duced because of psychiatric symptoms, such as auditory hallucinations and diarrhea. On the 12th day of perospirone treatment, Mr. A suddenly became confused and obtunded with severe muscle rigidity. When he was admitted to a psychiatry department, he had a high fever, tachycardia, and severe extrapyramidal symptoms. Laboratory tests showed an elevation of creatine phosphokinase (33,878 IU/liter) and leukocyte levels (30.9×10^9/liter). A urine analysis showed myoglobinuria. A brain computerized tomography scan showed no remarkable change, and the possibility of infectious disease was excluded.

Mr. A had a fulminant neuroleptic malignant syndrome complicated with rhabdomyolysis and acute renal failure. On the third day of admission, he was transferred to an intensive care unit. He received dantrolene treatment and hemodialysis. On the 19th day, the fever disappeared, and his serum creatine phosphokinase level was normal (112 IU/liter). On the 44th day, his muscle rigidity was resolved, and no psychiatric symptoms were observed. He was discharged on the 46th day.

This case report shows that neuroleptic malignant syndrome can occur after a switch to perospirone treatment from other antipsychotics and anticholinergic withdrawal. Perospirone is a novel serotonin-dopamine antagonist and also a partial serotonin (5-HT_{1A}) agonist (1). The receptor binding profile and pharmacological property of perospirone resemble those of risperidone, and side effects, such as extrapyramidal symptoms, tend to occur less often with perospirone (2). This patient did not develop neuroleptic malignant syndrome during previous risperidone and olanzapine treatment with anticholinergics. It has been reported that abrupt withdrawal of anticholinergic agents is associated with neuroleptic malignant syndrome (3). Special caution with regard to concomitant drugs is therefore necessary when switching between antipsychotic drugs.

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Trimipramine for Refractory Panic Attacks

TO THE EDITOR: Trimipramine, a tricyclic tertiary amine pharmacologically related to imipramine, which was the first pharmacological agent noted to treat panic disorders (1), provides significant rapid antipanic effects with minimal side effects at a low dose. Tricyclic drugs are less widely used than selective serotonin reuptake inhibitors (SSRIs) because tricyclic drugs generally have more severe adverse effects at the higher doses required for effective treatment of panic disorders (2). This is a case report about a man with a 20-year history of panic disorders who was unresponsive to all agents tried, with the exception of imipramine, which was discontinued because of side effects. Tripramine at 50 mg/day induced remission of the panic attacks without adverse effects.

Mr. A, a 41-year-old white man, had suffered from panic attacks since age 21. According to Mr. A, the medications tried to no avail (either were ineffective or were discontinued because of side effects) included amitriptyline, bupropion, amoxapine, fluoxetine, sertraline, citalopram, paroxetine, fluvoxamine, duloxetine, escitalopram, carbamazepine, valproic acid, lithium, olanzapine, aripiprazole, olanzapine plus fluoxetine, aripiprazole plus fluoxetine, clonazepam, lorazepam, alprazolam, and combinations of alprazolam with multiple SSRIs, atypical neuroleptics, tricyclics, tetracyclics, and anticonvulsants.

Mr. A was initially seen while taking fluoxetine, 20 mg/day, in addition to 8 mg/day of alprazolam with olanzapine, 10 mg/day, and he continued to experience 1–2 panic attacks every 10 days. He had been taking alprazolam for 14 years. Fluoxetine and olanzapine were discontinued, and mirtazapine, 15 mg at night, was begun with the longstanding alprazolam, 2 mg/day. Mirtazapine caused stimulation and was discontinued. Quetiapine, 25 mg/day, was tried in conjunction with 2 mg/day of alprazolam. Quetiapine had to be discontinued because of gastrointestinal side effects. Desipramine was started at 10 mg at bedtime. The dose was increased to 20 mg at bedtime within a 2-week span, but it had to be discontinued because of overstimulation and worsening of the panic attacks. Trimipramine was begun at 25 mg/day, together with alprazolam, 2 mg/day. In view of no side effects, the dose of trimipramine was increased to 50 mg in the morning together with 2 mg/day of alprazolam. Within 5 weeks, Mr. A indicated he was "much calmer" and had not experienced any panic attacks. He remained free of panic attacks for 6 months. Ongoing tapering of alprazolam was being pursued.

On the basis of this case report, further use of trimipramine may be warranted in the treatment of panic disorders unresponsive to the more commonly used treatment modalities.

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