Minor hallucinations in Parkinson disease
A subtle symptom with major clinical implications

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Abstract

Objective
Psychosis is one of the most debilitating complications of Parkinson disease (PD). Although research on PD psychosis has been focused on the study of well-structured visual hallucinations (VH), currently accepted National Institute of Neurological Disorders and Stroke–National Institute of Mental Health diagnostic criteria emphasize minor hallucinations (MH) as the most common psychotic phenomena in PD. The objective of this review is to comprehensively describe the clinical and research advances on the understanding of MH and to provide future directions for obtaining further insights into their potential major implications for PD management and prognosis.

Methods
A PubMed search was done in November 2018 to identify articles on minor psychotic phenomena in PD.

Results
MH often precede the onset of well-structured VH and are associated with other nonmotor symptoms such as REM sleep behavior disorder and depression. The pattern of functional brain connectivity changes associated with MH involve visual-processing areas and attention control networks, which overlap with abnormalities described in patients with well-structured VH. The dysfunction of cortical networks in patients with MH may be an early indicator of a more widespread form of the disease.

Conclusion
Although called "minor," MH may have major clinical and prognostic implications. Further research is needed to establish whether MH are associated with a higher risk of disabling psychotic complications, cognitive deterioration, or a more accelerated disease progression. Understanding the early neurobiological underpinnings of MH may provide the background for future studies to identify the progressive dysfunction of neural circuits leading to more severe forms of psychosis in PD.

Glossary

MH = minor hallucinations; **PD** = Parkinson disease; **PPMI** = Parkinson's Progression Marker Initiative; **RBD** = REM sleep behavior disorder; **UPDRS** = Unified Parkinson's Disease Rating Scale; **VH** = visual hallucinations.

Patients with Parkinson disease (PD) may develop several nonmotor symptoms during their disease course.[1] PD-psychosis is one of the most debilitating nonmotor symptoms and is associated with an overall higher nonmotor symptoms burden,[2] poor quality of life,[2] and higher mortality rate.[3] The spectrum of PD-psychosis symptoms is wide and includes minor hallucinations (MH), well-structured visual hallucinations (VH), delusions, and hallucinations in other sensory modalities (i.e., auditory, olfactory, gustatory, and tactile hallucinations).[4,5] The reported prevalence of PD psychosis ranges from 16% to 74%.[6] The wide variance in the prevalence reported is predominantly due to differences in the methodology used in the studies, especially the use of different scales and diagnostic criteria, the screening of minor psychotic phenomena, and the differences in clinical and demographic characteristics of the samples studied. In a large retrospective study of 445 patients with pathologically confirmed diagnoses of PD, half of the patients had a history of VH, indicating a lifetime prevalence of 50%.[7] Several longitudinal studies have reported that the point prevalence of psychosis in PD increases over time,[8,9] and may reach as high as 74% in a 20-year follow-up period.[10] As these longitudinal studies collected data largely on well-structured VH, the prevalence reported in these studies would appear underestimated if the full spectrum of psychosis were considered.

Of all the aforementioned symptoms, MH and VH are the most commonly observed manifestations of PD-psychosis.[11,12] Although research on PD-psychosis has been mostly focused on the study of well-structured VH, when the currently accepted diagnostic criteria for PD-psychosis proposed by the National Institute of Neurological Disorders and Stroke and National Institute of Mental Health are applied, MH appear as the most common manifestation of PD-psychosis.[13] The seminal studies by Fénelon et al.[4,5,14] showed MH to be the most frequent and early type of psychotic phenomena in PD.

MH encompass different types of hallucinatory experiences: (1) presence hallucinations (or feeling of presence), which refer to the vivid sensation that somebody (distinct from oneself) is present nearby, usually behind the patient's shoulder, in the absence of sensory clues revealing a presence; (2) passage hallucinations, which consist of a fleeting image or brief vision of a person, animal, or object passing sideways, within the periphery of the visual field; and (3) visual illusions, which are brief misperceptions of objects or living beings that differ from objective reality (e.g., mistaking a standing lamp in the room corner for a man).

MH have also a focus of interest as their development often precedes the onset of well-structured VH and can be observed from the earliest stages of the disease, even in drug-naive patients with PD.[15] It is not clear yet which clinico-demographic variables and other nonmotor symptoms are associated with the development of MH or whether their presence is a risk factor for the emergence of more severe psychotic symptoms, cognitive deterioration, or a more accelerated disease progression. In this article, we comprehensively review the phenomenology and clinico-demographic correlates of MH in PD, their relationship with well-structured VH, and the neuroimaging findings of patients with isolated MH. The clinical characterization and definition of the neurobiological underpinnings of MH may provide the background for future studies to identify the progressive dysfunction of neural circuits leading to more severe forms of psychosis in PD.

Methodology

In April 2019, the authors searched the pertinent literature in PubMed, using "Parkinson's disease" and a number of keywords ("minor hallucinations," "extracampine hallucinations," "passage hallucinations," "presence hallucinations," "illusions," and "misperceptions"). This step yielded 111 articles (table). During the initial screening of the abstracts/full texts, publications that were not relevant to this review, duplicates, and those that were published in languages other than English were excluded, leaving 40 articles for inclusion. The references from these articles and those excluded were reviewed, with an additional yield of 27 articles. In total, 67 articles pertaining to this topic were included in this review.

Data availability statement

The details of the literature search in PubMed are available in the table.

Epidemiology and phenomenology of MH in PD

Owing to the detailed description of MH published by Fénelon et al.[4] in 2000, the phenomenology and clinical correlates of MH have been mainly studied in patients with PD. The authors grouped together, under the term of "minor hallucinations/illusions," 3 types of phenomena. The sensation of a presence, a sideways passage, or illusions were present in 25.5% of a sample of patients with moderate to advanced PD. MH were present either in isolation or combined with well-structured VH, and one of the patients with vivid and recurrent presence hallucinations was receiving no treatment. Presence hallucinations were present in 64% of

Table Results of search for articles from PubMed using various key words and their combinations

Keywords and combinations	No. of publications		
	Total	Included	Excluded
"Parkinson's disease" and "Minor hallucinations"	19	12	8 (not in English: 1, not relevant: 7)
"Parkinson's disease" and "Extracampine hallucinations"	2	1	1 (not in English: 0, not relevant: 1)
"Parkinson's disease" and "Passage hallucinations"	11	6	5 (not in English: 1, not relevant: 4)
"Parkinson's disease" and "Presence hallucinations"	5	4	1 (not in English: 0, not relevant: 1)
"Parkinson's disease" and "Illusions"	58	15	43 (not in English: 8, not relevant: 35)
"Parkinson's disease" and "Misperceptions"	16	2	14 (not in English: 0, not relevant: 14)

patients with MH, passage hallucinations in 33%, and illusions in 16%. Previously, in a study analyzing the risk factors of hallucinations in PD, Sánchez-Ramos et al. had already succinctly stated that "many patients felt a sense of presence in the room."[16] As described in the studies by Fénelon et al., presence hallucinations refer to the vivid sensation that somebody (distinct from oneself), and more often a person than an animal, is present nearby, usually behind, beside, or next to the patient (always in close proximity), in the absence of any apparent and obvious sensory stimulus.[5] Patients usually feel a human presence, which is unidentified in most cases, but sometimes the presence is identified as a close relative. Presence hallucinations commonly occur while patients are alone, while sitting on a sofa, reading, cooking, or watching television.

Different from presence hallucinations, patients may also describe the sensation that someone uninvited is living in their home, but the feeling of that presence is not present nearby. This phenomenon is called phantom boarder syndrome, and it has been more frequently reported in patients with dementia than in PD itself.[17]

Passage hallucinations are described as the brief appearance of a person, an indefinite shadow, or an animal passing through the periphery of the visual field, moving forward from behind and close to patients' shoulders. Patients usually have an irrepressible urge to look towards the illusory moving perception, turning their head behind them. Passage hallucinations are most commonly described as the fleeting and poorly defined vision of a shadow passing sideways, or as a walking person or running animals (cats, rats, dogs), or as undefined moving objects.[15]

Visual illusions are the transient misperceptions of objects or living beings that differ from objective reality (e.g., a man is perceived in place of a standing lamp in the corner of a dimmed room). Visual illusions include the presence of pareidolias, the illusory perception of faces or animals from irrelevant visual stimuli, as might be seen with a face or an animal emerging out of a complex upholstering pattern in a couch.[18] A recent study highlighted that kineptosia (stationary objects perceived as

being in motion) and object misidentification illusions (objects misperceived as different objects) were the most common types of visual illusions in patients with PD.[19]

These minor phenomena are usually short-lasting (lasting a few seconds), occur more than once a week, are not distressing, and insight is completely preserved. Presence and passage hallucinations are more likely to occur indoors, while visual illusions are experienced also outdoors. In some cases, however, MH last longer (minutes to hours) and can be unpleasant, but rarely distressing. Patients usually experience isolated MH, but in all published series MH can coexist with well-structured hallucinations in other sensory modalities.

The actual prevalence of MH in PD remains unknown. In a prospective study of 50 drug-naive patients with PD, MH were screened using a structured interview covering all types of psychotic phenomena.[15] In this study, 21 of 50 (42%) patients with de novo untreated PD experienced minor hallucinatory phenomena. Both passage and presence hallucinations were documented in 57.1% of the patients with MH, and isolated passage hallucinations and isolated presence hallucinations were present in 28.6% and 14.3% of the patients, respectively. In addition, 7 patients reported visual illusions (e.g., kinetopsia of doorknobs and curtains, images emerging from patterned sofa and wallpaper). Interestingly, in one-third of patients with de novo MH, hallucinations manifested as a premotor symptom, starting before the onset of first parkinsonian motor symptoms. The early occurrence of MH in patients without dopaminergic medications indicates that hallucinations are indeed part of PD itself. It is not clear what is the effect of dopaminergic treatments on early MH. In this series, the use of levodopa and dopamine agonists was not associated with an impairment or improvement of MH. In a cross-sectional study of 414 patients with PD in the middle stages of the disease, screening VH by using a questionnaire circulated via an online patient community, presence or passage hallucinations were found in 50.3% of the cohort.[20] In particular, 45.9% reported a "feeling of movement" passing them, and 24.6% reported "feeling or imagining a presence" that was not truly there. Again, dopaminergic drugs did not appear to have any specific effect upon MH.[20]

In the only study that has compared the presence of MH in patients with PD and healthy controls,[15] healthy controls presented significantly fewer MH than patients with PD (5% vs 42%; $p < 0.0001$), indicating that mild psychotic symptoms are more frequent in PD than in the general population. The hallucinations found in healthy participants were very similar to those described by patients with PD.

The 5 healthy participants reported presence hallucinations very occasionally (1–3 times per year), and one of them also experienced passage hallucinations during the last year. Bereavement represents a stressful life event that has been associated with MH. One-third to half of healthy people who have lost their spouse report the presence of the deceased, or even seeing, hearing, and talking to the spouse.[21] MH are also experienced in other brain diseases such as epilepsy,[22] traumatic brain injury,[23] and in neurodegenerative disorders including dementia with Lewy bodies[24] and Alzheimer disease.[25] However, the prevalence in these conditions is largely unknown and further studies are needed to establish the phenomenology and frequency of MH in these conditions.

MH depict a larger problem in evolution

Associations of MH with VH

Several studies on the natural course of MH and VH have suggested a close association between both phenomena. In the seminal article by Fénelon et al.,[4] well-structured VH were present in association with MH in 11% of patients. Later, using the National Institute of Neurological Disorders and Stroke–National Institute of Mental Health criteria for PD psychosis, MH were found in 45% of 116 consecutive patients with PD, with 27% of the patients presenting both MH and VH.[14] The association of MH with VH was further established by another study by Fénelon et al.[5] that revealed the concomitant presence of VH in one-third of patients with PD with presence hallucinations. In that study, presence hallucinations were one of the predictive factors (odds ratio 4.5) for well-structured hallucinations. In a longitudinal community-based PD cohort of 250 patients, Mack et al.[16] reported MH in 20.4% patients with PD, and of those with MH, nearly 40% had concomitant structured VH. In another cross-sectional study by Lenka et al.,[26] isolated MH occurred in nearly 60% of patients with PD with hallucinations, and combined VH and MH was found in 14% of the sample. Interestingly, the onset of MH preceded that of VH in all patients with combined minor and well-structured hallucinations.[26] In the cross-sectional study by Wood et al.,[20] the questionnaire administered investigated the relationship between MH and VH. Although the information was collected retrospectively, MH onset was described as occurring around the same time as VH in 52.8% of reported cases, MH developed before VH in 25%, and after VH in 22.2%. Also, patients with MH were significantly more likely to report VH than those without them (26.6% vs 4.4%; $p < 0.001$). In the follow-up study of patients with drug-naive MH

by Pagonabarraga et al.,[15] 28.5% of the patients with MH developed well-formed VH after 4.4 years, suggesting that MH are perhaps the forme fruste of well-structured VH.

Although there are now abundant data suggesting that MH and VH are closely associated, showing that the onset of MH often precedes that of VH, no strong data are available yet to establish the natural course of MH, or whether the presence of MH is an independent risk factor for the development of well-structured or disabling hallucinations. In one study examining longitudinal evolution of well-structured VH, Goetz et al.[27] followed 48 patients with PD with benign visual hallucinations (with insight retained; Unified Parkinson's Disease Rating Scale [UPDRS] thought disorder score = 2) for at least 3 years, or until the UPDRS thought disorder score became 3 (loss of insight) or 4 (delusion). The study documented that 80% of the patients transitioned from benign hallucinations to more severe hallucinations during the follow-up. Considering this "malignant" outcome, the authors argued against the use of the term "benign hallucination" in the context of PD. Although this study demonstrated clearly that VH are progressive over time, the authors did not focus on patients with MH at study enrollment, so further longitudinal studies enrolling participants without hallucinations or with isolated MH at baseline are needed to establish the temporal relationship between the different psychotic phenomena over the natural course of PD, as well as their precise relationship with dopaminergic drugs.

It is well-established that VH have significant negative effects on health-related outcomes including quality of life that is independent of patients' motor symptoms.[12,16,28] In addition, VH in PD have been reported to be an independent risk factor for higher rates of nursing home placement,[29] caregiver distress,[30] and mortality.[3] Considering these negative outcomes related to VH in PD, disentangling the relationship between MH and VH conveys important clinical as well as research implications. Regarding treatment, one longitudinal study by Goetz et al.[31] revealed a positive effect of antipsychotic medications in patients with PD with hallucinations and retained insight. In this study, early treatment delayed the deterioration to more severe forms of hallucinations (39 vs 12 months; hazard ratio 0.156, $p < 0.0001$). Future prospective studies including patients with MH and hallucinations with insight retained would help to clarify the long-term effect of early treatments in patients with PD with milder forms of hallucinations.

Association of MH with other nonmotor symptoms

A number of clinical-epidemiologic studies have reported significant associations of MH with other nonmotor symptoms of PD. Fénelon et al.[4] reported a higher prevalence of depression in patients with MH, and Mack et al.[16] found patients with MH to have greater depressive symptoms and poorer quality of life. A recent study exploring the risk factors for early psychosis in the Parkinson's Progression Marker Initiative (PPMI) cohort revealed higher depression scores in patients with illusions or hallucinations.[32]

Interestingly, depression scores in the PPMI dataset were high even before the onset of hallucinations.[32] However, other longitudinal studies more focused on early VH have not seen such a clear relationship with depression, anxiety, or apathy.[33,34] As the use of antidepressants has been observed to improve hallucinations in PD in some clinical series,[35,36] further research on the relationship between minor psychotic phenomena and mood disorders may have important therapeutic implications.

The clinical observation of a relationship between altered dream phenomena, intrusion of REM sleep-related imagery into wakefulness, and REM sleep behavior disorder (RBD) with VH led to studies that showed a complex relationship between sleep disturbances and psychosis in PD.[37,38] RBD has been found to predict the development of hallucinations or delusions, and the severity of RBD symptoms has been associated with psychosis in patients with early PD without cognitive impairment.[39–41] Owing to all these studies, there is now robust evidence on the association between RBD and hallucinations in PD.[42] More recently, a significant association also has been found between RBD and MH.[15] Lenka et al.[26] identified RBD as one of the risk factors for early-onset psychosis and in a recently published study, Barrett et al.[43] revealed that RBD is an independent predictor of both MH and VH in patients with PD without dementia. Although there is burgeoning evidence to suggest close association between RBD and MH, the neurobiological underpinnings of this clinical relationship are yet to be fully understood.

Finally, although the relationship between cognitive impairment and well-structured hallucinations is clearly established in the literature,[11,44] no significant differences in global cognitive function or in any cognitive domain have been observed repeatedly in studies including patients with MH without dementia.[4,15,43,45,46] Morgante et al.,[47] however, observed in a longitudinal cohort of 495 patients that psychosis developed in the early stages of the disease predicted cognitive deterioration after 2 years of follow-up. These data indicate that the association of cognitive impairment with hallucinations remains controversial in the early stages of PD, and suggest that neural systems other than those involved in cognitive performance contribute to the genesis of milder hallucinations.[48] Considering the significant association of MH with RBD and formed VH, which are considered as risk factors for cognitive impairment, more studies are needed to establish the prognostic implications of MH in PD.[49] MH in PD could be understood as the tip of an iceberg beneath which reside debilitating nonmotor symptoms and potential progression to more widespread forms of the disease (figure).

Neuroimaging abnormalities in patients with MH

Several structural and functional neuroimaging studies have explored the neural correlates of VH in PD[50]; however, only a few have focused on MH. In patients with VH, structural imaging studies have revealed gray matter atrophy in brain regions corresponding to dorsal and ventral visual pathways,[51] cholinergic brain structures,[52,53] and the hippocampus.[54–56] Results of functional neuroimaging studies have suggested the presence of defective top-down control of visual processing and bottom-up dysfunction of visual perception systems.[50,57] In the first study to compare gray matter volume changes of patients with PD with isolated MH, Pagonabarraga et al.[48] revealed atrophy of brain regions corresponding to the dorsal visual stream (superior parietal lobe, precuneus), indicating the putative role of structures within posterior cortical areas in the genesis of hallucinations. The same group analyzed the structural and functional MRI changes in a new cohort of patients with early PD with isolated MH.[58] Compared to patients without MH, the MH group showed greater gray matter atrophy in secondary visual-processing areas (left middle occipital gyrus), areas within the dorsal visual stream (right precuneus) and ventral visual stream (right fusiform and right parahippocampal gyri), and in multimodal sensory processing areas (left supramarginal and left angular gyri). In addition, patients with PD–MH showed decreased gray matter volume in core regions of the default mode network (posterior cingulate cortex). This pattern of atrophy partially overlaps with that reported in patients with PD with well-structured VH,[50] suggesting that both phenomena perhaps share similar pathophysiologic processes. In addition, patients with MH showed significant alterations in functional connectivity within the default mode network. Greater functional connectivity was found between the posterior cingulate cortex with posterior regions of the task-positive network (bilateral superior parietal lobes, right precentral gyrus, and left middle cingulate cortex) and visual-processing areas specially involved in motion processing (bilateral posterior middle temporal gyrus including the V5/MT+ region). In parallel, reduced functional connectivity was found between the posterior cingulate cortex with prefrontal attentional areas and anterior temporal areas involved in high-level visual processing. Taken together, all these changes are in agreement with the attentional networks hypothesis proposed for well-structured VH,[59] which proposes that hallucinations in PD are related to deficient activation of the dorsal attentional networks when interpreting ambiguous percepts and shows that MH are associated with early disruption of bottom-up visual perceptual systems.

In a recently published study, Nishio et al.[60] investigated the commonalities and differences at the neural level between patients with PD with MH and those with well-structured VH. By using factor analysis, the authors first identified the phenomena of presence and passage hallucinations, and visual illusions as a single behavioral factor, indicating the validity of the concept "minor hallucinations/illusion" as an independent clinical entity. Then, to investigate the brain–behavior relationship for psychotic symptoms, correlation analyses were done with MRI morphometry and ^{18}F-fluorodeoxyglucose PET. The presence of MH significantly correlated posterior cortical atrophy and hypometabolism in the posterior cingulate

Figure Schematic representation of the iceberg phenomenon in patients with Parkinson disease and minor hallucinations
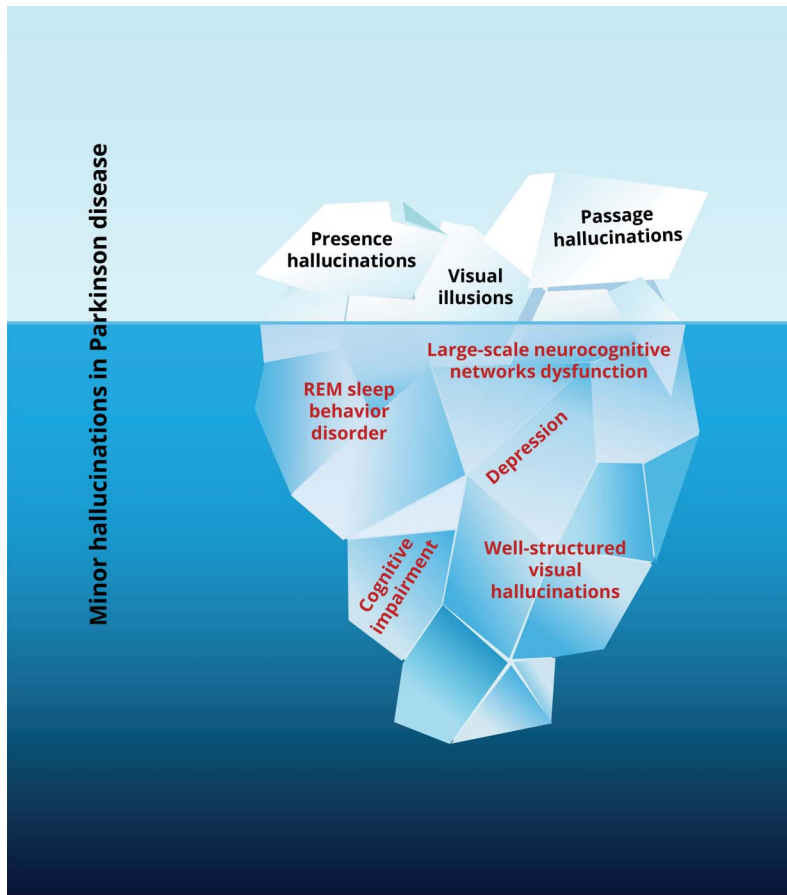
and the medial occipito-parietal cortices, further endorsing the notion that MH has distinctive neural substrates in patients with PD. All these studies indicate that patients with isolated MH share distinctive structural and functional brain abnormalities with patients with more structured hallucinations.

Finally, there is a need also to investigate the neurotransmitter abnormalities that may play a role in the functionality of these complex networks. Dysfunction of attentional and visuoperceptive areas has been associated with cholinergic defects in patients with early PD.[61] Also, the GABAergic system plays an important role in the visual cortex. By using magnetic resonance spectroscopy, GABAergic activity was measured in the occipital lobe of patients with PD with and without complex visual hallucinations, and in well-matched healthy participants.[62] In this study, the presence of VH was associated with reduced levels of GABA in the occipital cortex, together with structural changes in the ventral visual stream. The loss of GABA inhibition within the occipital cortex has additional translational implications, and remediation of GABAergic function may open new therapeutic avenues.[62,63]

The preliminary neuroimaging studies on isolated MH in PD help to understand the neurobiological underpinnings of very early psychosis in PD, and provide the background for future studies to identify the progressive dysfunction of neural circuits leading to more severe forms of psychosis in PD.

Future perspectives

Although previously described as "minor," current evidence suggests that MH are in fact closely associated, clinically and neurobiologically, with more complex forms of psychosis in PD. Future research is needed to describe better the natural course, effect of dopaminergic drugs, and relationships of MH with depression, sleep disorders, and cognitive impairment. If future longitudinal studies would establish MH as a risk factor for the disabling psychotic complications, cognitive deterioration, or a more accelerated disease progression, the early screening of MH could be important for selecting early candidate patients for clinical trials of PD-psychosis or disease-modifying therapies. An important issue that remains puzzling is the association of dopaminergic neurotransmission with psychosis in PD. Dopaminergic deficiency at the level of the striatum, prefrontal cortex, and posterior visuospatial areas has a direct effect on the attentional and visuoperceptive systems necessary to accurately and rapidly process the continuous incoming environmental visual information.[64] Dopaminergic deficiency has been seen to impair retinal

processing and to cause delayed visual evoked potentials.[65,66] Conversely, dopaminergic overstimulation of the limbic system, or even dopaminergic inputs to dysfunctional amygdala, is a well-known factor related to psychosis in PD. Hence, longitudinal studies taking into account all these variables are warranted to determine at what extent dopaminergic replacement can be useful or deleterious in the evolution of MH in PD.

There is burgeoning evidence to suggest that MH have major clinical and prognostic implications in PD. In spite of the nomenclature of MH, we hope that a general consensus emerges that they are "not so minor." Is it time for a new term, perhaps closer to the one utilized in the MDS-UPDRS item 1.2 score 1? Although the term "minor hallucinations" has been useful for unifying this clinical construct, new terms such as "nonformed" or "unformed hallucinations" could be adapted in the future in order not to consider these symptoms as mild neuropsychiatric complications. Further analysis of MH, and their neuropsychological, behavioral, and imaging characteristics, would not only enable early identification of patients with PD with a more widespread disease but would also help to establish their clinical implications and additional neurobiological underpinnings.

Acknowledgment

Study funding

Disclosure

Publication history

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Appendix (continued)

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References

1. Schapira AHV, Chaudhuri KR, Jenner P. Non-motor features of Parkinson disease. Nat Rev Neurosci 2017;18:435–450.
2. Kulick CV, Montgomery KM, Nirenberg MJ. Comprehensive identification of delusions and olfactory, tactile, gustatory, and minor hallucinations in Parkinson's disease psychosis. Parkinsonism Relat Disord 2018;54:40–45.
3. Forsaa EB, Larsen JP, Wentzel-Larsen T, Alves G. What predicts mortality in Parkinson disease? A prospective population-based long-term study. Neurology 2010;75:1270–1276.
4. Fénelon G, Mahieux F, Huon R, Ziégler M. Hallucinations in Parkinson's disease: prevalence, phenomenology and risk factors. Brain 2000;123(pt 4):733–745.
5. Fénelon G, Soulas T, Cleret de Langavant L, Trinkler I, Bachoud-Lévi AC. Feeling of presence in Parkinson's disease. J Neurol Neurosurg Psychiatry 2011;82:1219–1224.
6. Chang A, Fox SH. Psychosis in Parkinson's disease: epidemiology, pathophysiology, and management. Drugs 2016;76:1093–1118.
7. Williams DR, Lees AJ. Visual hallucinations in the diagnosis of idiopathic Parkinson's disease: a retrospective autopsy study. Lancet Neurol 2005;4:605–610.
8. Goetz CG, Wuu J, Curgian LM, Leurgans S. Hallucinations and sleep disorders in PD: six-year prospective longitudinal study. Neurology 2005;64:81–86.
9. Zhu K, van Hilten JJ, Putter H, Marinus J. Risk factors for hallucinations in Parkinson's disease: results from a large prospective cohort study. Mov Disord 2013;28:755–762.
10. Hely MA, Reid WG, Adena MA, Halliday GM, Morris JG. The Sydney multicenter study of Parkinson's disease: the inevitability of dementia at 20 years. Mov Disord 2008;23:837–844.
11. Ffytche DH, Creese B, Politis M, Chaudhuri D, Ballard C, Aarsland D The psychosis spectrum in Parkinson disease. Nat Rev Neurol 2017;13:81–95.
12. Clegg BJ, Duncan GW, Khoo TK, et al. Categorising visual Hallucinations in early Parkinson's disease. J Parkinsons Dis 2018;8:447–453.
13. Ravina B, Marder K, Fernandez HH, et al. Diagnostic criteria for psychosis in Parkinson's disease: report of an NINDS, NIMH work group. Mov Disord 2007;22:1061–1068.
14. Fénelon G, Soulas T, Zenasni F, De Langavant LC. The changing face of Parkinson's disease-associated psychosis: a cross-sectional study based on the new NINDS-NIMH criteria. Mov Disord 2010;25:763–766.
15. Pagonabarraga J, Martinez-Horta S, Fernández de Bobadilla R, et al. Minor hallucinations occur in drug-naive Parkinson's disease patients, even from the premotor phase. Mov Disord 2016;31:45–52.
16. Mack J, Rabins P, Anderson K, et al. Prevalence of psychotic symptoms in a community-based Parkinson disease sample. Am J Geriatr Psychiatry 2012;135(pt 8):2458–2469.
17. Kasahara H, Tsumura M, Kada H, et al. Perspectives on phantom boarder symptom. Psychogeriatrics 2005;5:103–107.
18. Diederich NJ, Fénelon G, Stebbins G, Goetz CG. Hallucinations in Parkinson disease. Nat Rev Neurol 2009;5:331–342.
19. Nishio Y, Yokoi K, Hirayama K, et al. Defining visual illusions in Parkinson's disease: kinetopsia and object misidentification illusions. Park Relat Disord 2018;55:111–116.
20. Wood RA, Hopkins SA, Moodley KK, Chan D. Fifty percent prevalence of extracampine hallucinations in Parkinson's disease patients. Front Neurol 2015;6:263.
21. Grimby A. Bereavement among elderly people: grief reactions, post-bereavement hallucinations and quality of life. Acta Psychiatr Scand 1993;87:72–80.
22. Zijlmans M, Van Eijsden P, Ferrier CH, Kho KH, Van Rijen PC, Leijten FSS. Illusory shadow person causing paradoxical gaze deviations during temporal lobe seizures. J Neurol Neurosurg Psychiatry 2009;80:686–688.
23. Persinger MA. Sense of a presence and suicidal ideation following traumatic brain injury: indications of right-hemispheric intrusions from neuropsychological profiles. Psychol Rep 1994;75(3 pt 1):1059–1070.
24. Uchiyama M, Nishio Y, Yokoi K, et al. Pareidolias: complex visual illusions in dementia with Lewy bodies. Brain 2012;135(pt 8):2458–2469.
25. Ruiz M, Arias A, Sánchez-Llanos E, et al. Minor hallucinations in Alzheimer's disease. J Alzheimers Dis 2018;64:543–549.

26. Lenka A, George L, Arumugham SS, et al. Predictors of onset of psychosis in patients with Parkinson's disease: who gets it early? Parkinsonism Relat Disord 2017;44:91–94.

27. Goetz CG, Fan W, Leurgans S, Bernard B, Stebbins GT. The malignant course of "benign hallucinations" in Parkinson disease. Arch Neurol 2006;63:713–716.

28. McKinlay a, Grace RC, Dalrymple-Alford JC, Anderson T, Fink J, Roger D. A profile of neuropsychiatric problems and their relationship to quality of life for Parkinson's disease patients without dementia. Parkinsonism Relat Disord 2008;14:37–42.

29. Aarsland D, Larsen JP, Tandberg E, Laake K. Predictors of nursing home placement in Parkinson's disease: a population-based, prospective study. J Am Geriatr Soc 2000;48:938–942.

30. Marsh L, Williams JR, Rocco M, Grill S, Munro C, Dawson TM. Comorbidities in patients with Parkinson disease and psychosis. Neurology 2004;63:293–300.

31. Goetz CG, Fan W, Leurgans S. Antipsychotic medication treatment for mild hallucinations in Parkinson's disease: positive impact on long-term worsening. Mov Disord 2008;23:1541–1545.

32. Ffytche DH, Pereira JB, Ballard C, Chaudhuri KR, Weintraub D, Aarsland D. Risk factors for early psychosis in PD: insights from the Parkinson's Progression markers initiative. J Neurol Neurosurg Psychiatry 2017;88:325–331.

33. Gibson G, Mottram PG, Burn DJ, et al. Frequency, prevalence, incidence and risk factors associated with visual hallucinations in a sample of patients with Parkinson's disease: a longitudinal 4-year study. Int J Geriatr Psychiatry 2013;28:626–631.

34. Lee AH, Weintraub D. Psychosis in Parkinson's disease without dementia: common and comorbid with other non-motor symptoms. Mov Disord 2012;27:858–863.

35. Voon V, Fox S, Butler TR, Lang AE. Antidepressants and psychosis in Parkinson disease: a case series. Int J Geriatr Psychiatry 2007;22:601–604.

36. Bergman J, Lerner PP, Sokolik S, Lerner V, Kreinin A, Miodownik C. Successful use of escitalopram for the treatment of visual hallucinations in patients with Parkinson disease. Clin Neuropharmacol 2017;40:246–250.

37. Kulisevsky J, Roldan E. Hallucinations and sleep disturbances in Parkinson's disease. Neurology 2012;63(8 suppl 3):S28–S30.

38. Pappert EJ, Goetz CG, Niederman FG, Raman R, Leurgans S. Hallucinations, sleep fragmentation, and altered dream phenomena in Parkinson's disease. Mov Disord 1999;14:117–121.

39. Gjerstad MD, Boeve B, Wentzel-Larsen T, Aarsland D, Larsen JP. Occurrence and clinical correlates of REM sleep behaviour disorder in patients with Parkinson's disease over time. J Neurol Neurosurg Psychiatry 2008;79:387–391.

40. Pacchetti C, Manni R, Zangaglia R, et al. Relationship between hallucinations, delusions, and rapid eye movement sleep behavior disorder in Parkinson's disease. Mov Disord 2005;20:1439–1448.

41. Forsaa EB, Larsen JP, Wentzel-Larsen T, et al. A 12-year population-based study of psychosis in Parkinson disease. Arch Neurol 2010;67:996–1001.

42. Lenka A, Hegde S, Jhunjhunwala KR, Pal PK. Interactions of visual hallucinations, rapid eye movement sleep behavior disorder and cognitive impairment in Parkinson's disease: a review. Parkinsonism Relat Disord 2016;22:1–8.

43. Barrett MJ, Smolkin ME, Flanigan JL, Shah BB, Harrison MB, Sperling SA. Characteristics, correlates, and assessment of psychosis in Parkinson disease without dementia. Park Relat Disord 2017;43:56–60.

44. Diederich NJ, Goetz CG, Stebbins GT. Repeated visual hallucinations in Parkinson's disease as disturbed external/internal perceptions: focused review and a new integrative model. Mov Disord 2005;20:130–140.

45. Llebaria G, Pagonabarraga J, Martínez-Corral M, et al. Neuropsychological correlates of mild to severe hallucinations in Parkinson's disease. Mov Disord 2010;25:2785–2791.

46. Lenka A, Hegde S, Arumugham SS, Pal PK. Pattern of cognitive impairment in patients with Parkinson's disease and psychosis: a critical review. Park Relat Disord 2017;37:11–18.

47. Morgante L, Colosimo C, Antonini A, et al. Psychosis associated to Parkinson's disease in the early stages: relevance of cognitive decline and depression. J Neurol Neurosurg Psychiatry 2012;83:76–82.

48. Pagonabarraga J, Soriano-Mas C, Llebaria G, López-Solà M, Pujol J, Kulisevsky J. Neural correlates of minor hallucinations in non-demented patients with Parkinson's disease. Park Relat Disord 2014;20:290–296.

49. Friedman JH. Editorial on: Pagonabarraga J, Martinez-Hora S, Fernandez de Bobadilla R et al. Minor hallucinations occur in drug-naïve Parkinson's disease patients even from the premotor phase. Mov Disord 2016;31:433.

50. Lenka A, Jhunjhunwala KR, Saini J, Pal PK. Structural and functional neuroimaging in patients with Parkinson's disease and visual hallucinations: a critical review. Park Relat Disord 2015;21:683–691.

51. Goldman JG, Stebbins GT, Dinh V, et al. Visuoperceptive region atrophy independent of cognitive status in patients with Parkinson's disease with hallucinations. Brain 2014;137(pt 3):849–859.

52. Janzen J, van't Ent D, Lemstra AW, Berendse HW, Barkhof F, Foncke EMJ. The pedunculopontine nucleus is related to visual hallucinations in Parkinson's disease: preliminary results of a voxel-based morphometry study. J Neurol 2012;259:147–154.

53. Shin S, Lee JE, Hong JY, Sunwoo M, Sohn YH, Lee PH. Neuroanatomical substrates of visual hallucinations in patients with non-demented Parkinson's disease. J Neurol Neurosurg Psychiatry 2012;83:1155–1161.

54. Ibarretxe-Bilbao N, Ramírez-Ruiz B, Tolosa E, et al. Hippocampal head atrophy predominance in Parkinson's disease with hallucinations and with dementia. J Neurol 2008;255:1324–1331.

55. Ibarretxe-Bilbao N, Ramirez-Ruiz B, Junque C, et al. Differential progression of brain atrophy in Parkinson's disease with and without visual hallucinations. J Neurol Neurosurg Psychiatry 2010;81:650–657.

56. Lenka A, Ingalhalikar M, Shah A, et al. Hippocampal subfield atrophy in patients with Parkinson's disease and psychosis. J Neural Transm 2018;125:1361–1372.

57. Lefebvre S, Baille G, Jardri R, et al. Hallucinations and conscious access to visual inputs in Parkinson's disease. Sci Rep 2016;6:36284.

58. Bejr-kasem H, Pagonabarraga J, Martínez-Horta S, et al. Disruption of the default mode network and its intrinsic functional connectivity underlies minor hallucinations in Parkinson's disease. Mov Disord 2018; 34:78–86;mds.27557.

59. Shine JM, Halliday GM, Gilat M, et al. The role of dysfunctional attentional control networks in visual misperceptions in Parkinson's disease. Hum Brain Mapp 2014;35:2206–2219.

60. Nishio Y, Yokoi K, Uchiyama M, et al. Deconstructing psychosis and misperception symptoms in Parkinson's disease. J Neurol Neurosurg Psychiatry 2017;88:722–729.

61. Bohnen NI, Kanel P, Müller MLTM. Molecular imaging of the cholinergic system in Parkinson's disease. Int Rev Neurobiol 2018;141:211–250.

62. Firbank MJ, Parikh J, Murphy N, et al. Reduced occipital GABA in Parkinson disease with visual hallucinations. Neurology 2018;91:e675–e685.

63. Onofrj M, Gilbert GJ. GABA and hallucinations in Parkinson disease: who is that sitting on my chair? Neurology 2018;91:293–294.

64. Shine JM, Bell PT, Matar E, et al. Dopamine depletion alters macroscopic network dynamics in Parkinson's disease. Brain 2019;142:1024–1034.

65. Bodis-Wollner I. Visual deficits related to dopamine deficiency in experimental animals and Parkinson's disease patients. Trends Neurosci 1990;13:296–302.

66. Onofrj M, Bodis-Wollner I. Dopaminergic deficiency causes delayed visual evoked potentials in rats. Ann Neurol 1982;11:484–490.

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