Review Article

Brain Conflux

Neuroimaging Advances in Neuropsychiatric Symptoms Associated with Parkinson's Disease

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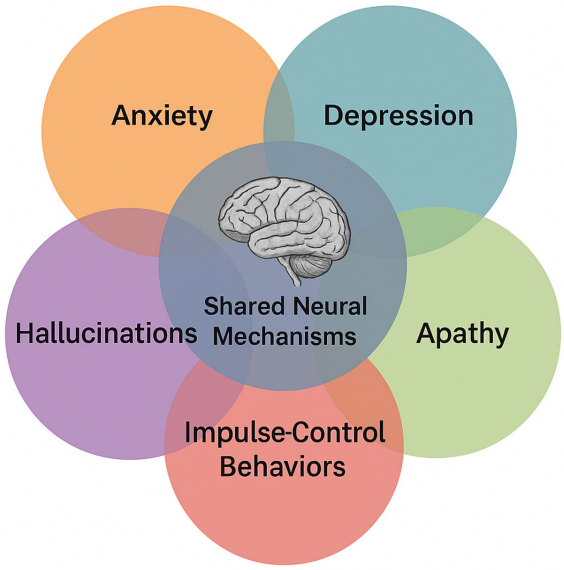
Graphical Abstract



- PD is increasingly viewed as a complex neuropsychiatric disorder.
- Neuroimaging has been used to reveal the neuropathological mechanisms underlying individual Parkinson's disease-related neuropsychiatric symptoms (PD-NPS).
- Neuroimaging studies employing multimodal techniques are pivotal in uncovering the distinct and shared neuropathological mechanisms in PD-NPS

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Neuroimaging Advances in Neuropsychiatric Symptoms Associated with Parkinson's Disease

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Abstract

Parkinson's disease (PD) is a prevalent neurodegenerative disorder traditionally defined by its motor symptoms yet increasingly recognized for its wide spectrum of neuropsychiatric symptoms (NPS) including anxiety, depression, apathy, impulse-control behaviors, and hallucinations. Recent neuroimaging advances have provided crucial insights into the neural substrates underlying these comorbidities. Structural imaging studies, using voxel-based morphometry and T1-weighted magnetic resonance imaging, have revealed regional atrophy in the frontal cortex, striatum, limbic areas, and occipital regions. In comparison, functional imaging using positron emission tomography, single-photon emission computed tomography, and resting-state functional MRI have identified abnormal network connectivity in circuits implicated in fear processing, reward regulation, and cognitive control. Overall, these imaging studies suggest shared and distinct pathophysiology of PD-related NPS, emphasizing the need for longitudinal, multimodal investigations to inform targeted therapeutic strategies and to improve clinical outcomes.

Introduction

Parkinson's disease (PD) is one of the most common neurodegenerative disorders worldwide, clinically defined by the presence of bradykinesia with at least one additional cardinal motor feature—rigidity or resting tremor—with a host of supportive and exclusionary criteria[1, 2]. Globally, PD affects approximately 0.3% of the population, with a mean onset in the early 50s; however, onset may occur as early as before age 40 and as late as in the 80s[3]. Incidence increases markedly with age, reaching about 1% in individuals over 65 and up to 3% in those older than 80[4]. With rising life expectancy, the number of PD cases and their associated socioeconomic burdens are projected to increase dramatically2. Furthermore, the incidence, prevalence, and mortality risk of PD is higher in men than in women by a ratio of approximately 1.4:1[2]. Early-onset PD—typically defined as onset before 45 years—is often associated with a genetic etiology, accounting for roughly 10% of cases, and up to 40% in those with onset before 30 years[5, 6].

While the motor features of PD remain its diagnostic cornerstone, non-motor symptoms are increasingly recognized as critical components of the disease and may even precede motor signs by several years. Non-motor signs include neuropsychiatric symptoms, disturbances in sleep–wake regulation, cognitive deficits (such as executive dysfunction, memory retrieval problems, dementia, and hallucinations), autonomic dysfunction (including orthostatic hypotension, urogenital disturbances, constipation, and hyperhidrosis), sensory deficits (most notably olfactory impairment), and pain[7]. These symptoms can significantly affect quality of life, overall disability, and need for long-term care[3].

Neuropsychiatric Symptoms in Parkinson's Disease

PD is increasingly viewed as a complex neuropsychiatric disorder. Neuropsychiatric symptoms (NPS) in PD can be broadly classified into three domains: affective (e.g., depression and anxiety), perceptual or cognitive (e.g., hallucinations), and motivational (e.g., impulse-compulsive behaviors (ICBs) and apathy)[8, 9]. Approximately 56% of early, untreated PD patients experience one or more neuropsychiatric disturbances[10]. Although patients may initially present with a single NPS, the progression of PD is frequently marked by an exacerbation and overlap of NPS, complicating both diagnosis and management[11, 12].

In patients with early-stage PD, depression and anxiety are the most common NPS. Depression often emerges in the early stages of the disease, with its prevalence increasing as age and disease duration. In advanced-stage PD patients, up to 60% exhibit comorbid depressive symptoms[13] Approximately 30-35% of PD patients may experience significant anxiety

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symptoms[14]. Mild hallucinations, particularly non-visual hallucinations, frequently occur in early PD [15]. Studies report that 25-40% of PD patients may experience mild psychosis, while around 4% develop delusions[16]. As the disease progresses, the prevalence of remission in late-stage PD can reach 60% [17, 18].Compared to other NPS, apathy has received relatively less research attention, with an average prevalence of approximately 35-40% [19, 20]. ICBs encompass impulse control disorders (pathological gambling, compulsive shopping, hypersexuality, and binge eating), dopamine dysregulation syndrome (compulsive use of anti-PD medications), and stereotyped behaviors (repetitive, non-goal-directed activities). Among patients receiving dopaminergic therapy, the prevalence of impulse control disorders is approximately 15% [21], with the incidence increasing over the course of the disease[22]; the cumulative incidence over five years reaches 46%[23]. Dopamine dysregulation syndrome and stereotyped behaviors often overlap with impulse control disorders and are predominantly observed in patients taking high doses of levodopa [8].

NPS in PD are heterogeneous, and debate persists over whether NPS in PD result from PD pathology or from nonspecific effects of psychosocial deterioration and secondary, psychological sequelae[8]. Epidemiological data reveal that PD patients exhibit distinct hallucinations and ICBs compared to general psychiatric conditions, suggesting a PD-specific pathophysiology. However, it remains uncertain if PD-related anxiety and depression differ from those in primary psychiatric disorders or in the general population[8, 24, 25]. Early-stage NPS, particularly depression and anxiety, may arise from psychosocial factors, whereas later-stage symptoms are more associated with PD pathology and dopaminergic treatment exposure[8]. Longitudinal studies have identified risk factors for NPS such as age, gender, disease severity, and dopaminergic therapy, with over 50% of PD patients developing three or more NPS within five years[26-28].

Although NPS significantly impair the quality of life in patients with PD, clinical research on their treatment lags far behind advancements in managing motor symptoms. Given the complex interplay between motor and non-motor symptoms, their high comorbidity, and the potential that treating one symptom will impact others, effective management requires clinical expertise and interdisciplinary collaboration[29, 30]. Dopamine therapy in PD patients may exacerbate psychosis[29, 30] and ICBs 31 while improving apathy[32, 33]. Non-dopamine treatment strategies primarily encompass pharmacotherapy and psychotherapy. Research has demonstrated the safety and efficacy of several antidepressants in PD patients, including selective serotonin reuptake inhibitors (SSRIs), serotonin-norepinephrine reuptake inhibitors (SNRIs), and tricyclic antidepressants, with comparable efficacy across these drug classes[34]. Pimavanserin has shown promising efficacy and tolerability in treating psychosis, with randomized controlled trials currently underway. Glutamatergic antagonist amantadine may improve apathy[35] and ICBs[31, 36]. Psychological interventions, such as cognitive behavioral therapy (CBT), have been shown to ameliorate depressive symptoms in PD patients[37, 38]. Other therapeutic approaches, including bilateral deep brain stimulation (DBS) of the medial globus pallidus or subthalamic nucleus, repetitive transcranial magnetic stimulation (rTMS), electroconvulsive therapy, light therapy, and aerobic exercise, are also generating considerable interest[39-42].

Utilizing neuroimaging to study the neuropathological mechanisms PD-NPS can help identify region-specific atrophy, dysfunctional network connectivity, and neurotransmitter abnormalities through multimodal techniques (structural MRI, fMRI, PET/SPECT). These insights uncover heterogeneous pathological substrates that clinical assessments alone cannot detect. By bridging gaps in behavioral research, neuroimaging enables targeted therapeutic development, intervention optimization, and disease trajectory prediction. Ultimately, neuroimaging can better inform precision medicine for PD-NPS. The following sections review recent imaging studies that have advanced our understanding of the neural substrates underlying anxiety, depression, apathy, ICBs and hallucinations in PD.

Neuroimaging Studies on Anxiety

Anxiety is among the most common non-motor symptoms in PD, with an estimated prevalence of 31%—higher than observed in the general population or in patients with other medical conditions[14]. Despite anxiety's clinical impact and association with reduced quality of life[43-45], its neural mechanisms in PD remain incompletely understood.

Neuroimaging has implicated two major neural circuits in anxiety pathogenesis within PD patients. The first is the fear circuit involving the amygdala, anterior cingulate cortex (ACC), medial prefrontal cortex (mPFC), insular cortex, hippocampus, and striatum[46]. The second circuit is the limbic-cortico-striatal-thalamocortical network connecting the prefrontal cortex (PFC), basal ganglia, and thalamus[47, 46]. In PD, degeneration of dopaminergic, noradrenergic, and serotonergic neurons within these circuits likely contributes to the high prevalence of anxiety[48].

Voxel-based morphometry (VBM) studies have demonstrated that higher anxiety scores—assessed using instruments such as the Beck Anxiety Inventory[49]—are associated with reduced gray matter volumes in the bilateral ACC, left amygdala, precuneus, and cerebellar structures[50-52]. Structural covariance analyses indicate that anxiety severity inversely correlates with the connectivity integrity between the striatum and prefrontal regions[53]. Functional MRI (fMRI) investigations have revealed aberrant low-frequency oscillatory activity in the right cerebellum and orbital frontal cortex (OFC) among PD patients with anxiety[54]. In addition, enhanced functional connectivity between the amygdala and OFC, as well as between parietal and medial temporal cortices, has been documented, suggesting that anomalous network interactions and anxiety symptoms are linked[55-57]. Neurotransmitter imaging with positron emission tomography (PET) and single-photon emission computed tomography (SPECT) further support these findings; decreased binding of dopamine transporters (DAT) in the caudate, thalamus, insula, ACC, and PCC is associated with increased anxiety[58-61].

Neuroimaging Studies on Depression

Depression in PD has been extensively studied using metabolic imaging techniques such as PET and SPECT. Most investigations report hypometabolism in the frontal and striatal regions in depressed PD patients, as compared to both non-depressed PD cohorts and healthy controls[62].

Abnormal metabolic patterns have been identified in the prefrontal cortex, thalamus, amygdala, hippocampus, ACC, insula, and raphe nuclei[62, 61]. In contrast, a few studies have observed hypermetabolism in subcortical structures (e.g., the caudate, putamen, and amygdala) with a positive correlation to depressive symptom severity[63, 64]. Dysregulated activity in the amygdala has also been observed in depressed PD patients[63, 65]. Moreover, both FDG-PET and $H_2{}^{15}O$ PET studies consistently report reduced metabolism in the OFC of these patients[66, 67].

Structural MRI studies using T1-weighted imaging have consistently demonstrated atrophy in the PFC, parietal, thalamus and limbic regions (notably the ACC, hippocampus and amygdala) in PD patients with depression[62, 68, 69]. Several studies emphasize marked atrophy in the OFC and ACC—regions pivotal for cognitive control and emotional regulation[70-73]. Diffusion tensor imaging (DTI) studies reveal decreased white matter integrity in tracts connecting the ACC, thalamus, and frontotemporal regions[74-76]. In addition, resting-state fMRI studies have shown that depressed PD patients exhibit both increased and decreased intrinsic activity across various networks, reflecting disrupted connectivity within cortico-limbic circuits[65, 77, 78]. Collectively, these imaging findings suggest that depression in PD arises from the dysregulation of frontal–limbic circuits and is compounded by abnormalities in dopaminergic, serotonergic, and noradrenergic neurotransmission[62].

Neuroimaging Studies on Apathy

Apathy is a common non-motor symptom in PD, defined as a lack of motivation that is not attributable to diminished consciousness, cognitive impairment, or emotional distress. Structural MRI studies indicate that apathy in PD is associated with cortical thinning and atrophy in the temporal lobe, frontal cortex, parietal regions, ACC and hippocampus; key subcortical structures such as the putamen, caudate nucleus and accumbens are also affected[79-82]. Resting-state fMRI studies revealing reduced functional connectivity between the striatum and frontal regions have implicated disruptions in cortico–subcortical limbic networks in apathy pathophysiology[83, 84]. Metabolic imaging studies generally report negative correlations between apathy and regional metabolism in the striatum, cerebellum, and various cortical areas; however, in some instances, increased metabolism in the OFC and ACC may represent compensatory responses[85, 86]. Further, PET studies have demonstrated that severe dopaminergic denervation—especially within the mesolimbic pathway—is associated with greater apathy, especially following deep brain stimulation[87, 88]. While serotonergic and cholinergic dysfunctions have also been implicated[89], the current evidence supports a multifactorial etiology of apathy in PD.

Neuroimaging Studies on Impulse-Control Behaviors

ICDs in PD, which include pathological gambling, hypersexuality, compulsive eating, and compulsive shopping, are frequently associated with long-term dopaminergic therapy[90]. The core clinical features of ICDs are repetitive, compulsive behaviors that are initially rewarding but progressively difficult to control[23]. Recent imaging studies suggest that ICDs may result from a combination of dopaminergic treatment and underlying vulnerability in reward-processing circuits.

Structural MRI findings in PD patients with ICDs are mixed. Some studies report cortical thinning in the frontal cortex and reduced volumes in the nucleus accumbens and OFC[91, 92]; other studies find increased cortical thickness in frontal and cingulate regions, potentially reflecting neuroplastic changes related to excessive dopaminergic stimulation[93, 94]. Further, functional MRI studies reveal an association between ICDs and disrupted connectivity within dopaminergic networks, notably between the striatum, prefrontal cortex, and limbic areas[95-97]. Task-based fMRI studies employing reward-based paradigms have consistently shown altered activation in the OFC and caudate nucleus; this evidence supports the hypothesis that impaired reward processing contributes to ICD development[95, 98, 99]. Importantly, imaging studies in drug-naïve PD patients suggest that pre-existing network abnormalities in mesolimbic and executive circuits may predispose individuals to ICDs upon initiation of dopaminergic therapy[100, 101].

Neuroimaging Studies on Hallucinations

Hallucinations in PD typically begin with minor phenomena—such as brief passage hallucinations, illusions, or the sense of a presence. However, they may evolve to be more visually complex, particularly in advanced stages of the disease. Patients often retain insight initially; however, as the disease progresses, insight diminishes while delusional thinking and multimodal hallucinations emerge [15, 102].

In PD patients with visual hallucinations, structural imaging studies consistently report atrophy in occipital regions—most notably within the lateral and ventral occipitotemporal cortices[103, 104]. Some investigations controlling for cognitive status also document atrophy in frontal, hippocampal, and thalamic regions[105, 106]. Although white matter studies are relatively sparse, recent evidence points to reductions in occipital and parahippocampal white matter volume in patients with psychosis[107-109]. Functional imaging studies during hallucinatory episodes are rare. Available data indicate that reduced occipital region activity, combined with aberrant frontal activation, may underlie the emergence of visual hallucinations[110, 111]. Furthermore, altered connectivity among occipital, frontal, striatal, and thalamic regions—and increased co-activation of default mode network nodes—appears to contribute to psychosis vulnerability in PD[112].

Neuroimaging Studies of Multiple Neuropsychiatric Symptoms

It is not uncommon for PD patients to experience a combination of anxiety, depression, and apathy—a constellation often described as the "non-motor triad" of PD[113]. Neuroimaging studies have revealed that the severity of these overlapping symptoms is linked to the degree of dopaminergic and noradrenergic denervation in key limbic regions such as the ACC, thalamus, amygdala, and ventral striatum[114]. Although several neurotransmitter imaging studies report overlapping mechanisms, others delineate distinct neural signatures for individual symptoms. For example, while anxiety and depression both correlate with diminished dopaminergic activity in the ventral striatum,

specific patterns of connectivity—such as differential involvement of the caudate nucleus versus other striatal subregions—may distinguish these conditions[55, 57, 115]. Moreover, the frequent co-occurrence of depression and ICDs in PD suggests that shared disturbances in ventral striatal dopaminergic transmission, and associated cortico-striatal-limbic circuits, may underlie both disorders[116]. These overlapping yet distinct imaging findings underscore the complexity of neuropsychiatric manifestations in PD and highlight the need for integrative, multimodal imaging approaches.

A recent study reported that five neuropsychiatric symptoms in PD exhibit coordinated effects on brain morphometric profiles. Apathy, ICBs, and hallucinations share structural abnormalities in the somatomotor and visual areas. In contrast, the cortical abnormalities associated with anxiety and depression were most prominent in the prefrontal cortex and default mode network regions. These findings indicate that distinct mechanisms underlie different NPS: apathy, ICBs, and hallucinations may be directly linked to the primary neuropathology of PD within motor circuits, whereas anxiety and depression—whose neurobiological bases are less clearly defined—may arise from a combination of PD-related pathology (e.g., a hypodopaminergic state) and psychological factors[117].

Conclusions

Neuroimaging studies employing multimodal techniques are pivotal in uncovering the distinct and shared neuropathological mechanisms of Parkinson's disease-related neuropsychiatric symptoms. These studies collectively advance therapeutic precision and mechanistic understanding through revealing region-specific atrophy, disrupted functional connectivity, and neurotransmitter dysregulation. Despite these advances, current research is often constrained by small sample sizes, cross-sectional designs, and a focus on individual symptoms rather than a broader spectrum of neuropsychiatric manifestations. Future research should prioritize larger-scale, longitudinal, and multimodal imaging studies to further elucidate the complex neural networks involved. A more comprehensive understanding of these mechanisms is essential to advance targeted therapies, enhance clinical management, and ultimately improve patient outcomes in PD.

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Competing Interests

The authors declare that they have no existing or potential commercial or financial relationships that could create a conflict of interest at the time of conducting this study.

Data Availability

All data needed to evaluate the conclusions in the paper are present in the paper or the Supplementary Materials. Additional data related to this paper may be requested from the authors.

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