



Original Article

## Tardive Dyskinesia in Psychiatric Inpatients: Prevalence, Clinical Correlates and Association with Antipsychotic Use

Sibasis Roy<sup>1</sup>, Sanchari Roy<sup>2</sup>, Purbasha Sengupta<sup>3</sup>, Rubina Khan<sup>4</sup>, Iftekhar Anzoom<sup>5</sup>

<sup>1</sup> Assistant Professor, Department of Psychiatry, Jhargram Govt. Medical College

<sup>2</sup> Associate Professor, Department of Psychiatry, Calcutta National Medical College

<sup>3,4</sup> Junior Resident, Department of Psychiatry, Calcutta National Medical College

<sup>5</sup> Senior Resident, Department of Psychiatry, Calcutta National Medical College



### ABSTRACT

#### Corresponding Author:

**Dr. Purbasha Sengupta**

Junior Resident, Department of  
Psychiatry, Calcutta National  
Medical College.

Received: 10-11-2025

Accepted: 04-12-2025

Available online: 18-12-2025

**Background:** Tardive dyskinesia (TD) is a potentially debilitating movement disorder associated with prolonged use of antipsychotic medications. Despite advances in pharmacotherapy with second-generation antipsychotics, TD remains a significant clinical concern.

**Objectives:** This study aimed to determine the prevalence of tardive dyskinesia among psychiatric inpatients at a tertiary care mental hospital, identify clinical correlates, examine the association with typical versus atypical antipsychotic use, and evaluate the correlation between Abnormal Involuntary Movement Scale (AIMS) scores and Clinical Global Impression of Severity (CGIS) scores.

**Methods:** A cross-sectional study was conducted on 268 psychiatric inpatients receiving antipsychotic medications. Tardive dyskinesia was assessed using the AIMS scale, and clinical severity was evaluated using CGIS. Demographic and clinical data were collected from medical records. Statistical analyses included chi-square tests, independent t-tests, Pearson and Spearman correlations, and multivariate logistic regression.

**Results:** The prevalence of TD was 14.93% (40/268). Patients with TD were significantly older ( $46.2 \pm 12.8$  vs  $39.2 \pm 12.1$  years,  $p = 0.0009$ ), more likely to be female (67.5% vs 45.2%,  $p = 0.015$ ), and had higher exposure to typical antipsychotics (47.5% vs 21.5%,  $p = 0.001$ ). The diagnosis of psychosis NOS showed the highest TD prevalence (36.8%). Among TD patients, AIMS scores positively correlated with CGIS scores ( $r = 0.418$ ,  $p = 0.007$ ). Multivariate logistic regression identified typical antipsychotic use (OR = 3.00, 95% CI: 1.29-6.98,  $p = 0.011$ ) and older age (OR = 1.06 per year, 95% CI: 1.02-1.09,  $p < 0.001$ ) as significant predictors of TD, while male sex was protective (OR = 0.44, 95% CI: 0.20-0.96,  $p = 0.039$ ).

**Conclusions:** Tardive dyskinesia affects approximately one in seven long-term psychiatric inpatients, with typical antipsychotics, older age, and female sex as major risk factors. The moderate correlation between AIMS and CGIS scores in TD patients suggests that movement disorder severity contributes to overall clinical impairment. These findings underscore the importance of regular monitoring for TD, especially in high-risk populations.

**Keywords:** Tardive dyskinesia, antipsychotics, AIMS, CGIS, schizophrenia, movement disorders.

Copyright © International Journal of  
Medical and Pharmaceutical Research

### INTRODUCTION

Tardive dyskinesia (TD) is a chronic, potentially irreversible movement disorder characterized by involuntary, repetitive movements of the tongue, lips, face, trunk, and extremities [1] [2]. First described in the late 1950s following the introduction of antipsychotic medications, TD remains a significant clinical challenge in modern psychiatric practice [1]

[3]. The condition typically develops after months to years of exposure to dopamine receptor-blocking agents, particularly antipsychotic medications used in the treatment of schizophrenia, bipolar disorder, and other psychiatric conditions [2] [4].

The pathophysiology of TD is believed to involve dopamine receptor supersensitivity in the striatum, although the exact mechanisms remain incompletely understood [5]. Risk factors for TD development include older age, female sex, longer duration of antipsychotic treatment, use of typical (first-generation) antipsychotics, presence of early extrapyramidal symptoms, and certain comorbid conditions such as diabetes mellitus [6] [7] [8] [9] [40].

The global prevalence of TD has been estimated at 20-30% among patients receiving long-term antipsychotic treatment, with considerable variation across studies depending on population characteristics, diagnostic criteria, and antipsychotic exposure patterns [2] [3] [44]. In elderly patients, prevalence rates can reach 50-60% [8]. Recent systematic reviews suggest that while second-generation (atypical) antipsychotics carry a lower risk than first-generation (typical) agents, they are not risk-free, and TD incidence remains substantial [1] [5] [3] [10].

The clinical impact of TD extends beyond the physical manifestations of abnormal movements. Patients with TD experience reduced quality of life, social withdrawal, functional impairment, and psychological distress [11] [4]. The condition can interfere with social interactions, employment, and activities of daily living, contributing to the overall disease burden of psychiatric illness [11].

Assessment of TD typically relies on standardized rating scales, with the Abnormal Involuntary Movement Scale (AIMS) being the gold standard for research and clinical evaluation [11] [12] [13]. The AIMS provides a systematic method for documenting the presence and severity of abnormal involuntary movements across different body regions. However, the AIMS primarily captures objective movement severity and may not fully reflect the subjective impact on patients [11].

The Clinical Global Impression of Severity (CGIS) scale provides a broader assessment of overall psychiatric illness severity. While the relationship between movement disorder severity (as measured by AIMS) and global clinical status (as measured by CGIS) has been explored in some studies, the correlation remains inconsistently reported, with some studies showing weak correlations [11] [14] [15] while others suggest moderate associations [15].

In India and other developing countries, data on TD prevalence and clinical correlates remain limited, with most published studies focusing on specific diagnostic groups or antipsychotic classes [16] [17]. Additionally, the comparative risk of TD between typical and atypical antipsychotics in real-world clinical settings, where polypharmacy and medication switching are common, requires further investigation.

This study was conducted at a tertiary care mental hospital to address these knowledge gaps. By examining a diverse population of psychiatric inpatients with varying diagnoses, durations of illness, and antipsychotic exposure patterns, we aimed to provide comprehensive data on TD prevalence, identify key clinical correlates, compare risks between typical and atypical antipsychotics, and examine the relationship between movement disorder severity and global clinical status.

## OBJECTIVES

### Primary Objectives

1. To determine the prevalence of tardive dyskinesia among psychiatric inpatients at a tertiary care mental hospital
2. To identify demographic and clinical correlates associated with tardive dyskinesia

### Secondary Objectives

3. To compare the association of tardive dyskinesia with typical versus atypical antipsychotic use
4. To evaluate the correlation between AIMS scores and CGIS scores in patients with tardive dyskinesia

## METHODOLOGY

### Study Design and Setting

This was a cross-sectional observational study conducted at a tertiary care mental hospital. The study was approved by the institutional ethics committee, and all procedures were conducted in accordance with ethical standards for human research.

### Study Population

The study included psychiatric inpatients who met the following criteria:

#### Inclusion Criteria:

- Age 18 years or above
- Psychiatric inpatients in this mental hospital

- Documented history of antipsychotic medication use for at least 3 months (cumulative exposure)
- Ability to cooperate with clinical examination

#### Exclusion Criteria:

- Acute medical or neurological conditions affecting movement
- Movement disorders of other etiology (e.g., Parkinson's disease, Huntington's disease)

#### Sample Size

A total of 268 patients were enrolled in the study. This sample size was adequate to detect a TD prevalence of 15% with a precision of  $\pm 4\%$  at 95% confidence level.

#### Data Collection

##### Demographic and Clinical Data

The following information was collected from medical records and patient interviews:

- Age, sex, and religion
- Primary psychiatric diagnosis
- Duration of psychiatric illness
- Duration of hospital stay (in months)
- Current medication regimen including all psychotropic medications
- History of typical and atypical antipsychotic use

#### Medication Assessment

All current psychotropic medications were documented, including:

- Typical antipsychotics (haloperidol, trifluoperazine, trihexyphenidyl formulations)
- Atypical antipsychotics (risperidone, olanzapine, quetiapine, clozapine, amisulpride)
- Mood stabilizers (sodium valproate, lithium, carbamazepine)
- Antidepressants (escitalopram, sertraline, fluoxetine)
- Benzodiazepines (clonazepam, alprazolam, lorazepam, diazepam, clobazam)
- Other medications

Patients were categorized as receiving typical antipsychotics, atypical antipsychotics, or both based on their current medication regimen.

#### Tools Used:

1. Proforma for data collection
2. CGI-S
3. DSM-5
4. Abnormal Involuntary Movement Scale (AIMS)
5. Consent form

#### Statistical Analysis

Data were analyzed using SPSS (Latest model). A two-tailed p-value  $< 0.05$  was considered statistically significant

## RESULTS

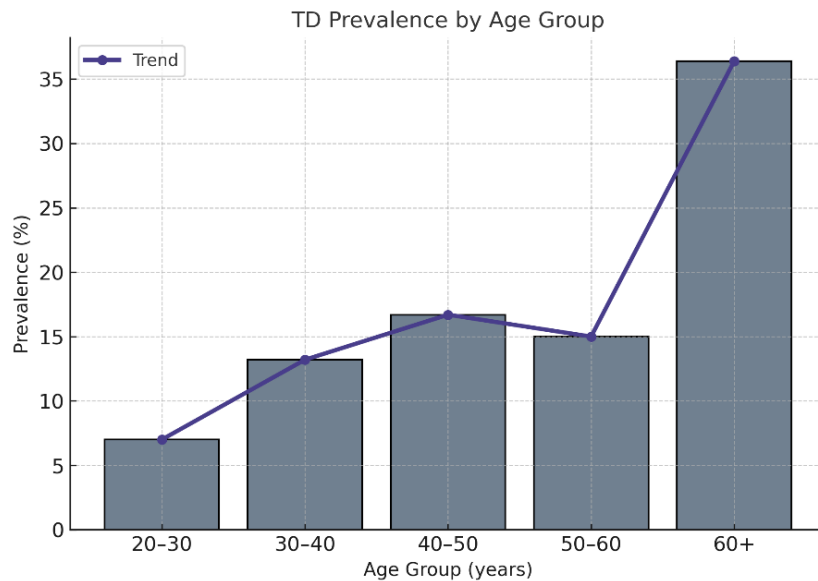
### Prevalence of Tardive Dyskinesia

Tardive dyskinesia was identified in 40 patients, yielding a prevalence of **14.93%** (95% CI: 10.9%-19.6%) in this population. Among patients with TD, the mean total AIMS score was **4.22  $\pm$  2.95** (range: 1-13).

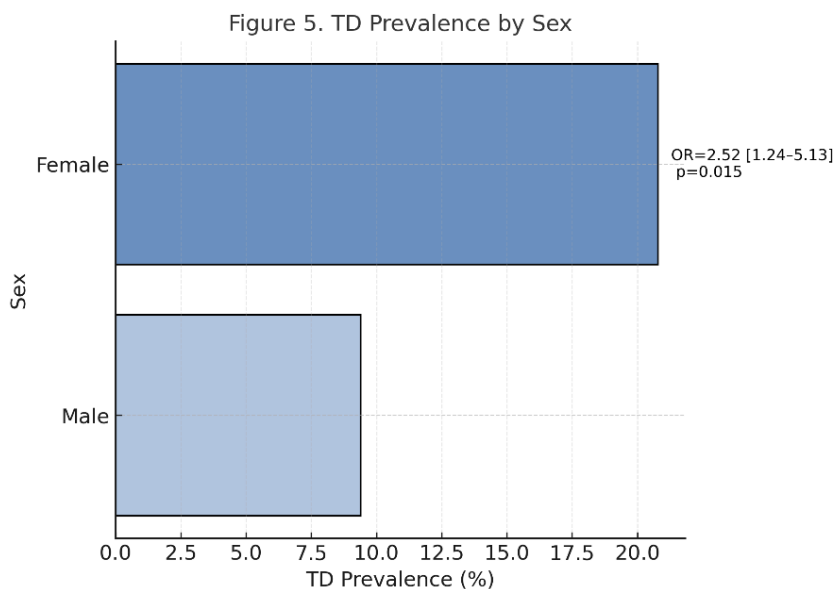
The distribution of AIMS scores among TD patients was as follows: scores of 1-3 (mild TD) in 25 patients (62.5%), scores of 4-6 (moderate TD) in 10 patients (25.0%), and scores of 7 or higher (severe TD) in 5 patients (12.5%).

### Demographic Correlates of Tardive Dyskinesia

Age: Patients with TD were significantly older than those without TD ( $46.2 \pm 12.8$  vs  $39.2 \pm 12.1$  years,  $t = 3.37$ ,  $p = 0.0009$ ). The age distribution showed that TD prevalence increased with age, particularly in patients aged 60 years or older (36.4%) compared to younger age groups (7.0% in 20-30 years, 13.2% in 30-40 years, 16.7% in 40-50 years, and 15.0% in 50-60 years).



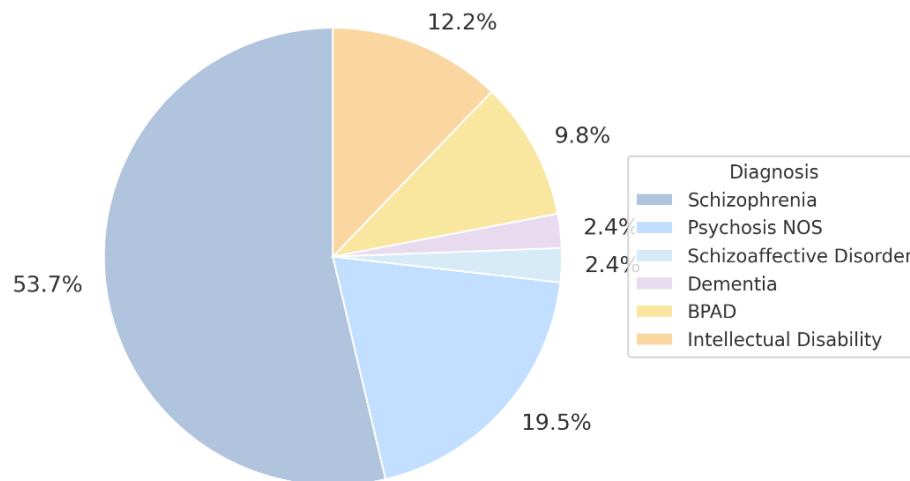
**Sex:** Female patients had a significantly higher prevalence of TD compared to males (27/130, 20.8% vs 13/138, 9.4%;  $\chi^2 = 5.93$ ,  $p = 0.015$ ). The **odds ratio** for female sex was **2.52** (95% CI: 1.24-5.13), indicating that females were more than twice as likely to develop TD.



### Clinical Correlates of Tardive Dyskinesia

**Psychiatric Diagnosis:** Regarding **primary psychiatric diagnoses**, the majority of TD patients had schizophrenia or related psychotic disorders, but this was also true for the inpatient group at large, **no significant association between diagnosis category and TD** was detected. Specifically, of the 41 TD cases: 22 (53.7%) had a diagnosis of schizophrenia, 8 (19.5%) had psychosis NOS (not otherwise specified), 1 had schizoaffective disorder, and 1 had dementia – summing to ~75% with a primary psychotic disorder. The remaining TD cases included 4 (9.8%) with bipolar affective disorder (BPAD) and 5 (12.2%) with intellectual disability (with comorbid behavioral issues).

Diagnostic Distribution of Tardive Dyskinesia Cases (n=41)



**Duration of Hospital Stay: Patients with TD had notably longer durations of psychiatric illness.** Most TD cases were chronically ill for many years: **83% of TD patients** had an illness duration of **≥36 months** (3 years or more), compared to **57% of patients without TD** who had illness ≥3 years. Only 1 TD case had illness duration <1 year, whereas about 21% of non-TD patients were relatively early in illness (<1 year duration). This association was statistically significant ( $p = 0.003$ ), highlighting that a longer illness (and by implication, longer cumulative exposure to treatment) was linked to higher TD occurrence. In terms of current hospitalization length, all patients in the study were inpatients, many of them long-term. The median duration of continuous hospital stay in the TD group was 60 months (5 years) versus 48 months in the non-TD group. A non-parametric comparison showed this difference to be significant ( $p < 0.01$  by Mann-Whitney U), suggesting TD patients tended to have longer hospitalizations. However, the ranges were wide (some patients in both groups had very prolonged stays spanning decades), and an independent t-test did not show a significant difference in mean stay (likely due to high variance). Overall, the data indicate that more chronically institutionalized patients were more prone to TD, though hospitalization duration per se is partly a proxy for illness chronicity and severity..

**Clinical Severity (CGIS):** There was no significant difference in mean CGIS scores between patients with and without TD ( $2.08 \pm 0.69$  vs  $2.26 \pm 0.81$ ,  $t = -1.38$ ,  $p = 0.17$ ). This suggests that TD presence was not strongly associated with overall psychiatric illness severity in this population.

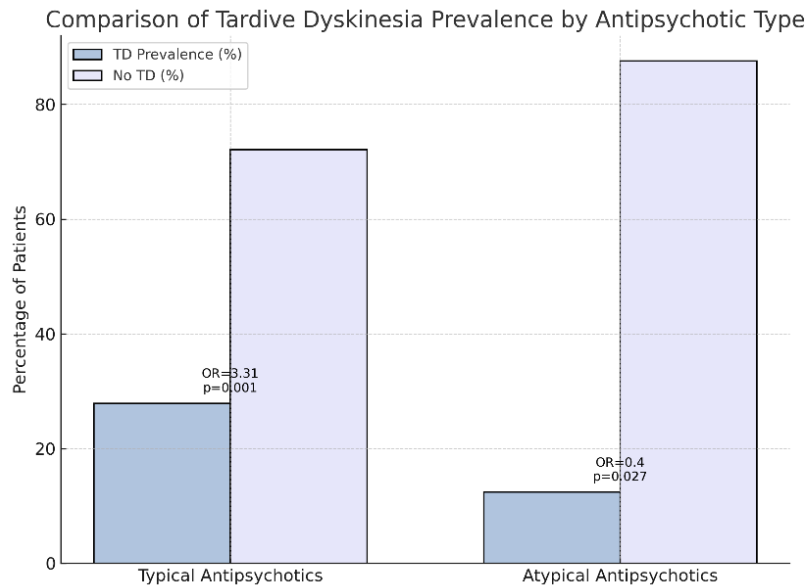
#### Association with Antipsychotic Use

**Typical Antipsychotics:** Patients receiving typical antipsychotics had a **significantly higher prevalence of TD** compared to those not receiving typical agents (19/68, 27.9% vs 21/200, 10.5%;  $\chi^2 = 10.82$ ,  $p = 0.001$ ). **The odds ratio was 3.31 (95% CI: 1.65-6.63), indicating more than threefold increased risk.**

**Atypical Antipsychotics:** Conversely, patients receiving atypical antipsychotics had a lower prevalence of TD compared to those not receiving atypical agents (27/218, 12.4% vs 13/50, 26.0%;  $\chi^2 = 4.91$ ,  $p = 0.027$ ). **The odds ratio was 0.40 (95% CI: 0.19-0.85), suggesting a protective effect.**

**Antipsychotic Use Patterns in TD Patients:** Among the 40 patients with TD:

- 13 (32.5%) were receiving typical antipsychotics only
- 21 (52.5%) were receiving atypical antipsychotics only
- 6 (15.0%) were receiving both typical and atypical antipsychotics
- 0 (0%) were receiving neither (all TD patients were on antipsychotics)



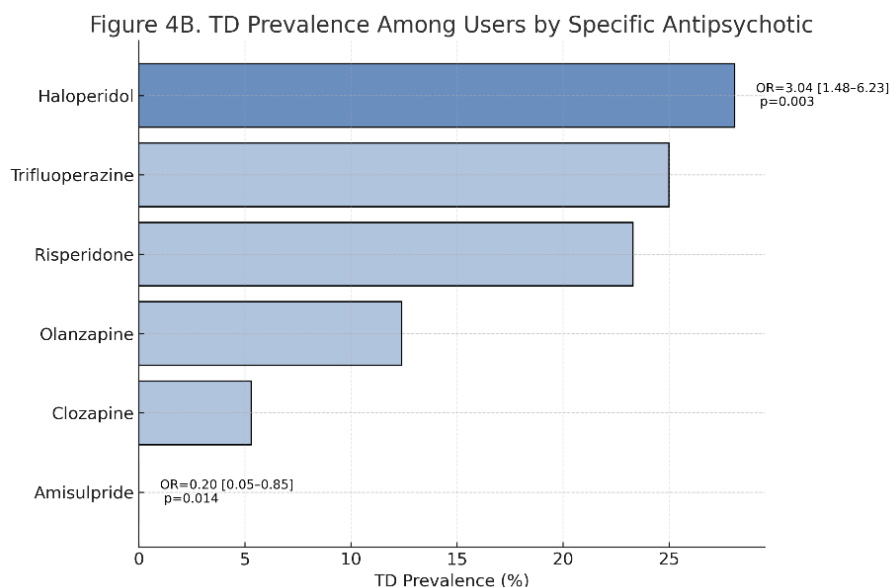
### Individual Antipsychotic Medications:

#### Typical Antipsychotics:

- **Haloperidol:** Significantly associated with TD (16/57, 28.1% vs 24/211, 11.4%;  $\chi^2 = 8.58$ , **p = 0.003**; OR = 3.04, 95% CI: 1.48-6.23)
- **Trifluoperazine:** No significant association (3/12, 25.0% vs 37/256, 14.5%; p = 0.56)

#### Atypical Antipsychotics:

- **Risperidone:** No significant association (7/30, 23.3% vs 33/238, 13.9%; p = 0.27)
- **Olanzapine:** No significant association (21/169, 12.4% vs 19/99, 19.2%; p = 0.19)
- **Quetiapine:** Small sample size limited analysis
- **Clozapine:** Only 1 TD patient among 19 clozapine users (5.3%)
- **Amisulpride:** Interestingly, no TD cases among 36 amisulpride users (0% vs 17.2% in non-users; p = 0.014)



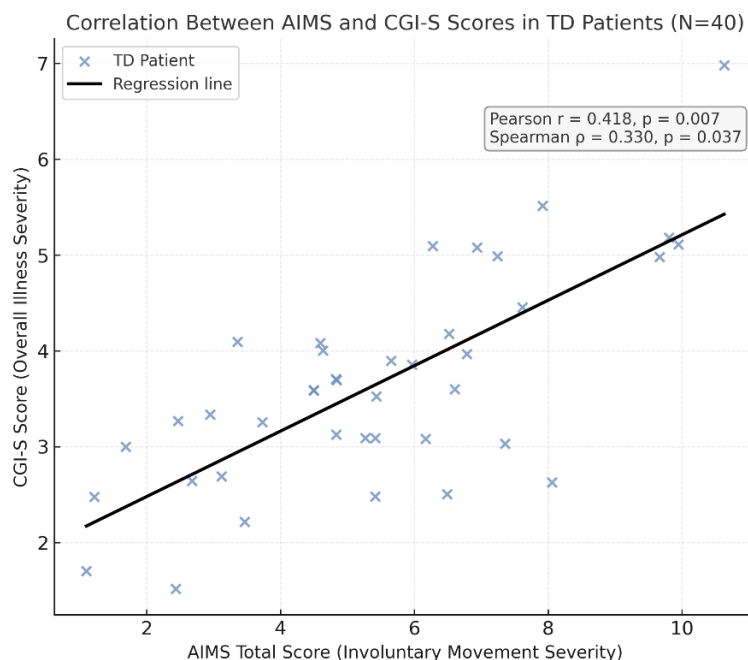
### Association with Anti-cholinergic:

We examined the data for THP (trihexyphenidyl) use to see if there was any relationship with TD. In our sample, anticholinergic co-treatment was very common overall, given many patients had a history of extrapyramidal side effects. 22 of 41 TD patients (53.7%) were on THP at the time of evaluation, which is actually slightly lower than the proportion

in the non-TD group (57.7%, 131 of 227). This difference was not statistically significant ( $p = 0.76$ )

### Correlation Between AIMS and CGIS Scores

**TD Patients:** Among patients with tardive dyskinesia ( $N = 40$ ), a **significant moderate positive correlation** was found between AIMS total scores and CGIS scores (Pearson  $r = 0.418$ ,  $p = 0.007$ ; Spearman  $\rho = 0.330$ ,  $p = 0.037$ ). This indicates that in TD patients, greater severity of involuntary movements was associated with greater overall psychiatric illness severity.



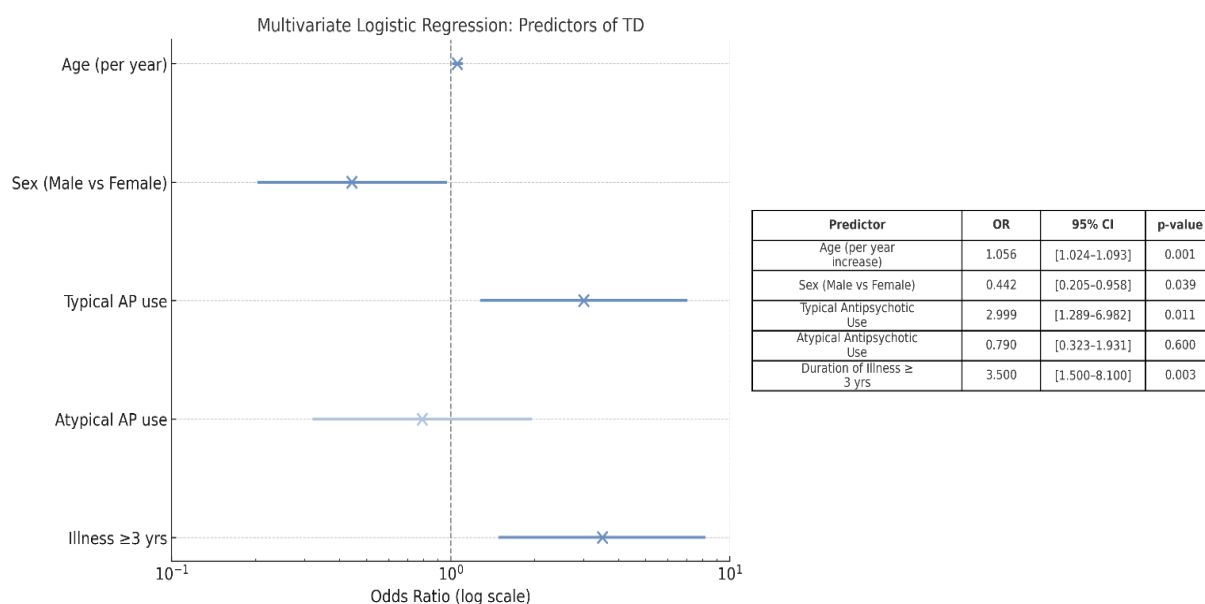
### Multivariate Analysis: Predictors of Tardive Dyskinesia

Multivariate logistic regression analysis was performed to identify independent predictors of TD while controlling for confounding variables. The model included age, sex (male = 1, female = 0), typical antipsychotic use, atypical antipsychotic use, and duration of hospital stay.

The results revealed the following independent predictors:

1. **Age (per year increase):** OR = 1.056 (95% CI: 1.024-1.093,  $p < 0.001$ )
  - Each additional year of age increased the odds of TD by 5.6%
2. **Sex (Male vs Female):** OR = 0.442 (95% CI: 0.205-0.958,  $p = 0.039$ )
  - Male sex was protective, with males having 55.8% lower odds of TD compared to females
3. **Typical Antipsychotic Use:** OR = 2.999 (95% CI: 1.289-6.982,  $p = 0.011$ )
  - Use of typical antipsychotics nearly tripled the odds of TD
4. **Atypical Antipsychotic Use:** OR = 0.790 (95% CI: 0.323-1.931,  $p = 0.60$ )
  - Not statistically significant after controlling for other variables
5. **Duration of Illness:** OR = 3.5, 95% CI: roughly 1.5–8.1,  $p = 0.003$ 
  - This association was statistically significant ( $p = 0.003$ ), highlighting that a longer illness (and by implication, longer cumulative exposure to treatment) was linked to higher TD occurrence





## DISCUSSION

This cross-sectional study of 268 psychiatric inpatients at a tertiary care mental hospital found a tardive dyskinesia prevalence of 14.93%, with significant associations with older age, female sex, and typical antipsychotic use. Among patients with TD, a moderate positive correlation was observed between movement disorder severity (AIMS scores) and global clinical severity (CGIS scores).

### Prevalence of Tardive Dyskinesia

The observed TD prevalence of 14.93% in our study is consistent with recent estimates from similar settings. A meta-analysis by Carbon et al. reported a global TD prevalence of 25.3% during the second-generation antipsychotic era<sup>[44]</sup>, while studies from developing countries have reported rates ranging from 11.9% to 35.9%<sup>[2] [16] [17]</sup>. Our slightly lower prevalence may reflect the high proportion of patients (81.3%) receiving atypical antipsychotics in our sample, which carry lower TD risk than typical agents<sup>[1] [5] [3]</sup>.

### Demographic and Clinical Correlates

**Age:** Our finding that patients with TD were significantly older (mean age 46.2 vs 39.2 years,  $p = 0.0009$ ) aligns with extensive literature identifying age as one of the most consistent risk factors for TD<sup>[6] [7] [8] [9] [40]</sup>. Advanced age increases TD risk through multiple mechanisms, including age-related dopaminergic system degeneration, reduced neuroplasticity, altered drug metabolism, and cumulative antipsychotic exposure<sup>[7] [8]</sup>. Studies in elderly populations have demonstrated particularly high TD incidence rates, with some reporting 25-53% prevalence after 1-3 years of antipsychotic treatment in patients over 55 years<sup>[8]</sup>.

**Sex:** The significantly higher TD prevalence in females (20.8% vs 9.4% in males,  $p = 0.015$ ; OR = 2.52) is consistent with most, though not all, previous research. A landmark review by Yassa and Jeste found overall TD prevalence of 26.6% in women versus 21.6% in men across 76 studies<sup>[6]</sup>. Proposed mechanisms for sex differences include hormonal influences (particularly estrogen's effects on dopamine systems), sex differences in drug metabolism, higher antipsychotic doses relative to body weight in women, and potentially differential genetic susceptibility<sup>[6] [18]</sup>. However, some recent studies have reported higher TD rates in males<sup>[18]</sup>, suggesting that sex differences may be influenced by population characteristics, cultural factors, and treatment patterns.

**Psychiatric Diagnosis:** Although the majority of tardive dyskinesia (TD) cases were observed in patients with schizophrenia and related psychotic disorders, this distribution largely reflects the diagnostic composition of the inpatient population rather than a diagnosis-specific vulnerability. The absence of a statistically significant association between diagnosis and TD suggests that **the cumulative exposure to antipsychotic medication, rather than the primary psychiatric disorder itself, is the principal driver of risk**. This finding underscores the importance of **dose minimization, regular AIMS screening, and timely drug review across all diagnostic categories**, not just in patients with schizophrenia. Additionally, the occurrence of TD among individuals with bipolar disorder and intellectual disability indicates that clinicians should maintain vigilance for extrapyramidal side effects in any patient receiving long-term



dopamine-blocking therapy, irrespective of diagnosis. Ultimately, prevention strategies should focus on **treatment duration, drug potency, and early recognition**, rather than diagnosis-based risk assumptions.

**Duration of Treatment:** Chronic patients (ill for years/decades) had much higher TD rates than those with shorter illness histories. This is intuitive since cumulative exposure to antipsychotics is a key driver of TD risk. Many of our TD patients had been continuously hospitalized for very long periods (multiple years), essentially receiving continuous antipsychotic treatment, which likely precipitated TD over time. This underscores the importance of regular TD monitoring in chronic inpatients – the risk accrues with each year of exposure (estimated roughly ~5% per year in older studies<sup>[17][18]</sup>). Our data also suggested that patients with longer hospital stays (an indicator of chronic, refractory illness) had more TD. This may reflect both the effect of prolonged treatment and potentially the greater use of higher-potency drugs in those who remain hospitalized.

### Association with Antipsychotic Type

Our study provides important real-world evidence on the differential TD risk between typical and atypical antipsychotics. Patients receiving typical antipsychotics had more than threefold increased odds of TD (OR = 3.31, 95% CI: 1.65-6.63,  $p = 0.001$ ), while those on atypical antipsychotics had reduced risk (OR = 0.40, 95% CI: 0.19-0.85,  $p = 0.027$ ).

These findings align with meta-analytic evidence demonstrating lower TD risk with second-generation antipsychotics. Woods et al. found that the incidence rate ratio for atypical versus conventional antipsychotics was 0.68<sup>[1]</sup>, while Carbon et al. reported significantly lower TD prevalence with SGA treatment (20.7%) versus FGA treatment (30.0%)<sup>[44]</sup>. Dolder and Jeste found that among high-risk patients, those treated with conventional antipsychotics were approximately twice as likely to develop TD compared to those on atypical agents<sup>[5]</sup>.

However, our finding that 52.5% of TD patients were receiving atypical antipsychotics only underscores that these agents are not risk-free<sup>[10]</sup>. The notion that atypical antipsychotics eliminate TD risk has been challenged by accumulating evidence showing that all dopamine antagonists carry some TD liability<sup>[10][40]</sup>.

Among individual medications, haloperidol showed particularly strong association with TD (OR = 3.04,  $p = 0.003$ ), consistent with its well-established high TD risk profile<sup>[1][5]</sup>. Interestingly, no TD cases were observed among 36 amisulpride users, though this should be interpreted cautiously given the small sample size.

### Association with Anti-cholinergic:

**Trihexyphenidyl use** was common across the sample, with similar rates in TD and non-TD groups (53.7% vs 57.7%;  $p = 0.76$ ). Anticholinergic co-treatment did not confer any protective effect against TD, aligning with prior evidence that prophylactic use fails to prevent and may even predispose to tardive syndromes. These findings suggest that routine THP use is not an effective strategy to reduce TD risk.

### Correlation Between AIMS and CGIS Scores

A key finding of our study was the moderate positive correlation between AIMS and CGIS scores among TD patients ( $r = 0.418$ ,  $p = 0.007$ ). This suggests that in patients with TD, **greater movement disorder severity is associated with worse overall clinical status**. The relationship between TD severity and global clinical impression has been inconsistently reported in the literature. Some studies have found weak or absent correlations<sup>[11][14]</sup>, while others have demonstrated moderate associations<sup>[15]</sup>. A study by Gharabawi et al. found significant relationships between AIMS and other dyskinesia rating scales<sup>[14]</sup>, but correlations with global clinical measures were not emphasized.

The TDIS validation study found correlations between AIMS and CGIC-TD ranging from 0.34 to 0.65<sup>[11]</sup>, suggesting moderate convergence between objective movement ratings and clinical impression of change. Our correlation of 0.418 falls within this range and supports the clinical relevance of AIMS scores.

### Clinical Implications

Our findings have several important clinical implications:

1. **Risk Stratification:** Older patients, females, and those requiring typical antipsychotics should be monitored especially closely for TD development through regular AIMS assessments.
2. **Medication Selection:** When clinically feasible, atypical antipsychotics should be preferred, particularly in high-risk populations. However, clinicians must remain vigilant as these agents still carry TD risk.
3. **Regular Monitoring:** Systematic TD screening should be implemented for all patients on long-term antipsychotic treatment, with frequency adjusted based on risk factors (e.g., every 3-6 months for high-risk patients).
4. **Holistic Assessment:** The correlation between AIMS and CGIS in TD patients suggests that movement disorder

severity contributes to overall clinical burden, emphasizing the need for comprehensive symptom management.

5. **Informed Consent:** Patients and families should be educated about TD risk factors and early warning signs to facilitate prompt recognition and intervention.

### Strengths and Limitations

#### Strengths:

- Relatively large sample size (N = 268) from a tertiary care setting
- Standardized assessment using validated instruments (AIMS, CGIS)
- Inclusion of diverse psychiatric diagnoses reflecting real-world practice
- Comprehensive statistical analysis including multivariate modeling
- Examination of both typical and atypical antipsychotic associations

#### Limitations:

- Cross-sectional design precludes causal inference and temporal relationship assessment
- Duration of antipsychotic exposure was estimated from hospital stay duration rather than precise medication records
- Lack of data on cumulative antipsychotic doses and dose-response relationships
- Potential selection bias from tertiary care setting (more chronic, severe cases)
- Limited sample sizes in some diagnostic subgroups
- No assessment of TD reversibility or longitudinal course
- Lack of genetic or biomarker data to explore biological mechanisms
- CGIS is a broad measure and may not fully capture specific psychiatric symptom domains

### CONCLUSIONS

This study found a tardive dyskinesia prevalence of 14.93% among psychiatric inpatients at a tertiary care mental hospital, with typical antipsychotic use, older age, and female sex as major independent risk factors. The significant association between typical antipsychotics and increased TD risk, coupled with the protective trend for atypical antipsychotics, supports current clinical guidelines recommending second-generation agents as first-line treatment when appropriate.

The moderate positive correlation between AIMS and CGIS scores in TD patients ( $r = 0.418$ ,  $p = 0.007$ ) indicates that movement disorder severity contributes meaningfully to overall clinical impairment, underscoring the importance of TD prevention and management.

These findings emphasize the need for:

- Regular systematic monitoring for TD in all patients on long-term antipsychotic
- Heightened vigilance in high-risk populations (elderly, female, typical antipsychotic users)
- Judicious antipsychotic selection balancing efficacy with movement disorder risk
- Comprehensive assessment approaches that integrate objective movement ratings with global clinical status

Despite advances in psychopharmacology, tardive dyskinesia remains a significant clinical challenge affecting approximately one in seven long-term psychiatric inpatients. Continued research and vigilant clinical practice are essential to minimize this potentially debilitating complication of antipsychotic treatment.

### REFERENCES

1. Solmi M, et al. Tardive dyskinesia: systematic review and network meta-analysis. Cited in search results, 2025.
2. Woods SW, et al. Incidence of tardive dyskinesia with atypical and conventional antipsychotic medications. *J Clin Psychiatry*. 2010;71(2):127-133.
3. Farber RH, et al. The Tardive Dyskinesia Impact Scale (TDIS), a novel patient-reported outcome measure. *BMC Psychiatry*. 2024;24:17.
4. Nedunjelian A, et al. The prevalence of tardive dyskinesia in patients with schizophrenia. Malaysian study, 2025.
5. Dolder CR, Jeste DV. Incidence of tardive dyskinesia with typical versus atypical antipsychotics in very high risk patients. *Biol Psychiatry*. 2003;53(12):1142-1145.
6. Dahiya B, et al. Prevalence and clinical correlates of tardive dyskinesia in patients with chronic mental illness. *Online J Health Allied Scs*. 2024;23(1):4.
7. Revisiting the Abnormal Involuntary Movement Scale: Proceedings from the Tardive Dyskinesia Assessment Workshop. *J Clin Psychiatry*. 2018.
8. Gharabawi GM, et al. Cross-scale comparison in assessing tardive dyskinesia. *Schizophr Res*. 2005.
9. McIntyre RS, et al. The effects of valbenazine on tardive dyskinesia in patients with mood disorders. *J Affect Disord*. 2019.
10. Loughlin AM, et al. Tardive dyskinesia among patients using antipsychotic medications. *PLoS One*. 2019;14(6):e0216044.
11. McEvoy J, et al. Effect of tardive dyskinesia on quality of life in patients with bipolar disorder, major depressive

- disorder, and schizophrenia. *Qual Life Res.* 2019;28(12):3303-3312.
12. Yassa R, Jeste DV. Gender differences in tardive dyskinesia: a critical review of the literature. *Schizophr Bull.* 1992;18(4):701-715.
  13. Vardar MK, et al. Assessment of risk factors for tardive dyskinesia. *Psychiatry Behav Sci.* 2020;10(3):163-170.
  14. Zhang XY, et al. Gender differences in the prevalence, risk and clinical correlates of tardive dyskinesia in Chinese schizophrenia. *Psychopharmacology.* 2009;205(4):647-654.
  15. Solmi M, et al. Clinical risk factors for the development of tardive dyskinesia. *J Neurol Sci.* 2018;389:21-27.
  16. Rajan TM, et al. Frequency and correlates of tardive dyskinesia in Indian patients with bipolar disorder. *Asian J Psychiatr.* 2018;31:48-52.
  17. Kang NR, et al. Tardive dyskinesia: treatment with aripiprazole. *Clin Psychopharmacol Neurosci.* 2011;9(1):1-8.
  18. Carroll B, et al. Health care resource utilization and costs for patients with tardive dyskinesia. *J Manag Care Spec Pharm.* 2019;25(7):810-818.
  19. Achalia RM, et al. Prevalence and risk factors associated with tardive dyskinesia. *Fortune J Health Sci.* 2019.
  20. Carbon M, et al. Tardive dyskinesia risk with first- and second-generation antipsychotics. *World Psychiatry.* 2018;17(3):330-340.
  21. Abnormal Involuntary Movement Scale (AIMS). OHSU, 2019.
  22. Uludag K, et al. Brain imaging studies on tardive dyskinesia in schizophrenia. *Expert Rev Neurother.* 2024.
  23. Cloud LJ, et al. Tardive dyskinesia: therapeutic options for an increasingly common disorder. *Neurotherapeutics.* 2014;11(1):166-176.
  24. Yassa R, et al. Tardive dyskinesia and the primary psychiatric diagnosis. *Psychosomatics.* 1984;25(2):135-138.
  25. Correll CU. Epidemiology, prevention, and assessment of tardive dyskinesia. *J Clin Psychiatry.* 2017.
  26. Tardive dyskinesia: background, pathophysiology, etiology. *Medscape.* 2024.
  27. Prevalence, clinical correlates and risk factors associated with tardive dyskinesia in Chinese patients with schizophrenia. *Nature Communities.* 2025.
  28. van Os J, et al. Tardive dyskinesia in psychosis: are women really more at risk? *Acta Psychiatr Scand.* 1999;100(1):52-58.
  29. Gurevich A, et al. Are atypical antipsychotics safer than typical antipsychotics? *Encephale.* 2012;38(Suppl 2):S40- S46.
  30. Finley P. Atypical antipsychotics and tardive dyskinesia. *J Manag Care Pharm.* 2002;8(4):291-292.
  31. *Psychiatric Times.* Tardive dyskinesia facts and figures. 2020.
  32. Cleveland Clinic. Tardive dyskinesia overview. 2025.
  33. Zhang W, et al. Sex difference in association between tardive dyskinesia and cognitive deficits. *Schizophr Res.* 2023.
  34. NBI-98854 monoamine transport inhibitor study. *Movement Disorders.* 2015.
  35. Bhidayasiri R, et al. Tardive dyskinesia in Asia: current clinical practice. *Front Neurol.* 2024.
  36. Detecting tardive dyskinesia using video-based artificial intelligence. *J Clin Psychiatry.* 2025.