Case Report *Open Access*

International Journal of Clinical Drug Practice & Pharmacotherapy

DOI: *doi.org/10.63721/26IJCDPP106*

Risperidone-Induced Drug-Induced Parkinsonism with Akathisia: A Clinical Case Report

Hanoakh Bhosale[1], Girish Joseph[2*], Neena Bhatti[2], Asmi Gamaliel[1] and Dinesh K Badyal[3]

[1]Student, Phase III Part 2 , Christian Medical College & Hospital, Ludhiana
[*2]Assistant Professor, Christian Medical College & Hospital, Ludhiana
[3]Professor & HOD, Christian Medical College & Hospital, Ludhiana

Citation: *Hanoakh Bhosale, Girish Joseph, Neena Bhatti, Asmi Gamaliel, Dinesh K Badyal (2026) Risperidone-Induced Drug-Induced Parkinsonism with Akathisia: A Clinical Case Report. Int J. of Cli Drug Pract & Pharmaco 1(1), 1-5. WMJ/IJCDPP-106*

Abstract

Drug-induced parkinsonism is a common extrapyramidal adverse effect associated with dopamine receptor-blocking agents. It clinically mimics Parkinson's disease and is primarily characterized by bradykinesia and rigidity, while tremor and gait instability are less prominent. Although second-generation antipsychotics are generally considered safer than first-generation agents, they may still produce extrapyramidal symptoms, particularly at higher doses. We report the case of a 35-year-old male who developed pseudoparkinsonism and akathisia following treatment with risperidone for a schizophrenia-like psychotic disorder. Causality assessment using the Naranjo Adverse Drug Reaction Probability Scale and WHO-UMC criteria indicated a probable association with risperidone. The patient showed significant clinical improvement following withdrawal of the offending drug. This case highlights the importance of early recognition of extrapyramidal symptoms and cautious use of antipsychotics, even those considered relatively safer, to prevent potentially reversible adverse drug reactions.

***Corresponding author**: Girish Joseph, Assistant Professor, Christian Medical College & Hospital, Ludhiana, India.

Submitted: 24.04.2026 **Accepted**: 29.04.2026 **Published**: 15.05.2026

Keywords: Risperidone, Pseudoparkinsonism, Second Generation Antipsychotics, Extrapyramidal Symptoms, Adverse Drug Reaction

Introduction

Adverse drug reactions (ADRs) represent a significant cause of morbidity and are an important concern in clinical practice. The World Health Organization defines an ADR as "a response to a drug that is noxious, unintended, and occurs at doses normally used in humans for prophylaxis, diagnosis, or treatment of disease" [1-2].

Drug-induced movement disorders constitute an important subset of ADRs and encompass a wide spectrum of involuntary motor abnormalities. These include parkinsonism, akathisia, dystonia, myoclonus, and tardive dyskinesia, many of which are reversible upon withdrawal of the offending agent. Among these, drug-induced parkinsonism (DIP) is one of the most commonly encountered extrapyramidal syndromes and is particularly associated with the use of dopamine receptor-blocking agents. Historically, DIP was first described in the 1950s as a complication of neuroleptic therapy used in psychiatric disorders [3-4].

Clinically, DIP closely mimics idiopathic Parkinson's disease, presenting with features such as bradykinesia, cogwheel rigidity, resting tremor, and postural instability. However, compared to Parkinson's disease, tremor may be less prominent, and the onset is often more acute or subacute. This resemblance frequently leads to diagnostic confusion, resulting in inappropriate long-term treatment with antiparkinsonian medications. The term "pseudoparkinsonism" is often used to describe this drug-induced phenomenon, emphasizing its reversible nature and distinction from neurodegenerative pathology [4-5].

The pathophysiology of DIP primarily involves blockade of dopamine D_2 receptors in the nigrostriatal pathway, leading to an imbalance between dopaminergic and cholinergic neurotransmission within the basal ganglia. Additional mechanisms, including alterations in GABAergic transmission and mitochondrial dysfunction, have also been proposed, contributing to the complexity of these movement disorders. The risk of developing DIP is influenced by several factors, including drug potency, dosage, duration of therapy, and patient-related factors such as age and underlying neurological vulnerability [5-6].

Schizophrenia-like disorders, often classified under acute and transient psychotic disorders, are characterized by the rapid onset of psychotic symptoms such as delusions, hallucinations, and disorganized thinking [3]. Second-generation antipsychotics (SGAs), including risperidone, olanzapine, ziprasidone, and aripiprazole, are widely used in the management of these conditions due to their comparatively lower risk of extrapyramidal side effects relative to first-generation agents. However, emerging evidence suggests that SGAs are not devoid of such adverse effects, particularly when used at higher doses or for prolonged durations [7-9].

Risperidone is a commonly prescribed SGA that exerts its therapeutic effects primarily through antagonism of dopamine D_2 and serotonin 5-HT_2A receptors. At higher doses, its dopamine receptor occupancy approaches that of first-generation antipsychotics, thereby increasing the risk of extrapyramidal symptoms, including parkinsonism and akathisia. Despite being well recognized, these adverse effects remain underreported in routine clinical practice, especially with SGAs, leading to delays in diagnosis and management [6-7].

Given the potential reversibility of DIP, early recognition and timely withdrawal or dose adjustment of the offending drug are crucial to prevent unnecessary morbidity. Reporting such cases plays a vital role in strengthening pharmacovigilance systems, improving clinician awareness, and guiding safer prescribing practices. In this context, we present a case of risperidone-induced pseudoparkinsonism and akathisia in a young adult, highlighting the importance of vigilance even with commonly used second-generation antipsychotics.

Case Report

This case was documented as part of a pharmacovigilance elective conducted by the Department of Pharmacology at Christian Medical College, Ludhiana, which serves as an Adverse Drug Reaction (ADR) Monitoring Centre. The case was reported from the Department of Psychiatry at the same institution. The adverse drug reaction was also reported to the Pharmacovigilance Programme of India (PvPI) through the ADR Monitoring Centre at Christian Medical College, Ludhiana.

A 35-year-old male from Himachal Pradesh, working as a school teacher, presented to the Department of Psychiatry on 15 April 2025 with complaints of suspiciousness, auditory hallucinations, decreased sleep, irritability, and poor self-care. Based on clinical evaluation, he was diagnosed with a schizophrenia-like psychotic disorder. Baseline assessment revealed a Hamilton Anxiety Rating Scale score of 22 and a Hamilton Depression Rating Scale score of 20. The patient was initiated on risperidone 4 mg twice daily, along with clonazepam 0.5 mg at bedtime and escitalopram/paroxetine 12.5 mg once daily.

At follow-up after three months (9 July 2025), the patient reported restlessness and an urge to move his legs for the preceding two weeks, suggestive of akathisia. On clinical examination, tremors and bradykinesia were also noted, raising suspicion of drug-induced parkinsonism (pseudoparkinsonism). Causality assessment using the Naranjo Adverse Drug Reaction Probability Scale yielded a score of 7, indicating a probable association with risperidone [10].

Risperidone was subsequently discontinued, and the patient was started on lorazepam 2 mg three times daily for symptomatic management. Following withdrawal of the suspected drug, the patient showed significant improvement in extrapyramidal symptoms. The adverse drug reaction was reported to the VigiFlow database and registered under IPC number 301229469.

A detailed description of causality, severity and preventability is shown in Table 1.

Table 1: Assessment of Adverse Drug Reaction Using Standard Pharmacovigilance Tools

Assessment Tool	Criteria/Parameters	Result	Interpretation
Naranjo Adverse Drug Reaction Probability Scale[10]	Temporal association, improvement on drug withdrawal (dechallenge), absence of alternative causes, previous reports	Score: 7	Probable ADR
Modified Hartwig and Siegel Severity Scale[11]	Drug discontinued; no prolongation of hospital stays	Level 3	Moderate severity ADR
Schumock and Thornton Preventability Scale[12]	High dose of risperidone; need for monitoring	Yes	Probably preventable ADR

Discussion

Drug-induced parkinsonism (DIP) is one of the most common extrapyramidal adverse effects associated with antipsychotic therapy [3-5]. Although second-generation antipsychotics (SGAs) are generally considered to have a lower risk of extrapyramidal symptoms compared with first-generation agents, several studies have demonstrated that these adverse effects can still occur, particularly with agents such as risperidone [7-9]. In the present case, the patient developed symptoms of akathisia and pseudoparkinsonism after approximately three months of risperidone therapy. The temporal association between drug administration and onset of symptoms, along with improvement following drug withdrawal, supports a causal relationship [3-4].

The pathophysiology of DIP is primarily attributed to blockade of dopamine D_2 receptors in the nigrostriatal pathway, leading to an imbalance between dopaminergic and cholinergic neurotransmission within the basal ganglia. This disruption results in characteristic motor features such as tremor, rigidity, bradykinesia, and akathisia. Risperidone exerts its therapeutic effects through dopamine D_2 receptor antagonism in the mesolimbic pathway. However, non-selective blockade of D_2 receptors in the nigrostriatal pathway contributes to the development of extrapyramidal symptoms. The risk of such adverse effects is dose-dependent, with higher doses associated with increased receptor occupancy and a greater likelihood of movement disorders [5-9]

In this case, the patient was prescribed risperidone at a dose of 8 mg/day, which is relatively high and may have contributed significantly to the development of extrapyramidal symptoms. This finding is consistent with previous studies reporting increased extrapyramidal risk with higher doses of risperidone [7] Causality assessment using the Naranjo Adverse Drug Reaction Probability Scale categorized the reaction as probable [10]. The severity of the reaction was assessed as moderate using the Modified Hartwig and Siegel Severity Scale, and it was considered probably preventable based on the Schumock and Thornton criteria [11].

Early recognition of DIP is essential, as timely withdrawal or dose reduction of the offending drug can lead to significant clinical improvement and prevent unnecessary long-term treatment with antiparkinsonian medications [4-5]. Failure to recognize this condition may result in misdiagnosis as idiopathic Parkinson's disease, leading to inappropriate management. This case underscores that although SGAs are widely perceived as safer alternatives, they are not devoid of extrapyramidal adverse effects. Clinicians should remain vigilant, particularly when prescribing higher doses of risperidone, and should monitor patients closely for early signs of movement disorders. Furthermore, this report highlights the importance of pharmacovigilance in identifying and documenting such adverse drug reactions. Systematic reporting contributes to improved drug safety data, enhances clinician awareness, and supports the development of safer prescribing practices [12].

Limitations

This case report has certain limitations. First, as a single-patient observation, the findings cannot be generalized to a broader population. Second, although a clear temporal relationship and clinical improvement following drug withdrawal support a causal association, rechallenge was not performed due to ethical considerations, thereby limiting definitive confirmation. Third, the patient was receiving multiple psychotropic medications, which may act as potential confounding factors in the development of extrapyramidal symptoms. However, the temporal association with risperidone and improvement following its discontinuation strongly suggest its primary role. Additionally, objective rating scales for extrapyramidal symptoms, such as the Simpson-Angus Scale or Barnes Akathisia Rating Scale, were not used, which could have provided more quantitative assessment of symptom severity. Serum drug levels were also not measured, limiting pharmacokinetic correlation.

Future Directions

Further research is required to better understand the incidence and risk factors associated with risperidone-induced extrapyramidal symptoms, particularly in younger patients and those receiving higher doses. Prospective studies evaluating dose-response relationships and individual susceptibility factors would help guide safer prescribing practices. There is also a need to promote the routine use of standardized assessment tools for early detection of drug-induced movement disorders in clinical practice. Incorporating structured monitoring protocols may improve early recognition and reduce the risk of misdiagnosis.

Strengthening pharmacovigilance systems through increased reporting of adverse drug reactions is essential to generate robust real-world data and improve drug safety profiles. Educational initiatives aimed at clinicians can enhance awareness regarding the potential for extrapyramidal symptoms even with second-generation antipsychotics. Finally, future studies should explore strategies for individualized antipsychotic therapy, including dose optimization and selection of agents with lower extrapyramidal risk, to improve patient outcomes while minimizing adverse effects.

Conclusion

This case highlights the occurrence of extrapyramidal symptoms such as pseudoparkinsonism and akathisia following treatment with risperidone. Although second-generation antipsychotics are considered safer than first-generation agents, clinicians should remain vigilant for these adverse reactions, particularly at higher doses [6-8]. Early recognition, prompt withdrawal of the offending drug, and appropriate management can lead to significant improvement in symptoms. Increased reporting of such adverse drug reactions is important for improving awareness and patient safety [1].

Patient Consent

The authors certify that appropriate patient consent was obtained for publication of this case report. The patient understands that efforts will be made to conceal identity, although anonymity cannot be completely guaranteed

Conflict of Interest

The authors declare that there are no conflicts of interest related to this report.

Learning Points

- Second-Generation Antipsychotics are considered to be much safer drugs for psychiatric disorders with minimal risk for extrapyramidal symptoms but they may still cause akathisia and pseudoparkinsonism at higher doses.
- Dose dependent dopamine D2 receptor blockage is the most important hypothesis to explain the causation of the symptom.

- Early recognition and prompt withdrawal or change of the offending drug can lead to significant improvement in the patient's condition without causing unnecessary long-term treatment or hospital stay.
- Emphasis should be given on reporting of ADRs at national level through pharmacovigilance Programme of India for availability of Data on the topic which will help improve drug safety monitoring and awareness.
- Causality Assessment scales like Naranjo Adverse Drug Reaction Probability Scale and WHO-UMC criteria are good tools for analyzing the likelihood of ADRs.

References

1. Marshal M, Aggarwal S, Joseph G, Bhatti N, Badayl DK (2026) Nimesulide-Associated Generalized Bullous Fixed Drug Eruption: A Rare Pharmacovigilance Case Report. Cureus 18: e101371.
2. Franz C, Handa D, Joseph G, Bhatti N, Badyal DK, et al. (2026) Amoxicillin-Clavulanate-Induced Pityriasis Rosea: A Pharmacovigilance Case Report. Cureus 18.
3. Morabito BD, Paulison B (2017) Drug-induced parkinsonism: a case report. Ment Health Clin 7: 65-68.
4. Factor SA (2001) Drug-induced parkinsonism. Neurol Clin 19: 419-38.
5. Shin HW, Chung SJ (2012) Drug-induced parkinsonism. J Clin Neurol 8: 15-21.
6. Sharma S, Joseph G, Bhatti N, Badyal DK, Abhilasha P (2025) Risperidone-Induced Polymenorrhea: An Under recognized Endocrine Adverse Reaction. J of Adv Clin Neu Res 1: 1-4.
7. Thomson SR, Chogtu B, Bhattacharjee D, Agarwal (2017) Extrapyramidal symptoms probably related to risperidone treatment: a case series. Ann Neurosci 24: 155-163.
8. Fusar-Poli P, Kempton MJ, Rosenheck RA (2013) Efficacy and safety of second-generation long-acting injections in schizophrenia: a meta-analysis of randomized controlled trials. Int Clin Psychopharmacol 28: 57-66.
9. Bo QJ, Li XB, Wang ZM, Li AN, Ma X, et al. (2016) Extrapyramidal symptoms during risperidone maintenance treatment in schizophrenia: a prospective multicenter study. J Clin Psychopharmacol 36: 125-129.
10. Naranjo CA, Busto U, Sellers EM, Sandor P, Ruiz I, et al. (1981) A method for estimating the probability of adverse drug reactions. Clin Pharmacol Ther 30: 239-245.
11. Hartwig SC, Siegel J, Schneider PJ (1992) Preventability and severity assessment in reporting adverse drug reactions. Am J Hosp Pharm 49: 2229-2232.
12. Pirmohamed M, James S, Meakin S, Green C, Scott AK, et al. (2004) Adverse drug reactions as cause of admission to hospital: prospective analysis of 18 820 patients. BMJ. 329: 15-19.