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The Prevalence of the Metabolic Syndrome in Ghanaian Psychiatric Patients on Antipsychotic (First versus Second Generation) Treatment in the Kumasi Metropolis

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Abstract
Metabolic syndrome (MetS), a predominant public health problem linked to cardiovascular and other morbidities, has acquired a significant precedence in clinical settings and patients with severe mental illnesses who are at higher risk for deviant components of this syndrome due to their illness and its treatment require careful and regular monitoring in this regard. Even though MetS has been established to be more prevalent among psychiatric patients than among any other population group, no data exist on its prevalence in Ghanaian psychiatric patients. This study seeks to find the prevalence of the MetS, in Ghanaian psychiatric patients on antipsychotics (first or second generation) compared to newly diagnosed psychiatric patients. This cross-sectional study of patients attending psychiatric department of the Komfo Anokye Teaching Hospital (KATH) in Kumasi, Ghana between February 2009 and July 2010. A total of 200 psychiatric patients comprising 100 newly diagnosed antipsychotic naïve patients and 100 patients on antipsychotic medication were sampled for the study. Prevalence of MetS diagnosed using the World Health Organization (WHO), International Diabetes Federation (IDF) and the National Cholesterol Education Programme, Adult Treatment Panel III (NCEP ATP III) criteria for defining MetS was employed.The prevalence was significantly higher among psychiatric patients on treatment in comparison with the treatment naïve group using NCEP ATP III (21.0% vs. 2.0%; $p < 0.0001$) and IDF (29.0% vs. 2.0%; $p < 0.0001$) criteria but not WHO (13.0% vs. 14.0%; $p = 0.8372$). Irrespective of the criteria used, the prevalence of MetS was higher among patients on second generation versus first generation antipsychotic medication (i.e. 44.4% vs. 18.7% for NCEP ATP III; 22.2% vs. 12.1% for WHO and 56.6% vs. 27.5% for IDF), however these did not reach a significant levelPrevalence of MetS was not only highly prevalent among Ghanaian patients treated with antipsychotic drugs, it was also higher among patients on second generation versus first generation antipsychotic medication.
Keywords: Diabetes, hypertension, dyslipidaemia, mental illness, antipsychotics.

1. Introduction

The designation of the combination of abnormalities which elevate the cardiovascular disease risk (CVD) leads to a multiple human systems disorder known as metabolic syndrome (MetS). Alterations in glucose homeostasis and metabolism, obesity, hyperlipidaemia and hypertension are some of the abnormalities (Holt et al., 2004; Lieberman, 2004; Toalson et al., 2004; Meyer et al., 2005; Blaha and Elasy, 2006). Sedentary lifestyles such as poor diet, decreased physical activity and smoking as well as decreased help-seeking (Phelan et al., 2001) are partial justifications as to why cardiovascular disease risk factors as well as rates of physical disorders in the psychiatric population are high . It has been deduced that mortality rates are about 2 to 3 times in psychiatric patients compared to the general population (Lesage et al., 1990; Brown et al., 2000). A research on the risk of coronary heart disease (CHD) and stroke in addition to lifestyle factors in psychiatric patients, McCreadie, (2003) noted an elevated (9.6%) mean 10-year risk of coronary heart disease compared to the general population (6.4%) as was the risk of stroke (4.1%).

Progressively, there has been an upsurging concern about the input of antipsychotic medication to the predominance of metabolic syndrome and its components especially since the introduction of the second generation (atypical) antipsychotic medications in a population with increased physical morbidity in comparison with the general population. An up rise in the prevalence of increase in weight, glucose intolerance and hyperlipidaemia and in a few cases, hypertension in subsequent antipsychotic use in psychiatric patients has been shown in current analysis. It was expressed by Heiskanen et al., (2003) that the metabolic syndrome was interpreted in 13 (37%) out of 35 patients with schizophrenia managed with antipsychotic medication and

Mackin et al., (2007) deduced elevated metabolic syndrome predominance and cardiovascular risk in 90 people managed with antipsychotics, compared to 92 age and gender matched controls. Correl et al., (2006) also disclosed that metabolic syndrome was in existence in 137 (37.3%) out of 367 adults treated with second generation antipsychotics and was significantly correlated with the 10-year risk of Coronary Heart disease (CHD) events.

The treatment basics of psychiatric disorder are with atypical antipsychotics. However their potency as compared to conventional (first generation) antipsychotics is being intensely debated and has been the subject of much fact-finding activity. The meta-analysis by Davis et al. (2003) hinted that some of the atypical antipsychotics (clozapine, amisulpride, risperidone and olanzapine) were more potent than conventional neuroleptics, but more current research indicate otherwise. Lieberman et al. (2005) pointed out that the second generation antipsychotic, olanzapine, was most adequate when rates of discontinuation were considered (but was also correlated with the most weight gain and dyslipidemia) and the conventional antipsychotic perphenazine was of similar potency to quetiapine, risperidone and ziprasidone in a Clinical Antipsychotic Trial of Intervention Effectiveness. According to McEvoy et al. (2006), clozapine was found to be more potent than the other atypical antipsychotics in the Phase 2 of the same study. Jones et al. (2006) established that atypical antipsychotic drugs did not present any significant advantage over the conventional antipsychotics use in terms of cost.

There has currently been substantial interest and concern about the metabolic abnormalities interrelated with atypical antipsychotic use (American Diabetes Association, 2004) alongside the potency debate. In discussion is whether these metabolic abnormalities only occur with management with antipsychotics, if there is dissimilarity between atypical and conventional antipsychotics in terms of these reactions and about the conflicting metabolic profiles of the various atypical antipsychotics.

2. Materials and Methods

2.1 Study population and setting

This cross-sectional study was carried out at the Psychiatric department of the Komfo Anokye Teaching Hospital, (KATH), Kumasi, Ghana. Patients attending the psychiatric department between February 2009 and July 2010 were recruited. A total of 200 psychiatric patients comprising 100 newly diagnosed antipsychotic naïve patients and 100 patients on antipsychotic medication were invited through a written informed consent to participate in the study.

2.2 Sampling

About 5 ml of venous blood sample was collected from the antecubital fossa of the study participants after an overnight fast (12 – 16 hours). One (1 ml) of the blood sample was dispensed into fluoride oxalate tube and the other 4 ml into vacutainer plain tubes. Serum and plasma were stored at -80^{0}C after centrifugation at 2000rpm for 15 minutes until assay was performed. Assay parameters include: fasting blood glucose (FGB), total cholesterol (TC), triglycerides (TG), high density lipoprotein (HDL) cholesterol and uric acid. Serum low density lipoprotein (LDL) cholesterol was estimated with the Friedewald equation (Friedewald et al., 1972). Reagent manufacturer's protocol was adopted for the estimation of analytes.

2.3 Anthropometric variables

Height to the nearest centimetre without shoes was measured against a wall-mounted ruler and weight to the nearest 0.1 kg in light clothing on a bathroom scale (Zhongshan Camry Electronics Co. Ltd. Guangdong, China). The body mass index (BMI) was calculated by dividing weight (kg) over the height squared (m2). Waist circumference (to the nearest centimetre) was measured with a Gulick II spring-loaded measuring tape (Gay Mill, WI) midway between the inferior angle of the ribs and the suprailiac crest. Hip circumference was measured as the maximal circumference over the buttocks in metres and the waist to hip ratio (WHR) calculated by dividing the waist circumference (m) by the hip circumference (m).

2.4 Statistical Analysis

Results are presented as Means ± SEM. Unpaired t-test was used to compare the means of all continuous variables. The Chi-square test statistic was used to assess the statistical significance of categorical variables. Odds analysis and confidence intervals of metabolic syndrome was done using the Odds ratio test statistic. Logistic regression test statistic was used to estimate the crude (c) and adjusted (adj) odds ratio (OR) for risk factors of metabolic syndrome. A p-value < 0.05 was considered to be statistically significant. All statistical analyses were performed using MedCalc® version 10.2.0.0 (www.medcalc.be) for windows.

3. Results and Discussion

The results of patients on treatment further classified into two categories, conventional and atypical based on the

composition of drug regimen are presented in Table 1. The mean age, weight, waist circumference, waist to hip ratio, body mass index and low density lipoprotein (LDL) values in the conventional treatment group were not significantly different from that in the atypical treatment group ($p > 0.05$). The mean systolic pressure (141.7 ± 8.3 mmHg), diastolic blood pressure (87.5 ± 4.5 mmHg) and mean fasting blood glucose concentration (7.2 ± 0.8 mmol L-1) in the atypical treatment group were significantly higher than in the conventional treatment group. Likewise, the lipid profile components of total cholesterol (TC) (5.9 ± 0.3 mmol L-1), triglycerides (TG) (2.1 ± 0.3 mmol L-1), HDL-C (1.5 ± 0.1 mmol L-1) and very low density lipoprotein (0.4 ± 0.1 mmol L-1) were significantly higher in the atypical treatment group compared to the conventional treatment group. On the contrary, the mean uric acid concentration in the conventional group (257.0 ± 8.2 µmol L-1) was significantly higher than the mean concentration in the atypical group.

In Figure 1 (Fig 1), when classified by the NCEP ATP III criteria, the prevalence of metabolic syndrome in the atypical treatment group was 44.4% compared to 18.7% in the conventional treatment group but the difference was not statistically significant ($p = 0.0703$). Likewise when classified by the WHO criteria, no significant differences in the prevalence of metabolic syndrome was observed in the conventional and atypical treatment groups (0.3198). The IDF criteria showed the same trend with a metabolic syndrome prevalence of 55.6% and 27.5% in the atypical and conventional treatment groups respectively but the difference was not statistically significant.

Table 2. gives the general overview of the prevalence of metabolic syndrome and its components as assessed with the NCEP ATP III criteria based on specific drugs administered to the patients on treatment. Out of the Sixty three (63) patients who were on a monotherapy regimen of conventional antipsychotics, 33.3% were on chlorpromazine, 20.6% were on trifluoperazine, 23.8% were on haloperidol and 6.3% were on fluphenazine. 11.1% on the antidepressant, amitriptyline and 4.8% were on the mood stabilizer, carbamazepine. The highest prevalence of metabolic syndrome (28.6%) was observed in patients on amitriptyline with raised triglyceride concentration being the most prevalent component (57.1%) of metabolic syndrome associated with amitriptyline usage. None of the patients on carbamazepine had metabolic syndrome (0.0%) but raised blood pressure was the most prevalent component (66.7%) linked with carbamazepine use. All the other monotherapy antipsychotics gave varying prevalence of metabolic syndrome with varying prevalence in the components of metabolic syndrome. Trifluoperazine and haloperidol were associated with raised waist circumference; fluphenazine with raised waist circumference, raised triglyceride, raised blood pressure and raised fasting blood glucose and chlopromazine with raised fasting blood glucose.

The prevalence of metabolic syndrome in patients on a polytherapy regimen of conventional antipsychotics was 25% with raised waist circumference being the most prevalent component of metabolic syndrome (35.7%). The highest prevalence of metabolic syndrome in patients on monotherapy of atypical antipsychotic drugs was observed in patients on risperidone (66.7%) with increased triglycerides and raised fasting blood glucose being the prevalent components of metabolic syndrome.

In Table 3, the prevalence of metabolic syndrome and its components was assessed using WHO criteria. The highest prevalence of metabolic syndrome in patients on monotherapy of conventional antipsychotics was seen in patients on fluphenazine (25.0%). With patients on polytherapy of conventional antipsychotics, the prevalence of metabolic syndrome was 10.7% with raised triglyceride concentration being the most prevalent component (28.6%). With patients on monotherapy of atypical antipsychotics, risperidone gave the highest prevalence of metabolic syndrome (33.3%) with raised triglyceride and raised fasting blood glucose levels being the most prevalent components of metabolic syndrome.

In using the IDF criteria, trifluoperazine gave the highest prevalence of metabolic syndrome (53.8%) among the monotherapy conventional antipsychotics with increased waist circumference being the most prevalent component of metabolic syndrome (69.2%). None of the patients on carbamazepine had metabolic syndrome and increased waist circumference and raised blood pressure were the most prevalent components of metabolic syndrome linked with the use of carbamazepine. Twenty five (25%) of the patients on polytherapy of conventional antipsychotics had metabolic syndrome with increased waist circumference being the most prevalent component of metabolic syndrome. Risperidone still turned out to be the atypical antipsychotic with the highest prevalence of metabolic syndrome (66.7%) with increased triglyceride concentration being the most prevalent component of metabolic syndrome associated with risperidone usage (Table 4).

After patients on treatment were classified into drug type (conventional and atypical), patients on atypical antipsychotics had a higher prevalence of metabolic syndrome compared to those on conventional antipsychotics. The predominance of metabolic syndrome determined with the NCEP ATP III and IDF criteria in patients on atypical antipsychotics was 44.4% and 55.6% respectively while the WHO criteria gave a predominance of 22.2%. Apart from the WHO criteria which gave prevalence rates within the range of what has been calculated in the general population, the NCEP ATP III and IDF criteria gave prevalence rates almost twice what has been calculated in the general population showing them to be an increased risk group for metabolic syndrome and its consequences.

Case reports have transpired which points to increased levels of hyperglycaemia and diabetes mellitus corelated with the use of atypical antipsychotics. Lindenmayer & Patel (1999) noted a case of olanzapine-induced diabetic ketoacidosis (DKA), which solved with substantial cessation of olanzapine treatment and further deliberated on the role of olanzapine in restraining insulin release and in generating a hyperglycaemic response. The mean fasting glycemic level in patients on atypical antipsychotics in this research was significantly higher in comparison with those on conventional antipsychotics. A high prevalence of elevated fasting blood glucose was associated with Risperidone use in comparison with olanzapine use in this study. Contrariwise, Koro et al., (2002b) in a large population based nested case-control study found olanzapine to be involved with a significant risk of diabetes compared with Risperidone.

Increase in weight, especially visceral adiposity as measured by waist circumference is one of the major components of metabolic syndrome and is the main criterion in the IDF definition. Reports on weight change in psychiatric patients during the course of a psychotic illness (Kraepelin, 1919) have raised significant interest in weight increase involved with the use of atypical antipsychotic drugs. Zhang et al., (2004) researched drug naïve Chinese psychiatric in-patients before and following 10 weeks of antipsychotic treatment by comparing them to well matched healthy controls. After 10 weeks of treatment the patient group demonstrated significant rise in abdominal subcutaneous fat and intra-abdominal fat, plasma leptin levels, plasma glucose levels and plasma lipid levels. Interestingly, no significant difference was found amidst Risperidone and Chlorpromazine and no significant interrelation was detected between change in BMI and clinical improvement. Similarly in this study, there were no significant differences in weight, waist circumference and BMI in patients on atypical antipsychotics compared to conventional antipsychotics. Upon adjusting for age in a logistic regression, however, waist circumference defined by the NCEP ATP III, IDF and WHO criteria and BMI were significant predictors of metabolic syndrome displaying the ability of atypical and conventional antipsychotics to have an almost equal tendency in inducing increase in weight in patients on medication.

Dyslipidaemia is an essential element of the metabolic syndrome and develops alongside with glucose dysregulation and weight gain in patients managed with atypical antipsychotics. In the North Finland 1966 Birth Cohort, subjects treated with antipsychotics, both conventional and atypical were found to have elevated lipid levels. A total of 8463 subjects from the original cohort were participants in this study. Out of 5654 (67%) of the total number who participated, 45 subjects were receiving antipsychotic treatment. 32 subjects (71%) were on conventional, 6 subjects (13%) were on atypical and 7 subjects (16%) were on both conventional and atypical antipsychotic treatment. The research found high predominance of total cholesterol and triglycerides in the 45 subjects treated with antipsychotics as compared to the 5609 who were not, even after adjusting for risk factors for hyperlipidaemia. Contrariwise to this finding, this study found significantly increased total cholesterol, triglyceride, HDL-cholesterol, LDL-cholesterol and VLDL in patients on atypical antipsychotics in comparison with those on conventional antipsychotics. A logistic regression analysis with adjustment for age also showed hypertriglyceridaemia and reduced HDL-cholesterol being significant risk factors for metabolic syndrome. Substantial case reports of increased lipids associated with antipsychotic treatment, Koro et al., (2002a) explored the interrelation using the General Practice Research Database (GPRD) and found patients managed with atypical antipsychotics being almost 3 times at risk of developing hyperlipidaemia in comparison with patients treated with conventional antipsychotics which is in agreement with the findings in this study. Tarricone et al. (2006) compared the prevalence of hyperglycaemia, hypercholesterolaemia and hypertriglyceridaemia and observed that patients treated with atypical antipsychotics had a significant predominance of hyperglycaemia and hypertriglyceridaemia compared to controls. Saari et al., (2004) in their research suggested that elevated lipids impair glucose metabolism resulting in hyperglycaemia and type 2 diabetes mellitus. This analysis could therefore explain the significant analysis of diabetes observed in patients on atypical antipsychotics compared to those on conventional antipsychotics.

Hypertension or high blood pressure is one of the risk factors of metabolic syndrome which has not been commonly correlated with treatment with atypical antipsychotics in general literature. In a retrospective chart review of 208 patients suffering from schizophrenia and treated with antipsychotics (conventional and atypical), Gupta et al., (2003) deduced elevated diabetes prevalence (17%), hypertension (29%) and hypertriglyceridaemia (44%). Contrariwise to those findings, patients on atypical antipsychotics in this study had significantly high systolic and diastolic blood pressure when compared to those on conventional antipsychotics. When blood pressure was analyzed as a risk component of metabolic syndrome, the prevalence of high blood pressure as determined by the NCEP ATP III, WHO and IDF criteria was twice higher in patients on atypical antipsychotics compared to those on conventional antipsychotics.

4. Conclusion

Risk factors for metabolic syndrome development in this research were hypertriglyceridemia, low HDL-cholesterol and high blood pressure.

Antipsychotic drugs that led to metabolic syndrome development in this study according to the ATP III,

WHO and the IDF definition criteria for metabolic syndrome were Amitryptyline (28.6%), Fluphenazine (33.3%) and Trifluoperazine (53.8%) respectively.

The use of antipsychotic medication, especially atypical antipsychotics should be re-evaluated with the knowledge that they can induce significant metabolic abnormalities and metabolic syndrome in psychiatric patients.

References

American Diabetes Association (2004) Consensus development conference on antipsychotic drugs and obesity and diabetes. Diabetes Care 27, 596-601.

Blaha M. and Elasy T.A. (2006) Clinical Use of the Metabolic Syndrome: Why the Confusion? Clinical Diabetes 24, 125-131.

Brown S., Inskip H. and Barraclough B. (2000) Causes of the excess mortality of schizophrenia. Br J Psychiatry 177, 212-217.

Correll C.U., Frederickson A.M., Kane J.M. and Manu P. (2006) Metabolic syndrome and the risk of coronary heart disease in 367 patients treated with second-generation antipsychotic drugs. J Clin Psychiatry 67, 575-583.

Davis J.M., Chen N. and Glick I.D. (2003) A meta-analysis of the efficacy of second-generation antipsychotics. Arch Gen Psychiatry 60, 553-564.

Friedewald W.T., Levy R.I. and Fredrickson D.S. (1972) Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. Clin Chem 18, 499-502.

Gupta S., Steinmeyer C., Frank B., Madhusoodanan S., Lockwood K., Lentz B. and Keller P. (2003) Hyperglycemia and hypertriglyceridemia in real world patients on antipsychotic therapy. Am J Ther 10, 348-355.

Heiskanen T., Niskanen L., Lyytikainen R., Saarinen P.I. and Hintikka J. (2003) Metabolic syndrome in patients with schizophrenia. J Clin Psychiatry 64, 575-579.

Holt R.I., Peveler R.C. and Byrne C.D. (2004) Schizophrenia, the metabolic syndrome and diabetes. Diabet Med 21, 515-523.

Jones P.B., Barnes T.R., Davies L., Dunn G., Lloyd H., Hayhurst K.P., Murray R.M., Markwick A. and Lewis S.W. (2006) Randomized controlled trial of the effect on Quality of Life of second- vs first-generation antipsychotic drugs in schizophrenia: Cost Utility of the Latest Antipsychotic Drugs in Schizophrenia Study (CUtLASS 1). Arch Gen Psychiatry 63, 1079-1087.

Koro C.E., Fedder D.O., L'Italien G.J., Weiss S., Magder L.S., Kreyenbuhl J., Revicki D. and Buchanan R.W. (2002a) An assessment of the independent effects of olanzapine and risperidone exposure on the risk of hyperlipidemia in schizophrenic patients. Arch Gen Psychiatry 59, 1021-1026.

Koro C.E., Fedder D.O., L'Italien G.J., Weiss S.S., Magder L.S., Kreyenbuhl J., Revicki D.A. and Buchanan R.W. (2002b) Assessment of independent effect of olanzapine and risperidone on risk of diabetes among patients with schizophrenia: population based nested case-control study. BMJ 325, 243.

Kraepelin E. (1919) Dementia praecox and paraphrenia. Chicago Chicago Medical Book Co.

Lesage A.D., Trapani V. and Tansella M. (1990) Excess mortality by natural causes of Italian schizophrenic patients. Eur Arch Psychiatry Neurol Sci 239, 361-365.

Lieberman J.A., 3rd (2004) Metabolic changes associated with antipsychotic use. Prim Care Companion J Clin Psychiatry 6, 8-13.

Lieberman J.A., Stroup T.S., McEvoy J.P., Swartz M.S., Rosenheck R.A., Perkins D.O., Keefe R.S., Davis S.M., Davis C.E., Lebowitz B.D., Severe J. and Hsiao J.K. (2005) Effectiveness of antipsychotic drugs in patients with chronic schizophrenia. N Engl J Med 353, 1209-1223.

Lindenmayer J.P. and Patel R. (1999) Olanzapine-induced ketoacidosis with diabetes mellitus. Am J Psychiatry 156, 1471

Mackin P., Bishop D., Watkinson H., Gallagher P. and Ferrier I.N. (2007) Metabolic disease and cardiovascular risk in people treated with antipsychotics in the community. Br J Psychiatry 191, 23-29

McCreadie R.G. (2003) Diet, smoking and cardiovascular risk in people with schizophrenia: descriptive study. Br J Psychiatry 183, 534-539.

McEvoy J.P., Lieberman J.A., Stroup T.S., Davis S.M., Meltzer H.Y., Rosenheck R.A., Swartz M.S., Perkins D.O., Keefe R.S., Davis C.E., Severe J. and Hsiao J.K. (2006) Effectiveness of clozapine versus olanzapine, quetiapine, and risperidone in patients with chronic schizophrenia who did not respond to prior atypical antipsychotic treatment. Am J Psychiatry 163, 600-610.

Meyer G.F., Wuerger S.M., Rohrbein F. and Zetzsche C. (2005) Low-level integration of auditory and visual motion signals requires spatial co-localisation. Exp Brain Res 166, 538-547.

Phelan M., Stradins L. and Morrison S. (2001) Physical health of people with severe mental illness. BMJ 322, 443-444.

Saari K., Koponen H., Laitinen J., Jokelainen J., Lauren L., Isohanni M. and Lindeman S. (2004) Hyperlipidemia in persons using antipsychotic medication: a general population-based birth cohort study. J Clin Psychiatry 65, 547-550.

Tarricone I., Casoria M., Gozzi B.F., Grieco D., Menchetti M., Serretti A., Ujkaj M., Pastorelli F. and Berardi D. (2006) Metabolic risk factor profile associated with use of second generation antipsychotics: a cross sectional study in a Community Mental Health Centre. BMC Psychiatry 6, 11.

Toalson P., Ahmed S., Hardy T. and Kabinoff G. (2004) The Metabolic Syndrome in Patients With Severe Mental Illnesses. Prim Care Companion J Clin Psychiatry 6, 152-158.

Zhang Z.J., Yao Z.J., Liu W., Fang Q. and Reynolds G.P. (2004) Effects of antipsychotics on fat deposition and changes in leptin and insulin levels. Magnetic resonance imaging study of previously untreated people with schizophrenia. Br J Psychiatry 184, 58-62.

Table 1. General characteristics of the study population stratified by type of treatment

Variables	Conventional (n = 91)	Atypical (n = 9)	P value
Age (yrs)	37.5 ± 1.4	41.7 ± 5.4	0.3817
WT (kg)	65.9 ± 1.5	67.7 ± 3.3	0.7181
WC (cm)	85.7 ± 1.3	86.1 ± 4.0	0.9211
HC (cm)	100.0 ± 1.5	104.0 ± 2.6	0.4131
WHR	1.4 ± 0.5	0.8 ± 0.0	0.7451
BMI (kg m^{-2})	24.8 ± 0.6	24.0 ± 1.2	0.6743
SBP (mmHg)	124.6 ± 2.1	141.7 ± 8.3	0.0420
DBP (mmHg)	79.1 ± 0.9	87.5 ± 4.5	0.0199
FBS (mmol L^{-1})	5.4 ± 0.3	7.2 ± 0.8	0.0541
UA (µmol L^{-1})	257.0 ± 8.2	195.8 ± 15.7	0.0441
TC(mmol L^{-1})	4.6 ± 0.1	5.9 ± 0.3	0.0018
TG(mmol L^{-1})	1.3 ± 0.1	2.1 ± 0.3	0.0011
HDL-C (mmol L^{-1})	1.2 ± 0.0	1.5 ± 0.1	0.0028
LDL-C (mmol L^{-1})	2.8 ± 0.1	3.4 ± 0.2	0.0711
VLDL(mmol L^{-1})	0.3 ± 0.0	0.4 ± 0.1	0.0024

Results are presented as mean ± SEM. P value (p<0.05) defines the level of significance when study population on treatment was compared to the newly diagnosed. WC = waist circumference, HC = hip circumference, WHR = waist to hip ratio, BMI = body mass index, SBP = systolic blood pressure, DBP = diastolic blood pressure, FBS = fasting blood glucose, TC = total cholesterol, HDL-C = high density lipoprotein cholesterol, LDL-C = low density lipoprotein cholesterol, VLDL = very low density lipoprotein.

Table 2. Prevalence of metabolic syndrome and components of metabolic syndrome defined by the NCEP ATP III criteria in the study population stratified by drugs.

National Cholesterol Education Programme Adult Treatment Panel III Criteria							
Variables	**n**	**MetS(%)**	**↑WC (%)**	**↑TG (%)**	**↓ HDL-C (%)**	**↑ BP (%)**	**↑ FBS (%)**
Typical AP (Monotherapy)							
TRIFLUOPERAZINE	13	2(15.4)	6 (46.2)	2 (15.4)	4 (30.8)	2 (15.4)	4 (30.8)
HALOPERIDOL	15	3(20.0)	6 (40.0)	5 (33.0)	2 (13.0)	2 (13.0)	5 (33.0)
FLUPHENAZINE	4	1(25.0)	1 (25.0)	1 (25.0)	0 (0.0)	1 (25.0)	1 (25.0)
CHLORPROMAZINE	21	2(9.5)	3 (14.3)	2 (9.5)	4 (19.0)	4 (19.0)	5 (23.8)
CARBAMAZEPINE	3	0(0.0)	1 (33.3)	0 (0.0)	0 (0.0)	2 (66.7)	0 (0.0)
AMITRIPTYLINE	7	2(28.6)	1 (14.3)	4 (57.1)	2 (28.6)	1 (14.3)	3 (42.9)
Typical AP (Polytherapy)	28	7(25.0)	10 (35.7)	8 (28.6)	5 (17.9)	7 (25.0)	6 (21.4)
Atypical AP (Monotherapy)							
OLANZAPINE	6	2(33.3)	2 (33.3)	3 (50.0)	0 (0.0)	3 (50.0)	3 (50.0)
RISPERIDONE	3	2(66.7)	2 (66.7)	3 (100.0)	0 (0.0)	1 (33.3)	3 (100.0)

AP = anti-psychotic, MetS = Metabolic syndrome, WC = waist circumference, TG = triglycerides, HDL-C = high density lipoprotein cholesterol, BP = blood pressure, FBS = fasting blood glucose

Table 3. Prevalence of metabolic syndrome and components of metabolic syndrome defined by the WHO criteria in the study population stratified by drugs

World Health Organization Criteria							
Variables	**n**	**MetS(%)**	**↑WC (%)**	**↑TG (%)**	**↓ HDL-C (%)**	**↑ BP (%)**	**↑ FBS (%)**
Typical AP (Monotherapy)							
TRIFLUOPERAZINE	13	2(15.4)	7 (53.8)	2 (15.4)	4 (30.8)	2 (15.4)	4 (30.8)
HALOPERIDOL	15	3(20.0)	7(47.0)	5 (33.0)	2 (13.0)	2 (13.0)	5 (33.0)
FLUPHENAZINE	4	1(25.0)	1 (25.0)	1 (25.0)	0 (0.0)	1 (25.0)	1 (25.0)
CHLORPROMAZINE	21	2(9.5)	7 (33.3)	2 (9.5)	4 (19.0)	3 (14.3)	5 (23.8)
CARBAMAZEPINE	3	0(0.0)	2 (66.7)	0 (0.0)	0 (0.0)	1 (33.3)	0 (0.0)
AMITRIPTYLINE	7	1(14.3)	1 (14.3)	4 (57.1)	2 (28.6)	0 (0.0)	3 (42.9)
Typical AP (Polytherapy)	28	3(10.7)	7 (25.0)	8 (28.6)	5 (17.9)	5 (17.9)	6 (21.4)
Atypical AP (Monotherapy)							
OLANZAPINE	6	1(16.7)	0 (0.0)	3 (50.0)	0 (0.0)	2 (33.3)	3 (50.0)
RISPERIDONE	3	1(33.3)	1 (33.3)	3 (100.0)	0 (0.0)	1 (33.3)	3 (100.0)

AP = anti-psychotic, MetS = Metabolic syndrome, WC = waist circumference, TG = triglycerides, HDL-C = high density lipoprotein cholesterol, BP = blood pressure, FBS = fasting blood glucose

Table 4. Prevalence of metabolic syndrome and components of metabolic syndrome defined by the IDF criteria in the study population stratified by drugs

International Diabetic Federation Criteria							
Variables	**n**	**MetS(%)**	**↑WC (%)**	**↑TG (%)**	**↓ HDL-C (%)**	**↑ BP (%)**	**↑ FBS (%)**
Typical AP (Monotherapy)							
TRIFLUOPERAZINE	13	7(53.8)	9(69.2)	2(15.4)	7(53.8)	2(15.4)	5(38.5)
HALOPERIDOL	15	5(33.0)	7(47.0)	6(40.0)	8(53.0)	2(13.0)	5(33.0)
FLUPHENAZINE	4	1(25.0)	2(50.0)	1(25.0)	1(25.0)	1(25.0)	1(25.0)
CHLORPROMAZINE	21	4(19.0)	7(33.3)	2(9.5)	13(61.9)	4(19.0)	5(23.8)
CARBAMAZEPINE	3	0(0.0)	2(66.7)	0(0.0)	1(33.3)	2(66.7)	0(0.0)
AMITRIPTYLINE	7	1(14.3)	2(28.6)	4(57.1)	4(57.1)	1(14.3)	3(42.9)
Typical AP (Polytherapy)	28	7(25.0)	13(46.4)	8(28.6)	12(42.9)	7(25.0)	7(25.0)
Atypical AP (Monotherapy)							
OLANZAPINE	6	2(33.3)	3(50.0)	3(50.0)	2(33.3)	3(50.0)	3(50.0)
RISPERIDONE	3	2(66.7)	2(66.7)	3(100.0)	0(0.0)	1(33.3)	2(66.7)

AP = anti-psychotic, MetS = Metabolic syndrome, WC = waist circumference, TG = triglycerides, HDL-C = high density lipoprotein cholesterol, BP = blood pressure, FBS = fasting blood glucose

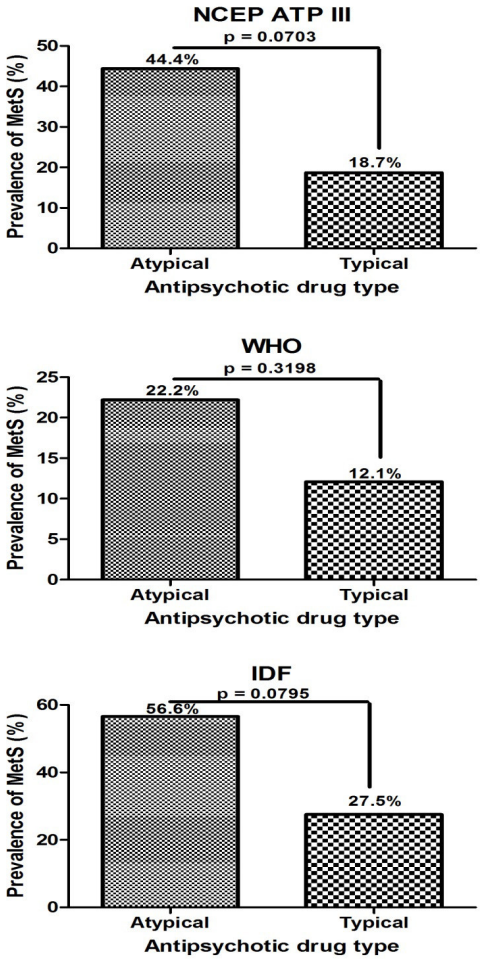

Figure 1. Prevalence of metabolic syndrome stratified atypical and typical antipsychotic medication