



# Delusional Misidentification Syndrome in Parkinson's Disease: A Systematic Review and New Approach to our Neurobiological Understanding

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## Abstract

Capgras syndrome (CS) describes a delusional misidentification syndrome whereby a person's loved one or property has been replaced by an imposter. It has commonly been reported in neurodegenerative diseases, but is less frequently reported in Parkinson's disease (PD). We report the case of a 77 year old male with PD, cognitive impairment and increasing visual hallucinations and symptoms of anxiety and depression. He developed CS reporting that his wife was intermittently replaced by an imposter. Whilst symptoms of anxiety and depression responded well to antidepressant treatment, and visual hallucinations responded to rivastigmine, the delusional belief still persists. We perform systematic literature review and identify 18 additional cases of CS in PD. The majority of cases identified describe concomitant cognitive impairment as well as visual hallucinations. Varied pharmacological strategies employed in treating the described patients make it difficult to distinguish the effect of any particular pharmacological intervention of underlying neurotransmitter disturbance underlying the delusion in PD. It is possible that a novel neurobiological mechanism is at play in these patients, and elucidating this further may increase our understanding of the neurobiological basis of the delusional misidentification syndrome.

## Keywords

Capgras syndrome, Cognitive impairment, Parkinson's disease, Visual hallucinations, Neurobiological mechanism

## Clinical Case

A 77 year old male with Parkinson's disease (PD), confirmed by DaTscan was referred to the older adult psychiatry clinic due to concerns regarding his mood and cognition. He was experiencing interrupted sleep and low mood, and his wife reported that during the night time hours the patient failed to recognise her. He had nil past psychiatric history and medical history included benign prostatic hypertrophy and one previous transient ischaemic attack only. He was receiving carbidopa, levodopa, entacapone as

well as ropinirole at maximum tolerated doses and his Addenbrooke's Cognitive Assessment-Revised score (ACE-R) was 79/100. CT head demonstrated normal age related cerebral atrophy with nil other pathology evident. Mirtazipine was commenced and a repeat cognitive assessment an improved ACE-R score at 97/100.

One year later he demonstrated dyskinesia, visual hallucinations of family members and increasing symptoms of anxiety. Ropinirole was reduced and rivastigmine commenced to good effect. Over the following two years he had increasing

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falls and his wife became his primary carer. During this period he frequently described how his wife was sometimes replaced by an imposter taking on the role of his career, who appeared and sounded identical but definitely “was not his real wife”. He was not overtly distressed by this “other wife” and when questioned in clinic as to who this other person might be he remained fixated on the idea that they were an imposter. The delusional misidentification later extended to other family members, namely confusing his daughter and his sister. Interestingly the visual hallucinations responded well to rivastigmine treatment and symptoms of anxiety responded well with addition of venlafaxine. Despite this combination of pharmacotherapy, the delusional misidentification syndrome remains.

### Introduction

PD is a neurological disorder characterised by classical motor symptoms associated with loss of dopaminergic neurons in the substantia nigra and Lewy bodies [1]. In addition, there are a number of non-motor symptoms which have been well characterised, including cognitive impairment, mood disturbance, autonomic dysfunction and sleep disorders. These have not only been attributed to subcortical dopaminergic dysfunction, with large parts of the limbic cortex and neocortex also being implicated [2]. Psychotic symptoms, which include delusional beliefs have been demonstrated to have a prevalence of 15%-30% in patients with PD [3-6].

Capgras syndrome (CS) describes the delusional belief that a person, usually closely related, has been replaced by an imposter or double. The delusion was first reported by J. Capgras in 1923, where his patients described the imposter as someone who looks similar to the original individual in question but was believed to be psychologically absent [7]. Commonly, subtle perceived differences in appearance and behaviour are used to rationalise the false belief and explain why the imposter is a different person to their true relative [8,9]. Whilst usually safe, a number of case reports demonstrate that patients with the delusion can pose a risk to others, with feelings of anger and hostility towards the imposter sometimes leading to violent behaviour [10-12].

This systematic literature review brings together all identifiable case reports of CS in patients with PD, including the most recent case as described.

It aims to outline common themes regarding the nature of the delusional belief in this patient population and place this in context of current neurobiological models of the delusion. It will also make suggestions for further research in this area.

### Methods

The following electronic Internet databases were searched from January 1970 to October 2016: PubMed, Medline, PsycINFO, EMBASE and Cochrane Library (Wiley). The search terms are listed in **Table 1**. Search terms from row 1 were combined with terms from row 2 using the “AND” operator. The titles and abstracts of matched records were screened by the first author according to the following inclusion and exclusion criteria:

#### ■ Inclusion criteria

Articles were included if they were (1) original research or case reports published as a paper, journal letter or conference abstract, (2) patients had a diagnosis of Parkinson’s disease and/or Parkinson’s disease dementia, (3) patients demonstrated symptomatology consistent with CS.

#### ■ Exclusion criteria

Articles were excluded if they were not (1) published in English or (2) were unpublished studies or articles.

### Results

The literature search generated 51 matches, of which 17 articles were deemed suitable based on the above inclusion/exclusion criteria so full text articles requested and reviewed. We were unable to obtain access to 2 articles, and 2 of the remaining 15 demonstrated repeated reporting of the same patients so were excluded. The only cross sectional study identified reported a prevalence of illusions and delusional misidentification of people of 7.3% of 191 patients with PD without dementia seen at a specialist PD clinic [6]. However, it should be noted that this paper failed to provide a more detailed definition for this phenomena or specifically describe the frequency of CS in their study population. The remaining 12 articles in combination of our case report describe 19 cases of CS in patients with PD. The summary of these patient’s demographic and clinical characteristics are outlined in **Table 2**.

**Table 1: Database search terms.**

1)	Parkinson OR Parkinsonian OR parkinsonism OR dopamine OR dopaminergic.
2)	Capgras OR delusional misidentification.

**Table 2: Summary of patient's demographic and clinical characteristics.**

Author, year	Age (yrs) sex (M/ F)	Diagnoses	Cognitive status	Psychiatric symptoms	Imaging findings	Nature of Capgras Syndrome	Pharmacotherapy	Author hypothesis
Roane et al. 1998 [14]	65 F	PD, diagnosed 3 years prior to CS onset.	MMSE=15/30	Visual hallucinations, depression, anxiety.	Nil reported.	Husband replaced by stranger, house did not belong to her.	Levodopa, lorazepam, bupropion, clonazepam, clozapine (diminished CS intensity)	<ul style="list-style-type: none"> <li>Delusional misidentification in PD results from combination of frontal lobe dysfunction and levodopa psychosis</li> <li>Delusional misidentification is likely to be common in patients with advanced PD.</li> <li>Clozapine was effective at reducing misidentification symptoms.</li> </ul>
	75 M	PD (bradykinesia, gait disturbance, nil resting tremor), diagnosed 6-9 years prior to CS onset.	MMSE=22/30	Visual hallucinations, delusional belief wife was unfaithful,	Nil reported	Another woman similar in appearance to wife was entering the house to cook and clean.	Levodopa, pergolide, thioridazine trialled (worsened parkinsonism), clozapine (resolved CS but symptoms returned when discontinued due to sedation)	
Hermanowicz et al. 2002 [18]	73 M	PD (tremor, rigidity, bradykinesia and postural instability), diagnosed 6 years prior to CS. Retinitis pigmentosa (unable to detect light since childhood)	MMSE=17/25	Visual hallucinations	MRI brain – diffuse symmetric cortical atrophy and minimal subcortical white matter ischaemic changes	Multiple imposters (male and female) replaced his wife	Levodopa, pramipexole, quetiapine (nil effect on delusion)	<ul style="list-style-type: none"> <li>Delusional misidentification does not require intact vision and other sensory modalities contribute to this phenomenon.</li> </ul>
Shiotsuki et al. 2010 [19]	64 F	PD (resting tremor, rigidity, bradykinesia, postural instability, wearing off phenomena), diagnosed 6 years prior to CS.	MMSE=20/30, Frontal Assessment Battery=11/18.	Anxiety in off state	MRI brain – slight frontotemporal cortical atrophy. SPECT – hypoperfusion in posterior cingulate cortex, precuneus and prefrontal cortex.	Husband replaced by imposter. Only occurred in the "off state" one hour prior to pharmacotherapy.	Levodopa/carbidopa. Treatment with both agents <b>decreased</b> CS. Nil effect with donepezil.	<ul style="list-style-type: none"> <li>CS is likely not secondary to dopaminergic psychosis, but imbalances in many non-dopaminergic transmitter systems.</li> </ul>
Brooks et al. 2010 [20]	69 M	PD (refractory), with bilateral subthalamic deep brain stimulator insertion. Depression, hyperlipidaemia.	Not reported.	Acute onset agitation and poor safety awareness 4 days post-operatively.	Nil reported.	Acute onset 4 days postoperatively. Wife had been replaced by imposter.	Risperidone reduced intensity of CS to some extent. Delusion resolved within 2 months.	<ul style="list-style-type: none"> <li>Case placed in context of transient post-operative neuro-cognitive changes frequently seen.</li> </ul>
Mori et al. 2011 [21]	4M, 1F; mean age 66.8 (SD not given)	All patients had PD with moderate motor symptoms.	Cognitive impairment in 3 of 5 patients.	5 of 5 had visual hallucinations	Nil reported.	Misidentification of family care giver in all cases.	CS delusion improved with reduced dopamine replacement and/or addition of atypical antipsychotic (medications not specified, cases described collectively)	<ul style="list-style-type: none"> <li>Dopaminergic pathways implicated.</li> </ul>

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Medic <i>et al.</i> 2012 [22]	75 M	PD (tremor, bradykinesia, rigidity), diagnosed 5 years prior to CS.	Mild dementia	Visual hallucinations	MRI brain – mild atrophy and leukoencephalopathy of unclear origin.	Family had been replaced by imposters, several versions of his wife.	Levodopa, rivastigmine + quetiapine improved CS slightly.	<ul style="list-style-type: none"> <li>Nil proposed.</li> </ul>
Cercy <i>et al.</i> 2012 [23]	68 M	PD with severe motor symptoms, diagnosed 11 years prior to CS.  Type 2 diabetes mellitus, hypothyroidism.	MMSE=24/30	Visual hallucinations	CT head – diffuse atrophy and mild microvascular disease.	Wife replaced by imposter, delusion disrupted by wife leaving the room.	Levodopa/carbidopa, midrodine, donepezil. CS remained despite quetiapine initiation.	<ul style="list-style-type: none"> <li>CS could be explained by altered habituation via increased dopaminergic stimulation diminished cholinergic activity.</li> </ul>
Moro <i>et al.</i> 2013 [24]	78 M	PD, moderate motor symptoms, diagnosed 14 years prior to CS.	Dementia (not otherwise specified)	Nil reported	Nil reported	Brother replaced by imposter	Levodopa/benserazide, entacapone, rasagiline, sertraline, rivastigmine. CS improved with decreased dose of levodopa/benserazide, discontinuation of rasagiline and addition of quetiapine.	<ul style="list-style-type: none"> <li>Case placed in the context of previous reports of CS.</li> </ul>
Apaydin <i>et al.</i> 2014 [13]	69 M	PD (tremor, bradykinesia, rigidity), diagnosed 13 years prior to CS. Right pallidotomy performed.	Rapid cognitive impairment post pallidotomy.	Acute onset visual hallucinations post pallidotomy.	Nil reported	Acute onset post pallidotomy, felt like his family members were strangers. Delusional misidentification of house.	Levodopa	<ul style="list-style-type: none"> <li>Combination of excess dopaminergic stimulation of mesolimbic system and frontal lobe impairment implicated.</li> </ul>
	77 M	PD (tremor, bradykinesia, rigidity)  REM sleep behaviour disorder.	Cognitive impairment (not otherwise specified)	Visual hallucinations.	Nil reported	Woman who looked like his wife had replaced her. Delusional misidentification of house.	Levodopa, pridedil and pramipexole.	
Islam <i>et al.</i> 2015 [16]	53 F	PD (confirmed on DAT-scan), diagnosed 3 years prior to CS.  Nil psychiatric diagnosis.	Cognitive impairment demonstrated by MoCA=21	Excessive goal directed behaviours, anxiety.	MRI brain- normal.	Patient's dogs replaced by imposters, cypress trees in garden replaced	Pramipexole, levodopa/carbidopa, quetiapine had no benefit on CS but clozapine did decrease delusion intensity.	<ul style="list-style-type: none"> <li>CS resulted from dopaminergic excess combined with impaired executive functioning.</li> </ul>
Kyrtsos <i>et al.</i> 2015 [15]	78 M	PD diagnosed 7 years prior to CS.  Bilateral DBS stimulation performed.  Previous traumatic brain injury.	MMSE=25/30, plus average scores on a battery of cognitive assessments. MMSE 14/30 6 months post-operatively.	Mild depressive symptoms, sleep disturbance, visual hallucinations.	MRI brain – extensive ischaemic microvascular disease. Normal brain volume.	Delusional misidentification of house following DBS surgery. 6 versions of his wife.	Carbidopa/levodopa, rasagiline (discontinued), amantadine (discontinued). Quetiapine, risperidone and sertraline commenced; improved cognition, anxiety and depression symptoms. Mild effect on CS. Acute psychotic episode halted by turning off DBS.	<ul style="list-style-type: none"> <li>Inadvertent stimulation of frontal associative or limbic circuits could explain CS in this case.</li> <li>Alternatively electrode induced disconnection between frontal lobe and other lobes could have triggered CS in this case.</li> </ul>

Ricciardi <i>et al.</i> 2016.[17]	62 M	PD diagnosed 14 years prior to CS.	MoCA=30/30 initially but severe cognitive impairment at later assessment (assessment score not reported)	Visual hallucinations, nocturnal arousal, anxiety, Othello syndrome.	Nil reported.	Believed his physician had been replaced by imposters when self-titrating levodopa infusion higher than prescribed.	Ropinirole (discontinued due to hypersexuality and compulsive behaviours), levodopa, entacapone, quetiapine added but no benefit reported. Cessation of levodopa infusion lead to CS resolution.	• Dopaminergic induced psychosis in the setting of cognitive impairment.
Our case report	77 M	PD (confirmed on DAT-scan)	ACE-R=79/100, improved to 97/100 with mirtazipine	Interrupted sleep, low mood, anxiety (responded well to venlafaxine), visual hallucinations	CT head age related cerebral atrophy only.	Wife intermittently replaced with an imposter.	carbidopa, levodopa, entacapone, ropinirole (dose reduced), mirtazapine, venlafaxine, rivastigmine. CS did not respond to ropinirole reduction, antidepressant addition or rivastigmine.	

Abbreviations: PD - Parkinson's disease; MMSE - Mini Mental State Examination; MoCA - Montreal Cognitive Assessment; ACE-R - Addenbrooke's Cognitive Examination-Revised; CS - Capgras syndrome; DBS - deep brain stimulation; SD - standard deviation; M - males; F - females.

In 17 of 19 cases CS was a late feature, developing at least 6 years following initial PD symptom onset. Of the 18 cases where cognition had been described, 15 cases of CS occurred in the context of fixed or fluctuating cognitive impairment. This may indicate that cognitive impairment in the context of PD may act as a risk factor for CS development. Where severity of motor symptoms of PD were described these were assessed as moderate or severe in nature, and in 15 out of 19 cases CS syndrome occurred in the context of concomitant visual hallucinations.

Many of the case-reports reviewed did not describe neuroimaging studies, but where such imaging had been reported, diffuse cortical atrophy and mild microvascular disease were most commonly described. The nature of the delusional belief was usually that a loved one, a close relative to the index case had been replaced by an imposter, either permanently or intermittently. Notable exceptions include the two cases reported by Apaydin, *et al.* the first case reported by Roane, *et al.* as well as the case reported by Kyrtsos, *et al.* whereby delusional misidentification of property were described [13-15]. In addition the case of delusional misidentification of animals and inanimate objects described by Islam, *et al.* and delusional misidentification of the patient's physician described by Ricciardi, *et al.* more recently [16,17].

Whilst the majority of case reports describe a gradual onset of delusional beliefs culminating in diagnosis of CS, the two cases of post-operative CS following deep brain stimulation surgery

(DBS) report sudden onset of the delusion within two weeks of surgery and subsequent resolution of the delusion either naturally or following disablement of the DBS device.

### Discussion

Following Capgras' initial report of the delusion, a series of psychodynamic explanations for the syndrome were hypothesised, until in the late 1980s increasing reports of the delusion with co-morbid neurological disease lead to a search for a neurochemical or neuroanatomical explanation. The range of co-morbid diseases described includes epilepsy [25,26], multiple sclerosis [27] and Alzheimer's disease [28]. CS has also been reported in patients with concomitant PD with and without Lewy body dementia [13,14,19,29].

In the mid-1980s researchers investigating models for the cognitive processes underlying facial processing suggested that facial recognition of familiar persons is associated with an effective response which is not seen when viewing the faces of non-familiar people [30]. Work investigating the neurological condition prosopagnosia, an inability to recognise faces, had demonstrated that some patients unable to recognise close family members still demonstrate increased autonomic activity when viewing faces of loved-ones compared to unknown faces, even though neither faces were recognisable. This was evidenced by changes in galvanic skin conductance, a frequently used measure of sympathetic arousal [31,32]. This work lead to the suggestion that two neuroanatomically

independent pathways of facial recognition exist: a pathway of conscious visual recognition and a distinct pathway with mesolimbic connections involved in subconscious emotional recognition [32]. Whilst it was postulated that lesions in the conscious visual recognition system explain the inability to recognise loved ones and preserved galvanic skin conductance in patients with prosopagnosia, researchers in the early 1990s hypothesised that CS was a mirror image of the condition [33]. They suggested that CS could be explained by damage to the subconscious visual recognition pathway with mesolimbic involvement with preservation of the conscious visual recognition system; these patients recognise the faces of their loved ones but do not experience the expected autonomic arousal. These patients may rationalise this decreased autonomic arousal by viewing their loved one as an imposter [34].

When mapping this dual facial recognition pathway model neuroanatomically, Bauer proposed a ventral facial recognition pathway which extended from the visual association area to the temporal lobe and amygdala via the inferior longitudinal fasciculus involved in the conscious perception of faces [32]. He proposed that subconscious visual recognition could be mapped to a dorsal pathway extending from the visual association area to the hypothalamus via the inferior parietal lobe and cingulate gyrus.

More recently, evidence from neuroimaging studies of patients with CS has led to alternative neurobiological models being postulated. A number of researchers noted that right hemispheric lesions are seen more commonly on neuroimaging compared to left sided lesions [35-38]. Due to the fact that CS is associated with abnormalities in facial processing, a right-hemispheric dominant process, it has been suggested that right hemispheric injury or dysfunction may underlie this delusional belief.

Further neuroimaging evidence has also identified that frontal lobe dysfunction may be involved in the pathogenesis of CS, due to high frequency of frontal lobe lesions in patients with the disorder [39-41]. This is particularly evident in evidence from Joseph, *et al.* who demonstrated that patients with schizophrenia and CS had significantly increased frontal atrophy compared to patients without the delusional belief [42]. Since the frontal lobes are involved with conflict resolution it could be argued that in addition to right hemispheric or mesolimbic dysfunction,

additional frontal lobe dysfunction leads to an inability to logically resolve the conflict between the reduced autonomic response experienced when viewing the face of a loved one and the expected response [43,44]. Such frontal lobe involvement could also explain the confabulation of the imposter as a separate, distinct person which is characteristic of the syndrome.

CS and the delusional misidentification syndromes have previously been described in patients with PD. Like many patients described in the literature, the two cases reported by Apaydin, *et al.* demonstrated marked cognitive impairment associated with the psychotic symptoms and dopamine agonists prescribed prior to the delusional belief [13]. Both patients had been treated with levodopa. The authors proposed that a combination of excess dopaminergic stimulation of the mesolimbic system in combination with frontal lobe impairment leads to the delusion.

Cercy and Marasia describe the case of a 68 year old man with PD and combined CS and intermetamorphosis syndrome [23]. This patient had been treated with levodopa and exhibited mild cognitive impairment. His delusional symptoms did not respond to antipsychotic therapy or donepezil, however his wife could successfully terminate the delusion by leaving the room and re-entering. The authors proposed that altered habituation could explain the phenomena, whereby excessive dopaminergic stimulation associated with pharmacotherapy in combination with a reduction in cholinergic stimulation leads to familiar visual stimuli being perceived as novel experiences. They cite the common presence of excess dopaminergic stimulation and decreased cholinergic stimulation in a wide variety of neuropsychiatric disorders as supporting evidence.

Conversely, Shiotsuki, *et al.* argue that dopamine deficiency as opposed to dopamine excess may explain CS in such patients [19]. They report the case of a 64 year old lady with cognitive impairment and PD who exhibited the delusion only in the hour before her L-dopa and carbidopa were due, whereby she developed the belief that her husband had been replaced by an imposter and her motor symptoms worsened. Interestingly the delusion resolved within one hour of medication administration and an increase in total L-dopa and carbidopa dose lead to a complete resolution of the delusion. The authors propose that the delusion in this patient

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was not iatrogenic dopaminergic psychosis as previous authors have postulated. They also cite evidence that dopaminergic loss combined with replacement can lead to widespread disturbances in a number of neurotransmitters.

The idea that non-dopaminergic pathways are implicated in patients CS is also supported in a paper by Speigel, *et al.* who report the case of a 54-year-old woman who developed CS after a right anterior cerebral artery thromboembolic stroke [45]. Her delusion responded significantly to the serotonin 2A receptor antagonist mirtazapine, implicating serotonin excess as an alternative underlying mechanism. It should be noted, however, that this patient did not have a concomitant diagnosis of PD when considering the relevance of these findings in patients with PD who experience a delusional misidentification syndrome.

### Conclusion

CS has been widely reported in patients with neurodegenerative disorders such as Alzheimer's disease and Lewy body dementia, however there are only a selection of case reports of the delusion in patients with idiopathic PD. The case report described here adds valuable information to the 18 case reports published previously of patients with CS and concomitant PD. Whilst nearly all cases of the delusion occur in the context of cognitive impairment, the varied pharmacological strategies employed in treating the described

patients makes it difficult to distinguish the effect of any particular pharmacological intervention of underlying neurotransmitter disturbance underlying the delusion in PD. Whilst both excess and insufficient levels of dopamine have been implicated as potential pathophysiological processes, interestingly in four of the cases identified, dopaminergic therapy modulation had no significant impact on the extent and pervasiveness of the delusional beliefs. Addition of atypical antipsychotics also had varying degrees of effectiveness in reducing the extent of the delusion in the cases described previously. Of further interest, in the case we describe, modulation of medications which effect serotonergic as well as noradrenergic pathways had no effect on the delusional symptoms reported by the patient or his family.

It is possible that a novel neurobiological mechanism is at play in these patients, and elucidating this further may increase our understanding of the neurobiological basis of the delusional misidentification syndrome. Further studies should attempt to study the effects of titrating medications which affect dopaminergic, serotonergic and noradrenergic transmission on the presence and extent of the delusion in a systematic manner. This should not only help clinicians in deciding how best to treat this intrusive and sometimes dangerous symptom in this patient group, but may elucidate neurobiological correlates for the underlying pathogenesis of CS.

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