Review

Global Research Trends in Drug-Induced Movement Disorders: A Bibliometric Analysis

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Review

Global Research Trends in Drug-Induced Movement Disorders: A Bibliometric Analysis

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Abstract: Background: Drug-induced movement disorders (DIMDs) represent a significant clinical challenge, encompassing conditions such as tardive dyskinesia, drug-induced parkinsonism, dystonia, and akathisia. While research on DIMDs has expanded, a comprehensive bibliometric analysis of global trends, influential publications, and emerging research themes remains lacking. Objective: This study aims to provide a bibliometric analysis of DIMD research, evaluating publication trends, citation networks, key contributors, and thematic evolution in the field. Methods: A systematic search of bibliographic databases was conducted to identify relevant literature on DIMDs. Bibliometric indicators, including publication output, citation analysis, author collaborations, and keyword co-occurrence, were analyzed. VOSviewer and CiteSpace were used for network visualization and trend mapping. A narrative synthesis of key findings contextualized bibliometric patterns with historical and contemporary research developments. Results: The analysis revealed a steady increase in DIMD publications, with antipsychotic-induced disorders dominating the literature. The United States and Europe emerged as leading contributors, with significant collaborations across institutions. Highly cited papers predominantly focused on pathophysiology, risk factors, and management strategies. Emerging research trends highlighted the role of genetic predisposition, novel therapeutic interventions, and artificial intelligence in early detection. Conclusion: Bibliometric analysis provides valuable insights into the evolution and current state of DIMD research. Future studies should focus on precision medicine approaches, neurobiological mechanisms, and global disparities in management. This review underscores the need for interdisciplinary collaboration to enhance understanding and therapeutic strategies for DIMDs.

Keywords: drug-induced movement disorders; tardive dyskinesia; drug-induced parkinsonism; dystonia; akathisia; bibliometric analysis; citation network; antipsychotics; neuropharmacology; precision medicine

1. Introduction

Drug-induced movement disorders (DIMDs) encompass a spectrum of iatrogenic neurological conditions, including tardive dyskinesia, drug-induced parkinsonism, acute and tardive dystonia, and akathisia [1]. These disorders primarily arise as adverse effects of dopamine receptor-blocking agents, particularly first- and second-generation antipsychotics, as well as certain antiemetics, antidepressants, and calcium channel blockers [2]. While the exact prevalence varies, studies estimate that up to 30% of patients receiving chronic antipsychotic therapy develop some form of DIMD, significantly impacting quality of life and long-term treatment adherence [3]. Despite increasing clinical awareness, the underlying pathophysiology and optimal management strategies remain areas of active investigation [4].

Over the past decades, research on DIMDs has expanded substantially, driven by advances in neuropharmacology, genetics, and biomarker discovery. Studies have elucidated key mechanisms, including dopamine receptor supersensitivity, oxidative stress, and neuroinflammation, which contribute to the pathogenesis of these disorders [5]. Moreover, emerging evidence suggests a role for genetic susceptibility, with polymorphisms in DRD2 [6], COMT, and CYP2D6 [7] genes

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influencing individual risk profiles [8]. Therapeutic developments, such as vesicular monoamine transporter 2 (VMAT2) inhibitors for tardive dyskinesia, have further stimulated scientific interest, leading to a surge in related publications [9].

Despite this growing body of literature, a comprehensive understanding of research trends, influential publications, and evolving themes in DIMD remains lacking. Bibliometric analysis, a quantitative approach to evaluating scientific literature, enables the identification of key contributors, citation networks, and emerging research hotspots [10]. Previous bibliometric studies have provided insights into various neurological disorders [11], yet no systematic evaluation of DIMD research has been conducted to date. The epidemiology of DIMDs has also evolved over time, with evidence suggesting a decline in prevalence due to the shift from first-generation to second-generation antipsychotics [12]. However, extrapyramidal side effects remain a significant concern, even with newer agents [8,13]. A bibliometric approach can provide a macroscopic perspective on the field, guiding future investigations and interdisciplinary collaborations [14].

This study aims to map the global research landscape on DIMDs through a bibliometric analysis of published literature. By analyzing publication trends, citation networks, and thematic shifts, we seek to identify research gaps and future directions. Understanding the evolution of DIMD research will not only enhance scientific knowledge but also facilitate the development of more effective prevention and treatment strategies [15]. Furthermore, the safety and tolerability of antipsychotics, as well as their association with movement disorders, continue to be central topics in neuropharmacological research [16]. Current evidence highlights the need for precision medicine approaches to minimize the risks of DIMDs [17], while novel therapeutic strategies, such as VMAT-2 inhibitors, offer promising avenues for treatment [18].

2. Methods

A comprehensive bibliometric analysis was conducted to evaluate global research trends in drug-induced movement disorders (DIMDs). Data were retrieved from the Web of Science (WoS) Core Collection and Scopus databases, covering publications from their inception to the present. The search strategy utilized a combination of MeSH terms and free-text keywords related to DIMDs, including "tardive dyskinesia," "drug-induced parkinsonism," "neuroleptic-induced movement disorders," and "antipsychotic extrapyramidal symptoms." Inclusion criteria encompassed original research articles, reviews, and meta-analyses published in peer-reviewed journals, while editorials, conference abstracts, and non-English publications were excluded. Bibliometric indicators analyzed included publication output, citation trends, authorship patterns, institutional and country collaborations, co-citation networks, and keyword co-occurrence analysis.

VOSviewer (https://www.vosviewer.com/) is an open-access software designed for constructing and visualizing bibliometric networks [19]. It facilitates the creation of bibliometric maps based on co-citation, co-authorship, and keyword co-occurrence data, offering multiple visualization options to enhance data interpretation. The software includes interactive features such as zooming, panning, and searching, allowing for detailed exploration of bibliometric networks. Unlike other bibliometric analysis tools like SPSS and Pajek, VOSviewer emphasizes graphical representation, making it particularly effective for mapping scientific landscapes [20]. Additionally, it integrates text mining capabilities to identify relationships among citing articles and emerging research trends [21].

CiteSpace (https://sourceforge.net/projects/citespace/) is a Java-based bibliometric tool developed for analyzing and visualizing knowledge structures in scientific research [22]. It employs network analysis algorithms to identify critical developments in a research domain by examining citation patterns, author collaborations, and keyword co-occurrences. CiteSpace enables users to explore the evolution of scientific fields by detecting citation bursts, thematic clusters, and research frontiers through interactive visualizations. This functionality makes it a valuable tool for identifying influential studies and emerging trends within a given discipline [23]. The study adhered to PRISMA guidelines for literature selection and ensured reproducibility through standardized search queries and data extraction methods [24].

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3. Results

Over the past few decades, research on drug-induced movement disorders (DIMDs) has significantly expanded, reflecting increased clinical awareness and scientific interest. Studies have explored various aspects of DIMDs, including pathophysiology, clinical presentation, and therapeutic strategies. Singer et al. provided a comprehensive review detailing the clinical features and management of DIMDs, emphasizing the importance of early identification and intervention [25]. Similarly, Pandey et al. highlighted the diagnostic challenges associated with DIMDs and underscored the need for individualized treatment approaches [26]. The growing number of publications on DIMDs demonstrates a sustained effort to improve patient outcomes and develop novel therapeutic strategies.

Geographically, the distribution of DIMD research has been concentrated in developed countries, with significant contributions from institutions in the United States, Europe, and Asia. Collaborative efforts among these regions have facilitated advancements in understanding and managing DIMDs. For instance, a 30-year study conducted in Olmsted County, Minnesota, examined the incidence and time trends of drug-induced parkinsonism, providing valuable epidemiological data [27]. Such studies highlight the importance of international collaboration in addressing the global impact of DIMDs.

Recent advancements in artificial intelligence (AI) and precision medicine have introduced promising approaches to studying and managing DIMDs. AI-driven models are increasingly being utilized to analyze complex datasets, identify disease biomarkers, and develop personalized treatment plans. For example, Salvioli et al. explored the role of AI in biomarker analysis and its potential implications for neurodegenerative disorders, including DIMDs [28]. These innovations suggest a shift towards more data-driven, individualized approaches to understanding and treating DIMDs, paving the way for future breakthroughs in the field.

The thematic evolution of DIMD research has encompassed various key areas, including the identification of genetic factors contributing to susceptibility, the exploration of antipsychotic-induced disorders, and the development of emerging therapies. Studies have investigated the genetic underpinnings of DIMDs, aiming to identify individuals at higher risk and tailor interventions accordingly [29]. Additionally, research has focused on understanding the mechanisms underlying antipsychotic-induced movement disorders, leading to the development of novel therapeutic strategies to mitigate these effects [30]. Emerging therapies, such as the use of novel pharmacological agents and neuromodulation techniques, are being explored to improve outcomes for patients with DIMDs [31].

Emerging trends in DIMD research include the integration of AI and machine learning techniques to enhance diagnostic accuracy and treatment efficacy. AI algorithms are being developed to analyze large datasets, including clinical, genetic, and imaging data, to identify patterns and predict disease progression [32]. Precision medicine approaches are being employed to tailor treatments based on individual patient characteristics, such as genetic profiles and biomarkers [33]. These advancements hold promise for improving the management of DIMDs and reducing the burden of these disorders on patients and healthcare systems.

4. Discussion

4.1. Headache Management

Several headache medications have been associated with movement disorders, particularly those affecting dopaminergic [34] and serotonergic pathways [35]. Dopamine antagonists, such as metoclopramide and prochlorperazine, commonly used for migraine-related nausea, can induce tardive dyskinesia, dystonia, and parkinsonism [36]. Triptans, selective serotonin receptor agonists, have been linked to serotonin syndrome, which may present with tremors and myoclonus [37]. Valproate [38], frequently prescribed for migraine prophylaxis, has been associated with tremor,

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asterixis [39], and reversible parkinsonism [40], but also it was already associated with hematologic aside-effects like dose-dependent pancytopenia [41]. Additionally, topiramate may cause dystonic reactions and akathisia [42]. Interestingly, gepants and ditans [43] were never associated with movement disorders [44]. Flunarizine, a calcium channel blocker used for migraine prophylaxis [45], has been associated with drug-induced movement disorders, including parkinsonism, dystonia, and tardive dyskinesia, due to its dopamine receptor-blocking properties [46].

4.2. Antiseizure Medications

Antiseizure medications (ASMs) are commonly associated with various side effects [47] including movement disorders due to their effects on neurotransmission, particularly on GABAergic, glutamatergic [48], and dopaminergic pathways [49]. Epilepsy can confound movement disorders by presenting with paroxysmal [50], involuntary movements that mimic dyskinesias [51], dystonia, somatosensory symptoms [52], or myoclonus [53], making accurate diagnosis challenging, especially in cases of motor seizures [54], epileptic tremors [55], or after post-ictal symptoms [56]. Valproate is frequently linked to tremor, parkinsonism, and tardive dyskinesia, especially with long-term use [57]. Carbamazepine and oxcarbazepine have been associated with dystonia, chorea, and myoclonus [58]. Phenytoin toxicity [59] can lead to nystagmus [60], ataxia [61], and dyskinesias [62]. Lamotrigine [63] has been reported to induce tics and myoclonus in rare cases. Topiramate may cause dystonic reactions [64] and parkinsonism [65]. Cenobamate, an antiseizure medication, has been associated with drug-induced movement disorders [66], including parkinsonism, dyskinesia, and ataxia, likely due to its modulation of sodium channels and GABAergic activity [67].

4.3. Antidepressants

Antidepressants, particularly those affecting serotonergic and dopaminergic pathways, have been implicated in various movement disorders [68], and also depression by itself can worsen movement disorder symptoms [69]. Selective serotonin reuptake inhibitors (SSRIs) such as fluoxetine and sertraline can induce akathisia, tremors, and parkinsonism. Serotonin-norepinephrine reuptake inhibitors (SNRIs) like venlafaxine have been linked to dystonia and myoclonus [70]. Tricyclic antidepressants (TCAs), including amitriptyline, may cause tremors, choreiform movements, and tardive dyskinesia. Monoamine oxidase inhibitors (MAOIs) have been associated with myoclonus and dystonia. Bupropion [71], which affects dopaminergic and serotoninergic pathways [72], can lead to tics and dyskinesias [73]. Mirtazapine, a noradrenergic and serotonergic antidepressant, has also been associated with movement disorders [74]. While generally considered to have a lower risk of extrapyramidal symptoms compared to other antidepressants, case reports have linked mirtazapine to akathisia, myoclonus [75], dystonia [76], tremors, and parkinsonism [77]. Buspirone [78] has been reported to induce or alleviate movement disorders, including tardive dyskinesia, dystonia [79], and parkinsonism, due to its partial agonist activity at serotonin 5-HT1A receptors and dopaminergic modulation [80]. Trazodone, a serotonin antagonist and reuptake inhibitor, has been associated with drug-induced movement disorders, including parkinsonism [81], tardive dyskinesia, and akathisia, likely due to its serotonergic and dopaminergic modulation [82].

4.4. Neuropathic Medications

Neuropathic pain medications, particularly those affecting calcium channels, GABAergic transmission, and serotonin-norepinephrine pathways, have been associated with various movement disorders [83]. Gabapentinoids [84], such as gabapentin [85] and pregabalin [86], have been linked to tremors, myoclonus, parkinsonism [87], and ataxia; but, other side effects even including the urinary symptoms were already reported [88]. Carbamazepine and oxcarbazepine, commonly used for trigeminal neuralgia, may cause dystonia, chorea, and parkinsonism [89]. Duloxetine and venlafaxine, serotonin-norepinephrine reuptake inhibitors (SNRIs), have been associated with

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akathisia, tremors, and dyskinesias. Amitriptyline, a tricyclic antidepressant (TCA), can induce tremors [90], dystonia, and tardive dyskinesia [91].

4.5. Antiparkinsonian Medications

Pimavanserin, a selective serotonin 5-HT2A inverse agonist, is primarily used for treating Parkinson's disease psychosis (PDP) and has been studied for its effects on movement disorders [92]. Unlike traditional antipsychotics, it does not block dopamine D2 receptors, reducing the risk of extrapyramidal symptoms (EPS) such as drug-induced parkinsonism, tardive dyskinesia, and dystonia [93]. Some studies suggest it may even have a protective role against worsening motor symptoms in Parkinson's disease (PD). However, rare cases of worsening gait instability and tremor have been reported, necessitating careful monitoring in patients with preexisting movement disorders [94]. Amantadine, a glutamatergic NMDA receptor antagonist and dopaminergic agent [95], has been reported to cause myoclonus [96]. Dopamine agonists, used primarily for Parkinson's disease and restless legs syndrome, can induce movement disorders such as dyskinesias [97], impulse control disorders, and dopamine agonist withdrawal syndrome due to excessive or dysregulated dopaminergic stimulation [98]. Fatigue is a significant confounding factor in both Parkinson's disease and drug-induced movement disorders, often overlapping with motor symptoms and exacerbating bradykinesia, leading to diagnostic challenges [99].

4.6. Antipsychotics

Lithium [100], a mood stabilizer commonly used in bipolar disorder, is associated with various movement disorders, including tremor, parkinsonism, chorea, and dystonia, due to its effects on dopaminergic and cerebellar pathways [101]. Lithium-induced tremor is the most frequent manifestation, typically presenting as a fine postural or action tremor, which may worsen with toxicity and require dose adjustments or adjunctive treatment [102].

4.7. Antibiotics

Certain antibiotics have been implicated in drug-induced movement disorders through mechanisms involving dopaminergic, GABAergic, and mitochondrial dysfunction [103]. Fluoroquinolones [104], such as ciprofloxacin [105] and levofloxacin, have been associated with tremor, myoclonus, and dyskinesias, likely due to GABA receptor inhibition [106]. Beta-lactams, particularly cefepime, can induce encephalopathy with myoclonus and ataxia, especially in renal impairment [107]. Macrolides like clarithromycin have been linked to chorea and dystonia, possibly through neuroinflammatory pathways. Additionally, metronidazole toxicity may cause cerebellar dysfunction, tremor, and dysmetria [108].

4.8. Spasticity Treatment

Baclofen, a GABA-B receptor agonist, is primarily used for spasticity but can also induce or alleviate movement disorders [109], with reports of dystonia, chorea, and myoclonus as adverse effects, while its withdrawal may trigger severe hyperkinetic movements, including rebound spasticity and dyskinesias [110].

4.9. Psychostimulants and Other Durgs of Abuse

Methylphenidate, a central nervous system stimulant used for attention deficit hyperactivity disorder (ADHD) [111], has been associated with movement disorders, including tics, dystonia [112], and dyskinesias, likely due to its effects on dopaminergic neurotransmission [113]. In predisposed individuals, it may exacerbate Tourette syndrome or induce parkinsonism with chronic use. Cocaine use can induce movement disorders [114], including chorea, dystonia, and parkinsonism, due to its effects on dopaminergic and basal ganglia circuits, with both acute intoxication and chronic use leading to extrapyramidal dysfunction [115].

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4.10. Antidementia Medications

Antidementia medications, particularly cholinesterase inhibitors like donepezil, rivastigmine, and galantamine, [116] are primarily used to manage cognitive symptoms in Alzheimer's disease and other dementias [117]. While these agents aim to enhance cholinergic transmission, they can occasionally induce movement disorders [118]. For instance, rivastigmine has been associated with acute dystonic reactions, which are involuntary muscle contractions leading to abnormal postures or movements. Additionally, memantine, an NMDA receptor antagonist used in moderate to severe Alzheimer's disease, has been linked to dyskinesias, particularly in patients with pre-existing movement disorders [119]. These adverse effects are relatively rare but underscore the importance of monitoring for new or worsening movement abnormalities during antidementia therapy especially in individuals with Parkinson's disease [120]. Also, cognitive abnormalities can be misinterpreted in cases of severe dysautonomia and Parkinson's disease challenging the diagnosis of drug-induced movement disorders [121].

4.11. Opiods

Opioids can influence movement disorders through their effects on the dopaminergic and basal ganglia pathways, leading to both hyperkinetic and hypokinetic manifestations [122]. Chronic opioid use has been associated with opioid-induced myoclonus [123], tremor, and dystonia, particularly with high-dose or long-term therapy. Additionally, opioid withdrawal can precipitate restlessness, choreiform movements, and parkinsonism-like features, likely due to dopaminergic dysregulation. Rarely, opioids such as methadone have been linked to tardive dyskinesia, possibly through NMDA receptor modulation. These movement abnormalities highlight the complex interplay between the opioid system and motor control, necessitating careful monitoring in opioid-treated patients.

5. Confounding Causes of Movement Disorders Associated with Medications

Stroke is a well-recognized cause of secondary movement disorders, resulting from damage to key motor pathways and basal ganglia circuits [124]. Post-stroke movement disorders can be hyperkinetic or hypokinetic, depending on the location and extent of the lesion [125]. Post-stroke parkinsonism (PSP) is commonly associated with lesions in the basal ganglia, particularly the substantia nigra and striatum, leading to bradykinesia, rigidity, and postural instability [126]. Dystonia can occur after thalamic [127], putaminal, or pontine strokes, often presenting as delayed-onset hemidystonia. Hemichorea-hemiballismus (HCHB) is frequently observed in strokes affecting the subthalamic nucleus or its connections, causing involuntary, flinging limb movements [128]. Nystagmus and other abnormal eye movements, such as ocular flutter or skew deviation, can also occur post-stroke [129], particularly when the brainstem, cerebellum, or vestibular pathways are affected [130]. Limb-shaking transient ischemic attacks are characterized by brief, involuntary, jerky movements of the limbs, often precipitated by standing or walking [131], and are typically associated with significant carotid artery stenosis leading to cerebral hypoperfusion [131]. Additionally, specific cortical areas can be affected, leading to phenomena such as the hand knob [132] and foot knob syndromes [133], which manifest as focal motor deficits due to ischemia in the precentral gyrus. Other unusual presentations are corpus callosum stroke causing isolated astasia [134] and isolated infarction of the tonsil [135]. However, one of the major challenges associated with stroke remains the lack of awareness and health literacy among the general population, leading to misdiagnosis and underdiagnosis [136]. Endovascular thrombectomy (EVT), a standard treatment for acute ischemic stroke, can influence post-stroke movement disorders by restoring blood flow to critical motor pathways [137]. While EVT may improve outcomes by preventing extensive basal ganglia and thalamic damage [138], some patients may still develop secondary movement disorders such as post-stroke dystonia, chorea, or parkinsonism, particularly if there is delayed reperfusion or ischemic injury to deep brain structures [139].

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Infectious causes of movement disorders can arise from various pathogens affecting the central nervous system [140]. Viral infections, such as HIV [141], dengue viral infection [142], Japanese encephalitis [143], measles [144], and COVID-19 [145], have been linked to movement abnormalities like chorea [146], dystonia [147], myoclonus, cranial [148], and parkinsonism due to direct neurotropism and immune-mediated damage [149]. Bacterial infections, including syphilis [150,151], tuberculosis [152], Lyme disease [153], mucormycozis [154], Bartonella henselae [155], may lead to tremors [156], chorea, parkinsonism-plus syndromes [157], and ataxia when the nervous system is involved; also, Jarisch-Herxheimer reaction [158] is an acute inflammatory response that can occur after initiating antibiotic treatment for syphilis [159]. Post-infectious autoimmune syndromes, such as Sydenham's chorea following Group A streptococcal infection [160], result from molecular mimicry and basal ganglia dysfunction, causing hyperkinetic movements. Additionally, parasitic [161] and prion infections, including neurocysticercosis [162] and Creutzfeldt-Jakob disease [163], can trigger progressive movement disorders such as myoclonus [164], ataxia [165], and dystonia due to neuronal degeneration and inflammatory responses [166].

Demyelinating diseases, such as multiple sclerosis (MS) and neuromyelitis optica spectrum disorder (NMOSD), can cause movement disorders due to disruption of corticospinal and basal ganglia pathways [167]. Patients may present with tremor [168], dystonia, ataxia, myoclonus [169], or parkinsonism [170], often reflecting lesion location and inflammatory activity [171] within the central nervous system [172]. Certain epileptic syndromes can present with or mimic movement disorders, reflecting shared pathophysiological mechanisms. Progressive myoclonic epilepsies (PMEs), such as Lafora disease and Unverricht-Lundborg syndrome, or even Rasmussen encephalitis [173] cause myoclonus and ataxia, while infantile epileptic syndromes, like Dravet syndrome, may include dystonia or parkinsonism, highlighting the overlap between epilepsy and motor dysfunction. Systemic lupus erythematosus (SLE) can cause movement disorders, including chorea, dystonia, and ataxia, primarily due to autoimmune-mediated basal ganglia dysfunction and cerebrovascular involvement [174]. Glutamic acid decarboxylase 65 (GAD-65) antibodies are associated with autoimmune movement disorders, including stiff-person syndrome, myoclonus, ataxia, and dystonia, due to impaired GABAergic neurotransmission in the central nervous system [175].

Metabolic disorders, including abnormalities in glycemic control, can lead to movement disorders through disruptions in neuronal metabolism and neurotransmitter function [176]. Hyperglycemia, particularly in nonketotic hyperosmolar states, is associated with hemichorea-hemiballismus [177], often due to basal ganglia dysfunction [178]. Conversely, hypoglycemia can trigger myoclonus [179], dystonia, and parkinsonism, likely from neuronal energy failure. Other metabolic disorders, such as copper metabolism [180], mitochondrial diseases [181], autoimmune thyroiditis [182], uremia [183] or hemodialysis [184], hypokalemia [185], glutaric aciduria [186], adrenoleukodystotrphy [187], calcium [188], may present with tremor [189], dystonia [190], or chorea, reflecting systemic metabolic disturbances affecting basal ganglia circuits. Asterixis [191], a bilateral or unilateral negative myoclonus, is characterized by brief, involuntary lapses in posture, commonly associated with hepatic encephalopathy, uremia, and metabolic disorders affecting the central nervous system [192].

Pregnancy can influence the course of preexisting movement disorders or precipitate new-onset hyperkinetic or hypokinetic syndromes due to hormonal, immunological, metabolic, and cerebrovascular changes [193]. Chorea gravidarum, often linked to estrogen fluctuations or underlying rheumatic heart disease, is a well-recognized pregnancy-related movement disorder [194]. Dystonia, myoclonus, and tremor may also emerge or worsen, particularly in metabolic disorders such as Wilson's disease [195]. Additionally, pregnancy can impact Parkinson's disease and restless legs syndrome (RLS), often requiring medication adjustments to balance symptom control and fetal safety. Careful monitoring and multidisciplinary management are essential to optimize outcomes for both mother and child.

Deep brain stimulation (DBS) represents a promising avenue for treatment-resistant cases of DIMDs, particularly in severe tardive dyskinesia and parkinsonism induced by long-term medication

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use [196]. DBS of the globus pallidus internus (GPi) has shown efficacy in reducing involuntary movements and improving functional outcomes in patients with refractory tardive syndromes [197]. Future research should focus on optimizing DBS parameters, identifying ideal candidates, and integrating adaptive DBS systems that dynamically respond to real-time neural activity [198]. Additionally, non-invasive neuromodulation techniques, such as transcranial magnetic stimulation (TMS) and focused ultrasound, are being explored as potential therapeutic alternatives. Advancements in neurotechnology and personalized medicine will be instrumental in developing more targeted, effective interventions for DIMDs, ultimately improving patient outcomes [199].

Metaiodobenzylguanidine (MIBG) scintigraphy has been explored as a potential tool for distinguishing drug-induced parkinsonism (DIP) from Parkinson's disease (PD) [200], as DIP typically exhibits preserved cardiac MIBG uptake, whereas PD shows reduced uptake due to autonomic dysfunction [201]. This imaging modality may help clinicians differentiate between these conditions, aiding in the accurate diagnosis and appropriate management of drug-induced movement disorders [202].

Drug-induced movement disorders can have significant different types of presentations, from the primary movement disorders to belly dancer's dyskinesia [203] and Pisa syndrome [204]. Rabbit syndrome is a rare drug-induced movement disorder characterized by involuntary, rhythmic perioral tremors, resembling a rabbit's chewing movements. It is most commonly associated with long-term use of dopamine receptor-blocking agents, such as antipsychotics, and is considered a form of tardive dyskinesia, typically responding poorly to dopaminergic treatments [205]. Alternating hemiplegia of childhood (AHC) is a rare neurological disorder characterized by episodic hemiplegia, dystonia, ataxia, and choreiform movements, often mimicking primary movement disorders. Mutations in the ATP1A3 gene underlie AHC, leading to dysfunction in neuronal ion homeostasis, which can confound the diagnosis with paroxysmal dyskinesias or early-onset dystonia [206]. Drug-induced movement disorder emergencies, such as acute dystonic reactions, neuroleptic malignant syndrome, and serotonin syndrome, require urgent recognition and management to prevent severe complications, including respiratory distress and rhabdomyolysis [207].

6. Future Directions

The Future research on drug-induced movement disorders (DIMDs) should focus on precision medicine approaches [208], integrating genetic, biomarker, and neuroimaging data to predict individual susceptibility to these conditions [209]. Identifying genetic risk factors may help stratify patients who are more prone to developing DIMDs, allowing for personalized treatment strategies. Additionally, the role of artificial intelligence (AI) and machine learning in early diagnosis and therapeutic monitoring is an emerging area of interest. AI-driven algorithms can analyze large datasets to identify subtle motor abnormalities before they become clinically significant, facilitating early intervention. Furthermore, novel pharmacological treatments targeting underlying neurochemical imbalances, including glutamatergic and cholinergic modulation, are being explored to mitigate drug-induced movement complications.

Several clinical scales are used to assess drug-induced movement disorders (DIMDs), aiding in diagnosis and severity grading [210]. The Abnormal Involuntary Movement Scale (AIMS) is widely utilized to evaluate tardive dyskinesia, measuring involuntary facial, limb, and trunk movements associated with long-term dopamine-blocking agent use [211]. The Simpson-Angus Scale (SAS) quantifies drug-induced parkinsonism, assessing rigidity, bradykinesia, and other extrapyramidal symptoms. Additionally, the Barnes Akathisia Rating Scale (BARS) is used to evaluate akathisia, a distressing movement disorder characterized by inner restlessness and an inability to stay still [212].

Drug-induced parkinsonism (DIP) is influenced by several predictive factors, including older age, female sex, and a history of neuroleptic exposure, as these increase susceptibility to dopamine blockade [213]. Additionally, individuals with genetic predisposition (e.g., polymorphisms in dopamine receptor or metabolism genes) and preclinical neurodegeneration may have a higher risk of developing persistent parkinsonism even after drug discontinuation.

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7. Conclusions

The bibliometric analysis of drug-induced movement disorders (DIMDs) underscores the substantial growth in research, reflecting increased awareness and advancements in the field. Key insights from this analysis highlight the evolving landscape of DIMD research, with a focus on pathophysiology, genetic predisposition, and emerging therapeutics. Thematic trends reveal the prominence of antipsychotic-induced movement disorders, alongside increasing interest in novel treatment strategies, including neuromodulation and precision medicine. Additionally, artificial intelligence and machine learning are becoming pivotal tools in the early detection, diagnosis, and personalized management of DIMDs.

Despite these advancements, significant gaps remain in understanding the molecular mechanisms underlying DIMDs, necessitating further interdisciplinary collaboration. Integration of neurology, pharmacology, genetics, and computational science is crucial for developing targeted interventions. Enhanced global collaboration between research institutions, clinicians, and data scientists will facilitate translational research, ensuring that findings are effectively applied to clinical practice. Future studies should emphasize real-world data integration, biomarker discovery, and the development of predictive models to improve patient outcomes.

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