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Case Report

The Many Faces of Blurry Vision in Parkinson's Disease: An Illustrative Case Series

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Abstract

Introduction: Ocular disorders constitute a major component of the non-motor symptoms of Parkinson's disease (PD). Blurry vision is commonly associated with PD, but often challenging to interpret.

Cases: We report two persons with PD who both experienced blurry vision, but each with a different underlying pathology that called for specific ophthalmological and neurological treatments. In case 1 the blurry vision was caused by diplopia as a result of strabismus and convergence insufficiency, while case 2 had blurry vision due to palinopsia, a higher order visual processing deficit. Adequate treatment improved vision in both cases.

Conclusion: The clinical spectrum of blurred vision is broad, and finding the underlying aetiology can be challenging. An incomplete diagnosis impedes therapeutic successes. Neurologists should be aware of the different underlying causes of blurred vision, should master the basic therapeutic approaches and know when to refer a patient to the ophthalmology department.

Keywords: Parkinson's Disease; Ophthalmology; Visual Function

Introduction

Ocular disorders are very common in patients with Parkinson's disease (PD). They can emerge with a variety of symptoms [1-3]. The most commonly reported ocular symptoms include double vision, blurry vision, watery eyes and visual hallucinations [1,4,5]. Ocular disorders may negatively affect daily functioning and quality of life [6]. Importantly, a good vision is especially vital for PD patients to visually compensate for their common loss of motor automaticity caused by basal ganglia dysfunction. The potential impact is emphasized by the fact that ophthalmological disorders combined with postural and gait instability increase the risk of falls and fall-related injuries [7]. Hence, it is important for physicians to recognize ocular problems. The clinical spectrum of ocular symptoms is broad and diagnosing the underlying pathology can be challenging. This is even more difficult in PD patients because ocular complains may arise as part the underlying disease pathology, as side effects of medication or as a completely unrelated comorbidity. Importantly, different underlying ocular and neurological disorders may cause similar ophthalmological symptoms. Here we report two patients with PD who experienced blurry vision, caused by different underlying ocular disorders. We show that detailed medical history taking and careful ophthalmological examination can distinguish the underlying ocular disorders from neurological causes of these ocular complaints.

Case 1

A 74-year-old man with a 15-year history of tremor-dominant PD participated in our study investigating the association between visual cueing and freezing of gait. He could not see the cues correctly due to visual problems. His main complaint was blurry vision, resulting in problems in daily life functioning. He experienced difficulties walking, experienced frequent falls and he had stopped driving a car because of insecurity in traffic.

His medical history included a strabismus correction surgery. Medications included carbidopa/levodopa 25/100 retard 7 times a day, carbidopa/levodopa with controlled release 25/100mg, ropinirole 5 mg 4 times a day, tamsulosin and pantoprazole.

When we took a detailed medical history and performed an ophthalmological examination, we discovered a diversity of ocular disorders explaining his blurry vision. First, the patient experienced double vision (although reporting it himself as blurry vision), resulting in incorrectly seeing the cues while walking. This also caused difficulties reading text. He experienced

this as "blurry vision". Second, there was an increase in visual problems when performing a task at near (e.g. reading a book) and towards the evening. Ophthalmological examination showed binocular horizontal diplopia due to convergence insufficiency and decompensation of his strabismus. Also, hypometric vertical saccades were found, consistent with an underlying diagnosis of PD. There was an abnormal Schirmer and tear film break up test (TFBUT), combined with a reduced eye blink rate (EBR) indicating keratoconjunctivitis sicca (dry eyes syndrome). Finally, we diagnosed beginning cataract formation.

A multicomponent treatment strategy was installed to improve the different aspects of his ocular problems. To ameliorate his diplopia and reading comfort, he was instructed to perform "pencil push up" exercises for his convergence amplitudes. Management of convergence insufficiency includes base-in prisms, monocular occlusion, orthoptic exercises or optimization of levodopa [8]. This intervention, along with ocular surface lubrication with artificial tears, improved his reading comfort. Unfortunately, his PD deteriorated quickly, therefore he declined cataract surgery.

Case 2

A 69-year-old man with a 6-year history of tremor-dominant PD visited the ophthalmological outpatient clinic because of blurry vision. The blurry vision led to falls, difficulties with reading text and driving a car. His medical history revealed early age-related macular degeneration (AMD), atrial fibrillation and hypertension. Medications included carbidopa/levodopa 50/200 retard 3 times a day carbidopa/levodopa 25/100 retard 2 times a day, carbidopa/levodopa with controlled release 25/100mg 3 times a day, pramipexole MVA 0,375 once a day, acenocoumarin, metoprolol and atorvastatin. Detailed medical history and ophthalmological examination revealed ocular problems that worsened in the dark and problems with contrast vision. The blurry vision increased when he was tracking a moving object (for example when watching soccer on television). He also reported after images (palinopsia). We also identified fluctuations during the day, with fewer problems 20-30 minutes after levodopa intake. Interestingly, patient did not report wearing-off based on the motor symptoms. In the evening there were also complaints of sore eyes, accompanied by worsening of the blurry vision.

Ophthalmological examination showed a low eye blink rate and a dry cornea of both eyes, indicating dry eyes syndrome. There was also beginning cataract formation in both eyes. The palinopsia and decreased contrast vision were, in absence of other ocular pathology, diagnosed as a higher order visual processing deficit in PD. Slow saccades may also contribute to seeing after images when visually tracking a moving object, as a motor symptom of PD. The treatment plan consisted of multiple components. The patient got a prescription for ocular surface lubrication drops. Dopaminergic treatment was optimised. Specifically, increasing levodopa therapy (dose and frequency) improved the patient's visual problems. He reported immediate improvement of contrast vision, disappearing of after images and smooth following of moving objects after medication intake. The patient also started with an extra levodopa doses before example e.g. driving a car or riding a bicycle at night. Together with treatment of dry eyes with eye drops, the blurry vision and sore eyes at night disappeared.

Discussion

Here we present two persons with PD who both volunteered a "blurry vision". Although this symptom seemed identical at first sight, each patient in fact had a different - and multifaceted - underlying pathology that called for a different diagnostic pathway and a personalized treatment approach. This illustrates the complex clinical presentation of ocular disorders in PD. Both patients experienced their visual problems as "blurry vision", but careful medical history taking revealed different symptoms and a diversity of contributing ocular disorders. The symptoms, diagnosis and possible treatments are summarised. Both cases shared a diagnosis of keratoconjunctivitis sicca or dry eyes syndrome, that can lead to eye strain and blurry vision mostly late in the day. Therapeutic intervention with ocular surface lubrication improved the visual problems. Two thirds of PD patients have clinical evidence of dry eyes [9]. This is thought to be usually induced by an inadequate tear production, compounded by a reduced eye blink rate [10].

On top of that, the first patient had a great variety of ocular disorders, of which diplopia intervened mostly with his daily life functioning. Symptoms of oculomotor disorders including diplopia are reported by 40-60% of patients with PD [3,11]. Binocular diplopia due to convergence insufficiency or pre-existing strabismus should be differentiated from monocular diplopia. Monocular diplopia generally results from refractive errors, corneal pathology, cataract formation or macular disorders. Convergence insufficiency is highly prevalent in PD (31%) and is associated with intermittent binocular diplopia in near vision [1]. It is often experienced during reading, and could lead to eye strain, and binocular blur. Patients with convergence insufficiency are unable to converge when a target is brought to the bridge of their nose, and orthoptic evaluation shows an exodeviation that is worse at near [12]. This may be improved by exercises of eye movements, prismatic correction and optimization of levodopa.

The second patient experienced problems that were most likely caused by a higher visual order disturbance, probably linked to levodopa deficiency of the visual cortex. The substantia nigra is involved in temporal processing, attributing to motor and perceptual tasks [13]. Hence, there could be deficits in the visual perception of rapidly moving stimuli in PD which could potentially cause problems in tracking fast moving targets. This could lead to after images. In PD, the dorsolateral prefrontal cortex is also disrupted as a result of decreased dopamine availability in the striatum, which is also likely to alter parietal-lobe functioning. As a result, visuospatial deficits and impaired contrast vision are reported [13]. Visual dysfunction may also be related to alterations in dopamine levels in the retina. Dopaminergic medication can

improve those difficulties in visual processing [1], and this was also the case in the second patient.

In conclusion, the clinical spectrum of blurred vision in PD is complex and diagnosing the underlying pathology and the various contributing factors can be challenging. Besides careful medical history taking, comprehensive ophthalmological examination may help to find the often-complex underlying causes of the complaints. An incomplete diagnosis impedes therapeutic successes and leaves PD patients not only in discomfort due to blurred vision, but possibly also causes unsafety due to insufficient visual feedback to compensate for loss of motor automaticity. Indeed, both patients that are presented here experienced significant gait disability as well as frequent falls in daily life, that both improved following treatment of the ocular problems. In such patients, an adequate basic treatment can already be installed by the neurologist (using table 1 for guidance), while for more treatment-resistant patients, a timely referral to the ophthalmology department should be considered.

Author Roles

Research Project: A. Conception, B. Organization, C. Execution;

Manuscript Preparation: A. Writing of the First Draft, B. Review and Critique.

C.B.: 1A, 1B, 1C, 2A, B.B: 1A, 2B, C.H.: 2B, N.d.V: 1A,2B, T.T: 1A, 2B

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Ethical Compliance Statement

a) Both patients gave their written informed consent.

All authors have read and complied with the Journal's Ethical Publication Guidelines. "We confirm that we have read the Journal's position on issues involved in ethical publication and affirm that this work is consistent with those guidelines."

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