Drug-induced Movement Disorders

Shyamal H. Mehta, MD, PhD[a], John C. Morgan, MD, PhD[b], Kapil D. Sethi, MD, FRCP (UK)[c,d,*]

KEYWORDS

• Movement disorder • Drugs • Dopamine receptor • Tardive dyskinesia
• Tardive Dystonia

KEY POINTS

- Drug induced movement disorders are most commonly associated with but not restricted to atypical & typical neuroleptics. Other drugs such as antidressants, antihistaminics and anti-arrhythmics, etc can also cause abnormal involuntary movements.
- Tardive dyskinesia (TD) can occur following a minimum of 3 months of neuroleptic expusure or even with a 1 month exposure in individuals over age 60.
- TD has not disappeared with the use of newer, more expensive antipsychotics; at higher doses, some of the newer atypical antipsychotics carry a substantially high risk similar to the older neuroleptics.
- It is important to distinguish classic TD from Tardive Dystonia due to treatment implications.

MOVEMENT DISORDERS CAUSED BY DOPAMINE RECEPTOR–BLOCKING AGENTS

The advent of atypical antipsychotics led to the hope that the incidence of drug-induced movement disorders will decrease significantly over time. However, the hope has not been realized, and the movement disorders caused by dopamine receptor–blocking agents (DBA) continue to be a significant problem. The introduction of chlorpromazine in 1952 was a major event in psychiatry but soon it became clear that this drug was associated with major significant acute side effects, such as akathisia, drug-induced parkinsonism, and acute dystonia.[1] A more persistent (late-appearing) dyskinesia was first recognized in the late 1950s.[2] Since then, extensive clinical experience has accumulated with the use of DBA, and the wide range of movement disorders that can be caused by these drugs (**Box 1**).

[a] Mayo Clinic Arizona, Scottsdale, AZ 85259, USA; [b] Georgia Health Sciences University, Augusta, GA 30912, USA; [c] Movement Disorders Program, Georgia Health Sciences University, Augusta, GA 30912, USA; [d] Merz Pharmaceuticals, 4215 Tudor Lane, Greensboro, NC 27410, USA
* Corresponding author. Movement Disorders Program, Georgia Health Sciences University, Augusta, GA 30912.
E-mail address: ksethi@gru.edu

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Box 1
Movement disorders induced by dopamine-blocking agents

1. Acute
   - Acute dystonia
   - Acute akathisia
   - Drug-induced parkinsonism
2. Chronic
   - Common:
     - Tardive dyskinesia
     - Tardive dystonia
     - Tardive akathisia
   - Uncommon:
     - Tardive myoclonus
     - Tardive tics
     - Tardive tremor
3. Miscellaneous
   - Neuroleptic malignant syndrome

ACUTE MOVEMENT DISORDERS CAUSED BY DOPAMINE RECEPTOR–BLOCKING AGENTS

Acute Dystonia

Acute dystonia occurs shortly after the introduction of DBA and occasionally after a dose increase or a switch to a more potent antipsychotic drug, particularly an injectable high-potency DBA. Often, there is a delay between the administration of the drug and the appearance of dystonia. About a half of patients experience the first signs of dystonia within 48 hours of drug intake, and in most patients signs appear within 5 days of drug initiation.[3] Acute dystonia is more likely to occur with typical DBA; however, the newer atypical drugs, including clozapine, are not devoid of this side effect.[4]

Dystonic reactions are variable in location and severity and are occasionally painful. The usual manifestations are orofacial dystonia, back arching, and neck extension. Life-threatening laryngospasm may occur.[5] Repeated acute dystonic reactions, in the absence of further exposure, has been observed even with a single dose of DBA. Acute dystonic reactions have rarely been reported with selective serotonin reuptake inhibitors (SSRIs), opioids, methylphenidate, rivastigmine, albendazole, gabapentin, cetirizine, foscarnet, quinine, and general anesthetics.[6] A form of subacute dystonic reaction appearing 3 to 10 days after starting DBA that results in truncal lateroflexion is called the Pisa syndrome (pleurothotonus).[7] However, Pisa syndrome may also be seen as a manifestation of Tardive Dystonia.[8]

An oculogyric crisis (OGC) is characterized by tonic conjugate ocular deviation that may last minutes to hours, and OGCs can occur in both acute and Tardive Dystonia.[9] Recurrent OGCs despite withdrawal of neuroleptic drugs are rare but have also been reported.[10] This finding may relate to the long half-life of these drugs or their metabolites.