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Review article

Pattern of cognitive impairment in patients with Parkinson's disease and psychosis: A critical review

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ABSTRACT

Psychosis is one of the debilitating non-motor symptoms (NMS) of Parkinson's disease (PD). Cognitive impairment is considered to be a risk factor for emergence of psychosis in PD. Early detection of relevant cognitive impairment may serve as a predictor for development of psychosis, with implications for prevention and early intervention. However, the exact pattern of cognitive impairment associated with psychosis is not clear. In this article, we aim to critically review the literature on case-control studies in PD patients with and without psychosis in order to understand the pattern of cognitive impairment in those with psychosis. Majority of studies conducted till date have focused on executive and visuospatial functions. Despite some inconsistencies, most of the studies found significant impairment in these domains in PD patients with psychosis compared to those without psychosis. Studies assessing for other cognitive functions such as attention, language and memory in PD patients have also found worse performance in those with psychosis. Although there is enough evidence to suggest that PD patients with psychosis have poor cognitive functioning, it is unclear if these deficits are generalized or specific. The available evidence, which is primarily in the form of cross-sectional studies assessing for specific cognitive deficits, is not adequate to indicate a clear demarcating pattern of cognitive deficits, which differentiates PD patients with and without psychosis. Longitudinal studies with extensive cognitive assessment are warranted.

1. Introduction

Parkinson's disease (PD) is characterized by core motor symptoms such as tremor at rest, rigidity, bradykinesia and postural instability [1]. In addition, patients with PD may also develop several non-motor symptoms (NMS) during the course of the illness. Psychosis, depression, cognitive impairment, autonomic dysfunction, sleep disturbances and hyposmia are few of the common NMS of PD [2]. Psychosis is one of the incapacitating NMS and it may be present in one third of the patients with PD. As per the diagnostic criteria proposed by the National Institute of Neurological Disorders and Stroke (NINDS) and the National Institute of Mental Health (NIMH) work group, psychosis in a patient with PD can be diagnosed if hallucinations or illusions or delusions or false sense of passage is/are present continuously for one month or have recurrent occurrence [3]. Psychosis commonly manifests as visual hallucinations (VH) and minor hallucinations such as sense of passage and sense of presence. Delusions, paranoid beliefs, illusions and hallucinations in other modalities may occur rarely, and when present, they usually co-exist with VH [4]. As VH is the commonest manifestation of psychosis in PD, majority of studies have focused on this symptom. Although older age, longer duration of PD, higher stage and severity of PD, long-term dopaminergic medications and cognitive impairment have been described as risk factors for emergence of psychosis in PD, the exact neurobiology and natural course still remains elusive [5]. Presence of psychosis has been described as an independent risk factor for poor functional outcome with increased nursing home placements [6], poor quality of life [7] and mortality [8] in patients with PD. Hence early identification of patients who are prone to have psychosis is of

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paramount importance. Several studies have been conducted to explore the neural and genetic correlates of psychosis in PD [9–11], however the findings have not been consistent. Studies based on neuroimaging, histopathology and neuropsychological evaluations have speculated a strong nexus of psychosis with cognitive impairment and rapid eye movement sleep behavior disorder (RBD) in patients with PD [12]. Psychosis and cognitive impairment often coexist in PD and their frequency and severity are greater in the postural instability and gait difficulty (PIGD) phenotype of PD compared to the tremor dominant phenotype [13,14]. Spectrum of cognitive impairment in PD may range from mild cognitive impairment (PD-MCI) to dementia (PDD). As MCI may be present in the patients with early and untreated PD [15], it might be possible to identify the pattern of impairment of the cognitive functions, which are possibly associated with emergence of psychosis later during the course of the illness. Early identification of pattern of cognitive impairment which has been described to have association with psychosis in PD is not only crucial for planning preventive therapies in future but also for a more stratified management of the ongoing symptoms.

This review aims to critically analyze the published literature focusing on pattern of cognitive impairment in patients with PD and psychosis.

2. Methodology

We conducted an electronic search in PubMed and PsycINFO database for publications focusing on cognitive impairment in patients with PD and psychosis published in last 30 years (from July1986 to July 2016). A broad search strategy was applied by using a number of key words and combinations, details of which are given in Table 1. Publications not relevant to the current review and duplicates were excluded after screening the titles, abstracts or full texts. The remaining articles were assessed for inclusion using the following criteria: (1) they were original studies, (2) full text was available in English, (3) cognitive parameters were compared between PD patients with and without psychosis. The flowchart depicting literature search is represented in Fig. 1.

3. Impairments in specific cognitive domains in patients with PD and psychosis

3.1. Executive dysfunction

Executive functions encompass the cognitive processes that underlie goal-directed behavior and are orchestrated by activity within the prefrontal cortex (PFC) [16]. Executive dysfunction in PD has been well established in the literature and may also be observed early in the disease [17]. Several studies have compared the results of tests corresponding to executive functions in PD patients with and without psychosis and have reported significant impairments in patients with psychosis. In a study involving 48 patients with PD (14 with hallucinations, 34 without hallucinations), Grossi et al. reported significant impairment in the executive functions in patients with psychosis [18]. The executive impairments were mainly in the tests assessing phonological and semantic fluency. One of the major limitations of this study was that the duration of disease of the two patient groups was not matched (patients with hallucinations had longer duration of disease). The authors did a two-year follow-up evaluation of 36 patients from this study (hallucinations at baseline = 9, developed hallucinations during follow up = 12, hallucination-free throughout follow up = 15) and reported significant decline in the executive functions in all the three groups [19]. Similar to the comparison during baseline, patients with hallucinations had significantly poor performance compared to the non-hallucinators. In this longitudinal study, impaired phonological fluency was the single independent predictor of onset of hallucinations during follow-up in patients who did not have hallucinations at base line. The result of this study was further reinforced by a study on 63 patients with PD (33 with hallucinations and 30 without hallucinations) by Ozer et al. [20]. In addition to the phonological fluency, Ozer et al. reported impairment in response inhibition in the hallucinating PD patients, which represents impairment in executive functions. Unlike the study by Grossi at al, the duration and stage of PD were matched between two patient groups. However, the total daily levodopa equivalent dose (LED) was significantly higher in the hallucinating PD patients. Later Imamura et al. also reported impaired performances in verbal fluency and in response inhibition suggestive of executive dysfunction in PD patients with hallucinations compared to those without hallucinations [21]. Interestingly, the executive dysfunction in PD patients with hallucinations was comparable to that of PD patients with dementia. Hence the authors speculated that presence of hallucinations might signal progression to dementia in follow-up. Barnes et al. [22], in a study involving 57 subjects (with VH: 17, without VH: 20, healthy controls: 20) compared the results of various tests assessing executive functions such as tests for (i) inhibitory ability (Stroop test and go-no-go test), (ii) category fluency (word generation in 6 categories) and (iii) working memory (2-back test and reading span). In this study, patients with VH had significantly poor performance on tests corresponding to inhibitory ability compared to patients without VH and the healthy controls. Katzen et al. [23] later compared performances across multiple cognitive domains in three groups of PD patients (i) with only VH (n = 35), (ii) with multimodal hallucinations (n = 12), (iii) without VH (n = 105). Similar to previously described studies, Katzen et al. observed significant executive dysfunction in the group with VH compared to the non-hallucinators. Interestingly there were no differences in the cognitive performances of PD patients with only VH and those with multimodal hallucinations. This suggests

Table 1
Results of PubMed and PsycINFO database search with various key words and combinations

Key words and combinations	Number of publications			
	PubMed	PsycInfo	Assessed for included	Inclusion for final review
Parkinson's Disease AND Psychosis AND Cognitive impairment	245 (6)	94 (4)	10	4
Parkinson's disease AND Psychosis AND Dementia	438 (6)	228 (4)	10	2
Parkinson's Disease AND Hallucinations AND Cognitive impairment	299 (9)	127 (11)	20	15
Parkinson's disease AND Hallucination AND Dementia	513 (14)	287 (9)	239	
Parkinson's disease AND Delusion AND Cognitive impairment	79 (3)	32 (2)	5	1
Parkinson's disease AND Delusion AND Dementia	124 (3)	75 (3)	6	1
Total	*1698(41)*	*843 (33)*	*74*	*30*
Total number of articles included for review after removing the duplicates				*13*
Total number of articles included from the reference sections of the shortlisted articles				*3*
Final number of article include for review				***16***

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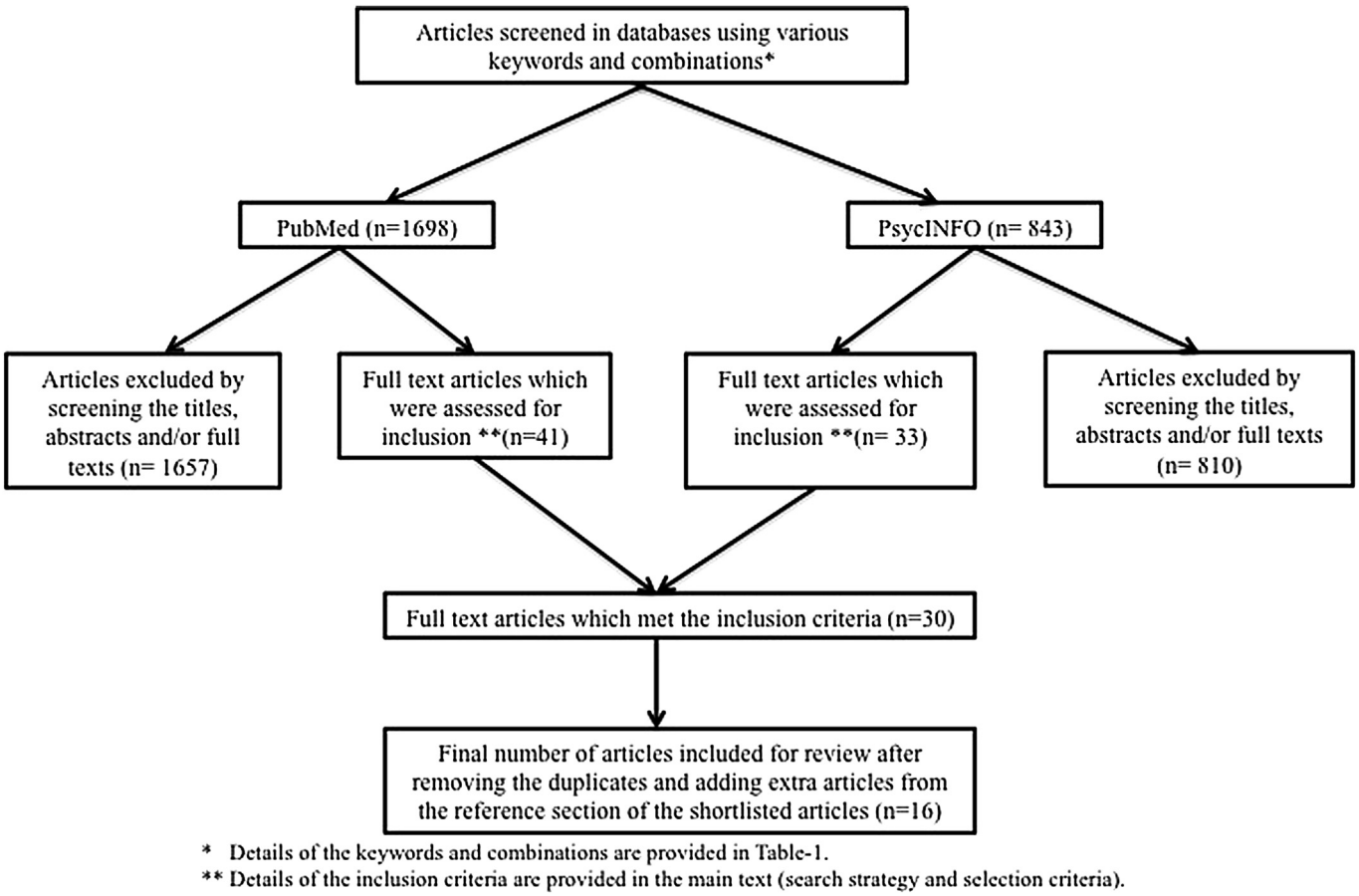

Fig. 1. Flow diagram describing the steps for shortlisting the articles for review.

hallucination in one modality may lead to significant cognitive impairment and those with hallucinations in multiple domains may not necessarily have worse cognitive performance than patients having hallucinations in a single domain. In addition to the executive dysfunction, patients with hallucinations also had impaired performance in tests assessing language, memory and visuospatial functions. Two major limitations of this study by Katzen et al. were the lack of details on the LED of the subjects and relatively lower sample size of the group with multimodal hallucinations.

Llebaria et al. were the first to compare the cognitive functions in PD patients across the two extremes of VH i.e. minor hallucinations and well formed VH [24]. Like the previous studies, the authors documented significant difference in the cognitive functions of patients with and without VH. There was no significant difference in the cognitive functions of patients with minor hallucinations and the non-hallucinators. This finding gets support from a neuroimaging study by Pagonabarraga et al. using voxel-based morphometry (VBM) [25], in which no significant atrophy in the frontal cortices which governs executive functions was observed in PD patients with minor hallucinations compared to the non-hallucinators. In the study by Llebaria et al. [24], patients with well-formed VH performed poorly in the tests measuring verbal fluency compared to those with minor hallucinations. This perhaps suggests that neurobiology of executive dysfunction in patients with PD runs parallel to that of the progression of hallucinations from lower extreme (minor hallucinations) to the higher extreme (well formed VH).

Factor et al. in a study involving 144 patients with PD compared the performances across several cognitive domains of three groups of patients with PD (i) without psychosis: 96, with only VH: 25, with predominantly delusions: 23) [26]. Similar to results of the previous studies, executive dysfunction was significant in patients with VH compared those without VH. However the authors did not find any significant cognitive dysfunction in patients with delusions compared to those without any psychotic symptoms. In addition to reinforcing the role of executive dysfunction in the genesis of VH in PD, this study by Factor et al. also suggests the possibility that VH and delusions in patients with PD may have different pathophysiological and anatomical correlates. This finding needs to be replicated in studies with larger sample size.

To summarize, executive functions have been described to be impaired in patients with PD and psychosis in most of the studies published so far. There are few studies in which no significant impairment was observed between PD patients with and without psychosis [27–30]. This conflict in results is probably secondary to differences in the demographic and disease characteristics. Although results have not been uniform, studies based on neuroimaging have reported atrophy of several regions in the frontal cortex in patients with PD and psychosis, which is in agreement with executive dysfunction reported by the studies described above [31,32]. Gama et al. in a VBM based study have reported significant atrophy of opercular frontal gyrus and superior frontal gyrus in the dominant hemisphere in PD patients with VH compared to those without VH [31]. However Gama et al. did not carry out neuropsychological evaluation of the patients in this study. Hence studies in future integrating both neuroimaging and neuropsychological evaluation may provide further insight into the role of executive

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dysfunction in emergence of psychosis in PD.

3.2. Visuoperceptive and visuospatial dysfunctions

Barnes et al. were the first to publish a study comparing visual object and space perception battery (VOSP) and source task in PD patients with and without VH [28]. In this study involving 57 subjects (with VH: 17, without VH: 20, controls: 20), it was observed that patients with PD had significantly poor performance in object recognition tasks in the VOSP as compared to the healthy controls. PD patients with VH performed poorly compared to those without VH on the same object recognition task. However, there was no significant difference in the results of tests assessing the source-memory task among the three groups. As all the tests assessing the object recognition requires the subjects to identify the silhouettes of a set of common objects and animals, the authors posited that the lack of fine details in the images perhaps contributed to the misidentification of the objects. The authors speculated an association of partial sensory deprivation with poor visuo-perception as crucial factors in the genesis of VH in PD. Subsequently several other studies also reported significantly poor visuo-perception/visuospatial functions in PD patients with VH compared to those without VH. Ramirez-Ruiz et al. compared the results of several neuropsychological tests in PD patients with ($n = 24$) and without VH ($n = 21$) and healthy controls ($n = 21$) [33]. In this study, patients with VH performed poorly in tests assessing visuospatial functions compared to those without VH and healthy controls. In addition, patients with VH also performed poorly in tests corresponding to language, verbal memory and visual memory compared the non-hallucinating counterparts. Ramirez-Ruiz et al. carried out another neuropsychological evaluation during 1-year follow up to explore pattern of cognitive decline over time [34]. The authors observed significant decline in all cognitive functions in the hallucinating group compared to the baseline performance and the largest decline was observed in the visual memory followed by visuoperceptive, visuospatial and frontal functions. Although abnormalities in the visuoperception and visuospatial functions were the major finding of the longitudinal study by Ramirez-Ruiz et al., the authors also noticed significant impairment in verbal memory, language and frontal executive functions in patients with VH. In the previously described study by Katzen et al. [23], in addition to impaired executive functions, patients with VH also had significantly poor performance in tests assessing visuospatial measures compared to the non-hallucinators. The authors did not observe any significant difference in the visuospatial functions in patients experiencing only VH compared to those having multimodal hallucinations.

There are studies, which did not observe any significant impairment in visuospatial and visuoperceptive functions in PD patients with psychosis compared to those without psychosis [20,24,27,29,30]. However these studies, which reported negative result, did not evaluate the visuospatoal functions as extensively as the studies, which reported, impaired functions. Difference in patient characteristics and medication dose might also have contributed to the conflict in the result.

To summarize, several studies have reported significant impairment in the visuospatial functions in patients with PD and hallucinations. Neuroimaging studies comparing the grey matter volumes of PD patients with and without hallucinations have also reported atrophy of regions of the brain corresponding to both dorsal and ventral visual stream [32,35]. Visuospatial dysfunction is present not only in PD patients with hallucinations, but also in PDD [36] and in other synucleinopathies such as rapid eye movement sleep behavior disorder (RBD) [37], dementia with Lewy bodies (DLBD) [38]. A recently published study by Quaranta et al. on a cohort of patients with Alzheimer's disease has also reported significant impairment in visuospatial functions in those with VH compared those without VH [39]. This perhaps suggests that alteration in visuospatial function is a common neuropsychological substrate of VH not only in PD but also in other synucleinopathies and in AD.

3.3. Alterations in attentional control

Several studies have hypothesized that genesis of hallucinations in PD is possibly secondary to the existence of an aberrant top-down visual processing which dominates the usual down-top visual processing [40]. In such a model describing the emergence of hallucinations, poor attention may play a crucial role. Multiple studies comparing the functions across several cognitive domains of PD patients with and without psychosis have reported poor attentional control in patients with psychosis. Bronnick et al. [27] in a study involving 172 patients with PDD compared the cognitive profile of patients with ($n = 86$) and without VH ($n = 86$). In this study, reaction time for several cognitive tasks was used as a measure of attentional control. The authors observed a significant poor attentional control in patients having VH and reported worse choice reaction time as an independent predictor of VH in patients with PDD. The major strength of this study was the individual matching of the patients with and without psychosis for age, gender, education, LEDs, duration of disease which has been described as risk factors for emergence of VH in PD. Factor et al. and Hepp et al. also reported impairments in tests assessing attention in PD patients with hallucinations compared to those without hallucinations [26,30]. In a recently published study, Hall et al. [41] compared the performance in attentional network test (ANT) in PD patients with ($n = 25$) and without VH ($n = 28$). ANT used in this study comprised of a cued reaction time task [42] and a flanker task [43]. In this study, the accuracy of PD patients with VH in the ANT was significantly lower than that of those without VH indicating possible alterations in the attentional network in the former group.

Studies based on neuroimaging also support the role of impaired attention in the genesis of hallucinations. Shin et al. in a study based on VBM have reported significant reduction in the grey matter volume of substantia innominata (SI) in PD patients with VH compared to those without VH [44]. SI is an important cholinergic structure of brain as the nucleus basalis of Meynert is located in SI. As cholinergic system has been described to play a vital role in sustained attention through interaction with frontal lobe and thalamo-cortical processing [45], poor attentional control can be posited to be one of the factors leading to emergence of hallucination in VH. Shine et al. in a review have emphasized that alteration in the dorsal attention network, which comprises the striatum and dorsolateral prefrontal cortex may play a crucial role in the pathogenesis of visual misperceptions and hallucinations [46]. As there is strong evidence of association of alterations in the neural network related to attentional control in patients with hallucinations, further studies integrating neuroimaging and neuropsychological evaluations in large number of patients may provide further insight into the role of impaired attention in the genesis of hallucinations.

3.4. Language, memory, and other cognitive functions

In addition to impairment in executive functions, visuospatial functions and attention, patients with PD and psychosis have been described to have deficits in language and memory functions in several studies. Chung et al. [29] compared the scores of Montreal cognitive assessment (MoCA) between patients with ($n = 26$) and without VH ($n = 32$). In this study, patients with VH had

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significantly lower total MoCA score, predominantly in the language domain. One of the demerits of this study is the lack of matching for stage of PD and the severity of depression (which was higher in patients with VH). In addition to impaired executive and visuospatial functions as described above, Factor et al. [26] and Katzen et al. [23] have also reported significantly poor performance in tests assessing language function (Boston naming test and phonemic fluency) in patients with hallucinations. Impairments in the verbal memory in patients with PD and VH was reported by both Factor et al. [26] and Ozer et al. [20]. Since impairments in

Table 2
Summary of the studies assessing cognitive performances in patients with Parkinson's disease and psychosis.

Authors	Aim of the study	Study cohort	Criteria for PD-Psychosis	Result
Barnes et al., 2003	To explore if patients with PD and VH have poor object perception and source monitoring compared to those without VH	PD + VH: 17 PD-VH: 20 HC: 20	PD: Details not mentioned VH history of at least one episode of VH in last three months	Patients having VH had poor object perception and recognition memory compared to those without VH
Grossi et al., 2005	To assess selected cognitive abilities in non-demented PD patients with and without Hallucinations	Hallucinators: 14 non-hallucinators: 34	PD: UK brain bank Psychosis: details not mentioned	PD patients with hallucinations had poor performance in tests assessing verbal learning semantic and phonological Fluency
Ramirez-Ruiz et al., 2006	To investigate the deficits in the neuropsychological domains in PD patients with and without VH	PD + VH: 24 PD-VH: 21 HC: 21	PD: Details not mentioned VH: History of VH in last one year	PD patients with VH had poor performances in language verbal learning, semantic fluency and visuoperceptive function
Ramirez-Ruiz et al., 2006	To evaluate the decline in specific neurocognitive functions in patients with PD and visual hallucinations in an one year follow up study	PD + VH: 20 PD-VH: 20 HC: 18	PD: Detais not mentioned VH: History of VH in last 1 year	Patients with VH had poor performance in tests assessing language, verbal memory,frontal and visuo-perceptive functions
Ozer et al. 2007	To assess the role of disease parameters and medications on cognitive impairment in PD patients with and without VH	PD + VH: 33 PD-VH: 30	PD: UK brain bank Psychosis: presence of definite VH in last 3 months	Patients with VH had significantly more frontal dysfunction and memory deterioration
Santangelo et al. 2007	To determine the progress of cognitive impairment in patients with and without VH	PD + VH: 21 PD-VH: 15	PD: not mentioned Psychosis: not mentioned	Patients with VH had poor phonological and semantic fluency tasks compared to those without VH
Imamura et al. 2008	To compare executive functions in non-demented PD patients with and without VH	PD: 23 PD + Vivid dreams:9 PD + VH:11 PDD:18	PD: UK brain bank VH: Details not mentioned	PD patients with VH have higher degree of executive dysfunction compared to those without VH and the pattern of impairment is similar to that observed in patients with PDD
Barnes et al. 2008	To investigate the link between executive dysfunction and occurrence of VH in PD	PD + VH: 17 PD-VH: 20 HC: 20	PD: Details not mentioned VH: Details not mentioned	Patients with hallucination have greater impairment in inhibitory ability compared to the non-hallucinators
Katzen et al. 2010	To examine the differences in neuropsychological and emotional function in patients with and without VH	PD + VH: 47 PD-VH: 105	PD: UK brain bank VH: history of one or more episode of VH in the past	Patients with hallcuinations have higher cognitive impairement compared to those without hallcuinations
Llebaria et al. 2010	To compare the pattern of impairment in neuropsychological assessment in patients with VH compared to those without VH	PD + VH: 29 PD-VH: 28	PD: Research diagnostic criteria Psychosis: MDS-UPDRS (I)	Patients with hallcuinations have cognitive impairments corresponding to froto-striatal and posterior cortical areas compared to those without VH
Bronnick et al. 2011	To compare the cognitive profile of demented PD patients with and without VH	PDD + VH: 86 PDD-VH: 86	PD: UK brain bank VH: Details not mentioned	Attentional control is an important cognitive correlate of VH in patients with PDD
Hepp et al., 2013	To investigate specific cognitive impairment in non-demented PD patients with VH	PD + VH: 31 PD-VH: 31	PD: UK brain bank Psychosis: by SCOPA-PC	Patients with VH have poor verbal learning and attention compared to those without VH
Factor et al., 2014	To determine the cognitive correlates of hallucinations and delusions in patients with PD	PD-VH/ delusion: 96 PD + VH/ delusion: 48	PD: UK brain bank Psychosis: by SAPS	Cognitive correlates of delusion and hallucination are different in patients with PD
Moustafa et al. 2014	To investigate cognitive functions in relation to occurrence of psychosis in patients with PD	PD + Psychosis: 21 PD-Psychosis: 23 HC: 22	PD: UK brain bank Psychosis: UPDRS-I (question-2)	Patients with psychosis have impaired transitive inference suggestive of dysfunction of hippocampus.
Chung et al. 2015	To analyze the domain of cognitive impairment in PD patients with VH	PD + VH: 26 PD-VH: 32	PD: UK brain bank Psychosis: NPI	Patients with psychosis have significant impairment in the language domain compared to those without psychosis
Hall et al. 2016	To explore deficits in attentional processing in patients with VH	PD + VH: 25 PD-VH: 28	PD: UK brain bank Based on MDS-UPDRS (question-2)	Patients with VH have significant impairment in attentional processing compared to those without VH

PD: Parkinson's disease, PDD: Parkinson's disease with dementia, VH: Visual Hallucinations, MDS-UPDRS: Movement Disorders Society Unified Parkinson's Disease Rating Scale, SAPS: Scale for assessment of positive symptoms, SCOPA-PC: SCales for Outcomes in PArkinson's disease-Psychiatric Complications.

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language and memory functions have not been reported as frequently as compared to executive and visuospatial dysfunctions, further studies involving larger sample size and coupled with neuroimaging may be essential to understand the role of language and memory dysfunction in the genesis of VH.

In a recent study involving 66 subjects, Moustafa et al. compared the performances of transitive interference tasks across three groups of subjects (PD with psychosis: 21, PD without psychosis: 23, healthy controls: 22) [47]. Transitive inference underscores a fundamental feature of relational memory, the ability to integrate experiences that share overlapping elements and then use this consequent relational network to guide novel judgments about elements that are related only indirectly within the network [48]. The authors observed significantly impaired performances in the patients with psychosis compared to the other groups. As transitive interference task represents hippocampal function, the authors postulated alterations in hippocampal functions in patients with PD and psychosis. It gets support from several VBM-based structural neuroimaging studies, which have reported hippocampal grey matter atrophy in PD patients with psychosis [49,50]. Further, Yao et al. [51] in a case-control study based on multimodal imaging of hippocampus in PD patients with and without VH have reported significant microstructural alterations in the white matter in posterior hippocampus and reduced functional connectivity of hippocampus with visual cortex in patients with VH.

The abnormal transitive inference task performance in PD patients with psychosis is yet to be replicated. Further studies integrating structural and functional imaging focusing on hippocampus may provide better insight.

Table 2 summarizes all the studies reviewed in this article, Table 3 summarizes the tests used to assess the cognitive functions in several domains and Table 4 summarizes the cognitive domains reported to be impaired in the studies reviewed.

Table 3
Summary of the cognitive domains tested and the neuropsychological tests used in the reviewed studies.

Authors	Neuropsychological tests employed						Domains affected in PD + VH
	Executive function	Attention	Language	Memory (Visual/verbal)	Visuoperception and Visuospatial	Others	
Grossi et al.	Phonological and Semantic fluency	–	–	RAVLT	–	Non-verbal reasoning (by RCPM)	Executive
Sanatangelo et al. (2-year follow-up study)	Phonological and semantic fluency	–	–	RAVLT	–	Non-verbal reasoning (by RCPM)	Executive
Ozer et al.	Stroop, WCST, clock Categorical fluency Clock drawing		–	SBST, Wechsler memory scale	JLO, BFR	–	Executive and memory
Imamura et al.	Stroop test Verbal fluency	Digit span	–	–	–	–	Executive
Barnes et al.	Stroop test, go-no-go Category fluency 2-back test, word span reading span	–	–	–	–	–	Executive
Katzen et al.	PASAT WCST SDMT	Trail making Digit span	Boston naming test controlled oral-word association test	CVLT BVRT	GET HVOT JLO	–	Visuospatial and executive
Liberia et al.	MDRS and PD-CRS	MDRS and PD-CRS	MDRS and PD-CRS	MDRS and PD-CRS	MDRS and PD-CRS	–	Executive
Factor et al.	WCST, Stroop Trail making-B	Digit span Trail making-A	Boston naming test and timed phonemic fluency.	Delayed story recall delayed recall of words	JLO	–	Global impairment
Barnes et al.	Verbal fluency	–	–	Recognition memory test for faces and words	VOSP	–	Visuoperception and memory
Ramirez-Ruiz et al.	Phonological Semantic fluency test	–	Token test Boston naming test	RAVLT Warrington's recognition memory	BFRT VFDT	–	Visuospatial language, memory and executive functions
Ramirez-Ruiz et al. (1-year follow-up study)	Phonological Semantic fluency test	–	Token test Boston naming test	RAVLT Warrington's recognition memory test	BFRT VFDT	–	Visuospatial language, memory and executive functions
Bronnick et al.	Verbal fluency (from ADAS-cog)	Choice and simple reaction time from CDR	–	Naming task (from ADAS-cog)	Visual construction task (from ADAS-cog)		Attention
Hepp et al.	Verbal fluency Backward disgit span Stroop test	Forward digit span and Trail making-A	Boston naming test	RAVLT Visual association test	Rey complex figure test		Verbal learning and attention
Hall et al.	–	Attention network test	–	–	–	–	Attention
Chung et al.	MoCA component	MoCA component	MocA component	MoCA component	MoCA component		Language
Moustafa et al.	Short and long delay working memory task	–	–	–	–	Transitive inference task	Hippcoampal function

RAVLT: Rey auditory verbal learning test, RCPM: Raven's colored progressive matrices, WCST: Wisconsin Card Sorting Test, SBST: SozelBellekSurecleriTest, JLO: Judgment Line Orientation, BFRT: Benton Facial Recognition Test, PASAT: Paced Auditory Serial Addition Task, SDMT: Symbol Digit Modalities Test), CVLT: California Verbal Learning Test, GET: Ghent Embedded figure Test, HVOT: Hooper Visual Orientation Test, MDRS: Mattis Dementia Rating scale, PD-CRS: Parkinson's Disease Cognitive Rating Scale, VOSP: Visual Object Space Perception battery, VFDT: Benton Visual form Discrimination, ADAS-cog, MoCA: Montreal Cognitive Assessment Scale.

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Table 4
Summary of cognitive domains affected in patients with Parkinson's disease and visual hallcuinations.

Cognitive domains	Total number of studies	Number of studies reporting Impaired function	Number of studies reporting no impairment
Executive function	15	10 Ref: [18–24], [26], [33–34]	5 Ref: [27–30]
Visuoperceptive /visuospatial	10	5 Ref: [23], [26], [28] [33–34]	5 Ref: [20], [24], [27], [29], [30]
Attention	8	4 Ref: [26], [27], [30] [41]	4 Ref: [21], [23] [24], [29]
Language	7	4 Ref: [26], [29], [33–34]	3 Ref: [23] [24], [30]
Memory	12	4 Ref: [20], [26] [33–34]	8 Ref: [18], [19], [21], [23], [24], [27], [29], [30]
Others (Transitive interference)	1	1 [47]	0

4. Conclusion and perspectives

Multitudes of case-control studies have compared the cognitive performances of PD patients with and without psychosis. It is evident from these studies that PD patients with psychosis have significantly more cognitive impairment compared to those without psychosis. Further, certain cognitive deficits have been described to be frequently associated with psychosis in patients with PD. The cognitive functions commonly reported to be impaired in PD patients with psychosis are in the executive, attention and visuospatial domains. Neuroimaging studies on PD patients with psychosis have found grey matter atrophy in frontal cortex and regions corresponding to dorsal and visual stream, which may be etiologically related executive and visuospatial dysfunctions respectively. However, Meppelink et al. [52] did not find significant difference in grey matter volumes between patients with and without VH. More studies integrating advanced neuroimaging with neuropsychological evaluation are warranted for better understanding of the neurobiology of psychosis in PD. Other studies have reported impaired attention, language and memory functions in the hallucinating PD patients. This is one of the limitations to better defining and understanding this complex topic, as the cognitive domains, which have been reported to have impairment in patients with PD and psychosis, are broad and non-specific. Most of the studies have focused on particular cognitive functions with exclusion of others. Studies with assessment of cognitive functions across multiple domains would help in delineating the deficits, which are specific to this population.

Most of the studies reviewed in this article were cross-sectional in nature. Hence it is difficult to predict if the patients who did not have any psychotic symptoms during their evaluation would develop psychosis in future or not. Hence longitudinal studies are of paramount importance for understanding the natural course of both cognitive impairment and psychosis in patients with PD. Majority of the studies have focused on PD patients with only VH. However, psychosis in few patients with PD may manifest as delusions, illusions, auditory hallucinations and tactile hallucinations. Although it would be interesting to explore the pattern of cognitive impairment specific to these forms of psychosis, their coexistence with VH would make such studies challenging. Minor hallucinatory phenomena (false sense of presence and passage) may precede the onset the VH in many patients. Hence studies investigating cognitive impairments in patients without VH, but with minor hallucinatory phenomena are highly essential, as these studies could possibly allow an earlier identification of patients at risk for VH and/or cognitive deterioration.

Although extensive research has been carried out to identify molecular, genetic and neuroimaging biomarkers for psychosis in PD, the concept of identifying the cognitive biomarkers for psychosis looks promising as they are non-invasive and less expensive. As deficits in executive functions, visuospatial functions and attention are the commonest neuropsychological alterations in patients with PD and psychosis, it would be interesting to carefully follow up the PD patients with deficits in one or more of these three domains for incidence of psychotic symptoms over time. Elucidation of the link between cognitive deficits and psychosis has therapeutic implications as well. As both psychosis and cognitive impairment are speculated to result from alterations in cholinergic and dopaminergic systems [53], these neurotransmitters may become appropriate target for future interventions. As certain drugs such as anticholinergics and dopamine receptor agonists (ropinirole and pramipexole) have the propensity to cause psychosis and cognitive dysfunction, use of these drugs may be closely monitored in patients with impairments of cognitive domains, which are possibly associated with psychosis. Moreover, if specific cognitive impairments are found to be precursors of psychosis, cognitive rehabilitation may play a role in the prevention or delay in onset of psychosis.

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