The Canadian Journal of Neurological Sciences (2025), 1–2
doi:10.1017/cjn.2025.10375

Canadian Journal of Neurological Sciences
Journal Canadien des Sciences Neurologiques

Letter to the Editor: New Observation

Metamorphopsia in a Male with Parkinson Disease: A Giant Problem?

Eric Noyes[1], Natasha Gattey[2], Jenna Reding[1] and Alex Rajput[1]

[1]Saskatchewan Movement Disorders Program, Division of Neurology, Saskatchewan Health Authority/University of Saskatchewan, Saskatoon, SK, Canada and [2]Division of Child and Adolescent Psychiatry, Department of Psychiatry, Saskatchewan Health Authority/University of Saskatchewan, Saskatoon, SK, Canada

Keywords: Connectomics; dementia; minor hallucinations; metamorphopsia; Parkinson disease

Minor hallucinations (MH) are commonly observed in Parkinson disease (PD) and are increasingly recognized as important markers of cognitive decline.[1–2] Metamorphopsia, a rare visual illusion phenotype, is infrequently reported in PD.[3] We report on a patient with PD who experienced metamorphopsia as part of an evolving cognitive profile, which preceded precipitous cognitive decline and progression of visual phenomena with loss of insight.

A male of European ancestry developed right-hand resting tremor at age 66. By age 68, he reported micrographia, hypophonia, and difficulty lifting his right leg while ambulating. Eight years prior to motor onset (age 58), he developed anosmia and dream re-enactment behaviors. Neurological examination at age 68 revealed resting tremor, bradykinesia, and rigidity, most pronounced on the right. His mini-mental state examination (MMSE) score was 30/30. The patient was 6'1" tall and weighed 182 lbs. He was diagnosed with PD but initially declined medications for treatment of motor symptoms. One year later, at age 69, he was started on oral levodopa (LD) with excellent results. Two years after initiating medication and titrating to a LD equivalent daily dose of 800 mg, he developed motor fluctuations and peak-dose dyskinesia. At age 72, his MMSE score was 29/30, and he now described "brain fog" during wearing off of LD. He denied any form of visual phenomena or other hallucinations at this time.

At age 73, he began experiencing features of metamorphopsia. This occurred in the absence of increasing levels of dopaminergic medications. He often perceived the staff members at his retirement community as "giants" despite them being much shorter in stature compared to himself. He estimated the height of these giants at 15–20 feet tall. Occasionally, he perceived rooms as being larger to accommodate these giants, causing him to feel small. During the first few weeks of experiencing these illusions, he frequently asked them to stand back-to-back with him to compare heights. After several encounters, he recognized that his perception of them as giants was distorted and thereafter maintained good insight into the phenomenon. Presence hallucinations emerged within 6 months, and repeat examination revealed polyminimyoclonus.

At age 74, his metamorphopsia resolved, but passage hallucinations developed alongside cognitive decline, with his MMSE score dropping to 21/30. He was previously adept with computers but had lost the ability to use this technology, except for playing simple games. His family also discovered that he was attempting to send emails from Microsoft Word. Within six months, he developed structured hallucinations (SH). These involved visual, auditory, and tactile hallucinations. He described a group of six people entering his home and making themselves comfortable by sitting in his favorite chair or eating his meals. He often became upset during these episodes, calling the police and attempting to physically remove the figures from his home. Despite numerous attempts by family and healthcare workers to explain the nature of these phenomena to him, efforts were futile as he no longer maintained insight. He was investigated for delirium twice within three months, but no medical etiology was identified for his rapid neurocognitive decline. Brain imaging with a CT scan was unremarkable. His symptoms of psychosis were refractory to quetiapine 50 mg PO BID. He was subsequently started on rivastigmine 3 mg PO BID, which led to improvement of his hallucinations after 4 weeks of therapy. His cognitive function was otherwise unchanged.

This case highlights the evolving nature of visual phenomena in PD, with a focus on metamorphopsia and its progression to SH with cognitive decline. Our patient's rate of decline was striking. Currently, there is limited evidence on the rate of minor hallucinations progressing to SH, but a 2016 study found that 28.5% of patients with MH had made this transition within 4.4 years.[4] We are not aware of any studies reporting the rate of progression to SH and dementia in PD patients from the onset of MH.

Our patient's abrupt cognitive decline within a year of developing metamorphopsia aligns with observations that minor visual phenomena may predict worsening cognitive function and progression to psychosis.[1–2] This observation has led to proposals that MH may be used as a biomarker in PD.[5] It is unclear if different types of MH (passage, presence, or visual illusions) carry a higher risk of progressing to SH with cognitive decline.

Multiple brain networks and their connectivity have been studied in relation to psychosis in PD.[1] In 2011, Shine et al proposed the "attentional network dysfunction" hypothesis. This hypothesis describes a failure of recruitment through the dorsal

Corresponding author: Eric Noyes; Email: eric.noyes@usask.ca

Cite this article: Noyes E, Gattey N, Reding J, and Rajput A. Metamorphopsia in a Male with Parkinson Disease: A Giant Problem?. *The Canadian Journal of Neurological Sciences*, https://doi.org/10.1017/cjn.2025.10375

attention network, which is responsible for sustained attention during ambiguous visual input, leading to incorporation of perceptual errors into the visual processing stream.[6] Unfortunately, longitudinal studies focusing on the dynamic transition between functional connectivity in different types of hallucinations are lacking. We are aware of a single study that attempted to measure the impact of early treatment of MH using rivastigmine.[7]The trial was prematurely discontinued due to a lack of funding and difficulty with patient enrollment. Another limitation was that participants in the study had a mean MH onset of 2.2 years (standard deviation 2.8 years) prior to enrollment.

Given the recent observations that both prevalence and incidence of PD are increasing,[8] further research is warranted to investigate if early intervention through a variety of treatments can modify brain connectomics. The goal is to produce a disease-modifying effect through preservation of normal functional connectivity. Additionally, further exploration of the prognosis surrounding different types of MH may help the rising tide of individuals with PD better prepare for the years ahead.

Metamorphopsia is a rare phenomenon in PD. Our case highlights a patient with precipitous cognitive decline progressing to psychosis after the onset of metamorphopsia. There is a lack of published data describing the rate of MH progressing to SH and dementia in patients with PD. Identifying this rate of progression may help predict prognosis and guide treatment in PD patients who develop MH. Furthermore, narrative descriptions of MH phenotypes may provide additional insights into prognosis and thus should be explored.

Acknowledgements. We are grateful to Linda Beatty for her patient and diligent preparation of the manuscript. We thank the Royal University Hospital Foundation for their continued support of the Saskatchewan Movement Disorders Program.

Author contributions. E.N. Writing – original draft, examination of patient, N.G. – review and editing, literature review contribution, J.R. – review and editing, literature review contribution, A.R. – review and editing, examination of patient. All authors have read and agreed to the final manuscript.

Funding statement. The authors do not report any funding for this project.

Competing interests. E.N. received support from Ipsen, Parkinson Canada, and AbbVie for attending conferences. A.R. received honoraria from Ipsen for moderating a presentation and the quebec consortium for drug discovery for reviewing an application. N.G. and J.R. have no conflict of interest.

References

1. Lenka A, Pagonabarraga J, Pal PK, Bejr-Kasem H, Kulisvesky J. Minor hallucinations in Parkinson disease: a subtle symptom with major clinical implications. *Neurology*. 2019;93:259–266. doi: 10.1212/WNL.0000000000007913.
2. Bejr-Kasem H, Sampedro F, Marin-Lahoz J, Martinez-Horta S, Pagonabarraga J, Kulisevsky J. Minor hallucinations reflect early gray matter loss and predict subjective cognitive decline in Parkinson's disease. *Eur J Neurol*. 2021;28:438–447. doi: 10.1111/ene.14576.
3. Sasaki C, Yokoi K, Takahashi H, et al. Visual illusions in Parkinson's disease: an interview survey of symptomatology. *Psychogeriatrics*. 2022;22:38–48. doi: 10.1111/psyg.12771.
4. Pagonabarraga J, Martinez-Horta S, Fernández de Bobadilla R, et al. Minor hallucinations occur in drug-naive Parkinsons disease patients, even from the premotor phase. *Mov Disord*. 2016;31:45–52. doi: 10.1002/mds.26432.
5. Pagonabarraga J, Bejr-Kasem H, Martinez-Horta S, Kulisevsky J. Parkinson disease psychosis: from phenomenology to neurobiological mechanisms. *Nat Rev Neurol*. 2024;20:135–150. doi: 10.1038/s41582-023-00918-8.
6. Shine JM, Halliday GM, Naismith SL, Lewis SJ. Visual misperceptions and hallucinations in Parkinsons disease: dysfunction of attentional control networks?. *Mov Disord*. 2011;26:2154–2159. doi: 10.1002/mds.23896.
7. van Mierlo TJM, Foncke EMJ, Post B, et al. Rivastigmine for minor visual hallucinations in Parkinson's disease: a randomized controlled trial with 24 months follow-up. *Brain Behav*. 2021;11:e2257. doi: 10.1002/brb3.2257.
8. Noyes E, Rajput AH, Kim M, Rajput A. Increased survival in contemporary Parkinson's disease: a 47-year autopsy study. *Neuroepidemiology*. 2024;14:1–8. doi: 10.1159/000541420.