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Seeing Through the Fog: Visual Symptoms in Parkinson's Disease and Dementia

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Abstract: ***Introduction**: Non - motor symptoms such as dementia and visual hallucinations are critical determinants of long - term outcomes and quality of life in Parkinson's disease (PD). This study was motivated by the need to better understand these symptoms, particularly the visual disturbances associated with PD and Parkinson's disease dementia (PDD). **Aim**: A central aim of this study was to characterise the spectrum of visual symptoms experienced by individuals with PD and PDD. In addition to complex visual hallucinations—already recognised for their prognostic significance—we investigated a broader range of phenomena, including illusory misperceptions, presence and passage hallucinations, and diplopia (double vision). Another major objective was to define key metrics of visual exploration strategies during visuocognitive tasks, and to examine their relationship with cognition, visual symptoms, and motor function. We also evaluated the usefulness of retina - specific assessments in exploring the potential contribution of retinal dysfunction to visual impairment in PD. **Result**: Our findings indicate that not all visual symptoms share a common pathophysiological mechanism. We propose that hallucinations should be categorised into distinct phenomenological groups to improve understanding of their causes and predictive value in future longitudinal research. Additionally, we found that patients with perceptual difficulties exhibited significantly less efficient visual exploration strategies, highlighting the interplay between cognitive deficits and eye movement patterns in PD. Retinal structure, as measured by optical coherence tomography (OCT), did not show significant alterations in PD, suggesting that caution is needed before using OCT as a biomarker for the disease. Nevertheless, our neurophysiological findings point to the retina as a potential origin of diminished visual acuity in PD, despite no marked differences in central or peripheral retinal responses between PD patients and controls.*

Keywords: Parkinson's Disease; Freezing of Gait; Visual Dysfunction; Eye Movement; Autonomic Nervous System; visual impairment; visuoperceptive deficit; visuospatial deficit; visual hallucinations; dopamine

1. Introduction

Visual symptoms are a common but frequently underrecognized point of Parkinson's complaint (PD) and Parkinson's complaint madness (PDD). These symptoms encompass a wide range of marvels, including difficulty reading, double vision (presbyopia), visual visions, sensations of presence and passage, and complex visual visions (CVHs). While CVHs have been extensively studied due to their prognostic counteraccusations — particularly their association with cognitive decline — the relationship between colourful visual symptoms, optical health, and cognitive status remains inadequately understood. Minor hallucinatory gests, similar as visions and passions of presence or passage, are constantly reported by cases with PD and are occasionally inaptly grouped with CVHs. still, analogous gests also do in the general population, particularly during transitions between sleep and insomnia, as well as in conditions similar as wakefulness and brainstem diseases. These overlaps suggest that visual symptoms in PD may arise from multiple, distinct mechanisms, potentially involving both cortical and brainstem dysfunction. Visual function is also compromised in PD, with impairments observed in visual perceptivity (VA), discrepancy perceptivity (CS), colour demarcation, and stir perception. Some studies have linked VA and CS poverties as threat factors for the development of CVHs. still, cognitive capability may also impact performance on visual testing, complicating the interpretation of these associations. also, the symptom of presbyopia, though generally reported, has not been totally delved in the environment of PD. Given the eventuality for different visual symptoms to reflect distinct underpinning pathologies, a better understanding of their frequence, characteristics, and clinical supplements is essential. madness with Lewy bodies (DLB) and Parkinson's complaint madness (PDD) are both classified as major neurocognitive diseases under DSM - 5 due to the accumulation of α - synuclein protein in the brain. still, their bracket and relationship continue to be batted. Although they partake several clinical symptoms similar as madness, cognitive oscillations, visions, and signs of parkinsonism — the timing of symptom onset is used to separate them. In DLB, cognitive decline generally occurs before or within one time of parkinsonian motor symptoms, while in PDD, madness develops only after a well - established opinion of Parkinson's complaint. Despite these temporal distinctions, there's substantial clinical and pathological imbrication between the two. Both conditions parade poverties in administrative function, visual - spatial capacities, and memory — however memory impairment tends to be more pronounced in DLB. Nearly all Parkinson's cases ultimately witness cognitive decline, occasionally indeed before motor symptoms appear. Again, utmost individualities with DLB will develop parkinsonian features over time, though a nonage may not. Neuropathological studies have shown that both diseases involve wide Lewy body pathology, frequently accompanied by Alzheimer's complaint - related changes like amyloid - beta pillars and tau befuddlements especially in DLB. This has led to an ongoing discussion about whether DLB and PDD represent distinct conditions, different clinical expressions within a complaint diapason, or variations of a participated underpinning pathology told by inheritable and molecular factors. PD is primarily characterized by motor impairments performing from the progressive loss of dopaminergic neurons in the substantia nigra pars compacta,

a growing body of exploration highlights the significance of non - motor symptoms (NMSs) in complaint progression and patient quality of life. Among these, visual and visuospatial disturbances have surfaced as particularly poignant and potentially prophetic features of PD. Visual impairments in PD encompass a wide range of dysfunctions, from introductory visual processing poverties similar as reduced discrepancy perceptivity, disabled colour demarcation, and oculomotor abnormalities to more complex visuospatial and perceptual poverties affecting spatial exposure, navigation, and visual working memory. These dysfunctions contribute to substantial limitations in diurnal living conditioning, including reading, driving, and walking, eventually reducing tone - efficacity and life satisfaction.

Aim

This review aims to give a comprehensive overview of the optical, visuoperceptive, and visuospatial disturbances observed in PD, examining their neurobiological underpinnings, progression across complaint stages, and applicability as early labels of complaint onset. Prevalence Parkinson's complaint is the alternate most current neurodegenerative complaint after Alzheimer's, affecting about 1 of people over 60 and over to 3 over 80 in industrialized countries. This study aimed to (1) characterize the full diapason of visual symptoms in PD and PDD, (2) examine their association with cognitive impairment and optical abnormalities, and (3) identify distinct clinical predictors for different types of visual marvels. By doing so, we hope to inform better individual, remedial, and prognostic approaches to managing visual symptoms in Parkinsonian diseases. Pathophysiology and Clinical Impact of Visual Impairments in Parkinson's Disease individualities diagnosed with Parkinson's complaint (PD) constantly witness a variety of visual and eye movement abnormalities. These issues may affect from the underpinning neurological changes associated with the complaint or be told by long - term drug use. Visual difficulties tend to consolidate as the complaint progresses, and studies estimate that nearly 70 of PD cases report recreating visual disturbances during the course of their illness. A wide range of eye movement dysfunctions have been noted in PD. These include reduced capability to align both eyes (binocular confluence), occurrences of double vision, and slower or Page 1 of 2 further limited eye shadowing during stir. Vertical aspect, especially overhead and over movement, can also be bloodied. also, cases may show detainments in initiating rapid - fire eye movements (saccades), reduced saccade breadth (hypometria), and involuntary quick shifts in aspect known as square surge pulls. These impairments can significantly hamper diurnal functioning, making tasks like reading, writing, driving, or moving through surroundings more delicate

2. A Clinical Overview of Visual Impairments in Parkinson's Disease

2.1 Retinal Changes

In individuals with Parkinson's disease (PD), both structural and functional alterations have been observed in the eye, particularly affecting the retina and optic nerve. Advances in imaging techniques, such as optical coherence tomography (OCT), have enabled detailed visualization of these changes. Studies utilizing OCT have frequently reported thinning in several retinal layers, including the macula, retinal nerve fibre layer (RNFL), ganglion cell layer, inner and outer plexiform layers, inner and outer nuclear layers, as well as the photoreceptor layer and retinal pigment epithelium. Despite these findings, reductions in macular thickness and volume are not universally observed across all investigations. Researchers have also examined the relationship between retinal thinning and clinical measures of disease progression. While some evidence suggests that RNFL thinning may be associated with longer disease duration and increased severity, other studies have not confirmed these associations. Notably, thinning of the parafoveal ganglion cell–inner plexiform layer (GCIPL) appears to have the strongest correlation with visual dysfunction in PD patients.

2.2 Pupil Reactivity in Parkinson's Disease

Patients with Parkinson's disease (PD) often exhibit changes in how their pupils respond to light. These changes include enlarged pupil size, noticeable differences between the two pupils after light exposure, and slower pupil reactions such as delayed constriction and reduced movement amplitude. Interestingly, some research indicates that these abnormalities might not be directly related to dopamine loss, as medications targeting the dopaminergic system do not appear to influence the pupil light reflex. It has been proposed that both branches of the autonomic nervous system—the parasympathetic and sympathetic—are affected in PD, with the parasympathetic system being more severely disrupted. This imbalance in parasympathetic function may emerge early in the disease process. As PD progresses, parasympathetic dysfunction tends to worsen, while changes in sympathetic activity develop more gradually. In addition to autonomic causes, impaired pupil reactions may also result from damage to the retina, optic nerve, or brainstem regions such as the locus coeruleus. Moreover, individuals with PD who also experience cognitive decline tend to show more pronounced pupil constriction deficits, a pattern similar to that observed in Alzheimer's disease. Finally, research has found that PD patients may demonstrate increased pupil responses when challenged with greater postural demands.

2.3 Glaucomatous Disturbances

Individuals show a higher tendency to develop glaucoma or visual field abnormalities resembling those seen in glaucoma, most commonly linked to open - angle glaucoma Nevertheless, current epidemiological research on this association is limited, and the strength of the available evidence remains weak.

2.4 Eyelid and Blink Reflex Abnormalities in Parkinson's Disease

Patients with Parkinson's disease often experience problems with eyelid function and blinking. These issues may include a slower or irregular blinking rate, difficulty opening the eyelids, involuntary eyelid spasms, and drooping of the upper eyelid. Such changes can be caused by damage to the nervous system, especially the loss of dopamine - producing cells. Many individuals also develop dry eyes due to reduced tear production and blinking, along with changes in the tear film

caused by Meibomian gland dysfunction. These symptoms can lead to discomfort and visual disturbances, affecting the quality of life in those with Parkinson's disease.

2.5 Visual hallucinations

Visual hallucinations are a common symptom in Parkinson's disease, often appearing as the disease progresses. Patients may see people, animals, or objects that are not actually present. These experiences can begin as mild illusions or a sense of presence and later develop into vivid, complex images. The exact cause is unclear, but it is believed to involve problems with brain networks that manage attention and visual processing, along with changes in chemicals like dopamine and serotonin. Some Parkinson's medications can increase the risk of hallucinations. These symptoms are important to monitor, as they are linked to faster cognitive decline and may signal a greater need for long - term care.

3. Visual Acuity, Contrast Sensitivity, and Colour Vision in Parkinson's Disease and Dementia

Visual disturbances are common in individuals with Parkinson's disease and often go beyond typical age - related changes. One of the most frequently reported complaints is reduced visual clarity or sharpness, known as impaired visual acuity. This problem is especially noticeable in low - contrast settings, such as reading light Gray text on a white background or seeing in dim lighting.

In addition to reduced acuity, many people with PD experience a decline in contrast sensitivity—the ability to distinguish between objects and their background when there is little difference in colour or brightness. This decline affects both the central (foveal) and peripheral areas of vision, and is especially prominent at intermediate and high spatial frequencies, which are important for detecting fine details and textures.

Colour vision is also affected in Parkinson's disease, particularly along the red - green (protan Deutan) axis. Patients may have difficulty distinguishing between certain colours, such as dark green, blue, and red, which can make visual tasks more challenging. These colour vision problems can be one of the early signs of PD and may worsen as the disease progresses.

Research suggests that these visual impairments may be linked to the loss of retinal ganglion cells and damage to the macula—the central part of the retina responsible for sharp vision. Dopamine, a key neurotransmitter that is deficient in PD, plays an important role in the retina's ability to process visual signals. A reduction in dopamine in the eye may contribute to the decline in both contrast sensitivity and colour perception.

3.1 Visuospatial impairment

Visuospatial impairment is a common non - motor symptom in Parkinson's disease, affecting the ability to understand and process visual and spatial information. Patients often struggle with tasks like judging angles, distances, or positions of objects. Tests such as the Judgment of Line Orientation (JLO) and the Visual Object and Space Perception Battery (VOSP) have shown reduced performance in individuals with PD, especially in those with cognitive decline. Some patients also have trouble with mental rotation, particularly when imagining body parts like hands, suggesting difficulty linking visual information with movement. These challenges likely result from changes in brain areas responsible for spatial awareness and visual - motor integration. Furthermore, some of these visual problems have been found to improve with dopaminergic medications such as levodopa or apomorphine, which may temporarily enhance contrast sensitivity. However, in many cases, these visual symptoms continue to progress and have been associated with a greater risk of cognitive decline and dementia in PD patients.

3.2 Visuoconstructive impairment

Visuoconstructive impairment in Parkinson's disease refers to difficulty in tasks that involve building, drawing, or copying shapes and objects. Tests like the Block Design from the WAIS, the Clock Drawing Test, and the Rey–Oster Rieth Complex Figure often reveal poor performance in PD patients. These difficulties are linked not only to visual processing issues but also to problems with planning and spatial awareness. A common behaviour seen is the "closing - in" phenomenon, where patients draw too close to the model they are copying. This may be due to problems with attention, working memory, or impaired control of motor actions, especially in those with more advanced cognitive decline.

4. Visual Processing Deficits in Parkinson's Disease: Influence of Motor Subtype and Hemispheric Onset.

The lateralization of motor symptom onset in Parkinson's disease (PD) has been increasingly recognized as a key factor influencing non - motor symptoms, particularly those related to visual and visuospatial processing. Since PD typically begins asymmetrically, with motor symptoms appearing on one side of the body, this asymmetry reflects differential involvement of the brain's hemispheres—most notably, the contralateral hemisphere to the affected side. Given the right hemisphere's dominant role in spatial awareness and visual attention, patients with left - side motor onset (LPD) may be more susceptible to visuospatial deficits. Research has demonstrated that individuals with LPD often perform worse on tasks requiring spatial navigation, attention, and orientation, and tend to rely more heavily on visual input compared to those with right - side onset (RPD). These deficits may contribute to functional impairments that go unrecognized if symptom laterality is not considered. Therefore, understanding the relationship between side - of - onset and visual function is crucial for developing more individualized assessment and intervention strategies in PD care. Emerging evidence suggests that both the side of motor symptom onset and the initial motor phenotype in Parkinson's disease (PD) significantly influence visual and visuospatial processing. Studies have found that patients with left - side onset (LPD), which corresponds to greater right - hemispheric dysfunction, exhibit impairments in perceiving global visual elements, while those with right - side onset (RPD) show deficits in processing finer, local details of objects. This

asymmetry has been linked to differential involvement of the contralateral temporoparietal junction, a region critical for spatial integration. Beyond laterality, the nature of the initial motor symptoms also appears to affect visual function. Patients with a bradykinesia/rigidity - dominant (B/R - D) phenotype demonstrate more pronounced deficits in visual domains such as acuity, depth perception, peripheral vision, and light/dark adaptation compared to both tremor - dominant (T - D) individuals and healthy controls. Furthermore, those with postural instability and gait difficulty (PIGD) exhibit worse performance on visuospatial tasks and may be at higher risk for visual hallucinations, particularly among those with rigid - akinetic features. These findings highlight the importance of considering both motor symptom laterality and phenotype when evaluating visual impairments in PD, as they may point to distinct underlying neural mechanisms and guide more personalized interventions.

5. Gender Influence in Visuospatial Symptoms in Parkinson's Disease

Research on gender differences in cognitive function among individuals with Parkinson's disease (PD) has yielded mixed findings, particularly concerning visuospatial abilities. Some studies have indicated that men with PD outperform women on spatial tasks, such as the Road Map Test of Direction Sense, which involves egocentric mental rotation. Similarly, gender - based disparities have been observed in tests of spatial navigation, motion perception, and line bisection, with males generally demonstrating superior performance. Tests like the Judgment of Line Orientation (JLO) and other visuospatial tasks have also shown that female patients may score lower than their male counterparts, especially during the early stages of the disease. One proposed explanation for this difference involves the neuroprotective role of oestrogen, which may help preserve cognitive function in females. However, not all studies support these findings. Some have reported no significant sex differences in visuospatial functioning, even among patients with or without mild cognitive impairment. Research using tasks such as mental rotation, global - local pattern perception, and visual problem - solving has failed to find consistent male–female disparities. These inconsistencies suggest that while gender may influence certain aspects of visuospatial cognition in PD, it is not a universal factor and may interact with other variables such as the side of symptom onset and disease progression.

6. Brain Network Alterations Associated with Visual and Spatial Deficits in Parkinson's Disease

Table 1: Neuroanatomical regions associated with visuoperceptual, visuospatial, and visuoconstructive deficits in Parkinson's disease

Cognitive Domain	Assessment Task	Associated Brain Regions
Visuoperceptual	Facial Recognition Test (FRT)	Fusiform Gyrus (BA 19, 36), Parahippocampal Region, Middle Occipital Gyrus, Inferior Frontal Gyrus (BA 47)
	Visual Form Discrimination Test (VFDT)	Left Lateral Occipital Cortex, Bilateral Superior Parietal Lobule (BA 7, 40), Superior Occipital Cortex (BA 19), Inferior Frontal Gyrus (BA 47)
Visuospatial	Judgment of Line Orientation (JLO)	Bilateral Superior Temporal Gyrus, Right Lateral Occipital Cortex
Visuoconstructive	Pentagon Copy Test (PCT)	Right Supplementary Motor Area, Left Rostral Middle Frontal Gyrus, Pars Triangularis, Left Cuneus

Table 2: Cortical areas associated with thinning in relation to visuospatial and perceptual deficits in Parkinson's disease and potential progression to dementia.

Cognitive Function	Task Used	Involved Brain Regions
Visuoperceptual	Facial Recognition Test (FRT)	Left lingual region, superior temporal gyrus (left), parahippocampal gyrus (both hemispheres)
	Symbol Digit Modalities Test (SDMT)	
Visuospatial	Judgment of Line Orientation (JLO)	Insular cortex (left), inferior and superior temporal gyri, fusiform area (right hemisphere)
Visuoconstructive	Pentagon Copy Test (PCT)	Entorhinal area (left), middle and inferior temporal regions, medial temporal pole, parahippocampus, fusiform and lingual gyri, lateral occipital cortex

Structural MRI studies have consistently shown that reductions in gray matter volume are associated with poorer visuospatial and visuoperceptual performance in individuals with Parkinson's disease (PD). Research by Pereira et al. found that Gray matter loss in the superior parietal and occipital regions correlated with lower visuospatial scores, while deficits in facial recognition and visual perception were linked to atrophy in the fusiform gyrus, parahippocampus, and middle occipital gyrus. These findings suggest that facial recognition difficulties are related to ventral occipitotemporal thinning, while visual form discrimination involves dorsal parietal areas. Garcia - Diaz et al. supported these distinctions, observing similar associations using the same tasks. Additionally, performance on tests like the Judgment of Line Orientation (JLO) and Pentagon Copy Test (PCT) has been linked to structural changes in temporal, frontal, and occipital regions, including the insula and supplementary motor areas. Cortical thinning appears more extensive in PD patients with cognitive impairment, particularly in the medial temporal, parietal, and occipital cortices. Variations in Gray matter loss are also influenced by the subtype of mild cognitive impairment, with amnestic patients showing greater atrophy and visuospatial deficits. Furthermore, left - side symptom onset is often associated with more severe visuospatial impairment and lower volume in the right dorsolateral prefrontal cortex. Brain asymmetry and white matter deterioration, particularly in major association tracts such as the corpus callosum

7. Functional Neuroimage Correlates of VS/VP Deficits in Parkinsons' Disease

Functional neuroimaging studies, including fMRI, PET, and SPECT, have consistently demonstrated abnormalities in both dorsal and ventral visual pathways in individuals with Parkinson's disease (PD), suggesting disruptions in typical bottom - up visual processing and the emergence of irregular top - down control. Early PET - based research by Eberling and Bohnen highlighted reduced glucose metabolism in visual association areas, primary visual cortex, and right parietal regions, even among PD patients without cognitive decline. Similarly, lower visuospatial task performance has been associated with hypometabolism or reduced perfusion in the occipital and frontal lobes, along with diminished right hemisphere activity observed via SPECT imaging. As PD advances and dementia emerges, there is a shift in metabolic patterns, notably in Brodmann area 18 (visual association cortex) and the posterior cingulate cortex. In contrast, non - demented patients primarily show reduced activity in BA 17 (primary visual cortex). The Benton Visual Retention Test, used to assess various visual and memory functions, shows strong correlation with BA 18 metabolism, with early hypometabolism in visual association and precuneus cortices potentially marking the onset of dementia. In fMRI studies, PD patients have shown decreased activation in the right insula, hippocampus, bilateral caudate, and left putamen during visuospatial tasks, accompanied by increased activation in the dorsolateral prefrontal cortex (DLPFC) and posterior parietal cortex—interpreted as compensatory top - down mechanisms. Resting - state fMRI further reveals functional connectivity deficits, particularly in right fronto - occipitoparietal circuits, which worsen with disease progression and contribute to visuospatial and visuoperceptual impairments. These disruptions become more widespread, eventually affecting bilateral prefrontal and frontoparietal networks in PD patients with mild cognitive impairment or dementia, with increased regional synchrony noted in the medial - superior occipital gyrus compared to healthy controls.

8. Neurobiological Basis of Visual Hallucinations in Parkinson's Disease

Neuroimaging studies investigating visual hallucinations (VHs) in Parkinson's disease (PD) have often been limited by small sample sizes and methodological differences, including variations in controlling for cognitive status, disease stage, and medication effects. Despite this, structural imaging has consistently identified gray matter atrophy in brain regions critical for visual perception, attention, and memory—such as the primary and association visual cortices, limbic areas, and cholinergic structures like the pedunculopontine nucleus and substantia innominata. Patients with VHs often show widespread cortical thinning, especially in occipital, parietal, temporal, frontal, and limbic lobes, with an observed asymmetry sparing the left ventral visual stream. Specific studies have noted reduced Gray matter in areas such as the left lingual gyrus, superior parietal lobules, inferior frontal and temporal lobes, thalamus, cingulate cortex, dorsolateral and rostral prefrontal cortices, and various visual processing regions. Atrophy in the hippocampus, particularly its head, has also been linked to hallucinations and the development of dementia. Additionally, degeneration in cholinergic systems, including the pedunculopontine nucleus and substantia innominata, appears to play a role. Structural deficits have also been reported in the cerebellum, notably in lobules VIII, IX/VII, and Crus I. Both the dorsal and ventral visual processing pathways are affected in hallucinating patients, with atrophy found in regions responsible for object recognition and spatial orientation, such as the cuneus, lingual and fusiform gyri, occipital and parietal cortices, and motor - related gyri. Importantly, these changes are not solely dependent on the presence of dementia, suggesting a distinct neuroanatomical basis for VHs in PD.

9. Genetic Factors of VS/VP Deficits in Parkinsons' Disease

Although genetic mutations are implicated in only a subset of Parkinson's disease (PD) cases, they offer valuable insights into the origins and characteristics of visuospatial and visuoperceptual (VS/VP) deficits. Certain genetic variants appear to influence cognitive and visual outcomes in PD. For instance, mutations in the LRRK2 and parkin genes have been associated with a reduced risk of cognitive decline and visual disturbances. In contrast, carriers of mutations in the GBA gene, which encodes a lysosomal enzyme, tend to exhibit poorer visuospatial abilities and a greater likelihood of experiencing visual hallucinations. Additionally, individuals carrying the H1 haplotype of the MAPT gene have an increased risk of developing dementia, demonstrate reduced accuracy in tasks involving complex spatial rotations, and show decreased functional activity in brain areas like the parietal cortex and caudate nuclei. These genetic findings suggest that hereditary factors may play a role in the severity and nature of VS/VP impairments in PD.

10. Conclusion

Individuals with Parkinson's disease (PD) frequently experience a variety of visual and perceptual challenges, many of which are overlooked in clinical practice and therefore remain inadequately treated. These difficulties arise from disruptions across multiple levels of the visual system—from early structures like the retina to more advanced cortical regions responsible for processing complex visual information. Impairments in basic visual functions such as contrast sensitivity and acuity can negatively influence higher - order cognitive tasks, especially since many standard cognitive assessments rely heavily on visual input. Cognitive deficits in attention and perception further compromise how visual scenes are interpreted, indicating a breakdown in top–down processing mechanisms. Structural imaging research has revealed degeneration in the posterior cortical areas and white matter, particularly in those with mild cognitive impairment, suggesting a link between cortical atrophy and worsening visual function. Functional brain imaging also shows altered activity patterns, with decreased activation in subcortical and limbic structures, and compensatory overactivity in frontal and parietal regions, especially in the right hemisphere. These changes reflect the brain's attempt to adapt to visual deficits. Additionally, asymmetries in visual processing have been noted depending on whether PD onset occurs on the left or right side of the body. Neurochemical

imbalances involving dopamine, serotonin, noradrenaline, and acetylcholine further contribute to visual dysfunction. Such deficits often progress alongside the disease and are strongly associated with visual hallucinations, a distressing symptom for many patients. These hallucinations correlate with structural changes in key visual and memory - related brain regions. Understanding these mechanisms through advanced imaging and genetic research could improve early diagnosis and guide new treatments aimed at managing visual symptoms in PD.

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