



SCIENTIFIC LETTER

Capgras syndrome as a psychiatric manifestation in Parkinson's disease: A case report and literature review



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Abstract We present the case of a 58-year-old male patient who had symptoms of anxiety after witnessing a case of social violence in his community in 2005. After that, he presented symptoms of Parkinson Disease and in 2006 we established this as the main diagnosis. In 2009 he presented neuropsychiatric symptoms such as apathy, anhedonia, social isolation, blunted affect, visual and auditory hallucinations, paranoid delusions, soliloquies, and the false belief that his wife and daughter had been replaced by identical impostors. We established the diagnosis of Capgras Syndrome. This case is clinically relevant because of the presentation of its symptoms, its evolution and its presenting comorbidity.

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Introduction

The ability to recognize faces and discriminate those which are not familiar to us is there since birth, and it is developed

and perfected through the first years of life. Delusional misidentification syndromes are a group of phenomena in which patients erroneously identify familiar people or places or even themselves, believing said objects or people to have been replaced or transformed. There are several subtypes, however, most authors divide it into 4 main syndromes (Capgras syndrome, Frégoli syndrome, Intermetamorphosis syndrome and syndrome of subjective doubles), Capgras syndrome being the most common among these syndromes.¹

Joseph Capgras described the first case of a syndrome characterized by the delusional belief that a close relative

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has been replaced by an impostor. In the beginning, it was believed to be associated to a higher degree with psychiatric disorders, but in fact it is more frequently associated with neurodegenerative diseases, especially with Lewy body disease, and it is commonly found in association with visual hallucinations.² In the case of Capgras syndrome, there is not only the misidentification of someone familiar to them, but a delusional belief that the person has been replaced by a double that is not quite exact. Study of the causes has been closely linked to the analysis of patients with brain injuries which would have developed prosopagnosia.³ This perceptual disorder causes the patient to be unable to recognize faces in a consistent way, despite the fact that he/she is able to recognize visual objects.⁴

Continuing along the lines of the study of Capgras syndrome, starting from the prosopagnosia phenomenon, it has been suggested that there would be a "mirror image" of prosopagnosia, in a way that, although the conscious ability to recognize faces would be intact, there would be damages to the system that produces automatic emotional excitement when observing familiar faces.⁵ This finding was later confirmed in patients with a Capgras syndrome diagnosis.

Traditionally, the origin of Capgras syndrome was considered to reside in a psychodynamic base; the premise was that people who received a great deal of affection from the patients were also recipients of their ambivalence, which made them the object of the projections and led to the patient's psychological disintegration.

On the other hand, in addition to prosopagnosia, Capgras syndrome has been linked with multiple pathologies. It has been described in psychiatric as well as organic disorders. Within psychiatric profiles, it is common to find this delusion in paranoid schizophrenia, as well as psychotic depression, delusional disorders, schizoaffective disorder or bipolar disorder, with a frequency of 1–15% in psychotic patients.

Regarding the organic conditions that occur in Capgras delusion, this appears mainly in various types of dementia like Alzheimer, Lewy bodies and Parkinson, as well as posterior to brain injuries. It is followed among other conditions by ictus, epilepsy, chronic alcoholism cases and encephalitis.⁶

Capgras syndrome has been considered a type of paranoid psychosis more frequently linked to Lewy body dementia, as well as psychiatric illnesses like schizophrenia. Nevertheless, there are reports in medical literature that even link it to dopaminergic deficiency, which appears during the "off" states, solving it with an increment in L-dopa and less frequently in an isolated form as a psychiatric manifestation of Parkinson's disease.⁷ Below, we describe a peculiar case of a patient who presented Parkinson's disease and Capgras syndrome simultaneously.

Clinical case

The patient is a 58-year-old male with a history of systemic arterial hypertension (since he was 45 years old), without a history of any other major health issue. His symptoms began in 2004 with a right upper limb tremor which did not affect his everyday life, and thus was not given importance. In 2005 he witnessed a situation of social violence having been caught in a crossfire and having his workplace

attacked with a grenade. As a result, he began displaying the following symptoms: anxiety, fear, recurrent memories of the event, affective bottling, hypervigilance and avoidance behavior, with a significant decline in his work and social activity. He was diagnosed with acute stress disorder and began treatment with 50 mg of sertraline every 24 h, with a remission of symptoms. Subsequently, other symptoms characterized by bradykinesia appeared, with an increase in the right upper limb rest tremor, fine motor clumsiness and gait and posture alterations being asymmetrical with uncoordinated arm movement. A Parkinson's disease diagnosis was established in 2006. In 2009 the patient displayed neuropsychiatric signs and symptoms, apathy, anhedonia, a tendency to isolation, affective bottling, and later aural and visual hallucinations with paranoid content, soliloquies, as well as a false belief that his wife and daughter were not them but had been replaced by other people, thus integrating a Capgras syndrome.

The following clinimetrics were performed: The Schwab & England Activities of Daily Living Scale, which evaluates the patient's functional state, with a value of 10%, meaning the patient is "Totally dependent, harmless; completely handicapped". Hoehn and Yahr evaluation scale, a descriptive staging scale of the progression of Parkinson's disease symptoms, placing the patient in a stage 4: Severe disability; still able to walk or stand unassisted. Freezing and "on-off" unpredictable (Stages 1–5, where stage 1: minimal or no functional disability and stage 5: the patient is confined to a bed). Montreal Cognitive Assessment battery (MoCa) evaluates mild cognitive impairment, with a score of 11, corresponding to a moderate cognitive impairment (normal range of 24–30). Hamilton anxiety rating scale of 10 (mild anxiety). Hamilton depression scale with 5 points (not depressed). Folstein Mini-Mental State Exam with 17 points, corresponding to moderate impairment.

Treatment was begun with levodopa/carbidopa 250 mg/25 mg orally, ½ pill every 6 h, and amantadine 100 mg orally ½ tsp every 8 h with a positive control of Parkinsonism symptoms. Regarding psychotic symptoms, we began treatment with quetiapine 150 mg with positive results. Currently the patient continues with the same treatment and is stable, with good control of Parkinson's symptoms, in addition to not presenting current active psychosis.

Discussion and conclusions

The presence of hallucinations and delusions is frequent in Parkinson's disease, affecting around 20–30% of the patients.⁸ Psychotic symptoms have been linked to the disease before there were treatments for it, but its prevalence has considerably risen since the generalization of the use of dopaminergic stimulation.⁹ There also have been reports of the presence of transient neurocognitive changes, secondary to the placement of a deep cerebral stimulator for resistant Parkinson's, causing symptoms characteristic of Capgras syndrome.¹⁰

One of the most relevant intrinsic factors in the development of psychosis is the cholinergic deficit. Other precipitant and risk factors linked to the disease itself are the age of onset, length, presence of depression and

especially the presence of cognitive impairment, because Capgras syndrome appears in the later stages of dementia in Parkinson's, which results in stressing situations for the patients and even more for the caregivers.¹¹

The most important factor is the direct and indirect stimulation of the corticolimbic dopaminergic receptors. In fact, these phenomena may appear even in patients being treated with L-Dopa or dopaminergic agonists in early stages, although its frequency increases considerably in more advanced patients and in patients with complications, especially if they develop dementia. Recent processing models highlight the dissociation between recognition and emotional response, based – from the neurobiological point of view – on the disconnection between the frontal lobe and the right temporal and limbic regions, along with the bilateral frontal damage. Some theories suggest a two-way intervention as an explanation, one lateral non-cortical and the other subcortical, located in the right hemisphere.¹²

Evidence that not all patients with right hemisphere affection develop a delusional syndrome has led to suggest the presence of a positive mechanism creator of delusions, which would require at least a certain preservation of the left hemisphere. This usually acts to inhibit the right side; it tries to give coherence and sense and interpret dissonant information between recognition and absence of familiarity, thus its affection would lead to delusion.¹³

Moreover, the monothematic delusion two-factor theory, among which there is the Capgras syndrome, suggests that the genesis is caused by a double failure: a right hemisphere injury, probably in the ventromedial zone of the right-frontal lobe, in addition to the failure of the beliefs comparison mechanism, which produces delusion persistence in spite of evidence against it, sustained also by the right frontal lobe.¹⁴ The positive mechanism in this case would be the beliefs evaluation system, similar to the reality monitoring functions.¹⁵

On the other hand, psychiatric manifestations of Parkinson's disease are: depression and anxiety. Mood disorders are probably Parkinson's disease's most frequent symptoms, so much that they may precede motor symptoms in up to 30% of cases.¹⁶

Significant depression prevalence presents itself in more than double the number of patients with Parkinson's (37%) than what is normally found in other diseases (18%). Less severe forms of depression are more frequent than major depression, however; the latter occurs in 5% of patients. Despite everything, the depressive symptomatology displayed in this context goes beyond the simple expected reaction caused by suffering an upsetting disease and being mostly disabled to a greater or lesser degree. Changes in the serotonergic system play a major role, a link with noradrenergic transmission, based on the presence of a deficit of noradrenergic neurons in the locus coeruleus. There is a dopamine deficit in mesocortico-limbic projections and of dopaminergic cells in the ventral tegmentum in depressed parkinsonians.¹⁷

The anxiety disorders are manifested through agitation, chronic anxiety, panic attacks and obsessive-compulsive disorders. The pathogeny of anxiety is explained by the increment caused by levodopa of the catecholamine and its metabolites of the brain. It is usually linked to the amount

and duration of treatment and it is more frequent in the "off" periods.¹⁸

Chronic anxiety can be an exclusive manifestation of the "off" periods and may be present during the whole day. Due to therapeutic implications, it is important to determine this symptom's presentation profile, because in the case of being secondary to the increment of catecholamine by levodopa, dopaminergic treatment must be adjusted, in addition to applying psycho-pharmaceuticals.¹⁹ Panic attacks are usually linked to prolonged treatments with levodopa. They constitute a rare complication and occasionally occur in concurrence with the "off" periods of levodopa dependents, much later than dyskinesia and motor fluctuations.

In this case, the patient has presented the three most frequent types of psychiatric manifestations, although what draws attention has been the Capgras syndrome, because this syndrome constitutes a complex process that is not limited to a simple facial processing problem, but a multiple cerebral dysfunction which sustains it, since it may become chronic. Due to the high frequency of the psychiatric manifestations of Parkinson's disease, linked or not to medical treatment, it is important to know the syndromes associated with the "off" periods of the disease and to know how to tell them apart from the medications' side effects and psychotic symptoms of other origin in order to implement the proper treatment, because it impacts quality of life and its functional prognosis. Clinical interviews are basic for establishing the order of the symptoms.

Conflict of interest

The authors have no conflicts of interest to declare.

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