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USE OF ATYPICAL ANTIPSYCHOTICS AND MOVEMENT DISORDERS: A CASE OF ARIPIPRAZOLE INDUCED CHOREA

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We describe a 66-year-old woman with a 33-year history of treatment with antipsychotic drugs (she was reportedly affected by psychiatric symptoms, dominated by “depression”), who presented with severe, generalized chorea, which started after shifting from haloperidol to aripiprazole.

Since 1974, she had been treated with several antipsychotics (including haloperidol and risperidone) and antidepressants, in different combinations;moreover she underwent electroconvulsive therapy. In 2003, after shifting from haloperidol to aripiprazole, she presented facial dyskinesias, which progressively diffused to all the body regions and, after a few months. In 2006, aripiprazole was discontinued and olanzapine was introduced, with a dramatic increase of choreic dyskinesias. The picture was so severe that the patient could barely stand, walk, talk and eat.

MRI of the brain revealed minimal signs of diffuse atrophy and chronic leucoencefalopathy.Genetic testing for HD and the screening for Wilson's disease were negative; so the diagnosis was a severe tardive syndrome (orolingual dyskinesias, pelvic dyskinesias and generalized chorea).

Olanzapine (10 mg/die) was discontinued, and a low dose of clozapine (12,5 mg/die) was introduced, without any exacerbation of the psychiatric disorder, and without modification of chorea. After a few days, tetrabenazine was introduced and gradually titrated up to 75 mg/die in two weeks. After two weeks, a dramatic improvement of dyskinesias was evident (video).

To explain the case of our patient, we hypothesize that chronic administration of neuroleptics would lead to dopamine D2-receptor hypersensitivity in the nigrostriatal pathway and favor the agonist profile of aripiprazole to promote the activation of dopamine D2-receptors.

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