Review

Secondary Movement Disorders: A Comprehensive Review of Drug-Related and Systemic Etiologies

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Posted Date: 28 July 2025

doi: 10.20944/preprints202507.2264.v1

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 doi:10.20944/preprints202507.2264.v1

Review

Secondary Movement Disorders: A Comprehensive Review of Drug-Related and Systemic Etiologies

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Abstract

Secondary movement disorders (SMDs) encompass a broad and clinically significant spectrum of motor abnormalities arising from diverse etiologies, including pharmacologic agents, metabolic imbalances, infections, vascular insults, and immune-mediated mechanisms. These disorders may present with hyperkinetic features such as chorea, dystonia, and myoclonus, or hypokinetic manifestations like parkinsonism, often mimicking primary neurodegenerative conditions and complicating diagnosis. This review synthesizes current knowledge on the pathophysiology, clinical presentation, and management of SMDs, with a particular focus on drug-induced syndromes, metabolic disturbances, infectious triggers, cerebrovascular events, and autoimmune phenomena. Advances in neuroimaging, pharmacogenomics, and biomarker development have enhanced diagnostic precision, while emerging therapies—including VMAT2 inhibitors, adaptive deep brain stimulation, and personalized pharmacologic strategies—offer new avenues for treatment. By integrating multidisciplinary insights, this review aims to improve early recognition, guide differential diagnosis, and support individualized care for patients with secondary movement disorders.

Keywords: drug-induced movement disorders; tardive dyskinesia; drug-induced parkinsonism; akathisia; dystonia; vmat2 inhibitors; pharmacovigilance; neuroleptics; adverse drug reactions; personalized medicine

1. Introduction

Drug-induced movement disorders (DIMDs) are a clinically significant and often underrecognized group of neurological syndromes resulting from exposure to various pharmacologic agents [1]. These disorders span a wide spectrum, including hyperkinetic manifestations such as tardive dyskinesia, dystonia, and tremor, as well as hypokinetic conditions like drug-induced parkinsonism [2]. The prevalence of DIMDs has increased in parallel with the widespread use of medications that affect central neurotransmitter systems, particularly antipsychotics, antiemetics, and antidepressants [3]. Despite their iatrogenic nature, DIMDs can lead to substantial morbidity, reduced quality of life, and diagnostic confusion with primary movement disorders [4].

Understanding the pathophysiology of DIMDs involves dissecting complex interactions within dopaminergic, serotonergic, and glutamatergic pathways [5], with emerging evidence pointing to genetic predispositions and neuroplastic changes as contributing factors [6]. Advances in neuroimaging, biomarker development, and pharmacogenomics *Brain Sci.*,, xhave begun to refine diagnostic accuracy and therapeutic strategies [7]. Notably, the introduction of vesicular monoamine transporter 2 (VMAT2) inhibitors has transformed the management of tardive syndromes [8], while novel approaches to continuous dopaminergic stimulation offer promise in mitigating drug-induced parkinsonism [9].

This review aims to provide a comprehensive and integrative overview of secondary movement disorders (SMDs), encompassing both drug-induced and non-drug-related etiologies. By synthesizing current evidence across pharmacologic, metabolic, infectious, vascular, and immune-mediated

 doi:10.20944/preprints202507.2264.v1

causes, the study seeks to enhance clinical recognition, diagnostic accuracy, and therapeutic decision-making. Special emphasis is placed on differentiating SMDs from primary neurodegenerative disorders, understanding underlying pathophysiological mechanisms, and exploring emerging diagnostic tools and personalized treatment strategies. Ultimately, this work aspires to support clinicians in delivering safer, more effective, and individualized care for patients presenting with secondary movement abnormalities.

2. Headache Medications

Several medications commonly used in the treatment of headaches, particularly migraines, have been implicated in the development of DIMDs [10]. Among the most frequently associated are dopamine receptor antagonists such as metoclopramide [11], prochlorperazine [12], and promethazine [13], which are often prescribed to manage migraine-associated nausea. These agents can lead to a range of movement disorders, including acute dystonia, parkinsonism, tardive dyskinesia, and akathisia [14]. These effects may appear within hours to days of administration or emerge after prolonged use, especially in older adults or those with underlying neurological vulnerability [15].

In addition to antiemetics, antiepileptic drugs like valproate [16] and phenytoin [17], which are used for migraine prophylaxis, have been associated with tremor, ataxia, and parkinsonism [18]. Though less common, these adverse effects can significantly impact quality of life and may be misattributed to primary neurological conditions [19]. Antihistamines such as hydroxyzine [20] and promethazine, sometimes used for tension-type headaches or as adjuncts in migraine therapy, have also been linked to extrapyramidal symptoms, particularly when used in combination with other central nervous system depressants [21]. Lasmiditan has rarely associated with abnormal movements [22].

Furthermore, the chronic overuse of acute headache medications—including triptans [23], NSAIDs [24], and combination analgesics—can lead to medication-overuse headache (MOH), a secondary headache disorder that, while not a movement disorder itself, reflects the broader spectrum of drug-induced neurological complications [25]. MOH can exacerbate headache frequency and severity, complicating diagnosis and treatment [26]. Recognizing the potential for DIMDs in patients receiving headache therapies is essential for early intervention, appropriate medication selection, and minimizing long-term neurological harm [27].

3. Antidepressants

Antidepressants, while essential in the management of mood disorders, have been increasingly recognized as potential contributors to DIMDs [28]. These adverse effects, though relatively rare, span a wide spectrum including tremor, akathisia, dystonia, parkinsonism, bruxism, and tardive dyskinesia [29]. A large postmarketing pharmacovigilance study using the WHO VigiBase database found significant associations between several antidepressants and movement disorders, with the highest reporting odds ratio (ROR) observed for bruxism and the lowest for tics [30]. Notably, selective serotonin reuptake inhibitors such as citalopram, paroxetine, fluoxetine, and sertraline [31] were frequently implicated, along with serotonin-norepinephrine reuptake inhibitors like duloxetine and venlafaxine [30,32].

Certain antidepressants appear to carry a higher risk for DIMDs. Agents such as mirtazapine [33,34], vortioxetine [35], amoxapine [36], trazodone [37], bupropion [38], phenelzine [39], and fluvoxamine [40] were associated with elevated RORs for multiple movement disorder subtypes [41]. These effects may be mediated by serotonergic and dopaminergic interactions within basal ganglia circuits [42], particularly in individuals with predisposing factors such as older age, polypharmacy, or underlying neurological vulnerability [43]. Interestingly, a 2025 study also highlighted that mild motor signs—including rigidity, dystonia, and hypokinesia—were present in a significant proportion of patients with major depressive disorder, and were associated with poor treatment response,

 doi:10.20944/preprints202507.2264.v1

independent of medication use [44]. This raises the possibility that some motor symptoms may reflect subclinical movement disorders rather than purely drug-induced phenomena [45]. Buspirone, an anxiolytic from the azapirone class [46], is primarily used to treat generalized anxiety disorder but has also been prescribed off-label for conditions such as depression, bruxism, and substance use disorders [47]. Although generally considered to have a favorable safety profile compared to benzodiazepines, buspirone has been associated with DIMDs in a small but notable number of cases [48]. A comprehensive literature review identified dyskinesia, akathisia, myoclonus, parkinsonism, and dystonia as the most frequently reported movement disorders linked to buspirone use. These symptoms typically emerged within the first month of treatment and were often reversible upon discontinuation of the drug. However, rare cases of persistent movement disorders, such as cervical-cranial dystonia and tremor, have also been documented following prolonged use [49]. The underlying mechanisms are not fully understood but may involve serotonergic modulation of dopaminergic pathways [50].

Amitriptyline, a tricyclic antidepressant, is widely used not only for depression but also for chronic pain and migraine prophylaxis [51]. Despite its therapeutic versatility, it has been associated with movement disorders such as tremor, akathisia, and parkinsonism [52], particularly in older adults or when used in combination with other serotonergic or anticholinergic agents [53]. These effects are thought to arise from its influence on central neurotransmitter systems, including serotonin, norepinephrine, and dopamine [54].

4. Antiseizure Medications

Antiseizure medications (ASMs) are essential in the management of epilepsy and various neurological conditions, but they are increasingly recognized as potential contributors to DIMDs [55]. These adverse effects include parkinsonism, tremor, myoclonus, dystonia, and dyskinesias [56], which may be misdiagnosed as primary movement disorders or event part of the aura phenomenon [57,58]. Noteworthy, the most common cause of ASM withdrawal is side effects to medications [59]. A comprehensive review identified that ASMs such as cenobamate [60], ethosuximide [61], felbamate [62], lamotrigine [63], phenytoin [64,65], tiagabine [66], and vigabatrin [67] are more frequently associated with the induction or worsening of movement disorders [68]. These effects are often dose-dependent and may be exacerbated by polypharmacy or underlying neurological vulnerability [69]. Some ASMs have a dual role, being both therapeutic for certain movement disorders and potential inducers of others [70]. For instance, clonazepam [71], gabapentin [72,73], levetiracetam [74], pregabalin [75,76], and topiramate [77] are sometimes used to treat tremor, myoclonus, or restless leg syndrome [78], yet they have also been reported to cause or worsen movement symptoms in susceptible individuals [79]. The mechanisms underlying these effects are complex and may involve alterations in GABAergic, glutamatergic, or dopaminergic neurotransmission [80]. Valproate [81] and carbamazepine [82,83] show variable effects, sometimes improving and other times exacerbating movement symptoms, depending on the clinical context and patient-specific factors [84].

Recent pharmacogenomic studies have highlighted the role of genetic variability in modulating susceptibility to ASM-induced adverse effects, including DIMDs [85]. Variants in genes affecting drug metabolism, neurotransmitter receptors, and ion channels may influence individual responses to ASMs [86]. This underscores the importance of personalized medicine approaches in epilepsy care, particularly in vulnerable populations such as the elderly or those with comorbid neurodegenerative conditions [87]. Clinicians should maintain vigilance for new-onset or worsening movement symptoms in patients on ASMs and consider dose adjustments, drug substitutions, or further neurological evaluation when appropriate [88].

5. Antipsychotics

Antipsychotic medications are a leading cause of DIMDs, which include a range of motor complications such as tardive dyskinesia (TD) [89], drug-induced parkinsonism, acute dystonia

 doi:10.20944/preprints202507.2264.v1

[90], akathisia [91], and in rare cases, neuroleptic malignant syndrome (NMS) [92] and belly dancer's dyskinesia [93]. These adverse effects are most commonly associated with first-generation (typical) antipsychotics like haloperidol and fluphenazine [94], but second-generation (atypical) antipsychotics such as risperidone, olanzapine, and aripiprazole [95] can also cause them, especially at higher doses or with long-term use [96]. Tardive dyskinesia, in particular, is a chronic and potentially irreversible condition characterized by involuntary, repetitive movements of the face, limbs, or trunk, and is estimated to affect at least 800,000 adults in the U.S. alone [97]. Rarely antipsychotics can be associated with oral vertical dyskinesia, also known as "rabbit" syndrome [98]. Also, some individuals with PD can have abnormal curvature of their spine related to antipsychotics use [99].

The risk of developing DIMDs increases with age, female sex, cumulative antipsychotic exposure, and co-administration of other dopamine-blocking agents [100]. Druginduced parkinsonism, which mimics idiopathic Parkinson's disease, often presents symmetrically and lacks the classic resting tremor seen in PD [101]. Acute dystonia and akathisia may occur within days to weeks of initiating treatment, while TD typically emerges after months or years of exposure [102]. Importantly, routine screening for abnormal movements is recommended—every 6 months for high-risk patients and annually for others—to ensure early detection and intervention [103]. Pimavanserin is a selective serotonin 5-HT2A receptor inverse agonist approved for the treatment of Parkinson's disease psychosis [104], offering antipsychotic benefits without exacerbating motor symptoms— a key advantage over traditional antipsychotics [105]. It has also shown promise in clinical trials for dementia-related psychosis and major depressive disorder, expanding its therapeutic potential [106]. Unlike dopamine-blocking agents, pimavanserin's mechanism allows for symptom control with a lower risk of drug-induced movement disorders, making it a valuable option in neuropsychiatric care [107].

Lithium, a mood stabilizer primarily used in the treatment of bipolar disorder, has been associated with a range of neurological side effects, including DIMDs [108]. While generally well-tolerated at therapeutic levels, lithium can cause tremor, which is the most common motor side effect and may present as a fine postural or action tremor [109]. At higher serum concentrations or in cases of chronic use, lithium may also lead to parkinsonism, ataxia, myoclonus, and rarely, choreoathetosis [110]. These effects are thought to result from lithium's interference with dopaminergic neurotransmission and cerebellar function [111]. Risk factors for lithium-induced movement disorders include advanced age, renal impairment, polypharmacy, and long-term exposure [112]. Monitoring serum lithium levels and renal function is essential to minimize toxicity, and dose adjustments or discontinuation may be necessary if movement symptoms emerge [113].

Management of antipsychotic-induced movement disorders involves dose reduction, switching to a lower-risk antipsychotic, or discontinuation when feasible [114]. For tardive dyskinesia, VMAT2 inhibitors such as valbenazine and deutetrabenazine have shown efficacy in reducing symptoms [115]. However, treatment must be individualized, as some DIMDs (e.g., drug-induced parkinsonism) may respond to anticholinergic agents, while others (e.g., TD or NMS) may worsen with such therapy [116]. Clinicians are advised to avoid lumping all DIMDs under the umbrella of "extrapyramidal symptoms" and instead tailor treatment based on the specific disorder and patient profile [117].

6. Antibiotics

Although antibiotics are not traditionally associated with movement disorders, several classes have been implicated in causing neurological side effects, particularly in vulnerable populations such as the elderly or those with renal impairment. Metronidazole, for example, is well-documented to cause cerebellar toxicity, leading to ataxia, dysarthria, and tremor, especially with prolonged use or high cumulative doses [118]. Neuroimaging in such cases often reveals bilateral cerebellar lesions, which are typically reversible upon discontinuation of the drug. Similarly, isoniazid, used in

 doi:10.20944/preprints202507.2264.v1

tuberculosis treatment, can cause parkinsonism, tremor, and peripheral neuropathy, particularly in patients with vitamin B6 deficiency, as it interferes with GABA synthesis.

Another notable group includes fluoroquinolones (e.g., ciprofloxacin [119], levofloxacin [120]), which have been associated with myoclonus, seizures, and chorea, likely due to their ability to antagonize GABA-A receptors and lower the seizure threshold [121]. These effects are more likely in patients with renal dysfunction or when fluoroquinolones are combined with other neurotoxic agents. Beta-lactam antibiotics, particularly cefepime, have also been linked to encephalopathy, myoclonus, and non-convulsive status epilepticus, especially in patients with impaired renal clearance [122]. These symptoms often resolve with dose adjustment or drug discontinuation, but early recognition is critical to prevent complications.

Clinicians should be aware of the potential for antibiotics to induce or exacerbate movement disorders, especially in patients with pre-existing neurological conditions or those on polypharmacy regimens. A thorough medication history, including recent antibiotic exposure, is essential when evaluating new-onset movement symptoms. In many cases, symptoms are reversible with prompt discontinuation of the offending agent, but failure to recognize the drug-related etiology can lead to unnecessary investigations or misdiagnosis as a primary movement disorder.

7. Immunotherapy

Immunotherapy, particularly immune checkpoint inhibitors (ICIs) and monoclonal antibodies, has revolutionized the treatment of cancer and autoimmune diseases. However, these therapies can also lead to immune-related adverse events (irAEs), including neurological complications such as movement disorders. Reported manifestations include parkinsonism, myoclonus, ataxia, chorea, and dystonia, often resulting from autoimmune inflammation of the basal ganglia, cerebellum, or peripheral nerves [123]. These effects may occur weeks to months after initiating therapy and can mimic primary neurodegenerative conditions, making diagnosis challenging. Early recognition is critical, as many cases respond to immunosuppressive treatment such as corticosteroids or IVIG [124].

Recent studies have highlighted the role of T-cell dysregulation and autoantibody production in the pathogenesis of immunotherapy-induced movement disorders. For example, immune checkpoint inhibitors like nivolumab and pembrolizumab have been associated with autoimmune encephalitis and paraneoplastic syndromes, which may present with subacute movement abnormalities. In some cases, antibodies against neuronal surface antigens (e.g., anti-NMDA receptor, anti-LGI1) have been detected, supporting an autoimmune mechanism [125]. These findings underscore the need for comprehensive neurological evaluation, including CSF analysis and antibody panels, in patients developing new-onset movement symptoms during or after immunotherapy [126].

Looking ahead, researchers are exploring biomarkers and genetic predictors to identify patients at higher risk for neurological irAEs. Advances in precision immunotherapy aim to balance efficacy with safety by modulating immune responses more selectively. As immunotherapy expands beyond oncology into neurology and psychiatry, clinicians must remain vigilant for atypical presentations of movement disorders and collaborate across specialties to ensure timely diagnosis and management.

8. Antiviral

While antiviral medications are primarily used to treat viral infections, emerging evidence suggests that some agents may contribute to neurological adverse effects, including movement disorders. This is particularly relevant in the context of COVID-19 therapies, where drugs like nirmatrelvir/ritonavir (NMVr) and remdesivir (RDV) have been widely used [127]. A study analyzing data from the European Medicines Agency's EudraVigilance database found that NMVr was associated with a significantly higher incidence of neuropsychiatric adverse drug reactions,

 doi:10.20944/preprints202507.2264.v1

including delirium, cognitive disturbances, and sleep disorders, as well as a higher reporting rate of movement disorders compared to other antivirals [128].

The mechanisms underlying antiviral-induced movement disorders are not fully understood but may involve central nervous system penetration, mitochondrial toxicity, or immune-mediated neuroinflammation [129]. Protease inhibitors like ritonavir are known to affect CNS neurotransmitter pathways, potentially leading to extrapyramidal symptoms or parkinsonism, especially in patients with pre-existing neurological vulnerabilities [130]. Although RDV had a lower incidence of neuropsychiatric effects than NMVr, it was still associated with movement-related symptoms in a subset of patients, highlighting the need for careful monitoring during antiviral therapy.

Clinicians should be vigilant for new-onset motor symptoms in patients receiving antiviral treatments, particularly in older adults or those with comorbid neurological conditions. Early recognition and discontinuation of the offending agent can lead to symptom resolution in many cases. As antiviral use expands beyond acute infections to chronic viral conditions and prophylaxis, understanding their neurological safety profile—including the risk of DIMDs—will be essential for optimizing patient care.

9. Psychostimulants

Psychostimulants—including amphetamines, cocaine [131], methylphenidate [132], and synthetic cathinones ("bath salts")—are increasingly recognized as triggers for a range of DIMDs. These substances can cause hyperkinetic symptoms such as chorea, tics, dystonia, myoclonus, and stereotypies, often emerging acutely during intoxication. In some cases, symptoms like chorea or tic-like movements may persist beyond the intoxication phase, especially with chronic use. Amphetamines, for example, have been linked to persistent motor abnormalities even after cessation, and phenomena like "crack dancing" reflect the stereotyped movements seen in stimulant users.

The pathophysiology of stimulant-induced movement disorders involves dopaminergic overstimulation, particularly in the striatum and basal ganglia, regions critical for motor control. Cocaine and amphetamines increase synaptic dopamine levels, which can disrupt normal motor signaling and lead to involuntary movements [133]. Additionally, synthetic stimulants may have unpredictable effects due to their variable potency and receptor profiles. These drugs can also lower the seizure threshold and contribute to neurotoxicity, further complicating the clinical picture. In emergency settings, stimulant-related movement disorders are a growing concern, often requiring supportive care and symptomatic management.

Management of psychostimulant-induced movement disorders involves cessation of the offending agent, symptomatic treatment (e.g., benzodiazepines for agitation or myoclonus), and in some cases, long-term neurological follow-up. For individuals with substance use disorders, addressing the underlying addiction is essential to prevent recurrence. Emerging therapies such as repetitive transcranial magnetic stimulation (rTMS) are being explored to reduce stimulant cravings and improve neuropsychiatric outcomes. Clinicians should maintain a high index of suspicion for DIMDs in patients presenting with new-onset motor symptoms and a history of stimulant use [134].

10. Antidementia Medications

Antidementia medications, primarily cholinesterase inhibitors (donepezil [135], rivastigmine, galantamine) and NMDA receptor antagonists (memantine [136]), are used to manage cognitive symptoms in Alzheimer's disease and other dementias [137]. These drugs aim to enhance cholinergic transmission or modulate glutamatergic activity, thereby improving memory, attention, and daily functioning. However, their effects on movement disorders are complex. While they may offer mild benefits in conditions like Parkinson's disease dementia by improving apathy and attention, they can also exacerbate motor symptoms such as tremors or rigidity in some patients due to increased cholinergic activity [138].

 doi:10.20944/preprints202507.2264.v1

Conversely, movement disorders can be both a side effect and a comorbidity in patients treated with antidementia medications. For example, cholinesterase inhibitors have been associated with extrapyramidal symptoms, including parkinsonism and dystonia, especially in elderly patients or those with preexisting motor dysfunction [139]. Additionally, the interplay between neurodegenerative diseases like Lewy body dementia and Parkinson's disease complicates treatment, as these conditions often present with both cognitive decline and motor symptoms [140]. Clinicians must carefully balance cognitive benefits with potential motor side effects, tailoring therapy to individual patient profiles and monitoring closely for adverse reactions.

11. Muscle Relaxants

Muscle relaxants are a diverse group of medications used to relieve muscle spasms, spasticity, and associated pain. They are broadly categorized into antispasmodics and antispastics. Antispasmodics, such as cyclobenzaprine, methocarbamol, and carisoprodol, are typically prescribed for acute musculoskeletal conditions like back pain or tension headaches [141]. These drugs act primarily on the central nervous system to reduce involuntary muscle contractions. Antispastics, including baclofen [142], tizanidine, and dantrolene, are used to manage chronic spasticity seen in conditions like multiple sclerosis, cerebral palsy, and spinal cord injuries [143]. Some agents, like diazepam and tizanidine, have both antispasmodic and antispastic properties.

The choice of muscle relaxant depends on the underlying condition, patient tolerance, and potential side effects. For example, baclofen is often preferred for spinal spasticity due to its central action on GABA-B receptors, while dantrolene, which acts directly on skeletal muscle, is favored when central side effects are a concern [144]. However, these medications can cause sedation, dizziness, and dependence, especially with long-term use. Therefore, they are usually prescribed for short durations or under close supervision in chronic cases [145]. Non-pharmacologic therapies like physical therapy and stretching exercises are often recommended alongside muscle relaxants for optimal outcomes.

12. Opioids

Opioids, while primarily used for pain management, have been increasingly recognized for their potential to induce or exacerbate movement disorders. These effects are often underreported but can include a range of involuntary movements such as myoclonus, tremor, dystonia, and even parkinsonism [146]. The pathophysiology is not fully understood but may involve opioid-induced alterations in dopaminergic pathways or direct effects on basal ganglia circuits [147]. For instance, chronic opioid use (tramadol [148]) has been associated with hyperkinetic movements like chorea and myoclonus, particularly in patients with substance use disorders [149].

A notable case involved a patient who developed transient choreoathetoid movements after a single dose of methadone or even oxycodone, despite no prior neurological history [150]. This suggests that even therapeutic doses of opioids can trigger movement disorders in susceptible individuals. The symptoms in this case resolved spontaneously, but such reactions highlight the need for clinicians to be vigilant when prescribing opioids, especially in populations with underlying neurological vulnerabilities. Other opioids, such as meperidine, have also been implicated in similar adverse motor effects, further supporting a class-wide concern [151]. Management of opioid-induced movement disorders typically involves discontinuation or dose reduction of the offending agent, along with symptomatic treatment using agents like benzodiazepines, antiepileptics, or antipsychotics depending on the movement type. In some cases, corticosteroids have been used to shorten the course of choreiform symptoms. As opioid prescriptions remain common, especially in chronic pain and palliative care settings, awareness of these potential neurological side effects is essential for early recognition and appropriate intervention.

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13. Metabolic

Metabolic movement disorders arise from both inherited and acquired disruptions in biochemical pathways that affect the central nervous system, particularly the basal ganglia and cerebellum. Inherited metabolic disorders (IMDs), also known as inborn errors of metabolism, are increasingly recognized as important causes of movement disorders across all age groups [152]. A 2025 review identified over 550 IMDs associated with movement disorders, including conditions such as glutaric aciduria type I [153], Wilson disease [154], adrenoleukodystrophy [155], mitochondrial disorders [156], Fahr's disease [157,158], and organic acidemias [159]. These disorders can present with a wide range of motor symptoms, including dystonia [160], chorea, myoclonus, ataxia, and parkinsonism [161], often in combination with developmental delay, seizures, or systemic features. Noteworthy, these clinical manifestations can be exacerbated by myelopathic findings [162].

Acquired metabolic disturbances—such as hypoglycemia, hyperglycemia, hepatic encephalopathy [163], uremia [164], copper [165], thyroid [166], and electrolyte imbalances [167,168]—can also lead to acute or subacute movement disorders [169]. For example, non-ketotic hyperglycemia is a well-known cause of hemichorea-hemiballismus [170], particularly in elderly patients with poorly controlled diabetes [171]. Similarly, hepatic encephalopathy may present with asterixis [172], tremor, or parkinsonism [173], while uremic encephalopathy can cause myoclonus and restless leg syndrome [174]. These conditions are often reversible with correction of the underlying metabolic derangement, making early recognition and treatment essential.

14. Infectious

Infectious diseases can play a significant role in the development or unmasking of DIMDs, particularly in vulnerable populations. Systemic infections such as HIV [175], herpes zoster [176], syphilis [177], dengue [178–180], zygomycosis [181], toxoplasmosis [182], tuberculosis [183], measles [184,185], leprosy [186], and neurocysticercosis [187,188] have been associated with basal ganglia dysfunction, which may increase susceptibility to movement disorders when patients are exposed to dopamine-blocking or serotonergic medications [189]. For example, individuals with HIV-associated neurocognitive disorders may develop parkinsonism or dystonia [190] when treated with antipsychotics or antiemetics, due to pre-existing subclinical damage to dopaminergic pathways [191]. Similarly, neurosyphilis [192] can mimic idiopathic Parkinson's disease and may worsen with medications that affect central neurotransmission [193]. Noteworthy, viral diseases can mimic several movement disorders [194].

Infections can also alter drug metabolism and pharmacodynamics, thereby increasing the risk of adverse neurological effects [195]. Systemic inflammation, fever, and hepatic or renal dysfunction during infections can impair the clearance of medications such as lithium [196], valproate [197], or antipsychotics, leading to toxic levels and subsequent movement disorders like tremor, myoclonus, or parkinsonism [198]. In some cases, infections may trigger autoimmune responses that affect the central nervous system [199], such as in post-streptococcal basal ganglia encephalitis [200], which can present with chorea or dystonia and be exacerbated by serotonergic or dopaminergic agents [201]. These interactions underscore the importance of careful medication management during acute or chronic infections [202].

15. Cerebrovascular Disorder

Cerebrovascular disorders are a significant cause of secondary movement disorders, particularly in older adults, especially in those with poor literacy [203]. But, they also can occur in other groups such as pregnant population [204]. These movement abnormalities can arise acutely following ischemic or hemorrhagic strokes [205], or more insidiously in the context of chronic small vessel disease [206]. The most common presentations include vascular parkinsonism [207], hemichorea-hemiballismus [208], dystonia [209], astasia [210], and tremor. But also, acute pseudobulbar palsy was observed [211]. Vascular parkinsonism typically presents with lower body-predominant

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bradykinesia and gait disturbance, often without resting tremor, and is frequently resistant to levodopa therapy [212]. Lesions in the basal ganglia, thalamus [213], or subthalamic nucleus are commonly implicated [214], and neuroimaging often reveals strategic infarcts or diffuse white matter changes consistent with cerebral small vessel disease [215]. Moreover, there are two interesting presentations called the cortical hand [216] and cortical foot [217]. Also, there is the occurrence of cerebral venous thrombosis rarely been associated with abnormal movements [218].

Recent studies have expanded the understanding of movement disorders associated with cerebral artery stenosis (CAS), even in the absence of overt stroke [219]. Chronic hypoperfusion due to CAS can lead to functional disruption of basal ganglia circuits, resulting in movement disorders such as chorea, myoclonus, or parkinsonism [220]. These symptoms may fluctuate with cerebral perfusion status and can improve following revascularization procedures or medical optimization of cerebral blood flow [221]. This highlights the importance of considering vascular etiologies in patients with atypical or rapidly progressive movement disorders, especially when traditional neurodegenerative markers are absent, especially in atrial fibrillation [222].

Techniques such as arterial spin labeling MRI and CT perfusion are increasingly used to detect subtle perfusion deficits in patients with unexplained motor symptoms [223]. These tools, combined with clinical assessment and vascular risk profiling, are helping to refine diagnostic accuracy and guide targeted interventions. As our understanding of the vascular-motor interface deepens, early recognition and treatment of cerebrovascular contributors to movement disorders may offer opportunities to improve outcomes and prevent progression [224].

16. How to Differentiate PD from DIP

Differentiating Parkinson's disease (PD) from drug-induced parkinsonism (DIP) can be clinically challenging, as both conditions share core motor features such as bradykinesia, rigidity, and tremor [225]. However, several distinguishing characteristics can aid in diagnosis. PD typically presents asymmetrically, with symptoms starting on one side of the body and progressing gradually [226]. In contrast, DIP often manifests symmetrically, affecting both sides of the body equally from the onset [227]. Additionally, resting tremor, a hallmark of PD, is less prominent or even absent in DIP. Non-motor symptoms such as anosmia, REM sleep behavior disorder, and autonomic dysfunction are more common in PD and usually precede motor symptoms, whereas they are rare in DIP [228].

The temporal relationship between symptom onset and medication exposure is another key differentiator [229]. DIP usually develops within weeks to months of initiating or increasing the dose of dopamine-blocking agents, such as antipsychotics or certain antiemetics [230]. If the offending drug is discontinued, symptoms of DIP often improve or resolve within weeks to 18 months, although in some cases, especially in older adults, symptoms may persist [231]. In contrast, PD is a progressive neurodegenerative disorder with no known cure, and symptoms typically worsen over time despite treatment. A detailed medication history is therefore essential in evaluating parkinsonian symptoms [232].

Cardiac ^{123}I-MIBG scintigraphy is a valuable imaging tool used to differentiate Parkinson's disease (PD) from drug-induced parkinsonism (DIP) by assessing cardiac sympathetic innervation [233]. MIBG, a norepinephrine analog, is taken up by sympathetic nerve terminals in the myocardium [234]. In PD, there is cardiac sympathetic denervation, resulting in reduced MIBG uptake, which can be quantified using the heart-to-mediastinum (H/M) ratio [235]. Studies have shown that PD patients typically exhibit significantly lower H/M ratios in both early and delayed imaging phases, with sensitivities around– 83% and specificities between 80–86% [236].

Advanced imaging techniques can further aid in differentiation. Dopamine transporter (DAT) imaging using SPECT or PET scans typically shows reduced striatal uptake in PD due to loss of dopaminergic neurons [237], whereas DAT imaging is usually normal in DIP, reflecting intact presynaptic dopamine terminals [238]. While not routinely required, DAT imaging can be particularly useful in ambiguous cases or when clinical features overlap [239]. Ultimately, a

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combination of clinical history, symptom pattern, medication exposure, and, when necessary, imaging studies provides the most accurate approach to distinguishing PD from DIP [240].

17. Deep Brain Stimulation

Deep brain stimulation (DBS) is a well-established surgical treatment for Parkinson's disease (PD), particularly effective in managing motor symptoms such as tremor, rigidity, and bradykinesia in patients with motor fluctuations or medication-refractory symptoms [241]. The procedure involves implanting electrodes into specific brain regions—most commonly the subthalamic nucleus (STN) or globus pallidus internus (GPi)—which deliver continuous electrical stimulation to modulate abnormal neural activity [242]. Traditional DBS systems operate in an open-loop fashion, delivering constant stimulation regardless of the patient's real-time brain state [243]. While effective, this approach can lead to side effects such as speech disturbances or gait impairment, and may not address non-motor symptoms.

Recent advances have led to the development and FDA approval of adaptive DBS systems, which represent a major leap forward in personalized neuromodulation [244]. These systems continuously monitor brain activity and adjust stimulation parameters in real time based on specific neural biomarkers [245]. For example, adaptative DBS can detect pathological beta oscillations in the STN and deliver targeted pulses only when needed, reducing side effects and improving symptom control [246]. UCSF researchers have developed both "fast" and "slow" adaptive algorithms, allowing clinicians to tailor therapy to individual patient profiles using Bluetooth-enabled software. This dynamic approach not only enhances motor symptom management but also shows promise in addressing non-motor symptoms such as mood disturbances and sleep dysfunction. Another 2025 breakthrough involves tailored DBS programming for gait dysfunction, a symptom often resistant to standard DBS. Researchers at UCSF used machine learning to optimize stimulation settings based on real-time gait analysis, significantly improving walking speed, step symmetry, and stability without worsening other symptoms [247]. By integrating neural recordings with kinematic data, they developed a Walking Performance Index (WPI) to guide individualized DBS adjustments. These innovations mark a shift toward precision neuromodulation, where DBS is no longer a one-size-fits-all therapy but a dynamic, data-driven intervention capable of adapting to the evolving needs of patients with Parkinson's disease.

18. Discussion and Future Directions

The Unified Parkinson's Disease Rating Scale (UPDRS) is the most widely used clinical tool for assessing the severity and progression of PD [248]. Developed originally in 1987 and later revised by the Movement Disorder Society (MDS-UPDRS), the scale is divided into four parts: Part I evaluates mentation, behavior, and mood; Part II assesses activities of daily living (ADL); Part III focuses on motor examination, including tremor, rigidity, bradykinesia, and postural instability; and Part IV addresses motor complications such as dyskinesias and fluctuations related to therapy. Each item is scored on a 0–4 Likert scale, with higher scores indicating greater impairment. The UPDRS provides a comprehensive, standardized framework for tracking disease progression, guiding treatment decisions, and evaluating therapeutic outcomes in both clinical and research settings [249]. The "liquid gold" formulation of levodopa–carbidopa stabilized with ascorbic acid offers a promising alternative for managing Parkinson's disease, particularly in patients with erratic gastrointestinal absorption or motor fluctuations [250]. This liquid solution allows for more consistent and rapid absorption compared to tablets, leading to smoother and more predictable symptom control. Ascorbic acid plays a crucial role in stabilizing levodopa and carbidopa, especially in patients using magnesium oxide-based laxatives, which can otherwise degrade these medications [251]. Additionally, the liquid form facilitates individualized dosing and may reduce OFF periods, improving overall quality of life in advanced Parkinson's disease [252].

 doi:10.20944/preprints202507.2264.v1

As the landscape of DIMDs continues to evolve, future research must prioritize the development of predictive tools that integrate pharmacogenomics, digital biomarkers, and real-world data. Personalized medicine approaches—leveraging genetic profiles, wearable technologies, and artificial intelligence—hold promise for identifying individuals at heightened risk for DIMDs before symptoms emerge. This proactive strategy could revolutionize pharmacovigilance by enabling clinicians to tailor drug regimens based on individual susceptibility, thereby minimizing neurological harm. Additionally, expanding the use of advanced neuroimaging and fluid biomarkers, such as α-synuclein seed amplification assays, may improve diagnostic accuracy and help differentiate DIMDs from idiopathic movement disorders in complex clinical scenarios [253].

Another critical direction involves refining therapeutic strategies to address both prevention and management of DIMDs. Innovations such as adaptive DBS, VMAT2 inhibitors, and liquid levodopa–carbidopa formulations exemplify the shift toward precision neuromodulation and individualized pharmacotherapy [254]. Future clinical trials should explore the efficacy of these interventions across diverse populations and drug classes, including underrepresented groups such as the elderly, pregnant individuals, and those with comorbid neurodegenerative or psychiatric conditions. Moreover, interdisciplinary collaboration among neurologists, psychiatrists, pharmacologists, and data scientists will be essential to translate emerging discoveries into clinical practice. By embracing a holistic, patient-centered approach, the field can move toward safer prescribing practices and improved neurological outcomes.

19. Conclusions

DIMDs represent a complex and evolving field within neurology, intersecting pharmacology, genetics, and clinical medicine. This review underscores the wide spectrum of DIMDs, ranging from hyperkinetic manifestations such as chorea, dystonia, and myoclonus to hypokinetic presentations like drug-induced parkinsonism. The diversity of implicated agents—including antipsychotics, antidepressants, antiseizure medications, opioids, immunotherapies, and even antibiotics—highlights the need for heightened clinical vigilance across specialties. While some movement disorders are transient and reversible, others may persist or become disabling, particularly with delayed recognition or continued exposure to the offending agent.

Recent advances in neuroimaging, pharmacogenomics, and biomarker development have enhanced diagnostic precision and opened new avenues for personalized treatment. The emergence of VMAT2 inhibitors, adaptive deep brain stimulation, and liquid levodopa–carbidopa ascorbic acid formulations exemplify the therapeutic innovations reshaping management strategies. However, challenges remain in differentiating DIMDs from primary neurodegenerative conditions, especially in older adults or those with polypharmacy. Moreover, the interplay between systemic diseases, infections, and pharmacologic exposures adds further complexity to diagnosis and treatment.

Looking forward, the integration of digital monitoring tools, real-world pharmacovigilance data, and individualized risk profiling will be essential to advance early detection and prevention. Multidisciplinary collaboration among neurologists, psychiatrists, pharmacists, and primary care providers is crucial to optimize outcomes and minimize iatrogenic harm. As our understanding of DIMDs deepens, a paradigm shift toward proactive, precision-based care is both necessary and achievable. Ultimately, recognizing and addressing DIMDs not only improves neurological health but also enhances the overall safety and efficacy of modern therapeutics.

Supplementary Materials: Not applicable.

Author Contributions: "Conceptualization, methodology, software, validation, formal analysis, investigation, resources, data curation, writing—original draft preparation, writing—review and editing, visualization, supervision, project administration, funding acquisition performed E.R.S. All authors have read and agreed to the published version of the manuscript.

Funding: This research received no external funding.

 doi:10.20944/preprints202507.2264.v1

Institutional Review Board Statement: Not applicable.

Informed Consent Statement: Not applicable.

Data Availability Statement: Not applicable.

Acknowledgments: None.

Conflicts of Interest: The authors declare no conflicts of interest.

Abbreviations

The following abbreviations are used in this manuscript:

DBS Deep brain stimulation
DIMD Drug-induced movement disorder
PD Parkinson's disease
MOH Medication-overuse headache
NMS Neuroleptic malignant syndrome ROR Reporting odds ratio
TD Tardive dyskinesia
VMAT2 Vesicular monoamine transporter 2

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