Acute-onset Orofacial Dyskinesia with a Single Low Dose of Oral Flupentixol: A Case Report

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ABSTRACT

Tardive dyskinesia are known to occur commonly among patients receiving neuroleptic drugs for prolonged periods. But, few reports of acute onset dyskinesia have also been reported in the literature. This report highlights one such case where the patient had dyskinetic movements with a single low dose of oral Flupentixol. Further, we examine the potential nosological status of acute onset dyskinesia associated with neuroleptic use.

Key words: Acute dyskinesia, flupentixol, single low dose, tardive dyskinesia

INTRODUCTION

Dyskinesias refer to abnormal involuntary movements. Tardive dyskinesia (TD), which commonly presents as oro-buccolingual movements or choreoathetoid movements of extremities, is associated with chronic antipsychotic treatment, especially the first-generation antipsychotics.[1] Rarely, have such movements been reported to occur acutely with the administration of neuroleptics.

CASE REPORT

A 35-year-old man was diagnosed as having a severe depressive episode without psychotic symptoms (International Classification of Diseases, Tenth Edition). There was no history suggestive of bipolarity or any other mental or physical symptoms. Neither was there any history of psychiatric or neurological illness in his family. He received trials of escitalopram (10–20 mg/day over 5 weeks), followed by fluvoxamine (50–200 mg/day over 4 weeks), without any significant improvement. Thereafter, sertraline 50 mg/day was started, with additional, flupentixol 0.5 mg/day for anxiety. Single dose of these medications was consumed at night, and by the next morning, he developed involuntary, rapid, to and fro movements of the lower jaw. No abnormal movements were noticeable in any other body parts. At this time, he was not receiving escitalopram or fluvoxamine. Sertraline and flupentixol were stopped immediately, but the movements continued unabated. These movements would occur throughout the day, except when asleep. There was...
no history of any movement disorders in this patient. He was examined by a neurologist, but no underlying cause could be revealed. He had not used any other dopamine-blocking agents. He underwent several investigations, including peripheral blood smear for acanthocytes, serum ceruloplasmin, urinary copper, and brain magnetic resonance imaging but no abnormality was detected. He was provisionally diagnosed as having flupentixol-induced dyskinesia and started on tetrabenazine 25 mg b.d. and clonazepam 0.5 mg t.i.d. No benefit was reported despite continued treatment for 1 month.

DISCUSSION

According to the Diagnostic and Statistical Manual of Mental Disorders Fourth Edition, Text Revision, TD occurs after neuroleptic exposure for at least 3 months (1 month if age ≥ 60 years). However, few case reports have described dyskinesia associated with short-term use (2–6 weeks) of antipsychotics (mostly in moderate- to high-oral doses). Only one case report so far, before ours, has described onset of dyskinesia (in a patient with intellectual disability) with a single dose of antipsychotic (flupentixol depot 50 mg intramuscular). However, unlike this report, our patient received a very low dose of flupentixol (0.5 mg orally) and had normal intelligence.

There are reports of escitalopram and fluvoxamine causing dyskinetic movements, but none of them have reported onset of such movements after stopping these drugs as happened in our case. Furthermore, there are no known reports of sertraline-associated TD. This makes it more likely that dyskinesia was associated with flupentixol, which is a known offender. Spontaneous dyskinesias, unrelated to neurological conditions, are also known to occur in patients with mental illness. However, the temporal association of the onset of dyskinesia with antipsychotic administration inclines us to think otherwise. For the same reason, and because of the absence of new stressors, and typical site and character of abnormal movements, dissociative symptoms were ruled out.

This case raises an important question: Is acute-onset dyskinesia a distinct clinical entity, different from acute-onset akathisia and dystonia, and TD? Clearly, the movement phenotype of dyskinesia is quite distinct from akathisia and dystonia, and the latter readily subside after stopping the offending drug, while the early- or acute-onset dyskinesia, as seen in most of the reports, including ours, tend to be persistent. However, the acute-onset dyskinesia is phenotypically similar to the tardive one, and little is known about its mechanism. TD has been theorized to result from increased receptor sensitivity or maladaptive neuronal plasticity. However, such changes occur over prolonged periods and are unlikely to explain the acute-onset dyskinesia.

This case highlights the need for research into the nosological status of neuroleptic-associated acute-onset dyskinesia and exploring neurobiological and treatment correlates. Furthermore, it demonstrates the importance of judicious use of antipsychotics as even low doses for short durations might cause disabling adverse effects.

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Conflicts of interest
There are no conflicts of interest.

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