VEP) were all conducted and were found to be normal. Computed tomography (CT) and magnetic resonance imaging (MRI) of the brain were also completed and were found to be normal.

There were no available records between 1991 and 2013. In addition, MJ was unsure of whether he was seen by an eyecare provider during that time. Starting in 2014, he re-initiated eye care with a few different private providers who had varied diagnoses. The last diagnosis made prior to transferring care to the West Haven VA Medical Center was macular degeneration OU.

Exam Findings

The patient received a comprehensive eye examination upon entering the West Haven VA Medical Center. MJ's BCVA was measured with a Feinbloom chart to be 10/80- with an eccentric view at 6 o'clock OD and 10/60- with an eccentric view at 11 o'clock OS. The refractive

error was compound myopic astigmatism: OD: -4.50-1.00x180 and OS: -3.00-2.25x155. Primary gaze acuity was recorded as 10/140 OD and OS.

Both entrance testing and anterior segment findings were found to be unremarkable OU. Fundus evaluation revealed mild RPE mottling of the maculae OU. The periphery was unremarkable OU. An optical coherence tomography (OCT) was deemed necessary as the patient's symptom of progressive vision loss OU did not equate with the clinical findings of mild macular retinal pigment epithelium (RPE) mottling OU. Surprisingly, the macular OCT showed a sub-retinal cystic space OD and a hyper-reflective lesion OS, with surrounding subretinal fluid (SRF; Figure 1). After an abnormal OCT finding, the patient was referred for further evaluation with the retinal specialist.

Upon further study of the posterior pole, the retinal specialist discounted the idea of macular degeneration based on demographics and clinical appearance. Fundus autofluorescence (FAF) imaging showed no abnormal hypo- or hyper-fluorescent lesions (Figure 2). Fluorescein angiography performed at this visit revealed no active leakage or ischemia OU; however, a mild RPE defect seen as subtle hyper-fluorescence was visible OS (Figures 3b and 4b). The lesions were measured using the macular OCT and were roughly 1300 um laterally/1000 um vertically OD and 700 um laterally/500 um vertically OS. Based on these clinical findings, the retinal specialist diagnosed the patient with presumed old central serous chorioretinopathy (CSCR) OU.

A Humphrey VF was completed (Figure 5) and revealed a central defect with a few supero-nasal defects OD. A central defect with superior and infero-nasal defects was shown OS. Temporal defects seen were likely associated with the optic nerve blind spot.

Although a tentative diagnosis of CSCR was made, MJ was referred to a neuro-ophthalmologist both because clinical findings were inconsistent with classic CSCR and because the BCVA did not match the size of the

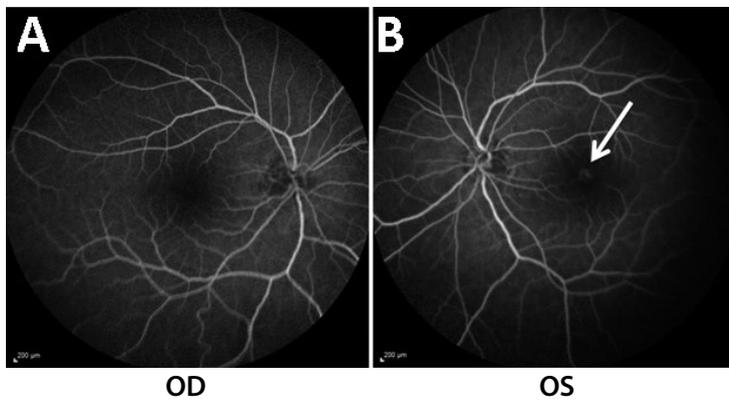


Figure 3. Fluorescein Angiography: Mid-phase-Fluorescein angiogram of the right eye taken at 2:40 min following injection (OD) and left eye taken at 3:13 min (OS). FA OD and OS showed normal choroidal, arterial, and venous filling. Good perfusion was noted OU. OD was unremarkable for hyper- or hypofluorescence. OS had faint central hyperfluorescence at the macula.

lesion. A thorough evaluation was conducted by neuro-ophthalmology. Of note, cranial nerve testing revealed no abnormalities, a retinal nerve fiber layer (RNFL) OCT OU was normal, and Ishihara color vision was reduced although asymmetric: 3/11 OD and 1/11 OS. The neuro-ophthalmologist concluded that there was no optic neuropathy and that in all likelihood, the reduced vision was due to the macular lesions OU. Because of the history of inconsistent findings and the fact that the clinical signs did not agree with the degree of vision loss, he also diagnosed MJ with functional vision loss.

Discussion

As previously mentioned, functional vision loss, or NOVL, is a visual disturbance manifesting as decreased acuity and/or visual field loss with no apparent organic disease in the structures of the visual system spanning from the cornea to the occipital cortex.⁵ It is divided into 3 subgroups: psychogenic, malingering, and factitious disorders. From an examination standpoint, the delineation is not crucial. Regardless of etiology, a complaint of vision loss should always elicit a complete ocular evaluation. The first step is to rule out refractive error and anterior or posterior segment abnormalities. Next, ancillary testing such as OCT, ERG, VEP, FA, and/or FAF should be ordered to aid in diagnosis. Only then should a functional vision loss diagnosis be considered.

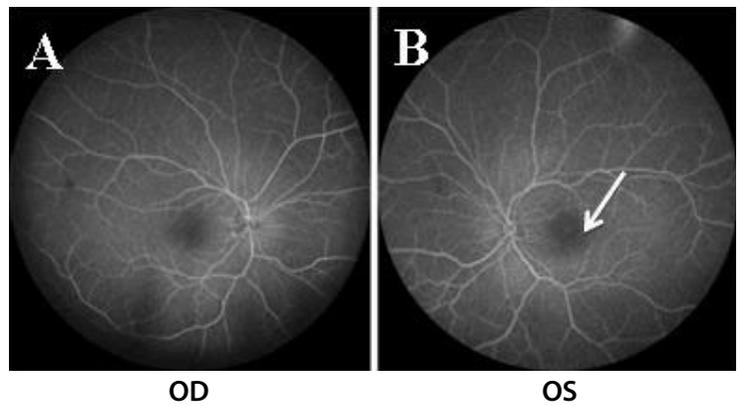


Figure 4. Fluorescein Angiography: Late-phase-Fluorescein angiogram of the right eye taken at 5:57 min following injection (OD) and left eye taken at 6:25 min (OS). FA OD and OS showed normal choroidal, arterial, and venous filling. Good perfusion was noted OU. OD remained unremarkable for hyper- or hypofluorescence. OS had a trace central hyperfluorescence at the macula, further muted when compared to the mid-phase images (arrows).

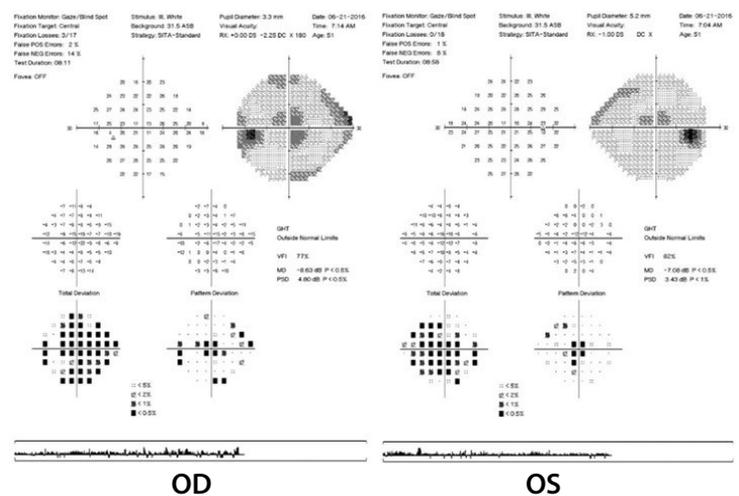


Figure 5. Humphrey Visual Field 24-2 SS of the right eye (OD – image to the right) and left eye (OS – image to the left). Reliability indices within normal limits, gaze tracker adequate OU. OD shows a central defect with a few sup/nasal defects. OS shows a central defect with sup and inf nasal defects. Temporal defects seen are likely associated with optic nerve blind spot.

Neuro-imaging is recommended in particular cases where a reproducible visual field defect is discovered and/or other neurological deficits are present. In addition to ruling out abnormalities, a common aspect is an inconsistency in exam findings, especially if the vision loss is worse than exam findings would predict. Management of NOVL is typically just reassurance, with some sources suggesting placebo visual exercises.⁶ A psychiatric referral may be appropriate if the patient is not already being followed and especially when other impairments of psychological behavior are present.⁶

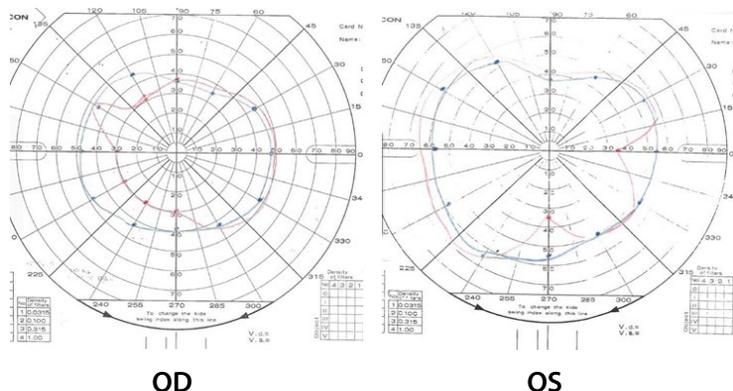


Figure 6. Tangent screen of the right eye (OD) and left eye (OS) measured in 1991. The blue lines represent measures taken using a 3mm target at 1 meter. The red lines represent measures taken using a 6mm target at 2 meters. Both eyes display a tubular visual field.

MJ was first seen by an eye care provider in August of 1991. Myriad testing was done at that initial exam to address the unexplained vision loss. After an extensive work-up, the provider had functional vision loss high on the differential. There were several critical exam findings that made that diagnosis more likely. The first finding was the near-normal stereopsis result, where MJ scored 8/9 on the Titmus circles stereotest. That score corresponds to 50 secs of arc. According to the study conducted by Levy, to achieve a stereoacuity of 50 secs of arc, a visual acuity of about 20/30 is required,⁷ better than MJ's BCVA of 20/50 OD/OS at that time. A second exam finding that could possibly suggest a functional vision loss component occurred during the refraction. Initially, MJ saw 20/200 OD/OS at distance; however, was able to read the 20/40 line at near. This represents an inconsistency as the angle of resolution should be the same at both distance and near. Finally, as seen in Figure 6, a tangent screen visual field test was performed at both one and two meters. In a patient with either intact vision or organic disease, visual field should expand with increasing distance in a characteristic cone shape. In a patient with NOVL, the field will not expand and will remain the same at both one and two meters, producing a tube shape.⁵ Figure 6 displays this tubular field OD and OS. Although these findings were suggestive of NOVL, a complete

Table 1. Testing Completed During Initial Examination, 1990
*****Abnormal findings are in bold**

Testing	Results
Refraction	BCVA OD: 20/200; 20/50 with coaxing BCVA OS: 20/200; 20/50 with coaxing
Stereo	8/9 on Titmus circles
Red Saturation	Normal
PIP (Ishihara)	2/14 OD/OS
Farnsworth D-15	Normal OD/OS
Amsler Grid	Normal OD/OS
OKN	Normal OU
Anterior Segment	Normal OU
C/Ds	OD: 0.5R OS: 0.5R
Tangent Screen	Tubular field (Figure 6)
HVF 30-2	Small central scotoma
HVF 10-2	Small central scotoma that fluctuated on repeat fields
HVF 30-60	Normal OU
Fluorescein Angiography	Normal OU; (-)leakage, hyper-/hypofluorescence
VEP	Normal OU (Borderline latencies)
CT/MRI	Normal
CBC	Normal
ESR	Normal, 19
Serum protein electrophoresis	Normal
FTA-ABS	Negative
ANA	Negative
24 hr urine collection	Hg, Pb, As normal
Folate, RBC, B12	Normal

work-up was executed to rule out any possible organic causes.

All testing and results from 1991 are depicted in Table 1. An extensive work-up was completed, including a dilated fundus exam, automated perimetry, brain imaging, blood work, electrodiagnostics, and urine analysis.

As mentioned earlier, following the diagnosis of NOVL, records for MJ's visits were not found until 2014, when an outside provider made a diagnosis of macular degeneration. Following a comprehensive exam by the retinal specialist at the West Haven VA, macular degeneration was placed much lower on the list of differential diagnoses. Age-related macular degeneration (AMD) is a disease that affects central vision. Typical demographics are Caucasian, male, and typically over 50 years old. Individuals aged 43-54 have an 8% chance of developing AMD,

whereas in those aged over 75, the risk jumps to 30%.⁸ In addition, diagnosis of macular degeneration is based on clinical appearance, with differentiating characteristics such as drusen, pigment migration, and geographic atrophy.⁸ Fundus evaluation revealed only mild RPE mottling, but no other characteristic findings for AMD. Furthermore, macular OCT (Figure 1) showed no characteristic drusenoid pigment epithelial detachments (PED). The patient's younger age and race made the diagnosis even less likely.

During the course of the examination with the retinal specialist, several other potential differential diagnoses were discussed: Leber's hereditary optic neuropathy, macular pattern dystrophy, Stargardt's, achromatopsia/cone dystrophy, and chronic central serous chorioretinopathy. A brief discussion of each disease entity and the pertinent exam findings follows.

Leber's hereditary optic neuropathy (LHON) is a mitochondrial genetic disease with preponderance for young adult males. Optic atrophy is a universal trait of LHON, accompanied by reduced vision not exceeding 20/200. A fluorescein angiography, while not essential for proper diagnosis, confirms the non-vascular origin of the atrophy when there is no leakage from the optic nerve. Pattern ERG can be used to confirm optic nerve dysfunction. Family history may be positive for visual dysfunction on the maternal side. CT and MRI scans are also usually unremarkable.⁹ Following the initial retinal evaluation, MJ was seen by a neuro-ophthalmologist to rule out LHON in addition to any other possible optic neuropathies. The lack of nerve head pallor and normal RNFL OCT excluded LHON. The neuro-ophthalmologist further stated that there was likely no other optic neuropathy and relegated the decreased vision to the outer retinal degeneration noted on the macular OCT in combination with NOVL.

Autosomal dominant macular pattern dystrophies comprise five similar types: adult-

onset foveomacular vitelliform dystrophy, butterfly-shaped pigment dystrophy, reticular dystrophy, multifocal pattern dystrophy, and fundus pulverulentus. These diseases, originating from a mutation in the peripherin/RDS gene, are characterized by pigment disruption and migration primarily affecting the RPE.¹⁰ Disease onset is typical in the 4th and 5th decades of life and may lead to progressive vision loss. Hannan et al. show that macular OCT typically reveals RPE disruption, with expanding hyper-reflectivity extending from the RPE towards the outer nuclear layer. Additionally, they indicate that the FA shows variable hyper- and hypo-fluorescence.¹¹ As indicated by Boon et al., FAF shows distinctive hyper- and hypo-fluorescent patterns, which allows for better differentiation of the 5 sub-types.¹² Although MJ's macular OCT (Figure 1) somewhat resembled the lesions seen in adult-onset foveomacular vitelliform dystrophy OD, macular pattern dystrophy was discounted after viewing the results of both the autofluorescence and fluorescein angiography; both imaging sets were devoid of any significant or typical hyper- or hypo-fluorescent lesions.

Stargardt's and fundus flavimaculatus are autosomal recessive macular dystrophies caused by a mutation on the ABCA4 gene. Differentiation of the two dystrophies is ill-defined; some believe that the dystrophies are part of a continuum of the same disease. Visual loss from either can vary but is usually no worse than 20/200. Macular OCT will show disruption of both the RPE and photoreceptor integrity line (PIL) in delineated areas.¹³ The fundus hallmark of the disease is yellowish-white flecks mainly located in the posterior pole. The flecks can be differentiated from drusen as they are generally pisciform (fish-like) in shape, as opposed to the commonly round shape of drusen. FAF is not highly diagnostic, as it will show areas of hyper- and hypo-fluorescence consistent with other macular dystrophies.

An FA provides diagnostic gravitas, as it will show the defining “dark choroid,” a clinical finding where background fluorescence of the choroid is blocked by the lipofuscin build-up within the RPE. In addition, flecks generally do not hyperfluoresce during FA, in contrast to drusen. Visual fields tend to have central deficits that spread more peripherally with further progression of the disease.¹⁴ Stargardt’s, like the autosomal dominant macular dystrophies, was removed from consideration in this case: the patient’s fundus exhibited no signs of the classic pisciform flecks, fluorescein angiography was clear of any hyper- or hypo-fluorescence, and the defining “dark choroid” was not present in MJ’s FA.

Cone dystrophies are conditions that generally affect the cones within the macula. Achromatopsia, also known as rod monochromatism, is a genetic condition where no functioning cones exist within the retina. This is considered a stationary cone dystrophy as it is present in infancy and does not tend to progress. As is expected, these patients have no color vision, and BCVA is generally around 20/200. Other characteristics include pendular nystagmus and photophobia.¹⁵ Progressive cone dystrophy, in contrast to the aforementioned stationary counterpart, typically presents in childhood or early adult life. Patients generally start with cone dysfunction; however, rods may begin to be affected in later stages of the condition (at times described as cone-rod dystrophies). A prominent early symptom is photophobia. With progression, vision can deteriorate to 20/200, and color vision can progress from a deficiency to total loss. Bull’s eye maculopathy may be observed but can be as seemingly insignificant as mild RPE disruption. Fundus autofluorescence may show hyper-fluorescence early in the disease and will show hypo-fluorescence due to atrophy late in the disease.¹⁶ As it pertains to MJ’s condition, achromatopsia was removed from consideration as he had a normal Farnsworth

D-15 at 26 years of age. That finding also made the progressive cone dystrophies less likely as well. Autofluorescence provided further evidence, as no atrophy was present in MJ’s condition. Although MJ did report longstanding photophobia, the finding was insufficient for diagnosis.

Central serous chorioretinopathy (CSCR) is a disorder caused by a dysfunctional RPE that is abnormally permeable to fluid. This permeability leads to fluid retention in both the sub-retinal and sub-RPE potential spaces. The demographics of this condition typically include middle-aged males averaging between 45-51 years old. Several associated risk factors have been identified, including use of glucocorticoids, pregnancy in the third trimester, type A personality, and psychotropic medication or psychological stress.¹⁷ Psychological stress induces release of cortisol, an endogenous steroid hormone, which is part of the corticosteroid family.¹⁸ CSCR can present in an acute or chronic form. Chronicity has a varied time interval, ranging from 3 to 6 months of persistent fluid presence. Acute forms may be recurrent but tend to resolve spontaneously with minimal damage. Chronic CSCR has the potential for further damage as the sub-retinal fluid is not absorbed in an efficient manner, leading to photoreceptor loss. Additionally, individuals with chronic CSCR are at risk for choroidal neovascularization.¹⁹ Fundus evaluation will usually identify a neurosensory detachment via elevation. Chronic cases can be further identified by RPE atrophy and RPE pigment clumping. Macular OCT will show the typical neurosensory detachment located in the posterior pole. It is not uncommon to have associated PEDs within the neurosensory detachment. A thickened choroid can be found with enhanced depth imaging (EDI) OCT. An FA can display the hallmark “smokestack” pattern but more often there is a single pinpoint leak that spreads with time.¹⁷ Color vision deficits, mostly blue deficits, have been

reported in patients with CSCR.²⁰ MJ's fundus evaluation revealed very mild hypo- and hyper-pigmentation in the macula of both eyes. Macular OCT showed a small, central neurosensory detachment in the right eye and a central neurosensory detachment in the left eye with an associated hyper-reflective lesion. Hyper-reflective lesions have been reported in the past and are due to subretinal fibrosis from a possible now-quiescent retinal neovascularization.²¹ FAF was not significant. The FA showed very mild hyperfluorescence in the OS only.

MJ's presentation was atypical for several reasons. First, the lesions were located symmetrically in the central macula, where generally they can show up anywhere in the posterior pole. Additionally, the area of the central lesions was very small, whereas CSCR lesions are generally larger. Despite these characteristics, chronic CSCR may be responsible for MJ's current clinical appearance. Unfortunately, possible treatments such as laser photocoagulation depend on the FA identifying leaks in acute CSCR. There was no identifiable leak on MJ's FA, making laser an unviable option.

Unfortunately for MJ, there were two conditions contributing to his vision loss: the longstanding functional vision loss in addition to the atypical chronic CSCR. Functional vision loss remained important despite the recognition of an organic pathology. Since optic neuropathy was ruled out, it stood to reason that beyond the outer retinal degeneration at the fovea of both eyes, healthy retina remained. As mentioned, at its widest, the outer retinal lesions spanned 1300 μm OD and 700 μm OS. One degree of visual field corresponds to approximately 288 μm ,²² which in turn calculates to 4.51 degrees OD and 2.43 degrees OS in our patient. MJ was seen in the low vision clinic and underwent extensive eccentric viewing training at every clock hour. Despite this training, MJ was unable to improve beyond 20/160 OD and 20/120 OS. According to the study by Wertheim, peripheral

visual acuity at 5 degrees beyond fixation, well beyond the lesions, is expected to be at least 20/66.²³ Even at 10 degrees beyond fixation, expected VA is 20/100. As can be seen, MJ's decreased BCVA cannot be fully accounted for by the lesions. When comparing the size in degrees of the lesions on OCT, 4.51 degrees OD and 2.43 degrees OS, these numbers were significantly less than the HVF 24-2 in Figure 5. Based on the HVF, the central scotomas in both eyes were at minimum 6 x 6 degrees in size. These findings supported the concurrent existence of both NOVL and central vision loss.

Interestingly, it seems that both NOVL and CSCR have a common correlation to psychological factors. For CSCR, patients report more psychosomatic problems, unfavorable stress coping skills, and increased critical life events.²⁴ All of these factors increase both physical and emotional stress on the body. In particular, elevated aggression was a risk factor for chronic CSCR. A look into MJ's medical record showed that he had a history of aggression, agitation, and involvement in verbal altercations. NOVL has also been found to be related to concomitant psychiatric disorders (30%) or psychosocial events (36%),⁴ although these percentages vary significantly from study to study. Psychosocial events include stressors such as physical trauma and sexual abuse.⁴ This etiological link between NOVL and CSCR has been noted before, where the author suggested that care should be taken, as CSCR could mimic NOVL due to a similar presenting history of a psychological event.⁶ Perhaps, then, it is not so happenstance that MJ, with his history of post-traumatic stress disorder and aggression, developed CSCR following his longstanding NOVL. The common link is MJ's longstanding history of psychological factors. For patients with longstanding NOVL, it may prove practical to screen for CSCR due to this possible relationship.

Once NOVL has been diagnosed, management should be focused towards stressing a good prognosis to encourage visual recovery.

As with MJ, co-existing ocular pathology and patients' concern for it may incite or exacerbate the functional component, so this must also be addressed. Care should be taken to voice concerns in a compassionate and supportive manner, as confrontations are rarely helpful.²⁵ Reassurance alone seemed more likely to result in recovery than would the addition of nonspecific treatments like eye exercises, glasses, eye drops, or placebo medicines.²⁶ Additionally, Chen et al. recommended at least one follow-up appointment to give the patient the perception that the problem was undergoing active management.²⁷ Good prognostic indicators for full recovery are young age and lack of associated psychiatric disease.⁵ Because MJ did not have these good prognostic indicators and therefore was at risk for having persistent NOVL, he was ultimately referred for psychiatric/psychological counseling. In general, because adults are more prone to having psychiatric disease,⁴ it is suggested that the focus of treatment be on the psychiatric condition itself rather than the visual dysfunction.²⁶

This case reiterates the importance of thorough examinations, even in individuals with well-established chronic conditions. In 1991, MJ did not have outer retinal lesions; his Amsler grid was clear, his color vision was normal, and his fundus examination was unremarkable. Fast-forwarding to 2016, definitive organic pathology was present with reduced color vision and a central scotoma on Amsler grid in both eyes. At some point during that 25-year gap, the organic condition developed. It is not clear whether the patient was seen regularly, but would a full work-up have been completed had the patient not already been diagnosed with functional vision loss? This case underscores a critical diagnostic bias that can strongly impact the outcome of an encounter. Premature closure bias occurs when further information is not sought after once a diagnosis is made.²⁸ MJ was impacted by this bias once he was identified to have NOVL.

Once that occurred, further organic pathology was not thoroughly investigated, which ultimately influenced diagnosis, management, and treatment.

Conclusion

Although chronic CSCR is not a progressive disease, and MJ will not likely experience further organic loss due to it, it was critically important to identify it. Knowing the diagnosis, particularly when the disease is treatable, is essential in proper patient management. MJ was diagnosed with functional vision loss in 1991, and that label stayed with him until just recently. It is possible that having this diagnosis allowed providers to forego additional testing as the vision loss was already accounted for. That assumption, however, runs significant risk.

In healthcare, providers strive to come to the correct diagnosis as quickly and efficiently as possible so as to direct proper treatment. Soaring healthcare costs make a quick diagnosis cost-effective. Once patients are diagnosed with a particular condition, they are then taken down that specific disease's treatment protocol. The inherent difficulty in this delivery method is the risk of missing newer conditions as they develop, a notion that is highlighted in this case report. Underlying this topic is the medically oriented Hickam's dictum, which states that patients can have as many diseases as they please. It is easy to fall back to medical parsimony and to assign all symptoms a patient has to an established diagnosis, but that is not always the most prudent. Providers must remain vigilant at all times, particularly in cases where the vision loss is multifactorial.

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