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Capgras syndrome in Dementia with Lewy Bodies

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Abstract

Background—Capgras syndrome is characterized by the recurrent, transient belief that a person has been replaced by an identical imposter. We reviewed clinical characteristics of Dementia with Lewy Bodies (DLB) patients with Capgras syndrome compared to those without Capgras.

Methods—We identified 55 consecutive DLB patients (11 cases with Capgras syndrome (DLB-C) and 44 cases without evidence of Capgras (DLB). Semi-structured interviews with the patient and an informant, neurological exams, and neuropsychological testing were performed. Caregivers were assessed for caregiver burden and depression. Primary comparisons were made between DLB-C and DLB. Exploratory analyses using stepwise logistic regression and bootstrap analyses were performed to determine clinical features associated with Capgras.

Results—DLB-C patients experienced more visual hallucinations and self-reported anxiety, had higher scores on the Neuropsychiatric Inventory, and were less likely to be treated with cholinesterase inhibitors at time of initial evaluation. Extrapyrarnidal symptoms and depression were not associated with Capgras. Caregivers of DLB-C patients had higher caregiver burden. DLB-C was associated with self-reported anxiety (OR 10.9; 95% CI 2.6-47.6). In a bootstrap analysis, clinical findings that were predictors of Capgras included visual hallucinations (log(OR) 18.3; 95% CI 17.9-19.3) and anxiety (log(OR) 2.9; 95% CI (0.31-20.2).

Conclusions—Our study suggests that Capgras syndrome is common in DLB and usually occurs in the presence of anxiety and visual hallucinations, suggesting related etiopathogenesis. Early appreciation of Capgras syndrome may afford the opportunity to alleviate caregiver burden and improve patient and caregiver outcomes.

Keywords

Lewy body; dementia; Capgras syndrome; caregiver burden; hallucinations; delusions

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DESCRIPTION OF AUTHOR' ROLES

J.Galvin designed the study; P Thaipisutikul, Y Zweig, A Gurnani and J Galvin collected the data, P Thaipisutikul drafted the manuscript, Y Zweig, I Lobach, and J Galvin edited the manuscript, J Galvin and I Lobach were responsible for statistical design and carrying out the statistical analyses.

CONFLICT OF INTEREST DECLARATION

None

INTRODUCTION

Capgras syndrome is characterized by the recurrent and transient belief that a person, usually someone closely related, has been replaced by an imposter. The imposter usually has features that are very similar to those of the original person, although subtle physical differences are used to differentiate the original person from the imposter (Todd *et al.*, 1981; Frazer and Roberts, 1994). Initially, Capgras was described in psychiatric diseases, including paranoid schizophrenia (Merrin and Silberfarb, 1976) and schizoaffective disorder (Haslam, 1973). Capgras syndrome has also been reported in neurodegenerative diseases such as Alzheimer's disease (Cummings *et al.*, 1987) and Dementia with Lewy Bodies (DLB) (Roane *et al.*, 1998). Capgras syndrome in patients without a neurodegenerative disease typically occurs at a younger age and can be associated with psychiatric disease, cerebrovascular events, and illicit drug use (Josephs, 2007). To date, it is unclear whether there are differences between Capgras syndrome as it occurs in neurodegenerative compared with non-neurodegenerative diseases (Josephs, 2007).

The etiology and pathogenesis of Capgras syndrome is not yet fully understood. Capgras has been associated with diffuse brain lesions, focal right hemispheric pathology (Weinstein and Burnham, 1991), and anatomic disconnection of temporal and limbic regions functionally from a damaged frontal lobe (Fienberg and Roane, 2005). Psychodynamic theories posit that Capgras syndrome may be due to anxiety-induced regression of cognitive and emotional functioning, pathological splitting of internalized object representation, inadequately repressed conflicting or ambivalent feeling toward the implicated person, or the projection of negative emotion arising from the conflicting feelings (Bourget and Whitehurst, 2004). However, there is still no single theory or hypothesis that can explain Capgras syndrome in the context of neurodegenerative disease.

Capgras syndrome in DLB nearly always co-exists with visual hallucinations (Josephs, 2007). The prevalence of Capgras syndrome in DLB reported in one study was 16.6% (Harciarek and Ketes, 2008). Half of these cases had an isolated Capgras syndrome and while the other half were accompanied by phantom boarder phenomena (Harciarek and Ketes, 2008). Hallucinations and misperceptions are common in DLB, often associated with Lewy body pathology in the amygdala, parahippocampus and inferior temporal lobe, and may involve disrupted cortical connections between the occipital and temporal lobes (Harding *et al.*, 2002). Prosopagnosia, the inability to recognize faces, is another perceptual disorder common in neurodegenerative disease also associated with occipitotemporal pathology (Barton, 2011). This study reviews the characteristics of DLB patients with Capgras syndrome compared to DLB patients without Capgras syndrome and its potential impact on DLB caregivers.

METHODS

Study Participants

Participants were drawn from a consecutive series of referrals to the Pearl I. Barlow Center for Memory Evaluation and Treatment, a dementia specialty practice at New York University Medical Center, from August 2010 to August 2011. Assessments were completed

by a multidisciplinary team of a neurologist, geriatric psychiatrist, geriatric nurse practitioner, social worker and psychometrician. We identified 11 cases of DLB with Capgras syndrome (DLB-C) and 44 cases of DLB without evidence of Capgras syndrome, evaluated during the study period. No patient-informant dyad contributed more than one observation to the dataset.

Clinical Evaluation

The neurologist conducted independent semi-structured interviews with the patient and a knowledgeable collateral source, usually the spouse or close family member (Carr *et al.*, 2000). The diagnosis of DLB was made based on the clinical interview, physical examination and cognitive evaluation in accordance with the 2005 revised version of DLB Consortium Criteria for the clinical diagnosis of probable DLB (McKeith *et al.*, 2005). The diagnosis of Capgras syndrome was made based on a clinical interview with the informant in accordance with the Diagnostic and Statistical Manual – IV TR (American Psychiatric Association, 1994).

The Clinical Dementia Rating (CDR) was used to determine the presence or absence of dementia and to stage its severity (Morris, 1993). The CDR rates cognitive function in each of six categories (memory, orientation, judgment and problem solving, and performance in community affairs, home and hobbies and personal care). A global CDR 0 indicates no dementia. CDR 0.5 represents questionable or very mild dementia; CDR 1, 2, or 3 corresponds to mild, moderate, or severe dementia (Morris, 1993). The sum of CDR boxes (CDR-SB) provides a quantitative expansion of the CDR ranging from 0 (no impairment) to 18 (maximum impairment) (Morris, 1993).

All patients underwent clinical neuroimaging with non-contrast MRI or CT. These were not research-grade scans and no quantitative measurements are available. The senior author (JEG) reviewed all films to provide a qualitative rating of atrophy, vascular burden, and white matter disease. These variables were not included in the analyses since images were collected in a non-standardized fashion.

Caregiver Evaluation

Psychosocial evaluations were completed by each caregiver to determine the presence of non-cognitive symptoms observed in the patient and to assess the impact of these symptoms on the caregiver. The Neuropsychiatric inventory (NPI-Q) (Kaufer *et al.*, 2000) assessed behavior and mood, the Mayo Sleep Questionnaire (MSQ) (Boeve *et al.*, 2011) assessed the presence of parasomnias (including REM sleep behavior disorder), the Mayo Fluctuation Questionnaire (MFQ) (Ferman *et al.*, 2004) assessed the presence of cognitive fluctuations, and the Epworth Sleepiness scale (Johns, 1991) assessed daytime sleepiness. In addition, caregivers completed the Zarit Burden Inventory (Herbert *et al.*, 2000) to evaluate for caregiver burden and the PHQ-9 (Kroenke *et al.*, 2001) to assess caregiver depression.

Neuropsychological Evaluation

Each patient was administered a 30-minute test battery at the time of the office visit to assess their cognitive status. The psychometrician was unaware of the diagnosis, presence or

absence of Capgras or the CDR. A brief global assessment was performed using the Mini Mental State Exam (MMSE) (Folstein *et al.*, 1975). The test battery also included measures of episodic memory (Wechsler Memory Scale Logical Memory-Story A (Wechsler, 1987); Hopkins Verbal Learning Task – immediate and delayed recall and cued recognition (Brandt *et al.*, 1992); semantic memory (Animal Fluency (Troyer *et al.*, 1998); 15-item Boston Naming Test (MacKay *et al.*, 2005); and working memory (Digit span forward and backward task (Conway *et al.*, 2005). Three timed measures addressed psychomotor, visuospatial and executive abilities: Wechsler Adult Intelligence Scale Digit Symbol (Wechsler, 1955), and Trailmaking A and B (Reitan, 1958). Construction was assessed with the Clock Drawing Task (Borson *et al.*, 1999). Mood was assessed with the Hospital Anxiety Depression Scale (Snaith, 2003) which provides subscale scores ranging from 0-21 with scores greater than 7 supporting the presence of depression or anxiety. Not all patients were able to complete all parts of the battery and few patients were able to complete the Trailmaking B tasks (not shown in results).

Statistical Analysis

Statistical analysis was performed using IBM SPSS v20 (Armonk, NY) and R program for Statistical Computing (<http://www.r-project.org/>). Descriptive statistics were used to report the demographic and clinical characteristics of the patients, informants' ratings, CDR, CDR-SB, and neuropsychological test results. Independent sample t-tests were used for continuous data and Chi-square analyses were used for categorical data. Primary comparisons were made between 11 DLB-C cases and 44 DLB cases without Capgras; p-values less than 0.05 were considered statistically significant. We also examined the possible interactive effects between anxiety and antidepressant use, hallucinations and cholinesterase inhibitor use, and hallucinations and neuroleptic use; none were significant. In an exploratory fashion, we examined the clinical characteristics associated with Capgras syndrome, accepting an elevated false positive rate ($p < 0.2$) in the modeling. Two statistical models were performed. First, stepwise logistic regression was performed with variables with p-values < 0.2 to determine which clinical features predicted the presence of Capgras with odds ratios (OR) reported. Logistic regression analysis is based on an assumption that the asymptotic distribution of parameter estimates is Gaussian. However, we anticipated that the finite sample distribution of $\log(\text{OR})$ estimates based on our small sample might not be Gaussian. Therefore, we also performed nonparametric bootstrap analyses to approximate the distribution of parameter estimates by an empirical distribution of the observed data reporting the $\log(\text{ORs})$ and confidence intervals. We examined variables with p-values < 0.2 based on 1,000 bootstrap samples.

RESULTS

Patient Characteristics

Fifty-five patients are included in this report with a mean age of 75.22 ± 7.4 years and 15.2 ± 3.4 years education. Men comprised 64% of the sample, 71% were married and 85% were right-handed. Informants included spouses (69%), adult children (24%) or paid caregivers (7%). Demographic data from 11 DLB-C and 44 DLB cases are shown in Table 1. There was no difference in age or education between DLB-C and DLB patients. Patients with Capgras

were more likely to have visual hallucinations ($p=.01$). Global dementia ratings were not different between DLB-C and DLB. There were no differences between groups on any of the neuropsychological tests.

Behavioral Ratings

We found that the DLB-C group had significantly higher scores on the NPI-Q (15.7 ± 4.8) compared to the group without Capgras (11.6 ± 5.7 , $p=.03$). DLB-C patients endorsed more symptoms of anxiety on the Hospital Anxiety and Depression Scale ($p=.05$) but no differences were seen in self-reported depression symptoms. Caregivers of patients with Capgras syndrome had higher caregiver burden on the Zarit Burden Inventory (26.2 ± 10.1) compared with caregivers of patients without Capgras syndrome (19.3 ± 8.4 , $p=.02$).

Medications

Medication usage by patients is documented in Table 2. At the time of initial evaluation, 80% of patients had been started on cholinesterase inhibitors, however DLB-C patients were more likely to have discontinued their use ($p=0.02$). No differences were noted for NMDA-receptor antagonists, antipsychotics, antidepressants, benzodiazepines, or dopaminergic agents.

Clinical Features Associated with Capgras Syndrome

Exploratory analyses were conducted to examine clinical features associated with the presence of Capgras. In a stepwise logistic regression using clinical variables that showed trends (p -values <0.2), the only clinical feature associated with Capgras syndrome in DLB was self-reported anxiety (OR 10.9; 95% CI 2.6-47.6). Bootstrap analyses demonstrated that visual hallucinations (log(OR) 18.3; 95% CI 17.95, 19.28) and self-reported anxiety (log(OR) 2.9; 95% CI (0.312, 20.24)) were significantly associated with Capgras ($p<0.05$), while REM sleep behavior disorder (log(OR) 1.67; 93% CI (0.02, 2.34)) and non-usage of cholinesterase inhibitors (log(OR) 1.73; 93% CI (0.025, 3.28)) were marginally associated with Capgras in DLB ($p=0.07$).

DISCUSSION

We found that Capgras syndrome was common in DLB. DLB-C patients were more likely to have visual hallucinations, their caregivers tended to report more symptoms of cognitive fluctuations and REM sleep behavior disorder, and patients reported more symptoms of anxiety. The presence of Capgras significantly increased caregiver burden and led to the caregiver (typically the object of the delusion) to express more depressive symptoms. The single best predictor of Capgras in DLB was the presence of anxiety with a 10-fold increased risk. While DLB is typically more common in men (Rosenberg *et al.*, 2001), we found no gender preference for Capgras.

The clinical features of DLB include cognitive fluctuations, recurrent visual hallucinations and spontaneous features of parkinsonism (McKeith *et al.*, 2005). We found a strong relationship between Capgras and visual hallucinations; however there was no relationship between the presence of parkinsonism and Capgras. These data agree with a previous study

that also found 100% co-occurrence of visual hallucinations and Capgras syndrome in DLB (Josephs, 2007). Our results suggest that visual hallucination and Capgras syndrome may have a related etiopathogenesis.

It is not surprising that the DLB-C had higher NPI-Q scores. The NPI-Q covers a range of neuropsychiatric and behavioral symptoms, and Capgras syndrome has been associated with several neuropsychiatric symptoms, including delusions, anxiety, irritability, agitation, and motor disturbances (Cummings *et al.*, 1987; Josephs, 2007). Indeed, patient self-reported anxiety was the strongest feature associated with the presence of Capgras. As a result, the caregiver who is often the target of the delusion and spends most of the time with the patient, reports significant burden and emotional strain related to this behavior as demonstrated in the higher scores on the Zarit Burden Inventory.

DLB caregivers experience significant burden and distress. Caregivers reported the highest burden when describing the impact of providing care on their personal lives, health, and emotional well-being with spouses reporting higher burden than non-spouse caregivers (Galvin *et al.*, 2010a). Three dimensions of burden in DLB caregivers have been described: role strain, personal strain, and worry about performance (Leggett *et al.*, 2011). We previously reported that DLB caregiver burden correlated strongly with mood (anxiety, depression and apathy), delusions, hallucinations and sleep disturbances (Galvin *et al.*, 2010a; Leggett *et al.*, 2011). We also previously reported that increased caregiver burden led to feelings of social isolation and lack of understanding and adequate support from family, friends, or healthcare providers (Galvin *et al.*, 2010a). Indeed, the diagnostic challenge of DLB may only heighten this sense of isolation and burden (Galvin *et al.*, 2010b).

We did not find differences in neuropsychological performance across semantic, episodic, or working memory, attention, psychomotor, executive, visuospatial or constructional praxis in DLB-C patients compared with DLB without Capgras. This conflicts with a previous report that greater impairment in executive abilities is usually present in Capgras patients (Bourget and Whitehurst, 2004). Global dementia ratings as measured by the MMSE, CDR or CDR-SB were not different between groups. It should be noted however, that few patients were able to complete the more complex tasks of the battery such as the Trailmaking B (only 17/55 could attempt the task).

Our study has limitations. The small sample may limit the power to detect statistical differences on clinical features using parametric techniques. We attempted to correct for this by performing bootstrap analyses. Both models (Logistic Regression and Bootstrap) support the main group effects demonstrated in Table 1 that visual hallucinations and anxiety are key clinical features associated with Capgras and that we did not miss other associated features because of a lack of power. The patients were referrals from community practices to a specialty clinic and the senior author is a recognized expert in the area of DLB. This may have led to a referral bias with more severely affected patients or those refractory to usual care being included in this study. However, we chose to use consecutive patients to limit the effects of referral bias and the extensive work-up demonstrated that the groups were well balanced in terms of demographic characteristics, neuropsychological performance and dementia severity (median CDR 1, median MMSE 21 suggesting most patients were in mild

to moderate stage of disease). The treatment of Capgras syndrome in DLB patients may be complicated because DLB patients may have adverse reactions to neuroleptic medication (McKeith *et al.*, 2005). Cholinesterase inhibitors are frequently used and may possess some ability to reduce psychiatric symptoms (Bourget and Whitehurst, 2004) although in the present study this course of action was apparently not well tolerated. A final limitation is the absence of biological markers of disease. It is possible that DLB-C has a different pathologic burden than patients without Capgras such as increased cerebrovascular disease or concurrent Alzheimer's disease, although qualitatively there were no differences noted on patients' clinical imaging studies in this study.

Caregivers of DLB patients experience significant burden that may be heightened by cognitive, functional and behavioral problems, a sense of isolation, and challenges with the diagnostic experience (Galvin *et al.*, 2010a; 2010b; Leggett *et al.*, 2011). Capgras syndrome, when present, significantly increases caregiver burden and distress. Early appreciation of DLB and Capgras syndrome may afford the opportunity to alleviate caregiver uncertainty, permit intervention by using both pharmacologic and non-pharmacologic approaches, and allow for future planning for the long-term care of patients and improving the well-being of their caregivers.

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Table 1

Patient Characteristics

Variable	DLB-C	DLB	p-value ^a
<i>Patient Characteristics</i>			
Age, y	77.1 (8.5)	74.7 (7.1)	.35
Education, y	16.4 (3.9)	14.8 (3.3)	.16
Gender, % male	63.6	63.6	1.0
Marital status, % married	81.8	68.2	.52
Family history of dementia, %	18.2	29.5	.71
Right-handed, %	81.8	86.4	.84
<i>DLB Clinical Features</i>			
Parkinsonism, %	90.9	88.6	1.0
Visual hallucinations, %	100.0	61.4	.01
Cognitive fluctuations, %	63.6	40.9	.19
REM sleep behavior disorder, %	72.7	47.7	.18
<i>Dementia Ratings</i>			
Mini-mental state exam	19.7 (5.2)	19.3 (6.7)	.87
Clinical dementia rating	1.6 (0.7)	1.5 (0.8)	.64
CDR Sum of boxes	10.6 (3.6)	9.0 (4.5)	.23
<i>Behavioral Ratings^b</i>			
Neuropsychiatric inventory	15.7 (4.8)	11.6 (5.7)	.03
Epworth sleepiness scale	8.9 (6.3)	6.8 (5.0)	.25
HADS-Depression, %	21.1	20.0	.61
HADS-Anxiety, %	31.8	8.3	.05
<i>Neuropsychological Performance</i>			
Animal naming	8.8 (4.8)	9.5 (5.3)	.71
15-item Boston naming	10.7 (1.9)	9.6 (3.5)	.35
WMS Logical Memory –Story A	5.1 (3.9)	3.7 (2.9)	.35
HVLT- Total recall	8.3 (5.2)	9.6 (4.8)	.48
HVLT-Delayed (free) recall	.78 (1.7)	.73 (1.8)	.94
HVLT-Cued recognition	4.3 (3.5)	5.5 (3.6)	.48
Digit Span Forward	5.9 (1.6)	5.5 (1.5)	.55
Digit Span Backwards	2.6 (1.3)	2.9 (1.4)	.42
Trailmaking A	123.0 (42.9)	111.7 (50.8)	.55
WAIS Digit Symbol	19.7 (11.7)	20.2 (11.6)	.94
<i>Caregiver Ratings</i>			
Zarit Burden Inventory	26.2 (10.1)	19.3 (6.7)	.02
PHQ-9	5.8 (5.8)	3.6 (3.8)	.21

Means (SD)

Key: DLB-C: Lewy body dementia with Capgras syndrome; DLB: Lewy body dementia without Capgras; mini-PPT: Mini Physical Performance Test; CDR: Clinical Dementia Rating; PHQ: Personal Health Questionnaire; WMS: Wechsler Memory Scale; HVL: Hopkins Verbal Learning Task; WAIS: Wechsler Adult Intelligence Scale

^a p-values derived from t-tests for continuous variables and Chi-square tests for categorical variables;

^b Neuropsychiatric Inventory and Epworth Sleepiness Scale are completed by caregiver; HADS is completed by the patient.

Table 2

Medication Usage

Medication	DLB-C	DLB	p-value ^a
Cholinesterase inhibitor, %	54.5	86.4	.02
NMDA receptor antagonist, %	36.4	47.7	.49
Antipsychotic, %	31.8	27.3	.77
Antidepressant, %	36.4	56.8	.22
Benzodiazepine, %	15.9	9.1	.57
Dopaminergic agent, %	36.4	38.6	.89

Key: DLB-C: Lewy body dementia with Capgras syndrome; DLB: Lewy body dementia without Capgras; NMDA: N-methyl-d-aspartate

^a Chi-square