Delirious Mania Associated With Bipolar Disease in a Brazilian Patient: Response to ECT and Olanzapine

To the Editor: The subtype of catatonia designated as delirious mania is characterized by excessive motor activity, combined with factors such as: unsystematic speech, disorientation, confusion, and psychosis, with acute onset of symptoms. Delirious mania is associated with bipolar disorder, and its symptoms encompass mania and acute mental confusion, when additional tests do not indicate other diseases. It was initially described by Calmeil[1] in 1832, but a subsequent case series published by Luther Bell[2] in 1849 described 40 patients with the condition among 1,700 admissions to McLean Hospital. Three-quarters of these patients died. Many authors have suggested that 15%–20% of all patients with acute mania show signs of delirium, contrary to the notion that it is not as common.[3] Patients with this syndrome experience significant morbidity[4] and a high rate of mortality without treatment.

There is no clear consensus on what clinical characteristics are associated with delirious mania or what treatments are effective. Recognition of this syndrome is further complicated by the fact that many cases in delirious psychiatric patients are precipitated by medical or neurological conditions or use of psychoactive substances.[5]

Case Report

We report the case of a 42-year-old man diagnosed with bipolar disorder, initiated when he was 27 years old, who experienced irregular treatment due to living in a rural area. There was 1 year without treatment. He came to the emergency hospital with acute onset (7 days) of severe symptoms such as irritable mood, reduced need for sleep, pressured speech, racing thoughts, distractibility, fluctuating levels of consciousness, disorientation, disorganized behavior, extreme psychomotor agitation, pressured speech, hypersexuality, visual hallucinations, stereotypy, echolalia, echopraxia, tachycardia, rapid heart rate, and grandiosity.

We started treatment with lorazepam, up to 10 mg, without improvement. Lithium carbonate, 900 mg per day, also failed to cause improvement. After haloperidol, 10 mg/day, and thioridazine, 600 mg/day, the symptoms worsened, and he became more hostile and angry and experienced increased psychomotor activity, resulting in more weight loss and delirium. His laboratory studies, including CBC, CPK, serology for syphilis and AIDS, thyroid function tests, and renal functions, were within normal limits. A brain CT revealed no pathological findings.

After 20 days without improvement, we indicated ECT. After the third session, he was better, chatting adequately and sleeping from 6 to 7 hours per night. However, there were technical problems and the ECT was suspended. After 2 weeks, he began to gradually worsen. We started a treatment of olanzapine, up to 30 mg/day. After 2 months, the patient showed marked clinical improvement in his symptoms of catatonia and delirious mania. The response to ECT was much faster. He was maintained on olanzapine, 15 mg/day, and was referred to the outpatient clinic for psychiatric follow-up.

Discussion

An antipsychotic, even atypical, should be used with great caution in catatonia because it can cause neuroleptic malignant syndrome or even aggravate the catatonia, especially the malignant catatonia. However, Nicolato et al.[6] have reported a response to olanzapine for catatonia associated with bipolar disorder.

Rodrigo Nicolato, M.D., M.Sc., Ph.D.
Neuropsychiatry, UNIFENAS-BH, Brazil; Instituto Raul Soares, Belo Horizonte, Minas Gerais, Brazil; Psychiatry, IPSEMG, Brazil
Alexandre Costa-Val, M.D.
Anderson Souza, M.D.
João Vinícius Salgado, M.D., M.Sc., Ph.D.
Instituto Raul Soares, Belo Horizonte, Minas Gerais, Brazil
Antônio Lúcio Teixeira, M.D., M.Sc., Ph.D.
Internal Medicine, Federal University of Minas Gerais, Brazil

References

1. Calmeil LF: Dictionnaire de Medicine: Our Repertoire General des Sciences Medicales Considerees Sous le Rapport Theorique et Pratique, 2nd ed. Bechet, Paris, 1832
2. Bell L: On a form of disease resembling some advanced stage of mania and fever. Am J Insanity 1849; 6:97–127
3. Fink M: Delirious mania. Bipolar Dis 1999; 1:54–60
4. Friedman RS, Mufson MJ, Eisenberg TD, et al: Medical and psychiatrically ill: the challenge of delirious mania. Harv Rev Psychiatry 2003; 11:91–98
5. Karmacharya R, England ML, Ongür D: Delirious mania: clinical features and treatment response. J Affect Disord 2008; 109:312–316
6. Nicolato R, Romano-Silva MA, Correa H, et al: Stuporous catatonia in an elderly bipolar patient: response to olanzapine. Aust N Z J Psychiatry 2006; 40:498

Psychiatric Comorbidities in Dandy-Walker Variant Disorder

To the Editor: The term "Dandy-Walker complex" has been used to

denote Dandy-Walker malformation, the Dandy-Walker variant, and megacisterna magna[1] together. The Dandy-Walker variant is defined as cerebellar dysgenesis, without enlargement of the posterior fossa and with variable degree of hypoplasia of the cerebellar vermis.[2] The commonly mentioned symptoms are developmental delay, mental retardation, cerebellar ataxia, and symptoms of hydrocephalus.[3] The psychiatric dimensions of the Dandy-Walker syndrome have not been elaborated adequately in the literature. We report a case of Dandy-Walker variant presenting with a multitude of psychiatric manifestations with a special focus on the causative role of cerebellar defect in attention deficit-hyperactivity disorder (ADHD).

Case Report

A 14-year-old-male was brought in with complaints of remaining overactive; not sitting in place; poor comprehension at school and at home since the ages of 4 and 5; throwing temper tantrums; abusive, assaultive, and destructive behavior; lying and stealing; and continuously rubbing both index fingers around the respective thumbs for the past 5 years. His birth history was normal. He didn't have any contributory family history. He had multiple earlier psychiatric consultations, but he did not become compliant on any of them. The mental status examination revealed an overactive patient, not remaining in one place for more than a couple of minutes. His stereotypical movement of rubbing both index fingers around his thumbs would disappear when he was involved in other activities, but physical examination revealed hardening at the sites of the fingers and thumbs where he rubbed them. His IQ came to be 58. Subsequently he was diagnosed with hyperkinetic conduct disorder (Conners Rating Scale score=18) with mild mental retardation, stereotypic movements, and nocturnal enuresis. His routine investigations as well as ultrasound of abdomen and electroencephalogram were normal. His CT brain scan revealed the following abnormalities: the fourth ventricle was more prominent; the fourth ventricle was communicating with a small posterior fossa cyst; the cerebellar tonsils were separated by more than usual distance; the most inferior vermis was not clearly seen, implicating that it was hypoplastic; and the cerebral sulci, bilateral sylvian fissures, and bilateral cisterns were prominent (Figure 1 and Figure 2). The posterior fossa was normal in size. The impression made by the consulting radiologist was that it was Dandy-Walker variant. The patient was given symptomatic pharmacological treatments for his nocturnal enuresis and for his hyperactivity along with behavioral therapy for his destructive behavior, hyperactive behavior, and his repetitive finger movements. After 2 weeks of treatments, his night time bedwetting stopped completely, as well as his overactive and destructive behavior after 1.5 months (a fall of 80% in Conners Rating Scale score). His repetitive finger movements reduced about 50%.

Discussion

Our case can be explained just on the basis of comorbidities in ADHD. However, the neuropathological bases of these coexistences are not clearly known.

As mentioned before, mental retardation is fairly common with the malformations of Dandy-Walker complex. Other features presented in our patient (i.e., the conduct disorder, hyperkinetic disorder, stereotypic movements, and nocturnal eneuresis) have not been reported together in association with Dandy-Walker variant in the literature to the best of our knowledge. This could have been due to the rarity of this condition. The exact role of the cerebellum

FIGURE 1. CT Scan of the Patient Showing Enlarged Fourth Ventricle and Separated Cerebellar Tonsils Along With Mild Dilation of Other CSF Spaces

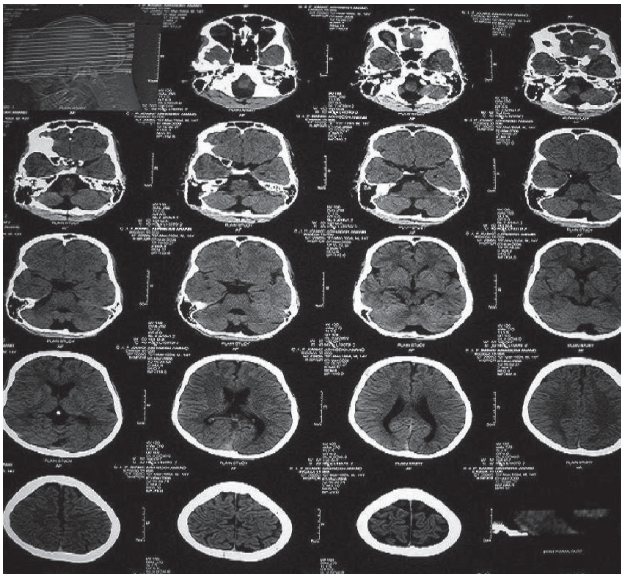
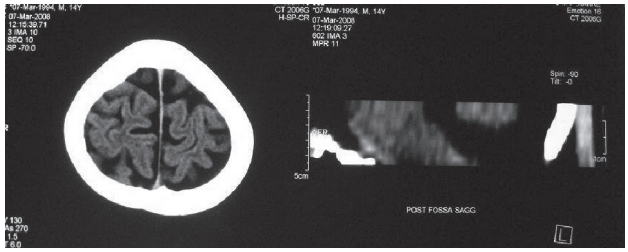


FIGURE 2. Close Up of the Sagittal Section of Posterior Fossa Showing Hypoplasia of the Lower Part of Cerebellar Vermis



in these disorders is not known. It has been suggested by various studies that the cerebellum is involved in subserving attention. However, cerebellocerebral connections are more important rather than cerebellum in this context.[4] More direct relation of a vermian defect comes from the morphometric MRI studies which have shown that the vermal volume is less in individuals with ADHD. This reduction has been found to involve mainly the posterior inferior lobe but not the posterior superior lobe,[5] consistent with the CT finding of our patient. However, such studies have been few and the vermian defect has not been severe enough so as to be called hypoplasia. Association of cerebellar abnormality with stereotypic movements is an atypical finding and any link between them is a matter of speculation.

Ravi Prakash
Psychiatry, Central Institute of Psychiatry, Udaipur, India
Lokesh Kumar Singh
Jatinder Kour
Biswaranjan Mishra
Psychiatry, Central Institute of Psychiatry, Ranchi, Jharkhand, India
Anuja Lakra
Pathology, Rajendra Institute of Medical Sciences, Ranchi, Jharkhand, India

References

1. Barkovich AJ, Kjos BO, Norman D, Edwards MS: Revised classification of posterior fossa cysts and cystlike malformations based on the results of multiplanar MR imaging. Am J Neuroradiol 1989; 10:977–988
2. Harwood-Nash DC, Fitz CR: Neuroradiology in infants and children, vol 3. St Louis, Mo, Mosby, 1976, pp 1014–1019
3. Notaridis K, Ebbing P, Giannakopoulos C, et al: Neuropathological analysis of an asymptomatic adult case with Dandy-Walker variant. Neuropathol Appl Neurobiol 2006; 32:503–522
4. Timmann D, Daum I: Cerebellar contributions to cognitive functions: a progress report after two decades of research. Cerebellum 2007; 6:159–162
5. Berquin PC, Giedd JN, Jacobsen LK, et al: Cerebellum in attention-deficit hyperactivity disorder: a morphometric MRI study. Neurology 1998; 50:1087–1093

Organizational Role of Retina Horizontal Cells

To the Editor: In the complex organization of neural cells of the retina, there are five different cell types. One of them is the horizontal cell, which is connected laterally with the synaptic bodies of the rods and cones, as well as with the dendrites of the bipolar cells.[1] Most researchers claim that the outputs of the horizontal cells are always inhibitory and provide the "lateral inhibition" by sending feedback to cones negatively,[2] which is important in all sensory systems and helps to ensure the transmission of sensory patterns with proper contrast. However, it seems that more studies are needed to clarify the exact role of horizontal cells.

On the other hand, chaos synchronization in physical and biological systems has been widely studied over the last few years. Chaos, which is a universal phenomenon in nonlinear dynamics, exists in a variety of neural systems ranging from the simple to the complex. Chaotic oscillations of individual neurons may be responsible for many regular regimes of operation. Researchers have developed many neuronal models to simulate chaos of real neurons and obtained many significant results. In addition, experimental evidence demonstrates that synchronous neuronal oscillations underlie many cortical processes. Ensembles of neurons can synchronize in order to accomplish critical functional goals, such as the biological information processing or the production of regular rhythmical activity.[3,4] To achieve chaos synchronization of neuronal systems, many control methods, such as feedback control, have been developed.[4]

As a clue for synchronization in vertebrate retina, it can be emphasized that such a process has been seen in the inner plexiform layer of retina, where amacrine cells, bipolar cells, and ganglion cells have synapses, and it has been shown that amacrine cells play a synchronization role by giving feedback on ganglion cells for generating spontaneous activity in developing vertebrate retina.[5]

In the physiological view of human retina, such a synapse exists in outer plexiform layer between photoreceptors, horizontal cells, and bipolar cells. We hypoth-