Delusional Misidentification Post–Traumatic Brain Injury Responding to Clozapine

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In this interesting case, a patient with a traumatic brain injury (TBI) later developed Capgras delusions. The patient had supersensitivity to multiple antipsychotic trials with no response. When shifted to low-dose clozapine, his delusions remitted entirely with great tolerability. This case might open new treatment venues for complicated clinical scenarios of organic psychoses.

Case Report

A 24-year-old Kuwaiti man was escorted by his elder brother for paranoid ideations of 3-month duration. He is the youngest of 5 siblings of a nonconsanguineous monogamous family with no family history of neuropsychiatric disorders. He had unremarkable developmental trajectories with an outstanding scholastic achievement. He previously worked as a police officer. He was a smoker but had no history of illicit drug use. He had no medical history of note. He had a motor vehicle accident 1 year ago, incurred a severe TBI, and was admitted to the intensive care unit with loss of consciousness and serial seizures. He was discharged on valproate 1,500 mg/d. He was doing well following this accident, as reported by his parents, apart from occasional complaints of low-grade headaches, giddiness, and poor concentration. Lately, he started to display nonsensical talk that his family had been replaced by imposters (Capgras delusions) and were coming after him. He was panic stricken by the idea, felt threatened, had fragmented sleep, lost his appetite, and locked himself in his room. He had not worked since then. He was seen in a private psychiatric facility wherein a diagnosis of psychotic depression was made, and he was started on escitalopram 10 mg, aripiprazole 5 mg, and clonazepam 0.5 mg. He developed severe akathisia, and, thus, risperidone was used in lieu. He experienced recurrent torticollis. Quetiapine was then tried and uptitrated to 600 mg/d. Escitalopram was increased to 20 mg/d and clonazepam to 1.5 mg/d. The patient's sleep improved, but the core delusions were unchanged after 8 weeks on this regimen. He was admitted to our inpatient facility.

Table 1. Clozapine Adverse Reactions

Metabolic syndrome
Agranulocytosis
Cardiomyopathy and myocarditis
Pulmonary embolism
Seizures
Orthostatic hypotension
Dementia-related mortality
Sialorrhea
Pancreatitis
Constipation and ileus

Thorough laboratory investigations, sodium valproate, toxic screen, electroencephalography, and brain magnetic resonance imaging were all unremarkable. A neuropsychological battery was conducted, and slowing of processing speed, a borderline full-scale intelligence quotient score of 78, and problems with divided attention were noted. Valproate was continued, but other psychotropics were gradually phased out over 2 weeks. TBI-related psychosis was the working diagnosis. Clozapine (as Versacloz solution) was introduced, with weekly total leucocytic count protocol, at 12.5 mg/d at night and uptitrated to 150 mg/d (divided) over another 2 weeks. No extrapyramidal symptoms were reported. After 2 weeks, his delusions disappeared with no residua. He was discharged and followed up in the outpatient department. At the time of this writing, 16 weeks have elapsed, and he continues to do well with great tolerability. He is currently enrolled in cognitive remediation sessions with the occupational therapy team and most importantly is back to work.

Discussion

Capgras syndrome describes a delusional misidentification that a person closely related to the patient has been replaced by an imposter.[1] The patient accepts the resemblance but believes they are different people. It can involve inanimate objects as well and is more common in females. The syndrome was originally described by Capgras and Reboul-Lachaux in 1923 and is also called *l'illusion de sosies*.[1] It is commonly associated with schizophrenia (based on delusional percept), mood disorders (eg, bipolar), and organic brain syndromes (eg, frontal lobe dysfunction, right hemispheric dysfunction). Psychoanalytically, it was regarded as the result of ambivalent attitude to the person implicated. A disconnection between fusiform gyrus

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(concerned with face recognition) and the limbic system (concerned with emotions) has been postulated in a patient with Capgras following TBI.

As TBI sequelae, psychotic symptoms may occur early or late. In the early postinjury period, psychotic symptoms are most commonly manifestations of posttraumatic delirium. In the late postinjury period, these symptoms may be part of a schizophrenia-like psychosis, comprising predominantly persecutory delusions, auditory hallucinations, and a dearth of negative symptoms, or may be associated features of a mood disorder. The best available epidemiologic evidence suggests that TBI confers a modest increased risk for schizophrenia-like psychosis, especially if coupled with a positive family history of psychosis and male sex.[2] Of note, TBI renders patients more susceptible to neurologic side effects of antipsychotics.[3]

As the present case portrays, the patient was resistant to treatment due to either efficacy or tolerability issues. Superiority of clozapine in refractory psychosis is well established.[4] Clozapine has a unique and composite pharmacologic portfolio. Antiglutamate actions might account for clozapine having an edge over other antipsychotics (Ahmed Naguy, MBBch, MSc, personal communication, 2021). Clinical use, however, is usually fraught with a multitude of serious adverse drug reactions (Table 1), and there have been 5 black-box warnings.[5] Apart from dose-dependent seizures, clozapine is almost devoid of neurologic side effects. An impressive response at this relatively low dose is typical of organic psychoses.

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