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Covert Dyskinesia With Aripiprazole: A Case Report

To the Editor: Covert dyskinesia is a masked form of tardive dyskinesia that emerges only after antipsychotic drugs are withdrawn or reduced in dosage. Covert dyskinesia is distinguished from other forms of withdrawal-emergent dyskinesia by its persistence for more than 3 months following discontinuation of antipsychotics.¹ Only 1 prior case has suggested its occurrence with aripiprazole.²

Case report. A 53-year-old man with history of depression and 8-year remission of alcohol, opiate, and cocaine abuse was referred for depressive symptoms associated with several recent emergency visits for reported low mood and suicidal ideation. He had 3 hospitalizations in the prior 4 years for similar symptoms but never had suicide attempts. His medical problems included hypertension, diabetes mellitus, obesity, hyperlipidemia, gastroesophageal reflux disease, history of deep vein thrombosis, pulmonary embolism, and a motor vehicle accident with no loss of consciousness or intracranial bleeding. He had prior trials of carbamazepine, bupropion, mirtazapine, and aripiprazole. He had taken aripiprazole for 2 years, targeting mood symptoms and impulsivity. He was maintained on mirtazapine 45 mg/d, bupropion extended release 150 mg/d, aripiprazole 15 mg/d, and trazodone 50 mg/d. His medical records for the prior 2 years showed no mention of abnormal movements, but no Abnormal Involuntary Movement Scale (AIMS)³ results were found. As his mood remained stable and he was having metabolic side effects, aripiprazole was tapered and discontinued over a period of 3 months between July and September of 2014. He was seen 6 weeks later with no significant mood problems or abnormal movements noted.

He presented for follow-up 3 months after cessation of aripiprazole in early January 2015 with new-onset involuntary repetitive lip smacking and chewing. He was edentulous, did not use dentures, and had no history of oral infection. His score on the AIMS was 4. His other medications included amlodipine 10 mg/d, fenofibrate 145 mg/d, glipizide extended release 10 mg/d, hydrochlorothiazide 25 mg/d, lisinopril 10 mg/d, melatonin 5 mg/d, metformin 1,000 mg/d, omeprazole 20 mg/d, and rivaroxaban 20 mg/d. His mood remained euthymic. At the next visit 8 weeks later, his dyskinesia persisted with an AIMS score of 5. He was lost to follow-up but was seen 8 months later with depression and suicidal ideation. He was now taking citalopram 40 mg/d, trazodone 50 mg/d, and haloperidol 15 mg/d (for reported psychosis). He had drooling, marked rigidity, and tremors in his arms and legs. No dyskinesia was seen, and his AIMS score was 0. His perioral movements had improved a few months prior, but he

was unsure if this followed taking haloperidol. Haloperidol was discontinued, and involuntary movements reemerged within 24 hours. His AIMS score was 11 five days later and 13 three weeks later with movements extending to the left hand.

The DSM-5⁴ defines withdrawal-emergent dyskinesia as lasting less than 4–8 weeks after neuroleptic cessation, while tardive dyskinesia persists beyond 2 months. On the basis of the time course of late emergence of the movement disorder 3 months after cessation of aripiprazole, our case is one of covert dyskinesia¹ that was suppressed by later use of haloperidol as expected for dyskinesia.

One prior report² described possible covert dyskinesia emerging only 2 weeks after discontinuation of aripiprazole. This briefer interval raises a question of separation from withdrawal dyskinesia. Our patient had at least 3 months without aripiprazole before emergence of the movements, which represents a clearer designation of covert dyskinesia than dyskinesia in the previously reported case.² Of note, their patient² shared similar risk factors for dyskinesia, including older age, long use of neuroleptics, and mood disorder. These cases suggest at least some risk for delayed and sustained movement disorders after prolonged use of aripiprazole.

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