Drug-induced movement disorders

Marcel Dekker - Itqān fī ʻulūm al


Table 3
Risk Factors for Drug-Induced Movement Disorders

Akathisia	Tardive Dyskinesia	Dystonia	Parkinsonism
Advanced age	Advanced age	*Acute Dystonia*	Acquired immune
Affective disorder	Affective disorder	High-potency neuroleptics	deficiency syndrome
Cognitive impairment	Alcoholism	History of electroconvulsive	Advanced age
Female sex	Diabetes mellitus	therapy	Dementia
High-potency neuroleptics	Duration of treatment	Male sex	Female sex
High neuroleptic dosage	Electroconvulsive treatment	Mental retardation	
History of akathisia	Female sex	Young age	
Iron deficiency	History of extrapyramidal	*Tardive Dystonia*	
Mental retardation	reaction	Male sex	
Negative symptoms	Intermittent neuroleptic	Presence of tardive	
of schizophrenia	treatment	dyskinesia	
Rapid neuroleptic	Iron deficiency	Young age	
dosage escalation	Mental retardation		
	Organic brain disorder		
	Total daily drug dosage		

Description: -
-
New York (N.Y.) -- Hospitals
Womens hospitals -- New York (State) -- New York
Dyskinesia, Drug-Induced.
Tardive dyskinesia -- Etiology.Drug-induced movement disorders
-
Neurological disease and therapy -- v. 62Drug-induced movement disorders
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. In the majority of cases, DIP is a subacute process that develops within three months of initiating the offending agent or, in some cases, after a dosage increase, and it is slowly reversible upon drug withdrawal.

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Anticholinergic prophylaxis of acute haloperidol-induced acute dystonic reactions. For patients with severe, refractory EPS, clozapine may be used specifically to treat the EPS, if they are judged to be severe enough to be disabling or potentially life threatening. Conventional neuroleptics and phenothiazine antiemetics are most commonly implicated.

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Akathisia: a review and case report following paroxetine treatment.

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Acute dystonic reactions peak at 24 to 48 hours from the initiation of therapy.

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In the patient experiencing acute akathisia, the causative agent should be discontinued, if possible.

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