

Management of drug-induced movement disorders in psychiatry: an update

Abstract

Drug-induced movement disorders (DIMD) represent a variety of iatrogenic and clinically distinct movement disorders, including akathisia, tardive dyskinesia, dystonia, and Parkinsonism. DIMD remain a significant burden especially among certain patient populations receiving psychotropic medication. Knowledge of DIMDs will allow clinicians and healthcare professionals to better identify and manage patients with these conditions. Here we discuss the important features and current practical management steps of different types of DIMD in psychiatry.

Keywords: Akathisia. Tardive Dyskinesia. Dystonia. Parkinsonism. Neuroleptic. Antidepressant.

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Introduction

Drug-induced movement disorders (DIMD) are a group of neurologic motor disturbances associated with the use of neuroleptic agents.[1] Based on the duration they are mainly classified into three groups. Firstly, there are acute onset movement disorders, typically occurring on drug initiation and less commonly with changes in dose, for example acute dystonic reactions and neuroleptic malignant syndrome (NMS). Secondly, that emerges during drug treatment but stop when the drug is withdrawn, for example drug-induced Parkinsonism (DIP) or tremors. Thirdly, the 'tardive' movement disorders (TMD), emerging typically after chronic treatment and often not remitting, even when the drug is stopped. The drugs associated with DIMD[2] include antipsychotics, antidepressants,[3] antiepileptics, antihistaminics, antiemetics, central anticholinergics, dopamine agonists, and mood stabilisers. DIMD are disabling and disfiguring, may result in noncompliance and subsequent relapse and rehospitalisation. They can rarely be life threatening, particularly when affecting respiratory or bulbar muscles.[4] The fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5)[5] lists

eight categories of medication-induced movement disorders. The DSM-5 categories include: 1) Neuroleptic-induced parkinsonism, other medication-induced parkinsonism; 2) NMS; 3) Medication-induced acute dystonia; 4) Medication-induced acute akathisia; 5) Tardive dyskinesia (TDk), tardive dystonia (TDt); 6) Medication-induced postural tremor; 7) Other medication-induced movement disorder; 8) Antidepressant discontinuation syndrome; 9) Other adverse effects of medication.

Being an iatrogenic condition, prevention and early recognition of the symptoms comprise the best method of managing DIMD. Best preventive strategies should include weighing risk and benefit of treatment before instituting therapy with a drug known to cause DIMD.

Drug-induced Parkinsonism

DIP is characterised by the classic triad of tremor, rigidity, and bradykinesia. Other features like difficulty in rising from a seated position, gait imbalance, masked facies, micrographic, slow shuffling gait, and stooped posture may be observed. It usually developed days to weeks after

initiating of antipsychotic drugs treatment, mainly typical antipsychotics.[2]

Management

The best treatment of DIP is prevention, including the avoidance of prescription of causative drugs whenever it is not strictly necessary. Special precautions are needed in patients who are at risk. DIP remits slowly after drug withdrawal or reducing the dose, but some patients may develop persistent symptoms. Other options include change to another drug with lower propensity for DIP. Second generation neuroleptics are associated with lower risk than first generation antipsychotic drugs.[6] Anticholinergic agents, such as benztropine, amantadine (especially in elderly or in patients already having early signs of Parkinson's disease), or diphenhydramine are prescribed if symptoms persist. Night dose prescription of anticholinergic agents is not recommended as the symptoms are usually absent during sleep. Nicotine and nicotinic receptor drugs have been tried recently in patients with DIP with variable success.[7] Use of drugs should be reviewed regularly. Slow withdrawal should be attempted after the acute phase of treatment or following any lowering of antipsychotic dose. Prophylactic use of antiparkinsonian agent at the start of neuroleptic treatment remains controversial.[8]

Neuroleptic malignant syndrome

The syndrome, first reported in 1968 by Delay and Deniker, is considered as a most serious potentially life-threatening but also the rarest and least known complication of neuroleptic chemotherapy.[9] The four defining features that characterise NMS are hyperthermia, altered mental status, motor symptoms, and autonomic instability. The onset of symptoms varies from hours to days. The diagnosis is often missed in the early stages, and the withdrawal or agitation may mistakenly be considered to reflect an exacerbation of the psychosis. The important differential diagnosis includes infectious diseases (meningitis or encephalitis), psychiatric or neurological (e.g. idiopathic malignant catatonia), toxic or pharmacological (e.g. malignant hyperthermia, serotonin syndrome), endocrine (e.g. thyrotoxicosis, pheochromocytoma), environmental (e.g. heatstroke).[10]

Management

Supportive therapy: In mild cases it is self-limited after cessation of antipsychotic medication. Most of the individual recover within one week and nearly all within 30 days. Medical management required only in complicated cases. The offending agent must be withdrawn immediately. Volume resuscitation, correction of electrolyte abnormalities, physical cooling and serial monitoring should be aggressive. Intensive medical care may involve support of cardiac, respiratory, and renal functions in severe cases.[10-12]

Pharmacological treatments: The most commonly used medications for the condition are dantrolene 1-2.5 mg/kg body weight administered intravenously and bromocriptine 2.5 mg orally two or three times a day, although amantadine is sometimes used.[13] Benzodiazepines should be considered if not already prescribed; 1-2 mg parenteral lorazepam has been used.[14] Levodopa, apomorphine,

and carbamazepine have also been used among many other drugs.[15] Electroconvulsive therapy (ECT) may be effective if symptoms are refractory.

Rechallenge and recurrence: Antipsychotic treatment will be required in most instances and rechallenge is associated with acceptable risk. Antipsychotics should be withheld for at least five days, preferably longer. Consider using an antipsychotic structurally unrelated to that previously associated with NMS. Avoid depots (of any kind) and high-potency conventional antipsychotics.[12]

Acute dystonia

It was first reported in the United States in 1960. Dystonias are brief or prolonged contractions of muscles that result in obviously abnormal movements or postures, including oculogyric crises, tongue protrusion, trismus, torticollis, oromandibular dystonias, and dystonic postures of the limbs and trunk. It can be life-threatening in some cases if laryngeal muscles are affected.[4] Symptoms usually develop within days of starting or increasing the oral antipsychotic dose. Differential diagnosis includes catatonia, Huntington's disease, Wilson's disease, or conversion disorder.[16,17]

Management

Acute dystonia resolves when the offending drug is discontinued or the dose is reduced or switching to an antipsychotic with a low propensity for extrapyramidal symptoms (EPS). In severe cases dystonia can be effectively relieved with a short course of a potent antimuscarinic agent such as benztropine, diphenhydramine, and trihexyphenidyl, administered orally, intramuscularly, or intravenously.[16,17] For focal dystonic symptoms, local injections of botulinum toxin are preferred. In refractory cases, intrathecal baclofen, deep brain stimulation, ablative pallidotomy, or intrathecal pump implantation has been tried with some success.[11,18]

Drug-induced akathisia

The phenomenon of akathisia (from the Greek meaning "never to sit down") was first described by Haskovec (1904). It is described as subjective feelings of restlessness, objective signs of restlessness, or both. The core features are subjective complaints of restlessness on the legs. It usually develops within first four weeks of starting or increasing the dose of medication. It has been associated with aggression, suicidal behaviour, and treatment non adherence.[19]

Management

When left untreated, the symptoms of acute akathisia may gradually subside or wax and wane over time. Reduction of dose or switching to another less potent first generation antipsychotic or second generation antipsychotics are one of the first options.[12] Administration of a lipophilic β -blocker, such as propranolol (30-120 mg/day), is effective and well-tolerated. Nevertheless, a Cochrane review reports of insufficient data to recommend β -blocking drugs for akathisia.[20] Administration of antimuscarinic agents (e.g. benztropine, diphenhydramine), benzodiazepines are also found to be effective. Serotonergic agonist or antagonist

(ritanserin, cyproheptadine, zolmitriptan) may be preferred if sedation is desired. Miscellaneous agents like amantadine, clonidine, mianserin, mirtazapine, trazodone, carbamazepine, gabapentin, pramipexole, cabergoline, and clozapine have been suggested to be effective in patients with chronic and persistent akathisia.[21,22]

Drug-induced tremor

Tremor is an easily recognisable movement disorder, which can be sub acutely induced by numerous psychotropic drugs.[3] Well-known causative agents are neuroleptics, sympathomimetics, tricyclic antidepressants, selective serotonin re-uptake inhibitors (SSRI), lamotrigine, lithium, and valproate.[1]

Management

Treatment is required if the tremor becomes bothersome to the patient and affecting social or occupational functioning. The therapeutic strategies include use of lowest possible dose, reducing the dose or stop the drug if possible. Use of sustained release drugs, switching to an effective less tremorogenic drugs, and taking medicines at bedtime are also a good therapeutic choice in some patients. Among pharmacological treatment, β -blocker such as propranolol is the most commonly used drug, while primidone, benzodiazepines, gabapentin, and topiramate are occasionally used.[23]

Rabbit syndrome (perioral tremor)

Rabbit syndrome (RS) is a rare movement disorder generally associated with prolonged use of first generation neuroleptics, either as monotherapy or in combination with anticholinergic agents.[24] It is characterised by fine, rapid, rhythmic movements of oral and masticatory muscles along the vertical axis of the mouth.[25]

RS typically responds favourably to anticholinergic agents such as benztropine, biperiden, procyclidine, and trihexyphenidyl.[25] RS disappears a few days after the introduction of an anticholinergic agent, but may reappear after stopping anticholinergic medications. The other option includes reducing the dose or change of antipsychotic agent.

Drug-induced tardive syndromes

Tardive syndromes (TS) encompass a broad spectrum of abnormal involuntary movements due to chronic exposure to dopamine receptor blocking agents;[26] may occur with other agents such as antiepileptics, lithium, oral contraceptives, and SSRI. The TS include TDk, TDt, tardive akathisia (TA), tardive tremor, tardive tics or tourettism, and tardive myoclonus.[27]

Tardive dyskinesia

It was first reported in the United States in 1960. The disorder consists of abnormal, involuntary, irregular choreoathetoid movements of the muscles of the head, limbs, and trunk. TDk may also occur after discontinuation of neuroleptic or reduction in dose in which case the condition is called neuroleptic withdrawal emergent-dyskinesia. The condition is usually time limited lasting less than four to eight weeks.[5]

Factors associated with exacerbation of TDk symptoms include administration of antimuscarinics or sympathomimetics, stimulants, and emotional extremes. As with most dyskinesias, symptoms subside during sleep, and in mild cases, patients may be unaware of the movements. After TDk develops, remission rates are low if the antipsychotic is continued. Most common pattern is mild to moderate severity with waxing and waning course.[26]

Management

Prevention is the best strategy which includes identifying the risk factors, early diagnosis, and use of antipsychotic medications only when clearly indicated, that too in the lowest effective doses. Once TDk is recognised, withdrawal of any anticholinergic drugs and a reduction in the dose of antipsychotic drugs has been recommended as initial steps. However, Cochrane found little support for this approach and the American Academy of Neurology does not recommend it.[28,29] Alternatively, the clinician may switch the patient to clozapine or change to an antipsychotic with lower propensity for TDk.[30] A controversial strategy for treating TDk is to continue or increase the dose of the dopamine antagonist. Switching or withdrawing antipsychotics is not always effective and so additional agents are often used. Most frequently prescribed additional drugs for treatment of TDk is propranolol, clonazepam, tetrabenazine. The evidenced-based guidelines of the American Academy of Neurology recommend the use of clonazepam and ginkgo biloba.[31,32] Other therapeutic agents for which there is some anecdotal support include vitamin E, levodopa, benzodiazepines, botulinum toxin, reserpine, tetrabenazine, propranolol,[26] and dopamine-depleting agents. Use of ondansetron, amantadine, levetiracetam, pregabalin, gabapentin, valproate, vigabatrin, donepezil, fish oils, pyridoxine (B₆), and baclofen have been suggested based on small open labelled trials and case reports. Recently, there has been a resurgence of stereotactic pallidal surgery. Although the efficacy of both pallidotomy and deep brain stimulation (sub thalamic nucleus, globus pallidus) has been encouraging, the evidence is still limited.[33]

Tardive dystonia

TDt develops after months to years of treatment with a neuroleptic or within three months after treatment discontinuation and many often coexists with TDk. Generally the diagnosis of TDt can be made in the presence of chronic dystonia (persistent for more than one month), clear history of neuroleptic use, and absence of a general medical or neurologic condition that may account for dystonia. Symptoms may or may not be painful and may be isolated to one body part or spread to contiguous body parts (segmental dystonia) or even generalise to multiple body parts.[34]

Antimuscarinics such as trihexyphenidyl is recommended, a maximum dose of 32 mg of trihexyphenidyl. The dopamine-depleting agents, such as reserpine and tetrabenazine, are considered the most effective drugs. Clozapine has been reported to be effective in the treatment of idiopathic and in TDt. In medically intractable cases of TDt, alternatives such as botulinum toxin injections, ECT,

pallidotomy, thalamotomy, and intrathecal baclofen can be employed.[32,34]

Pisa syndrome

It is a rare form of dystonia associated with neuroleptic treatment characterised by sustained truncal lateroflexion with backward rotation that gets exacerbated while walking.[35] Symptoms of Pisa syndrome generally abate after withdrawal of the causative agent. Thus Pisa syndrome may be considered as an atypical subtype of TDt.

Tardive akathisia

The symptoms of TA are the same as in acute akathisia.[27] General principles of management of other TSs is recommended including prevention and changes in the neuroleptic treatment. If not successful, following pharmacological agents have been recommended: Anticholinergic drugs (benztropine), catecholamine-depleting drugs such as reserpine, tetrabenazine, and oxyperetine. Other drugs such as benzodiazepines and opiates with limited utility have been used.

Tardive tourettism

Drug-induced tardive tourettism is a neuro-behavioural disorder characterised by motor and vocal tics, and a wide range of behavioural problems following chronic neuroleptic treatment. The symptoms exhibited by the patients were indistinguishable from those of classic Tourette syndrome.[27] Management includes substitution of typical antipsychotics with an atypical antipsychotics or removal of the causative neuroleptic if possible. Pimozide, clonidine, and haloperidol can be helpful in some patients with tardive tourettism.

Tardive myoclonus

Myoclonus is a movement disorder characterised by abrupt, brisk, shock like, involuntary, repetitive, synchronous or asynchronous contractions of a muscle group of axial or appendicular muscles. Late-onset (tardive) myoclonus represents a distinct movement disorder secondary to neuroleptic exposure. The original report of tardive myoclonus has been reported as postural myoclonus in the upper extremities in 32 of 133 psychiatric patients who had been treated with neuroleptics for at least three months. The study revealed a slight male to female preponderance.[27]

Myoclonus may improve spontaneously over years or be unrelenting. When myoclonus is particularly disabling, treatment may include clonazepam, valproic acid, and levetiracetam.[36]

Tardive tremor

Stacy and Jankovic[37] described tardive tremor as a distinct clinical variant of TDk characterised by lack of other parkinsonian features, lack of response to conventional medications typically used for essential tremor, and marked improvement with tetrabenazine, differentiates this tremor from all other types of hereto-described rhythmic movement disorders.

Tardive dysmentia

Wilson *et al.*[38] had proposed the concept of tardive dysmentia (unstable mood, loud speech, and approach to the examiner) and attributed the aetogenesis of this syndrome to long-term exposure to neuroleptics. It has been described as the behavioural equivalent of TDk. Its association with TDk and psychopathology has been controversial.[39]

Antidepressant discontinuation syndrome

Antidepressant discontinuation syndrome is a set of symptoms that occur after an abrupt cessation or marked dose reduction of an antidepressant drug that was taken continuously for at least one month.[5] Symptoms generally begin within two to four days and typically include specifically emotional, neurosensory, neuromotor, vasomotor, neurological, or gastrointestinal.

Management

Symptoms are usually mild and self-limiting, but can occasionally be severe and prolonged. Agomelatine and vortioxetine are associated with a very low, if any risk of discontinuation symptoms.[40] For more severe symptoms, reintroduction of original antidepressant (or another with a longer half-life from the same class) and gradual tapering is recommended along with monitoring for symptoms.[12]

Serotonin syndrome

Serotonin syndrome results most often from the combination of two or more agents that enhance serotonergic activity or concentration in the central nervous system.[41] It is characterised by a triad of behavioural, autonomic, and neurological symptoms. Onset is typically abrupt but some patients report insidious or recurrent subtle cognitive decline, behavioural abnormalities, and tremor with postural changes days to weeks before the full-blown syndrome develops. The most commonly implicated agents reported have been combinations of monoamine oxidase inhibitors and tricyclic antidepressants with any other serotonergic agent.[42] Major differential diagnoses include NMS, infectious causes, hepatic encephalopathy, heatstroke, myocardial necrosis, delirium tremens, and intoxication by adrenergic or anticholinergic agents. It can be differentiated from NMS by relatively sudden onset of symptoms, presence of myoclonus and hyperreflexia.[43]

Management

Withdrawal of serotonergic agents is sufficient to improve symptoms in half of all cases. Persistent or severe symptoms require pharmacologic treatment. Intravenous electrolyte solution is administered 50-100 mL/h to avoid the risk of myoglobinuria. Benzodiazepines may be prescribed to reduce anxiety. Resuscitation (cooling off, mechanical ventilation, anticonvulsants, and antihypertensive agents) may be required for serious cases.[43] Cyproheptadine and chlorpromazine have been described as possible therapy for serotonin syndrome. Most patients improve completely within 24 hours after being treated with cyproheptadine or chlorpromazine but in some patients symptoms persist longer.

Conclusion

The various DIMD (i.e. akathisia, TDk, dystonia, and Parkinsonism) pose a significant burden to patients, often resulting in nonadherence, disease relapse, and decreased quality of life. For many of these disorders, treatment inconsistently provides benefit, and therefore, primary prevention is essential. Knowledge of DIMD should allow healthcare professionals to better identify patients with DIMD, or those at risk, and implement prevention and treatment plans.

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