LETTER TO THE EDITOR

Two Instances of Improvement in Tardive Dyskinesia After Administration of Aripiprazole in a Single Patient

To the Editor: Tardive dyskinesia (TD) is a serious involuntary movement syndrome characterized by purposeless, repetitive movements that involve several locations such as the buccolinguo-masticatory region.¹ It often develops as a result of taking antipsychotic drugs, and the prevalence rate reaches 20% with typical antipsychotics.^{2,3} The atypical antipsychotics have smaller risk, but a percentage of patients treated with them still develop TD.

Some physicians, especially the neurologists, have tried different strategies for this serious syndrome. There is literature available that presents several cases of improvement after aripiprazole therapy.^{4,5} Here, we report a patient who had improvement in TD with aripiprazole use.

Case report. Ms A, a 68-year-old woman who is prone to anxiety and obsession, was initially diagnosed with DSM-IV-TR major depressive disorder 15 years prior to this report. Due to persistent depression and anxiety symptoms, her previous doctor had prescribed low-dose sulpiride and flupentixol/melitracen as adjunctive therapy since 9 years ago (she had received antidepressant treatment with fluoxetine and venlafaxine in the past). She has fair compliance with drug treatment, but sometimes she adjusted the dosage herself.

The oral-lingual TD developed in April 2007, at which time she went to another hospital, where the physician discontinued all typical antipsychotics and administrated aripiprazole for treatment of dyskinetic symptoms after admission. Oral aripiprazole 3.75 mg at bedtime was initiated in June 2007. The dyskinetic symptoms were well controlled with the strategy a few weeks later. After she was discharged, the outpatient department doctor continued her aripiprazole treatment for about a month and then shifted to previous medications, including sulpiride.

The oral-lingual dyskinesia relapsed in February 2009, and the physician added risperidone to her regimen. The TD symptoms did not respond to risperidone, and Ms A started to visit our psychiatric section in September 2009. We arranged admission for her after evaluation in February 2010 due to persistent suicidal ideation and stopped the typical antipsychotic agent soon after admission. After a few days' observation, we noted that Ms A had severe and refractory depression (DSM-IV-TR major depressive disorder, recurrent, severe with psychotic features) and TD symptoms. We chose to add aripiprazole (10 mg once per day) as adjuvant therapy for depression. Her Hamilton Depression Rating Scale⁶ score diminished from 17 to 7 after 3 weeks' treatment. At the same time, we noted that her TD symptoms improved. Her score on the Abnormal Involuntary Movement Scale⁷ diminished from 25 to 6 after 4 weeks' treatment.

Aripiprazole is a unique atypical antipsychotic agent: its pharmacologic mechanism has a partial agonist effect on dopamine D₂ and D₃ and 5-HT_{1A} receptors and an antagonistic effect on 5-HT_{2A} receptors.^{8,9} It has alterative affinity to the dopamine receptor and may have the D₂ receptor up-regulation effect.¹⁰ Some suggested that the property of partial dopamine D₂ and D₃ agonist

of aripiprazole has a stabilization effect on dopamine receptors. This atypical antipsychotic agent has several possible mechanisms that can have a therapeutic effect on TD patients. There have been several clinical reports and some additional research,^{11–13} but there was no single principle or guideline for clinical use on treating TD patients. In this case, we used aripiprazole as adjunctive therapy to an antidepressant; it also helped to diminish TD symptoms. From her medical history at the previous hospital, we also noted that she had been treated before with aripiprazole. The combination of that outcome and the current one suggests that aripiprazole potentially has efficacy in patients with recurrent TD. However, more research is needed in this direction.

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