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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

frightened facial expression. There are hints that patients diagnosed with progressive supranuclear palsy have an increased risk for developing depressive-like syndromes within the next years.[1] However, to our knowledge, there is no data recording major depression as an antecedent symptom of later diagnosed progressive supranuclear palsy.

Case Report

A 57-year-old man was admitted to our hospital for a major depressive syndrome without psychotic features. His main symptoms were depressed mood, anhedonia, apathy, loss of activity, difficulties in concentration, and self-reproach feelings. Attention and short-term memory were slightly impaired.

He indicated that this first depressive episode began 4 years earlier. No neurological, general medical, or psychiatric diseases had been described or diagnosed before. Within the first 3 months of the episode, depressive symptoms improved spontaneously; however, slightly depressed mood and concentration difficulties persisted until the time of admission. During the last year, decreased motor activity with slowed movements was observed. Moreover, he sometimes stumbled, had a tendency to fall backward, and had difficulties in walking down stairs. During the last month before admission these symptoms worsened. His wife described a change in his manner: he had become more and more apathetic, had increasing cognitive declines, and neared almost complete motor inactivity. He was admitted to our hospital for further diagnosis.

Neurological examination showed moderate hypomimia with a frightened facial expression, pathological eye movements with voluntary saccades, ocular pursuit movements, and vertical gaze palsy (impairment of voluntary vertical eye movements with preservation of reflex vertical eye movements). His motor activity was decreased; he had a slight rigor of the left arm and an increased axial tone with neck extension. Furthermore, we found a slight bradykinesia of the left arm and an unstable gait. He also described writing difficulties, but the type face was not changed in terms of a micrography.

No pathological findings were obtained by physical examination or standard laboratory tests including the liquor cerebrospinalis, EEG, ECG, and MRI of the head. To differentiate progressive supranuclear palsy from Parkinson's disease and other related disorders, we tested the response to L-dopa. The intake of dopamine (Madopa® 500 mg/day) did not lead to any improvements. This lack of appreciable response to L-dopa and dopaminergic drugs characterizes progressive supranuclear palsy.

Taking all findings together, we diagnosed a progressive supranuclear palsy which started with a major depressive episode according to the DSM-IV criteria.

Comment

As far as we know, this case report is the first published report of a patient with a depressive episode as a first ("soft") sign of a progressive supranuclear palsy. There are reports and even studies of progressive supranuclear palsy-associated depressive-like symptoms, especially apathy, disinhibition, dysphoria, and anxiety.[2] Awareness of possibly preceding psychiatric aspects like a depressive state as the first symptom of developing progressive supranuclear palsy may be helpful for an early diagnosis of this and other neurodegenerative diseases.

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Marked Hyperphagia Associated With Total Loss of Satiety in Alzheimer's Disease

To the Editor: The neuroanatomical basis for hyperphagia seen in patients with dementia is not well understood. We describe here a patient with marked hyperphagia associated with complete loss of satiety who provides clues to the site of involvement.

Case Report

An 84-year-old man with progressive memory loss for 4 years was diagnosed to have Alzheimer's disease and followed clinically over 3 years. His Mini-Mental State Examination (MMSE) score was 19. Language functions were normal. Cranial MRI scan revealed mild cortical atrophy. Serum B_{12}, Folate, T_3, T_4, and thyroid stimulating hormone levels were normal. Marked hyperphagia (confirmed by detailed history[1] and direct observation) was noted about 1 year after onset. Under direct observation, the patient was noted to complete a heavy meal (estimated 1800 calories) and a few minutes later to be eating