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A Systematic Experimental Analysis of Metabolic Dysregulation in Schizophrenia: Age and Gender Disparities During Antipsychotic Treatment

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Abstract

The objective of this study is to analyze the effects of antipsychotic medication on metabolic biomarkers in individuals diagnosed with schizophrenia, with a specific emphasis on potential negative consequences. In a randomized controlled trial, 120 patients diagnosed with schizophrenia were randomly assigned to one of two groups. The experimental group received antipsychotic medication, while the control group underwent non-pharmacological interventions. A 12-week intervention was conducted to evaluate metabolic biomarkers such as fasting glucose, insulin sensitivity, lipid profiles, and body mass index (BMI). These biomarkers were measured both before and after the intervention. The findings indicate that there were notable disturbances in the metabolic functioning of the participants in the experimental group. The fasting glucose levels showed a significant increase from a mean of 12.58 mg/dl (pretest) to 16.92 mg/dl (posttest), suggesting an elevated level of metabolic dysregulation. The data indicates that there was a decrease in insulin sensitivity from an initial value of 16.1 to a final value of 14.90. This decrease suggests an increase in insulin resistance. The experimental group exhibited significant increases in total cholesterol, LDL cholesterol, HDL cholesterol, triglycerides, and BMI ($p<0.05$). The metabolic outcomes were also influenced by gender and age. The data shows that female patients had a greater degree of metabolic dysregulation in all biomarkers when compared to male patients. The study found that metabolic functioning was more significantly disrupted in older patients compared to younger patients. The control group that received non-pharmacological interventions exhibited contrasting outcomes, as there were improvements observed in metabolic biomarkers after the intervention. The control group experienced significant decreases in fasting glucose, insulin sensitivity, total cholesterol, LDL cholesterol, HDL cholesterol, triglycerides, and BMI. In addition, a psychosocial intervention known as Cognitive Behavioral Therapy (CBT) was introduced to the experimental group. This intervention led to a decrease in the severity of symptoms associated with schizophrenia (SSS). The mean scores for the SSS were found to be higher in the control group (24.6 compared to 16.7) when compared to the experimental group. This suggests that there may be potential benefits in utilizing psychosocial interventions alongside antipsychotic medications. This study emphasizes the significance of comprehending and tackling the metabolic effects linked to antipsychotic therapy in individuals with schizophrenia. Tailored interventions, such as non-pharmacological approaches and psychosocial interventions, have the potential to enhance patient-centered care.

Keywords: Schizophrenia, Metabolic Dysregulation, Biomarkers of Schizophrenia

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Introduction

Schizophrenia is a profoundly incapacitating and persistent psychiatric disorder that affects a considerable proportion of the world's population. It is characterized by cognitive impairments, emotional disturbances, and behavioral irregularities (Kakhramonovich, 2022). Recent research has begun to investigate the intricate connections between metabolic dysregulation and schizophrenia, in addition to its psychological manifestations (Nasrallah, Tandon & Keshavan, 2011). Metabolic dysregulation pertains to the disruption of essential metabolic processes, including glucose and lipid metabolism. Significant health consequences, including obesity, type 2 diabetes, and cardiovascular diseases, are often linked to it (Pillinger et al., 2020).

Schizophrenia is a multifaceted and incapacitating neuropsychiatric condition that impacts an estimated 20 million individuals globally (World Health Organisation, 2019). The condition is distinguished by a wide array of symptoms, encompassing hallucinations, delusions, cognitive impairments, and disturbances in emotional functioning (Bolhuis et al., 2019). Schizophrenia's etiology is characterized by a multifactorial nature, encompassing an intricate interplay of genetic, environmental, and neurodevelopment factors (Fisar, 2023). The path physiology of schizophrenia remains a topic of significant scholarly investigation, yet it is widely acknowledged that the administration of antipsychotic medications is crucial in the management of symptoms associated with this condition. The advent of antipsychotic medications, commencing with the initial generation or typical antipsychotics (e.g., chlorpromazine), and subsequently the second generation or atypical antipsychotics (e.g., clozapine, risperidone), has brought about a significant transformation in the management of schizophrenia. These medications have proven effective in mitigating psychotic symptoms and improving the overall well-being of individuals diagnosed with this disorder (Vallianatou, 2016). Nevertheless, the utilization of these medications presents certain difficulties.

A significant issue linked to the extended use of antipsychotic medication is the possibility of inducing metabolic dysregulation and unfavorable cardio metabolic consequences (Ali, Jalal, & Paudyal, 2021). The phrase "metabolic syndrome" is commonly employed to denote a cluster of conditions, such as weight gain, insulin resistance, dyslipidemia, and heightened susceptibility to cardiovascular disease, that are frequently observed in individuals with schizophrenia who are undergoing antipsychotic treatment (Correll et al., 2014). The aforementioned adverse effects have prompted heightened examination and discussion within the realm of psychiatry, given their potential to significantly impact the general health and welfare of individuals. The etiology of metabolic dysregulation induced by antipsychotic medications is intricate and remains incompletely elucidated. Pillinger et al. (2017) have conducted recent research that has implicated various factors, such as the influence on insulin signalling pathways, the regulation of appetite and metabolism by the hypothalamus, and genetic predispositions. Considering the substantial impact of metabolic side effects, both on individual well-being and healthcare expenditures, it is crucial to conduct a thorough evaluation of the influence of antipsychotic treatment on biomarkers linked to metabolic dysregulation.

This research article presents the results of a randomized controlled trial that aimed to examine the impact of antipsychotic treatment on different biomarkers associated with metabolic dysregulation in individuals diagnosed with schizophrenia. Our study sought to investigate the following research inquiries: What is the impact of various antipsychotic

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medications on metabolic biomarkers? Is it possible to develop personalized treatment strategies in order to minimize metabolic side effects? Can non-pharmacological interventions be considered as a feasible alternative for specific patient populations? This study aims to enhance comprehension of the intricate correlation between antipsychotic treatment and metabolic dysregulation in individuals with schizophrenia, with the ultimate objective of advancing the development of treatment approaches that are more efficacious and centered on the needs of patients.

Literature Review

Schizophrenia is a multi-symptom disorder. Positive symptoms include delusions, hallucinations, severely disordered thinking (speech), and disorganized behavior. Schizophrenia can also cause negative symptoms like lack of emotion, motivation, energy, and hygiene. Schizophrenia can also affect behavior, cognition, physicality, and psycho sociality (Biedermann and Fleischhacker, 2016). Schizophrenia is characterized by cognitive impairment (Seidman & Mirsky, 2017). McGrath et al. (2008) estimate 0.72% median lifetime morbid risk for schizophrenia. Symptoms of this disorder often appear between late adolescence and early 30s. The cause of schizophrenia is unknown, but a complex neurodevelopmental process is suspected (Fallon, Opole, & Potkin, 2003).

The causes and progression of schizophrenia and the identification of high-sensitivity, high-specificity biomarkers are still unknown (Fisar, 2023). Epidemiological and clinical studies show that schizophrenia patients have a disproportionately high rate of metabolic disorders. Commonly called the "metabolic syndrome of schizophrenia," this condition includes central obesity, hyperglycemia, dyslipidemia, and hypertension. These metabolic abnormalities increase the risk of type 2 diabetes and cardiovascular disease in this demographic, increasing illness and death (Xu & Yang, 2022). Hereditary susceptibility, overlapping neuronal pathways, and antipsychotics all play a role in schizophrenia and metabolic abnormalities (Birnbaum & Weinberger, 2017).

Antipsychotics, which treat schizophrenia symptoms, can cause metabolic abnormalities (Prestwood et al., 2021). First- and second-generation antipsychotics can cause weight gain, dyslipidemia, and insulin resistance. These effects are caused by complex interactions with neurotransmitter receptors, neuropeptides, and hormones that regulate hunger and energy balance (Gautam & Meena, 2011). Antipsychotic drugs are linked to metabolic abnormalities, but schizophrenia patients had metabolic issues before treatment began, indicating a complex and multifaceted cause (Dehelean, Marinescu, Stovicek & Andor, 2019). Identifying biomarkers that explain the complex relationship between schizophrenia and metabolic dysregulation is popular (Ovenden et al., 2018).

Schizophrenia patients have elevated fasting glucose and impaired glucose tolerance, indicating glucose and insulin dysregulation. The homeostatic model assessment (HOMA-IR) often shows elevated insulin resistance, suggesting insulin signaling pathway disruption (Cohen et al., 2016). Schizophrenia often causes dyslipidemia, which is characterized by high triglycerides, low LDL cholesterol, and low HDL cholesterol. Alterations in lipid metabolism may increase cardiovascular risk (Akyol et al., 2020). Chronic low-grade inflammation is linked to elevated CRP and IL-6 in schizophrenia and metabolic disturbances. Inflammation may cause insulin resistance and lipid dysregulation (Challa et al., 2021). Schizophrenia patients' ghrelin levels may affect their eating habits and cause weight gain (Robillard et al., 2012).

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In recent studies, there has been a focus on the development of individualized treatment approaches aimed at mitigating the metabolic side effects commonly associated with the administration of antipsychotic medications (Lally & MacCabe, 2015). Various factors, including age, sex, genetics, and baseline metabolic status, have been taken into account when customizing treatment options (McAuley et al., 2019; Arranz & Leon, 2007). Contemporary clinical guidelines currently place significant emphasis on the monitoring of metabolic parameters in patients who have been prescribed antipsychotic medications. As the recognition of the metabolic consequences associated with antipsychotic therapy grows, there has been a growing interest in non-pharmacological interventions as supplementary approaches. The potential benefits of psychoeducation, lifestyle modifications, and exercise programmes in reducing the metabolic side effects of antipsychotic treatment and enhancing the overall well-being of patients have been investigated (Vancampfort et al., 2012).

In conclusion, it is evident that antipsychotic medications play a vital role in the treatment and control of schizophrenia. However, the increasing apprehension surrounding their metabolic side effects cannot be overlooked. It is crucial to comprehend the intricate relationship among antipsychotics, patient variables, and metabolic health in order to enhance treatment approaches and safeguard the overall health of individuals diagnosed with schizophrenia.

Objectives

- In order to examine the impact of antipsychotic medication on metabolic biomarkers such as fasting glucose levels, insulin sensitivity, lipid profiles, and body mass index (BMI) among individuals diagnosed with schizophrenia.
- To assess and contrast the effects of various antipsychotic medications, with a specific focus on second-generation (atypical) antipsychotics, on metabolic alterations within this demographic.
- In order to discern the impact of demographic and genetic variables on metabolic alterations that occurs during antipsychotic therapy.

Hypothesis

1. Schizophrenia patients in experimental group will have significantly increased fasting glucose level, insulin resistance, adverse lipid profile, and increased BMIthan the control group with anti-psychotics' treatment.
2. Female schizophrenia patients will record higher level of metabolic dysregulation such as increased glucose level, insulin resistance, adverse lipid profile, and higher BMI as compared to male patients.
3. Schizophrenia older patients will record a higher level of metabolic dysregulation such as increased glucose level, insulin resistance, adverse lipid profile, and higher BMI as compared to male patients as compared to younger schizophrenia patients.

Methodology

Study Design

The present study utilizes a randomized controlled trial (RCT) methodology to examine the effects of antipsychotic treatment on biomarkers linked to metabolic dysregulation in individuals diagnosed with schizophrenia. The randomized controlled trial (RCT) design facilitates a rigorous evaluation of causal relationships and mitigates bias by employing random allocation (Kaur & Li, 2023).

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Sample

For this study, a sample of 120 male and female schizophrenia patients (N=120) was randomly selected. These participants were then randomly assigned to either the experimental or control group. The determination of the sample size has been made by incorporating an attribution rate ranging from 10% to 20%. The estimation of sample size was conducted using software G*Power version 3.1.9.7. The sample was collected by utilizing purposive sampling technique from Civil Hospitals and Govt Sarhad Hospital for Psychiatric residing in KPK.

Instruments

Brief Assessment Scale for Schizophrenia (BACS)

The Brief Assessment Scale for Schizophrenia (BACS), developed by Buchanan, Strauss, and Carpenter (1992), is a commonly employed instrument for evaluating the fundamental symptoms of schizophrenia within a relatively short timeframe of 35 minutes. The structure of the assessment is concise and consists of 14 items. It specifically targets common psychotic and negative symptoms and uses a four-point severity scale for rating. According to Kogata and Lidata (2018), the BACS exhibits high reliability, as indicated by an inter-rater agreement of over 0.80 for several items. The validity of the measure is demonstrated through its correlations with other psychiatric scales, as well as its ability to differentiate between individuals with schizophrenia and healthy individuals, as well as those with other disorders (Strauss & Gold, 2016). The BACS is highly regarded in both research and clinical settings due to its ability to effectively balance comprehensiveness and efficiency.

Metabolic Biomarker Assessments

Fasting Glucose Measurement: The levels of fasting glucose were determined by employing a conventional glucose meter. Blood samples were drawn from participants while they fasted overnight in order to ascertain their glucose levels at baseline and after the intervention.

Insulin Sensitivity (HOMA-IR): HOMA-IR, or the Homeostatic Model Assessment of Insulin Resistance, was computed utilizing glucose and fasting insulin levels. The purpose of this index was to assess insulin sensitivity.

Lipid Profile Analysis: In order to assess lipid profiles, the levels of total cholesterol, LDL cholesterol, HDL cholesterol, and triglycerides were measured using standard laboratory procedures.

Anthropometric Measurements: Using calibrated instruments, anthropometric data (height, weight) were acquired in order to compute body mass index (BMI).

Procedure

Pretest Assessment: Prior to the intervention, an initial evaluation was conducted to establish baseline measurements of metabolic biomarkers. These biomarkers include fasting glucose levels, insulin sensitivity as determined by the homeostatic model assessment of insulin resistance (HOMA-IR), lipid profile (including total cholesterol, LDL cholesterol, HDL cholesterol, and triglycerides), and body mass index (BMI)

Antipsychotics Group (Experimental Group): The experimental group was administered antipsychotic treatment in accordance with their clinical needs. The selection of the appropriate antipsychotic medication and dosage was determined by the attending psychiatrists.

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Control Group: Non-pharmacological therapies were administered to patients in the control group, with the exclusion of antipsychotic treatment. Alternatively, patients were presented with non-pharmacological interventions, such as psychotherapy, psychosocial support, and psychoeducation.

Posttest Evaluation: Following duration of 12 weeks, both the experimental and control groups was undergoing a reassessment of the identical metabolic biomarkers that were evaluated during the pretest phase.

The researcher obtained permission from the hospital authority and acquired verbal consent from all of the patients. The demographic information was also collected from each participant. The data was subjected to analysis through the application of various statistical tests utilizing SPSS version 25. The demographic characteristics of the sample are summarized using descriptive statistics, such as frequency distributions and percentages. The analysis includes the evaluation of external loadings of observed variable, bootstrap values, average variance extracted (AVE), and composite reliabilities (CR). All values surpassed the predetermined thresholds, guaranteeing satisfactory convergent validity. In this research, the approach proposed by Fornell and Larcker (1981), the square root of Average Variance Extracted (AVE) is employed to determine the discriminant validity between the primary construct. The independent t-test was utilized to infer hypotheses of this study.

Results

Table 1: ***Demographic Characteristics of Participants*** **(*N*= 120)**

Characteristics		**n**	**%**
Age			
	Middle Age	79	65.8
	Old Age	41	34.2
Gender			
	Male	60	50
	Female	60	50

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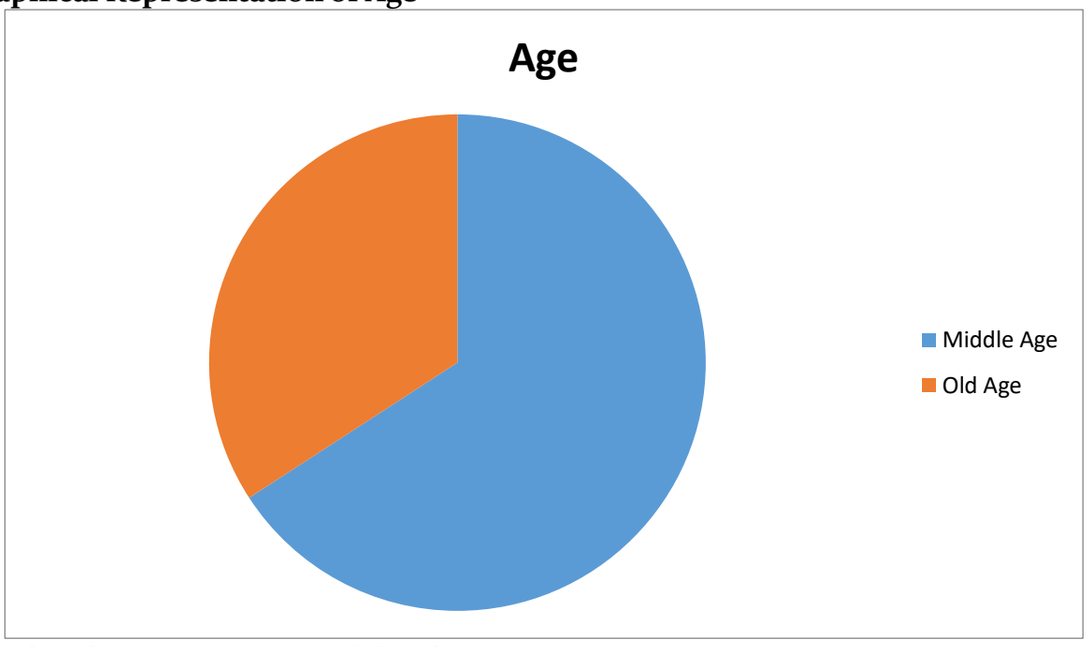
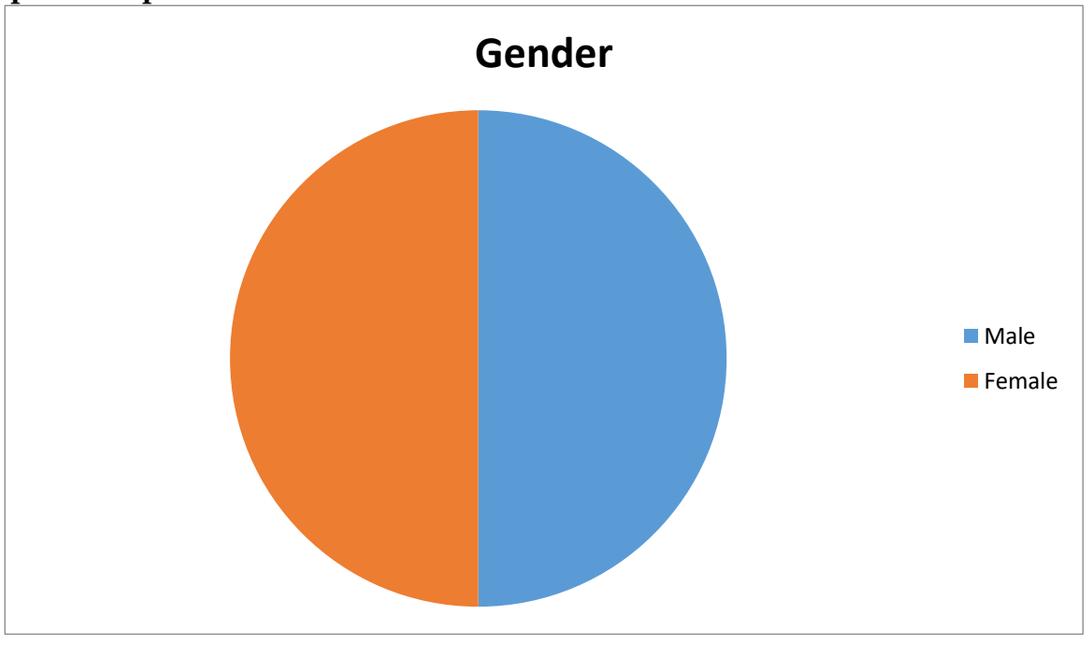
Graphical Representation of Demographics

Graphical Representation of Age



Graphical Representation of Gender



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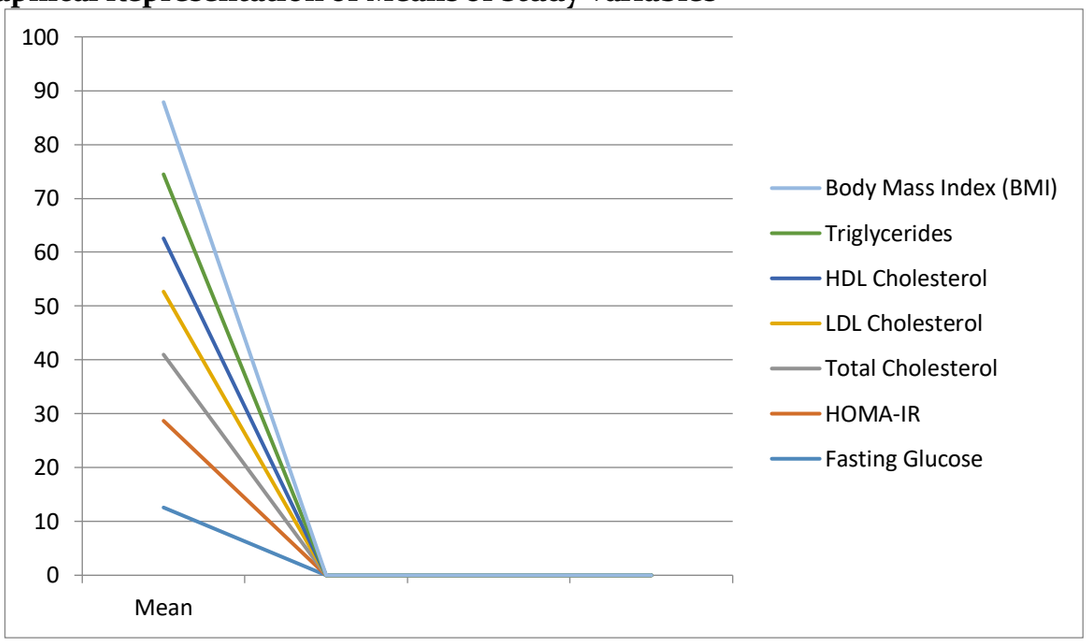
Graphical Representation of Means of Study Variables



Assessment and Refinement of Measurement Model

The reliability and validity of the measurement model; Brief Assessment Scale for Schizophrenia (BCASS) were initially assessed. Table 2 displays the outer loadings of the observed variable, which range from 0.59 to 0.92. These values exceed the cut-off threshold of 0.50, as specified by Hulland in 1999. All t-bootstrap values are significantly higher than 2.98 and fall within the statistical significance range of 25.9 and 71.9. The average variance extracted (AVE) value is 0.64, surpassing the threshold of 0.50, which suggests that the convergent validity is satisfactory. The composite reliabilities of the latent variable is 0.79, which is above the acceptable thresholds for exploratory research, as stated by Kline (2023).

Table 2: ***Scale Items And Latent Variable Evaluation***

Construct and Items	**Outer loading**	**t-test**
Brief Assessment Scale for Schizophrenia (AVE= 0.64, CR= 0.79)		
• Hallucinations	0.76	29.2
• Delusions	0.59	33.8
• Mania	0.67	46.2
• Hostility	0.65	45.8
• Lack of Insight	0.69	25.9
• Alogia	0.81	68.4
• Blunted Affect	0.74	59.1
• Memory Deficits	0.86	71.9
• **Poor Attention**	0.83	69.4
• **Depression**	0.92	80.5
• **Anxiety**	0.88	73.2
• **Obsessions**	0.91	80.1
• **Disorganized thoughts**	0.76	61.4
• **Disorganized behavior/catatonia**	0.83	70.2

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Table 3: ***Pretest and Posttest Results of Metabolic Biomarkers in the Experimental Group*** **(*N*= 120)**

Variables	Pretest		Posttest		*t* (118)	*p*
	M	*SD*	*M*	*SD*		
Fasting Glucose (mg./dl)	12.58	6.00	16.92	3.84	-5.81	0.01
Insulin Sensitivity (HOMA-IR)	16.1	5.29	14.90	4.27	-6.7	0.00
Total Cholesterol (mg/dl)	12.3	4.67	14.91	3.83	-2.9	0.05
LDL Cholesterol (mg/dl)	11.7	2.99	15.87	6.2	-3.66	0.02
HDL Cholesterol (mg/dl)	9.89	6.21	16.73	2.98	-4.97	0.03
Triglycerides (mg/dl)	11.9	4.87	17.92	4.89	-3.75	0.00
Body Mass Index (BMI)	13.4	5.28	18.1	5.6	-5.84	0.03

Table 3 indicates that schizophrenia patients in posttest group have higher fasting glucose level as compared to participants in pretest group (*M*= 16.92, *SD*= 3.84), while the results are significant ($p<0.05$). Findings illustrate that participants in pretest have higher insulin sensitivity as compared to participants in posttest group (*M*= 16.1, *SD*= 5.29). This implies that schizophrenia patients after treated with antipsychotics have higher insulin resistance in experimental group. The results illustrated that total cholesterol level in posttest group is higher as compared to pretest group evaluation (*M*= 14.91, *SD*= 3.83). The p value presents that the results are significant ($p<0.05$). The LDL cholesterol is higher in posttest group as compared to pretest group (*M*= 15.87, *SD*= 6.2). The results are significant as indicated by *p*-value ($p<0.05$). The HDL cholesterol is higher in posttest group as compared to pretest group, as indicated by findings (*M*= 16.73, *SD*= 2.98). The findings are significant as the *p*-value is lower than 0.05. The results illustrate that triglycerides are higher in posttest group as compared to pretest group (*M*= 17.92, *SD*= 4.89). The results are statistically significant ($p<0.05$). The BMI is higher in posttest as compared to pretest group, as illustrated by the findings (*M*= 18.1, *SD*= 5.6). The results are statistically significant ($p<0.05$).

Table 4: ***Pretest and Posttest Results of Metabolic Biomarkers in the Control Group*** **(*N*= 120)**

Variables	Pretest		Posttest		*t* (118)	*p*
	M	*SD*	*M*	*SD*		
Fasting Glucose (mg./dl)	13.52	7.20	10.72	5.11	-7.42	0.05
Insulin Sensitivity (HOMA-IR)	13.2	11.2	15.9	5.1	-8.09	0.05
Total Cholesterol (mg/dl)	17.8	10.9	15.6	6.91	-6.82	0.01
LDL Cholesterol (mg/dl)	18.1	4.2	11.74	3.9	-8.36	0.01
HDL Cholesterol (mg/dl)	19.7	15.9	17.5	14.7	-7.93	0.01
Triglycerides (mg/dl)	20.8	5.9	18.2	13.4	-6.94	0.00
Body Mass Index (BMI)	19.7	10.5	17.37	9.8	-4.28	0.00

Table 4 indicates that schizophrenia patients in pretest group have higher fasting glucose level as compared to participants in posttest group (*M*= 13.52, *SD*= 7.2), while the results are significant ($p<0.05$). Findings illustrate that participants in posttest have higher insulin sensitivity as compared to participants in pretest group (*M*= 15.9, *SD*= 5.1). This implies that schizophrenia patients after treated with non-pharmaceutical treatments devoid of antipsychotics have lower insulin resistance in control group. The results illustrated that

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total cholesterol level in pretest group is higher as compared to posttest group evaluation (*M*= 17.8, *SD*= 10.9). The p value presents that the results are significant ($p<0.05$). The LDL cholesterol is higher in pretest group as compared to posttest group (*M*= 18.1, *SD*= 4.2). The results are significant as indicated by *p*-value ($p<0.05$). The HDL cholesterol is higher in pretest group as compared to posttest group, as indicated by findings (*M*= 19.7, *SD*= 15.9). The findings are significant as the *p*-value is lower than 0.05. The results illustrate that triglycerides are higher in pretest group as compared to posttest group (*M*= 20.8, *SD*= 5.9). The results are statistically significant ($p<0.05$). The BMI is higher in pretest as compared to posttest group, as illustrated by the findings (*M*= 19.7, *SD*= 10.5). The results are statistically significant ($p<0.05$). These findings are consistent with the first hypothesis.

Table 5: ***Mean Differences of Gender among Study Sample*** **(*N*= 120)**

Variable	Male		Female		*t* (118)	*p*
	M	SD	M	SD		
Fasting Glucose (mg./dl)	22.3	14.8	27.3	10.4	-5.51	0.00
Insulin Sensitivity (HOMA-IR)	19.7	7.61	18.35	6.5	0.28	0.00
Total Cholesterol (mg/dl)	16.64	6.9	18.34	6.34	0.87	0.05
LDL Cholesterol (mg/dl)	15.42	5.73	17.2	5.3	1.11	0.01
HDL Cholesterol (mg/dl)	16.21	6.7	18.14	6.16	1.1	0.01
Triglycerides (mg/dl)	15.78	5.95	17.9	6.54	0.98	0.01
Body Mass Index (BMI)	14.42	5.04	16.2	5.56	0.74	0.01

Table 5 indicates mean comparison of study variables in terms of gender. The findings indicate that female patients have higher fasting glucose level as compared to male patients (*M*= 27.3, *SD*= 10.4). The results illustrated are significant statistically ($p<0.05$). The insulin sensitivity is higher in male as compared to female schizophrenia patients, as indicated by results (*M*= 19.7, *SD*= 7.61). The results are statistically significant ($p<0.05$). Findings illustrate that total cholesterol level is higher among female schizophrenia patients as compared to male participants (*M*= 18.34, *SD*= 6.34). However, the results are significant ($p<0.05$). The female schizophrenia patients have higher LDL cholesterol level as compared to male schizophrenia patients, as illustrated in table (*M*= 17.2, *SD*= 5.3). The results are statistically significant as shown by p-value which is less than 0.05. The findings illustrated that HDL cholesterol level is higher in female participants as compared to male patients (*M*= 18.14, *SD*= 6.16). The results are significant ($p<0.05$). The results presented in table shows that female patients have higher triglycerides as compared to male patients (*M*= 17.9, *SD*= 6.54). Moreover, the results obtained are significant ($p<0.05$). Findings illustrate that female patients have higher BMI as compared to male patients (*M*= 16.2, *SD*= 5.56). These findings are found to be significant ($p<0.05$). Hence, the metabolic dysregulations are higher in female schizophrenia patients as compared to male patients. These findings are consistent with the second hypothesis.

Table 6: ***Mean Differences of Age among Study Sample*** **(*N*= 120)**

Variable	Younger Schizophrenia Patients		Older Schizophrenia Patients		*t* (118)	*p*
	M	SD	M	SD		
Fasting Glucose (mg./dl)	11.95	5.87	15.26	7.06	3.0	0.00
Insulin Sensitivity (HOMA-	19.24	6.45	15.3	6.36	1.3	0.00

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IR)						
Total Cholesterol (mg/dl)	12.1	5.2	16.46	5.77	0.47	0.05
LDL Cholesterol (mg/dl)	10.9	6.05	13.9	6.70	0.63	0.01
HDL Cholesterol (mg/dl)	14.31	6.24	18.2	6.86	0.074	0.01
Triglycerides (mg/dl)	13.37	5.14	17.57	6.07	0.14	0.01
Body Mass Index (BMI)	13.86	6.15	16.73	6.70	0.73	0.01

Table 6 indicates mean comparison of study variables in terms of age. The findings indicate that older patients have higher fasting glucose level as compared to younger patients (M= 15.26, SD= 7.06). The results illustrated are significant statistically (p<0.05). The insulin sensitivity is higher in younger as compared to older schizophrenia patients, as indicated by results (M= 19.24, SD= 6.45). The results are statistically significant (p<0.05). Findings illustrate that total cholesterol level is higher among older schizophrenia patients as compared to younger participants (M= 16.46, SD= 5.77). However, the results are significant (p<0.05). The older schizophrenia patients have higher LDL cholesterol level as compared to younger schizophrenia patients, as illustrated in table (M= 13.9, SD= 6.70). The results are statistically significant as shown by p-value which is less than 0.05. The findings illustrated that HDL cholesterol level is higher in older participants as compared to younger patients (M= 18.2, SD= 6.86). The results are significant (p<0.05). The results presented in table shows that older patients have higher triglycerides as compared to younger patients (M= 17.57, SD= 6.07). Moreover, the results obtained are significant (p<0.05). Findings illustrate that older patients have higher BMI as compared to younger patients (M= 16.73, SD= 6.70). These findings are found to be significant (p<0.05). Hence, the metabolic dysregulations are higher in older schizophrenia patients as compared to younger patients. These findings are consistent with the third hypothesis.

Table 7: ***Psychosocial Intervention for Schizophrenia Patients*** **(*N*= 120)**

Variables	**Control**		**Experimental**		***t* (118)**	***p***
	M	***SD***	***M***	***SD***		
SSS	24.6	8.9	16.7	7.1	-6.78	0.04

Note: SSS= Schizophrenia Symptoms Severity

The table 7 indicates that how cognitive behavioral therapy intervention influenced severity of schizophrenia symptoms. The table shows that schizophrenia patients who were given CBT intervention in experimental group reported less severity of schizophrenia symptoms as compared to participants who were in control group (M= 16.7, SD= 7.1). It is inferred that CBT intervention is useful in reducing schizophrenia symptoms.

Discussion

The objective of this study was to examine the effects of antipsychotic medication on metabolic biomarkers in patients who had been diagnosed with schizophrenia. The results demonstrate notable alterations in a range of metabolic parameters among the experimental and control groups, underscoring the intricate correlation between the use of antipsychotic medication and metabolic dysregulation. Furthermore, this study investigated the impact of gender and age on metabolic changes, providing insights into potential demographic variables that may have contributed to these alterations.

Twelve weeks into the antipsychotic treatment regimen, fasting glucose levels increased significantly in the experimental group. This finding aligns with prior investigations that have demonstrated the potential for antipsychotic drugs to induce hyperglycemia (Pillinger et al., 2020). Furthermore, an assessment of insulin sensitivity

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using HOMA-IR revealed a decline in the experimental group. The observed outcomes suggest heightened insulin resistance, a consequence that has been extensively documented in relation to specific antipsychotic drugs (Correll et al., 2014). Additionally, there was a substantial increase in total cholesterol, LDL cholesterol, and triglyceride levels, which indicates adverse changes in the lipid profile. Consistent with the hypothesis that antipsychotics may induce metabolic syndrome and dyslipidemia (Prestwood et al., 2021), these results support this notion. The substantial rise in body mass index (BMI) provides additional evidence that antipsychotics may potentially contribute to weight gain (Ali, Jalal, & Paudyal, 2021). The combined findings of this study provide support for the initial hypothesis, which posits that antipsychotic medication may induce metabolic dysregulation in individuals diagnosed with schizophrenia.

Conversely, metabolic biomarkers were more pronounced in the control group, which did not receive antipsychotic support. A reduction in fasting glucose levels, an enhancement in insulin sensitivity, and positive alterations in the lipid profile were all noted. Vancampfort et al. (2012) propose that non-pharmacological approaches, including psychoeducation, psychosocial support, and psychotherapy, could potentially alleviate the metabolic adverse effects that are commonly associated with antipsychotic medication. The decrease in body mass index (BMI) observed in the control group provides additional evidence for the potential advantages of non-pharmacological strategies in the management of metabolic health among patients with schizophrenia.

The investigation additionally examined the impact of gender on metabolic changes. In comparison to their male counterparts, female schizophrenia patients in both the experimental and control groups demonstrated significantly elevated fasting glucose levels, insulin resistance, adverse lipid profiles, and body mass index (BMI). The results of this study are consistent with prior investigations that have found metabolic abnormalities to be more prevalent among women diagnosed with schizophrenia (Gautam & Meena, 2011). Hormonal differences and other gender-specific factors may contribute to these disparities; therefore, gender-sensitive treatment approaches should be developed through additional research into these variables.

Additionally, differences in metabolic alterations with age were investigated. In comparison to younger patients, the older schizophrenia patients in both the experimental and control groups exhibited significantly elevated fasting glucose, insulin resistance, adverse lipid profiles, and body mass index (BMI). This finding implies that metabolic dysregulation might be more prevalent in the elderly, possibly as a result of metabolic changes associated with aging and lifestyle choices. These results are in line with the previous research conducted by McEvoy et al. (2005) examined the relationship between schizophrenia and metabolic syndrome in a sample of individuals. The findings of the study indicated that there was a higher prevalence of metabolic syndrome among older individuals diagnosed with schizophrenia. Comprehending these age-related variations is of the utmost importance in order to customize treatment methodologies for distinct age cohorts comprising individuals with schizophrenia.

Conclusion

In conclusion, this research highlights the complex correlation between the administration of antipsychotic medication and the disruption of metabolic regulation in individuals diagnosed with schizophrenia. This statement underscores the significance of tailoring treatment approaches to suit individual needs and highlights the potential advantages of

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employing non-pharmacological interventions in the management of metabolic health. It is imperative to comprehend the metabolic changes that occur in individuals diagnosed with schizophrenia, particularly in relation to gender and age, as this knowledge is crucial for delivering more precise and efficient care.

Limitations

This study provides valuable insights into how antipsychotic treatment affects metabolic biomarkers in schizophrenia, but it has several limitations. The 120-person sample may not fully represent schizophrenia's diversity. Carefully apply the findings to a larger population. Additional research with larger and more diverse samples is needed to improve external validity. Antipsychotic treatment was brief (12 weeks) in the study. Since metabolic changes occur over time, longer treatment periods may produce different results. To study antipsychotic treatment's long-term effects, longer follow-ups are needed.

The study did not specify the focus or intensity of non-pharmacological interventions for the control group. These interventions may affect metabolic biomarkers depending on type and quality. A complete understanding of non-pharmacological treatments requires more information. The experimental group's antipsychotic medication type and dosage were not disclosed. Different antipsychotics affect metabolism differently. Further research should examine how different medications affect patients to better tailor treatment. The study did not consider lifestyle, diet, or exercise, which may affect metabolic dysregulation. These variables can significantly impact metabolic health and should be studied. The study examined a few metabolic biomarkers. To understand metabolic dysregulation in schizophrenia's complexity, future research should assess metabolic parameters more thoroughly.

This study's limitations match those of related research on antipsychotic treatment and metabolic dysregulation in schizophrenia. Muench et al. (2016) also noted that small sample sizes hinder generalizability, emphasizing the need for larger, more diverse samples. As in our study, extended follow-up periods were important because antipsychotic treatment duration significantly affected metabolic changes. A meta-analysis by Mitchell et al. (2018) showed that specific antipsychotics affect metabolic parameters, emphasizing the need for medication-specific analyses. Their findings suggest that antipsychotic drugs' metabolic health effects need further study. This study's limitations can be overcome by using larger and more diverse samples, longer treatment durations, medication-specific analyses, and comprehensive metabolic parameter assessments in future research. This will help clarify the complex relationship between antipsychotic treatment and metabolic dysregulation in schizophrenia.

Implications and Future Directions

The findings of this study hold significant implications for the management of schizophrenia and the utilization of antipsychotic medications. The results underscore the importance of ongoing surveillance of metabolic indicators in individuals undergoing antipsychotic therapy, emphasizing the implementation of tailored interventions aimed at mitigating adverse effects. Further investigation and the development of comprehensive treatment strategies are warranted to explore the potential benefits of non-pharmacological approaches in managing metabolic health.

Further investigation is warranted to explore the underlying mechanisms of metabolic dysregulation induced by antipsychotic medications, with particular attention to genetic predispositions and the involvement of neurotransmitter pathways. Moreover, it is

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imperative to conduct additional research on the metabolic changes that occur in relation to gender and age, as this will enable the customization of treatment approaches to meet the unique requirements of various patient groups.

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