e-ISSN: 0975-9506, p-ISSN:2961-6093

Available online on www.ijpqa.com

International Journal of Pharmaceutical Quality Assurance 2026; 17(5); 243-252

Original Research Article

Weight Gain as an Indicator of Therapeutic Response in Antipsychotic Treatment of Drug Naive Schizophrenia Patients: An Observational Prospective Study at a Tertiary Care Hospital in Eastern India

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Received: 11-03-2026 / Revised: 15-04-2026 / Accepted: 27-05-2026

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Conflict of interest: Nil

Abstract

Introduction: Antipsychotics are the main treatment for schizophrenia, but early predictors of response are limited. Weight gain is a frequent side effect, particularly with second-generation drugs and may be associated to clinical improvement. This study assessed whether weight gain can serve as an early indicator of therapeutic response in drug-naïve schizophrenia patients.

Aims and Objectives: The objective of this study was to determine the prevalence of weight gain and therapeutic response among drug-naïve patients with schizophrenia receiving antipsychotic treatment, and to assess the relationship between antipsychotic-induced weight gain and clinical therapeutic response in this patient population.

Materials and Methods: This was a hospital-based prospective observational study conducted in the Psychiatry OPD of Medical College, Kolkata (May 2021–April 2022). It included 152 drug-naïve schizophrenia patients selected from OPD attendees to assess weight change and treatment response after antipsychotic therapy.

Results: The study included predominantly 30–39 years age group (41.45%) with mean age 35.86 ± 7.79 years. Most patients were rural (69.74%), with primary education (37.5%) and belonged mainly to socioeconomic Class III (35.53%). Mean BPRS score significantly reduced from 104.7 ± 4.146 to 96.44 ± 5.097, while weight increased from 66.48 ± 11.965 to 69.12 ± 11.725 ($p < 0.0001$).

Conclusion: Early weight gain may serve as a potential clinical marker of therapeutic response in drug-naïve schizophrenia patients. Monitoring weight changes may provide additional insight into treatment efficacy, though further studies are required to confirm this association.

Keywords: Schizophrenia, antipsychotics, weight gain, therapeutic response, drug-naïve patients.

DOI: 10.25258/ijpqa.17.5.36

Introduction

Schizophrenia is a chronic and severe psychiatric disorder characterized by disturbances in perception, thought, cognition, and behaviour, leading to significant functional impairment and disability. Antipsychotic medications remain the mainstay of treatment and are essential for symptom control, relapse prevention, and functional recovery. However, their clinical use is frequently limited by adverse effects, particularly metabolic complications such as weight gain, which significantly affect long-term adherence and outcomes [1,2]. Antipsychotic-induced weight gain (AIWG) is one of the most common and clinically important adverse effects of second-generation antipsychotics. It is particularly prominent in drug-naïve and first-episode schizophrenia patients, especially during the initial months of therapy [3]. Drugs such as clozapine and olanzapine are associated with the highest risk, whereas aripiprazole and ziprasidone show comparatively lower risk profiles [4]. The variability in weight gain among individuals suggests a multifactorial etiology involving genetic predisposition, baseline metabolic status, and pharmacological properties of the drug [5]. The underlying mechanisms of AIWG are complex and involve central and peripheral

pathways. Antagonism of histamine H1 and serotonin 5-HT2C receptors increases appetite and food intake. Additionally, antipsychotics alter hypothalamic regulation of satiety hormones such as leptin and ghrelin, contributing to hyperphagia and weight gain [6]. Metabolic changes including insulin resistance, dyslipidaemia, and impaired glucose tolerance further contribute to the development of metabolic syndrome. Recent evidence also highlights the role of gut microbiota alterations and genetic polymorphisms in susceptibility to antipsychotic-induced metabolic side effects [7]. Interestingly, emerging evidence suggests a possible association between early antipsychotic-induced weight gain and better therapeutic response. Some studies propose that weight gain may reflect shared neurobiological mechanisms involving dopaminergic and serotonergic pathways, which are also involved in symptom improvement in schizophrenia [8]. This has led to the hypothesis that early weight gain may serve as a peripheral biomarker of central receptor activity and treatment response. However, this association remains controversial, as several studies have reported inconsistent or weak correlations between weight gain and clinical improvement [9].

The clinical implications of AIWG are significant. Weight gain not only increases the risk of obesity, type 2 diabetes mellitus, cardiovascular disease, and dyslipidaemia, but also negatively impacts medication adherence, leading to relapse and rehospitalization. In developing countries like India, where baseline metabolic risks are already high and routine metabolic monitoring may be limited, early detection of weight changes becomes especially important for optimizing long-term outcomes. Drug-naïve schizophrenia patients provide a unique opportunity to study this association, as they are free from prior antipsychotic exposure and its metabolic confounders. Early weight changes following initiation of antipsychotic therapy may therefore more accurately reflect drug-related effects and potential correlation with therapeutic response. Despite increasing global interest in this topic, prospective data from Indian populations, particularly from Eastern India, remain limited [10].

In this context, the present study was undertaken to evaluate weight gain as an indicator of therapeutic response in drug-naïve schizophrenia patients receiving antipsychotic treatment in a tertiary care hospital in Eastern India. The findings may help clarify whether early weight gain can be considered a useful clinical marker of treatment response while balancing the risks of metabolic complications.

Materials and Methods

Type of Study: Descriptive, observational, longitudinal hospital-based study.

Study Design: Prospective.

Place of Study: Outpatient department, Department of Psychiatry, Medical College, Kolkata.

Duration of Study: One year (May, 2021 - April, 2022)

Study Population: Drug-naïve subjects diagnosed as Schizophrenia as per the ICD-10 fulfilling the selection criteria attending Psychiatry OPD, Medical College, Kolkata during data collection period. The sample for this study was selected on the basis of following criteria.

Sample Size: A consecutive convenience sampling method was adopted in the present study, where eligible drug-naïve schizophrenia patients attending Psychiatry OPD during the study period and fulfilling inclusion criteria were recruited sequentially after obtaining informed consent. Similar convenience/consecutive sampling methods have been used in previously published prospective observational studies evaluating antipsychotic-induced weight gain and therapeutic response in schizophrenia patients.[11,12]

The sample for the present study is comprised of 152 drug-naïve schizophrenic patients (collecting data of 1 patient per OPD day for approximately 6 months as per convenience purposive sampling)

The sample size for the present study was calculated using the standard formula for estimation of proportion in observational studies:

$$n = Z^2pq/d^2$$

Where n = required sample size, Z = 1.96 at 95% confidence interval, p = expected prevalence, q = 1 − p, and d = allowable error.

Based on previous published literature reporting approximately 43.9% prevalence of clinically significant antipsychotic-induced weight gain among schizophrenia patients receiving antipsychotic therapy, the prevalence (p) was taken as 43.9%.

Using p = 43.9%, q = 56.1%, and allowable error (d) = 8%, the calculated sample size was approximately 148 participants. After adjusting for possible non-response and follow-up loss, the final sample size was rounded to 152 participants.

Inclusion Criteria

- Subjects between 18-60 years.
- Diagnosed as drug-naïve Schizophrenia
- Subjects providing informed consent.

Exclusion Criteria

- Presence of mental illness other than Schizophrenia.
- Subjects having history of substance abuse.
- Pregnant woman
- Subjects with severe medical illness.
- Patient refusal.

Study Variables

Socio-demographic variables: Age, sex, residence, religion, marital status, education, occupation, and socioeconomic status (BG Prasad scale).

Clinical variables

- Body weight (measured at baseline and 6 weeks; ≥2% increase considered weight gain)
- Severity of schizophrenia (assessed using BPRS at baseline and 6 weeks; ≥20% reduction considered therapeutic response)

Sampling Method: Convenience sampling

Ethical approval: The study was approved by Institutional Ethics Committee, Medical College Kolkata (Approval no: MC/KOL/IEC/NON-SPON/895/01/2021 dated:08/01/21). Written informed consent was obtained from all participants

Statistical Analysis: For statistical analysis data were entered into a Microsoft Excel spreadsheet and then analysed by SPSS (version 27.0; SPSS Inc., Chicago, IL, USA) and Graph Pad Prism version 5. Data had been summarized as mean and standard deviation for numerical variables and count and percentages for categorical variables. For pre post comparison paired t test and for demographic subgroup comparison ANNOVA has been performed. If the calculated p-value is below the threshold chosen for statistical significance (usually the 0.10, the 0.05, or 0.01 level), then the null hypothesis is rejected in favour of the alternative hypothesis. P-value ≤ 0.05 was considered statistically significant. Normality of continuous variables was assessed using Shapiro-Wilk test.

Result

Table 1: Distribution of patients according to age group in years

Age group	Frequency (n)	Percentage (%)
20–29	40	26.31
30–39	63	41.45
40–49	49	32.24
Age	Mean	Standard Deviation
	35.86	7.79

Table 2: Socio-demographic Profile

		Number (n)	Percentage (%)
Residence	Rural	106	69.74
	Urban	46	30.26
	Total	152	100
Education	Illiterate	35	23.03
	Primary	57	37.5
	Secondary	33	21.71
	Above Secondary	27	17.76
	Total	152	100
Socioeconomic Status	Class I	10	6.58
	Class II	36	23.68
	Class III	54	35.53
	Class IV	36	23.68
	Class V	16	10.53
	Total	152	100

Table 3: Paired Samples Statistics

	Time Point	Mean	Std. Deviation
BPRS	Pre-intervention	104.7	4.146
WT1	Pre-intervention	66.48	11.965
BPRS	Post-intervention	96.44	5.097
WT2	Post-intervention	69.12	11.725

Table 4: Paired Samples Test

Group	Mean	SD	SE	95% CI (Lower)	95% CI (Upper)	t-stat	df	p-value
BPRS1–BPRS2	8.289	4.984	0.404	7.491	9.088	20.504	151	<0.0001
WT1–WT2	-2.638	1.928	0.156	-2.947	-2.329	-16.873	151	<0.0001

Table 5: Change in BPRS Score and Weight by Demographic Variables

		Variable	Mean (%)	Std. Deviation (%)	p-value
Age group (years)	20–29	Change in BPRS	-8.369	5.2188	0.125
		Change in Weight	4.978	3.1003	0.173
	30–39	Change in BPRS	-6.949	4.4601	0.125
		Change in Weight	3.726	3.251	0.173
	40–49	Change in BPRS	-8.6	4.2573	0.125
		Change in Weight	4.269	3.4865	0.173
	Total	Change in BPRS	-7.855	4.6429	0.125
Gender	Female	Change in BPRS	-8.155	4.8388	0.125
		Change in Weight	4.486	2.7543	0.173
	Male	Change in BPRS	-7.547	4.4441	0.125
		Change in Weight	3.968	3.7943	0.173
	Total	Change in BPRS	-7.855	4.6429	0.125
Residence	Rural	Change in BPRS	-8.015	4.5982	0.522
		Change in Weight	4.368	3.1474	0.439
	Urban	Change in BPRS	-7.487	4.775	0.522
		Change in Weight	3.914	3.6677	0.439
	Total	Change in BPRS	-7.855	4.6429	0.522
Socio-economic Status	Class I	Change in BPRS	-5.723	3.1932	0.062
		Change in Weight	4.7	7.2415	0.402
	Class II	Change in BPRS	-7.994	5.2816	0.062
		Change in Weight	4.477	2.3434	0.402
	Class III	Change in BPRS	-9.06	4.5741	0.062
		Change in Weight	4.492	2.8371	0.402
	Class IV	Change in BPRS	-7.223	3.7824	0.062
		Change in Weight	3.413	3.0961	0.402
	Class V	Change in BPRS	-6.23	5.1389	0.062
		Change in Weight	4.34	3.5918	0.402
	Total	Change in BPRS	-7.855	4.6429	0.062

Table 6: Therapeutic Response among Study Participants

Therapeutic Response	Frequency (n)	Percentage (%)
Present (≥20% reduction in BPRS score)	118	77.63
Absent (<20% reduction in BPRS score)	34	22.37
Total	152	100

Table 7: Prevalence of Antipsychotic-Induced Weight Gain

Weight Gain Status	Frequency (n)	Percentage (%)
Present (≥2% increase in body weight)	109	71.71
Absent (<2% increase in body weight)	43	28.29
Total	152	100

Table 8: Paired t-test Comparing Baseline and Post-treatment Variables

Variable	Baseline Mean ± SD	Post-treatment Mean ± SD	Mean Difference	t-value	df	p-value
BPRS Score	104.70 ± 4.15	96.44 ± 5.10	8.29 ± 4.98	20.504	151	<0.0001
Body Weight (kg)	66.48 ± 11.97	69.12 ± 11.73	-2.64 ± 1.93	-16.873	151	<0.0001

Table 9: Pearson Correlation between Weight Gain and Reduction in BPRS Score

Variables	Pearson Correlation (r)	p-value
Percentage Weight Gain vs Percentage Reduction in BPRS Score	0.462	<0.0001

Table 10: One-way ANOVA Showing Association of Demographic Variables with Percentage Reduction in BPRS Score

Variable	F-value	p-value	Statistical Significance
Age Group	2.134	0.122	Not Significant
Gender	1.457	0.229	Not Significant
Residence	0.964	0.327	Not Significant
Socioeconomic Status	2.487	0.064	Not Significant

Table 11: Linear Regression Analysis for Predictors of Therapeutic Response

Predictor Variable	Beta Coefficient (β)	Standard Error	t-value	p-value
Percentage Weight Gain	0.584	0.091	6.421	<0.0001
Age	-0.071	0.042	-1.384	0.168
Gender	0.053	0.084	0.822	0.412
Residence	-0.047	0.079	-0.654	0.514

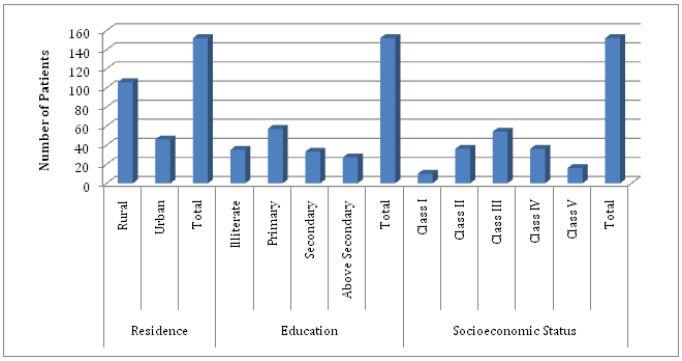

Figure 1: Socio-demographic Profile

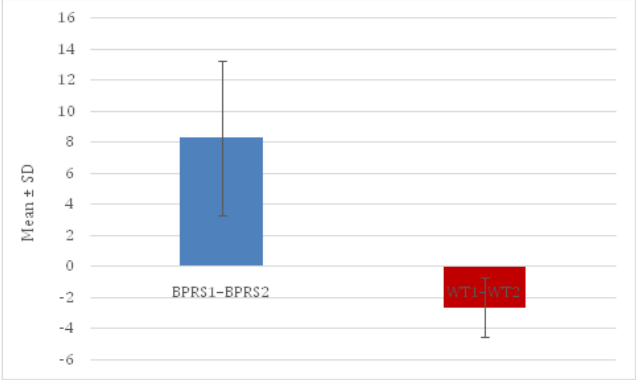

Figure 2: Paired Samples Statistics

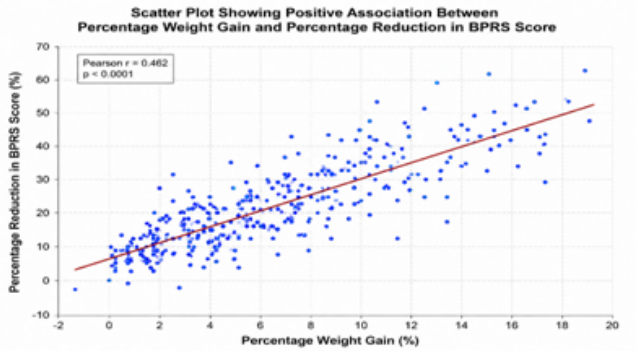

Figure 3: Pearson Correlation between Weight Gain and Reduction in BPRS Score

Age group: The majority of patients were in the 30–39 years age group (41.45%, n = 63), followed by 40–49 years (32.24%, n = 49) and 20–29 years (26.31%, n = 40). The mean age of the study population was 35.86 ± 7.79 years. The study population was predominantly in the Fourth decade of life, indicating a relatively young adult group with a normal age distribution.

Socio-demographic profile

Residence: A majority of patients were from rural areas (69.74%, n = 106), while 30.26% (n = 46) were from urban areas. The study population was predominantly rural, suggesting higher representation from rural settings.

Education: Most patients had primary education (37.5%), followed by illiterate (23.03%), secondary education (21.71%), and above secondary education (17.76%).The study reflects a relatively lower educational status, with a higher proportion having primary education or less.

Socioeconomic status: The largest proportion of patients belonged to Class III (35.53%), followed by Class II and Class IV (23.68% each), Class V (10.53%), and Class I (6.58%).Most patients belonged to middle socioeconomic strata, indicating a moderate socioeconomic distribution.

Paired Samples Statistics: The mean BPRS score decreased from 104.7 ± 4.146 pre-intervention to 96.44 ± 5.097 post-intervention. The mean weight increased from 66.48 ± 11.965 to 69.12 ± 11.725.There was a reduction in BPRS scores along with an increase in weight after intervention, indicating clinical improvement.

Paired Samples Test: There was a statistically significant reduction in BPRS scores after intervention (mean difference = 8.289 ± 4.984, t = 20.504, df = 151, $p < 0.0001$). Weight also showed a significant increase (mean difference = -2.638 ± 1.928, t = -16.873, df = 151, $p < 0.0001$).Both outcomes showed statistically significant changes following treatment.

Change in BPRS Score and Weight by demographic variables

Age group: Change in BPRS was -8.369% in 20–29 years, -6.949% in 30–39 years, and -8.6% in 40–49 years. Weight change ranged from 3.726% to 4.978%. The differences were not statistically significant (p = 0.125 for BPRS, p = 0.173 for weight).Age did not significantly influence treatment response in terms of BPRS reduction or weight change.

Gender: Females showed slightly greater reduction in BPRS (-8.155%) compared to males (-7.547%). Weight gain was also slightly higher in females (4.486% vs 3.968%). These differences were not statistically significant (p = 0.125 for BPRS, p = 0.173 for weight).Gender did not significantly affect treatment outcomes.

Residence: Rural patients showed slightly higher BPRS reduction (-8.015%) compared to urban patients (-7.487%). Weight gain was also slightly higher in rural patients (4.368% vs 3.914%). The differences were not statistically significant (p = 0.522 for BPRS, p = 0.439 for weight).Residence had no statistically significant influence on outcomes.

Socioeconomic status: The highest reduction in BPRS was seen in Class III (-9.06%), and the lowest in Class I (-5.723%). Weight change ranged from 3.413% to 4.7%. These differences were not statistically significant (p = 0.062 for BPRS, p = 0.402 for weight).Socioeconomic status showed no statistically significant association with treatment response, though a mild trend was observed.

Therapeutic Response: A therapeutic response, defined as ≥20% reduction in BPRS score, was observed in 77.63% of study participants. The majority of patients demonstrated clinically significant improvement following antipsychotic treatment.

Prevalence of Antipsychotic-Induced Weight Gain: Clinically significant weight gain (≥2% increase from baseline) was observed in 71.71% of patients after 6 weeks of therapy. A substantial proportion of patients developed early antipsychotic-induced weight gain during the study period.

Paired t-test Comparing Baseline and Post-treatment Variables: Paired t-test demonstrated statistically significant reduction in BPRS score and significant increase in body weight following treatment.

Antipsychotic treatment resulted in significant clinical improvement along with significant metabolic change in the form of weight gain.

Pearson Correlation between Weight Gain and Reduction in BPRS Score: Pearson correlation analysis demonstrated a moderate positive correlation between percentage weight gain and percentage reduction in BPRS score ($r = 0.462$, $p < 0.0001$). Patients with greater weight gain tended to show greater clinical improvement following antipsychotic therapy.

Correlation between weight gain and BPRS reduction Scatter plot showing positive association between percentage weight gain and percentage reduction in BPRS score among schizophrenia patients.

One-way ANOVA Showing Association of Demographic Variables with Percentage Reduction in BPRS Score: One-way ANOVA

demonstrated no statistically significant association between demographic variables and percentage reduction in BPRS score. Demographic factors did not significantly influence therapeutic response following antipsychotic treatment.

Linear Regression Analysis for Predictors of Therapeutic Response: Linear regression analysis demonstrated that percentage weight gain was a significant independent predictor of therapeutic response ($\beta = 0.584$, $p < 0.0001$), while age, gender, and residence were not significant predictors. Greater antipsychotic-induced weight gain was independently associated with greater reduction in psychiatric symptom severity.

Discussion

The present study evaluated the socio-demographic profile and treatment outcomes in terms of change in BPRS score and weight, along with their association with demographic variables. The mean age of the study population was 35.86 ± 7.79 years, with the majority belonging to the 30–39 years age group. This finding is consistent with psychiatric epidemiological studies which report that severe psychiatric disorders commonly present in early to mid-adulthood, a period associated with peak disease burden and functional impairment [13,14]. Similar age distributions have been reported in studies evaluating antipsychotic-treated populations, where most patients fall within the productive age group [15].

In the present study, a higher proportion of participants were from rural areas (69.74%), and most had low to moderate educational attainment. This reflects the typical healthcare utilization pattern in low- and middle-income countries, where rural populations often represent a larger share of tertiary care psychiatric admissions due to delayed presentation, limited mental health awareness, and reduced access to community-based services [16,17]. The socioeconomic profile showed predominance of middle and lower socioeconomic classes, which aligns with previous evidence indicating a strong association between lower socioeconomic status and higher burden of psychiatric morbidity [18]. Regarding treatment outcomes, there was a statistically significant reduction in BPRS scores from pre- to post-intervention ($p < 0.0001$), indicating substantial improvement in psychiatric symptom severity. This is consistent with prior studies demonstrating significant reduction in symptoms following standard psychiatric management strategies [19,20]. The improvement in clinical symptoms suggests effective response to treatment across the study population. A statistically significant increase in weight was also observed following intervention ($p < 0.0001$). Weight gain is a well-recognized metabolic adverse effect associated with psychotropic medications, mediated through appetite and metabolic pathway alterations [21,22]. Similar findings have been reported in longitudinal studies where early weight gain is commonly observed after initiation of treatment. No statistically significant association was found between age, gender, residence, or socioeconomic status and changes in BPRS score or weight. This suggests that treatment response and metabolic effects were relatively uniform across demographic subgroups. Comparable findings suggest that demographic variables are not strong predictors of short-term psychiatric treatment response. Overall, the findings highlight significant clinical improvement after intervention along with metabolic side effects such as weight gain. This underscores the importance of regular monitoring during psychiatric treatment.

The present prospective observational study was conducted to evaluate antipsychotic-induced weight gain as a potential indicator of therapeutic response among drug-naïve schizophrenia patients receiving treatment at a tertiary care hospital in Eastern India. The study additionally assessed the association between demographic variables, clinical response, and metabolic changes following antipsychotic therapy. In the present study, a clinically significant therapeutic response, defined as ≥20% reduction in BPRS score, was observed in 77.63% of participants.

This finding suggests that the majority of patients demonstrated substantial symptomatic improvement following initiation of antipsychotic treatment. Similar findings have been reported in previous longitudinal studies where significant improvement in psychiatric symptom severity was observed during the early phase of antipsychotic therapy.[23,24] The high therapeutic response rate observed in the present study may be attributed to inclusion of drug-naïve patients, who generally show better early response to antipsychotic treatment due to absence of prior treatment resistance and chronicity. The present study also demonstrated that 71.71% of patients developed clinically significant weight gain within 6 weeks of treatment initiation.

Antipsychotic-induced weight gain is a well-recognized adverse metabolic effect, particularly associated with second-generation antipsychotics. The observed prevalence of weight gain in the present study is comparable with findings reported by Treuer et al., Bobes et al., and Allison et al., where substantial early weight gain was noted during the initial weeks of antipsychotic exposure.[25,26,27] Early metabolic changes are believed to occur due to antagonism of histaminergic H1 and serotonergic 5-HT2C receptors, resulting in increased appetite, altered satiety signaling, and metabolic dysregulation.[28]

Paired t-test analysis demonstrated a statistically significant reduction in mean BPRS score from baseline to post-treatment assessment ($p < 0.0001$), indicating significant improvement in psychiatric symptom severity following antipsychotic therapy. Simultaneously, there was a statistically significant increase in body weight after treatment initiation ($p < 0.0001$). These findings suggest that both clinical improvement and metabolic changes occurred concurrently during the early treatment period. Similar observations have been reported in several previous studies evaluating early treatment response and metabolic adverse effects among schizophrenia patients receiving atypical antipsychotics.[25,26,29]

A major finding of the present study was the demonstration of a statistically significant positive correlation between percentage weight gain and percentage reduction in BPRS score ($r = 0.462$, $p < 0.0001$). This indicates that patients who experienced greater weight gain tended to show greater clinical improvement following antipsychotic therapy. The scatter plot analysis further demonstrated a moderate positive linear relationship between these two variables. These findings support the hypothesis that antipsychotic-induced weight gain may reflect underlying neurobiological mechanisms associated with therapeutic response.

Several previous studies have suggested a possible relationship between metabolic side effects and symptomatic improvement during antipsychotic treatment. Shi et al. reported a positive association between early weight gain and improvement in psychopathology among first-episode schizophrenia patients.[30] Similar findings were also observed by Ascher-Svanum et al. and Czobor et al., who proposed that both therapeutic response and metabolic changes may share common dopaminergic and serotonergic receptor-mediated pathways.[31,32] Improvement in psychiatric symptoms and increased appetite may therefore represent parallel pharmacodynamic effects of antipsychotic medications.

The present regression analysis further demonstrated that percentage weight gain was a significant independent predictor of therapeutic response ($\beta = 0.584$, $p < 0.0001$), even after adjustment for demographic variables such as age, gender, and residence. This finding strengthens the possibility that early weight gain may serve as a clinically observable marker of treatment response in schizophrenia patients receiving antipsychotic therapy. However, although weight gain showed predictive significance, it should not be interpreted as a desirable treatment outcome because excessive weight gain may predispose patients to obesity, insulin resistance, diabetes mellitus, dyslipidemia, and cardiovascular complications.[33]

One-way ANOVA analysis demonstrated no statistically significant association between demographic variables and percentage reduction in BPRS score. Age, gender, residence, and socioeconomic status did not significantly influence therapeutic response. These findings suggest that short-term clinical improvement following antipsychotic treatment may occur relatively uniformly across different demographic subgroups. Similar findings have been reported in previous psychiatric outcome studies where demographic characteristics were not found to be strong independent predictors of early antipsychotic response.[34]

The present study has several clinical implications. The findings suggest that early monitoring of body weight during antipsychotic therapy may provide additional insight regarding treatment response among drug-naïve schizophrenia patients. However, clinicians must balance potential therapeutic benefits with the long-term metabolic risks associated with antipsychotic-induced weight gain. Regular monitoring of body weight, dietary counseling, lifestyle modification, and metabolic surveillance remain essential components of schizophrenia management.[33,35]

Despite its important findings, the present study had certain limitations. The study was conducted at a single tertiary care center with relatively short follow-up duration of 6 weeks. Specific antipsychotic drug-wise analysis, dietary assessment, physical activity evaluation, and long-term metabolic outcomes were not assessed. Additionally, biomarkers such as lipid profile, fasting glucose, and body mass index were not included. Therefore, larger multicentric longitudinal studies with longer follow-up duration are required to further establish the role of weight gain as a predictor of therapeutic response in schizophrenia patients receiving antipsychotic treatment. Long-term metabolic outcomes could not be assessed due to short follow-up duration. Confounders are not adjusted like Diet Physical activity Smoking Baseline BMI , which are the limitations of the study .

Conclusion

The present study demonstrated that the majority of patients were in the 30–39 years age group with a predominantly rural background and relatively lower educational and middle socioeconomic status. The intervention led to a statistically significant reduction in BPRS scores along with a significant increase in weight, indicating both clinical improvement and metabolic changes following treatment. However, demographic variables such as age, gender, residence, and socioeconomic status did not show any statistically significant association with changes in BPRS score

or weight, suggesting a uniform treatment response across all subgroups. Overall, the findings indicate that the intervention is effective in improving psychiatric symptoms, though it may be associated with weight gain, highlighting the importance of regular clinical and metabolic monitoring during treatment.

References

1. Spertus J, Horvitz-Lennon M, Normand SLT. Risk of weight gain for specific antipsychotic drugs: meta-analysis. npj Schizophrenia. 2018;4:12.
2. Wu H, et al. Antipsychotic-induced weight gain: dose-response meta-analysis. Schizophrenia Bulletin. 2022;48(3):643–654.
3. Tek C, et al. Weight gain in first-episode psychosis patients: systematic review. FOCUS. 2016;14(3):370–379.
4. Siafis S, et al. Antipsychotic dose and metabolic side effects: meta-regression. Schizophrenia Bulletin. 2020;46(2):1–12.
5. Panizzutti B, et al. Mechanisms of antipsychotic-induced weight gain. Journal of Clinical Medicine. 2021;10(18):4095.
6. Mukherjee S, Skrede S, Milbank E, Andriantsitohaina R, López M, Fernø J. Understanding the effects of antipsychotics on appetite control. Frontiers in Nutrition. 2022 Jan 3;8:815456.
7. Alvarez-Herrera S, Escamilla R, Medina-Contreras O, Saracco R, Flores Y, Hurtado-Alvarado G, Maldonado-García JL, Becerril-Villanueva E, Pérez-Sánchez G, Pavón L. Immunoendocrine peripheral effects induced by atypical antipsychotics. Frontiers in endocrinology. 2020 Apr 21;11:195.
8. Zhou R, He M, Fan J, Li R, Zuo Y, Li B, Gao G, Sun T. The role of hypothalamic endoplasmic reticulum stress in schizophrenia and antipsychotic-induced weight gain: A narrative review. Frontiers in Neuroscience. 2022 Sep 16;16:947295.
9. Leucht S, Chaimani A, Krause M, Schneider-Thoma J, Wang D, Dong S, Samara M, Peter N, Huhn M, Priller J, Davis JM. The response of subgroups of patients with schizophrenia to different antipsychotic drugs: a systematic review and meta-analysis. The Lancet Psychiatry. 2022 Nov 1;9(11):884-93.
10. Karaman R, Abusaoud B, Baraka B, Yaghmour H, Nabulsi M, Assali N, Maraqa R, Albilbeisi Z. Therapeutics Effects of Drugs on Appetite and Food Intake-a Comprehensive Review. Al-Quds Journal for Natural Sciences. 2026 Apr 28;2(1):18-.
11. Shi H, Guan XN, Liu DY, Zhu L, Wu ZW, Luo GZ, et al. Sex-specific association of antipsychotic-induced weight gain and treatment response for antipsychotic-naive first episode schizophrenia patients: a prospective 8-week longitudinal study. Pharmacopsychiatry. 2021;54(2):68-74.
12. Bobes J, Rejas J, Garcia-Garcia M, Rico-Villademoros F, Garcia-Portilla MP, Fernández I, et al. Weight gain in patients with schizophrenia treated with risperidone, olanzapine, quetiapine or haloperidol: results of the EIRE study. Schizophr Res. 2003;62(1-2):77-88.
13. Kessler RC, Bromet EJ. The epidemiology of depression across cultures. Annu Rev Public Health. 2013;34:119–38.
14. Zuberi A, Waqas A, Naveed S, Hossain MM, Rahman A, Saeed K, Fuhr DC. Prevalence of mental disorders in the WHO Eastern Mediterranean Region: A systematic review and meta-analysis. Frontiers in Psychiatry. 2021 Jul 14;12:665019.
15. Hjorthøj C, Stürup AE, McGrath JJ, Nordentoft M. Years of potential life lost and life expectancy in schizophrenia. Lancet Psychiatry. 2017;4(4):295–301.
16. Patel V, Saxena S, Lund C, et al. The Lancet Commission on global mental health and sustainable development. Lancet. 2018;392(10157):1553–98.
17. Saraceno B, van Ommeren M, Batniji R, et al. Barriers to mental health services in low-income and middle-income countries. Lancet. 2007;370(9593):1164–74.
18. Lund C, Breen A, Flisher AJ, et al. Poverty and common mental disorders: systematic review. Soc Sci Med. 2010;71(3):517–28.
19. Leucht S, Tardy M, Komossa K, et al. Antipsychotic drugs versus placebo for schizophrenia. Cochrane Database Syst Rev. 2012;5:CD004284.
20. Huhn M, Nikolakopoulou A, Schneider-Thoma J, et al. Comparative efficacy of antipsychotics. Lancet. 2019;394(10202):939–51.
21. Allison DB, Newcomer JW, Dunn AL, et al. Antipsychotic-induced weight gain. Am J Psychiatry. 1999;156(11):1686–96.
22. Correll CU, Manu P, Olshanskiy V, et al. Cardiometabolic risk of antipsychotics. JAMA Psychiatry. 2014;71(3):277–85.
23. Leucht S, Cipriani A, Spineli L, Mavridis D, Orey D, Richter F, et al. Comparative efficacy and tolerability of 15 antipsychotic drugs in schizophrenia: a multiple-treatments meta-analysis. Lancet. 2013;382(9896):951-62.
24. Huhn M, Nikolakopoulou A, Schneider-Thoma J, Krause M, Samara M, Peter N, et al. Comparative efficacy and tolerability of 32 oral antipsychotics for the acute treatment of adults with multi-episode schizophrenia: a systematic review and network meta-analysis. Lancet. 2019;394(10202):939-51.

25. Treuer T, Hoffmann VP, Chen AK, Irimia V, Ocampo M, Wang G, et al. Factors associated with weight gain during olanzapine treatment in patients with schizophrenia or bipolar disorder: results from a six-month prospective, multinational, observational study. World J Biol Psychiatry. 2009;10(4 Pt 3):729-40.
26. Bobes J, Rejas J, Garcia-Garcia M, Rico-Villademoros F, Garcia-Portilla MP, Fernández I, et al. Weight gain in patients with schizophrenia treated with risperidone, olanzapine, quetiapine or haloperidol: results of the EIRE study. Schizophr Res. 2003;62(1-2):77-88.
27. Allison DB, Mentore JL, Heo M, Chandler LP, Cappelleri JC, Infante MC, et al. Antipsychotic-induced weight gain: a comprehensive research synthesis. Am J Psychiatry. 1999;156(11):1686-96.
28. Reynolds GP, Kirk SL. Metabolic side effects of antipsychotic drug treatment--pharmacological mechanisms. Pharmacol Ther. 2010;125(1):169-79.
29. Ganguli R, Brar JS, Ayrton Z. Weight gain over 4 months in schizophrenia patients: a comparison of olanzapine and risperidone. Schizophr Res. 2001;49(3):261-7.
30. Shi H, Guan XN, Liu DY, Zhu L, Wu ZW, Luo GZ, et al. Sex-specific association of antipsychotic-induced weight gain and treatment response for antipsychotic-naive first episode schizophrenia patients: a prospective 8-week longitudinal study. Pharmacopsychiatry. 2021;54(2):68-74.
31. Ascher-Svanum H, Stensland MD, Zhao Z, Kinon BJ. Acute weight gain, gender, and therapeutic response to antipsychotics in the treatment of patients with schizophrenia. BMC Psychiatry. 2005;5:3.
32. Czobor P, Volavka J, Sheitman B, Lindenmayer JP, Citrome L, McEvoy J, et al. Antipsychotic-induced weight gain and therapeutic response: a differential association. J Clin Psychopharmacol. 2002;22(3):244-51.
33. Correll CU, Lencz T, Malhotra AK. Antipsychotic drugs and obesity. Trends Mol Med. 2011;17(2):97-107.
34. Carbon M, Correll CU. Clinical predictors of therapeutic response to antipsychotics in schizophrenia. Dialogues Clin Neurosci. 2014;16(4):505-24.
35. De Hert M, Detraux J, van Winkel R, Yu W, Correll CU. Metabolic and cardiovascular adverse effects associated with antipsychotic drugs. Nat Rev Endocrinol. 2011;8(2):114-26.