Case Report # ____

Stop the Spinning: An Approach to the Dizzy Patient

Candidate # ____

Category B: Ocular Disease / Surgery

Topic V: Diagnosis & Management of Ocular Disease – Posterior Segment

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ABSTRACT

Purpose: The purpose of this article is to offer the eye care provider a systemic approach to the dizzy patient.

Case report: A 50-year-old Caucasian male presented for an ocular examination with the complaints of periodic dizziness and diplopia. Ophthalmic findings and subsequent testing revealed undiagnosed multiple sclerosis.

Discussion: A systematic approach to the dizzy patient with attention to the history and examination red flags is important to correctly diagnose the underlying etiology.

Conclusion: Communication of ophthalmic findings with the healthcare team is invaluable for accurate diagnosis, treatment, and management.

Key Words: *Dizziness, vertigo, diplopia, nystagmus, multiple sclerosis*

INTRODUCTION

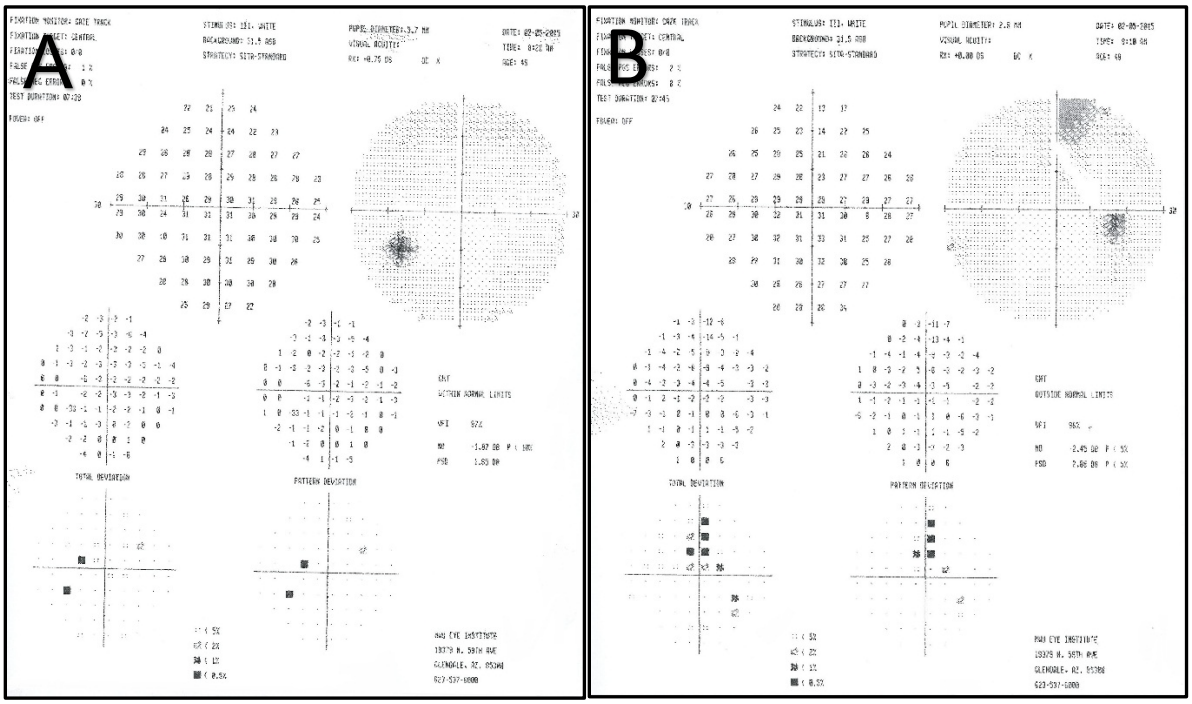
Dizziness is a term used to describe a variety of symptomatology including lightheadedness, presyncope, disequilibrium, and vertigo.[1] Vertigo and dizziness account for 2.5% of US emergency department visits per year.[2] This is an estimated 3.9 million encounters.[3] Genetic factors may play a role the development as in Ménière disease and migraine. The most common risk factors include advancing age and previous dizziness episode. Symptoms include painless head discomfort as in lightheadedness, feeling like one is going to pass out as in presyncope, feeling off balance as in disequilibrium, or feeling like the environment is spinning as in vertigo. Associated causes are generalized to inner ear problems, neurologic, cardiac, and psychiatric conditions, medications, blood disorders, and trauma. Differential diagnosis is primarily determined via history and physical exam. Management strategies focus on treating the underlying etiology; antiemetic medications are typically prescribed to mitigate the negative symptoms of nausea and vomiting.

While the majority of acute dizziness cases will present to the emergency department, the eye care provider may be consulted to evaluate for ocular pathology, and in some cases, may be the first to encounter it. This article will offer the eye care provider a systematic approach to the dizzy patient outlining the importance of a detailed case history with attention to the red flags that signal a serious underlying etiology.

CASE REPORT

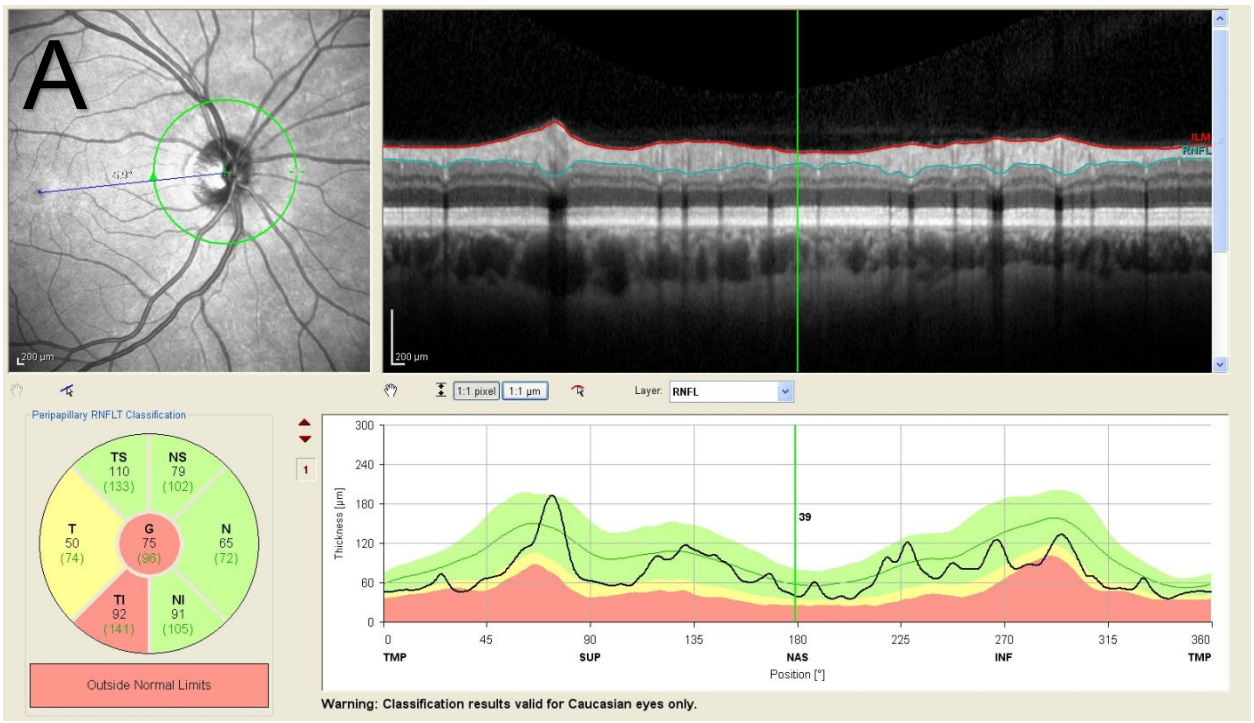
A 50-year-old Caucasian man presented for an ocular examination with the complaint of periodic dizziness and binocular horizontal diplopia in extreme right gaze for two months. Ocular history revealed presbyopia OU. Medical history included allergic rhinitis, childhood asthma, depression, insomnia, and vitamin D deficiency. Medications included clonazepam (Klonopin®, Genentech, CA), meclizine (Antivert®, Pfizer, NY), multivitamin (Centrum OTC®, Wyeth Consumer Healthcare, South Africa), Nicoderm CQ (nicotine patch, GSK, Middlesex, UK), and vitamin D3 (Caltrate®, Pfizer, NC). He had no known drug allergies. Family history was positive for diabetes, heart disease, hypertension, and lung cancer. Social history was positive for cigarette use; he denied alcohol or illicit drug use.

Best corrected visual acuity was 20/20 OD, OS. Pupils were normal without afferent pupillary defect. Extraocular muscles revealed gaze-evoked and torsional nystagmus as well as right horizontal gaze palsy. Visual fields are shown in Figures 1A and 1B. Cover test was orthophoric at distance and 6 exophoria at near. Ishihara color vision was reduced at 0/12 OD and 2/12 OS. Anterior segment examination was normal. Goldmann applanation tonometry was 19 mmHg OD and 17 mmHg OS. Angles were open gonioscopically OD, OS. Posterior segment examination revealed trace temporal pallor of the optic nerve heads bilaterally; the cup-to-disc ratio measured 0.30 x 0.30 OD and 0.50 x 0.50 OS. All other findings were normal. Spectralis optical coherence tomography (OCT) of the retinal nerve fiber layer (RNFL) is shown in Figures 2A & 2B.



FIGURES 1A & 1B

Humphrey visual field 30-2 SITA STD demonstrating superior defects extending from the blindspot of the right eye (1B, right) and extension of the blindspot of the left eye (1A, left). Both studies are reliable.



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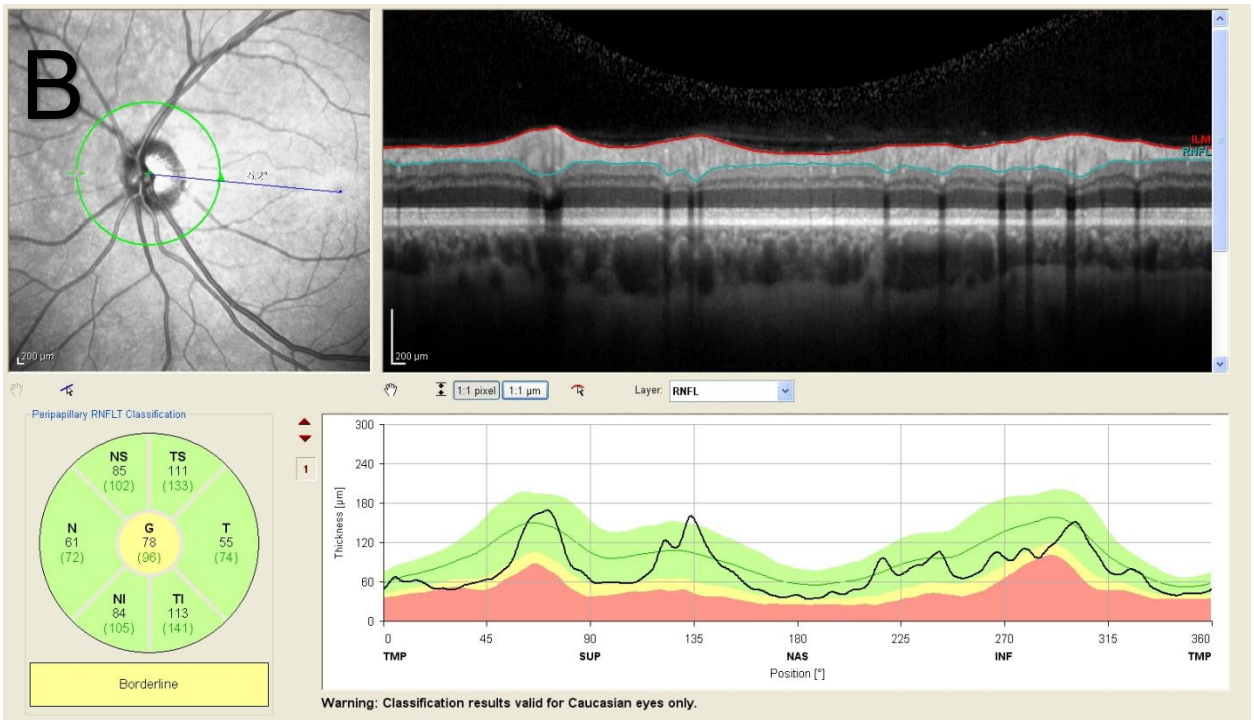

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57 **Figures 2A & 2B**

58 Spectralis OCT of the right eye demonstrating abnormal thinning of the inferior-temporal retinal nerve fiber layer

59 and borderline thinning temporally (2A); the left eye exhibits borderline generalized thinning (2B).

Given the ocular and systemic findings, the patient was advised to present to the emergency department for STAT neurology evaluation and testing. Magnetic resonance imaging (MRI) of the head and orbits with and without contrast revealed periventricular white matter, pontomedullary junction and brainstem demyelinating lesions as shown in Figures 3A-3H. Spinal MRI revealed multiple abnormal T2 foci within the cervical and thoracic regions as shown in Figure 4. Lumbar puncture and cerebrospinal fluid analysis revealed positive oligoclonal banding. Complete blood count with differential, CMP, Westergren ESR, CRP, ANA, FTA-ABS, RPR, vitamin B12, folate, homocysteine, MMA, NMO antibody, JC virus, and lipid panel were normal or negative. Clinical findings of dizziness, gaze palsy, and nystagmus correlated with brain lesions due to multiple sclerosis (MS). Bilateral optic atrophy was attributed to previous asymptomatic episodes of demyelinating optic neuritis and/or active MS-associated accelerated retinal nerve fiber layer loss. He was started on Tysabri® (natalizumab, Biogen, MA) and referred for physical, occupational, and speech therapy. Smoking cessation counseling was provided. Dizziness and eye movement abnormalities improved significantly with treatment; the optic atrophy remained stable.

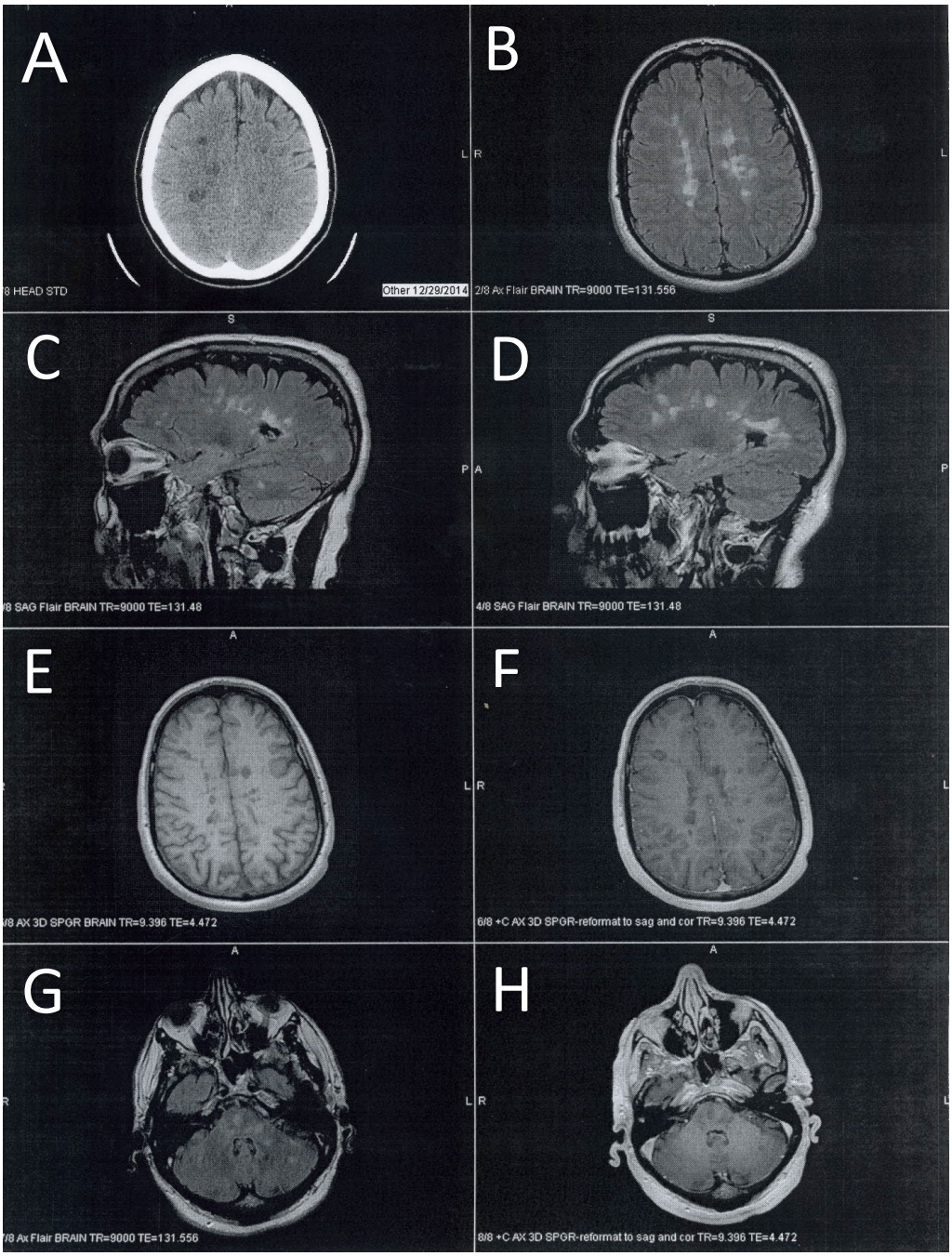

Figure 3A-3H

Axial MRI brain showing multiple cortical white matter lesions (A); T2-axial fluid attenuated inversion recovery (FLAIR) image (B); sagittal T2-axial FLAIR highlighting multiple periventricular white matter lesions and 1 focal cerebellar lesion (C); sagittal T2-axial FLAIR exhibiting Dawson's fingers (D); axial T1-weighted 3D spoiled gradient (SPGR) echo (E); axial T-1 weighted 3D SPGR-reformat to sagittal and coronal (F); T2-weighted axial FLAIR image

depicting brainstem and cerebellar demyelinating lesions (G); and axial 3D SPGR reformat to sagittal and coronal (H).

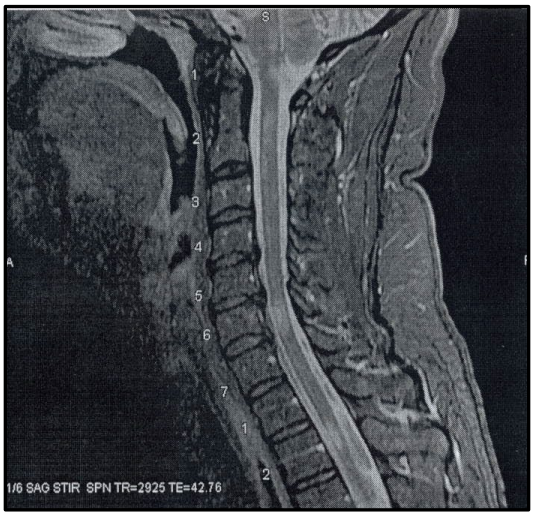

Figure 4

Sagittal short T1-inversion recovery (STIR) MRI cervical and upper thoracic spine indicating multiple foci of high signal intensity consistent with demyelinating lesions.

DISCUSSION

The historical approach to dizziness involved the practitioner asking of the patient, "What do you mean by dizzy?" If the patient responded with spinning or motion implying vertigo, a search for a vestibular cause was evaluated.[1] Impending faint, or presyncope, a cardiovascular disease was sought. Disequilibrium, or unsteady gait, a neurologic etiology was most concerning, and non-specific dizziness or any other dizziness sensation, a search for psychiatric or metabolic cause would be examined.

Newer approaches to the dizzy patient focus on triage, timing, triggers, and telltale signs in a search for an accurate diagnosis.[4, 5] Triage involves focusing on clinical red flags that immediately point to a serious cause of dizziness requiring *emergent* evaluation as outlined in Table 1.

Table 1. Clinical Red Flags that warrant emergent evaluation

Triage: Dizziness Red Flags
Abnormal vital signs
Confusion / impaired mental status
Associated pain
Neurologic symptoms (e.g., diplopia, dysarthria, dysphagia, etc.)
Cardiovascular symptoms (e.g., chest pain, dyspnea, syncope)

Abnormalities of vital signs or mental state may be manifestations of Addisonian crisis, anemia, carbon dioxide poisoning, decompression sickness, herpes simplex virus encephalitis, hypercapnia, hypoglycemia, hypoxia, hypotension, isoniazid intoxication, mountain sickness, myxedema, subdural hematoma, thyroid storm, or Wernicke syndrome. Pain, depending on location (abdomen, back, chest, ear, head, neck), may be a harbinger of abscess, carbon monoxide poisoning, carotid or vertebral dissection, giant cell arteritis, herpes zoster, intracranial hemorrhage, intracranial pressure abnormality, malignant otitis externa, mastoiditis, meningitis, myocardial infarction (MI), otitis media, pituitary apoplexy, pneumonia, pulmonary embolism (PE), spinal cord compression, thoracic/abdominal aortic aneurysm or dissection. Finally, neurologic or cardiovascular symptoms should prompt an emergent neurologic or cardiac evaluation, respectively.

The remainder of patients not exhibiting emergent red flag symptomatology are further divided by the timing of dizziness as transient / episodic (lasting seconds to hours) or persistent / continuous (lasting days to weeks). Transient / episodic dizziness is more commonly benign in nature and may include benign paroxysmal positional vertigo, benign orthostatic hypotension, Ménière disease, panic attack, reflex syncope, or vestibular migraine. The provider should be cautioned that more dangerous mimics of benign causes presenting as transient / episodic dizziness include cardiac arrhythmia, cardiovascular emergencies (e.g., MI, aortic dissection, PE, and occult GI bleed), neuro-humoral

neoplasm (e.g., insulinoma, pheochromocytoma), transient ischemic attack, and toxic exposure (e.g., carbon monoxide). Persistent / continuous dizziness etiologies of the more common, benign nature include drug toxicity (e.g., anticonvulsants), herpes zoster oticus, vestibular neuritis, or viral neuritis. The provider should be cautioned to exclude the dangerous mimics that may also present with persistent / continuous dizziness including bacterial labyrinthitis / mastoiditis; brainstem encephalitis (e.g. listeria, herpes simplex) or Miller Fisher syndrome; brainstem, cerebellar, or labyrinthine stroke; or Wernicke syndrome.

For patients with transient dizziness less than 24 hours duration, a detailed history and exploration for triggers will help to further elucidate an etiology. In general, transient dizziness that is exertional and spontaneous, or untriggered, is most likely related to a dangerous cause. A trigger such as a change in head position is more likely related to a benign cause and may be replicated on physical exam (e.g., Dix-Hallpike maneuver).

For those cases with persistent dizziness greater than 24 hours, a neurologic examination with attention to the vestibulo-ocular reflex responses, ocular alignment, and the presence or absence of nystagmus is necessary as these patients are at high risk for stroke.

In this case, the symptom of dizziness in conjunction with multiple neurologic and ocular signs warranted a STAT evaluation for a serious underlying systemic etiology. The differential diagnosis for gaze-evoked nystagmus, tortional nystagmus, horizontal gaze palsy, and bilateral optic atrophy was considered to arrive at the diagnosis and will be discussed individually.

Gaze-evoked nystagmus is the most common type of jerk nystagmus.[6] It is characterized by large amplitude movements beating in the direction of gaze. It should be contrasted with physiologic endpoint nystagmus where a small degree of nystagmus in extreme gaze is seen in 50% of normal subjects but in not sustainable. Gaze-holding structures of the brain include the midbrain, lateral medulla, and the cerebellar flocculus. Smooth pursuit and gait abnormalities are often present.

Rebound nystagmus may also be present briefly which suggests a vestibulocerebellar lesion. One of the most common etiologies is due to toxic or metabolic dysfunction secondary to anticonvulsant medication such as phenytoin or other commonly prescribed medications such as lithium and sedatives. Other causes include: spinocerebellar degeneration, MS, stroke, posterior fossa tumor, gluten sensitivity with anti-gliadin antibodies, spinocerebellar ataxia, myasthenia gravis (MG), and Miller-Fisher syndrome.[7, 8]

Rotary or torsional nystagmus is caused by dysfunction of vertical semicircular canal inputs from one side or by otolith dysfunction. It often presents as a constant nystagmus with oscillopsia that may dampen with convergence. Smooth pursuits may be poor; a skew deviation is present in approximately 30% of patients. The most common etiologies are lesions of the pontomedullary junction involving the vestibular nucleus including stroke, MS, or tumor of the pons or cerebellum.[9] Other rare causes include: vascular compression, trauma, syringobulbia, seizures, and congenital.

Horizontal gaze palsy involves loss of all conjugate movements toward the side of the lesion including saccades, pursuits, and vestibulo-ocular reflex (VOR) with sparing of vertical movements and vergence. Horizontal gaze-evoked nystagmus occurs on looking contralesionally, with fast phases directed away from the lesion. Causes include congenital, brainstem lesions, cerebellar mass, or neurodegenerative (e.g. Wernicke encephalopathy, Gaucher disease, Leigh Syndrome).[8]

The differential diagnosis of optic atrophy includes the following general categories: congenital, inflammatory, demyelinating, ischemic, compressive, infiltrative, paraneoplastic, traumatic, nutritional, toxic, radiation, papilledema, and hereditary.[10, 11] Clinical characteristics and test results of this case suggest the most likely etiology of the bilateral optic atrophy was due to former asymptomatic episodes of demyelinating optic neuritis and/or active MS-associated accelerated retinal nerve fiber layer loss. Longitudinal OCT studies have shown RNFL axonal loss in the absence of acute optic neuritis in MS

patients.[12, 13] Active MS has also been associated with accelerated retinal ganglion cell and inner plexiform layer thinning.[14]

So, what is the role of the primary eye care provider in the patient presenting with dizziness? A detailed history is critical with questions pertaining to the dizziness red flags. Onset date, duration, frequency, context, triggers, aggravating or alleviating factors, and any associated signs or symptoms are important. The ocular examination should focus attention on the cover test and extraocular muscle findings looking for evidence of smooth pursuit, saccadic, cranial nerve, nystagmus or saccadic intrusion abnormality. Additional optokinetic nystagmus, Halmagyi head thrust maneuver, oculocephalic response (e.g., Doll's eye maneuver), Dix-Hallpike maneuver, cranial nerve screen, and gait assessment tests are helpful in isolating where in the brain a problem is most likely. Physical examination may also include blood pressure measurement (sitting and supine), heart rate, and temperature.

CONCLUSION

The primary eye care provider may be the first physician to encounter the patient with a complaint of dizziness. Awareness of trigger red flags is useful for prompt emergent evaluation. A careful ophthalmic examination with attention to history, cover test, and extraocular muscles as well as ancillary testing results are valuable to ascertain an underlying etiology. Communication of the ophthalmic findings to other medical specialists is important in the differential diagnosis of dizziness.

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