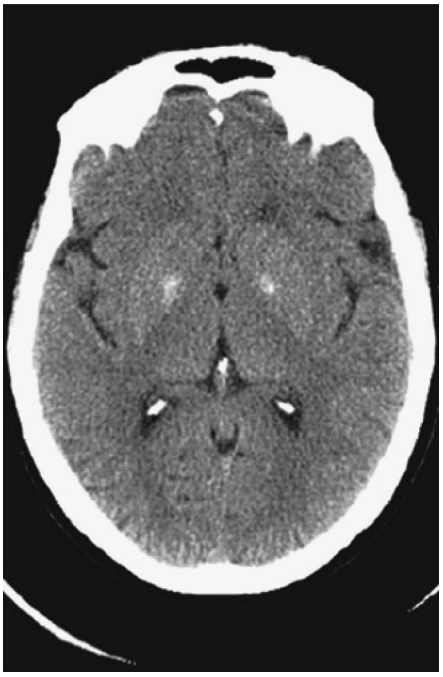
Case Report

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,

leading von Meduna[1] to introduce convulsive therapy for schizophrenia. Although modern research questions this postulate, there is a complex and understudied interplay between seizures and behavioral disorders. I report on a woman with epilepsy who used self-induced seizures to treat her depression.

Case Report

A 43-year-old woman was diagnosed with epilepsy at age 29. She experienced 6–8 complex-partial or generalized tonic-clonic seizures per month for many years, failing to respond to a range of anticonvulsant drugs. However, when she was treated with a combination therapy of phenytoin (300 mg/day) and gabapentin (1600 mg/day), her seizures were completely suppressed. Regrettably, after 6 months without seizures, she noticed the onset of depression. Her personality underwent a profound change; she became sad, feeling that life was no longer worth living. She had trouble sleeping and had early morning awakening. Food lost its taste and she lost weight. Although she was depressed, there were no symptoms of psychosis. Her family confirmed the marked change in her mood. Although she had experienced some depressive episodes in her early 20s, since developing epilepsy she had been a happy, positive-thinking person, despite the personal difficulties arising from her seizure disorder.

Several antidepressants were tried, but none were of benefit; she remained depressed for more than a year. Ultimately, due to the depression, she quit taking her anticonvulsants stating that "life just is not worth it." This led to a series of complex partial and generalized tonic-clonic seizures, after which her mood promptly became cheerful and optimistic again. She felt that seizures had done more for her mood than the antidepressant drugs. She was restarted on phenytoin and gabapentin, but did not receive any further antidepressants. After several years of unsupervised self-experimentation, she disclosed a new approach to her seizure disorder. She would abruptly stop taking gabapentin for five consecutive days during the last week of every fourth month. This would result in her having several complex-partial and generalized tonic-clonic seizures (at 3–4 days postwithdrawal); she did not disclose these periodic seizures to family or friends. Since embarking upon this strategy of self-induced "therapeutic seizures," her depression had completely resolved.

Discussion

In the 1950s, Landolt[2] observed people with epilepsy who became psychotic when their seizures were controlled—an observation which he attributed to the electroencephalographic phenomenon of "forced normalization." Conversely, Tellenbach[3] used the term "alternative psychoses" to describe people whose psychosis resolved with the return of seizures. Although these notions are now regarded as outdated and inaccurate, there is a close biological link between seizures and psychiatric (not necessarily psychotic) disorders.

This case demonstrates the relationship between seizures and depression. In particular, it demonstrates that the dynamic neurochemical milieu of the postseizure state may exhibit prolonged anticonvulsant properties. Postseizure increases in brain levels of dopamine and serotonin, as well as alterations in norepinephrine metabolism, have been consistently observed in animal models of epilepsy.[4,5] Analogously, studies concerning the use of electroconvulsive seizures to treat depression likewise reveal increased release of norepinephrine and increased serotonergic activity (including upregulated 5-HT_{2a} receptors and increased 5-HT_{2a} mRNA levels) in the postseizure state.[6] Since such neurochemical changes have antidepressant effects, it is reasonable to assert that "naturally occurring" seizures also produce antidepressant effects.

Although abrupt alterations in anticonvulsant dosing cannot be recommended, this woman with epilepsy used periodic self-induced seizures precipitated by intermittent medication withdrawal to treat her depression.

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