cific neural substrate has not yet been established.[1]

Case Report

The patient, a 61-year-old right handed woman with no psychiatric history, presented 1-year history of increasingly jealous behavior. She had no evidence of premorbid jealousy or paranoid personality traits. Her husband had been a trading company employee, and frequently made business trips. In 1985, she suffered from tuberculum sellae meningioma. The right orbitofrontal cortex was removed in the process of extirpating the meningioma through intrahemisphere approach. Following the operation, she became irritable and pathologically persistent. She persisted in saying that her son should marry in haste, which ultimated in his running away from home. In 2001, she started to accuse her husband of infidelity. She insisted that he was having an affair with a 70-year-old woman who was a member of their ground golf circle. Also, she believed that they held a wedding ceremony and had more than 10,000 children. At this point, she had a medical examination. She was normotensive and blood chemistry and hematology were normal. The Hasegawa Dementia Rating Scale–Revised score was 30/30. An MRI scan revealed a deficit of the right orbitofrontal cortex.

In 2001, her accusations became more severe and her husband consulted our clinic. Mental state examination revealed that she tended to confabulate and was easily distracted. Her speech was grammatically correct. In addition to delusional jealousy, she experienced auditory hallucinations that told her her husband was unfaithful. She believed that her husband's mistress would frequently visit their house and steal things. She pinched a piece of paper in the door to see if someone came into her home when she went out. Her Mini-Mental State examination (MMSE) score was 29/30. She could remember five items and her autobiographical memory was intact. Her family history for mental illness was negative.

Her husband reported that she bought unnecessary articles and sometimes coerced him to shoplift. Her son complained that she repeatedly called him and tried to persuade him to marry. In addition, she sometimes made sexual jokes to him and complained about her sexless life. Because she refused medicine, we offered a supportive psychotherapy to her husband to relieve his burden. As he became adept at dealing with her accusations, the frequency and severity of her accusations gradually decreased.

Comment

The present patient was unique in having localized cerebral pathology. In previous reports of organic Othello syndrome, lesion areas were rather broad.[2–4] This case suggests that the Othello syndrome may be caused solely by right orbitofrontal lesions. In men, it is suggested that sexual dysfunction plays an important role in the development of this syndrome. Although our patient did not have sexual dysfunction, hypersexuality might cause an excessive sex drive. In addition to hypersexuality, she revealed frontal lobe syndrome, including antisocial behavior and distractibility. Dysfunction of the frontal system might preclude her from correcting her misbelief in the face of contradictory evidence, which was suggested in other content-specific delusions.[5] Finally, the additional role of psychological stress, such as solitude, which is common in middle-aged woman, should also be underlined.

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Schizophrenia-Like Psychosis Following Right Putaminal Infarction

SIR: Schizophrenic symptoms are sometimes caused by physical disorders affecting brain function. Neuroimaging findings in these cases give useful information for understanding the neuropsychiatric background of schizophrenic symptoms, especially in early stage neurodegenerative disease and focal cerebrovascular disorders. Here, we report a case of schizophrenia-like psychosis following right putaminal infarction.

Case Report

The patient was a 40-year-old right-handed Japanese high-school graduate without a history of psychotic illness. Following her father's

death, she developed subacute auditory hallucinations and persecutional delusions (for example, she thought someone was intruding into and wiretapping her house, or believed that her child had been kidnapped). A few days later, she became excitable and tried to strangle her husband, after which she was restrained by her family and admitted to our hospital. On admission, she was alert, and physical and neurological examinations revealed no abnormalities. Laboratory tests were normal. A magnetic resonance imaging (MRI) scan revealed a subacute right putaminal infarction, which was partially extended to the right caudate and insula. The infarction was treated conservatively. Administration of risperidone (5mg/day) was initiated and the psychotic symptoms gradually disappeared. Two months later, the psychotic symptoms had completely disappeared and she was discharged. The regimen of risperidone was tapered off over the following 6 months and no relapse was observed.

Comment

Cummings[1] noted that subcortical structures (striatum [caudate and putamen], globus pallidus, and thalamus) and the prefrontal cortex form three circuits (dorsolateral prefrontal, lateral orbital, and anterior cingulate circuits) and these circuits mediate many aspects of human behavior. On the other hand, recent neuroimaging studies in schizophrenia have demonstrated morphological and functional abnormalities of the frontal-subcortical circuits, although these results remain ambiguous.[2,3]

In our patient, a right putaminal infarction, which affects the dorsolateral prefrontal and lateral orbital circuits, caused schizophrenia-like hallucinations and delusions. In contrast, a case of schizophrenia in which hallucinations and delusions disappeared following left putaminal hemorrhage was previously reported.[4] In this previous case, it could be speculated that excessive left frontal-subcortical function was associated with schizophrenic symptoms, and that correction of the excess function by left putaminal hemorrhage extinguished the symptoms. Collectively, these findings suggest that left and right frontal-subcortical functional asymmetry (i.e., relative right hemispheric hypofunction or relative left hemispheric hyperfunction) may play a critical role in the appearance of schizophrenic symptoms, especially for hallucinations and delusions.

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Psychogenic Movement Disorder Masquerading as CJD

SIR: Creutzfeldt-Jakob Disease (CJD) is a rare, rapidly progressive prion disease characterized by dementia, dysarthria, and movement disorders, and typically culminates in death within 6 months of contraction.[1] We describe a patient who presented with a conversion disorder resembling CJD confounded by 14-3-3 proteins in his CSF.

Case Report

A 58 year-old man with a history of major depression complained of having memory impairment, difficulty walking, choppy speech, and incoordination for 1 month. There was no family history of movement disorders or dementia.

Initial examination by other physicians revealed intact immediate and recent memory and normal speech and language. He was noted to have complex choreoathetotic movements in his arms, startle myoclonus, bilateral intention tremor, and a wide-based slow gait.

Given subjective subacute memory decline, startle myoclonus and gait ataxia, the possibility of CJD was entertained. EEG and brain magnetic resonance imaging (MRI) were normal; however, 14–3–3 proteins were present in his CSF and he was diagnosed with CJD by other neurologists.

Despite complaints of new or worsening symptoms, there was no change in his examination; a repeat EEG was normal and neuropsychological testing was not consistent with a progressive dementia. His Mini-Mental State Examination