Case Report

The patient, a 57-year-old right-handed man, sustained his first cerebral vascular accident in the right middle cerebral artery region at the age of 45, which resulted in right-sided hemiparesis that resolved completely within 3 months. He continued to run his private business successfully while living with his mother.

The patient lost his father in early childhood. There was no evidence of an emotional or conduct disorder during school years, and the patient eventually obtained his university degree. He continued to manage his successful practice until he sustained the second cerebral vascular accident in the left middle cerebral artery region at age 53.

The patient became aware of his homosexual orientation in his early teens and had several gay partners. He suffered a major depressive episode at age 26 that resolved within a few months. He also had a diagnosis of excessive harmful use of alcohol, but there was no evidence of dependence.

The patient started complaining of his changed personality and heterosexual orientation 6 months after his second stroke. At the same time he complained of excessive mood swings and changed interests. He became preoccupied with photography and had a successful photographic exhibition a year after his second stroke. His sexual orientation remained heterosexual 4 years following the second stroke, and he preferred to describe himself as bisexual because of his previous homosexual orientation.

Discussion

The mechanism by which a person acquires his sexual orientation is complex and ranges from pure psychological theories to more complex biological concepts. Our patient was aware of his homosexual orientation beginning in his early teens. He always enjoyed his gay relationships and had had at some point a live-in partner. He grew up with an absent father and had a strong bond with his mother. He went back to live with his mother after separating from his partner 4 years before his first stroke. It is unlikely that his psychological reaction to his first and/or second stroke could explain his altered sexual orientation, and his sexuality was accepted by his social network and family members.

Taking into consideration the interval between his first and second stroke, it is likely that an organic process within the left middle cerebral artery region is the cause of his altered sexual orientation.

The sexual needs of patients suffering from a brain injury are centered on hyper- and hyposexuality rather than altered sexual orientation. The alteration of sexual orientation raises serious challenges to patients and their care. It may be essential to address the issue of sexual orientation in assessing patient needs following brain injury in addition to other possible behavioral changes that might be encountered.

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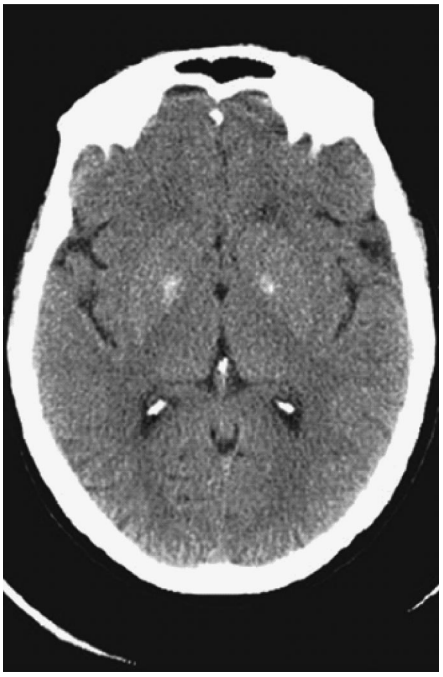
Capgras Syndrome Associated With Fahr's Disease

To the Editor: Capgras syndrome is a specific misidentification syndrome in which the person believes that another person, with whom he or she has close emotional ties as well as ambivalence at the same time, has been replaced by an persecutory imposter. Capgras syndrome has been found in a number of neuropsychiatric conditions like Alzheimer's disease, parkinsonism, vascular dementia, stroke, multiple sclerosis, and schizophrenia-like-psychosis.[1] Fahr's disease is a clinical entity with idiopathic bilateral basal ganglia calcification in the absence of any clinical or biochemical abnormality. Up to 50% of idiopathic bilateral basal ganglia calcification present with neuropsychiatric manifestations, which include auditory and visual hallucinations, complex delusions, and schizophrenia-like psychosis.[2] We describe an association between idiopathic bilateral basal ganglia calcification and Capgras syndrome, which has not been reported earlier.

Case Report

A 30-year-old woman diagnosed with paranoid schizophrenia for 6 years was maintaining well on olanzapine, 15 mg/day, with good compliance. She presented with a 6-month history of misidentifying her husband as an imposter with malice, suggestive of Capgras syndrome. There was no other associated psychopathology. A detailed physical and neurological examination suggested bilateral pedal pitting edema and positive left-sided palmomental reflex, without any evidence of cognitive impairment or movement disorder. A contrast-enhanced CT brain scan revealed bilateral basal ganglia calcification, involving the pallidal region (Figure 1). Laboratory tests including hemogram, thyroid function test, and serum calcium were within normal limits. A neurologist, whose opinion was sought in view of the CT findings, suggested the calcification as idiopathic. The patient's olanzapine was increased to 25 mg/day for the next 3 months; following this the Capgras phenomenon resolved. However, she developed delusions of infidelity during follow-up, which resolved after increasing olanzapine to 30 mg/day.

FIGURE 1. CT Scan of the Brain Showing Bilateral Hyperdense Lesions in Basal Ganglia



Discussion

In Capgras syndrome there is a disruption of facial recognition circuitry, resulting in facial misidentification.[3] The structural and functional neuroimaging studies in Capgras syndrome have localized the involvement of the bilateral parietal and posterior frontal regions with more frequent involvement of the nondominant cerebral hemisphere.[4] Till now, no particular circuit involving basal ganglia has been implicated in Capgras syndrome, although hypodensity of lenticular nucleus has been reported.[5] The CT scan of our patient revealed idiopathic bilateral basal ganglia calcification involving the pallidum. We hypothesize that, in our case, basal ganglia calcification could have disrupted one of the cortico-subcortical circuits, which might have some contribution in facial processing systems. Basal ganglia calcification leading to disruption of the thalamo-cortico-striatal circuit has been reported to manifest as schizophrenia-like psychosis. The isolated Capgras phenomenon in our case could be a part of the schizophrenia process resulting from the same mechanism.[6] A dysfunctional input of basal ganglia to the prefrontal cortex as seen in Capgras syndrome in parkinsonism[7] could be a third proposition.

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Self-Induced "Therapeutic Seizures" for the Treatment of Depression

To the Editor: Early 20th century research postulated an antagonism between epilepsy and psychosis,