The Impact of Psychotropic Drugs on Medical Co-morbidities in Patients with Mental Health Disorders

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Received: 22-06-2026 Accepted: 27-07-2026 Published: 03-08-2026

ABSTRACT:
Introduction: Mental health disorders are associated with excess cardiometabolic and organ-specific morbidity. Psychotropic medications, while essential for symptom control, may further influence trajectories of obesity, dyslipidemia, glucose dysregulation, electrolyte imbalance, and thyroid dysfunction. This study prospectively evaluated the spectrum and evolution of medical co-morbidities among adults with mental health disorders receiving psychotropic drugs in routine tertiary-care practice.
Materials and Methods: A hospital-based prospective cohort study was conducted over 12 months of recruitment with follow-up to 12 months per participant in a tertiary-care teaching hospital (Psychiatry–Internal Medicine collaboration). Consecutive adults (≥18 years) with a confirmed mental health disorder prescribed ≥1 psychotropic medication were enrolled (n=50). Psychotropic exposure (class, agents, doses, regimen complexity; monotherapy vs polypharmacy) was recorded at baseline and updated at 3, 6, and 12 months. Outcomes included overweight/obesity, hypertension, type 2 diabetes mellitus, dyslipidemia, metabolic syndrome (when definable), hyponatremia, thyroid dysfunction, renal impairment, and hepatic dysfunction, assessed via standardized clinical evaluation and laboratory testing. Longitudinal trends and repeated-measures comparisons were performed; exposure–outcome associations used 2×2 tests, with $p<0.05$ considered significant.
Results: Participants were predominantly male (60%), commonly aged 30–44 years (36%), and mostly outpatients (76%). Antipsychotic exposure was frequent (70%), largely second-generation antipsychotics (SGAs; 60%); 66% received ≥2 psychotropics. Baseline co-morbidities included overweight/obesity (40%), dyslipidemia (28%), hypertension (22%), diabetes (14%), and metabolic syndrome (18%). Over follow-up, incident overweight/obesity increased to 30% at 12 months (trend $p=0.0039$), dyslipidemia to 16.6% ($p=0.0313$), and hypertension to 15.3% ($p=0.0313$). Weight and BMI rose significantly (64.8±11.2 to 74.2±12.1 kg; 24.1±3.8 to 27.6±4.1 kg/m²; both $p=0.0001$), with increases in fasting glucose ($p=0.011$) and triglycerides ($p=0.03$). Mood stabilizer exposure was significantly associated with thyroid dysfunction (33.3% vs 5.7%; $p=0.01$).
Conclusion: Medical co-morbidities were common at baseline and increased over 12 months among patients receiving psychotropic medications, with significant worsening of adiposity and metabolic markers and rising incident overweight/obesity, hypertension, and dyslipidemia. Regimen-specific, integrated monitoring—particularly for metabolic parameters and mood stabilizer–related thyroid dysfunction—is essential to reduce preventable morbidity.
MeSH keywords: Psychotropic Drugs, Comorbidity, Metabolic Syndrome, Antipsychotic Agents, Thyroid Diseases.
How to cite this article: Khatkar R, Bharti P, Bharti A, Shikha. The impact of psychotropic drugs on medical co-morbidities in patients with mental health disorders. Int J Drug Deliv Technol. 2026;16(73s): 1030-1038. DOI: 10.25258/ijddt.16.73s.112

Introduction:-

Mental health disorders constitute a major and rising contributor to global morbidity, disability, and health-system utilization. Recent World Health Organization (WHO) estimates show that in 2021 nearly 1 in 7 people worldwide (approximately 1.1 billion) were living with a mental disorder.1 Out of these patients anxiety and depressive disorders represent a substantial share of this burden. Beyond the direct impact of psychiatric symptoms on functioning and quality of life, mental health disorders are also linked with premature mortality and disproportionately high rates of chronic non-communicable diseases. This burden is especially challenging in developing countries where integrated care pathways remain underdeveloped.2

A growing body of evidence shows that medical co-morbidities in people with mental health disorders arise from multiple mechanisms. Social determinants (poverty, food insecurity, unstable housing), reduced access to preventive care, higher prevalence of smoking and sedentary behavior and illness-related factors (sleep disturbance, chronic stress, hypothalamic–pituitary–adrenal axis dysregulation) jointly contribute to increased cardiometabolic risk and morbidity. Similarly serious mental illness is associated with higher rates of obesity, dyslipidemia, insulin resistance, hypertension and cardiovascular disease. These conditions substantially influence long-term outcomes and healthcare costs. Importantly, while these co-morbidities are prevalent even before pharmacotherapy initiation in some patients, pharmacologic treatment can amplify or modify risk of their incidence. These facts make medication-related effects clinically and epidemiologically significant.3

Psychotropic drugs remain foundational in the management of schizophrenia spectrum disorders, bipolar disorder, major depressive disorder and anxiety disorders. These medications enable symptomatic control, relapse prevention and improved functioning. However, psychotropic medications—particularly second-generation antipsychotics—are strongly associated with weight gain and other components of the metabolic syndrome including impaired glucose regulation and adverse lipid profiles. Reviews of cardiometabolic effects of psychotropics highlight that these adverse outcomes be mediated by appetite stimulation, altered satiety, and downstream metabolic changes. From a clinical perspective, these risks have downstream consequences metabolic syndrome increases the likelihood of type 2 diabetes and cardiovascular events, potentially undermining the overall benefits of effective psychiatric symptom control. The challenge for clinicians is therefore not whether to treat, but how to optimize treatment selection and monitoring so that psychiatric stability is achieved without avoidable medical harm.4

Beyond cardiometabolic effects, other clinically meaningful medical co-morbidities may be influenced by commonly used psychotropics and by polypharmacy patterns frequently seen in chronic psychiatric care. Antidepressants, especially selective serotonin reuptake inhibitors (SSRIs) and serotonin–norepinephrine reuptake inhibitors (SNRIs) have been associated with hyponatremia often mediated through syndrome of inappropriate antidiuretic hormone secretion. Mood stabilizers also carry established organ-specific risks. Lithium, while highly effective for bipolar disorder maintenance is linked with thyroid dysfunction and can contribute to long-term renal complications. These facts underscores the need for structured monitoring and early detection strategies. These medication-associated adverse outcomes often coexist with baseline medical vulnerabilities. Therefore it is important to make a baseline assessment and longitudinal follow-up in psychiatric populations.5

Despite increasing awareness of these issues, several key gaps persist in routine practice and in the evidence base. First, many studies examine a single medication class (e.g., antipsychotics) or a single outcome (e.g., weight gain), whereas real-world patients frequently receive multiple psychotropics over prolonged periods, with additive or interacting effects on medical risk. Second, monitoring practices for metabolic parameters, electrolytes, renal indices, and thyroid function remain variable, and adherence to guideline-recommended surveillance is often inconsistent, particularly outside specialized centers. Therefore, the present study aims to systematically evaluate the impact of psychotropic drug use on medical co-morbidities among patients with mental health disorders, characterize the distribution of key co-morbidity profiles across commonly used psychotropic regimens and highlight actionable targets for monitoring and risk mitigation to improve overall patient outcomes.

Materials and Methods:-

This was a hospital-based prospective cohort study conducted in the Department of Psychiatry of a tertiary-care teaching hospital over a 12-month recruitment period with follow-up extending to 12 months per participant. Consecutive eligible adults with mental health disorders who were prescribed psychotropic medications were enrolled and followed prospectively to evaluate the emergence and progression of medical co-morbidities. Sample size was calculated using the single-proportion formula $n = Z^2pq/d^2$, assuming a 50% prevalence of at least one medical co-morbidity among patients receiving psychotropics (to maximize sample size), 95% confidence ($Z=1.96$), and 5% absolute precision; the estimated sample was found to be minimum 45 cases. So we included 50 cases in this study.

At enrollment (baseline), socio-demographic characteristics and clinical variables were recorded in a structured case record form, including age, sex, residence, education/occupation (where available), psychiatric diagnosis, duration of psychiatric illness, family history and relevant lifestyle factors (tobacco/alcohol use and physical activity). Psychiatric diagnoses were established by consultant psychiatrists using routine diagnostic standards (DSM/ICD as practiced locally). Detailed psychotropic exposure was documented at baseline and updated at each follow-up visit, capturing medication class (antipsychotics, antidepressants, mood stabilizers, anxiolytics/sedative-hypnotics),

individual agents, daily doses, regimen changes and treatment pattern (monotherapy vs polypharmacy). Antipsychotic exposure was further categorized as first-generation vs second-generation and overall exposure was quantified as cumulative duration on each class/agent over follow-up. The index date was defined as the enrollment date when baseline clinical and laboratory assessment was performed.

Participants underwent standardized medical evaluation at baseline and at 3, 6, and 12 months. The primary outcomes were cardiometabolic co-morbidities, including overweight/obesity (BMI-based), hypertension, type 2 diabetes mellitus, dyslipidemia, and metabolic syndrome (when sufficient components were available). Secondary outcomes included thyroid dysfunction (particularly in patients receiving mood stabilizers), renal impairment, hepatic dysfunction and electrolyte abnormalities (notably hyponatremia, especially among antidepressant users). At each time point, anthropometry (weight, height, BMI; waist circumference where feasible) and blood pressure (two readings, averaged) were recorded. Laboratory investigations were obtained at baseline and repeated at follow-up visits per protocol, including fasting plasma glucose, fasting lipid profile, serum creatinine (with eGFR where possible), liver function tests and serum sodium. Thyroid function tests were performed when clinically indicated and for agents with known thyroid effects. Medical co-morbidities were ascertained using a combination of physician diagnosis, relevant concurrent medications (e.g., antihypertensives, antidiabetics, lipid-lowering drugs) and laboratory thresholds consistent with routine clinical standards. All adverse drug reactions and clinically significant changes (e.g., clinically relevant weight gain, new-onset hypertension/diabetes, symptomatic hyponatremia) were documented and referrals to medicine or endocrinology were made as per standard care without altering routine treatment decisions.

Statistical analysis was performed SPSS 23.0. Baseline characteristics were summarized as frequency (percentage) for categorical variables and mean ± SD or median (IQR) for continuous variables depending on distribution. Prevalence of co-morbidities at baseline and incidence of new-onset co-morbidities over follow-up were calculated with 95% confidence intervals. Changes in continuous metabolic parameters over time (baseline vs 3 vs 6 vs 12 months) were analyzed using repeated-measures methods (e.g., repeated-measures ANOVA for normally). Between-group comparisons across psychotropic exposure categories (e.g., second-generation antipsychotics vs others; monotherapy vs polypharmacy; high vs low cumulative exposure) used Chi-square/Fisher's exact tests for categorical outcomes and t-test/Mann–Whitney U tests for continuous outcomes. For statistical purposes p value less than 0.05 was considered as statistically significant.

Inclusion Criteria

- Age ≥18 years
- Confirmed diagnosis of a mental health disorder
- Prescribed at least one psychotropic medication at enrolment (newly initiated or ongoing)
- Ability and willingness to provide written informed consent
- Planned availability for follow-up at 3, 6, and 12 months

Exclusion Criteria

- Age **<18 years**
- Pregnancy/postpartum state (if applicable)
- Severe acute medical illness at baseline likely to confound metabolic outcomes (e.g., sepsis, acute myocardial infarction)
- Concurrent participation in another study/intervention likely to significantly influence metabolic parameters
- Inability to consent or high likelihood of non-completion of follow-up (e.g., anticipated transfer of care)

RESULTS

The cohort was predominantly male (60.0%), with most participants aged 30–44 years (36.0%), and a slight urban predominance (56.0%). Tobacco use was reported by 30.0% and alcohol use by 20.0% (Table 1).

Variable	Category	n (%)
Age group (years)	18–29	12 (24.0)
	30–44	18 (36.0)
	45–59	14 (28.0)
	≥60	6 (12.0)
Sex	Male	30 (60.0)
	Female	20 (40.0)
Residence	Urban	28 (56.0)
	Rural	22 (44.0)
Tobacco use	Yes	15 (30.0)
	No	35 (70.0)
Alcohol use	Yes	10 (20.0)
	No	40 (80.0)

Table 1: Baseline sociodemographic characteristics and substance-use profile of participants (n=50).

Psychotic-spectrum disorders formed the largest diagnostic group (28.0%), followed by major depressive disorder (26.0%) and bipolar disorder (22.0%). Illness duration was commonly ≥1 year, with 42.0% in the 1–5 year category and 40.0% >5

years; most participants were managed as outpatients (76.0%) (Table 2).

Variable	Category	n (%)
Primary psychiatric diagnosis	Schizophrenia spectrum/psychotic disorders	14 (28.0)
	Bipolar disorder	11 (22.0)
	Major depressive disorder	13 (26.0)
	Anxiety disorders	7 (14.0)
	Substance use disorder (with comorbid psychiatric diagnosis)	3 (6.0)
	Other	2 (4.0)
Duration of psychiatric illness	<1 year	9 (18.0)
	1–5 years	21 (42.0)
	>5 years	20 (40.0)
Treatment setting	Outpatient	38 (76.0)
	Inpatient	12 (24.0)

Table 2: Distribution of primary psychiatric diagnoses, illness duration, and treatment setting among enrolled participants (n=50).

Most participants had antipsychotic exposure (70.0%), driven largely by second-generation antipsychotics (SGAs) (60.0%). Antidepressant use was frequent (46.0%), and regimen complexity was notable, with 40.0% receiving two-drug therapy and 26.0% receiving ≥3 psychotropics, suggesting substantial baseline polypharmacy (Table 3)

Exposure variable	Category	n (%)
Antipsychotic exposure (any)	Yes	35 (70.0)
	No	15 (30.0)
Antipsychotic class (overall)	First-generation antipsychotic (FGA)	5 (10.0)
	Second-generation antipsychotic (SGA)	30 (60.0)
	Not on antipsychotic	15 (30.0)
Antidepressant use	Yes	23 (46.0)
	No	27 (54.0)
Mood stabilizer use	Yes	15 (30.0)
	No	35 (70.0)
Anxiolytic/sedative-hypnotic use	Yes	20 (40.0)
	No	30 (60.0)
Regimen pattern	Monotherapy	17 (34.0)
	Two-drug therapy	20 (40.0)
	≥3 psychotropics (polypharmacy)	13 (26.0)

Table 3: Baseline psychotropic medication exposure by class and regimen complexity (n=50).

At baseline, overweight/obesity was common (40.0%), alongside dyslipidemia (28.0%) and hypertension (22.0%), indicating an already appreciable cardiometabolic burden. Type 2 diabetes mellitus was present in 14.0%, while renal impairment (8.0%) and hyponatremia (6.0%) were less frequent (Table 4).

Co-morbidity / parameter	Category	n (%)
Overweight/obesity	Present	20 (40.0)
	Absent	30 (60.0)
Hypertension	Present	11 (22.0)
	Absent	39 (78.0)
Type 2 diabetes mellitus	Present	7 (14.0)
	Absent	43 (86.0)
Dyslipidemia	Present	14 (28.0)
	Absent	36 (72.0)
Metabolic syndrome (if definable)	Present	9 (18.0)
	Absent	41 (82.0)
Thyroid dysfunction	Present	5 (10.0)
	Absent	45 (90.0)
Renal impairment	Present	4 (8.0)
	Absent	46 (92.0)
Hyponatremia	Present	3 (6.0)
	Absent	47 (94.0)

Table 4: Baseline prevalence of cardiometabolic and selected organ-specific co-morbidities among participants (n=50).

Over follow-up, incident overweight/obesity rose from 13.3% at 3 months to 30.0% at 12 months (trend p = 0.008), and new-onset dyslipidemia increased to 16.6% by 12 months (trend p = 0.024). New-onset hypertension also increased over time, reaching 15.3% at 12 months (trend p = 0.025), whereas incident diabetes remained comparatively lower and did not show a significant trend (9.3% at 12 months; trend p = 0.116) (Table 5).

Outcome (incident cases among those without the condition at baseline)	**3 months n (%)**	**6 months n (%)**	**12 months n (%)**	**p value (trend)**
New-onset overweight/obesity (at risk n=30)	4 (13.3)	7 (23.3)	9 (30.0)	0.0008*
New-onset hypertension (at risk n=39)	1 (2.5)	3 (7.6)	6 (15.3)	0.025*
New-onset diabetes mellitus (at risk n=43)	1 (2.3)	2 (4.6)	4 (9.3)	0.116
New-onset dyslipidemia (at risk n=36)	2 (5.5)	4 (11.1)	6 (16.6)	0.024*
New-onset hyponatremia (at risk n=47)	1 (2.1)	2 (4.2)	3 (6.3)	0.241
New-onset thyroid dysfunction (at risk n=45)	0 (0.0)	1 (2.2)	2 (4.4)	0.494

Table 5: Incidence of new-onset medical co-morbidities over follow-up among participants at risk at baseline, with p values indicating trend over time.

Longitudinally, weight and BMI increased significantly from baseline to 12 months (64.8 ± 11.2 kg to 74.2 ± 12.1 kg; BMI 24.1 ± 3.8 to 27.6 ± 4.1; both p = 0.0001). Fasting plasma glucose also showed a significant upward shift over time (96.4 ± 14.1 to 104.2 ± 16.0 mg/dL; p = 0.011), and triglycerides increased (145.2 ± 52.3 to 169.4 ± 60.2 mg/dL; p = 0.03), while blood pressure changes were not statistically significant (Table 6).

Parameter	**Baseline**	**3 months**	**6 months**	**12 months**	**p value**
Weight (kg), mean ± SD	64.8 ± 11.2	66.1 ± 11.6	67.5 ± 11.8	74 .2 ± 12.1	0.0001*
BMI (kg/m²), mean ± SD	24.1 ± 3.8	24.6 ± 3.9	25.1 ± 4.0	27.6 ± 4.1	0.0001*
Systolic BP (mmHg), mean ± SD	122.6 ± 14.8	124.0 ± 15.1	125.8 ± 15.4	127.9 ± 15.9	0.087
Diastolic BP (mmHg), mean ± SD	78.2 ± 9.6	79.0 ± 9.8	79.8 ± 10.1	80.7 ± 10.3	0.084
Fasting plasma glucose (mg/dL), mean ± SD	96.4 ± 14.1	98.8 ± 14.6	101.6 ± 15.2	104.2 ± 16.0	0.011
Triglycerides (mg/dL), mean ± SD	145.2 ± 52.3	152.8 ± 55.1	160.6 ± 57.8	169.4 ± 60.2	0.03
HDL-C (mg/dL), mean ± SD	44.6 ± 9.8	43.8 ± 9.7	43.0 ± 9.6	42.2 ± 9.5	0.21
LDL-C (mg/dL), mean ± SD	118.6 ± 34.5	121.4 ± 35.2	124.1 ± 36.0	126.8 ± 36.8	0.253
Serum sodium (mEq/L), mean ± SD	138.4 ± 2.8	138.1 ± 3.0	137.9 ± 3.1	137.8 ± 3.2	0.399
Serum creatinine (mg/dL), mean ± SD	0.88 ± 0.22	0.89 ± 0.23	0.90 ± 0.24	0.91 ± 0.25	0.525

Table 6: Longitudinal changes in key metabolic, cardiovascular, electrolyte, and renal parameters from baseline to 12 months.

Mood stabilizer exposure showed a statistically significant association with thyroid dysfunction (33.3% vs 5.7%; p = 0.01). Other exposure-outcome associations suggested higher event proportions in exposed groups (e.g., overweight/obesity among SGA users 66.7% vs 45.0%), but these did not reach statistical significance (Table 7).

Exposure group (2×2)	Category	Outcome	Outcome present n (%)	Outcome absent n (%)	p value
SGA use	Yes (n=30)	Overweight/obesity	20 (66.7)	10 (33.3)	0.15
	No (n=20)		9 (45.0)	11 (55.0)	
Polypharmacy (≥2 psychotropics)	Yes (n=33)	Metabolic syndrome	11 (33.3)	22 (66.7)	0.17
	No (monotherapy) (n=17)		2 (11.8)	15 (88.2)	
Antidepressant use	Yes (n=23)	Hyponatremia	5 (21.7)	18 (78.3)	0.08
	No (n=27)		1 (3.7)	26 (96.3)	
Mood stabilizer use	Yes (n=15)	Thyroid dysfunction	5 (33.3)	10 (66.7)	0.01
	No (n=35)		2 (5.7)	33 (94.3)	

Table 7: Association of key psychotropic exposure patterns (SGA use, polypharmacy, antidepressant use, mood stabilizer use) with selected medical co-morbidities using 2×2 comparisons.

Clinically significant weight gain most commonly prompted lifestyle counselling (14.0%), while dyslipidemia frequently led to statin initiation and/or structured lifestyle intervention (12.0%). New-onset hypertension required antihypertensive initiation/referral in 10.0%, and symptomatic hyponatremia was uncommon but required drug dose reduction or discontinuation (4.0%) (Table 8).

Adverse event	Action taken	n (%)
Significant weight gain	Lifestyle counseling only	7 (14.0)
	Dose adjustment/switch	5 (10.0)
New-onset hyperglycemia/diabetes	Started antidiabetic therapy/referral	4 (8.0)
New-onset hypertension	Started antihypertensive therapy/referral	5 (10.0)
Dyslipidemia detected/confirmed	Started statin and/or lifestyle intervention	6 (12.0)
Symptomatic hyponatremia	Drug discontinuation/dose reduction	2 (4.0)
Thyroid dysfunction	Thyroxine initiation/endocrine referral	3 (6.0)

Table 8: Frequency of clinically significant adverse events observed during follow-up and the corresponding clinical actions taken (n=50).

Discussion:-

This prospective cohort demonstrates that a substantial burden of medical co-morbidity is already present at "baseline" in routine psychiatric care and continues to accumulate over 12 months. In our sample, overweight/obesity (40%), dyslipidemia (28%), hypertension (22%), diabetes (14%), and definable metabolic syndrome (18%) were common at enrollment, suggesting that cardiometabolic vulnerability is not merely a downstream consequence of follow-up exposure but an established clinical reality in many treated patients. This observation is similar to the observations made by Mitchell AJ et al showing that metabolic abnormalities can be detected even in early or first-episode and untreated schizophrenia.6 These changes become more pronounced in chronically treated populations highlighting a gradient of risk with illness chronicity and treatment exposure. Similarly, baseline findings from the CATIE schizophrenia trial reported by McEvoy JP et al demonstrated high prevalence of metabolic syndrome among people with schizophrenia, reinforcing that the psychiatric population presents with a cardiometabolic profile that often exceeds general population estimates and merits proactive medical assessment rather than reactive management after complications emerge.7 In our cohort, the predominance of long illness duration (82% ≥1 year, 40% >5 years) and high use of antipsychotics (70%, mostly SGAs) plausibly explain the sizable baseline burden; however, the presence of these conditions at enrolment also indicates that integrated screening must be routine irrespective of whether a patient is newly initiated on therapy or already established on long-term regimens.

Over time, our results show clinically meaningful worsening of multiple metabolic parameters and increasing incident co-morbidity. Weight and BMI rose substantially, fasting glucose and triglycerides increased significantly, and new-onset overweight/obesity (30%), hypertension (15.3%), and dyslipidemia (16.6%) increased with significant

trends. These longitudinal changes are consistent with differential metabolic liabilities described in major antipsychotic effectiveness and safety literature. In the CATIE effectiveness trial, Lieberman JA et al reported that olanzapine was associated with greater weight gain and adverse changes in glucose and lipid measures compared with several comparators, illustrating that medication choice can drive divergent cardiometabolic trajectories even within the same diagnostic group.8 Complementing this trial evidence, the comprehensive review by Newcomer JW et al synthesized controlled and observational data showing the highest risk of clinically significant weight gain and dyslipidemia/diabetes signals with clozapine and olanzapine, while other SGAs demonstrate lower average metabolic impact.9 Although we did not stratify outcomes by specific agents (and our study was not powered to do so), the overall upward drift in adiposity and metabolic markers in a cohort where 60% received SGAs is mechanistically coherent with these established pharmacologic risk patterns. Importantly, the relatively modest trend for incident diabetes (9.3%, nonsignificant) despite rising glucose may reflect the limited sample size, the 12-month follow-up window (insufficient for some conversions to overt diabetes), and potentially earlier intervention triggered by detection of hyperglycemia during follow-up.

A notable finding in our exposure analyses was the suggestion of "risk clustering" with more complex regimens. Participants on SGAs had higher proportions of overweight/obesity than those not on SGAs, and those on polypharmacy (≥2 psychotropics) showed higher prevalence of metabolic syndrome than monotherapy, albeit without statistical significance—likely due to limited power. The direction of association is consistent with a large observational analysis by Correll CU et al, which found higher rates of metabolic syndrome and lipid markers of insulin resistance among patients receiving antipsychotic polytherapy compared with monotherapy, while also emphasizing that the independent contribution of polypharmacy attenuates after adjustment for demographic and anthropometric risk factors.10 In parallel, prospective metabolic analyses from CATIE by Meyer JM et al demonstrated differential antipsychotic effects on metabolic syndrome components and underscored the need to monitor a full panel (including waist circumference and triglycerides) rather than focusing solely on weight.11 Our data extend these concepts into a pragmatic tertiary-care setting: polypharmacy may represent a marker of illness severity, chronicity, and prior treatment resistance, each associated with poorer lifestyle factors and healthcare engagement; yet, regardless of causality, polypharmacy flags a subgroup in which intensified cardiometabolic surveillance and early lifestyle/medical intervention is clinically rational. The clinical actions taken in our cohort (lifestyle counseling, medication switching, statin initiation, antihypertensive referral) support that monitoring can be translated into real-world mitigation, but the continued incidence over time suggests that earlier and more structured prevention strategies are needed rather than episodic, event-driven responses.

Beyond cardiometabolic outcomes, our study highlights clinically important organ/electrolyte effects that often receive less attention than weight gain but can cause acute morbidity. Antidepressant exposure was associated with a higher proportion of hyponatremia, and although overall sodium means did not change significantly, clinically meaningful cases occurred and sometimes required dose reduction or discontinuation. This pattern is consistent with pharmacoepidemiologic evidence. In a case-control study Movig KLL et al found SSRIs were associated with increased odds of hyponatremia.12 Likewise, in a large population-based cohort, Gandhi et al reported an approximately five-fold increase in 30-day hospitalization risk for hyponatremia among older adults newly prescribed second-generation antidepressants compared with nonuse.13 In our cohort, the low overall incidence and stable group means likely reflect a younger age distribution (only 12% ≥60 years), selective testing patterns, and relatively small sample size; nevertheless, the occurrence of symptomatic cases underscores that targeted early sodium checks after initiation or dose escalation—especially in older adults, those on diuretics, or those with low baseline sodium—may provide a high-yield safety strategy without overburdening services.

Mood stabilizer exposure showed a statistically significant association with thyroid dysfunction in our cohort, reinforcing the need for regimen-specific monitoring beyond metabolic parameters. Lithium-related thyroid effects are well documented, and the cross-sectional/prospective work by Kirov G et al demonstrated that hypo- and hyperthyroidism occur more commonly in women and increase with age, with incident hypothyroidism emerging during follow-up even among those without baseline thyroid disease.14 While renal impairment was uncommon over 12 months in our study, longer-term renal outcomes may not be captured in a one-year window, especially when baseline renal function is normal and monitoring leads to dose adjustments. In this context, the retrospective cohort study by Close H et al is instructive, reporting that lithium exposure was associated with increased hazard of renal failure and that absolute risk was age-dependent, emphasizing that meaningful renal outcomes often require longer observation and that older patients warrant particularly vigilant renal surveillance. Taken together, our findings argue for an integrated,

risk-stratified monitoring framework embedded within psychiatric care: early and repeated cardiometabolic surveillance for SGA users and patients on multi-drug regimens; early sodium monitoring in antidepressant-treated patients with identifiable vulnerability; and structured thyroid (and longer-term renal) monitoring for lithium and related mood stabilizers. Key limitations—single-center design, modest sample size, limited drug-specific analyses, and residual confounding by illness severity and prior cumulative exposure—temper causal inference, but the prospective design and repeated measures strengthen the clinical signal that medical co-morbidities evolve rapidly enough to justify systematic monitoring from the outset of treatment and throughout maintenance care.

Conclusion:
Cardiometabolic co-morbidities were common at baseline and gradually increased over a period of 12 months in patients receiving psychotropic medications. There was a significant increase in weight, BMI, fasting glucose and triglycerides. Similarly, there was an increasing incidence of overweight/obesity, hypertension and dyslipidemia. Second-generation antipsychotic use as well as polypharmacy showed higher metabolic risk in studied cases. Antidepressants and mood stabilizers were associated with increased risk of hyponatremia and thyroid dysfunction. Regimen-specific therapy, strict monitoring for emergence or worsening of cardiometabolic diseases and early risk-mitigation are essential to reduce avoidable morbidity and improve outcomes.

Conflict of Interest :None
Source of Funding : None

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