Mania as a Possible Prodrome to Dementia

SIR: There has been much debate over whether late onset depression may be a prodrome to dementia.[1] Kessing and Fleming[2] have also suggested a link between affective disorders and dementia. The authors could not find a report in the literature that described mania preceding the diagnosis of dementia. Below we describe an elderly patient who was diagnosed with dementia following a solitary episode of mania.

Case Report

"Mr. V" is a 76-year-old Caucasian man who presented to our facility with an isolated manic episode prior to obtaining a definitive diagnosis of dementia. Besides a self-limited episode of depression that occurred in his thirties, he had no other notable past psychiatric history. The patient was reported to be euthymic and relatively cognitively intact until he underwent the removal of a basal cell carcinoma for which he received a 2 to 3 week course of corticosteroids. Approximately 6 weeks following the discontinuation of the corticosteroids, Mr. V exhibited symptoms of mania, which included insomnia, hyperreligiosity, hypersexuality, hyperverbosity, irritability, and increased cleaning behavior which led to his first psychiatric hospital admission. Upon admission to the hospital, Mr. V's Mini-Mental State Examination (MMSE) was 21/30. He was treated with a vast array of mood stabilizers and his mania eventually abated 2 months into his hospital stay. Following the aforementioned manic episode, the patient then became apathetic while in a euthymic state. He would sleep the majority of the day and night and express no desire to participate in activities. His apathy was mood congruent and there was no indication of any affective disturbance. The patient would also frequently confabulate his activities and experiences. Neuropsychological testing revealed impairments in verbal and visual learning and memory, executive functioning, and complex aural comprehension. Object naming and word finding tasks were preserved, suggesting a diagnosis of Alzheimer's dementia. His MMSE at this time was 23/30. A full medical work-up was also conducted and revealed no vitamin deficiencies, thyroid dysfunction, or source of infection. His EEG was normal and a magnetic resonance image of his brain revealed small vessel ischemic changes. Furthermore, a single photon emission computed tomography scan revealed hypoperfusion of both frontal lobes and mild bilateral temporal lobe perfusion; both of which were deemed nondiagnostic findings.

Comment

The presentation of the above patient suggests that affective disorders in general may precede the diagnosis of dementia. Such a prodrome may not be confined to depression and may, in fact, include mania.

Brian S. Appleby, M.D.
Paul Rosenberg, M.D.
Johns Hopkins Hospital, Baltimore, Md.

References

1. Scheitzer I, Tuckwell V, O'Brien J, et al: Is late onset depression a prodrome to dementia? Int J Geriatr Psychiatry 2002; 17:997–1005
2. Kessing LV, Flemming MN: Increased risk of developing dementia in patients with major affective disorders compared to patients with other medical illnesses. J Affect Disord 2003; 73:261–269

A Rare Case of Epilepsy in a 16-Year-Old Girl With Fallot Tetralogy Attributed to CNS Heterotopia and Pachygyria

SIR: We report a case of a 16-year-old girl who was admitted to our clinic because of partial seizures. The clinical neurological examination and the neuropsychological tests were normal. The laboratory tests (i.e., CBC, blood biochemistry, urine tests, immunologic assay tests) were normal. The CSF analysis and cultures, as well as the CSF protein electrophoresis, were normal. Cerebral computed tomography (CT) was normal and ECG showed RBBB (right bundle branch block) and LAH (left anterior hemiblock). However, the EEG revealed abundant generalized slow wave activity with occasional focal and diffuse spikes and spike wave activity.

The brain magnetic resonance imaging (MRI) scan revealed subcortical heterotopia of the gray matter extending across the right ventricle. More specifically, along the surface of the occipital horn of the right ventricle and at its posterior part as well as at the right temporal horn, there was an irregularly lobulated mass of gray matter that extended into the hemispheric white matter giving signals similar to the cerebral cortex. There were also similar

signals of the cerebral cortex at the temporal horn of the left ventricle. The cortex overlying the heterotopia was abnormal, too. Pachygyria at the cortex of the right temporal parietal occipital area was identified.

Both heterotopia and pachygyria which presented at the brain MRI of our patient may reflect possible anatomic epileptogenic areas.[1] The above conditions represent developmental abnormalities of the CNS caused by neuronal migration defects. The embryogenetic period when the above developmental abnormalities took place is estimated to be between the sixth and the seventh gestational week when Fallot tetralogy was developed.

Comment

This is the first case report in the literature of comorbid CNS migration defect and Fallot tetralogy. The heart development defect allows us to not only estimate the possible embryogenetic period but also to raise suspicion for implication of the hypoxic blood supply of the defective heart in the etiology of the neuronal migration defect.

Defective neuronal migration results in the formation of a disorganized cerebral cortex in which neurons are not normally connected with one another.[2] Neurons that failed to reach their destination at the cortex remain at subcortical positions and differentiate composing islands of mature nerve cells, resembling cortical neurons (subcortical band heterotopia) separated from the overlying cortex by an intervening band of white matter. The gyral pattern is also abnormal and is the basis for the morphologic classification of neuronal migration defects into lissencephaly, pachygyria, and polymicrogyria.

Smaragda Katsoulakou, M.D.
Argyro Kiamili, M.D.
Dimitris Karaiskos, M.D.
Department of Neurology, Tzaneio General Hospital, Pireas, Greece

References

1. Raymond AA, Fish DR, Sisodiya SM, et al: Abnormalities of gyration, heterotopias, tuberous sclerosis, focal cortical dysplasia, microdysgenesis, dysembryoplastic neuroepithelial tumour and dysgenesis of the archicortex in epilepsy: clinical, EEG and neuroimaging features in 100 adult patients. Brain 1995; 118:629–660
2. Barkovich J, Kuzniecky RI: Gray matter heterotopia. Neurology 2000; 55:1603–1608

A Comment on "Exertion" After Sports-Related Concussion

SIR: The Concussion in Sport (CIS) group[1,2] recommended that athletes with concussions undergo complete rest and be asymptomatic prior to beginning any rehabilitative therapy program. CIS recommended that concussed athletes avoid any exertional physical activity after a sports-related concussion, as such activity can reactivate and/or exacerbate the symptoms of concussion. A stepwise rehabilitation program is recommended, with successful completion of each graduated step being a prerequisite for transition to the next, more intensive activity level. The initial CIS statement in 2002 focused on physical exertion (e.g., walking, stationary cycling, weight training, running, skating), while the second CIS position paper, in 2005, added cognitive exertion as a potentially aggravating factor postconcussion. I report two clinical cases of concussed professional athletes whose recovery from a sports-related concussion was probably complicated by exertional activity typically not considered "exertional"—sexual behavior.

Case Report

A 21-year-old hockey player sustained a concussion in a fight during a game. There was no loss of consciousness or amnesia; immediate symptoms included headache, dizziness, nausea, blurred vision, and a feeling of "fogginess" for several days. His score on the Post-Concussion Syndrome (PCS) scale was 23, with a preseason baseline PCS score of 2. He was removed from the game and did not return to play. His concussion history included one concussion 3 years prior with no loss of consciousness or amnesia and complete recovery after 2 weeks. The athlete was advised not to engage in any exertional activities (physical or cognitive). The concussive symptoms dissipated within a week, and the Athletic Trainer began him on a rehabilitative program of stationary cycling. Stationary cycling led to a recurrence of symptoms, so a return to complete rest and restriction from physical and cognitive exertion was recommended. One week later the athlete was again asymptomatic. He underwent neuropsychological testing, with results being consistent with his preseason baseline testing. MRI of the brain was reported as normal, and neurological examination was benign. He began light aerobic activity on a Wednesday, with no recurrence of symptoms. On Friday his activity level was increased from stationary cycling to skating, and he remained symptom free.

However, upon reporting to practice the following Monday, the athlete complained of a recurrence of symptoms (PCS score of 25). He denied any unusual or atypical exertional activity during the weekend to the athletic trainer. Careful questioning, however, revealed that his