gether with valproate because the latter decreases the clearance of lamotrigine.

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Electric Sensations: Neglected Symptom of Escitalopram Discontinuation

TO THE EDITOR: We make reference here to a patient who experienced electric sensations after discontinuing escitalopram treatment.

Ms. A was first referred to our clinic by her family doctor for depressive symptoms (asthenia, insomnia) in the context of a psychosocial stress situation. Treatment with citalopram, 20 mg/day, was initiated, which was soon replaced by escitalopram, 10 mg/day, the enantiomer of citalopram. Because she was responding well, Ms. A reduced her dose to 5 mg/day and, 3 weeks later, stopped treatment altogether. About a week later, she began to experience electric shock-like sensations or visual flashes lasting for about 1 second each. This was followed by a phase of spatial disorientation that lasted for about 30 seconds and was experienced as highly unpleasant and frightening. The sensations were only felt when Ms. A was in an upright position; she had no history of loss of tonicity. These episodes occurred up to three times a day over a period of 2 weeks. Prodromal symptoms or specific triggers were not reported. While Ms. A was taking citalopram/escitalopram, no side effects were observed. Interactions with other substances could be eliminated because this was her only medication.

Ms. A's depressive symptoms soon returned, resulting in more therapy with escitalopram (10 mg/day). She began to feel better and thus decided to reduce her medication, although it was well tolerated, to 5 mg/day and, after 4 weeks, again ceased taking it. One week later, the electric shocks reappeared, although they were now less intense. Ms. A's family doctor continued to urge pharmacological intervention to treat her depression. However, she refused to cooperate out of fear of becoming addicted to antidepressants because of the sensations that she interpreted as withdrawal symptoms. An EEG as well as a detailed neurological examination revealed no abnormalities.

There is not much relevant literature on the subject of electric sensations as a discontinuation symptom of selective serotonin reuptake inhibitors and nothing at all in connection with citalopram or escitalopram, although comparable symptoms have been described as occurring after treatment with paroxetine and sertraline (1). The pathophysiology is not clear; the down-regulation of serotonin 5-HT_2 receptors and the desensitization of both the 5-HT_2 receptor transmembrane signaling system and the 5-HT autoreceptors seem to be of particular concern (2). There are two therapeutic options: resuming the medication and tapering it off even more slowly (2) or allowing the syndrome to run its course. However, the patient would need to be reassured that the symptoms are likely to disappear within 2–8 weeks without serious consequences. None has been reported so far (3).

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Aripiprazole-Induced Movement Disorder

TO THE EDITOR: Aripiprazole is a dopamine D_2 partial receptor agonist (1). Trials have shown rates of extrapyramidal side effects similar to those of placebo administration, as seen with other atypical antipsychotic agents (1). Some case reports have found that atypical antipsychotics may even be helpful in treating tardive dyskinesia (2, 3). Although the occurrence of extrapyramidal symptoms seems to be less frequent with atypical antipsychotics, it is important to note that these effects can still occur. To our knowledge, only one case of aripiprazole-associated dyskinesia has been reported (4). We report a case of pseudoparkinsonism and rabbit syndrome that occurred in an antipsychotic-naive patient during treatment with aripiprazole.

Ms. A was a 27-year-old woman diagnosed with bipolar I disorder. Before she received care through our clinic, her condition was maintained with 150 mg/day of extended-release bupropion, 100 mg/day of lamotrigine, and 75 mg/day of extended-release venlafaxine. She had an emergence of manic symptoms and was administered aripiprazole, 10 mg/day. Ms. A had never been treated with an antipsychotic in the past. One month later, her dose of aripiprazole was increased to 20 mg/day. During the course of the next month, she was tapered from lamotrigine and venlafaxine to consolidate her drug regimen. She was seen for follow-up, where she complained of muscle stiffness and tongue movements that had grown increasingly worse over the last 2 months. She was observed by her outpatient psychiatrist to have muscle rigidity, a masked face, a shuffling gait, and orofacial movements consistent with rabbit syndrome.

It was decided to taper the aripiprazole and start ziprasidone, 60 mg b.i.d., along with benztropine, 1 mg b.i.d. Ms. A was seen 5 days later with a marked improvement in symptoms. She had no orofacial movements, decreased muscle rigidity, and only a slight tremor. At this visit, aripiprazole was completely stopped, and ziprasidone was increased to 80 mg b.i.d. Two weeks later, Ms. A was seen with no muscle rigidity or orofacial movements. Benztropine was discontinued, and her condition was maintained with ziprasidone without any reemergence of symptoms. When we evaluated these events with the