ORIGINAL ARTICLE

Spectrum and Clinical Burden of Drug-induced Ataxia in Low-resource Settings: An Observational Study

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Abstract

Background: The clinical manifestation of drug-induced ataxia (DIA) is well established clinically but often becomes unrecognized until actual ataxia occurs. DIA may include ataxia as its primary clinical symptom or may be one of multiple manifestations. This study aimed to characterize the development of DIA types and the timing of clinical resolution.

Materials and methods: Between January 2016 and March 2025, a prospective observational study of subjects diagnosed with DIA based on the development of ataxia after starting a drug and no prior history of ataxia was conducted. Collected data included the drug involved, timing to onset of symptoms, type of DIA (cerebellar vs sensory), and time required for resolution of symptoms.

Results: A total of 63 subjects met the criteria for inclusion in this analysis. Ataxia was most commonly attributed to antiepileptic drugs (AED) (44 subjects, 69.8%), with the remaining 19 subjects having ataxia after the use of chemotherapeutic or other drugs (30.2%). The most frequent drug associated with ataxia was phenytoin (22 subjects). The majority of ataxia experienced by the subjects was of the cerebellar variety (58.7%), whereas only 41.3% experienced a sensory form of ataxia. Symptoms of ataxia following the use of a drug were most commonly noted >72 hours after starting the drug (79.4%). In subjects with symptomatic improvement after intervention, 39.7% of subjects demonstrated improvement within 72 hours after intervention.

Conclusion: DIA may typically be caused by AEDs and chemotherapeutic drugs, with symptoms commonly manifesting >72 hours after initiation of drug therapy. Early recognition and treatment of DIA may improve clinical outcomes for individuals diagnosed with DIA.

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Introduction

Ataxia, derived from the Greek meaning "lack of order," is defined as the loss of coordinated voluntary movement, affecting gait, limb use, and speech. This impairment typically results from dysfunction of the cerebellum or its afferent/efferent pathways, including the sensory proprioceptive system. The etiology of ataxia is broad, encompassing genetic disorders, neurodegenerative conditions, vascular events, and metabolic disturbances.[1]

Drug-induced ataxia (DIA) is a significant iatrogenic condition that is often underrecognized in clinical practice despite being a prevalent adverse effect of numerous medications.[2] A systematic review has highlighted that DIA can result from a wide array of pharmaceuticals, including but not limited to antiepileptic drugs (AEDs), chemotherapeutic agents, psychotropic medications, and certain antibiotics.[2] The true incidence of DIA is difficult to ascertain, but it represents a considerable burden, particularly in polypharmacy and specialized clinical settings.

The pathophysiological mechanisms of DIA are complex and drug specific. Common mechanisms include: (1) direct neurotoxicity, particularly to the Purkinje cells of the cerebellum, which are highly susceptible to metabolic and excitotoxic injury; (2) disruption of key neurotransmitter pathways, such as the gamma-aminobutyric acid (GABA)-ergic system, leading to impaired cerebellar inhibition; and (3) damage to the peripheral sensory nerves or dorsal root ganglia, resulting in sensory ataxia.[3,4] For instance, phenytoin is believed to cause cerebellar toxicity through voltage-gated sodium channel blockade and subsequent Purkinje cell loss, while chemotherapeutic agents such as cytarabine can cause a dose-dependent cerebellar syndrome.[5,6]

Clinically, DIA can present on a spectrum from acute onset following a single dose or dose adjustment to an insidious, chronic progression with prolonged exposure.[2] The clinical phenotype can be purely cerebellar (e.g., gait ataxia, nystagmus, dysmetria), sensory (e.g., positive Romberg sign, impaired proprioception), or a mixed picture. While many cases of DIA are reversible with dose reduction or drug withdrawal, some agents, particularly certain chemotherapies, can cause permanent neurological sequelae.[7]

Despite its clinical importance, there is a paucity of comprehensive data on the clinical spectrum, temporal profiles, and outcomes of DIA from low-resource settings such as India. Most existing literature consists of case reports or reviews from high-income countries, limiting the generalizability of findings to populations with different genetic backgrounds, drug prescription patterns, and monitoring capabilities. This study was therefore conducted to provide a detailed characterization of the clinical and temporal profile of DIA in a tertiary care cohort in India, with the objective of enhancing clinical surveillance and enabling earlier therapeutic intervention.

Aims and Objectives

Primary Objective

This study was designed with the primary aim of characterizing the clinical and temporal profile of DIA in patients receiving antiepileptic and chemotherapeutic agents.

Secondary Objectives

The secondary objectives were to identify and quantify the most common drugs associated with ataxia within our clinical cohort and to classify the predominant type of ataxia—sensory versus cerebellar—while describing its associated clinical features. Furthermore, the study sought to analyze the temporal patterns of both symptom onset and clinical resolution following drug intervention, as well as to evaluate potential risk factors for DIA, including dose duration, drug levels, and patient demographics. Finally, we aimed to compare the clinical profiles and outcomes of ataxia induced by AEDs vs chemotherapeutic or other agents to determine whether distinct patterns exist between these drug classes.

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Materials and Methods

Study Design and Setting

This was a prospective, longitudinal observational study conducted in the Department of Neurology and General Medicine at Mahatma Gandhi Medical College and Research Institute, Puducherry, a tertiary care hospital in South India, from January 2016 to March 2025. The study protocol was approved by the Institutional Ethics Committee (MGM/IHEC/85/2016). Written informed consent was obtained from all patients willing to participate in the study.

Inclusion and Exclusion Criteria

The inclusion criteria for this study required patients to be aged 4 years or older, presenting with new-onset ataxia—as previously defined—following the initiation or dose modification of a drug. Eligible patients must have had no prior history of ataxia, cerebellar disease, or hereditary ataxia and must have had normal neuroimaging [computed tomography (CT) or magnetic resonance imaging (MRI) of the brain] to exclude structural causes. Exclusion criteria included a history of significant alcohol use disorder as per World Health Organization (WHO) criteria,[8] uncontrolled metabolic disorders such as hypothyroidism or Wilson disease, or an active central nervous system infection. Additionally, patients with preexisting peripheral neuropathy from causes other than the suspected drug—such as diabetes mellitus or vitamin B12 deficiency—were not eligible. Those with psychiatric or cognitive disorders that would preclude the ability to provide reliable clinical history or informed consent were also excluded.

Data Collection

For all enrolled participants, a standardized case report form was used to collect the following data:

Demographics: Age, sex.

Drug history: Name of the suspected drug, indication for use, daily dose, duration of therapy before symptom onset, and any recent dose modifications.

Clinical assessment: All patients underwent a comprehensive neurological examination performed by a neurologist. The type of ataxia was classified based on predefined clinical criteria adapted from standard neurological texts.[9]

Cerebellar ataxia: Presence of at least two of the following: gait ataxia (broad-based, unsteady), limb ataxia (dysmetria on finger-to-nose or heel-to-shin test), dysdiadochokinesia (impaired rapid alternating movements), hypotonia, and cerebellar ocular motor signs (e.g., gaze-evoked nystagmus, saccadic dysmetria).

Sensory ataxia: Presence of ataxia with impaired proprioception [positive Romberg's sign (which worsens with eye closure)] and signs of peripheral neuropathy (diminished or absent ankle jerks, impaired vibration sense), in the absence of classical cerebellar signs.

Investigations: Serum drug levels were measured where clinically indicated and available. These were categorized as therapeutic or supratherapeutic based on standard laboratory reference ranges.

Outcome: The time to clinical resolution (defined as return to premorbid baseline neurological function or significant improvement in ataxia symptoms) was recorded following drug withdrawal, dose reduction, or specific intervention (e.g., levocarnitine for valproate toxicity). Patients were followed until clinical resolution or for a maximum of 30 days.

Definitions and Categorization

Onset time: The time from the last drug dose modification (initiation or dose increase) to the first appearance of ataxia symptoms, categorized as follows:

Group A: ≤24 hours

Group B: 24–72 hours

Group C: >72 hours

Resolution time: The time from drug intervention (withdrawal or dose reduction) to documented clinical improvement, categorized as follows:

Group I: ≤72 hours

Group II: 72–144 hours

Group III: >144 hours

Sample Size Calculation

The sample size was calculated based on the primary outcome of mean recovery time between the antiepileptic and chemotherapy groups. Assuming a two-sided alpha of 5% and power of 80%, and anticipating a clinically significant difference of 3 days in recovery time with a pooled standard deviation (SD) of 2.5 days (based on pilot data and previous literature), the minimum required sample size was estimated to be 11 patients per group (using the formula for comparing two means). Our final sample of 44 patients in the AED group and 19 in the chemotherapy group was therefore adequately powered to detect the observed differences between the drug classes.

Statistical Analysis

Data were analyzed using Statistical Package for the Social Sciences (SPSS), version 26.0 (IBM Corp., USA). Descriptive statistics were presented as mean ± standard deviation (SD) for continuous variables and frequencies (percentages) for categorical variables. The primary outcome of resolution time was compared between drug classes (AEDs vs chemotherapeutic/other drugs) using an independent-samples *t*-test. Categorical variables were analyzed using the Chi-squared test or Fisher's exact test, as appropriate. A p-value < 0.05 was considered statistically significant for all tests. Risk ratios (RRs) along with 95% confidence intervals (CIs) were computed where applicable to quantify effect sizes.

Results

Participant Demographics and Clinical Profile

A total of 63 patients met the inclusion criteria and were enrolled in the study. The mean age was 48.2 years (SD 21.5, range: 4–89 years), with a near-equal sex distribution (34 males, 29 females). The primary drug class implicated was AEDs in 44 cases (69.8%), while chemotherapy and other drugs accounted for the remaining 19 cases (30.2%). Baseline demographics are detailed in Table 1.

Pharmacological Agents Linked to Ataxia

Phenytoin was the single most common offending agent, responsible for 34.9% (n = 22) of all cases. Carbamazepine was implicated in 19% (n = 12) of cases. Other AEDs included gabapentin (n = 3), zonisamide (n = 2), and lamotrigine (n = 2). Among chemotherapeutic and miscellaneous drugs, cytarabine and lithium each caused ataxia in 6.3% (n = 4) of cases. The antidepressant amitriptyline was implicated in 7.9% (n = 5) of cases. Other medications noted were ketamine, acyclovir, methotrexate, and mebendazole,

Table 1: Baseline demographic and clinical characteristics of study cohort (N = 63)

Characteristics	*Value*
Total patients	63
Mean age (±SD)	48.2 ± 21.5 years
Age range	4–89 years
Gender (male:female)	34:29
Primary drug category	
AEDs	44 (69.8%)
Chemotherapeutic/ other drugs	19 (30.2%)
Most common indication	Seizure disorder (n = 28)

Table 2: Frequency and percentage of drugs associated with ataxia

Drug	*Number of cases (N = 63)*	*Percentage (%)*	*Drug class*
Phenytoin	22	34.9%	Antiepileptic
Carbamazepine	12	19%	Antiepileptic
Amitrypylline	5	7.9%	Antidepressant
Cytarabine	4	6.3%	Chemotherapeutic
Lithium	4	6.3%	Mood stabilizer
Gabapentin	3	4.8%	Antiepileptic
ketamine	4	6.3%	Anesthetic
5-fluorouracil	2	3.2%	chemotherapeutic
Methotrexate	2	3.2%	Chemotherapeutic
Others*	5	7.9%	Miscellaneous

Others*—Zonisamide, Lamotrigine, Acyclovir, Mebendazole, Neem Oil, Immunoglobulin, Hep B Vaccine, Amiodarone, Cyclophosphamide

Table 3: Distribution of ataxia type and temporal onset groups

Variable	*Sensory ataxia (n = 26)*	*Cerebellar ataxia (n = 37)*	*Total (N = 63)*
Onset group, *n* (%)			
Group A (≤24 hours)	3 (11.5%)	4 (10.8%)	7 (11.1%)
Group B (24–72 hours)	2 (7.7%)	4 (10.8%)	6 (9.5%)
Group C (>72 hours)	21 (80.8%)	29 (78.4%)	50 (79.4%)
Most common signs			
Impaired proprioception	22 (84.6%)	–	–
Positive Romberg	19 (73.1%)	–	–
Dysmetria/dysdiadochokinesia	–	28 (75.7%)	–
Nystagmus/abnormal eye movements	–	18 (48.6%)	–
Ataxic gait	26 (100%)	37 (100%)	63 (100%)

Table 4: Time to clinical resolution after intervention

Resolution group	*AEDs (n = 44)*	*Chemotherapeutic/other drugs (n = 19)*	*Total (N = 63)*
Group I (≤72 hours)	19 (43.2%)	6 (31.6%)	25 (39.7%)
Group II (72–144 hours)	13 (29.5%)	4 (21.1%)	17 (27%)
Group III (>144 hours)	12 (27.3%)	9 (47.4%)	21 (33.3%)
Mean resolution time (days)	5.1 ± 2.3	8.2 ± 3.1	6.0 ± 2.9

Table 5: Clinical improvement by onset group

Onset group	*Resolution <72 hours (group I)*	*Resolution 72–144 hours (group II)*	*Resolution >144 hours (group III)*	*Total*
Group A (≤24 hours)	5	2	0	7
Group B (24–72 hours)	3	2	1	6
Group C (>72 hours)	17	13	20	50
Total	25	17	21	63

Table 6: Risk factor analysis in phenytoin induced ataxia (*n* = 22)

Factor	*Sensory ataxia (n = 14)*	*Cerebellar ataxia (n = 8)*	*Total (n = 22)*
Dose duration >1 week	12 (85.7%)	7 (87.5%)	19 (86.4%)
High serum drug level	10 (71.4%)	5 (62.5%)	15 (68.2%)
Dose increased prior to ataxia	6 (42.9%)	3 (37.5%)	9 (40.9%)
Mean resolution time (days)	6.2 ± 1.8	5.8 ± 1.5	6 ± 1.7
Peripheral neuropathy present	9 (64.3%)	2 (25%)	11 (50%)

highlighting the wide range of drugs that can precipitate ataxia (Table 2).

Classification and Clinical Presentation of Ataxia

The most frequent disorder observed in this group was cerebellar ataxia, with 37 patients (58.7%) affected. Other common cerebellar signs included dysmetria (*n* = 28, 75.7%) and dysdiadochokinesia (*n* = 25, 67.6%). Nystagmus or other oculomotor deficits were present in 18 patients (48.6%). Sensory ataxia was diagnosed in 26 patients (41.3%), characterized by impaired proprioception (*n* = 22, 84.6%), a positive Romberg sign (*n* = 19, 73.1%), and diminished tendon reflexes (*n* = 17, 65.4%). While phenytoin was associated with both types, it produced sensory ataxia more frequently (14/22 patients, 63.6%). In contrast, cytarabine caused exclusively cerebellar manifestations. The distribution of ataxia subtype by onset is presented in Table 3.

Timing of Symptom Onset and Recovery

A striking finding was the delayed onset of symptoms, with the vast majority of patients (*n* = 50, 79.4%) experiencing ataxia >72 hours after the last drug modification (group C). Only 11.1% (*n* = 7) had onset within 24 hours (group A), and 9.5% (*n* = 6) had onset between 24 and 72 hours (group B).

Following drug intervention, clinical recovery occurred within 72 hours (group I) in 39.7% (*n* = 25) of patients. A further 27.0% (*n* = 17) recovered within 72–144 hours (group II), and 33.3% (*n* = 21) had a prolonged recovery taking >144 hours (group III). Ataxia related to chemotherapeutic agents was associated with significantly longer recovery periods. The time to clinical recovery by drug class is shown in Table 4, and a cross-tabulation of onset and resolution groups is presented in Table 5.

Analysis of Contributing Factors

Within the phenytoin subgroup (*n* = 22), 15 subjects were determined to have elevated serum concentrations of phenytoin. The combination of high serum levels and a therapy duration of >3 weeks appeared to enhance symptom severity and prolong recovery time. Other risk factors associated with phenytoin-induced ataxia are shown in Table 6. Similar to phenytoin, carbamazepine demonstrated a comparable risk profile. For drugs that are not typically monitored, duration of treatment and cumulative dosage were the primary identified risk factors. Age >60 years was associated with both an increased frequency of ataxia and a longer

Table 7: Comparative profile of ataxia by drug class ($N = 63$)

Parameter	*AEDs (n = 44)*	*Chemotherapeutic/other drug (n = 19)*	*p-value*
Mean age (years)	45.3 ± 19.2	54.1 ± 24.7	0.12
Male:female ratio	22:22	12:7	0.32
Ataxia type, *n* (%)			0.03
Sensory	21 (47.7%)	4 (21.1%)	
Cerebellar	23 (52.3%)	15 (78.9%)	
Onset group C (>72 hours)	36 (81.8%)	14 (73.7%)	0.46
Resolution group III (>144 hours)	12 (27.3%)	9 (47.4%)	0.02
Abnormal drugs levels	28/34 (82.4%)	5/12 (41.7%)	<0.01

mean recovery time across all medication types.

Comparison between Drug Classes

The clinical profile of ataxia due to AEDs was different from that attributed to chemotherapy. Sensory ataxia was more frequent with AEDs (47.7%) than with chemotherapy (21.1%, $p = 0.03$). Conversely, cerebellar ataxia was the dominant phenotype in the chemotherapy group (78.9%). While the onset of symptoms was delayed (>72 hours) in the majority of both groups, the mean time to recovery from AED-related ataxia was significantly shorter than that from chemotherapy-related ataxia (5.1 vs 8.2 days, $p = 0.02$). A comparison of clinical features according to drug class is shown in Table 7.

Discussion

The current study describes the clinical and temporal characteristics of DIA and supports its identification as a preventable iatrogenic disorder.[2] The predominance of AEDs, particularly phenytoin, aligns with global literature that identifies these agents as common culprits due to their narrow therapeutic index and direct effects on the central nervous system (CNS).[5] The high proportion of phenytoin-related cases in our cohort likely reflects its widespread and often unmonitored use in the management of epilepsy in resource-limited settings.[10] Our finding that elevated serum phenytoin levels were present in a majority of these cases (68.2%) underscores the critical need for accessible and routine therapeutic drug monitoring to prevent neurotoxicity.[5]

The significant association between AEDs and sensory ataxia, as opposed to the predominantly cerebellar ataxia associated with chemotherapy, suggests divergent pathophysiological pathways.[3,4] Phenytoin's neurotoxicity, while classically linked to cerebellar Purkinje cell damage, also involves peripheral nerve injury, possibly through folate antagonism or direct axonal damage, leading to the sensory deficits observed in our patients.[6] In contrast, chemotherapeutic agents such as cytarabine are known for their selective toxicity to cerebellar granule cells, explaining the pure cerebellar syndrome seen in our cohort.[11]

Importantly, the majority (79.4%) of the subjects experienced a delayed onset of symptoms, indicating that DIA cannot solely be considered an acute toxic event. The presence of delayed symptom expression indicates that there may be other mechanisms at work; for example, cumulative neurotoxicity, progressive neuronal injury, or the unmasking of previously compensated subclinical deficits.[2,12] This finding reinforces the need for ongoing clinical monitoring of patients receiving chronic therapy because symptoms may develop many weeks after initiation of pharmacological treatment. This latency period creates a diagnostic challenge, as the temporal link between the drug and the symptom may not be immediately apparent to the clinician, potentially leading to delayed diagnosis and unnecessary investigations.[12]

The observed differences in recovery times between drug classes are clinically important. The relatively rapid recovery (mean 5.1 days) following withdrawal of most AEDs supports the concept of functional, reversible neurotoxicity in many cases. However, the significantly prolonged recovery (mean 8.2 days) and higher proportion of patients in Resolution group III for chemotherapy-associated ataxia highlight the potential for more irreversible, structural damage from these agents.[7,11] This finding is consistent with studies on high-dose cytarabine, where neurotoxicity can be dose limiting and recovery incomplete.[11]

Our risk factor analysis reinforces the importance of both pharmacokinetic and pharmacodynamic factors. The strong link between high serum drug levels and ataxia, even within the "therapeutic window" for some patients, highlights the interindividual variability in drug metabolism and CNS susceptibility.[13] Furthermore, age emerged as a significant nonmodifiable risk factor. The increased vulnerability in older adults is likely multifactorial, stemming from age-related declines in hepatic and renal clearance, reduced neurological reserve, and an increased likelihood of polypharmacy.[14]

Clinical Implications

In low-resource settings where access to frequent serum drug-level monitoring may be limited, our findings underscore the importance of high clinical vigilance. Physicians should maintain a high index of suspicion for DIA in any patient presenting with new-onset ataxia, particularly those receiving long-term AED therapy or chemotherapy. A detailed drug history, including recent dose changes, is important. Given the delayed onset, the absence of a recent dose change does not rule out DIA. Patients receiving high-risk medications, such as phenytoin, should undergo regular neurological assessments of gait and proprioception and serum monitoring. Patient and caregiver education regarding the early warning signs of ataxia is also vital to facilitate early reporting and intervention.

Strengths and Limitations

The main strength of our study is its prospective, longitudinal design with a well-characterized cohort from a real-world clinical setting. The systematic neurological phenotyping of ataxia is another key strength. However, several limitations must be acknowledged. First, as a single-center study, the findings may not be fully generalizable to other populations. Second, the observational nature of the study precludes the establishment of definitive causality, although the Naranjo Adverse Drug Reaction Probability Scale[15] was used implicitly in clinical assessment. Third, serum drug levels were not available for all patients, particularly for non-AEDs,

limiting our ability to perform a complete pharmacokinetic analysis for all cases. Finally, the sample size, while adequate for descriptive analysis, limited the statistical power for subgroup analyses of rarer drugs.

Future Directions

Multicenter prospective studies with standardized drug monitoring and biomarker integration should be the focus of future research. Additionally, exploring genetic polymorphisms in drug-metabolizing enzymes and drug transporters may support personalized medicine approaches to identify at-risk individuals before drug exposure, ultimately moving toward the prevention of DIA.[16]

Conclusion

Ataxia secondary to pharmacotherapy demonstrates specific clinical and temporal characteristics. Antiepileptic medications, especially phenytoin, account for the majority of cases, and chemotherapeutic agents lead to longer recovery courses. The frequently delayed onset of symptoms mandates ongoing, vigilant clinical surveillance throughout the course of therapy, not just immediately after initiation. Optimal management involves timely identification, prompt dose modification or drug withdrawal, and an understanding of the unique neurotoxic mechanisms of each causative agent. To protect patient safety and improve outcomes, the implementation of structured neurological assessments into the care plans of patients receiving high-risk medications is essential.

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