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CASE REPORT

Capgras syndrome in a very late onset, treatment resistant schizophrenia

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ABSTRACT

We report a Malay man, with underlying chronic medical illnesses, presenting with positive symptoms of schizophrenia, including Capgras syndrome (CS) at the age of 73. Physical examination and blood investigations were normal and brain CT scan showed age-related cerebral atrophy. Neuropsychological assessment showed probable right hemisphere lesions but relatively intact memory and intellectual functions. Several neuroleptics including depot injections were tried but ineffective. Positive symptoms including CS eventually improved with clozapine before his death from myocardial infarction. This case report highlights the uncommon occurrence of CS in treatment resistant schizophrenia (TRS) of very late onset and its treatment challenges.

Key words: Capgras syndrome, elderly, treatment resistant schizophrenia, very late onset schizophrenia

Introduction

CS also known as ''illusion des sosies'' or ''illusion of doubles'' (Sadock and Sadock, 2008), is characterized by a delusional belief that someone significant or well known to an individual, has been replaced by an imposter with a strong physical resemblance (Todd *et al.*, 1981). CS has been associated with organic illnesses and neurological disorders. It occurs in up to 4% of psychotic patients and has been observed mostly in schizophrenia, especially of the paranoid subtype (Kirov *et al.*, 1994).

This case report illustrates the rare occurrence of CS in a patient with treatment resistant, very late onset schizophrenia (VLOS), and challenges in its treatment.

Case report

Mr. M, a retired 76-year-old Medical Assistant was first hospitalized in his hometown in 2009 following behavioral changes of a month duration. He was later referred to Universiti Kebangsaan Malaysia Medical Center in May 2010 when his symptoms did not remit. He experienced second and third person auditory hallucinations, such as "M is a gifted Prophet." There were no other Schneiderian First Rank symptoms.

He was often seen to be talking to himself, which he attributed to having private conversations or "teletalk" (a form of neologism) with famous world leaders. He was hostile towards his wife whom he believed had conspired with their children to withdraw all his bank accounts worth millions of dollars. Besides grandiose delusions, there were no other features of mania. He also presented with CS, in which he insisted that all his family members had died and had been replaced by doubles. There was no decline in his cognitive functions or daily functioning. There was no past psychiatric history or any identifiable psychosocial stressors contributing to the illness.

On admission, mental state examination showed an overweight elderly man with poor rapport, blunted affect and euthymic mood, looseness of associations, persecutory and grandiose delusions, and auditory hallucinations. He exhibited hallucinatory behavior of mumbling to himself, which he clarified as having a friendly "teletalk" with the Queen of England. There were delusions of his real family being replaced by impostors. Depressive and manic symptoms could not be elicited. His judgment was impaired and insight of illness poor. Cognitive testing with Mini-Mental State Examination (MMSE) was within normal range

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with a score of 29/30. Repeated MMSE done during his hospitalizations did not show any significant changes in the scores to suggest any acute or chronic organic brain disorders. Complete physical examinations, including neurological examination, visual and hearing tests were unremarkable.

Mr. M had been on treatment for diabetes mellitus type 2, hypertension and hyperlipidemia for more than 20 years. In the ward, his blood pressure and blood sugar levels were fairly stable, within the range of 110–140/ 70–90mmgHg and HbA1c of 5.1–6.0%. His lipids profile showed only slight rise in LDL level. His latest prescriptions on admission were gliclazide 40 mg bd, metoprolol 25 mg bd, amlodipine 10 mg od and lovastatin 2 mg nocte. However, his renal profile showed raised urea and creatinine levels despite being asymptomatic of any urinary or constitutional symptoms to suggest acute or chronic renal disease. Abdominal x-ray and ultrasound did not show any renal or liver pathology. He was diagnosed by the nephrology team to have Stage 3 renal disease, not requiring dialysis.

Except for low hemoglobin of 11.8 g/dL, his total white cell count, thyroid function test, folate and vitamin B12 levels, RPR and HIV screenings were within normal limits. Brain computerized tomography (CT) scan showed presence of age-related cerebral atrophy. Neuropsychological tests revealed equivocal findings of probable right temporal or frontal lesions based on his low performance on the similarities subtest of the Wechsler Adult Intelligence Scale (WAIS-III^{UK}). Overall his intelligence and memory functions were intact.

Treatment was a challenge in this patient. Initially, quetiapine was prescribed in slow increment up to 800 mg daily. Later, haloperidol was added as an adjunct, up to 5 mg bd. Due to poor response, both anti-psychotics were tailed off and replaced with aripiprazole ten daily. Later on, risperidone 3mg was added as a combination therapy. All his oral medications were later replaced with intramuscular risperidal consta 37.5 mg two weekly as patient was not compliant to his oral medications upon discharge from hospital. Over a span of more than a year, he had four admissions for aggressive tendencies. His psychotic symptoms finally improved after his medication was changed to clozapine. He was beginning to accept his "fake family." During his latest hospital admission, he was closely monitored with weekly hematology investigations and ECG monitoring which showed no abnormalities. Unfortunately, Mr. M died of an acute, extensive, anterior myocardial infarction in March 2012, during the ninth week of treatment with clozapine at a dose of 225 mg daily.

Discussion

CS was initially reported to be associated with psychiatric illness (schizophrenia of paranoid type) but over the years, reports of its association with neurological disorders especially in neurodegenerative diseases have increased (Burn *et al.*, 1987). Therefore, it is crucial to rule out a possible organic etiology when the onset is at an older age. This case report describes an elderly man presenting with CS and positive symptoms of schizophrenia for the first time in late life. A provisional diagnosis of Schizophrenia was made based on the absence of cognitive impairment from history and mental state assessments which could not support a diagnosis of neurocognitive disorder.

Interestingly, his neuropsychological assessment showed an average memory function and a probable right hemisphere lesions (right temporal and/or right frontal region) which were not detected in his brain CT scan. These findings correlated with studies emphasizing the importance of bilateral frontal pathology in patients with CS (Alexander MP *et al.*, 1979). It is also said that the frontal/executive and memory deficit has a role in delusional misidentification syndrome (Staton *et al.*, 1982). Schizophrenia has long been referred to as "a left hemisphere disorder" (Flor-Henry *et al.*, 1976) and neuroimaging has confirmed presence of small and subtle brain abnormalities in schizophrenia (Shenton *et al.*, 2001).

There were continuing concerns about the use of neuroleptics, combination therapy and depots and their potential cardiovascular and cerebrovascular complications in this patient, who had concomitant chronic medical illnesses. As Mr. M met the Kane criteria of TRS, clozapine was commenced after full cardiac screening. Clozapine is known to be relatively safe and well tolerated in elderly psychiatric patients (Barak *et al.*, 1999), hence supporting the rationale use of clozapine in this patient.

Most of his psychotic symptoms eventually improved after a few weeks of clozapine, with decline in the brief psychiatric rating scale (BPRS) scores from 64 to 27. It was unfortunate that patient passed away before the optimal dose of clozapine could be achieved. The improvement in positive symptoms with clozapine helps support the diagnosis of TRS in this patient.

However, there were several limitations in this report. Postmortem assessment was not conducted as his family had refused permission for one. An autopsy study conducted by Josephs *et al.*, (2007) on two deceased subjects with CS showed pathological changes consistent with Alzheimer's disease and Lewy bodies dementia. A brain magnetic resonance

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imaging (MRI) was not also done as his family could not afford to pay for it.

Description of authors' roles

M. K. Nurul Ain treated this patient and wrote this report. R. Rosdinom assisted in patient's management and revision of this report while M. Raynuhar conducted the neuropsychological assessment.

Conflict of interest

None.

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