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Olanzapine-induced akathisia in OCD

Sir – Akathisia is a distressing side-effect of neuroleptic medication. As an atypical neuroleptic, olanzapine is less likely to cause extrapyramidal side-effects, and the prevalence of akathisia after treatment with olanzapine in clinical trials of patients with schizophrenia was about the same as after treatment with clozapine, at 6.3%.¹ There have been case reports of akathisia in patients with schizophrenia being treated with olanzapine, but none of akathisia in patients with other disorders receiving olanzapine. We present a case of akathisia in a patient with obsessive-compulsive disorder (OCD) receiving olanzapine. Akathisia resolved on discontinuation of olanzapine, and reappeared when the drug was recommenced.

Case report

A 30 year-old woman with severe OCD was admitted to hospital for treatment. Her symptoms included obsessive thoughts that "something bad will happen", and compulsions designed to ward off harm. These included performing bathroom rituals, walking in and out of doors, and writing the letter "n" (for "not") on walls. She also had repeating, counting, and checking compulsions. Rituals were repeated four times or in multiples of four. At its most severe, the disorder necessitated that the patient stay in the bathroom all night, performing endless rituals.

There were comorbid depressive symptoms intermittently. The patient did not have tics. There was no other psychiatric history, no medical history, and no family history of OCD or Gilles de la Tourette syndrome.

Initial treatment with clomipramine could not be tolerated by the patient who experienced severe side-effects. Paroxetine 50mg daily was unhelpful. At the time of her admission to hospital, the Yale-Brown Obsessive Compulsive Scale (YBOCS) score was 32. The paroxetine dose was maintained at 50mg, and olanzapine 10mg was added. This was titrated to 15mg and after two weeks of treatment there was a dramatic response with marked improvement of all OCD symptoms. YBOCS score was eight.

After three weeks of treatment however, severe akathisia developed, the patient scoring 24 on the Hillside Akathisia Scale (subjective and objective subscale scores of 12 and 12 respectively). There were no other extrapyramidal symptoms. Treatment with benztropine was unhelpful, and the patient self-discontinued olanzapine. All OCD symptoms returned, and the patient was as disabled as ever.

Olanzapine was recommenced, again with good effect on the OCD symptoms, and akathisia, which reappeared after a few days, was successfully treated with propranolol, 20mg bid. OCD symptoms remain in remission.

Significant OCD symptoms persist in 40%-60% of patients after a trial of a serotonin reuptake inhibitor (SRI).² There is evidence to suggest a role for antipsychotic drugs in the pharmacologic management of OCD. In a study of fluvoxamine augmented with the typical neuroleptic haloperidol, improvement occurred in 65% of patients with refractory OCD.³

There is less data regarding the use of atypical neuroleptics in refractory OCD. Preliminary reports suggest that

risperidone might alleviate OCD symptoms when added to an SRI, and in one study risperidone in addition to an SRI led to a significant improvement in 50% of SM-refractory OCD patients.

Olanzapine, another atypical neuroleptic, has been reported to improve OCD symptoms when added to fluoxetine in one case report. In clinical trials of olanzapine in patients with schizophrenia, akathisia has been reported. There have been only two reports describing individual cases of olanzapine-induced akathisia, and these also occurred in patients with schizophrenia.

To our knowledge, this is the first report of olanzapine-induced akathisia in OCD. SRIs have independently been reported to cause akathisia,⁷ however SRI-induced akathisia was felt to be unlikely in this case as the patient had been taking paroxetine for four months prior to the addition of olanzapine, without ill-effect.

In view of the very dramatic response to olanzapine in our patient, a role for olanzapine in the treatment of patients with refractory OCD is further suggested. Our report highlights both the advantages of a trial of an atypical neuroleptic in OCD patients, and the fact that atypical neuroleptics may cause akathisia in patients with OCD, as in patients with schizophrenia. Since neuroleptic-induced akathisia is frequently associated with compliance difficulties, it is important to consider the akathisia risk of the newer antipsychotic drugs, even though it is much lower than that found with typical neuroleptics.

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