

## Case Report

# Visual Hallucinations in Parkinson Disease relates to Profession: A Case Report

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## Abstract

Visual Hallucinations (VH) are common in Parkinson Disease (PD) and are likely related to changes in the visual pathways as shown by increased serotonergic binding and changes on structural and functional brain studies. We present a case of a retired pediatrician with PD who presented with persistent hallucinations of seeing small children in the setting of dopaminergic medication use. The content of his hallucinations was very consistent throughout. We review the literature related to visual hallucination and suggest that contents of visual hallucinations in PD are likely related to prior strong emotional visual memories including those generated through the patient's profession.

## INTRODUCTION

Parkinson disease (PD) is the second most common neurodegenerative disorder, affecting 1% of individuals older than 65 years of age, with similar prevalence across the globe [1]. The four cardinal symptoms of PD are motor, namely bradykinesia, resting tremor, rigidity and postural instability. However, PD is strongly associated with non-motor symptoms such as cognitive impairment, behavioral disturbances, autonomic instability, hallucinations, depression and sleep disorders [2]. Parkinson Disease Psychosis (PDP) is a common late-stage complication of PD with nearly 50% life time risk in this population [3]. This is characterized by presence of hallucinations or delusions, lasting more than one month in patient with a diagnosis of PD. PDP symptoms progress over time [4] and are associated with significant burden for patients and caregivers including increased hospitalization [5], increased attention by caregivers and decline in physical health [6].

Hallucinations are defined as having sensory perceptions in the absence of external sensory stimuli and can be visual, auditory, tactile, olfactory, gustatory and somatic. Visual hallucinations are by far the most common symptom in PDP [3]. Risk factors for PDP include long duration of disease, cognitive impairment, sleep-wake disturbances and use of dopaminergic agents [7]. Impairment in motor status, ocular disorders and depression are other minor risk factors [8]. Treatment for PD psychosis is complex, and includes non-pharmacological and pharmacological options. However, the long-term benefits and complications of the available therapeutics have not been ascertained [9].

Visual hallucinations in PD may be related to lack of

suppression of visual association cortex from primary visual cortex due to decrease in visual input due to retinal dopamine deficiency [10,11]. Although PD medications also play a role in the production of visual hallucinations, the mechanism for this observation is still unclear [12,13]. Due to excessive activation of existing pathways, it is quite possible that hallucinations can 're-live' a prior visual experience. Patients have often reported seeing a deceased loved one, including deceased pets. In our experience, we have noted findings quite often related to prominent prior visual experiences which in most cases relate the work habits. Here we summarize a case report to support and discuss our hypothesis that VH in PD are commonly associated with prior visual experiences especially regarding patients' profession.

## CASE REPORT

78 years old male, retired pediatrician with a history of PD since 2006, presented to the neurology clinic in May 2015 with hallucinations. He had Idiopathic Parkinson's Disease since 2009 and had postural imbalance with gait problems, poor posture, drooling, Rapid Eye Movement Sleep Behavior Disorder (RBD) and freezing of gait. His primary complaint, however, was visual hallucinations that started in 2011. He saw images of one or two small children playing at some distance, usually on waking up from sleep. He reported these images were sharp, colored and very vivid. These occurred 2-3 times/day, lasting for few seconds to couple of minutes and usually during the medication ON-phase. He had intact insight and hallucinations were not frightening. He had no interaction with them and they were consistently the same children. No triggers were noted and images easily disappeared by diverting the attention. In 2013

(2 years after onset of hallucinations), he started to see a man standing or sitting at some distance. Now he became disturbed and reacted to him saying “get out” and “go away”, but insight was retained most of the time. On examination, he had Hoehn and Yahr Stage 3 Parkinsonism with partial ON UPDRS-Part III score of 52, with a rare right hand typical resting tremor and mainly rigidity with akinesia. Cervical dystonia (mainly anterocollis) and drooling was noted. Other significant findings included hypometric saccades, occasional square wave jerks, mild finger to nose dysmetria with no upper motor neuron findings. He was taking Carbidopa-levodopa 25-250 mg 0.5 tablet six times daily along with Carbidopa-levodopa Extended release 50-200 mg tablet at 3 am and ropinirole 2 mg three times daily. He reported hallucinations started after ropinirole was added. After decreasing ropinirole to 4 mg daily, the hallucinations improved significantly but did not resolve. On follow up, he reported consistent improvement with decrease in frequency and he was maintained on same prescription.

## DISCUSSION

Different assumptions have been laid forward regarding visual hallucinations in PD such as overstimulation of mesolimbic structures with dopaminergic medications, Lewy body deposition in temporal lobe and abnormality in cortical areas involving visual cortex and visual pathway [10,11]. However most accepted hypothesis is related to impair visual processing in the setting of dopaminergic and cholinergic degeneration.

To our knowledge, Diederich NJ et al, were the first ones who proposed theory of impaired visual processing in PD patients with hallucinations in 1998. They also explained that VH in PD are due to re-emergence of previously stored percepts which normally suppressed in the presence of sufficient visual input due to retinal dopamine deficiency [10]. There is evidence that 5-HT<sub>2A</sub> receptor binding is increased in PD patients with hallucinations in certain regions of the brain including the ventral visual pathway, the bilateral dorsolateral prefrontal cortex, medial orbitofrontal cortex, and insula [14]. Although PD medications also play a role in the production of visual hallucinations, the mechanism for this observation is still unclear [12,13]. Diederich and Holroyd have individually put forward the theory that visual hallucinations may be related to lack of suppression of visual association cortex from primary visual cortex due to decrease in visual input seen in PD due to retinal dopamine deficiency [10,11]. This theory was later supported by Holroyd and Wooten's work with fMRI in PD patients with and without hallucinations that examined visual cortices as well as visual pathways. They noted enhanced activity in visual association cortex but activation deficient in primary visual cortex along with retinal dopamine deficiency in patients with hallucinations as compared to those without [11].

A voxel-based morphometry (VBM) analysis of brain Magnetic Resonance Imaging (MRI) revealed that PD patients with hallucinations, especially visual, show reduced grey matter volume in frontal, temporal and thalamic areas. Structural abnormalities to these areas can be responsible for impaired visual processing leading to visual hallucinations [15]. Grey matter volume reductions in the lingual gyrus and superior parietal lobe areas were also observed in hallucinated PD patients, which are both involved in higher visual processing.

These findings support the idea of impaired visual processing as a mechanism of visual hallucinations in PD patients.

Commonly the contents of VH are people, animals, objects, whole faces, colors, motion, scenery and sometimes vague which confirm the involvement of ventral and dorsal visual pathways responsible [8]. In our experience patients may report seeing small or little people but they are usually of adult age. Our patient, a pediatrician, hallucinated mostly about children. This raises questions as to whether these hallucinations are related to his profession or prior memories. Similar experience has been described by farmers in rural communities where they reported seeing farm animals which is an otherwise highly rare finding (personal experience and communications). In the same way, a textile artist hallucinates about colored strings lying near her (personal communication). Some other authors have reported that VH in PD patients may be related to something of importance to patients, such as seeing a pet dog which had died 5 years ago [16].

Conventional theories believe only limbic system of the medial temporal lobe is responsible for storage and processing of memories. However, Lashley's research shows that long term memories are stored all over the cortex with respect to their stimulus after consolidation in hippocampus e.g. neuronal groups in visual cortex will store a sight memory. Recent studies support the fact that the hippocampus is only a relay center not a reservoir/storage place for long term memories. It might act as a binding area for different cortical regions that participated in the initial encoding of memory [17]. Association of visual memory storage in visual cortex and visual processing impairment in PD supports our suggestion that VH contents in PD are related to patient's prior memories which are often related to profession and patient's emotional attachments.

## CONCLUSION

Current work suggests that visual hallucinations in Parkinson Disease Psychosis are associated with impairment in visual cortices and visual processing areas and we propose that these are usually consistent with prior visual memories of significant for patients and commonly relates to their professional life.

## REFERENCES

1. Alexis Elbaza, James H Bowerb, Demetrius M Maraganoreb, Shannon K McDonnell, Brett J Petersona, J.Eric Ahlskogb, et al. Risk tables for parkinsonism and Parkinson's disease. J Clin Epidemiol. 2002; 55: 25-31.
2. Hauser RA, Biaggioni I, Hewitt LA, Vernino S. Integrated Analysis of Droxidopa for the Treatment of Neurogenic Orthostatic Hypotension in Patients with Parkinson Disease. Mov Disord Clin Pract. 2018; 5: 627-634.
3. Forsaa EB, Larsen JP, Wentzel-Larsen T, Goetz CG, Stebbins GT, Aarsland D, et al. A 12-Year Population-Based Study of Psychosis in Parkinson Disease. Arch Neurol. 2010; 67: 996-1001.
4. Ravina B, Marder K, Fernandez HH, Friedman JH, McDonald W, Murphy D, et al. Diagnostic criteria for psychosis in Parkinson's disease: Report of an NINDS, NIMH work group. Mov Disord. 2007; 22: 1061-1068.
5. Klein C, Prokhorov T, Miniovitz A, Dobronevsky E, Rabey JM. Admission of Parkinsonian patients to a neurological ward in a community

- hospital. *J Neural Transm.* 2009; 116: 1509-1512.
6. Schrag A, Hovris A, Morley D, Quinn N, Jahanshahi M. Caregiver-burden in parkinson's disease is closely associated with psychiatric symptoms, falls, and disability. *Parkinsonism Relat Disord.* 2006; 12: 35-41.
  7. Zahodne LB, Fernandez HH. Pathophysiology and Treatment of Psychosis in Parkinson's disease: A Review. *Drugs & Aging.* 2008; 25: 665-682.
  8. Mosimann UP, Rowan EN, Partington CE, Collerton D, Littlewood E, O'Brien JT, et al. Characteristics of Visual Hallucinations in Parkinson Disease Dementia and Dementia with Lewy Bodies. *Am J Geriatr Psychiatry.* 2006; 14: 153-160.
  9. Bhatti D, Torres-Russotto D. Pharmacological Management of Psychosis in Parkinson Disease: A Review. *CDTH.* 2012; 7: 151-163.
  10. Diederich NJ, Goetz CG, Raman R, Pappert EJ, Leurgans S, Piery V. Poor visual discrimination and visual hallucinations in Parkinson's disease. *Clin Neuropharmacol.* 1998; 21: 289-295.
  11. Holroyd S, Wooten GF. Preliminary FMRI Evidence of Visual System Dysfunction in Parkinson's disease Patients With Visual Hallucinations. *J Neuropsychiatry Clin Neurosci.* 2006; 18: 402-404.
  12. Kuzuhara S. Drug-induced psychotic symptoms in Parkinson's disease. Problems, management and dilemma. *J Neurol.* 2001; 248: 28-31.
  13. Banerjee AK, Falkai PG, Savidge M. Visual hallucinations in the elderly associated with the use of levodopa. *Postgrad Med J.* 1989; 65: 358-361.
  14. Ballanger B, Strafella AP, van Eimeren T, Zurowski M, Rusjan PM, Houle S, et al. Serotonin 2A Receptors and Visual Hallucinations in Parkinson Disease. *Arch Neurol.* 2010; 67: 416-421.
  15. Shin S, Lee JE, Hong JY, Sunwoo MK, 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.
  16. Barnes J. Visual hallucinations in Parkinson's disease: a review and phenomenological survey. *J Neurol Neurosurg Psychiatry.* 2001; 70: 727-733.
  17. Kanthamalee S, Sripankaew K. Effect of neurobic exercise on memory enhancement in the elderly with dementia. *JNEP.* 2013; 4: 69.

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