"I Can't Read": A review of the ophthalmological features of Parkinson's disease

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Chief Complaint: Difficulty Reading

History of Present Illness: A 67-year-old man presented to the strabismus clinic with increased difficulty reading for the past year. He experienced intermittent, horizontal, binocular diplopia and blurring of his vision while reading, accompanied by fatigue and headaches. He had been prescribed numerous bifocal glasses with different strengths of lower segment reading adds with no relief.

Past Ocular History: Bilateral ocular surface irritation for which he used preservative-free artificial tears.

Medical History: Multiple minor falls for which the patient uses a cane.

Medications: None

Family History: None

Social History: Lives with his wife in a two-bedroom apartment. No smoking or alcohol consumption.

Physical Exam

- Visual Acuity (with correction) 20/30 OD and 20/30 OS
- Extraocular Motility: Ductions were full, and upward saccades were hypometric. Frequent small amplitude horizontal saccadic movements interrupted fixation, taking his gaze off target for less than a second.
- Orthoptic Examination (with correction): There was a 5 prism diopter exodeviation in primary position at distance, which increased to 12 prism diopters at near. At near there was an intermittent exotropia which

was not present at distance. Near point of convergence (NPC) measured greater than 10 cm.
- Pupils: 4 mm dark, 2 mm light OD; 4 mm dark, 2 mm light OS; no relative afferent pupillary defect (RAPD) OU
- Intraocular Pressure: 16 mm Hg OU
- Confrontation Visual Fields: Full OD and OS
- Hertel Exophthalmometry: 16mm OD and 16 mm OS
- External Examination: There was a 4 Hz hand tremor at rest, staring facial expression, and a reduced blink rate.
- Anterior Segment Exam: Bilateral meibomian gland inspissation, mild conjunctival hyperemia and inferior punctate erosions.
- Dilated Fundus Exam: Occasional drusen, otherwise normal disc, macula, and periphery OU

Clinical Course

Convergence insufficiency was suggested by the patient's history of binocular horizontal diplopia occurring while reading and examination finding of an exodeviation that was worse at near with an increased NPC. The patient's pill-rolling resting tremor, reduced blink rate, hypometric vertical saccades, and square-wave jerks were consistent with an underlying diagnosis of idiopathic Parkinson's disease. The patient was referred to a neurologist who confirmed the diagnosis and initiated dopaminergic therapy. He was initially instructed to perform "pencil push up" exercises to improve his convergence amplitudes. When these did not help, he was prescribed a separate pair of reading glasses fit with base-in prism. He was given a reading stand with improved lighting to reduce the impact of hand tremor on reading stability. These interventions, along with continued ocular surface lubrication, significantly improved his reading comfort.

Discussion

The classic motor features of Parkinson's Disease (PD) include resting tremor, bradykinesia, rigidity, and postural instability; these may accompany non-motor features, such as dysautonomia and depression. In PD, it is thought that there is reduced dopaminergic input from the substantia nigra in the midbrain to the stratum. However, mounting evidence from neuroimaging, pathology, and electrophysiology suggests that involvement of the fronto-striatal system goes beyond alterations in dopamine-mediated circuits alone.[1, 2]

Ophthalmic features of PD are diverse. Abnormalities in contrast sensitivity, color vision, and impaired spatial processing are reported in the literature. The most common complaint of patients with PD relates to ocular surface irritation, and two thirds of patients have clinical evidence of dry eye.[3] Biousse *et al.* found a reduced tear film break-up time in patients with early PD, reflecting impaired function of the mucin layer of the tear film.[3] The ocular surface may also be affected by a reduction in blink rate and amplitude. Some authors suggest that blink rate may increase with systemic dopaminergic therapy.[4] Evidence of blepharitis may be found in 75% of patients with PD, but this did not significantly differ from controls in one study.[3] Management of the ocular surface consists of avoidance of systemic medications that contribute to dry eye such as anticholinergics, management of coexisting blepharitis with warm compresses, eyelid scrubs, dietary omega-3 acids, and liberal use of preservative-free artificial tears. Punctal occlusion should be reserved for patients with ocular surface disease refractory to more conservative options.

Convergence insufficiency is a common cause of eye strain, binocular blur, and diplopia.[1, 3] Patients with convergence insufficiency are unable to converge when a target is brought closer than 10 cm from the bridge of their nose (NPC > 10 cm), and orthoptic evaluation will reveal an exodeviation that is worse at near. The lack of diplopia at distance distinguishes convergence insufficiency from a decompensated exophoria. Biousse *et al.* found reduced convergence amplitudes in 80% of patients with early PD versus 25.8% of controls but did not

identify significant differences in NPC.[3] Many strategies can be employed to improve the reading comfort of patients with PD: the use of a reading stand can eliminate the contribution of instability from postural tremor. Optimizing ambient lighting conditions may assist patients with reduced contrast sensitivity and color discrimination. Bifocal lenses actually may contribute to convergence insufficiency in presbyopes; thus, a trial of reading glasses fitted with base-in prism can improve reading comfort. Improvement in convergence amplitudes and NPC have been reported after the initiation of dopaminergic therapy.[1, 3, 5]

Ocular motility dysfunction is common in patients with PD. In the early stages of PD, voluntary saccades may be hypometric with normal latency and velocity, with upward saccades being affected first. In contrast, in progressive supranuclear palsy (PSP), slowing of vertical saccades is observed, and downward saccades are the first to be compromised. Eyelid retraction is a classic feature of PSP which does not occur in PD. The eventual difficulty with downward saccades in both PD and PSP may make the use of bifocal lenses impractical.[1-3]. Patients who demonstrate hypometric saccades may be instructed to draw a finger across the text to improve fixation stability and improve reading comfort.

Square-wave jerks are small saccadic intrusions (amplitude typically 0.1-0.4 degrees) that take the eye off the target. There is a subsequent inter-saccadic interval, after which the eyes return to the target by an opposing horizontal saccade. Square-wave jerks are thought to arise from disinhibition of saccade-related cells in the brainstem, including those of the superior colliculus which receive inhibitory signals from nigro-tectal pathways. Square-wave jerks are more frequent in patients with both PD and PSP. In patients without PD, square wave jerks may be observed, although they generally occur less frequently than five per minute. In PD, they are seen more frequently than five per minute, and in PSP, they may be observed on average 30 times per minute. The ratio of square-wave jerks to blink rate is thought to be a defining clinical characteristic that differentiates PD and PSP; in PD, the ratio is generally < 2, while PSP generally demonstrates a ratio > 3. [2]

Hallucinations, almost certainly underreported, occur in 9-44% of patients with PD and may occur early in the course of the disease. Visual hallucinations last between seconds and minutes and commonly take the form of an object or form which passes sideways across the patient or the vivid sensation that a another person or form is present in the vicinity. Hallucinations may also be auditory, tactile, or paranoid in nature and can be worsened by dopaminergic therapy.[6-9]

Blepharospasm and apraxia of eyelid opening (ALO) are frequently observed in patients with advanced PD and PSP. Blepharospasm is characterized by bilateral uncontrolled involuntary spasms of the eyelid protractor muscles and brows. Contractions of the procerus, corrugator, and orbicularis oculi are readily observed on clinical examination. Blepharospasm is exquisitely sensitive to injections of botulinum toxin into the eyelid protractors.[10] ALO is a condition which may occur concurrently with blepharospasm, or independently. ALO is characterized by the intermittent inability to open the eyelids after closure in the absence of apparent contraction of the orbicularis oculi muscle. Like blepharospasm, ALO is usually responsive to injections of botulinum toxin into the eyelid protractors, but other approaches include augmenting systemic dopaminergic therapy for PD, eyelid crutches, and surgical orbicularis myectomy.[11-13] A separate review on blepharospasm and ALO can be found on EyeRounds (207-blepharospasm.htm).

Parkinson's disease encompasses characteristic ophthalmic features which include reduced contrast sensitivity, color discrimination, reduced blink rate and amplitude, ocular surface disease, reduced convergence amplitudes, eye movement dysfunction and square wave jerks, and hallucinations. Blepharospasm and ALO are also observed in patients with advanced PD and PSP and are often responsive to injections of botulinum toxin into the eyelid protractors. Specific interventions can be offered by the ophthalmologist for ocular surface dysfunction and convergence insufficiency which may improve the visual function and quality of life of patients with Parkinson's disease.

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