Use of Clinical Markers to Identify Metabolic Syndrome in Antipsychotic-Treated Patients

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Objective: Metabolic syndrome (MetS) is prevalent among antipsychotic-treated patients; however, in psychiatric clinics, scarce resources often limit the feasibility of monitoring all 5 criteria that are necessary for diagnosing MetS. As one goal of the MetS definition is to facilitate the clinical identification of insulin-resistant individuals, other biomarkers of insulin resistance have been explored. However, there are relatively few data from antipsychotic-treated patients, especially on the association between these markers and the clinical MetS diagnosis.

Method: We analyzed data from 196 psychiatric patients over age 40 years enrolled in an ongoing study of antipsychotic-related metabolic effects that began in August 2005. In addition to anthropometric measures and MetS criteria, levels of certain metabolism-related peptides (ghrelin, adiponectin, peptide YY, leptin, and insulin) were measured. The utility of these clinical and metabolic markers to identify individuals with MetS was evaluated by constructing receiver operating characteristic curves. Optimal cutoff values were calculated for markers with the greatest area under the curve on the basis of sensitivities and specificities for MetS diagnosis.

Results: Ninety-nine subjects (50.5%) met MetS criteria. The receiver operating characteristic analysis found that waist circumference, triglyceride to high-density lipoprotein (TG:HDL) ratio, and body mass index had the greatest area under the curve. The waist circumference cutoff value of 40 inches, TG:HDL ratio of 2.6, and body mass index of 28 kg/m^2 yielded sensitivities and specificities of 73% and 80%, 74% and 78%, and 75% and 74%, respectively, for MetS diagnosis.

Conclusions: Waist circumference, TG:HDL cholesterol ratio, or body mass index could be used as screens for identifying possible MetS in antipsychotic-treated patients to prompt complete investigation into all MetS criteria.

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Numerous studies worldwide have raised concern over the public health importance of metabolic disorders among severely mentally ill individuals, focusing clinical attention on prevention of type 2 diabetes mellitus in these patients.[1] Through appropriate screening, early intervention measures can be undertaken, including behavioral modification, switching to metabolically lower-risk medications, or use of adjunctive pharmacotherapy. The metabolic syndrome (MetS) concept has proved useful in this regard, by highlighting a clustering of clinical characteristics among certain insulin-resistant prediabetic patients, including central obesity, hypertension, atherogenic dyslipidemia (decreased high-density lipoprotein [HDL] cholesterol, elevated triglycerides), and increased levels of prothrombotic proteins and inflammatory markers.[1] This clinical picture has been codified in the MetS diagnosis, and individuals who meet MetS criteria represent a patient cohort at increased future risk for type 2 diabetes mellitus and cardiovascular events.[2]

The US National Cholesterol Education Program (NCEP) guidelines stipulate that at least 3 of the following 5 criteria can be used for a diagnosis of MetS[3]: waist circumference > 40 inches (102 centimeters) in men or > 35 inches (88 centimeters) in women, blood pressure ≥ 130/85 mm Hg, fasting glucose ≥ 100 mg/dL, fasting triglycerides ≥ 150 mg/dL, and HDL cholesterol < 40 mg/dL in men or < 50 mg/dL in women. However, the International Diabetes Federation has adopted MetS criteria that mandate increased waist circumference as a necessary condition,[4] thereby allowing this measure to be the focus of initial screening. The prevalence of the MetS diagnosis and that of its individual components have been shown to be higher in individuals with schizophrenia,[5–7] bipolar disorder,[5,7–9] and major depressive disorder[5,7,10] compared to nonpsychiatric controls. The reported MetS prevalence in schizophrenia and other psychotic disorders ranges from 40% to 60%, roughly twice the expected age- and sex-matched prevalence. Although the literature on MetS in psychotic patients is relatively recent,[6] numerous studies over several decades have documented increased prevalence of related conditions, including type 2 diabetes mellitus, overweight, and hyperlipidemia, and also increased cardiovascular mortality in this patient population.[11–14] While lifestyle factors such as smoking, inactivity, dietary habits, and possibly the disease itself contribute to cardiometabolic risk among the severely mentally ill individuals,[1] in recent years, there has been a considerable focus on the role of atypical antipsychotics in MetS risk.[15,16] Clinical studies indicate that certain antipsychotics (eg, clozapine and olanzapine) carry a higher risk of treatment-related metabolic dysfunction,[17,18] but metabolic monitoring is recommended for all antipsychotic-treated patients, even those on treatment with relatively lower-risk agents (eg, high-potency typical antipsychotics, ziprasidone, aripiprazole).[17]

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For Clinical Use

- Metabolic syndrome is common in patients with severe mental illness, especially in those taking antipsychotics.
- Metabolic syndrome increases the future risk of diabetes and cardiovascular events.
- Clinicians can use several clinical markers as screening tools to identify possible metabolic syndrome in antipsychotic-treated patients and initiate close monitoring as well as modify the treatment plans accordingly.

Despite recommendations for routine metabolic screening as the standard of care for management of patients receiving antipsychotic treatment,[14,17] evaluation of each MetS component is not often feasible in many outpatient psychiatry care settings due to the associated personnel costs and the limited time afforded for clinical care. Evidence for this is seen in recent data that document low levels of metabolic screening among antipsychotic-treated patients.[19,20] While complete screening for all 5 MetS criteria remains the standard, the characterization of brief screening tools focusing on a small subset of MetS criteria may have significant clinical benefit by allowing consolidation of screening to target those parameters with greatest predictive power for the MetS diagnosis.

The purpose of the present study was to examine which clinical and laboratory measures have the greatest predictive power for MetS diagnosis among antipsychotic-treated patients. This would enable us to identify which criteria, or other markers, might serve as MetS screens for mental health clinicians and staff working with antipsychotic-treated patients at high risk for MetS and other insulin-resistant states.

METHOD

Subjects and Assessments

The data were drawn from a National Institute of Mental Health–funded longitudinal study of antipsychotic metabolic, cardiovascular, and cerebrovascular effects of atypical antipsychotic medications in patients over age 40 years. This ongoing study began in August 2005 (clinicaltrials.gov identifier: NCT00245206). Qualifying diagnoses included schizophrenia; psychosis associated with mood disorder, dementia, or posttraumatic stress disorder; and psychotic disorder not otherwise specified. The study was approved by the University of California, San Diego (UCSD) Institutional Review Board, and all participants provided written informed consent. Subjects were recruited from psychiatric clinics at UCSD and Veterans Affairs San Diego Healthcare System (VASDHS), as well as from nursing homes and board-and-care homes in San Diego County.

Participants enrolled in this study completed a baseline evaluation and had follow-up assessments at week 6, week 12, and every 3 months thereafter up to 2 years. This article pertains to baseline data, which included the following: (1) medical history, use of psychotropic and other medications, and results of neurologic and screening physical examination; (2) anthropometric measurements for obesity; (3) psychopathology ratings, assessment of medication side effects, and ratings of everyday functioning; (4) venous blood for routine clinical laboratory metabolic parameters (glucose, insulin, lipid panel, high sensitivity C-reactive protein); and (5) venous blood for metabolic-related biomarkers (ghrelin, adiponectin, peptide YY, leptin, and insulin). Medical history and physical examinations, including anthropometric measurements for body mass index (BMI) and waist circumference, were performed by 2 trained physician assistants.

The clinical metabolic laboratory assessments were performed in a certified clinical laboratory at the UCSD Medical Center. Homeostatic model assessment of insulin resistance (HOMA-IR) was calculated using the standard formula: ([insulin level {μIU/mL}] × [glucose {mmol/L}]) / 22.5 and an improved model (HOMA-IR2) (www.OCDEM.ox.ac.uk).[21] Peptide biomarkers (adiponectin, leptin, ghrelin, and peptide YY) were assayed in the core laboratory of the UCSD General Clinical Research Center. Levels of adiponectin, leptin, ghrelin, and peptide YY were measured using assay kits manufactured by Linco Research, Inc (St Charles, Missouri). Insulin (catalog no. HI-14 K), ghrelin (catalog no. GHRT-89HK), leptin (catalog no. HL-81 K), and peptide YY (catalog no. PYYT-66HK) were assayed by radioimmunoassay, and adiponectin (catalog no. EZHADP-61 K) was assayed by enzyme immunoassay. Measuring ranges (MRs) and lower limits of detection (LLODs) are as follows: (1) insulin: MR = 2–200 μIU/mL, LLOD = 2 μIU/mL; (2) ghrelin: MR = 96–6150 pg/mL, LLOD = 93 pg/mL; (3) leptin: MR = 0.5–100 ng/mL, LLOD = 0.5 ng/mL; and (4) peptide YY: MR = 10.5–1350 pg/mL, LLOD = 10 pg/mL.

Clinical MetS diagnosis was made using standard American Heart Association–modified NCEP guidelines.[3] Subjects were considered as having met the glucose or blood pressure criterion if they were receiving antidiabetic medication or insulin or antihypertensive medication, respectively. Framingham 10-year cardiovascular absolute risk as well as age- and sex-matched Framingham relative risk were also calculated and compared between patients with and without metabolic syndrome.

Statistical Analysis

Continuous variables were assessed for normality of distribution; appropriate transformations were employed for the following nonnormally distributed variables: insulin, leptin, adiponectin, ghrelin, peptide YY, and HOMA-IR. Two-group comparisons were performed with either the

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Table 1. Demographic Characteristics and Biomarkers in Patients With Versus Without Metabolic Syndrome

Variable	N	Range	Metabolic Syndrome: Yes Mean (SD), n = 99	Metabolic Syndrome: No Mean (SD), n = 97	*t* Value	*df*	*P* Value
Continuous variable							
Age, y	196	40–94	64 (12)	66 (14)	0.596	194	.552
Education level, y	196	4–22	14 (3)	13 (3)	1.71	194	.088
Weight, kg	196	38–153	95 (19)	76 (20)	6.89	194	.001
Systolic blood pressure, mm Hg	196	80–178	134 (19)	122 (15)	4.99	194	.001
Diastolic blood pressure, mm Hg	196	46–97	75 (10)	69 (9)	4.09	194	.001
Waist, in	196	27–59	44 (5)	37 (6)	8.81	194	.001
Body mass index, kg/m^2	193	18–50	33 (6)	26 (6)	7.55	191	.001
Framingham 10-year risk, % risk	190	2.0–47.0	17 (9.3)	11.1 (6.6)	10.43	188	.001
Framingham relative risk,[a] % risk	190	0.4–5.7	2.06 (1.06)	1.37 (0.76)	5.12	189	.001
Fasting glucose, mg/dL	196	53–509	125 (67)	99 (37)	3.25	194	.001
HDL cholesterol, mg/dL	196	20–111	40 (12)	52 (16)	5.97	194	.001
LDL cholesterol, mg/dL	196	11–216	114 (34)	111 (37)	0.537	191	.592
Triglycerides, mg/dL	196	23–807	181 (123)	99 (53)	6.07	194	.001
					Mann-Whitney *z* score		
Highly sensitive C-reactive protein, mg/L	194	0.5–8.0	0.83 (0.80)	0.76 (0.71)	2.47		.013
Insulin, μIU/mL	181	3–131	26 (24)	16 (16)	4.35		.001
Leptin, ng/mL	183	0.5–104.0	31 (24)	13 (17)	6.73		.001
Adiponectin, μg/mL	181	2–49	10 (7)	15 (10)	4.31		.001
Ghrelin, pg/mL	182	62–3,688	691 (302)	825 (506)	1.47		.144
Peptide YY total, pg/mL	87	10–1,377	356 (312)	238 (193)	1.99		.089
HOMA-IR[b]	181	0.62–65.00	9 (10)	4 (5)	5.05		.001
HOMA-IR2[c]	181	0.4–7.1	2.8 (1.6)	1.9 (1.2)	4.29		.001
Categorical variable			n (%)	n (%)	χ^2	*df*	*P* Value
Sex, male			69 (69.7)	64 (66.0)	0.310	1	.577
History of smoking			68 (68.7)	71 (73.2)	0.483	1	.531
Race/ethnicity							
White			70 (70.7)	69 (71.1)	6.936	5	.225
African American			16 (16.2)	15 (15.5)			
Hispanic			11 (11.1)	5 (5.2)			
Asian			1 (1.0)	5 (5.2)			
Native American			1 (1.0)	1 (1.0)			
Biracial/multiracial			0 (0.0)	2 (2.1)			
Diagnosis					4.152	4	.386
Schizophrenia			36 (36.4)	34 (35.1)			
Mood disorder with psychosis			23 (23.2)	24 (24.7)			
Dementia with psychosis			17 (17.2)	23 (23.7)			
Posttraumatic stress disorder with psychosis			19 (19.2)	10 (10.3)			
Psychosis not otherwise specified			4 (4.0)	6 (6.2)			

[a]Age- and sex-matched 10-year relative risk of developing cardiovascular disease. [b]HOMA-IR = ([insulin {μIU/mL}] × [glucose {mmol/L}])/22.5.
[c]HOMA-IR2 model was calculated on the basis of an application provided at www.OCDEM.ox.ac.uk.
Abbreviations: HDL = high-density lipoprotein, HOMA-IR = homeostatic model assessment of insulin resistance, HOMA-IR2 = improved homeostatic model assessment of insulin resistance, LDL = low-density lipoprotein.

Student *t* test or the nonparametric Mann-Whitney test (depending on whether the assumptions for parametric statistics were met) on continuous variables and the χ^2 test on dichotomized variables. For all statistical tests, a 2-sided α value < .05 was deemed statistically significant.

Predictive utility of clinical and metabolic markers to identify individuals with metabolic syndrome was evaluated by constructing receiver operating characteristic (ROC) curves. Those clinical and metabolic markers that were significantly better performers on ROC (based on greater areas under the curve [AUCs]) were selected for cutoff value analysis to identify specific values that would be useful in predicting MetS diagnosis. The clinical and laboratory variable cutoff values diagnostic of MetS were based on the formula $M = (w \times s) + ([1 - w] \times p)$, in which w = prevalence of the disease (metabolic syndrome), s = sensitivity, and p = specificity of the variable in question.[22] The cutoff value identified was the value that maximized M. This represented the optimal combination of sensitivity and specificity for the variable in the particular study sample.

RESULTS

Table 1 presents the demographic and metabolic characteristics of the 196 patients who were taking antipsychotics and completed all metabolic diagnostic assessments at study baseline. Fifty-one percent (99/196) met MetS criteria, and there were no statistically significant differences in mean age, sex, racial distribution, educational level, and psychiatric diagnostic distribution between subjects with and without MetS. With the exception of LDL cholesterol, ghrelin, and peptide YY, there were significant differences in levels and prevalence of metabolic markers and MetS criteria between those with and without MetS (see Table 1).

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Table 2. Comparison of Area Under the Receiver Operating Characteristic (ROC) Curves for Clinical and Metabolic Markers of Metabolic Syndrome

Marker	Area Under the ROC Curve ± SE	95% Confidence Interval
Waist circumference	0.82 ± 0.032	0.76–0.88
Triglyceride:HDL cholesterol ratio	0.81 ± 0.033	0.74–0.87
Body mass index	0.80 ± 0.034	0.73–0.86
Leptin	0.78 ± 0.034	0.72–0.85
Weight	0.76 ± 0.036	0.69–0.83
Triglyceride	0.76 ± 0.037	0.69–0.83
HDL cholesterol	0.74 ± 0.038	0.67–0.81
HOMA-IR	0.71 ± 0.039	0.64–0.79
HOMA-IR2	0.69 ± 0.041	0.61–0.77
Glucose	0.69 ± 0.041	0.61–0.77
Insulin	0.69 ± 0.040	0.61–0.76
Adiponectin	0.69 ± 0.040	0.61–0.75
Systolic blood pressure	0.67 ± 0.040	0.61–0.75
Diastolic blood pressure	0.64 ± 0.041	0.59–0.75
Ghrelin	0.55 ± 0.044	0.56–0.72
LDL cholesterol	0.51 ± 0.044	0.43–0.60

Abbreviations: HDL = high-density lipoprotein, HOMA-IR = homeostatic model assessment of insulin resistance, HOMA-IR2 = improved homeostatic model assessment of insulin resistance, LDL = low-density lipoprotein.

Clinical and laboratory metabolic markers that were significantly different between those with and without MetS were evaluated with ROC curves. Table 2 depicts the curves in rank order of AUC. The greatest AUCs were for waist circumference (AUC = 0.82; 95% CI, 0.76–0.88; $P < .001$), triglyceride to HDL (TG:HDL) ratio (AUC = 0.81; 95% CI, 0.74–0.87; $P < .001$), and BMI (AUC = 0.80; 95% CI, 0.73–0.86; $P < .001$). The ROC AUC analyses for insulin resistance measured by HOMA-IR, HOMA-IR2, glucose, insulin, blood pressure, and other peptide biomarkers showed that these performed significantly worse than the 3 top predictor variables, with ghrelin, blood pressure, adiponectin, and insulin ranking near the bottom. Leptin was the only exception, and it ranked fourth among the variables examined, with AUC = 0.78 (95% CI, 0.72–0.85).

The optimal cutoff values for the 3 top variables that maximized M (ie, MetS diagnosis) were waist circumference > 40 inches, TG:HDL ratio ≥ 2.6, or BMI > 28 kg/m^2. The sensitivity and specificity results for the cutoff values are presented in Table 3. Among the 99 subjects with MetS, 72 had waist circumference > 40 (73% sensitivity), whereas 78 of the 97 without MetS had waist circumference ≤ 40 (80% specificity). For TG:HDL ratio ≥ 2.6, the sensitivity and specificity were 74% and 78%, respectively, and for BMI cutoff value > 28 kg/m^2, 75% sensitivity and 74% specificity. As nearly 70% of the study subjects were receiving ongoing treatment with antidiabetic, antihypertensive, or lipid-lowering medication, we ran a second analysis in the subgroup of subjects who were on any of these treatments to determine if the prior relationships held true. The results indicated that waist circumference, TG:HDL ratio, and BMI remained the optimal predictors of MetS, and the proposed cutoff value for each marker yielded equivalent sensitivity and specificity to what was found in the larger sample. After identification of the 3 optimal predictors, we subsequently analyzed the sensitivity and specificity for combinations of any 2 of these 3 markers to see if this improved on the results. Although the specificities increased into the range of 84% to 97%, the sensitivities decreased to the range of 55% to 64%. Use of all 3 measures combined increased specificity to 97%, but the sensitivity was only 50% (Table 3).

DISCUSSION

While the metabolic syndrome diagnosis itself confers no greater value for cardiovascular risk prediction than standard algorithms,[23] the concept retains significant value by highlighting clinical findings that, in isolation, may not generate significant attention, but that are associated with future risk for heart disease and type 2 diabetes mellitus.[24]

Although 5 common criteria have emerged for most definitions of the MetS, research has demonstrated that not all criteria are equally predictive of insulin resistance. Central adiposity is considered a core feature of MetS but is weighted equally with the other 4 criteria in the NCEP MetS diagnostic scheme. Given the strong association between central adiposity and insulin resistance in children and adults,[25,26] the International Diabetes Federation MetS diagnostic scheme mandates that waist circumference must be 1 of at least 3 of 5 criteria met. To further examine this issue, McLaughlin et al[27] studied the predictive value of common metabolic markers, including BMI, total cholesterol, HDL cholesterol, fasting glucose, fasting insulin, and fasting triglycerides, in identifying overweight patients who have high levels of insulin resistance. Using frequently sampled intravenous glucose tolerance testing to measure the predictor variable (insulin sensitivity), followed by ROC analysis, these investigators found that fasting triglyceride levels, TG:HDL ratio, and fasting insulin were the most useful metabolic markers to identify highly insulin-resistant individuals. Although not monitored for clinical purposes, certain cytokines have shown strong relationships with obesity and insulin resistance, including leptin, ghrelin, adiponectin, and peptide YY, with a small number of articles exploring the impact of antipsychotic treatment on these markers.[28]

The current analysis was motivated in part by the desire to confirm whether the expected association between markers identified by McLaughlin et al[27] and the MetS diagnosis (as a surrogate marker for insulin resistance) holds for antipsychotic-treated patients and to explore whether measurement of cytokines related to metabolic risk provides superior predictive power than more traditional measures. A recent review notes that changes in cytokine levels can be seen during antipsychotic treatment,[28] but only 1 article specifically examined the relationship with MetS diagnosis. Bai and colleagues[29] examined the association between serum adiponectin levels and MetS in a cohort of 188 clozapine-treated Chinese patients with schizophrenia. A logistic regression model did find that adiponectin levels inversely correlated with MetS diagnosis (OR = 0.85, $P < .01$), but BMI was more robust (OR = 1.42, $P < .001$).

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Table 3. Sensitivity and Specificity of Selected Clinical and Metabolic Markers for Predicting Metabolic Syndrome

Marker	Metabolic Syndrome,[a] n		Sensitivity, %	Specificity, %	Metabolic Syndrome,[b] n		Sensitivity, %	Specificity, %
	Yes	No			Yes	No		
1. Triglyceride:HDL cholesterol ratio < 2.6	26	76	74	78	22	42	73	82
Triglyceride:HDL cholesterol ratio ≥ 2.6	73	21			60	9		
2. Waist circumference ≤ 40 in	27	78	73	80	21	41	74	80
Waist circumference > 40 in	72	19			61	10		
3. Body mass index ≤ 28 kg/m^2	25	70	75	74	19	35	77	71
Body mass index > 28 kg/m^2	73	25			63	14		
Both 1 and 2 greater than cutoff points	54	3	55	97	45	1	55	98
Either one or both less than cutoff points	45	94			37	50		
Both 1 and 3 greater than cutoff points	55	6	56	95	48	48	59	94
Either one or both less than cutoff points	44	91			34	3		
Both 2 and 3 greater than cutoff points	63	16	64	84	54	10	66	80
Either one or both less than cutoff points	36	81			28	41		
All 3 greater than cutoff points	49	3	50	97	42	1	51	98
Any of the 3 less than cutoff points	50	94			40	51		

[a]All subjects in the study. [b]In subjects taking antidiabetic, antihypertensive, or lipid-lowering medications.
Abbreviation: HDL = high-density lipoprotein.

Although the mechanisms for antipsychotic-induced metabolic dysfunction are unclear, the present analysis underscores the fact that waist circumference, TG:HDL ratio, and BMI are valuable tools for metabolic screening in antipsychotic-treated patients and are markedly superior to cytokines, insulin, or fasting glucose for prediction of MetS diagnosis. Our study also suggests that, as a screening tool for identifying the metabolic syndrome, the use of 2 or 3 combined measures (among the top 3 predictors) did increase the specificity, but also decreased the sensitivity, and overall did not provide more optimum sensitivity and specificity than did the individual measures. In addition, our study found that the sensitivities and specificities of the top 3 predictors of metabolic syndrome held up well even in the patients who were on treatment with antidiabetic medications, antihypertensives, and lipid-lowering medications. This indicates that these clinical markers could be used for screening of metabolic syndrome in patients regardless of concomitant medications used for blood pressure, dyslipidemia, or diabetes. Given the difficulties mental health providers may face in obtaining waist circumference measurements, the importance of BMI as a predictor of MetS provides support for use of basic weight measures as a reasonable surrogate for assessment of MetS risk. The greater challenge is to obtain fasting lipid profiles, but our results, combined with the recent data on low rates of lipid and glucose monitoring for antipsychotic-treated patients,[20] should impel the mental health community to redouble its efforts toward consistent monitoring of laboratory metabolic parameters among patients receiving antipsychotic therapy.

Our study has several limitations. First, this was a sample of patients who were over age 40 years, with different psychotic disorders, taking a variety of antipsychotic medications, and who were enrolled in a study of metabolic and other effects of atypical antipsychotics, so the relationships and cutoff values noted in these analyses may not apply equally well in all settings. Second, some of the results might be influenced by ongoing medication intervention for metabolic conditions. While our analysis of the cohort receiving pharmacotherapy for hypertension, dyslipidemia, or diabetes found cutoff values that were similar to those for the sample as a whole, the impact of specific treatments could not be ascertained due to the paucity of individuals taking medications from 1 class (eg, antihypertensives) who did not also take medications from other treatment categories. The mean age of our sample was 65 years, so the results found here may not necessarily be generalizable to younger adults or child or adolescent populations. Lastly, in most ROC analyses, the outcome of interest should be independent from the test measurement of the diagnosis. In our study, we intentionally included the individual components of MetS diagnosis in the ROC analysis to examine whether, among antipsychotic-treated patients, all criteria were equally likely to be present in those with MetS, since none by itself is sufficient for the NCEP-defined MetS diagnosis. Our analysis confirmed the International Diabetes Federation model for MetS diagnosis, as waist circumference emerged as the leading predictor, but BMI was nearly equal in this regard and is, for screening purposes, more easily obtainable.

In conclusion, our study identified several common clinical and laboratory markers including waist circumference, TG:HDL cholesterol ratio, and BMI that each can be used to predict MetS with good sensitivity and specificity. Body mass index in particular is easily available in psychiatric clinical settings, so the presence of elevated BMI should prompt complete investigation of all MetS criteria, with considerable efforts put forth to obtain fasting lipid and glucose levels. It is only through consistent screening that early interventions can be implemented to decrease the metabolic comorbidities and related risks in antipsychotic-treated psychiatric patients.

Drug names: aripiprazole (Abilify), clozapine (FazaClo, Clozaril, and others), olanzapine (Zyprexa), quetiapine (Seroquel), risperidone (Risperdal and others), ziprasidone (Geodon).
Disclosure of off-label usage: The authors have determined that, to the best of their knowledge, aripiprazole is not approved by the US Food and Drug Administration for the treatment of dementia or posttraumatic stress disorder (PTSD); clozapine is not approved for the treatment of dementia,

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PTSD, bipolar disorder, or unipolar depression; and olanzapine, quetiapine, risperidone, and ziprasidone are not approved for the treatment of dementia, PTSD, or unipolar depression.

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