rior parietal cortex. Orbitofrontal cortex dysfunction during negative emotional processing with subsequent hyperactivity of the anterior cingulated cortex and medial prefrontal cortex may mediate catatonic stupor and akinetic mutism. Similarly, selective mutism, a form of social phobia/social anxiety disorder, can be regarded as a "motivational disorder" suggesting an involvement of the dorsolateral prefrontal cortex as an essential part of the "willed action system." Indeed, Tillfors et al.[8] found an activation of the amygdala, dorsolateral prefrontal cortex and inferior temporal cortex when subjects with social anxiety disorder were exposed to a public speaking task to provoke anxiety-related symptoms.

Although the etiology remains unclear, patient's speech and language disturbance fits in a "congenital" form of Broca's type or nonfluent aphasia (left inferior frontal cortex). Cognitive deficits occurred mainly in the executive domain (sustaining and dividing attention, verbal learning strategy, working memory, and cognitive flexibility), while verbal memory, concept formation, and reasoning were spared. According to Stuss et al.,[9] these executive deficits can be attributed to dorsolateral and medial prefrontal dysfunction.

In conclusion, anatomic regions suggested to be affected in our patient include prefrontal association cortex regions that have often been associated with neuropsychiatric disorders (dorsolateral, ventrolateral and medial prefrontal cortex, orbitofrontal cortex, and anterior cingulated cortex), but also parts of the motor cortex including the left inferior frontal lobe and the supplementary motor area. Because cortical/subcortical structural damage in the MRI scan as well as signs of "hypofrontality" in the PET study were absent in our patient, it remains open whether impaired connectivity between different brain regions might have caused both functional hyperactivity of some regions (e.g., dorsolateral prefrontal cortex in selective mutism) and hypoactivity of others (e.g., orbitofrontal cortex and supplementary motor area in executive dysfunction), respectively. Thus, regional frontal lobe syndromes, including neuropsychiatric symptoms, may be present without any signs of structural abnormalities. In contrast to the case reported by Ferrara et al.,[10] structural and functional neuroimaging was not a helpful tool for diagnosis in our patient, but detailed neuropsychological and psychopathological examinations were.

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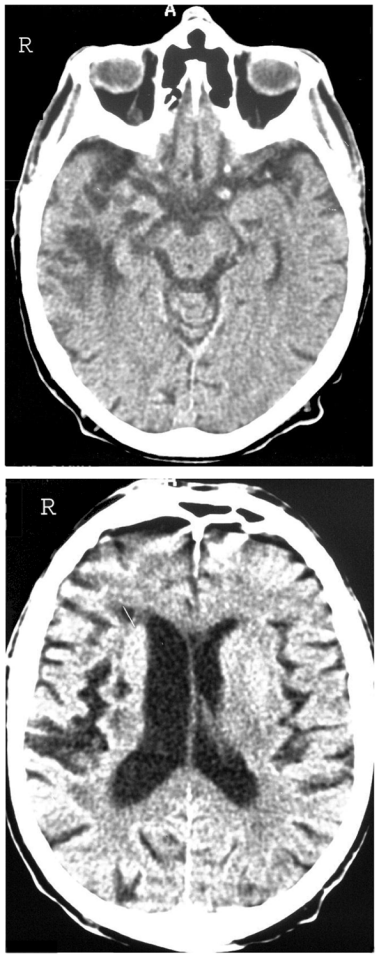
Neuroimaging Correlates of Chronic Delusional Jealousy after Right Cerebral Infarction

To the Editor: Although there have been accounts of delusional jealousy occurring in patients with structural brain damage, the phenomenon has rarely been reported following right cerebrovascular infarction. In this new case we will endeavor to describe the possible pathogenic role of right hemisphere localization.

During hospitalization for a transient left-sided hemiplegia after a right middle cerebral artery infarct, a 77-year-old man became convinced that members of the medical staff wanted to steal his house. Although he was rapidly discharged (5 days later), he subsequently declared that his wife had had a love affair with a school master many

years before. The patient had always been jealous and envious of the teacher, and he believed that his wife was still seeing him. During the 3 years following his infarct, he had to be hospitalized on several occasions because of verbal and physical abuses toward his wife as well as bouts of severe depression, during one of which he made a serious suicide attempt. On examination, 1 month after his infarct, the patient presented as mildly intellectually deteriorated and the recall test of the Rey figure demonstrated defects in visuospatial memory. Although outwardly calm, he showed a marked lack of emotional control when discussing his wife. It transpired that the evidence he gave to support his beliefs regarding her infidelity were false interpretations of trivial past events. A CT head scan (Figure 1) showed a low density defect of the right temporal and parietal lobes as well as mild cortical atrophy. Single photon emission computed tomography(SPECT) using hexamethyl-propyleneamine oxime (HMPAO) (Figure 2) revealed a large hypoperfusion of the right hemisphere, involving the right frontal lobe, the possible result of a deafferentation (or diaschisis) effect.

FIGURE 1. Widening of the Sulci Lesions of the Right Temporal Lobe (Top) and of the Right Parietal Lobe (Bottom)



In their study of the anatomical basis of delusions after right cerebral infarction, Levine and Grek [1] argued that delusions depended primarily on premorbid brain atrophy rather than location and size of the lesion. However, this conclusion has been challenged by Westlake and Weeks[2] who described a case, after right infarction, in a much younger patient. Furthermore, Levine and Grek's conclusions were only based on the results of CT scans, and did not take into account the possible functional effects of the lesions. Malloy and Richardson[3] underlined the role of the lesions of the frontal lobes and the right hemisphere in various content-specific delusions and suggested that, in at least some cases, a dysfunctional frontal system may be a necessary component in the development of fixed delusions. In a case of spatial delirium (reduplicative paramnesia), Nighoghossian et al.[4] demonstrated with SPECT the pathogenic role of functional right hypofrontality. We submit that our case indicates that right frontal involvement is crucial in the emergence of delusional jealousy. This would explain why a similar delusion appeared after a right frontal biopsy in the case described by Silva and Leong[5] when the infarct was in the left frontal lobe. Of course, such neuroimaging correlates do not exclude, particularly in older patients, the etiological role of psychological factors, such as premorbid jealousy and/or paranoid personality traits.

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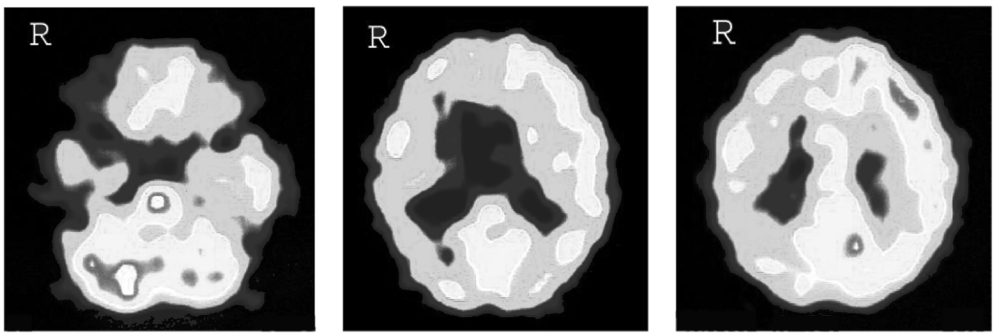
FIGURE 2. Right Severe Temporal and Parietal Hypoperfusion; Relative Right (Mainly) and Left Frontal Hypoperfusion



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Risperidone and Valproate for Mania Following Stroke

To the Editor: Poststroke depression is common. In contrast, mania is rare after a stroke.[1,2] We report a case of mania following a stroke with a lesion in the nondominant (right) temporal cortex, which was successfully treated with a combination of risperidone and valproate.

Case Report

Mr. A, a 55-year-old, right-handed man, presented sudden nonfluent aphasia lasting 10 minutes, which was investigated in the emergency department. CT scan revealed focal atrophy in the right temporal cortex. According to the general practitioner and the emergency department, the patient developed mania within 24 hours, characterized by psychomotor agitation, insomnia, a euphoric mood, distractibility, an irritable affect, disorganized thought, denial of illness, and flight of ideas. He had been receiving treatment for hypertension, coronary atherosclerotic vascular disease (chronic stable angina), peripheral arterial disease, and diabetes for 10 years from his general practitioner. His treatment included 10 mg/day of amlodipine, 5 mg/day of trinitrine, and 5 mg/day of glibenclamide. There was no previous personal or family history of psychiatric disorder.

He was admitted to our psychiatric department without his consent 9 days later. He fullfilled the DSM-IV criteria for a manic episode. He was correctly orientated. Cognitively, he scored 28 out of 30 on the Mini-Mental State Examination. B_{12} and folates levels were within the normal range. Syphilis serology was negative. A second CT scan showed ischemic focal changes in the right temporal lobe. An angiography revealed a thrombotic stenosis in the right vertebral artery. Mr. A was started on 1000 mg/day of valproate and 2 mg/day of risperidone and had a good response. Over a 15-day period, his elevated mood returned to an euthymic level.

Comment

This case report was characterized by a strong temporal relationship between the neurological symptoms and the onset of the manic episode (24 hours). As in previous studies of poststroke mania, the clinical presentation was the same as for primary mania.[1,2] However, since no CT imaging was available prior to the first manic episode, it is not possible to make any definitive conclusions.

Mania and bipolar affective disorder are strongly associated with right hemisphere lesions, particularly in the limbic or limbic-related areas, which have strong connections with frontal lobe, right orbital frontal lobe, basotemporal, basal ganglia, or thalamic lesions.[1,2]

There is currently no clear consensus about the most effective treatment for poststroke mania. Evans et al.[3] suggested that secondary manic episodes differ from typical bipolar states and are often particularly difficult to treat. This preliminary report suggests that risperidone and valproic acid can be used in the management of secondary mania. Controlled clinical trials are necessary to confirm the efficacy and tolerance of these drugs in the treatment of secondary mania.

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Depression Preceding the Onset of Progressive Supranuclear Paralysis: A Case Report

To the Editor: Progressive supranuclear palsy is a movement disorder which often causes parkinsonian symptoms. Typical clinical findings in progressive supranuclear palsy are supranuclear vertical gaze palsy, postural instability, gait abnormality, bradykinesia, dysarthria, an increased axial tone, and a