DIAGNOSTIC AND STATISTICAL MANUAL OF MENTAL DISORDERS

FOURTH EDITION

TEXT REVISION

DSM-IV-TR™

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Research criteria for 333.7 Neuroleptic-Induced Acute Dystonia

A. One (or more) of the following signs or symptoms has developed in association with the use of neuroleptic medication:

   (1) abnormal positioning of the head and neck in relation to the body (e.g., retrocollis, torticollis)
   (2) spasms of the jaw muscles (trismus, gaping, grimacing)
   (3) impaired swallowing (dysphagia), speaking, or breathing (laryngeal-pharyngeal spasm, dysphonia)
   (4) thickened or slurred speech due to hypertonic or enlarged tongue (dysarthria, macroglossia)
   (5) tongue protrusion or tongue dysfunction
   (6) eyes deviated up, down, or sideward (oculogyric crisis)
   (7) abnormal positioning of the distal limbs or trunk

B. The signs or symptoms in Criterion A developed within 7 days of starting or rapidly raising the dose of neuroleptic medication, or of reducing a medication used to treat (or prevent) acute extrapyramidal symptoms (e.g., anticholinergic agents).

C. The symptoms in Criterion A are not better accounted for by a mental disorder (e.g., catatonic symptoms in Schizophrenia). Evidence that the symptoms are better accounted for by a mental disorder might include the following: the symptoms precede the exposure to neuroleptic medication or are not compatible with the pattern of pharmacological intervention (e.g., no improvement after neuroleptic lowering or anticholinergic administration).

D. The symptoms in Criterion A are not due to a nonneuroleptic substance or to a neurological or other general medical condition. Evidence that the symptoms are due to a general medical condition might include the following: the symptoms precede the exposure to the neuroleptic medication, unexplained focal neurological signs are present, or the symptoms progress in the absence of change in medication.

333.99 Neuroleptic-Induced Acute Akathisia

Diagnostic Features

The essential features of Neuroleptic-Induced Acute Akathisia are subjective complaints of restlessness and at least one of the following observed movements: fidgety movements or swinging of the legs while seated, rocking from foot to foot or "walking on the spot" while standing, pacing to relieve the restlessness, or an inability to sit or stand still for at least several minutes. In its most severe form, the individual may be unable to maintain any position for more than a few seconds. The subjective complaints include a sense of inner restlessness, most often in the legs; a compulsion to move one's legs; distress if one is asked not to move one's legs; and dysphoria and anxiety. The symptoms typically occur within 4 weeks of initiating or increasing the dose of a neuroleptic medication and can occasionally follow the reduction of medi-

cation used to treat or prevent acute extrapyramidal symptoms (e.g., anticholinergic agents). The symptoms are not better accounted for by a mental disorder (e.g., Schizophrenia, Substance Withdrawal, agitation from a Major Depressive or Manic Episode, hyperactivity in Attention-Deficit/Hyperactivity Disorder) and are not due to a nonneuroleptic substance or to a neurological or other general medical condition (e.g., Parkinson's disease, iron-deficiency anemia).

Associated Features and Disorders

The subjective distress resulting from akathisia is significant and can lead to noncompliance with neuroleptic treatment. Akathisia may be associated with dysphoria, irritability, aggression, or suicide attempts. Worsening of psychotic symptoms or behavioral dyscontrol may lead to an increase in neuroleptic medication dose, which may exacerbate the problem. Akathisia can develop very rapidly after initiating or increasing neuroleptic medication. The development of akathisia appears to be dose dependent and to be more frequently associated with particular neuroleptic medications. Acute akathisia tends to persist for as long as neuroleptic medications are continued, although the intensity may fluctuate over time. The reported prevalence of akathisia among individuals receiving neuroleptic medication has varied widely (20%–75%). Although the atypical neuroleptic medications are less likely to cause akathisia than the typical neuroleptics, nonetheless, these medications do cause akathisia in some 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.

Differential Diagnosis

Neuroleptic-Induced Acute Akathisia may be clinically indistinguishable from syndromes of restlessness due to certain neurological or other general medical conditions, to nonneuroleptic substances, and to agitation presenting as part of a mental disorder (e.g., a Manic Episode). The akathisia of **Parkinson's disease** and **iron-deficiency anemia** are phenomenologically similar to Neuroleptic-Induced Acute Akathisia. The frequently abrupt appearance of restlessness soon after initiation or increase in neuroleptic medication usually distinguishes Neuroleptic-Induced Acute Akathisia.

Serotonin-specific reuptake inhibitor antidepressant medications may produce akathisia that appears to be identical in phenomenology and treatment response to Neuroleptic-Induced Acute Akathisia. Akathisia due to nonneuroleptic medication can be diagnosed as **Medication-Induced Movement Disorder Not Otherwise Specified.** Other situations that might be included under Medication-Induced Movement Disorder Not Otherwise Specified are acute akathisia with only subjective or only objective complaints, but not both; and akathisia occurring late in the course of treatment (e.g., 6 months after initiation of, or increase in the dose of, a neuroleptic). **Neuroleptic-Induced Tardive Dyskinesia** also often has a component of generalized restlessness that may coexist with akathisia in an individual receiving neuroleptic medication. Neuroleptic-Induced Acute Akathisia is differentiated from Neuroleptic-Induced Tardive Dyskinesia by the nature of the movements and their relationship to the initiation of medication. The time course of symptomatic presentation relative to neuroleptic dose changes may aid in this distinction. An increase in neuroleptic med-

ication will often exacerbate akathisia, whereas it often temporarily relieves the symptoms of Tardive Dyskinesia.

Neuroleptic-Induced Acute Akathisia should be distinguished from symptoms that are better accounted for by a mental disorder. Individuals with **Depressive Episodes, Manic Episodes, Generalized Anxiety Disorder, Schizophrenia and other Psychotic Disorders, Attention-Deficit/Hyperactivity Disorder, dementia, delirium, Substance Intoxication** (e.g., with cocaine), or **Substance Withdrawal** (e.g., from an opioid) may also display agitation that is difficult to distinguish from akathisia. Some of these individuals are able to differentiate akathisia from the anxiety, restlessness, and agitation characteristic of a mental disorder by their experience of akathisia as being different from previously experienced feelings. Other evidence that restlessness or agitation may be better accounted for by a mental disorder includes the onset of agitation prior to exposure to the neuroleptic medication, absence of increasing restlessness with increasing neuroleptic medication doses, and absence of relief with pharmacological interventions (e.g., no improvement after decreasing the neuroleptic dose or treatment with medication intended to treat the akathisia).

Research criteria for 333.99 Neuroleptic-Induced Acute Akathisia

A. The development of subjective complaints of restlessness after exposure to a neuroleptic medication.

B. At least one of the following is observed:

   (1) fidgety movements or swinging of the legs
   (2) rocking from foot to foot while standing
   (3) pacing to relieve restlessness
   (4) inability to sit or stand still for at least several minutes

C. The onset of the symptoms in Criteria A and B occurs within 4 weeks of initiating or increasing the dose of the neuroleptic, or of reducing medication used to treat (or prevent) acute extrapyramidal symptoms (e.g., anticholinergic agents).

D. The symptoms in Criterion A are not better accounted for by a mental disorder (e.g., Schizophrenia, Substance Withdrawal, agitation from a Major Depressive or Manic Episode, hyperactivity in Attention-Deficit/Hyperactivity Disorder). Evidence that symptoms may be better accounted for by a mental disorder might include the following: the onset of symptoms preceding the exposure to the neuroleptics, the absence of increasing restlessness with increasing neuroleptic doses, and the absence of relief with pharmacological interventions (e.g., no improvement after decreasing the neuroleptic dose or treatment with medication intended to treat the akathisia).

E. The symptoms in Criterion A are not due to a nonneuroleptic substance or to a neurological or other general medical condition. Evidence that symptoms are due to a general medical condition might include the onset of the symptoms preceding the exposure to neuroleptics or the progression of symptoms in the absence of a change in medication.