met allele of the BDNF gene (BDNF polymorphism) on the vulnerability of depression, there is strong evidence regarding the effect of the BDNF polymorphism on cognitive performance, hippocampal volume, and the prefrontal cortex which have been implicated as important structures in mood regulation and memory.[4,5]

In our previous study evaluating the association of the BDNF gene val66met polymorphism with the serum BDNF levels in drug-free depressed patients, we showed that depressed patients had significantly lower mean values of serum BDNF relative to the comparison group ($p<0.05$). Beyond suggesting a significant negative correlation between the Hamilton Depression Rating Scale (HAM-D)-17 scores and serum BDNF levels in the study group, we also showed that depressed patients with met allele had significantly lower serum BDNF levels correlated with more severe depression scores ($p<0.05$).

Although we failed to support the BDNF polymorphism as a susceptibility factor in major depression, our results indicated a strong link between the BDNF polymorphism, the depression severity, and the serum BDNF levels in depressed patients, which suggests its role as a prognostic factor during depression. However, the dilemma of whether the BDNF polymorphism predisposes an individual to an earlier age of developing depression or only increases the risk in the context of other genetic risk factors for depression or other hippocampal impairment should be evaluated with further neuroimaging/genetic studies, including antidepressant treatment outcomes.

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Antipsychotic Induced Catatonia: A Case of Probable Dementia With Lewy Bodies

To the Editor: This report describes the case of a patient who presented with catatonia after rapid escalation of antipsychotic dose. The patient was eventually diagnosed with probable dementia with Lewy bodies (DLB).

Case Report

Mr. A was a 51-year-old man who presented with distressing visual hallucinations and paranoia. After outpatient psychiatric evaluation, risperidone, 0.5 mg twice daily, was changed to paliperidone, 6 mg once daily. After a single dose, he became confused and ataxic and was taken to the emergency room for evaluation. No medical causes for his symptoms were found, and he was admitted to an inpatient psychiatric unit.

His examination was notable for mutism, constricted affect, and lead-pipe rigidity of the upper extremities. His responses were limited to intermittent staring at examiners. When assisted to a standing position, he remained standing but would not move. Catatonia was diagnosed, and lorazepam, 1 mg twice a day, and aripiprazole, 5 mg once daily, were started. Paliperidone was discontinued.

The following day, his mutism persisted and he would not respond to multiple interview attempts. Although he made scant eye contact, he continued to be noninteractive. He required staff assistance with transferring from bed and continued to decline food intake. Vital signs were well within normal limits. Laboratory studies were normal. CT of the head revealed no parenchyma changes or other acute abnormalities. Aripiprazole was discontinued after a single dose due to concern for neuroleptic malignant syndrome (NMS), while lorazepam was increased to 2 mg three times a day. The patient became less responsive after a total of 8 mg of lorazepam, and he was transferred to the neurology service. Laboratory studies and CSF analysis were unremarkable. EEG showed diffuse slowing with no epileptiform discharges. Lorazepam was discontinued.

Over the subsequent 48 hours, the patient's level of consciousness improved to where he started to

converse minimally. Examination revealed bradykinesia, asymmetric rigidity, shuffling gait, and hypophonia. A history of cognitive decline was obtained, and a diagnosis of dementia with Lewy bodies (DLB) was made. His motor symptoms improved significantly, and he was eventually discharged with donepezil, L-dopa/carbidopa, and low-dose quetiapine.

Discussion

Dementia with Lewy bodies is considered the second most common cause of dementia after Alzheimer's disease, although it is still under recognized.[1] The extreme negativism and motor findings found in catatonia involve frontal and extrapyramidal systems.[2] Dementia with Lewy bodies patients are highly sensitive to antipsychotics because neurodegeneration of the same neurological pathways occurs. Catatonia and mutism in DLB patients have also been reported.[3,4] Paliperidone, the principal active metabolite of risperidone, is roughly equipotent to risperidone. The patient's previously undiagnosed DLB predisposed him to acute catatonia when an antipsychotic with a relatively high extrapyramidal side effect profile was increased by up to times six.[5] This is the first case in the literature that we are aware of that demonstrates a possible link between paliperidone and catatonia in a patient with DLB. This case affirms the importance of slow titration of high potency antipsychotics with special consideration for DLB in atypical presentations of psychosis.

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Obsessive-Compulsive Disorder Following Cavernous Sinus Thrombosis

To the Editor: Injury to the brain is known to result in psychiatric disorders. Several authors have reported obsessive-compulsive disorder (OCD) following different types of brain injury, including head trauma, cerebrovascular accidents, brain infections, and brain tumors.[1] Studies of OCD following cerebrovascular accidents are few and mostly constitute case reports of strokes.

We present a case of a 51-year-old man who developed OCD after cavernous sinus thrombosis and was successfully treated with paroxetine.

Case Report

The patient was admitted to a neurological clinic because of a severe headache and brought to an intensive care unit 12 hours later because he became lethargic. Brain MRI and magnetic resonance angiogram (MRA) (Figure 1) showed right cavernous sinus thrombosis and possible meningoencephalitis. He received broad-spectrum antibiotic therapy and low mol weight heparin for 6 weeks. Surgical sinus drainage was not performed. The patient was discharged 2 months after the admission with only ophthalmoplegia. A second MRI showed no evidence of meningoencephalitis or structural brain damage.

During his hospitalization the patient progressively developed obsessive symptoms, which prompted him to seek psychiatric help a few days after the hospital discharge. He had repeated thoughts that "something terrible," such as a thunderbolt, would cause him somatic damage and had thoughts to kill his wife with a knife. He recognized that his obsessions were unreasonable; however, they caused him excessive distress. No compulsive symptoms were present in any time of the disorder. There was no history of psychiatric illness or substance abuse in the patient or his family.

The patient received an axis I diagnosis of OCD according to DSM-IV criteria (Yale-Brown Obsessive Compulsive Scale score=17) and was treated with up to 60 mg/day of paroxetine. Two