# DIAGNOSTIC AND STATISTICAL MANUAL OF MENTAL DISORDERS

FOURTH EDITION

TEXT REVISION

# DSM-IV-TR™

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# 333.82 Neuroleptic-Induced Tardive Dyskinesia

## Diagnostic Features

The essential features of Neuroleptic-Induced Tardive Dyskinesia are abnormal, involuntary movements of the tongue, jaw, trunk, or extremities that develop in association with the use of neuroleptic medication. The movements are present over a period of at least 4 weeks and may be choreiform (rapid, jerky, nonrepetitive), athetoid (slow, sinuous, continual), or rhythmic (e.g., stereotypies) in nature. The signs or symptoms develop during exposure to a neuroleptic medication or within 4 weeks of withdrawal from an oral (or within 8 weeks of withdrawal from a depot) neuroleptic medication. There must be a history of the use of neuroleptic medication for at least 3 months (or 1 month in individuals age 60 years or older). Although a large number of epidemiological studies have established the etiological relationship between neuroleptic use and Tardive Dyskinesia, any dyskinesia in an individual who is receiving neuroleptic medication is not necessarily Neuroleptic-Induced Tardive Dyskinesia. The movements must not be due to a neurological or other general medical condition (e.g., Huntington's disease, Sydenham's chorea, spontaneous dyskinesia, hyperthyroidism, Wilson's disease), to ill-fitting dentures, or to exposure to other medications that can cause acute reversible dyskinesia (e.g., L-dopa, bromocriptine). The movements should also not be better accounted for by a neuroleptic-induced acute movement disorder (e.g., Neuroleptic-Induced Acute Dystonia, Neuroleptic-Induced Acute Akathisia).

Over three-fourths of the individuals with Tardive Dyskinesia have abnormal orofacial movements, approximately one-half have limb involvement, and up to one-quarter have axial dyskinesia of the trunk. All three regions are affected in approximately 10% of individuals. Involvement of other muscle groups (e.g., pharyngeal, abdominal) may occur but is uncommon, especially in the absence of dyskinesia of the orofacial region, limbs, or trunk. Limb or truncal dyskinesia without orofacial involvement is more common in younger individuals, whereas orofacial dyskinesias are typical in older persons.

## Associated Features

The symptoms of Tardive Dyskinesia tend to be worsened by stimulants, neuroleptic withdrawal, and anticholinergic medications and may be transiently worsened by emotional arousal, stress, and distraction during voluntary movements in unaffected parts of the body. The abnormal movements of dyskinesia are transiently reduced by relaxation and by voluntary movements in affected parts of the body. They are generally absent during sleep. Dyskinesia may be suppressed, at least temporarily, by increased doses of neuroleptics or sedatives.

The overall prevalence of Neuroleptic-Induced Tardive Dyskinesia in individuals who have received long-term neuroleptic treatment ranges from 20% to 30%. The overall incidence among younger individuals ranges from 3% to 5% per year. Middle-age and elderly individuals appear to develop Neuroleptic-Induced Tardive Dyskinesia more often, with prevalence figures reported up to 50% and an incidence of

25%–30% after an average of 1 year's cumulative exposure to neuroleptic medication. Prevalence also varies depending on setting, with Tardive Dyskinesia tending to be more common among inpatients (especially chronically institutionalized individuals). Variations in reported prevalence may be due to a lack of consistency in the definition of caseness, neuroleptic prescribing practices, study design, and the demographics of the population being studied.

There is no obvious gender difference in the susceptibility to Tardive Dyskinesia, although the risk may be somewhat greater in postmenopausal women. Greater cumulative amounts of typical neuroleptics and early development of extrapyramidal side effects are two of the most consistent risk factors for Tardive Dyskinesia. Mood Disorders (especially Major Depressive Disorder), neurological conditions, and Alcohol Dependence have also been found to be risk factors in some groups of individuals. There is growing evidence that the newer atypical neuroleptics are associated with a much lower incidence of Tardive Dyskinesia than the typical neuroleptics.

Onset may occur at any age and is almost always insidious. The signs are typically minimal to mild at onset and escape notice except by a keen observer. In a majority of cases, Tardive Dyskinesia is mild and is primarily a cosmetic problem. In severe cases, however, it may be associated with general medical complications (e.g., ulcers in cheeks and tongue; loss of teeth; macroglossia; difficulty in walking, swallowing, or breathing; muffled speech; weight loss; depression; and suicidal ideation). If the individual with Tardive Dyskinesia remains off neuroleptic medication, the dyskinesia remits within 3 months in one-third of the cases and remits by 12–18 months in more than 50% of cases, although these percentages are lower in older persons. When individuals receiving neuroleptic medication are assessed periodically, Tardive Dyskinesia is found to be stable over time in about one-half, to worsen in one-quarter, and to improve in the rest. Younger individuals generally tend to improve more readily; in older individuals there is a greater likelihood that Tardive Dyskinesia may become more severe or more generalized with continued neuroleptic use. When neuroleptic medications are discontinued, it is estimated that 5%–40% of all cases remit and between 50% and 90% of mild cases remit.

## Differential Diagnosis

Dyskinesia that emerges during neuroleptic withdrawal may remit with continued withdrawal from neuroleptic medication. If the dyskinesia persists for at least 4 weeks, a diagnosis of Tardive Dyskinesia may be warranted. Neuroleptic-Induced Tardive Dyskinesia must be distinguished from other causes of orofacial and body dyskinesia. These conditions include **Huntington's disease; Wilson's disease; Sydenham's (rheumatic) chorea; systemic lupus erythematosus; thyrotoxicosis; heavy metal poisoning; ill-fitting dentures; dyskinesia due to other medications such as L-dopa, bromocriptine,** or **amantadine;** and **spontaneous dyskinesias.** Factors that may be helpful in making the distinction are evidence that the symptoms preceded the exposure to the neuroleptic medication or that other focal neurological signs are present. It should be noted that other movement disorders may coexist with Neuroleptic-Induced Tardive Dyskinesia. Because spontaneous dyskinesia can occur in more than 5% of individuals and is also more common in elderly persons, it may be difficult to prove that neuroleptic medications produced Tardive Dyskinesia in a given individ-

ual. Neuroleptic-Induced Tardive Dyskinesia must be distinguished from symptoms that are due to a neuroleptic-induced acute movement disorder (e.g., **Neuroleptic-Induced Acute Dystonia** or **Neuroleptic-Induced Acute Akathisia**). Neuroleptic-Induced Acute Dystonia develops within 7 days and Neuroleptic-Induced Acute Akathisia develops within 4 weeks of initiating or increasing the dose of a neuroleptic medication (or reducing the dose of a medication used to treat acute extrapyramidal symptoms). Neuroleptic-Induced Tardive Dyskinesia, on the other hand, develops during exposure to (or withdrawal from) neuroleptic medication in individuals with a history of neuroleptic use for at least 3 months (or 1 month in middle-age and elderly persons).

### Research criteria for 333.82 Neuroleptic-Induced Tardive Dyskinesia

A. Involuntary movements of the tongue, jaw, trunk, or extremities have developed in association with the use of neuroleptic medication.

B. The involuntary movements are present over a period of at least 4 weeks and occur in any of the following patterns:

   (1) choreiform movements (i.e., rapid, jerky, nonrepetitive)
   (2) athetoid movements (i.e., slow, sinuous, continual)
   (3) rhythmic movements (i.e., stereotypies)

C. The signs or symptoms in Criteria A and B develop during exposure to a neuroleptic medication or within 4 weeks of withdrawal from an oral (or within 8 weeks of withdrawal from a depot) neuroleptic medication.

D. There has been exposure to neuroleptic medication for at least 3 months (1 month if age 60 years or older).

E. The symptoms are not due to a neurological or general medical condition (e.g., Huntington's disease, Sydenham's chorea, spontaneous dyskinesia, hyperthyroidism, Wilson's disease), ill-fitting dentures, or exposure to other medications that cause acute reversible dyskinesia (e.g., L-dopa, bromocriptine). Evidence that the symptoms are due to one of these etiologies might include the following: the symptoms precede the exposure to the neuroleptic medication or unexplained focal neurological signs are present.

F. The symptoms are not better accounted for by a neuroleptic-induced acute movement disorder (e.g., Neuroleptic-Induced Acute Dystonia, Neuroleptic-Induced Acute Akathisia).

## 333.1 Medication-Induced Postural Tremor

### Diagnostic Features

The essential feature of Medication-Induced Postural Tremor is a fine postural tremor that has developed in association with the use of a medication. Medications with