ber following the sensations in about 50% of cases. Some parietal seizures may resemble panic attacks and be misdiagnosed as a psychiatric disorder, but showing poor response to antidepressants, as with our patient. Parieto-occipital seizures may be provoked by stimuli involving the receptive, interpretive, and connective function of the parietal and occipital lobes. The EEG may show focal discharges in the posterior regions, but the seizures are often electrically silent.[3] Even though MRI and functional imaging often reveal underlying pathology,[3] a normal exam, as in our patient, should not exclude the presence of organic lesion. The constructional apraxia, revealed as a disturbance in drawing without correct spatial relationships, existed despite the absence of apraxia for single movements.[6] The copying deficit was particularly evident in drawing three-dimensional figures, and was not simply one of formulating the sequence of steps necessary to produce a complex abstract form. This may be due to a difficulty in encoding the pictorial structure of a model depicting a three-dimensional object, suggesting the existence of a parietooccipital lesion. In summary, although a rare outcome from TBI, posttraumatic parieto-occipital epilepsy may occur even after minor head trauma and few risk factors, and respond poorly to anticonvulsants. Symptom presentation is pleomorphic, and can be misdiagnosed as psychiatric. The neuropsychological evaluation is useful in the diagnosis, especially when functional and neuroimaging exams show no anomalies, and may help establishing a causative role for TBI in producing neuropsychiatric disorders, which is important from clinical, scientific, and legal perspectives.

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Patau Syndrome

SIR: Patau syndrome is a congenital disorder which was reported by Dr. Klaus Patau in 1960.[1] The syndrome is caused by presence of an extra copy of chromosome 13. Other changes in chromosome 13, such as translocation, can also result in the characteristics classified as Patau syndrome. The syndrome causes serious physical and mental abnormalities, including neurological impairment, structural facial defects, heart defects, and mental retardation.

The incidence of Patau syndrome is approximately one per 12,000 live births.[2–4] The median survival age for children with the syndrome is 2.5 days. The most common cause of death is cardiopulmonary complications. Forty-five percent of the patients die within the first month of life and 70% die by 1 year of age. Survival to adulthood is extremely rare.

Case Report

We would like to report a 51-year-old Caucasian woman with Patau syndrome. Her chromosomal analysis showed partial translocation of chromosome 13 (unbalanced rearrangement between chromosome 13 and chromosome 14).

She had had 17 siblings in all. Eight of them died within the first few weeks of life. Another five died between the ages of 10 and 34 as a result of the medical complications of the syndrome; these siblings had mental retardation and psychosis. One living sibling, a 30-year-old man, suffered from mental retardation and psychosis. Three other siblings, two men and one woman, had no chromosomal abnormalities and are healthy.

The patient was born at full term with six toes on each foot, a small head, small eyes, low-set ears, and a cleft lip. Although all of her developmental milestones were delayed, she graduated from a special education high school. Her Wechsler Intelligence Scale for Children at 4 years old showed an IQ of 61 and moderate mental retardation. Her Wechsler Adult Intelligence Scale (WAIS) at 18 years old showed an IQ of 74 and educatable mental retardation while Rorschach testing showed severe immaturity. Her WAIS at 34 years old showed an IQ of 72 and borderline intellectual capabilities. The Halstead-Reitan Neurophysiological Battery showed serious neurological impairment. Her EEG showed complex partial seizures, and her computed tomography (CT) scan showed blunting of the frontal horns of the ventricular system.

Comment

What makes this case noteworthy, apart from the rarity of the syndrome and survival to adulthood, is the evolution of the psychosis, which is not a feature of Patau syndrome. The patient started to develop auditory hallucinations at the age of 15, for which she had multiple admissions and received different antipsychotic medications.

She started to decompensate after her antipsychotic medication was changed because of questionable seizures. She presented in the emergency room with agitated behavior. She was observed conversing with God, the devil, and her deceased siblings. She was delusional; she believed that people were taking her strength. The patient was medicated and admitted to the psychiatric floor. She was put on a regimen of valproic acid, 500 mg twice daily, aripiprazole, 20 mg daily, and olanzapine, 20 mg daily.

She was discharged to an assisted living facility after she stabilized. Since then, she has not had any hospitalization.

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Pisa Syndrome Resolved After Switching to Olanzapine

SIR: Pisa syndrome, or pleurothotonus, is a rare dystonic reaction commonly associated with prolonged antipsychotic medication. The treatment of Pisa syndrome at present is empirical, reflecting a poor understanding of its underlying pathophysiology.[1] The first-line treatment for Pisa syndrome remains a reduction in dose or discontinuation of antipsychotics, and the second-line treatment is an anticholinergic medication.[1] Here, we present a case of Pisa syndrome that developed during treatment with risperidone. Although both therapies were ineffective, the problem was resolved successfully after the medication was switched to olanzapine.

Case Report

"Mr. A," a 29-year-old man with a 3-year history of DSM-IV schizophrenia, had no history of head trauma or other neurological problems, and had no family history of dystonia or other movement disorders. In October 1999, he was admitted to the psychiatric unit for 9 months because of a severe psychotic exacerbation resulting in self-injuries of the tongue with scissors. A regimen of bromperidol, a maximum of 27 mg/day, and biperiden, 3 mg/day, was implemented with consequently good results. In the outpatient clinic, bromperidol was switched to risperidone at 4 mg/day, and then biperiden was discontinued.

In January 2001, 4 months after the initiation of risperidone, Mr. A was observed walking with a tilt toward the left. Physical examination showed tonic flexion of the trunk toward the left along with a slight backward axial rotation, classically referred to as Pisa syndrome. No evidence of other extrapyramidal symptoms was found. Secondary dystonias resulting from metabolic disorder, organic disorder, or infection were ruled out. The regimen of trihexyphenidyl was attempted for 4 weeks but no benefit was seen. A regimen of risperidone, 4 mg/day, was then reduced, step by step and cautiously, to 1 mg/day in 2 months. No improvement in Pisa syndrome was observed after 2-month observation with this dose. Because of the risk of psychotic relapse, risperidone, 1 mg/day, was successively switched to olanzapine, 5 mg/day, despite its discontinuation in August of 2001. Pisa syndrome gradually improved, and the symptoms disappeared within 2 months. Two years after olanzapine treatment, the patient was found to be in remission from the psychosis with no signs of Pisa syndrome.

Comment

The substitution to atypical antipsychotics, particularly clozapine, may provide alternatives for the treatment of patients with tardive dystonia.[2] Olanzapine, which has pharmacological similarities to clozapine, has been also suggested to be effective in the treatment of tardive dystonia.[3] For the treatment of Pisa syndrome, the usefulness of atypical antipsychotics has been mentioned only in a few case reports on clozapine[4] and amisulpride.[5] In this case, it remains unclear whether the clinical resolution of Pisa syndrome represents an antidystonic effect of olanzapine or simply a spontaneous remission of Pisa syndrome after the withdrawal of the offending drug, risperidone. Similar claims have been made for clozapine in the treatment of tardive dystonia.[4]

Although the pathophysiological mechanism is still unclear, this case