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Indian Journal of Clinical and Experimental Ophthalmology

Journal homepage: www.ijceo.org

Review Article

Through the eyes of Parkinson's: A narrative review of clinical spectrum and ophthalmological challenges in Parkinson's disease

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ARTICLE INFO

Article history:
Received 28-08-2023
Accepted 28-09-2023
Available online 29-12-2023

Keywords:
Parkinson's disease
Ophthalmological impairments
Vision
Retina
Neuro ophthalmology

ABSTRACT

The ophthalmic non-motor dysfunctions are most common among individuals with Parkinson's disease, manifesting both prior to motor symptoms and throughout the progression of the neurodegenerative condition. Detecting these impairments early on holds significant potential for disease identification, particularly in its nascent stages. Given the comprehensive nature of ocular involvement, encompassing both extraocular and intraocular elements of the visual system, a comprehensive evaluation stands to benefit patients. Considering the shared embryonic origin of the retina and central nervous system, exploring retinal changes in Parkinson's disease could yield insights applicable to the broader brain context. Consequently, identifying these manifestations could enhance medical assessments of Parkinson's disease and offer predictive value regarding its progression.

The impact of these ophthalmological issues extends to the quality of life of Parkinson's disease patients, accentuating the importance of addressing them. This review presents a comprehensive overview of the primary visual impairments linked to Parkinson's disease, reflecting a substantial portion of the visual challenges encountered by affected individuals and for the overall well-being of those living with the condition.

1. Introduction

Parkinson's disease is 2nd most common neurodegenerative disorder characterised by the motor abnormalities including bradykinesia, tremors and muscle stiffness. Early motor symptoms often show asymmetry and respond well to dopaminergic medications. Sleep disturbances, including REM sleep behavior disorder, commonly occur prior to motor symptom onset.[1] Other early non-motor signs encompass constipation, seborrheic dermatitis, hyposmia, and dysautonomia. Despite being primarily associated with movement issues, Parkinson's disease includes visual impairment among its reported non-motor symptoms.[2] Various studies have highlighted visual abnormalities such as color discrimination, visual acuity, contrast sensitivity, blurry vision, motion perception, and vision loss, attributed to reduced dopamine in the retina's amacrine and inner plexiform cells.[3] Dopamine is crucial for brain control, retinal function, and visual signaling. Disrupted dopamine and loss of retinal amacrine cells lead to ganglion cell

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https://doi.org/10.18231/j.ijceo.2023.094

receptive characteristics anomalies, affecting informational perception in Parkinson's.[4]

Research indicates aggregation of Alpha-Syn in inner retinal layers, suggesting its role in dopamine loss. Iron is also linked to Alpha-Syn aggregation and dopamine neuron reduction in retinal cells.[5] Establishing retinal biomarkers is crucial for early Parkinson's detection. This article evaluates lesser-recognized visual aspects of Parkinson's, stressing their clinical importance as initial disease indicators and potential outcome predictors. Special attention is given to visual processing impairments and oculomotor abnormalities. The aim is to assess relevant research and emphasize the need for retinal biomarkers in detecting this neurodegenerative disorder early.

2. Parkinson's Disease Pathophysiology

2.1. Genetic mutations and protein abnormalities

Genetic mutations hold a prominent role in the underlying mechanisms of Parkinson's disease. Specific genes such as PINK1, parkin, DJ-1, and UCH-L1 oversee essential processes like mitochondrial functioning and the breakdown of proteins through the ubiquitin proteasome system (UPS). Mutations in these genes result in misfolded proteins. The collaboration between the autophagy-lysosomal system and UPS works towards eliminating these misfolded proteins. Gene mutations including SNCA, PARK2, PINK1, DJ-1, and LRRK2 lead to profound protein misfolding and hinder the production of vital neurotrophic factors essential for maintaining protein integrity. The accumulation of misfolded alpha-synuclein proteins gives rise to structures known as "Lewy Bodies," affecting mitochondrial activity, autophagy, vesicle equilibrium, and sparking neuroinflammation.[6]

2.2. Impact of environmental toxins

Environmental toxins, such as rotenone and MPTP, interfere with the functionality of mitochondrial complex-I. This disruption results in ATP depletion in crucial brain regions and activates pathways that promote inflammation and cellular apoptosis. This process leads to the death and apoptosis of dopamine neurons. Additionally, 6-hydroxydopamine can breach the protective blood-brain barrier, causing significant damage to dopaminergic neurons. Reactive oxygen species (ROS) and free radicals like para-quinone, hydrogen peroxide, and hydroxyl radicals exacerbate cellular oxidative stress, ultimately culminating in apoptosis.[7,8]

2.3. Oxidative stress and dysfunction of mitochondria

Oxidative stress arises from an imbalance between the production of ROS and the body's capacity to counteract them through antioxidants. The primary sources of ROS in the brain are neuronal and glial mitochondria. The disruption of mitochondrial function by ROS leads to energy depletion and neurodegeneration, mediated through both caspase-dependent and caspase-independent pathways. The involvement of microglia, a type of immune cell in the brain, is pivotal in the inflammatory response. Oxidative stress negatively affects the UPS and activates microglia, resulting in reduced levels of neurotrophic factors and the release of inflammatory cytokines like TNF-α, IL-1, and IL-6. Oxidative stress-induced neuroinflammation, along with protein aggregation, apoptosis, and mitochondrial impairment, collectively contribute to the loss of dopaminergic neurons.[9,10]

3. Ocular Signs in Parkinson's Disease

Initial detection of ocular symptoms often occurs in Parkinson's disease (PD), potentially serving as early indications of the condition. Visual deficits have been proposed as potential diagnostic markers for PD. Various disorders like progressive supranuclear palsy (PSP), corticobasal degeneration (CBD), and multiple system atrophy (MSA) exhibit ophthalmological clinical signs and ocular motor impairments, contributing to the diagnosis of atypical Parkinsonism.[11]

3.1. Dopamine's role in vision

Dopamine significantly impacts several visual functions such as light adaptation, oculomotor function, contrast sensitivity, color perception, visuospatial construction, and spatial working memory.[12] Insufficient dopamine in PD leads to various visual abnormalities, including diplopia. PD patients are also prone to eyelid apraxia, blepharospasm, and dry eyes. Visual issues can be attributed to the dopamine-sensitive amacrine and inner plexiform cells within the retina.[13] The loss of dopamine-producing cells in PD patients' retinas corresponds with visual impairments. Moreover, misfolded and phosphorylated alpha-synuclein deposits have been identified in retinal tissues, possibly contributing to visual impairment.[14]

3.2. Impaired color and contrast perception

Color vision deficits are common in untreated PD patients. Reduced contrast sensitivity, particularly in spatiotemporal contrast, is observed in PD patients.[15] Both color and contrast vision deficits correlate with disease duration, and levodopa treatment may improve these impairments. PD patients might have difficulties in perceiving depth (stereopsis), which is associated with cognitive decline.[16]

3.3. Saccades and tremors

Saccadic movement abnormalities are prevalent in early PD stages, potentially serving as clinical diagnostic signs.

Aberrant eye movements, including hypometric saccades, are commonly observed in PD patients[17][17]. Ocular oscillations with rhythmic patterns resembling tremors have been reported, although their relation to disease progression and dopamine medication remains to be explored.[18]

3.4. Retinal implications

Retinal modifications are evident in PD, with alpha-synuclein accumulation contributing to neuronal damage. PD patients exhibit thinner retinal layers, especially in the inner retinal layers. Ganglion cell layer alterations and optic degeneration are observed in PD retinas. Optic abnormalities and irregular retinal thickness further characterize PD.[19,20]

3.5. Other ocular abnormalities

PD can lead to reduced blinking rates, exacerbating dry eye symptoms. Stereopsis deficits have been linked to disease severity and cognitive decline.[21] Visual acuity may also decline in PD, contributing to hallucinations. Corneal thickness and subbasal nerve density in the cornea are reduced in PD, indicating potential neuropathy.[22]

4. Ophthalmologic Diseases in Parkinson's Disease

4.1. Dry eye syndrome in Parkinson's disease

Dry eye syndrome is prevalent among individuals with Parkinson's disease, characterized by reduced tear secretion and alterations in the corneal tear film. This could stem from a lowered blinking rate and degeneration of lacrimal, meibomian, and other glandular structures.[23] A notable link has been established between the severity of dry eye symptoms and the progression of PD. Interestingly, increased lacrimation can also coexist with dry eyes in PD, potentially due to variations in eyelid movements.[24]

In cases where dry eyes result from decreased blink rates or blepharitis, consulting an ophthalmologist is advisable. Standard treatments involve artificial tears and maintaining eyelid hygiene, with potential adjustments in dopaminergic medication dosages proving beneficial. For instances of blepharospasm, subcutaneous injections of botulinum toxin may offer relief.[25]

4.2. PD and glaucoma

Individuals with Parkinson's disease face a 30% higher likelihood of developing glaucoma. Glaucoma patients exhibit lower levels of catecholamines, including dopamine, in aqueous humor and lacrimal fluid, hinting at a disruption in the eye's dopaminergic circuitry due to glaucoma. Both Parkinson's disease and glaucoma involve intricate origins. Shared pathogenic processes such as oxidative stress and microglial involvement in the central nervous system might contribute to the neurodegeneration seen in both conditions.[26,27]

4.3. PD and cataract

People with Parkinson's disease have a 1.48-fold higher chance of cataract development compared to the general population.[28] In the lenses of PD patients with cataracts, the function of glyceraldehyde-3-phosphate dehydrogenase, which plays a role in glycolysis and apoptosis activation, is diminished compared to those with cataracts but not PD.[26] Notably, cataract patients with PD demonstrate elevated levels of alpha-synuclein in their lenses after cataract surgery, suggesting a shared pathway in the pathogenesis of both diseases. The increase in alpha-synuclein and its cytotoxic aggregates in the neurons of Parkinson's disease patients and in the crystalline lens of cataract patients implies a common pathway in the development of these conditions.[29]

5. Visual Challenges in PD and Quality of Life

Many Parkinson's disease (PD) patients commonly report visual difficulties that can impact their daily activities. Reading, a sequential process involving fixations and saccades, might be affected by PD-related slow saccades and longer delays, particularly in patients experiencing cognitive challenges. Consequently, PD patients tend to read fewer words per minute compared to controls, with cognitive impairment likely contributing more to slower reading rates than motor dysfunction. Reading involves multiple cognitive areas, with the cortical reading network comprising frontal and temporal regions. Early PD stages may show impaired frontostriatal dopaminergic function, affecting executive functions, while visuospatial issues might indicate cognitive decline.[30]

Extended fixation durations in PD suggest difficulties in processing visual information and planning subsequent eye movements. More regressions in saccades might compensate for missed information during initial reading, while the increasing variation in regression amplitudes could reflect cognitive impairment with visuospatial disorder. PD individuals often engage spontaneous visual movements to compensate for impairments, like using floor patterns to prevent gait freezing. Ineffectiveness in noticing such visual cues can lead to immediate functional impact. Visual problems, combined with postural instability and gait issues, could elevate fall risk and social isolation, negatively affecting PD patients' quality of life.[31]

A battery of visual tests, including color discrimination, contrast sensitivity, and other non-motor aspects, can distinguish early PD patients from healthy individuals. These visual anomalies might arise not only during the disease's early stages but potentially before its onset. Early visual impairments may serve as predictors of the disease process, where color vision deficits correlate with

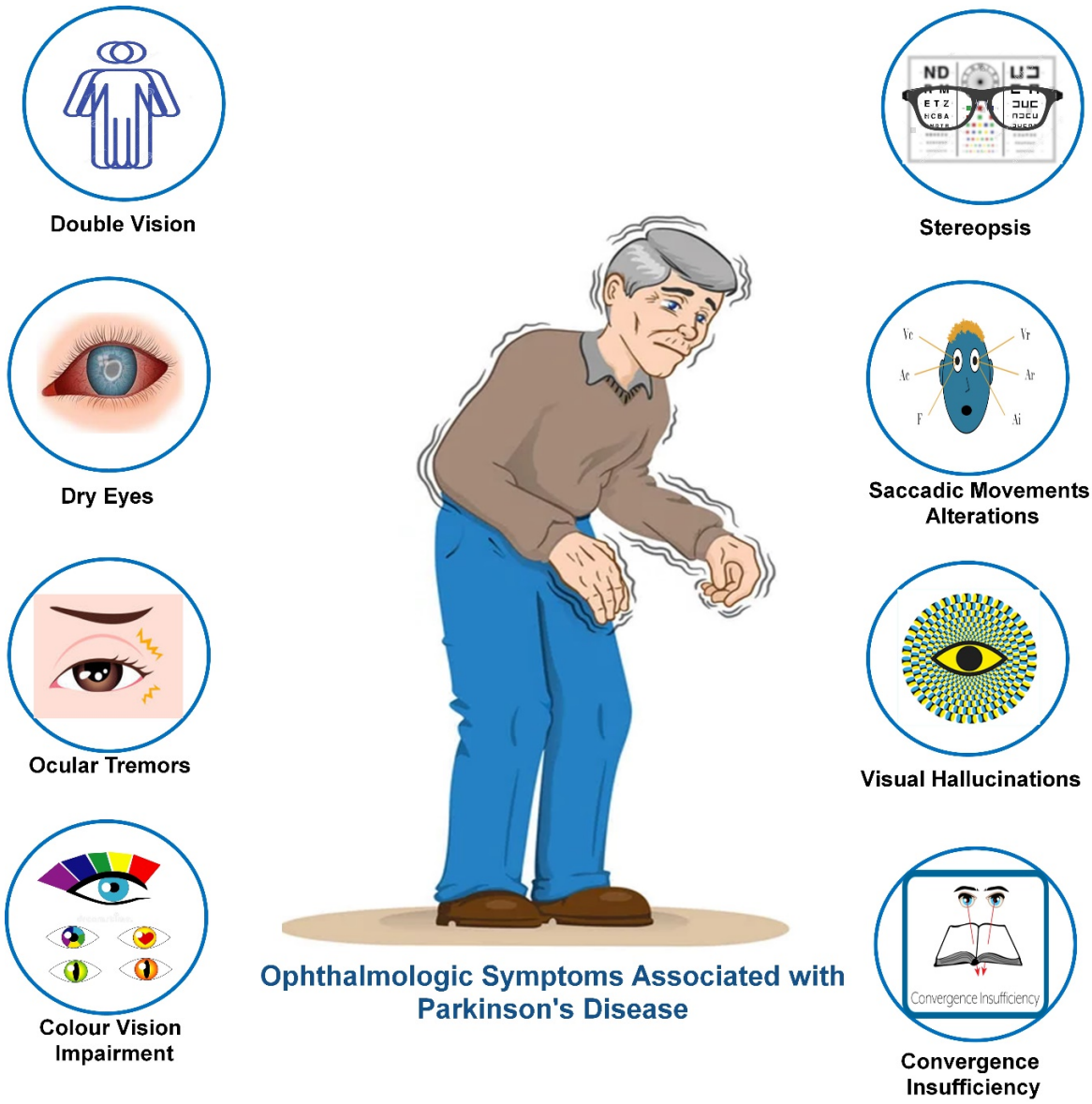

Figure 1: Ophthalmologic symptoms associated with Parkinson's disease

increased dementia risk, and stereopsis defects predict faster cognitive decline. Visual deficiencies could influence total motor performance, contributing to postural instability and increased fall risk. Phenotypic variations, such as tremor-predominant cases showing milder outcomes, remain unclear in terms of underlying causes.[32,33]

With disease progression, visual abnormalities become more pronounced. Color vision, contrast sensitivity, and stereopsis deficits correlate with disease advancement. Dopaminergic treatment's impact on visual manifestations underscores the role of retinal dopaminergic neuron decline in visual problems. Color vision alterations in pre-diagnostic rapid eye movement sleep behavior disorder and their association with progression hint at color vision deficits being a potential early diagnostic sign for Parkinson's disease.[34]

6. Management and Therapeutic Approaches

Patients afflicted with Parkinson's disease (PD) frequently communicate ocular discomfort and a sense of visual aberration. This can lead to mutual dissatisfaction for both the physician and patient, given the intricate nature of definitive diagnosis. Identifying the characteristic yet sometimes overlooked ocular manifestations in early PD empowers clinicians to prioritize optimal therapeutic strategies and reassure patients. In certain instances, fundamental interventions yield remarkable outcomes.

Physicians should discontinue medications that hinder tear production and accommodation. Concurrently, addressing conditions like blepharitis and ocular surface disorders becomes imperative. The recommendation of artificial tears to ensure appropriate corneal lubrication is pivotal.

Individuals experiencing tremors may derive benefit from utilizing a book holder for reading. As contrast sensitivity and color vision may be compromised, ensuring adequate ambient illumination while reading becomes crucial. Identifying and addressing convergence deficits and reduced convergence amplitudes might necessitate the incorporation of a base-in prism for reading glasses. Should achieving suitable single vision remain elusive despite prismatic correction, monocular occlusion during reading could offer a viable solution.[23]

Patients grappling with asthenopia or diplopia warrant a comprehensive refraction assessment and may gain from refraining from bifocals or progressive lenses. Opting for distinct eyeglasses for diverse activities such as distant vision, reading, and computer usage could prove advantageous. For those with tremors, dyskinesias, or a history of falls, prioritizing the spherical equivalent over extensive astigmatic correction in prescription eyeglasses may be judicious. Advising patients to employ their finger to guide eye movements on the page when saccadic velocity is compromised can enhance reading comfort and efficiency.[35]

Individuals reporting central hemianopia-related challenges following pallidotomy and expressing reading difficulties should undergo formal visual field assessments to discern subtle anomalies. Blepharospasm and apraxia of eyelid movement necessitate intervention with botulinum toxin and/or surgical measures if they persist despite ocular surface irritation therapy.[36] In the context of Parkinson's disease, abrupt onset visual hallucinations need not necessarily prompt modifications to therapy. By accurately identifying the root causes of patient concerns and delivering tailored interventions, potentially vexing physician–patient interactions can be circumvented, fostering a harmonious therapeutic alliance.[37]

7. Conclusions

In conclusion, the comprehensive assessment of ophthalmic non-motor dysfunctions in Parkinson's disease (PD) provides valuable insights into both early disease identification and potential predictors of disease progression. Visual impairments are pervasive and varied, impacting various aspects of the visual system, from extraocular to intraocular elements. Given the shared embryonic origin of the retina and the central nervous system, studying retinal changes can offer a unique perspective on broader brain-related processes, potentially yielding crucial diagnostic and prognostic markers. The significance of these ocular manifestations extends beyond their role in PD diagnosis. They directly affect the quality of life of individuals living with PD, potentially exacerbating mobility issues, fall risks, and social isolation. As these visual impairments may emerge even before motor symptoms, their recognition holds the promise of facilitating early medical assessments and interventions, ultimately leading to improved patient outcomes.

In the future, further interdisciplinary research is warranted to deepen our understanding of the intricate relationship between PD and its associated ophthalmic manifestations. Exploring the molecular and cellular mechanisms underlying retinal changes and their correlation with brain pathology can lead to the identification of reliable retinal biomarkers for PD diagnosis. Furthermore, continued advancements in ophthalmological diagnostic techniques, such as optical coherence tomography and novel imaging modalities, offer the potential for noninvasive and accurate monitoring of retinal changes over time. As we unravel the complex interplay between PD and visual abnormalities, collaborative efforts between neurologists and ophthalmologists are pivotal. Clinicians must remain vigilant for ocular signs in PD patients, and researchers should strive to uncover novel therapeutic approaches targeting the visual system to alleviate the burden of visual impairments in PD.

In summary, this comprehensive review highlights the prevalence, clinical significance, and potential future directions of research in the realm of ophthalmic non-motor dysfunctions in Parkinson's disease. By shedding light on the intricate relationship between the visual system and PD, we aim to drive advancements in both early disease detection and the development of innovative therapeutic strategies, ultimately enhancing the well-being and quality of life for individuals affected by this neurodegenerative disorder.

8. Source of Funding

Not applicable

9. Conflict of Interest

None.

10. Acknowledgement

Not applicable.

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Cite this article: Sonawane S, Thorat V, Bhabad S, Lamkhade G, Jadhav S, Jain R, Bhagwat A, Doke R. Through the eyes of Parkinson's: A narrative review of clinical spectrum and ophthalmological challenges in Parkinson's disease. *Indian J Clin Exp Ophthalmol* 2023;9(4):489-494.