paranoid and catastrophic delirious symptoms associated with logorrhea, irritability, excitability, and insomnia. He was admitted to our psychiatric unit. Physical examination indicated hypertension (180/100 mmHg, 100 b.b.p.). Neurological exploration, laboratory, and electrocardiography tests did not reveal any significant abnormalities. Sibutramine was suspended and quetiapine (400mg b.i.d.) and hidrosaluretil (50mg/day) were prescribed.

The delirious relapse remitted for over 3 weeks post-sibutramine discontinuation and the patient did not report any symptoms thereafter. He remained stable on a regimen of thioridazine (50mg/day) in posterior controls. Given that the symptoms began shortly after sibutramine was initiated or readministered, and were relieved after it was discontinued, sibutramine was thought to be the cause.

Comment

Since overweight and obesity have become emergent pathologies, classical treatment based on dietetic guidelines has improved with drug therapies. The heart valve problems arising from the use of fenfluramine and phentermine, raises questions about the safety and effectiveness of diet drugs. The FDA (Food and Drug Administration) took them off the market in 1997. Recently, French Pharmacovigilance Centres reported depression and suicidal tendencies associated with sibutramine in patients without such previous episodes.[5] In Spain, cautioned by some fatal cases in Italy, government agencies produced an advertisement outlining strict sibutramine prescription. This case report is confirmation that any ongoing use of sibutramine in psychiatric patients should be closely monitored during and prior to treatment, although the exact mechanism for this reaction in unknown.

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Aripiprazole-Associated Acute Dystonia

SIR: Aripiprazole is the latest addition to the armamentarium of antipsychotic agents available in the United States. It has been heralded as a third generation antipsychotic agent owing to its dual action as a dopamine autoreceptor agonist as well as the more conventional postsynaptic D_2 antagonist.[1]

This is the first report, to our knowledge, of acute dystonia associated with aripiprazole in an adult, although it has been described in a young adult before.[2]

Case Report

"Ms. W," a 19-year-old Caucasian woman with normal developmental milestones, considered bright by her parents, presented with a history of obsessive concerns about getting "sick," "poisoned," and "contaminated" which had started at about age 9. Her school performance progressively declined to the degree that she barely graduated from high school, despite home schooling. Her parents described lack of ambition, volition, and sociality, with an indifferent attitude towards their concern over her functioning—a change from her previous personality. Substance and organic history was negative. The patient had been treated with fluoxetine and therapy before, and was taking sertraline, 200mg/day, and clonazepam, 0.25mg b.i.d., at presentation, with no benefit. A diagnosis of schizophrenia, undifferentiated type, was made, and the patient was started on a regimen of aripiprazole, 15mg/day. Sertraline and clonazepam were continued with the intent that these would be tapered off after aripiprazole was started. After the third day of taking aripiprazole, the patient complained (by e-mail), "My neck wants to stay sideways all the time." She also complained of her lower jaw protruding to her right, the same side as her neck was turning to. There was no oculogyria or tongue dystonia. Ms. W stopped taking aripiprazole on her own and later reported that her side effects disappeared within 24 hours of discontinuation. There was no previous history of any movement disorder. She demonstrated the abnormal movements, consistent with acute dystonia, during a subsequent office visit.

Acute dystonia with atypical antipsychotic agents, although relatively rare, has been reported with other atypical agents, including with clozapine. Their 5-HT_{2A} antagonism, as a group, likely explains their low potential for extrapyramidal side effects.[3]

A likely explanation for acute dystonia in this case could be an increased plasma aripiprazole level

due to inhibition of cytochrome P4502D6 by sertraline, for which both are substrates.[4] Further, there is a generally decreased clearance of aripiprazole in women.[4] Since aripiprazole levels were not obtained, this hypothesis remains untested. Caution when using aripiprazole with sertraline or another P450D26 inhibitor, especially in women, would seem appropriate.

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Paroxetine-Induced Bruxism Effectively Treated With Tandospirone

SIR: Several case reports have shown that selective serotonin reuptake inhibitors (SSRIs) induced bruxism.[1] This letter reports a patient with paroxetine-induced bruxism who was effectively treated with tandospirone, a partial agonist of the 5-hydoxytryptamine $(5\text{-HT})_{1A}$ receptor. Tandospirone is less potent at dopamine D_2 receptor than buspirone.[2]

Case Report

"Mr. Y," an 81-year-old depressed Japanese man, received a 4-month trial of sulpiride, a common antidepressant medication used in primary care clinics in Japan. Since his depressive symptoms did not improve, he was referred to the psychiatry clinic. At that time, he experienced all nine criteria for major depressive disorder outlined in DSM-IV.[3] He agreed to a trial which replaced sulpiride with paroxetine.

A regimen of paroxetine was started with a bedtime dose of 10mg. After 7 days at this dosage with no reported adverse effects, the dosage was increased to 20mg/day. At the 14-day follow-up, depressive symptoms remained, although his mood had improved. The dosage was again increased to 30mg/day. At the 21-day follow-up, his depressive symptoms were under better control. He was very pleased with this treatment. However, he complained of "night-and-day jaw clenching" and "sore jaws and teeth." His wife also witnessed his symptoms. Within a week of adding tandospirone, 10mg p.o. t.i.d., to the regimen, he reported that his jaws no longer clenched and that sore jaws and teeth improved.

Comment

This case report shows that the patient developed bruxism after starting on a regimen of paroxetine. It has been postulated that disturbances in the central dopaminergic system, especially within the mesocortical tract, are linked to bruxism.[4] SSRI-induced bruxism is considered to be a consequence of serotonergically mediated inhibition of the dopaminergic system. In addition, in this case, elderly age and prior neuroleptic exposure (sulpiride: D_2 and D_3 antagonist) before starting paroxetine predisposed him to develop bruxism.

It has been reported that buspirone, a partial agonist of the 5-HT_{1A} receptor, ameliorates SSRI-induced bruxism.[1,4] This case suggests that tandospirone also improves paroxetine-induced bruxism. Several mechanisms can be considered in explaining why 5-HT_{1A} agonists relieve SSRI-induced bruxism. It has been shown that systematic administration of 5-HT_{1A} receptor agonists increases dopamine release in the prefrontal cortex.[5] This effect is considered a major factor in ameliorating bruxism.

The precise mechanism of how 5-HT_{1A} receptors regulate dopamine release is not known; however, several mechanisms have been postulated. Presynaptically, agonist activation of the somatodendritic 5-HT_{1A} autoreceptors in the raphe, which reduces 5-HT cell firing, synthesis, and release of 5-HT, is considered to increase dopaminergic neuron firing in the ventral tegmental area and synaptic release of dopamine in the prefrontal cortex.[4] Postsynaptic effects are also suggested since 5-HT_{1A} receptors are localized on nonserotonergic neurons in various brain regions, including the prefrontal cortex. It has been shown that dopamine release is modulated by postsynaptic 5-HT_{1A} receptors in the prefrontal cortex.[6]

This case adds to the literature by suggesting that SSRI-induced bruxism can be treated with tandospirone, a 5-HT_{1A} receptor agonist.

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