Dear Editor,

It is well known that some classes of neuropsychiatric drugs can cause secondary movement disorders. The class of drugs that is primarily associated with these adverse effects is the antipsychotics, but some antiepileptics and antidepressants, especially the selective serotonin recapture inhibitors, are also able to cause extrapyramidal syndromes. However, movement disorders are rarely described as a side effect of tricyclic antidepressants. The case described in this report represents a rare consequence of a tricyclic antidepressant.

An 8-year-old boy attended a local psychiatric service due to changes in behavior, including sadness, irritability, decreased interest in playing, disrupted sleep and diminished appetite. The symptoms began approximately 4 months previously, and even teachers at school noticed the change in his mood. Clinical examination and laboratorial exams were unremarkable. He was diagnosed with a major depressive episode, and imipramine 25 mg/day was initiated. Imipramine was chosen because tricyclics were the only class of antidepressants available in the Brazilian public health system when treatment was initiated. The patient had a positive response to the drug, and depressive symptoms remitted during the following 6 weeks. However, after 3 months, the boy presented dystonic involuntary movements characterized by sideways turning of the neck, mouth opening and twisted facial muscles. The movements were painful and interfered significantly with basic activities, such as eating and drinking. The patient did not report any urge to perform these movements. There was no history of neuropsychiatric disorders in his family. Computerized tomography and laboratory exams, such as complete blood count, electrolytes, tests for kidney, liver and thyroid disease, ceruloplasmin and acanthocyte analysis, were requested to rule out heredodegenerative diseases, and they were negative. The patient was only taking imipramine, and no other medication had been used during this period. He was diagnosed with transient drug-induced dystonia, and imipramine was immediately suspended. The dystonia remained for more than 2 days after the last intake. The boy attended follow-up appointments with both a psychiatrist and neurologist for over a year and remained free of abnormal movements and psychopathological changes.

Extrapyramidal symptoms (particularly tardive dyskinesia, akathisia, and Parkinsonism) induced by imipramine and other tricyclic antidepressants have already been described, but there is no report of dystonia with imipramine. Although drug-induced dystonia is a common adverse reaction to other classes of drugs, including some antidepressants, this side effect is rare with tricyclic antidepressants, possibly due to its intrinsic anticholinergic action. Thus, the pharmacodynamic explanation for this idiosyncratic reaction is unclear, but unknown genetic factors most likely played an important role in this case. One possible explanation that has been proposed to describe other rare cases of drug-induced dystonia is the contamination of the drug with other substances during manufacturing. This seems plausible, as Brazilian public health services purchase drugs that are either produced locally or imported from industries with different levels of manufacturing practices and quality control, and contamination can occur.

Physicians must be highly suspicious when psychiatric symptoms co-occur with movement disorders, and other primary diseases must be excluded. Additionally, this case report emphasizes the relevance of considering movement disorders as a possible side effect of tricyclic antidepressants, although the pharmacodynamic mechanism for this reaction is still unclear.

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