

OPHTHALMOLOGICAL FEATURES OF PARKINSON DISEASE

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ABSTRACT

Parkinson's disease is a common neurological disease common in middle aged and elderly men. Parkinson's disease involves the degeneration of the extrapyramidal motor system resulting in movement related symptoms such as tremor, rigidity, postural instability, ataxia etc.^[1]. Some patients may also develop and experience sensory changes involving olfactory disturbances and auditory and visual disturbances. This paper will explore the ophthalmologic and overall visual problems experienced by PD patients in a study conducted at the Berat and Pogradec Ambulatory Clinics in Albania. Recent advances in ocular imaging technologies, particularly optical coherence tomography (OCT) and retinal vascular imaging, have revealed retinal nerve fiber layer thinning and other retinal alterations that may reflect underlying neurodegenerative processes.^[2] As the retina is considered an extension of the central nervous system, ophthalmological biomarkers are increasingly being investigated for their potential role in the early diagnosis, disease monitoring, and prognosis of PD. Ophthalmological manifestations in PD encompass a wide range of abnormalities such as reduced visual acuity, impaired contrast sensitivity, colour vision deficits, dry eye syndrome, decreased blink rate, convergence insufficiency, ocular motility disturbances, and retinal structural changes.

Keywords:

Parkinson's disease, ophthalmology, visual disturbances, retinal dysfunction, eye movement abnormalities, visual hallucinations, non-motor symptoms.^[3]

INTRODUCTION

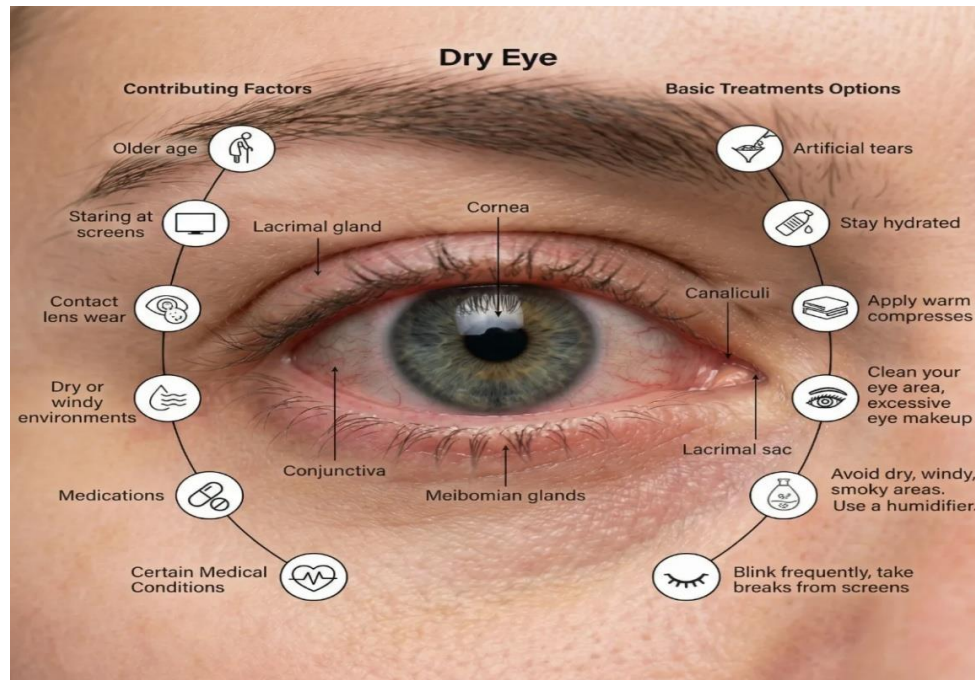
Visual disorders such as visual acuity, defects, dry eyes and visual hallucination are often under accounted for within Parkinson's Disease (PD). It is believed to be underreported by patients and also not acknowledged by their treating physicians. This study aims to identify the prevalence and the clinical effects of ophthalmic symptoms in patients with PD using a basic neurophthalmic screening tool.^[4] In a non-PD patient, decisions about movements take place in the motor cortex within the cerebellum. Via intra-cranial neurons, the action potential for the movement to take place travels to the basal ganglia. At the basal ganglia, the caudate and putamen receive the action potential for the movements required. The substantia nigra and subthalamic nucleus stimulate dopaminergic neurons to cause the binding of dopamine to the GABA receptors at the globus pallidus.^[5] The lack of inhibition now from the globus pallidus allows the firing of the thalamus to take place and so allows the generation of action potentials. These action potentials will travel along efferent neurons through the extrapyramidal pathway in order to generate movement.^[6]

1.OPHTHALMOLOGICAL FEATURES OF PARKINSON DISEASE

1.1 DRY EYE SYNDROME

Dry eyes affect the layers of tears that cover your cornea. It happens when you don't have enough tears or when your tears evaporate too quickly. Symptoms include burning, itching and a gritty feeling in your eye. You may need to try several different treatments to find a solution that works for you. Dry eye is a condition that affects your tear film, the three layers of tears that cover and protect the surface of your eyes.^[7] You need a smooth and

stable tear film to help you see clearly and comfortably. Disruptions to your tear Film can cause uncomfortable symptoms, like burning, itching, watering or blurred vision. Many different factors can prevent your tear film from working as it should and lead to dry eyes. Its causes and risk factors are numerous and complex.^[8] So, it may take a while to get to the root of the problem and figure out what's causing your symptoms. If you have dry eye syndrome, it's important to establish care with an ophthalmologist or optometrist to manage your condition.



A. SYMPTOMS

- A stinging, burning or scratchy sensation in your eyes
- Stringy mucus in or around your eyes
- Sensitivity to light
- Eye redness



- A sensation of having something in your eyes
- Difficulty wearing contact lenses
- Difficulty with night time driving
- Watery eyes, which is the body's response to the irritation of dry eyes
- Blurred vision or eye fatigue ^[9]

B. CAUSES

- Reduced blink frequency
- Autonomic dysfunction affecting tear secretion
- Meibomian gland dysfunction
- Medication-related effects ^[10]

C. CLINICAL SIGNIFICANCE

Dry eye symptoms negatively impact reading ability and overall visual comfort ^[11]

1.2 REDUCED BLINK RATE

A reduced blink rate—often caused by high visual concentration, stress, or extended digital screen use—leads to severe tear film instability. Consequences include dry eye syndrome, ocular surface exposure, digital eye strain (DES), and temporary visual blurring. ^[12]

CONSEQUENCES

- Increased tear evaporation
- Dry eye development
- Ocular discomfort
- Exposure keratopathy

Reduced blink frequency is considered a characteristic ocular manifestation of Parkinson disease. ^[13]

INCREASED TEAR EVAPORATION

In Parkinson's disease (PD), increased tear evaporation is a major contributor to dry eye disease (DED). This occurs primarily because of a reduced blink rate, impaired blink efficiency, and meibomian gland dysfunction (MGD). Blinking normally spreads the tear film evenly across the ocular surface and prevents excessive evaporation. ^[14] In PD, dopamine deficiency leads to fewer and less effective blinks, resulting in tear film instability and accelerated evaporation. Additionally, meibomian gland dysfunction, which is common in PD, disrupts the lipid (oil) layer of the tear film. The lipid layer normally slows evaporation; when it is abnormal, tears evaporate more rapidly, leading to ocular surface dryness and inflammation. ^[15]

DRY EYES DEVELOPMENT

Dry eye disease (DED) is one of the most common ophthalmological manifestations of Parkinson's disease (PD). The development of dry eye in PD is multifactorial and involves abnormalities in tear production, tear film stability, and blinking mechanisms. Dopaminergic deficiency in Parkinson's disease reduces the spontaneous blink rate and causes incomplete blinking. Normally, blinking spreads the tear film evenly across the ocular surface and prevents tear evaporation. ^[16] When blinking is reduced, tears evaporate more rapidly,

resulting in tear film instability and ocular surface desiccation. In addition, autonomic nervous system dysfunction may impair lacrimal gland secretion, leading to decreased aqueous tear production. Meibomian gland dysfunction further contributes to tear film instability by reducing the lipid layer that normally prevents excessive tear evaporation.^[17]

As dry eye progresses, increased tear osmolarity and ocular surface inflammation develop, causing symptoms such as burning, foreign-body sensation, redness, photophobia, fluctuating vision, and ocular discomfort. These symptoms can significantly affect quality of life and visual function in patients with Parkinson's disease.^[18]

OCULAR DISCOMFORT

Ocular discomfort is a common but often underrecognized symptom in patients with Parkinson disease (PD). It usually results from abnormalities in tear production, blinking, eyelid function, and ocular surface health.^[19]

EXPOSURE KERATOPATHY

Exposure keratopathy is a corneal disorder caused by inadequate protection of the ocular surface due to incomplete eyelid closure or reduced blinking. In Parkinson disease (PD), decreased spontaneous blink rate, impaired eyelid movements, and facial muscle rigidity can lead to prolonged corneal exposure, resulting in ocular surface damage. Reduced blink frequency is considered a characteristic ocular manifestation of Parkinson disease.^[20]

1.4 Reduced Blink Rate

Reduced blink rate is one of the most common ocular manifestations of Parkinson disease (PD). Healthy individuals blink approximately 15–20 times per minute, whereas patients with PD often exhibit a significantly reduced spontaneous blink rate, sometimes falling below 5–10 blinks per minute.^[21]

- The basal ganglia dysfunction and dopamine deficiency characteristic of PD impair the automatic control of blinking. As a result:

- Blinks occur less frequently.
- Blinks may be incomplete.
- Dry eye development
- Blinks occur less frequently.
- Blinks may be incomplete.
- Tear film renewal is reduced.
- Ocular surface exposure increases.

Consequences

- Increased tear evaporation
- Dry eye development
- Ocular discomfort
- Exposure keratopathy

Reduced blink frequency is considered a characteristic ocular manifestation of Parkinson disease.^[22]

1.5 COLOUR SENSITIVITY IMPAIRMENT

Contrast sensitivity impairment is the reduced ability to distinguish an object from its background, especially in low-light, fog, or glare. While standard eye charts test high-contrast visual acuity, contrast sensitivity measures functional vision. Impairment causes difficulties with activities like night driving, reading, and recognizing faces.^[23]

CLINICAL FEATURES

- Difficulty seeing in dim lighting
- Problems recognizing facial expressions
- Reduced ability to navigate complex environments Contrast sensitivity impairment often appears before significant visual acuity changes.^[23-24]

1.6 COLOUR VISION DEFICIENCY

Colour blindness or colour vision deficiency (CVD) is the decreased ability to see color, differences in color, or distinguish shades of color. The severity of color blindness ranges from mostly unnoticeable to full absence of color perception. Color blindness is usually a sexlinked inherited problem or variation in the functionality of one or more of the three classes of cone cells in the retina, which mediate color vision.^[25] The most common form is caused by a genetic condition called congenital red–green colorblindness (including protan and deutan types), which affects up to 1 in 12 males (8%) and 1 in 200 females (0.5%). The condition is more prevalent in males because the opsin genes responsible are located on the X chromosome. Rarer genetic conditions causing color blindness include congenital blueyellow colour blindness (tritan type), blue cone monochromacy, and achromatopsia.^[26] Colour blindness can also result from physical or chemical damage to the eye, the optic nerve, parts of the brain, or from medication toxicity. Colour vision also naturally degrades in old age.

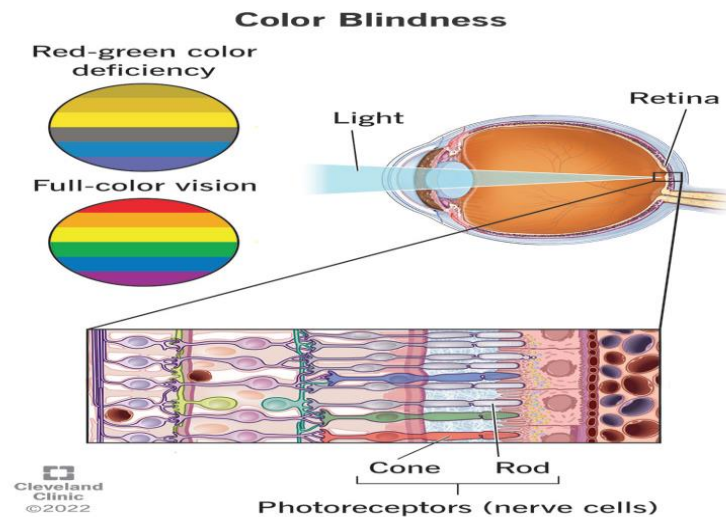
Pathophysiology

- Dopaminergic dysfunction in retinal amacrine cells
- Impaired retinal signal transmission

Colour vision abnormalities may precede motor symptoms and could represent an early^[27]

1.7 RETINAL STRUCTURAL CHANGES

Retinal structural changes refer to alterations in the cellular layers, vasculature, and tissues of the retina. They are critical clinical biomarkers for both localized eye diseases (like glaucoma and macular degeneration) and systemic neurodegenerative conditions (like Alzheimer's or Parkinson's disease). Recent retinal imaging studies have demonstrated structural abnormalities in Parkinson disease.^[28]



a. Findings

Retinal nerve fiber layer (RNFL) thinning
Ganglion cell layer loss Macular thinning

b. Diagnostic Tool

Optical Coherence Tomography (OCT) These retinal changes may correlate with disease severity and duration.^[29]

1.8 OCULAR MOTOR ABNORMALITIES

Oculomotor abnormalities refer to disorders affecting eye movements, tracking, alignment, and fixation. They range from subtle tracking difficulties impairing reading to severe ocular misalignment, double vision, and involuntary eye oscillations Eye movement abnormalities are frequently observed.

a. Saccadic Dysfunction

- Increased latency
- Reduced velocity
- Hypo metric saccades
- Smooth Pursuit Deficits
- Difficulty tracking moving objects
- Interrupted pursuit movements

b. Clinical Impact

- Reading difficulties
- Impaired visual scanning
- Reduced driving performance^[30]

1.9 CONVERGENCE INSUFFICIENCY

Convergence insufficiency refers to difficulty maintaining binocular alignment during near tasks.^[31]

Symptoms

- Diplopia (double vision)
- Eye strain
- Reading fatigue
- Headaches Symptoms may improve following dopaminergic therapy.^[32]

2. DIPLOPIA

Double vision affects a significant proportion of Parkinson disease patients Causes Convergence insufficiency.

- Ocular motor dysfunction
- Medication fluctuations
- Diplopia can significantly impair daily activities and mobility.^[33]

2.1 VISUAL HALLUCINATIONS

Visual hallucinations are particularly common in advanced disease stages.^[34]

- **Risk Factors**
 - a. Cognitive impairment
 - b. Dopaminergic medications
 - c. Sleep disturbances
 - d. Disease progression
- e. Clinical Presentation
 - f. Seeing people or animals
 - g. Complex visual scenes
 - h. Preserved insight in early stages
 - i. Visual hallucinations may indicate progression toward cognitive decline.^[35]

2.2 IMPAIRED VISUOSPATIAL FUNCTION

Impaired visuospatial function refers to a reduced ability to identify, analyze, and remember the spatial relationships, forms, and dimensions of objects. It affects how the brain perceives the world, causing difficulties with depth perception, navigation, face recognition, and assembling objects. Parkinson disease affects higher-order visual processing.^[36]

MANIFESTATIONS

- Difficulty judging distances
- Navigation problems
- Object recognition deficits
- Impaired spatial orientation
- These deficits increase fall risk and reduce independence.^[37]

3. PATHOPHYSIOLOGICAL MECHANISMS

Pathophysiology examines how normal physiological processes are altered by disease or injury. It bridges physiology (normal function) and pathology (abnormal states) by explaining the cellular, molecular, and genetic mechanisms that disrupt homeostasis to produce symptoms. Several mechanisms contribute to ophthalmological manifestations in Parkinson disease:^[38]

a. Dopamine Deficiency

Dopamine plays an essential role in retinal neurotransmission and visual processing.

b. Retinal Neurodegeneration

Accumulation of α -synuclein and neuronal loss affect retinal integrity.

C. Basal Ganglia Dysfunction

Abnormal basal ganglia activity disrupts ocular motor control.

d. Autonomic Dysfunction

Impaired autonomic regulation affects tear production and pupil responses.

e. Cortical Involvement

Visual association cortices may undergo neurodegenerative changes, contributing to visuospatial deficits and hallucinations.^[39]

4. CLINICAL SIGNIFICANCE

The practical, real-world importance of a research finding or treatment effect. Unlike statistical significance (which simply shows an effect is not due to chance), clinical significance evaluates whether the intervention makes a noticeable, meaningful difference in a patient's daily life, health, or quality of life.^[40] Ophthalmological abnormalities in Parkinson disease are clinically important because they:

- Affect quality of life
- Increase fall risk
- Impair reading ability
- Reduce driving safety
- Serve as potential early biomarker
- Provide insight into disease progression

Routine ophthalmological evaluation should therefore be included in comprehensive Parkinson disease management.^[41]

5. DIAGNOSIS

"Diagnosis reference" can mean a few different things depending on your context. Whether you are looking for medical codes, disease information, mental health criteria, or vehicle diagnostics, Clinical Assessment^[42]

a. Visual acuity testing

Colour vision assessment

b. Contrast sensitivity testing

Ocular motility examination

c. Imaging Techniques

Optical coherence tomography (OCT) Fundus photography

Retinal nerve fiber layer analysis

d. Specialized Testing

Visual evoked potentials Electroretinography Eye-tracking studies^[43]

5. MANAGEMENT

a. Medical Treatment

- Optimization of dopaminergic therapy
- Artificial tears for dry eye
- Management of visual hallucinations
- Vision Rehabilitation
- Prism glasses
- Reading aids
- Environmental modifications
- Multidisciplinary Care^[44]

Collaboration among neurologists, ophthalmologists, optometrists, and rehabilitation specialists improves patient outcomes.^[45]

6.CONCLUSION

Ophthalmological manifestations are common and clinically significant features of Parkinson disease. Visual disturbances may involve the ocular surface, retina, ocular motor system, and higher visual processing pathways. These abnormalities contribute substantially to disability and reduced quality of life. Advances in retinal imaging and visual assessment techniques have improved understanding of the relationship between ocular abnormalities and neurodegeneration. Early recognition and management of ophthalmological features can enhance patient care, improve functional outcomes, and potentially provide biomarkers for disease progression.

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