Article

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Evaluation of Clinical Correlation between Insulin Resistance and Antipsychotic Drug Therapy in Patients with Schizophrenia

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Abstract

Background: Treatment with different antipsychotics can lead to various metabolic side effects in patients with psychosis, impacting long-term prognosis. This study aimed to compare the changes and clinical efficacy of insulin resistance in patients treated with olanzapine and ziprasidone.

Method: A retrospective analysis was conducted on the clinical data of 80 patients with schizophrenia. The patients were divided into olanzapine treatment group and ziprasidone treatment group. Parameters including body weight, body mass index (BMI), fasting plasma glucose (FPG), fasting plasma insulin (FPI), cholesterol (CHO), triglyceride (TG), high-density lipoprotein (HDL), low-density lipoprotein (LDL), insulin resistance index, and Positive and Negative Syndrome Scale (PANSS) scores were recorded and compared before and after treatment.

Results: BMI, FPG, FPI, homeostatic model assessment of insulin resistance (HOMA-IR), CHO, TG and LDL in both groups were significantly higher than before treatment ($p < 0.05$). These parameters were significantly higher in the olanzapine group than in the ziprasidone group ($p < 0.05$). The level of HDL in both groups was significantly decreased after treatment, and the level of HDL in the olanzapine group was significantly lower than that in the ziprasidone group after treatment ($p < 0.05$). After treatment, the total score and score of PANSS in both groups were significantly lower than before treatment ($p < 0.05$). After treatment, there was no significant difference in total score and PANSS score between both groups ($p > 0.05$). The incidence of insulin resistance (IR) was significantly higher in the olanzapine group compared to the ziprasidone group ($\chi^2 = 4.021$, $p < 0.05$). In the IR group, BMI, FPG, FPI, TG, and LDL levels were higher than in the non-IR group ($p < 0.05$). Multivariate analysis indicated that BMI, FPG, FPI, TG, and LDL were independent risk factors for IR (odd ratio (OR) >1, $p < 0.05$).

Conclusions: Treatment with olanzapine and ziprasidone improves clinical symptoms in patients with schizophrenia, but increases the risk of insulin resistance. The metabolic side effects of olanzapine are more pronounced.

Keywords

schizophrenia; antipsychotics; insulin resistance

Introduction

Schizophrenia is a common mental illness characterized by disturbances in thinking, emotions, and behavior, marked by a dissonance between cognitive processes and the environment [1]. It is a devastating developmental and chronic disease that typically begins in late adolescence or early adulthood [2]. This complex disorder arises from gene-environment interactions [3]. Non-genetic factors, including substance abuse, parasitic infections, and stress, are significant risk factors for the onset of schizophrenia in young people [4]. The disease affects approximately 1% of the population in most countries and impacts both men and women. Children of parents with schizophrenia are at a higher risk of developing the disorder, likely due to a combination of genetic and epigenetic mechanisms, as well as environmental risk factors [5].

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The diagnostic criteria for schizophrenia encompass three primary symptom categories: positive symptoms, negative symptoms, and cognitive impairments. Positive symptoms include hallucinations, delusions, and disordered thought processes, often leading to aggressive and dangerous behavior. Many patients also suffer from enduring negative symptoms (deficits), such as language difficulties, social withdrawal, emotional flatness, and anhedonia. Cognitive dysfunction, which affects language, working memory, information processing speed, reasoning, and attention, is another prominent feature of the disorder. Approximately 40% of patients experience adverse symptoms, while around 80% exhibit significant cognitive impairment [6]. Schizophrenia profoundly impacts cognitive abilities, daily functioning, and social interactions, posing numerous challenges and burdens for families, communities, and society at large [1].

Antipsychotics are crucial in the treatment of schizophrenia, aiming to reduce the severity and frequency of symptoms. Consistent, long-term use of antipsychotics is vital for achieving and sustaining symptom management. However, the discontinuation rate of oral antipsychotics is estimated to be between 26% and 44%, with up to two-thirds of patients failing to adhere to their prescribed regimen [7]. Interruptions in treatment adherence are linked to an elevated risk of symptom recurrence and hospitalization. Psychosis relapse is profoundly distressing for patients and their families, with numerous ramifications for disease progression and brain structure [8], including progressive deterioration in the structure and volume of gray and white matter. Relapse may also lead to diminished responsiveness to previously effective antipsychotics, potentially leading to treatment resistance [9]. Despite their efficacy, antipsychotic drugs often have adverse effects. It is essential to monitor the immune response of the patient, the activation of inflammatory pathways, and self-regulatory mechanisms. Therefore, enhancing health outcomes for these patients, selecting appropriate antipsychotic medication, and mitigating the adverse effects of treatment and related factors pose considerable challenges [10].

First-generation antipsychotic drugs (FGAs) revolutionized the treatment of severe mental illnesses, making it possible to stabilize conditions that previously required prolonged hospitalization. Although FGAs transformed psychiatric practice, they are associated with severe adverse reactions related to physical exercise [11]. Second-generation antipsychotic drugs (SGAs) have become more commonly used clinically due to their efficacy in treating the negative symptoms of schizophrenia [12]. Compared to FGAs, SGAs quickly became the preferred choice due to their reduced exercise-related side effects and improved efficacy in addressing negative symptoms [11]. However, long-term SGA therapy is frequently accompanied by metabolic adverse effects, such as weight gain, insulin resistance, and the development of type 2 diabetes mellitus (T2DM), which significantly impact the quality of life of patients [12]. In recent years, as research has delved deeper into the efficacy of SGAs for treating schizophrenia, there has been a growing scholarly focus on the adverse reactions associated with these medications.

Olanzapine, one of the most commonly utilized SGAs, is an effective treatment for schizophrenia but is known to cause severe metabolic side effects, including weight gain, insulin resistance, and T2DM. These effects have become a significant public health concern [13,14]. Olanzapine is one of the primary SGAs currently sold in China but has recently come under increasing scrutiny [15]. Newer SGAs, such as ziprasidone, exhibit more favorable metabolic profiles but demonstrate inferior overall clinical efficacy compared to olanzapine [16]. Abnormal glucose metabolism and lipid metabolism are closely related to schizophrenia. By studying the correlation between antipsychotic drugs and alterations in insulin resistance among individuals with schizophrenia, along with their clinical impact, this paper aimed to discuss the clinical significance of relevant indicators and provide valuable insights for evaluating the effectiveness of psychiatric treatments.

Materials and Methods

Participants

The clinical data of 80 patients with schizophrenia who received standard medication at Wuyi County First People's Hospitalfrom June 2022 to May 2023 were retrospectively analyzed. Forty patients were treated with olanzapine (15–20 mg/day) for 12 weeks. Additional 40 patients who had been taking ziprasidone (initial dose: 20 mg/time, 3 times/day, adjusted within one week to 60 mg/time, twice/day) for a long time were selected as the control group.

Inclusion criteria: (1) Diagnosis of schizophrenia according to the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5); (2) No history of allergy to the tested drugs; (3) No diagnosis of diabetes or use of antidiabetic drugs, with a stable atypical antipsychotic regimen for at least 6 months. Exclusion criteria: (1) Concurrent serious epilepsy or other diseases; (2) Severe heart, lung, or other organ diseases; (3) Severe blood diseases. The research protocol received approval from Wuyi County

First People's Hospital Ethics Committee (Approval No. 2024516) and adhered to the principles of the Declaration of Helsinki. All participants provided informed consent after receiving a complete description of the study.

Detection Index

Blood samples were obtained from all patients before treatment initiation and after 12 weeks of consistent medication. All participants fasted for a minimum of 6 hours before blood collection. Samples were refrigerated immediately and underwent serum/plasma separation within 30 minutes, then frozen at –80 °C for subsequent batch processing.

Blood Samples

Blood samples were taken, typically in a fasting state, between 7–8 a.m., approximately 12 hours after the last olanzapine administration. Patients avoided greasy foods the day before blood sampling. Five milliliters of venous blood samples were collected from inpatients at 6:00 a.m., following an overnight fast (avoided food for >10 hours), and stored at –40 °C. An automatic biochemical analyzer (Beckman AU5800, Beckman Coulter, Brea, CA, USA) was used to measure fasting plasma glucose (FPG) (ml095386, Shanghai Enzyme-linked Biotechnology Co., Ltd., Shanghai, China), fasting plasma insulin (FPI) (ml060484, Shanghai Enzyme-linked Biotechnology Co., Ltd., Shanghai, China), cholesterol (CHO) (TR13421, Thermo Fisher Scientific, Waltham, MA, USA), triglyceride (TG) (EEA028, Invitrogen, Carlsbad, CA, USA), high-density lipoprotein (HDL) (ml092768, Shanghai Enzyme-linked Biotechnology Co., Ltd., Shanghai, China), and low-density lipoprotein (LDL) (L3486, Invitrogen, Carlsbad, CA, USA) levels.

Body Mass Index (BMI) Calculation

BMI is determined by dividing the weight of an individual (in kilograms) by the height (in square meters).

Homeostatic Model Assessment of Insulin Resistance (HOMA-IR) Calculation

The HOMA index was used to evaluate insulin resistance by calculating the values of insulin and glucose concentration. HOMA-IR = (FPG (mmol/L) × FPI (mIU/L))/22.5. Based on the Metabolic Syndrome components, the threshold value of HOMA-IR was set at 1.7 [17]. Patients with HOMA-IR values lower than 1.7 were assigned to the non-IR group, while those with HOMA-IR values >1.7 were assigned to the IR group.

Clinical Assessments

All participants were evaluated for clinical symptoms of schizophrenia using the Positive and Negative Syndrome Scale (PANSS) [18]. These symptoms comprise of seven positive symptoms, seven negative symptoms, 16 general pathological symptoms, and three additional symptoms, totaling 33 items. Each item is scored on a scale of 1–7, with a higher total score indicating greater severity of symptoms. The last three additional symptoms were not included in the total score. The PANSS provides a comprehensive assessment of psychiatric symptoms and reflects the overall clinical phase of patients.

Statistical Analysis

Statistical analyses were conducted using SPSS 23.0 (IBM, Armonk, NY, USA). Quantitative data meeting normal distribution and homogeneity of variance were expressed as mean ± standard deviation ($\bar{x} \pm s$). Independent sample *t*-tests were used for inter-group analysis of continuous variables and Chi-square or Fisher exact tests for categorical variables. The prevalence of IR was described using percentages and analyzed using the Chi-square test. Multiple logistic regression analysis was performed to evaluate the risk factors of IR. All *p*-values were two-tailed, with $p < 0.05$ indicating statistical significance.

Results

Changes in Related Indexes before and after Treatment

The study comprised a total of 80 subjects, with 40 patients receiving long-term olanzapine treatment for schizophrenia and 40 patients receiving ziprasidone. Table 1 presents the baseline characteristics of the subjects. There were no statistically significant differences in age, gender, weight, BMI, FPG, FPI, HOMA-IR, CHO, TG, HDL, or LDL between the two groups ($p > 0.05$). After treatment, the two groups had no significant difference in body weight ($p > 0.05$). Both groups showed significant increases in BMI, FPG, FPI, HOMA-IR, CHO, TG, and LDL levels compared to before treatment ($p < 0.05$). BMI, FPG, FPI, HOMA-IR, CHO, TG and LDL in the olanzapine group were significantly higher than those in the ziprasidone group, with statistical significance ($p < 0.05$). The

Table 1. Changes in related indexes in each group before and after treatment.

Parameters	Before treatment		χ^2/t-value	p-value	After treatment		χ^2/t-value	p-value
	Olanzapine	Ziprasidone			Olanzapine	Ziprasidone		
Age (years) mean ± SD	43.5 ± 4.9	43.6 ± 4.4	0.096	0.924	/	/	/	/
Gender N (%)	/	/	0.205	0.651	/	/	/	/
Male	22 (55)	24 (60)	/	/	/	/	/	
Female	18 (45)	16 (40)	/	/	/	/	/	/
Weight (kg) mean ± SD	76.0 ± 7.9	76.1 ± 7.7	0.057	0.954	78.9 ± 7.7	77.2 ± 7.5	0.968	0.336
BMI (kg/m^2)	25.5 ± 1.5	25.4 ± 1.3	0.319	0.751	26.7 ± 1.2*	26.1 ± 1.4*	2.058	0.043
FPG (mmol/L)	4.89 ± 0.55	4.90 ± 0.49	0.086	0.932	5.56 ± 0.34*	5.19 ± 0.61*	3.351	0.001
FPI (mIU/L)	7.44 ± 0.64	7.43 ± 0.73	0.065	0.948	8.80 ± 0.53*	8.40 ± 0.70*	2.881	0.005
HOMA-IR	1.62 ± 0.22	1.63 ± 0.25	0.190	0.850	2.17 ± 0.19*	1.92 ± 0.28*	4.671	<0.001
CHO (mmol/L)	4.08 ± 0.60	4.11 ± 0.50	0.243	0.809	4.98 ± 0.61*	4.68 ± 0.46*	2.483	0.015
TG (mmol/L)	1.14 ± 0.27	1.16 ± 0.28	0.325	0.746	1.57 ± 0.17*	1.41 ± 0.24*	3.441	0.001
HDL (mmol/L)	1.49 ± 0.07	1.50 ± 0.06	0.686	0.495	1.25 ± 0.07*	1.38 ± 0.06*	8.918	<0.001
LDL (mmol/L)	2.30 ± 0.22	2.35 ± 0.16	1.162	0.249	3.28 ± 0.24*	2.91 ± 0.19*	7.645	<0.001

Note: BMI, body mass index; FPG, fasting plasma glucose; FPI, Fasting plasma insulin; HOMA-IR, homeostatic model assessment of insulin resistance; CHO, cholesterol; TG, triglyceride; HDL, high-density lipoprotein; LDL, low-density lipoprotein. *$p < 0.05$ compared with the same group before treatment.

Table 2. Comparison of PANSS scores between the two groups before and after treatment.

Parameters	Before treatment		t-value	p-value	After treatment		t-value	p-value
	Olanzapine	Ziprasidone			Olanzapine	Ziprasidone		
PANSS Total	79.5 ± 7.9	79.6 ± 6.9	0.060	0.952	74.7 ± 7.4*	73.8 ± 6.5*	0.578	0.565
PANSS Positive	16.4 ± 4.1	16.5 ± 3.6	0.116	0.908	15.3 ± 3.7*	14.8 ± 3.4*	0.629	0.531
PANSS Negative	24.5 ± 3.5	24.6 ± 3.3	0.131	0.896	22.8 ± 3.3*	22.6 ± 3.2*	0.275	0.784
PANSS General	38.6 ± 4.5	38.4 ± 4.6	0.197	0.845	36.6 ± 4.2*	36.4 ± 4.3*	0.210	0.834

Note: PANSS, Positive and Negative Syndrome Scale. *$p < 0.05$ compared with the same group before treatment.

level of HDL in both groups was significantly decreased after treatment, and the level of HDL in the olanzapine group was significantly lower than that in the ziprasidone group after treatment, with statistical significance ($p < 0.05$).

Comparison of PANSS Scores between the Two Groups before and after Treatment

The PANSS scores for both groups before and after treatment are shown in Table 2. The PANSS has three dimensions of schizophrenia symptoms. There were no statistically significant differences in total PANSS score, PANSS Positive score, PANSS Negative score, or PANSS General score between the two groups before treatment ($p > 0.05$). After treatment, both groups exhibited significantly reduced total scores and scores of PANSS compared to pretreatment ($p < 0.05$). However, there were no significant differences in these scores between the two groups posttreatment ($p > 0.05$).

Comparison of IR Incidence

Before treatment, there was no significant difference in IR incidence between the two groups. After treatment, the incidence of IR in the olanzapine group was significantly higher than in the ziprasidone group ($\chi^2 = 4.021$, $p < 0.05$; Table 3).

Table 3. Comparison of IR incidence.

	Before treatment		After treatment	
	IR	Non-IR	IR	Non-IR
Olanzapine	8 (20%)	32 (80%)	36 (90%)	4 (10%)
Ziprasidone	14 (35%)	26 (65%)	29 (72.5%)	11 (27.5%)
χ^2-value	2.257		4.021	
p-value	0.133		0.045	

IR, insulin resistance.

Table 4. Comparison of clinical data between IR and non-IR groups.

Parameters	IR	Non-IR	χ^2/*t*-value	*p*-value
Age (years) mean ± SD	43.7 ± 4.6	43.0 ± 4.9	0.465	0.643
Gender N (%)			0.046	0.831
Male	40	6		
Female	29	5		
Weight (kg) mean ± SD	78.2 ± 7.6	77.2 ± 7.7	0.405	0.687
BMI (kg/m^2)	26.3 ± 1.4	25.3 ± 1.3	2.220	0.029
FPG (mmol/L)	5.46 ± 0.38	4.53 ± 0.73	6.500	<0.001
FPI (mIU/L)	8.76 ± 0.53	7.78 ± 0.66	5.504	<0.001
CHO (mmol/L)	4.87 ± 0.57	4.58 ± 0.46	1.603	0.113
TG (mmol/L)	1.51 ± 0.24	1.33 ± 0.20	2.357	0.021
HDL (mmol/L)	1.28 ± 0.08	1.31 ± 0.05	1.203	0.233
LDL (mmol/L)	3.12 ± 0.28	2.93 ± 0.25	2.118	0.037
PANSS Total	74.4 ± 6.9	73.3 ± 7.2	0.488	0.627
PANSS Positive	15.1 ± 3.5	14.7 ± 4.3	0.341	0.734
PANSS Negative	22.7 ± 3.2	22.7 ± 4.2	0.019	0.985
PANSS General	36.6 ± 5.6	35.8 ± 5.7	0.439	0.662

Table 5. Multivariate analysis of IR influencing factors.

Item	B value	Standard error	Wald value	*p*-value	OR value	95% CI
BMI (kg/m^2)	0.197	0.091	4.687	0.030	1.218	1.019–1.456
FPG (mmol/L)	1.013	0.277	13.374	<0.001	2.754	1.600–4.739
FPI (mIU/L)	1.021	0.283	13.016	<0.001	2.776	1.594–4.834
TG (mmol/L)	0.023	0.011	4.372	0.037	1.023	1.001–1.046
LDL (mmol/L)	0.341	0.158	4.658	0.031	1.406	1.032–1.917

Note: OR, odd ratio; CI, confidence interval.

Comparison of Clinical Data between IR and Non-IR Groups

After treatment, BMI, FPG, FPI, TG, and LDL levels were significantly higher in the IR group compared to the non-IR group ($p < 0.05$, Table 4).

Multivariate Analysis of IR Influencing Factors

IR occurrence was taken as the dependent variable (occurrence = 1, non-occurrence = 0), and factors displaying statistically significant differences in Table 4 were used as independent variables. The multivariate analysis indicated that BMI, FPG, FPI, TG, and LDL were independent risk factors for the occurrence of IR (odd ratio (OR) >1, $p < 0.05$, Table 5).

Discussion

In this study, individuals diagnosed with schizophrenia were treated with olanzapine and ziprasidone over 12 weeks. The changes of body weight, BMI, FPG, FPI, HOMA-IR, CHO, TG, HDL, LDL and other indexes of the participating patients were mainly recorded. The results indicated that treatment with olanzapine and ziprasidone significantly impacted these essential markers in patients with schizophrenia. After treatment, both groups exhibited significantly higher body weight compared to baseline, although there was no notable difference in mean body weight between the two groups. Additionally, BMI, FPG, FPI, HOMA-IR, CHO, TG, and LDL levels significantly increased in both groups post-treatment. These indices were notably higher in the olanzapine group compared to the ziprasidone group. Conversely, HDL levels significantly decreased in both groups, with the olanzapine group showing markedly lower HDL levels compared to the ziprasidone group.

Long-term use of antipsychotic medications is typically required for managing schizophrenia. While these drugs are essential for disease control, they can elevate the risk of metabolic syndrome and cardiovascular events as adverse effects [19]. Olanzapine and ziprasidone are commonly used antipsychotics, but they differ in mechanism of action, efficacy, and side effects. Olanzapine, an atypical

SGA, primarily acts on dopamine and serotonin receptors. It antagonizes dopamine D2 receptors in the mesencephalic limbic pathway, blocking dopamine effects on postsynaptic receptors. Olanzapine binds loosely to the receptor and separates easily, allowing normal dopamine neurotransmission. The effect on the D2 receptor led to a reduction in positive symptoms in patients, including hallucinations, delusions, language, thought, and behavior disturbances. It also acts as an antagonist to the serotonin-2A (5HT2A) receptor in the frontal cortex, reducing negative symptoms like anhedonia, low mood, pain, vomiting, and inattention [20,21].

Ziprasidone, an atypical antipsychotic, exhibits binding affinity for dopaminergic (DA), serotonergic (5HT), adrenergic (α1), and histaminergic (HA) receptors. Antagonism of the D2 receptor in the mesolimbic pathway effectively reduces positive symptoms, while antagonism of the 5HT2A receptor reduces negative symptoms of psychosis. However, antagonism of histaminergic and adrenergic (α1) receptors can lead to side effects such as drowsiness and postural hypotension [22,23].

Moreover, studies have indicated a notable prevalence of prediabetes and metabolic irregularities among schizophrenia patients treated with olanzapine, placing them at an elevated risk for subsequent cardiovascular diseases and type 2 diabetes [24]. Olanzapine is associated with significant peripheral metabolic complications, including hyperglycemia, insulin resistance, hyperlipidemia, weight gain, and the onset of T2DM, all of which can contribute to increased mortality [25,26]. In our study, body weight, fasting blood glucose, and the insulin resistance (IR) index of patients significantly increased after olanzapine treatment compared to pre-treatment. Similar trends were observed in the ziprasidone group. Additionally, the post-treatment data for the olanzapine group were higher than for the ziprasidone group, although the differences did not reach statistical significance.

Weight gain (at least 7% from baseline) is a side effect of almost all antipsychotics, though the likelihood varies across drugs. Olanzapine is primarily associated with weight gain. Individual susceptibility to weight changes on the same antipsychotic also varies [27]. Our study observed varying degrees of weight gain among patients receiving the same medication.

Levels of FPG, FPI, HOMA-IR, CHO, TG, and LDL significantly increased in both groups post-treatment, with the olanzapine group showing significantly higher levels than the ziprasidone group. HDL levels decreased significantly in both groups, with the olanzapine group showing a more notable decrease.

The levels of LDL and TG can predict IR in patients with schizophrenia [18]. Abnormal glucose metabolism is common in these patients. For example, studies have found that fasting blood glucose, insulin levels, and insulin resistance indices in patients with schizophrenia are higher than in healthy individuals [28,29]. Lipid metabolism is also closely related to schizophrenia [30]. Liu *et al*. [31] found that the incidence of elevated TG, TC, and LDL-C in patients with schizophrenia was lower than that in the general population, but the level of HDL-C was generally lower, suggesting early-onset lipid abnormalities in patients with schizophrenia.

Acute administration of olanzapine triggers IR by increasing hepatic glucose production, reducing glucose uptake, and rapidly triggering adverse metabolic reactions, even preceding weight gain. Individuals treated with olanzapine showed notably elevated insulin and glucose levels compared to those receiving conventional antipsychotics, suggesting a direct stimulatory effect of olanzapine on insulin secretion from pancreatic beta cells or as a consequence of insulin resistance [32]. A meta-analysis investigating the side effects of olanzapine and ziprasidone in patients with schizophrenia observed changes in TG, LDL, HDL, CHO, and FPG. Significant differences were observed between the two antipsychotics in terms of metabolic side effects, with olanzapine exhibiting more severe side effects and ziprasidone showing milder side effects [33]. Schoretsanitis *et al*. [34] found that all patients treated with olanzapine were at risk for weight gain. In addition to the metabolic impacts, olanzapine treatment has been associated with less common side effects, including elevated prolactin [35]. Therefore, it is recommended that patients should undertake lifestyle adjustments, such as diet control and physical exercise in the early stage. Drug intervention should also be considered as appropriate to reduce the risk of developing abnormal glucose metabolism.

In this study, it was observed that the treatment with olanzapine and ziprasidone in patients with schizophrenia significantly reduced PANSS scores. The PANSS score of olanzapine treatment group was significantly higher than that of ziprasidone treatment group. These findings indicate that olanzapine and ziprasidone are effective in improving clinical symptoms in patients with schizophrenia.

The study highlights the need for further research into the mechanisms of insulin resistance caused by the long-term use of olanzapine and ziprasidone to identify suitable drug intervention measures. Additionally, due to the small sample size of this study, further research is required to explore the effects of olanzapine and ziprasidone on patients with schizophrenia in the later stage.

Conclusions

Treatment with olanzapine and ziprasidone improves clinical symptoms in patients with schizophrenia but increases the risk of insulin resistance, and the metabolic side effects of olanzapine are more pronounced. By comprehensively evaluating the levels of FPG, FPI, TG, LDL, and other indicators in patients with schizophrenia, valuable insights can be provided for the diagnosis, prevention, and management of the condition. Additionally, lifestyle adjustments such as diet control and physical exercise, and appropriate drug interventions should be considered to mitigate these metabolic side effects.

Availability of Data and Materials

The data used to support the findings of this study are included within the article, and during the present study are available from the corresponding author on reasonable request.

Author Contributions

FW designed the research study. FW, FYW and XQT performed the research. WXN and WXL analyzed the data. JL helped perform the analysis with constructive discussions. All authors contributed to the drafting or important editorial changes in the manuscript. All authors read and approved the final manuscript. All authors have participated sufficiently in the work and agreed to be accountable for all aspects of the work.

Ethics Approval and Consent to Participate

The study protocol was approved by the Wuyi County First People's Hospital Ethics Committee (Approval No. 2024516) and the study was carried out in accordance with the Declaration of Helsinki. After receiving a full study description, all participants were given informed consent.

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Conflict of Interest

The authors declare no conflict of interest.

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