The psychiatry service was called to evaluate a patient in the emergency department (ED) for suicidal ideation secondary to restlessness. The patient was a 41-year-old woman with a history of depression who was taken to the ED directly from her outpatient psychiatry appointment, where she had stated she would rather die than deal with her “jitteriness and nausea.”

When she initially arrived in the ED, she was given a one-time oral dose of 8 mg of Zofran (GlaxoSmithKline, London, England) to relieve her nausea. The initial medical work-up included routine laboratory tests, abdominal X-ray series, tibia and fibula X-ray series, and venous Doppler ultrasound of the right leg. The results were all within normal limits except for a urine drug screen that was positive for opiates. This was explained by a one-time dose of hydromorphone given on arrival in the ED. However, the indication was not clear, as the patient did not complain of pain on arrival and she did not request pain medication.

Our initial evaluation revealed an obese woman lying down on her side with a staff sitter present in the examination room. Her grooming was suboptimal and possibly limited by her physical condition. She was restless, fidgety, and fretful. She was moving her arms and legs relentlessly and was not able to lie down for more than a minute without sitting up and then lying back down again. She was restless, fidgety, and fretful. She was moving her arms and legs relentlessly and was not able to lie down for more than a minute without sitting up and then lying back down again. She was cooperative but visibly frustrated by her constant need to move. She made no eye contact. There was no indication of disordered mentation. She described her mood as “depressed.” She became tearful when speaking about preferring death over continuing to feel the jitteriness of recent days. Her facial expressions reflected the emotions she reported. All thoughts appeared to be colored by her physical state. She had fair insight about her symptoms. Although somewhat dramatized by the patient, the core information she presented appeared to be accurate and valid for evaluation purposes.

Three days prior to this admission, she began feeling the need to constantly move. This feeling worsened in the ensuing days, to the point that she could not sleep and began to contemplate suicide to relieve her distress. She repeatedly said she did not want to “live like this (restlessness) anymore.” She also reported feeling that “ants [were] crawling all over” her. She denied recent drug or alcohol use and all previous urine drug screen results were negative.

The patient’s medical history included recent treatment for giant cell tumor, an endoprosthetic tibial replacement, and hypertension. She was also admitted to the same ED for nausea and vomiting 2 days prior to this admission and was discharged with a prescription for Zofran. In the days that followed, she could not tolerate any food or medications by mouth. Her home medications, which she reported taking daily, included 50 mg/day of trazodone for...

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insomnia and 20 mg/day of escitalopram for depression. She was also prescribed 50 mg/day of tramadol after tibial replacement surgery, which she had been taking intermittently.

She was medically cleared by the ED staff and, on our recommendation, was admitted to a medical unit for more evaluation due to her unresolved restlessness and stated wish for suicide. Prior to being transferred to the medical unit, she was given a one-time dose of 1 mg of lorazepam orally to decrease her restlessness. Soon after her first dose, she was subjectively and objectively less restless overall, although she was still seen turning on the bed frequently and not able to stay still. Escitalopram, tramadol, and trazodone were not restarted due to concern for possible serotonin syndrome as a possible, although unlikely, cause of her distress.

On the medical unit she required a continuous one-to-one staff sitter for the first 72 hours of admission due to concern that she may be actively suicidal.

The patient’s previous hospital psychiatry notes recorded a new diagnosis of major depression by the psychiatry consult service 6 weeks prior to this admission for cancer treatment. At that time, she endorsed a long history of depressive symptoms with passive suicidal thoughts. She was prescribed 10 mg/day of escitalopram, which had been increased to 20 mg/day 9 days prior to this admission.

On arrival to the medical unit, she was prescribed lorazepam at 1 mg twice a day. During the next 3 days she subjectively and objectively showed a gradual decrease in her complaints of restlessness. By day 4, her lorazepam dosage was decreased to 1 mg once per day. Her Barnes Akathisia Rating Scale (BARS) score1 (Global Clinical Assessment component) was 5 of 5 on the day of admission to the medical unit. This decreased to a score of 2 of 5 by day 3. It was notable that once her restlessness subsided she no longer complained of nausea or suicidal thoughts. On the day of discharge, her BARS Global Clinical Assessment score was 0 of 5. At this point, she endorsed no suicidal ideation and reported that her mood was “good.” At discharge she was advised to follow-up with her usual psychiatrist within 1 week to re-evaluate antidepressant medication because of her extensive history of depressive symptoms.

Therefore, we gave the patient a diagnosis of medication-induced acute akathisia. The Diagnostic and Statistical Manual of Mental Disorders, fifth edition2 identifies it as being “subjective complaints of restlessness, often accompanied by observed excessive movements (e.g. fidgety movements of the legs, rocking from foot to foot, pacing, inability to sit or stand still), developing within a few weeks of starting or raising the dosage of a medication (such as neuroleptic) or after reducing the dosage of a medication used to treat extrapyramidal symptoms.”

To date, there is a dearth of studies or case reports evaluating escitalopram-induced acute akathisia with secondary suicidal ideation. One previous case report describes a similar case.3

### DISCUSSION

In a review of the literature, 71 cases of selective serotonin reuptake inhibitor (SSRI)-associated extrapyramidal symptoms were found.4 Of these cases, akathisia was the most common, followed by dystonia, parkinsonism, and tardive dyskinesia–like states. It has been theorized to be caused by stimulating 5-hydroxytryptamine 2 receptors in the serotonin pathway that projects to the basal ganglia.5 This may cause a serotonin-mediated inhibition of dopamine release from the basal ganglia, resulting in a dopamine deficiency that leads to akathisia.

It is particularly interesting to note that most studies of SSRI-induced movement disorders have been associated with fluoxetine, sertraline, and paroxetine. These include one report of three patients...
who were re-exposed to fluoxetine after previously making a suicide attempt during fluoxetine treatment. They developed severe akathisia during re-treatment with fluoxetine and reported that the development of akathisia made them feel suicidal and precipitated their prior suicide attempts.

Only a few incidents regarding escitalopram and akathisia appear in the literature. By reporting this case, we hope physicians will become aware that, although rare, patients treated with escitalopram may develop akathisia. By being aware of the possibility of escitalopram-induced akathisia and treating it promptly, physicians will prevent the distressing and serious consequences of untreated akathisia in patients.

In this case, we have two important reasons to associate the patient’s symptoms with her increase in escitalopram dosage. First, her symptoms began 6 days after her dosage was doubled. This dose-dependent appearance of adverse effects is further validated by a study that shows adverse events occurring in the 20-mg/day escitalopram group with an incidence that was approximately twice that of the 10-mg/day group and approximately twice that of the placebo group. Second, the resolution of her symptoms after the administration of lorazepam and discontinuation of escitalopram is consistent with one previous case report in which akathisia resolved after 3 days of escitalopram discontinuation and treatment with diazepam. These studies support our assessment that the patient’s symptoms were caused by the increase in her escitalopram dosage.

The Naranjo adverse drug reaction probability scale (used to classify the probability that an adverse event is related to drug therapy) yielded a score of 6, which indicates a probable causal association.

Our case study has two main limitations with potential to affect the quality of our findings and our ability to answer any research questions. First, we did not obtain an escitalopram level. This may have helped us confirm the adverse event by providing objective daily levels to correlate with symptoms. This test was not available and it was impractical in terms of cost. Second, we did not ask the patient if she had ever had a similar adverse reaction to any other SSRI or similar medication. That information may have strengthened our case.

CONCLUSION

Escitalopram is an effective and widely used antidepressant, and akathisia is a rare side effect of its use. It should be suspected in any patient who presents with akathisia with recent initiation of or dosage increase of escitalopram. This case adds to the sparse literature on escitalopram-induced acute akathisia with secondary suicidal ideation.

REFERENCES