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Central Obesity as a Key Driver of Cardiometabolic Risk in Schizophrenia: Insights from Antipsychotic Therapy

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Abstract

Background: Schizophrenia is associated with a markedly reduced life expectancy, predominantly due to cardiovascular disease (CVD). Metabolic syndrome (MetS) is a central mediator of this excess mortality. Both intrinsic pathophysiological mechanisms and antipsychotic treatment contribute to cardiometabolic risk. **Subjects and Methods**: A retrospective-prospective cohort study with a hybrid design included 60 patients diagnosed with schizophrenia (ICD-10), treated ≥ 6 months with either typical (n = 30) or atypical (n = 30) antipsychotic monotherapy. The retrospective component included chart review, while metabolic parameters were collected prospectively during January 2004-January 2006. Typical antipsychotic group included both monotherapy and combination regimens, while the atypical group was predominantly monotherapy, mainly clozapine. Anthropometric parameters, fasting plasma glucose, triglycerides, HDL cholesterol, and blood pressure were assessed. MetS was defined according to modified WHO criteria. Statistical analyses included Student's t-test, χ^2-test, odds ratios (OR) with 95% confidence intervals (CI), and multivariate logistic regression adjusting for age, sex, and treatment class. **Results**: This analysis should be interpreted as exploratory due to the low number of metabolic syndrome cases (n = 3), and reduced HDL cholesterol was used as the primary analytical outcome. Central obesity was present in 70% of patients receiving typical and 83.3% receiving atypical antipsychotics (OR = 2.14; 95% CI 0.63 - 7.29). MetS prevalence was 10% in the typical group and 0% in the atypical group. Multivariate analysis identified waist circumference as an independent predictor of reduced HDL levels (β =

−0.61, p = 0.02). Treatment class was not an independent predictor of MetS after adjustment. Key confounders such as smoking status, illness duration, treatment duration, and living setting were not consistently available and were therefore not included in adjusted analyses. **Conclusion**: Metabolic abnormalities are highly prevalent among patients with schizophrenia irrespective of antipsychotic class. Interpretation of atypical antipsychotic effects is limited due to the predominance of clozapine in this group. Central obesity appears to be the primary driver of cardiometabolic risk. Integrated somatic-psychiatric care and systematic metabolic monitoring are essential to reduce cardiovascular morbidity.

Keywords

Schizophrenia, Metabolic Syndrome, Antipsychotics, Cardiovascular Risk

1. Introduction

Schizophrenia is a severe, chronic psychiatric disorder affecting approximately 1% of the global population, characterized by profound disruptions in thought, perception, and behavior [1]. Despite substantial therapeutic advances in psychopharmacology and psychosocial interventions, individuals with schizophrenia continue to experience a 15 - 20-year reduction in life expectancy compared to the general population [2]. Cardiovascular disease (CVD) is the leading cause of this premature mortality, accounting for 40% - 50% of deaths in most cohorts [3] [4]. Metabolic syndrome (MetS) originally conceptualized as "insulin resistance syndrome" [5], encompasses central obesity, dyslipidemia, hypertension, and impaired glucose metabolism. In the general population, its prevalence ranges from 20% - 30%, varying by age, ethnicity, and diagnostic criteria [6]. In schizophrenia, however, pooled prevalence estimates from meta-analyses consistently exceed 40%, with rates as high as 50% - 60% in long-term treated patients [7] [8]. Recent global data confirm this disparity: a 2024 systematic review and meta-analysis reported a worldwide MetS prevalence of 37% - 63% in schizophrenia patients [9], while a 2025 review highlighted underlying mechanisms linking the disorder itself to metabolic dysregulation [10]. The cardiometabolic burden in schizophrenia is multifactorial. Lifestyle contributors—sedentary behavior, poor diet, high smoking rates (up to 70% - 80%), and substance use—play a prominent role [11]. Yet intrinsic biological mechanisms are increasingly evident and independent of treatment. Drug-naïve patients with first-episode schizophrenia (FES) already exhibit insulin resistance, elevated inflammatory markers (e.g., IL-6, TNF-α), and altered adipokine profiles [12] [13]. A 2024 meta-analysis of 1009 FES patients found a MetS prevalence of 13%—2.5-fold higher than matched controls—suggesting disease-related vulnerability [14]. Shared genetic architecture further bridges schizophrenia and metabolic traits: genome-wide association studies identify overlapping loci for schizophrenia and traits like body mass index and type 2 diabetes

[15] [16]. Emerging evidence frames schizophrenia as a disorder of impaired metabolic flexibility, with brain and peripheral bioenergetic deficits present from illness onset [17] [18]. Antipsychotic medications exacerbate this risk, though heterogeneity exists. Second-generation (atypical) agents, particularly clozapine and olanzapine, are strongly associated with weight gain (mean 4 - 7 kg in the first year), dyslipidemia, and glucose dysregulation via H1-histamine and 5-HT2C receptor antagonism, leptin/ghrelin disruption, and direct mitochondrial interference [19]-[21]. A 2023 network meta-analysis of 18 antipsychotics confirmed clozapine and olanzapine as highest risk, with lurasidone, aripiprazole, and ziprasidone showing minimal effects [22] [23]. Typical antipsychotics carry lower metabolic liability but higher extrapyramidal risks. Real-world studies in transitional settings, like Bosnia and Herzegovina, often compare broad classes due to limited agent diversity and monitoring resources. This study evaluated MetS and its components prevalence in schizophrenia patients on typical versus atypical antipsychotic monotherapy, using multivariate modeling to identify predictors of cardiometabolic risk.

2. Subjects and Methods

2.1. Study Design

This retrospective-prospective cohort study with a hybrid design was conducted at the Department of Psychiatry, University Clinical Center Tuzla, and the Duje Asylum Center, Doboj Istok, Bosnia and Herzegovina. The retrospective component included chart review, while metabolic parameters were collected prospectively during January 2004-January 2006. Typical antipsychotic group included both monotherapy and combination regimens, while the atypical group was predominantly monotherapy, mainly clozapine. The study adhered to the Declaration of Helsinki principles and was approved by the local Ethics Committee.

2.2. Participants

Sixty adult patients recruited consecutively (37 males, 23 females; mean age 44.5 ± 12.6 years) diagnosed with schizophrenia per ICD-10 criteria were included. Group allocation: typical antipsychotics (n = 30; predominantly haloperidol, fluphenazine, and combinations) and atypical antipsychotics (n = 30; 27 on clozapine, 2 risperidone, 1 olanzapine). All were on stable monotherapy for ≥6 months. The typical antipsychotic group included both monotherapy and combination regimens, while the atypical group was predominantly monotherapy, mainly clozapine. Antipsychotic doses ranged from 300 to 600 chlorpromazine equivalents. Atypical group included all eligible patients, while the typical group was randomly selected from a larger pool of eligible patients.

2.3. Inclusion Criteria

Age ≥ 18 years; confirmed schizophrenia diagnosis; stable antipsychotic regimen ≥ 6 months; informed consent.

2.4. Exclusion Criteria

Pre-existing diabetes mellitus; active malignancy; severe renal disease; known familial dyslipidemia.

2.5. Clinical and Laboratory Assessment

Parameters measured: waist circumference (cm), systolic/diastolic blood pressure (mmHg), fasting plasma glucose (mmol/L), triglycerides (mmol/L), HDL cholesterol (mmol/L). MetS was defined by modified WHO criteria: fasting glucose ≥ 6.1 mmol/L plus ≥ 2 of waist circumference ≥ 94 cm (males)/ ≥ 88 cm (females); dyslipidemia (triglycerides ≥ 1.7 mmol/L or HDL < 0.9 mmol/L); blood pressure ≥ 140/90 mmHg or antihypertensive use.

2.6. Statistical Analysis

Key confounders such as smoking status, illness duration, treatment duration, and living setting were not consistently available and were therefore not included in adjusted analyses). Continuous variables: mean ± SD. Categorical: percentages. Tests: Student's t-test, χ^2-test, OR with 95% CI, multivariate logistic regression (MetS as dependent; predictors: age, sex, waist circumference, treatment class). Significance: $p < 0.05$. Analyses used SPSS 26.0. This analysis should be interpreted as exploratory due to the low number of metabolic syndrome cases (n = 3), and reduced HDL cholesterol was used as the primary analytical outcome.

3. Results

Baseline metabolic parameters were compared between patients receiving typical and atypical antipsychotics. Table 1 summarizes the mean values for key anthropometric and biochemical measures, along with the prevalence of hypertension, and includes p-values from Student's t-test or χ^2-test to assess group differences. No statistically significant differences were observed across parameters, though waist circumference approached significance. Interpretation of atypical antipsychotic effects is limited due to the predominance of clozapine in this group. Key confounders such as smoking status, illness duration, treatment duration, and living setting were not consistently available and were therefore not included in adjusted analyses.

Table 1. Baseline metabolic parameters.

Parameter	Typical (n = 30)	Atypical (n = 30)	p-value
Waist circumference (cm)	96.4 ± 11.26	102.3 ± 11.94	0.053
Triglycerides (mmol/L)	2.02 ± 1.15	2.42 ± 1.52	0.26
HDL cholesterol (mmol/L)	1.16 ± 0.32	1.06 ± 0.28	0.21
Fasting glucose (mmol/L)	5.31 ± 2.22	5.07 ± 0.55	0.53
Hypertension (%)	13.3%	23.3%	0.34

Central obesity prevalence: typical 70%, atypical 83.3% (OR = 2.14; 95% CI 0.63 - 7.29). Interpretation of atypical antipsychotic effects is limited due to the predominance of clozapine in this group.

The prevalence of individual components of metabolic syndrome was evaluated in both treatment groups. Table 2 presents the percentages of patients meeting criteria for each component, highlighting a high burden of central obesity and dyslipidemia across cohorts.

Table 2. Prevalence of metabolic syndrome components.

Component	Typical (%)	Atypical (%)
Central obesity	70	83.3
Elevated triglycerides	43.3	50
Reduced HDL	36.7	46.7
Elevated fasting glucose	16.7	13.3
Hypertension	13.3	23.3

MetS prevalence: typical 10%, atypical 0%. Interpretation of atypical antipsychotic effects is limited due to the predominance of clozapine in group treated with atypical antipsychotics.

Multivariate logistic regression was performed to identify associated factors of metabolic abnormalities, with metabolic syndrome as the dependent variable. Table 3 displays the regression coefficients (β), odds ratios (OR), 95% confidence intervals (CI), and p-values for selected predictors, adjusted for age, sex, and treatment class. This analysis should be interpreted as exploratory due to the low number of metabolic syndrome cases (n = 3), and reduced HDL cholesterol was used as the primary analytical outcome.

Table 3. Multivariate logistic regression (Predictors of Metabolic Abnormalities).

Predictor	β	OR	95% CI	p
Waist circumference	0.08	1.08	1.01 - 1.16	0.02
Age	0.03	1.03	0.97 - 1.09	0.29
Male sex	0.21	1.23	0.41 - 3.67	0.71
Atypical treatment	0.56	1.75	0.38 - 7.94	0.47

Multivariate regression as an exploratory analysis suggests waist circumference as an associated factor of cardiometabolic risk, rather than drug class. The analysis is limited by the small number of cases of complete MetS.

4. Discussion

This study reveals a substantial cardiometabolic burden in schizophrenia patients, with central obesity affecting 70% - 83% and MetS components highly prevalent regardless of antipsychotic class. Notably, no patients in the atypical group met

full MetS criteria, contrasting with 10% in the typical group—a finding that challenges the conventional view of greater metabolic liability with atypicals [22] [23]. These results align with broader literature. Meta-analyses report MetS rates of 40% - 60% in schizophrenia, often driven by central adiposity [7] [9]. A 2025 review emphasized that intrinsic factors—impaired glucose tolerance and inflammation—precede antipsychotics in FES [10]. Our clozapine-heavy atypical cohort (90%) may explain the lack of difference, as clozapine's superior efficacy in treatment-resistant cases could offset some risks in select patients. Recent data from Africa and Asia confirm high MetS rates (23% - 50%) with atypicals, but also highlight lifestyle and genetic confounders [9]. Central obesity emerged as the dominant driver, independently predicting low HDL ($\beta = -0.61$, $p = 0.02$). Visceral fat promotes insulin resistance, chronic inflammation, and atherogenesis via adipokine dysregulation [24] [25]. In schizophrenia, this vulnerability is amplified by shared pathophysiology: mitochondrial dysfunction, oxidative stress, and dopamine-serotonin imbalances [26]. CVD mortality remains disproportionately high: a 2024 study reported 4-fold increased sudden cardiac death risk in schizophrenia [27], while a Swedish register analysis showed CVD as the leading cause, with 10-year earlier onset [4]. Our findings underscore that antipsychotic class alone does not dictate risk, but abdominal adiposity does so with caution in interpretation due to the predominance of clozapine in the group treated with atypical antipsychotics. The results should not be generalized to the entire class of atypical antipsychotics, and that the observed metabolic profile may reflect agent-specific effects (clozapine) rather than class effects.

Limitations of the study include modest sample size, clozapine predominance in group treated with atypical antipsychotics, cross-sectional design, and absence of baseline data or biomarkers (e.g., CRP, adiponectin). Agent-specific analyses were infeasible due to small subgroups.

5. Conclusion

Metabolic abnormalities pervade schizophrenia irrespective of antipsychotic class, with central obesity as the pivotal driver. Treatment class was not an independent predictor after adjustment. These data reinforce the need for systematic metabolic screening from illness onset, per updated guidelines [28]-[30]. Integrated care models, lifestyle interventions, and adjunctive therapies like metformin [31] [32] are critical to mitigate CVD risk and narrow the mortality gap. Early intervention targeting visceral fat may yield the greatest benefit.

Author's Contribution

All authors were involved in all steps of preparing this manuscript, including final proofreading.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

References

[1] McCutcheon, R.A., Reis Marques, T. and Howes, O.D. (2020) Schizophrenia—An Overview. *JAMA Psychiatry*, **77**, 201-210. https://doi.org/10.1001/jamapsychiatry.2019.3360

[2] Hjorthøj, C., Stürup, A.E., McGrath, J.J. and Nordentoft, M. (2017) Years of Potential Life Lost and Life Expectancy in Schizophrenia: A Systematic Review and Meta-analysis. *The Lancet Psychiatry*, **4**, 295-301. https://doi.org/10.1016/s2215-0366(17)30078-0

[3] Olfson, M., Gerhard, T., Huang, C., Crystal, S. and Stroup, T.S. (2015) Premature Mortality among Adults with Schizophrenia in the United States. *JAMA Psychiatry*, **72**, 1172-1181. https://doi.org/10.1001/jamapsychiatry.2015.1737

[4] Laursen, T.M., Nordentoft, M. and Mortensen, P.B. (2014) Excess Early Mortality in Schizophrenia. *Annual Review of Clinical Psychology*, **10**, 425-448. https://doi.org/10.1146/annurev-clinpsy-032813-153657

[5] Reaven, G.M. (1988) Banting Lecture 1988. Role of Insulin Resistance in Human Disease. *Diabetes*, **37**, 1595-1607. https://doi.org/10.2337/diabetes.37.12.1595

[6] Saklayen, M.G. (2018) The Global Epidemic of the Metabolic Syndrome. *Current Hypertension Reports*, **20**, Article No. 12. https://doi.org/10.1007/s11906-018-0812-z

[7] Vancampfort, D., Stubbs, B., Mitchell, A.J., De Hert, M., Wampers, M., Ward, P.B., *et al.* (2015) Risk of Metabolic Syndrome and Its Components in People with Schizophrenia and Related Psychotic Disorders, Bipolar Disorder and Major Depressive Disorder: A Systematic Review and Meta-Analysis. *World Psychiatry*, **14**, 339-347. https://doi.org/10.1002/wps.20252

[8] Mitchell, A.J., Vancampfort, D., Sweers, K., van Winkel, R., Yu, W. and De Hert, M. (2011) Prevalence of Metabolic Syndrome and Metabolic Abnormalities in Schizophrenia and Related Disorders—A Systematic Review and Meta-Analysis. *Schizophrenia Bulletin*, **39**, 306-318. https://doi.org/10.1093/schbul/sbr148

[9] Manta, A., Georganta, A., Roumpou, A., Zoumpourlis, V., Spandidos, D., Rizos, E., *et al.* (2025) Metabolic Syndrome in Patients with Schizophrenia: Underlying Mechanisms and Therapeutic Approaches (Review). *Molecular Medicine Reports*, **31**, 1-16. https://doi.org/10.3892/mmr.2025.13479

[10] Salari, N., Maghami, N., Ammari, T., Mosafer, H., Abdullahi, R., Rasoulpoor, S., *et al.* (2024) Global Prevalence of Metabolic Syndrome in Schizophrenia Patients: A Systematic Review and Meta-Analysis. *Journal of Prevention*, **45**, 973-986. https://doi.org/10.1007/s10935-024-00798-8

[11] Pillinger, T., Beck, K., Gobjila, C., Donocik, J.G., Jauhar, S. and Howes, O.D. (2017) Impaired Glucose Homeostasis in First-Episode Schizophrenia: A Systematic Review and Meta-Analysis. *JAMA Psychiatry*, **74**, 261-269. https://doi.org/10.1001/jamapsychiatry.2016.3803

[12] Fernandes, B.S., Steiner, J., Bernstein, H.G., Dodd, S., Pasco, J.A., Dean, O.M., *et al.* (2016) C-Reactive Protein in Schizophrenia: A Review. *Molecular Psychiatry*, **21**, 431-440. https://doi.org/10.1038/mp.2015.87

[13] Garrido-Torres, N., Ruiz-Veguilla, M., Alameda, L,. Rodriguez, V., Gómez-Pablos, M., Alameda, L., *et al.* (2021) Prevalence of Metabolic Syndrome in First-Episode Psychosis. *Schizophrenia Research*, **232**, 1-8.

[14] So, H.C., Chau, C.K., Chiu, W.T., Ho, K.S., Lo, C.P., Yim, S.H., *et al.* (2019) Genetic Overlap between Schizophrenia and Metabolic Traits. *Translational Psychiatry*, **9**, Article No. 298.

[15] Hackinger, S., Prins, B., Mamakou, V., Zengini, E., Marouli, E., Brčić, L., *et al.* (2018) Evidence for Genetic Contribution to the Increased Risk of Type 2 Diabetes in Schizophrenia. *Translational Psychiatry*, **8**, Article No. 252. https://doi.org/10.1038/s41398-018-0304-6

[16] Sarnyai, Z. and Ben-Shachar, D. (2024) Schizophrenia, a Disease of Impaired Dynamic Metabolic Flexibility: A New Mechanistic Framework. *Psychiatry Research*, **342**, Article ID: 116220. https://doi.org/10.1016/j.psychres.2024.116220

[17] Cuesta, M.J., Papiol, S., Giné-Servén, E., Ballesteros, A., Gil-Berrozpe, G.J., Peralta, D., *et al.* (2025) Can Polygenic Risk Scores for Metabolic Syndrome Contribute to Long-Term Syndrome Development in First-Episode Psychosis Patients? A Naturalistic 21-Year Follow-Up Study. *Schizophrenia Bulletin*. https://doi.org/10.1093/schbul/sbaf158

[18] Newcomer, J.W. and Haupt, D.W. (2006) The Metabolic Effects of Antipsychotic Medications. *The Canadian Journal of Psychiatry*, **51**, 480-491. https://doi.org/10.1177/070674370605100803

[19] McIntyre, R.S., Mancini, D.A. and Basile, V.S. (2018) Mechanisms of Antipsychotic-Induced Weight Gain. *The Journal of Clinical Psychiatry*, **62**, 23-29.

[20] Holt, R.I. and Mitchell, A.J. (2021) Antipsychotics and Diabetes. *Diabetes Medications*, **38**, e14532.

[21] Pillinger, T., McCutcheon, R.A., Vano, L., Mizuno, Y., Arumuham, A., Hindley, G., *et al.* (2020) Comparative Effects of 18 Antipsychotics on Metabolic Function in Patients with Schizophrenia, Predictors of Metabolic Dysregulation, and Association with Psychopathology: A Systematic Review and Network Meta-Analysis. *The Lancet Psychiatry*, **7**, 64-77. https://doi.org/10.1016/s2215-0366(19)30416-x

[22] Huhn, M., Nikolakopoulou, A., Schneider-Thoma, J., Krause, M., Samara, M., Peter, N., *et al.* (2019) 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. *The Lancet*, **394**, 939-951. https://doi.org/10.1016/s0140-6736(19)31135-3

[23] Correll, C.U., Manu, P., Olshanskiy, V., Napolitano, B., Kane, J.M. and Malhotra, A.K. (2009) Cardiometabolic Risk of Second-Generation Antipsychotic Medications during First-Time Use in Children and Adolescents. *JAMA*, **302**, 1765-1773. https://doi.org/10.1001/jama.2009.1549

[24] Després, J. (2012) Body Fat Distribution and Risk of Cardiovascular Disease: An Update. *Circulation*, **126**, 1301-1313. https://doi.org/10.1161/circulationaha.111.067264

[25] Shirai, K. (2004) Obesity as the Core of the Metabolic Syndrome and the Management of Coronary Heart Disease. *Current Medical Research and Opinion*, **20**, 295-304. https://doi.org/10.1185/030079903125003008

[26] Khandaker, G.M., Cousins, L., Deakin, J., Lennox, B.R., Yolken, R. and Jones, P.B. (2015) Inflammation and Immunity in Schizophrenia: Implications for Pathophysiology and Treatment. *The Lancet Psychiatry*, **2**, 258-270. https://doi.org/10.1016/S2215-0366(14)00122-9

[27] Mujkanovic, J., Warming, P.E., Kessing, L.V., Køber, L.V., Winkel, B.G., Lynge, T.H., *et al.* (2024) Nationwide Burden of Sudden Cardiac Death among Patients with a Psychiatric Disorder. *Heart*, **110**, 1365-1371. https://doi.org/10.1136/heartjnl-2024-324092

[28] De Hert, M., Correll, C.U., Bobes, J., Cetkovich-Bakmas, M., Cohen, D., Asai, I., *et al.* (2011) Physical Illness in Patients with Severe Mental Disorders. I. Prevalence, Im-

pact of Medications and Disparities in Health Care. *World Psychiatry*, **10**, 52-77. https://doi.org/10.1002/j.2051-5545.2011.tb00014.x

[29] National Institute for Health and Care Excellence (2023) Psychosis and Schizophrenia: Exceptional Surveillance. NICE. https://www.nice.org.uk/

[30] U.S. Department of Veterans Affairs/Department of Defense (2023) Clinical Practice Guideline for the Management of First-Episode Psychosis and Schizophrenia. U.S. Government Publishing Office. https://www.healthquality.va.gov/guidelines/MH/scz/

[31] Zheng, W., Li, X.B., Tang, Y.L., Xiang, Y.Q., Wang, C.Y. and de Leon, J. (2015) Metformin for Weight Gain and Metabolic Abnormalities Associated with Antipsychotic Treatment: Meta-Analysis of Randomized Plac*ebo-Controlled Trials. Journal of Clinical Psychopharmacology*, **35**, 499-509. https://doi.org/10.1097/JCP.0000000000000392

[32] Carolan, A., Hynes-Ryan, C., Agarwal, S.M., Bourke, R., Cullen, W., Gaughran, F., *et al.* (2024) Metformin for the Prevention of Antipsychotic-Induced Weight Gain: Guideline Development and Consensus Validation. *Schizophrenia Bulletin*, **51**, 1193-1205. https://doi.org/10.1093/schbul/sbae205