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

Carlijn D.J.M. Borm a  Bastiaan R. Bloem a  Carel Hoyng b
Nienke M. de Vries a  Thomas Theelen b

aDonders Institute for Brain, Cognition and Behaviour, Department of Neurology, Centre of Expertise for Parkinson & Movement Disorders, Radboud University Medical Centre, Nijmegen, The Netherlands; bDepartment of Ophthalmology, Radboud University Medical Centre, Nijmegen, The Netherlands

Keywords
Parkinson’s disease · Ophthalmology · Visual function

Abstract
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. The clinical spectrum of blurred vision is broad, and finding the underlying aetiology can be challenging. An incomplete diagnosis impedes therapeutic successes. 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 presumably caused by strabismus and convergence insufficiency, while case 2 had blurry vision partly due to palinopsia, a higher order visual processing deficit. Adequate treatment improved vision in both cases. 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.
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 2 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 quite larger cues projected with augmented reality glasses on the floor in front of him correctly, due to visual problems. These cues consisted of a large yellow sphere displayed with an angle of 6° at 175 cm distance [8]. 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 repeated strabismus surgery at the age of 71 and 72 years. Medications included carbidopa/levodopa 25/100 retard 7 times a day, carbidopa/levodopa with controlled release 25/100 mg, 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 blurry vision presumably due to strabismus with a small angle, resolving when covering either eye, indicating the ocular misalignment. This resulted in incorrectly seeing the cues while walking. This also caused difficulties reading text. Second, there was an increase in visual problems when performing a task at near (e.g., reading a book) and towards the evening.

Alignment testing revealed a comitant exophoria at distance that increased at near. The near point of convergence was 11 cm, indicating a mild convergence insufficiency. 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 [9]. This intervention, along with ocular surface lubrication with artificial tears, improved his reading comfort. Unfortunately, not only his PD but his general health 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 25/100 mg 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 (e.g., when watching soccer on television). He also reported after images (palinopsia). We also identified fluctuations during the day, with fewer problems 20–30 min 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.

The treatment plan consisted of multiple components. The patient got a prescription for ocular surface lubrication drops. Dopaminergic treatment was optimized. 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, 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 summarized in Table 1. 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 [10]. This is thought to be usually induced by an inadequate tear production, compounded by a reduced eye blink rate [11].

On top of that, the first patient had a great variety of ocular disorders, of which binocular blur due to ocular misalignment intervened mostly with his daily life functioning. Symptoms
of oculomotor disorders including diplopia are reported by 40–60% of patients with PD [3, 12]. It is important to differentiate between binocular diplopia due to convergence insufficiency or pre-existing strabismus and monocular diplopia, which in contrast to binocular 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 [13]. 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 [14]. 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 [14]. Visual dysfunction including blurry vision may also be related to alterations in dopamine levels in the retina. It may be thus possible that the blurry vision in case 2 was partly due to changed 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

| Detailed clinical history taking | Diagnosis after examination | (Possible) treatment |
|----------------------------------|-----------------------------|---------------------|
| **Case 1**                        |                             |                     |
| Symptoms worsen when performing tasks at near | Convergence insufficiency | Pencil push up therapy, base-in prisms, increase levodopa therapy |
| Binocular diplopia                | Decompensated strabismus    | Correcting refractive error, prescribing prisms |
| Symptoms worsen in the evening    | Dry eyes syndrome           | Artificial tears     |
| **Case 2**                        |                             |                     |
| Seeing after images impaired contrast vision | Higher visual processing deficit | Increase levodopa therapy |
| Fluctuations of symptoms during the day, simultaneously with levodopa intake | Dry eyes syndrome | Artificial tears |
| Sore eyes in the evening          | Refraction error            | Correcting refractive error |

The third column summarized the treatment recommendations – these can also guide clinicians aiming to support patients with visual issues in their own clinical practice.
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, which 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.

**Statement of Ethics**

Both patients gave their written informed consent to publish their case and any accompanying images. All authors have read and complied with the Journal’s Ethical Publication Guidelines. Ethical approval was not required for this study in accordance with local/national guidelines.

**Conflict of Interest Statement**

The authors have no conflicts of interest to declare.

**Funding Sources**

This study is funded by a research grant from the Stichting Parkinson Fonds (Grant No. 38000). The funders had no role in study design, data collection and analysis, decision to publish, or preparation of the manuscript.

**Author Contributions**

Carlijn Borm: design and conceptualized study; major role in the acquisition of data; analysed the data; and drafted the manuscript for intellectual content. Bas Bloem: conceptualized study; interpreted the data; and revised the manuscript for intellectual content. Carel Hoyng, Nienke de Vries, and Thomas Theelen: interpreted the data and revised the manuscript for intellectual content.

**Data Availability Statement**

Requests for data from this study will be considered by Bas Bloem in line with data protection laws. Since our data are specific patient data, the general policy is that as long as the proposed use of the data is scientifically valid and if ethics approval permits, suitably anonymized data can be shared with other researchers.

**References**

1. Ekker MS, Janssen S, Seppi K, Poewe W, de Vries NM, Theelen T, et al. Ocular and visual disorders in Parkinson’s disease: common but frequently overlooked. *Parkinsonism Relat Disord*. 2017;40:1-10.
2. Weil RS, Schrag AE, Warren JD, Crutch SJ, Lees AJ, Morris HR. Visual dysfunction in Parkinson’s disease. *Brain*. 2016 Nov 1;139(11):2827-43.
3 Borm C, Visser F, Werkmann M, de Graaf D, Putz D, Seppi K, et al. Seeing ophthalmologic problems in Parkinson disease: results of a visual impairment questionnaire. Neurology. 2020 Apr 7;94(14):e1539–47.
4 Archibald NK, Clarke MP, Mosimann UP, Burn DJ. Visual symptoms in Parkinson’s disease and Parkinson’s disease dementia. Mov Disord. 2011;26(13):2387–95.
5 Buhmann C, Kraft S, Hinkelmann K, Krause S, Gerloff C, Zangemeister WH. Visual attention and saccadic oculo-motor control in Parkinson’s disease. Eur Neurol. 2015;73(5–6):283–93.
6 Santos-Garcia D, de la Fuente-Fernandez R. Impact of non-motor symptoms on health-related and perceived quality of life in Parkinson’s disease. J Neurol Sci. 2013;332(1–2):136–40.
7 Azulay JP, Mesure S, Amblard B, Pouget J. Increased visual dependence in Parkinson’s disease. Percept Mot Skills. 2002;95(3 Pt 2):1106–14.
8 Janssen S, Bolte B, Nonnekes J, Bittner M, Bloem BR, Heida T, et al. Usability of Three-dimensional augmented visual cues delivered by smart glasses on (Freezing of) gait in Parkinson’s disease. Front Neurol. 2017;8:279.
9 Racette BA, Gokden MS, Tychsen LS, Perlmutter JS. Convergence insufficiency in idiopathic Parkinson’s disease responsive to levodopa. Strabismus. 1999;7(3):169–74.
10 Tamer C, Melek IM, Duroman T, Oksüz H. Tear film tests in Parkinson’s disease patients. Ophthalmology. 2005;112(10):1795.
11 Bowen RC, Koeppel JN, Christensen CD, Snow KB, Ma J, Katz BJ, et al. The most common causes of eye pain at 2 tertiary ophthalmology and neurology clinics. J Neuroophthalmol. 2018 Sep;38(3):320–7.
12 Visser F, Vlaar AMM, Borm C, Apostolov V, Lee YX, Notting IC, et al. Diplopia in Parkinson’s disease: visual illusion or oculomotor impairment? J Neurol. 2019 Oct;266(10):2457–64.
13 Almer Z, Klein KS, Marsh L, Gerstenhaber M, Repka MX. Ocular motor and sensory function in Parkinson’s disease. Ophthalmology. 2012;119(1):178–82.
14 Armstrong RA. Oculo-visual dysfunction in Parkinson’s disease. J Parkinsons Dis. 2015;5(4):715–26.