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A Report of Successful Treatment of Psychosis in Epilepsy With Risperidone

SIR: Several research studies[1] have assessed the prevalence of psychiatric disorders in epilepsy and reported that 2% TO 7% of patients with epilepsy have comorbid psychosis. Clinically, psychotic symptoms of epilepsy are categorized into ictal, posticta, and interictal psychoses.[2,3]

A family history of psychosis, earlier age of onset of epilepsy, complex partial seizures, temporal lobe lesion, and borderline intellectual functioning were the most identified risk factors for the development of interictal psychosis.[4–7] The onset of the psychotic symptoms in epilepsy is variable and the most common features are hallucinations and paranoid delusions.[8,9]

Case Report

A 42-year-old Caucasian male was diagnosed between the ages of 3 and 5 years with complex partial seizure disorder after a traumatic brain injury and he had received antiepileptic medications. The patient was the only sibling to a couple who did not have a family history of seizure disorder or other neurological disease or psychiatric illness. He had borderline intellectual functioning, received special education, attended vocational school, and was able to hold a part-time job.

At the age of 37, the patient experienced increased frequency of his seizure activity (6 to 8 incidents of activity per month), despite his adherence to the antiepileptic medication regimen. When his antiepileptic medications were adjusted by his neurologist, he reported a decrease in frequency of his seizure activity (none or 1 to 2 incidents per month).

At that time, he started having episodes of auditory hallucinations, hearing incoherent voices and paranoid delusions, and believing that people are against him. No history of affective flattening or thought blocking or lack of motivation or social withdrawal was reported.

His magnetic resonance image of the head was normal and his electroencephalogram indicated slowing and sharp waves originating in the right temporal region. His CBC and blood chemistries were within normal limits. His urine toxicology was negative.

The patient was hospitalized for the above-noted psychotic symptoms seven times in 3 years. He was prescribed different antipsychotic medications, in sufficient doses and for an appropriate duration of time, on both an inpatient and outpatient basis. Despite his compliance, he did not show a significant improvement in his psychotic symptoms, especially his paranoid delusions. A regimen of risperidone was prescribed for his psychosis and was titrated to 4 mg daily. The patient demonstrated amelioration of his psychotic symptoms and he has been free of psychosis for 2 years.

Comment

The mechanism by which epilepsy may be associated with the psychotic episodes is not well known; it is likely that structural brain abnormalities cause both epilepsy and psychosis.[3] Several studies reported that seizures modify the presentation of the psychosis; psychosis complicates epilepsy when seizures are frequent.[3]

Researchers reported that about 50% of epileptic patients with psychosis could be diagnosed with schizophrenia.[8] Psychosis of epilepsy is distinguishable from schizophrenia by the lack of negative symptoms of schizophrenia and better premorbid personality.[8]

Treatment of psychosis of epilepsy requires more careful pharmacological treatment, considering the ability of the antipsychotic medications to provoke seizures.[10]

In our case, risperidone was associated with a clinically significant improvement of the psychotic symptoms without exacerbation of the seizure activity.

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A Case of Idiopathic Basal Ganglia Calcification Presenting With Only Acting-Out Attacks and Mild Cognitive Impairment: PET Findings

SIR: A patient with idiopathic calcification of the basal ganglia manifested acting-out attacks and mild cognitive impairment beginning at age 34. Computed tomography (CT) showed bilateral calcifications in the putamen and globus pallidus consistent with the diagnosis of Fahr's disease. In contrast, reduced glucose uptake in positron emission tomography (PET) was not just confined to the areas mentioned above, but extended to the temporal and parietal and cerebellar regions. Our findings suggest that the cerebellum, striatum, and their cortical connections perform a global functional unit capable of influencing not only the motor behavior but also the cognitive and emotional functions.

Fahr's disease (basal ganglia calcification) is a rare neurodegenerative disorder characterized by bihemispherical calcium deposits, especially in the basal ganglia.[1] Its clinical manifestations are a rigid hypokinetic syndrome, psychiatric symptoms, and cognitive impairment.[1] Clinical diagnosis is facilitated by the presence of bilateral calcifications in the basal ganglia in CT. However, for the correct diagnosis, these calcifications may not be explained by any particular disorder of the calcium phosphorus metabolism or other diseases.

Case Report

The patient was admitted to the University of Uludag with acting-out attacks and difficulty in concentration. The patient also had episodes of violence precipitated by little provocation. During the examination the patient showed appropriate emotional display. The speech was found to be normal except that it was spoken in an aggressive voice and interrupted sometimes by word-finding difficulties. Psychiatric and neurological examinations were otherwise normal. By neuropsychological evaluation, the Benton test (Instruction F)[2] revealed mild to moderate disturbance in selective attention and concentration. On the Wechsler Memory Intelligence Scale, the patient exhibited mild to moderate impairment regarding logical memory, working memory, visual memory, and attention.

CT and magnetic resonance imaging (MRI) showed a bilateral calcification of the putamen and globus pallidus consistent with Fahr's disease, but no pronounced brain atrophy was observed. In contrast, reduced glucose uptake in PET was not only restricted to the left basal ganglia, but also involved the right temporoparietal and the right cerebellar cortices corresponding to the impaired cognitive flexibility, figural, and working memory. Secondary causes of the calcifications were excluded by laboratory testing.

Comment

The basal ganglia and cerebellum have traditionally been associated with motor performance. Though the neuronal activity within the basal ganglia and cerebellar loops with motor areas of the cerebral cortex were found to be highly correlated with parameters of movement, the aspects of cognitive function were found to be more related to the activity within the basal ganglia and cerebellar loops.[3]

In our case, functional abnormalities did not entirely parallel morphological changes, and were also found in the temporal parietal and cerebellar regions which appeared to be rather unaffected in CT and MRI.

This indicates that the reduced glucose uptake observed in the respective regions may reflect secondary deficits due to diminished functions of circuits involving the basal ganglia. However, in addition to suggesting the existence of an functional network between the ipsi and contralateral corticosubcortical regions, our findings also indicate the possible role of the cerebellum in emotional and cognitive functions.[4–6]

In conclusion, we present an atypical presentation of presumed cognitive impairment and emo-