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METABOLIC SIDE EFFECTS OF AMISULPRIDE IN PSYCHIATRIC PATIENTS: A RETROSPECTIVE STUDY

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ABSTRACT

Background: Amisulpride, a second-generation antipsychotic, is widely used for treating schizophrenia and schizoaffective disorders due to its efficacy against positive and negative symptoms and lower risk of extrapyramidal side effects compared with first-generation agents. However, metabolic side effects such as weight gain, dyslipidemia, hyperglycemia, and hyperprolactinemia remain concerns, potentially increasing the risk of cardiovascular disease and diabetes. Limited data exist on these effects in Indian populations, where metabolic syndrome is rising due to urbanization and genetic factors. **Methods:** We conducted a record-based cross-sectional retrospective study using health records from the Department of Psychiatry at KVG Medical College and Hospital, Sullia, India, spanning January 2024 to December 2024. We included 60 patients aged 18 to 65 years with DSM-5 diagnoses of schizophrenia or schizoaffective disorder on amisulpride monotherapy (approximately 600 mg per day) for at least 6 months, with available baseline and follow-up metabolic data. Exclusion criteria included pre-existing endocrine or metabolic disorders, concurrent use of other antipsychotics or metabolism-altering medications, incomplete Health records data, and pregnancy or breastfeeding. Metabolic parameters (weight, body-mass index [BMI], fasting blood glucose, glycated haemoglobin [HbA1c], lipid profile, and prolactin levels) were compared between baseline (within 1 month of initiation) and follow-up (at least 6 months) using paired t-tests, with subgroup analyses by sex. Multivariate regression identified predictors of changes. The significance level was $P<0.05$. **Results:** The mean age was 32.1±8.0 years; 58.3% were male. Mean treatment duration was 14.4±5.2 months,. Significant increases occurred in weight (71.6±15.5 kg to 72.8±15.3 kg, $P<0.001$; mean gain, 1.2 kg), BMI (24.6±6.4 to 25.0±6.4, $P<0.001$), total cholesterol (170.1±37.2 mg/dl to 186.8±43.8 mg/dl, $P<0.001$), and low-density lipoprotein (LDL) cholesterol (110.6±28.5 mg/dl to 116.7±32.6 mg/dl, $P=0.006$). No significant changes were observed in fasting blood glucose (91.0±9.9 mg/dl to 90.2±10.6 mg/dl, $P=0.226$), HbA1c (5.0±0.6% to 5.0±0.6%, $P=0.165$), high-density lipoprotein (HDL) cholesterol (50.7±8.3 mg/dl to 50.9±7.6 mg/dl, $P=0.623$), or triglycerides (129.1±49.8 mg/dl to 129.2±56.1 mg/dl, $P=0.976$). Prolactin levels increased markedly (17.5±5.9 ng/ml to 112.4±74.9 ng/ml, $P<0.001$), more so in females (21.1±4.4 ng/ml to 189.2±55.3 ng/ml) than males (14.9±5.5 ng/ml to 57.6±15.0 ng/ml; $P<0.001$ for sex difference). Hyperprolactinemia was prevalent in 95% of patients, with clinical symptoms noted in 10%. No new-onset diabetes or dyslipidemia requiring intervention was documented. **Conclusions:** Amisulpride is associated with minimal weight gain and no significant alterations in glucose metabolism or most lipid parameters but causes significant increases in total and LDL cholesterol and prolactin levels, particularly in females. These findings support its use in Indian patients at metabolic risk but underscore the need for routine monitoring of lipids and prolactin to mitigate long-term health risks.

KEYWORDS :

INTRODUCTION

Schizophrenia and schizoaffective disorders are chronic psychiatric conditions affecting approximately 1% of the global population, characterized by positive symptoms (e.g., hallucinations, delusions), negative symptoms (e.g., social withdrawal, apathy), and cognitive impairments. These disorders impose a substantial burden on individuals, families, and healthcare systems, with lifetime prevalence rates similar in India, where cultural, socioeconomic, and genetic factors may influence presentation and outcomes. Antipsychotic medications form the cornerstone of treatment, but their use is complicated by side effects that can affect adherence and long-term health.

First-generation antipsychotics, while effective against positive symptoms, are notorious for extrapyramidal side effects such as parkinsonism and tardive dyskinesia, stemming from high dopamine D2 receptor blockade in the nigrostriatal pathway. Second-generation antipsychotics (SGAs), introduced in the 1990s, offered improvements by incorporating serotonin 5-HT2A antagonism, reducing extrapyramidal risks and enhancing efficacy against negative symptoms. Amisulpride, a unique SGA, selectively antagonizes dopamine D2 and D3 receptors without significant serotonin affinity, making it effective for both positive and negative symptoms at doses of 400 to 1200 mg per day. Clinical trials have demonstrated its superiority over placebo and equivalence or superiority to other SGAs like risperidone in symptom control, with a favourable tolerability profile.[1,2]

Despite these advantages, SGAs are associated with metabolic disturbances, including weight gain, insulin resistance, dyslipidaemia, and hyperglycaemia, which contribute to metabolic syndrome—a cluster of conditions increasing the risk of type 2 diabetes, cardiovascular disease,

and premature mortality.[3,4] The prevalence of metabolic syndrome in patients with schizophrenia is 2 to 3 times higher than in the general population, attributed to lifestyle factors, disease-related sedentary behaviour, and medication effects. Hyperprolactinemia, another common side effect due to dopamine blockade in the tuberoinfundibular pathway, can lead to sexual dysfunction, amenorrhea, galactorrhoea, and osteoporosis, particularly in women.[10]

Existing literature on amisulpride's metabolic profile is encouraging but limited. A 24-week prospective study by Kotan et al. involving 18 patients reported minimal weight gain (0.8 kg), significant total cholesterol elevation, but no changes in glucose parameters or triglycerides.[1] A meta-analysis by Leucht et al. confirmed lower weight gain (1.27 kg over 6 months) compared with olanzapine (4.45 kg) or clozapine (4.9 kg).[2] Newcomer's comprehensive review highlighted amisulpride's lower risk for dyslipidaemia and hyperglycaemia relative to high-risk agents like olanzapine.[3] Tschoner et al.'s cluster analysis classified amisulpride as "non-high-risk" for metabolic dysregulation, with no significant insulin or lipid changes.[4] Comparative trials, such as Peuskens et al.'s randomized controlled trial (RCT) of 377 patients, showed amisulpride causing less weight gain (1.6 kg) and glucose elevation than olanzapine (3.9 kg and +4.42 mg/dl glucose).[5] Rettenbacher et al. found no glucose metabolism alterations over 16 weeks, unlike clozapine.[6] Systematic reviews by Taylor and McAskill, and Russell and Mackell, ranked amisulpride lowest for weight gain (0.2-1.4 kg).[7,9] Consensus guidelines by De Hert et al. recommend it for patients at metabolic risk.[8] However, Bushe et al. noted high hyperprolactinemia rates (89%), often asymptomatic but warranting monitoring.[10]

In India, a low- to middle-income country (LMIC), the burden of schizophrenia is compounded by rising metabolic syndrome prevalence, driven by urbanization, dietary shifts to high-calorie processed foods, physical inactivity, and genetic predispositions like higher visceral adiposity in South Asians.[3,4] A 2023 national survey estimated metabolic syndrome in 30-40% of urban Indians, with psychiatric patients at even higher risk due to poor monitoring and resource constraints. Despite amisulpride's widespread use in India for its affordability and efficacy, real-world data on its metabolic effects are scarce, with most studies from Western populations.[1-10] This gap is critical, as ethnic differences in drug metabolism (e.g., CYP1A2 polymorphisms) and environmental factors may modulate side effects. Poor adherence due to undetected metabolic issues can exacerbate psychosis relapses, hospitalizations, and socioeconomic strain.

This retrospective study addresses these gaps by analysing Health records from a rural Indian hospital setting, evaluating the prevalence and severity of metabolic side effects in patients on long-term amisulpride monotherapy. By focusing on a diverse cohort, we aim to inform evidence-based prescribing, enhance monitoring protocols, and improve outcomes in resource-limited settings. Understanding these effects could guide interventions like lifestyle counselling or adjunctive therapies, ultimately reducing the dual burden of mental and physical illness.

METHODS

Study Design and Setting

This was a record-based cross-sectional retrospective study conducted at the Department of Psychiatry, KVG Medical College and Hospital, a tertiary care facility in Sullia, Dakshina Kannada, Karnataka, India. The hospital serves a predominantly rural population with mixed socioeconomic backgrounds, providing comprehensive psychiatric care including outpatient and inpatient services. The study utilized health records from January 2024 to December 2024. Ethical approval was obtained from the Institutional Ethics Committee of KVG Medical College and Hospital (approval number:), and the study adhered to the Declaration of Helsinki principles. As a retrospective analysis of de-identified data, informed consent was waived, but patient confidentiality was maintained through coded identifiers.

Participants

Eligible patients were identified using consecutive sampling from Health records until the target sample size was reached. Inclusion criteria were: adults aged 18 to 65 years; both males and females; DSM-5 diagnosis of schizophrenia or schizoaffective disorder confirmed by a psychiatrist; amisulpride monotherapy at a mean dose of approximately 600 mg per day for at least 6 months; and availability of baseline metabolic data (within 1 month of treatment initiation) and follow-up data (at 6 months or the latest available up to 24 months). Exclusion criteria included pre-existing endocrine or metabolic disorders (e.g., diabetes mellitus, hyperlipidaemia, thyroid dysfunction); concurrent use of other antipsychotics, mood stabilizers, antidepressants, or medications altering metabolism (e.g., corticosteroids, beta-blockers); incomplete or unreliable Health records data (e.g., missing key parameters or inconsistent documentation); and pregnancy or breastfeeding during treatment, as these could confound metabolic assessments.

Sample Size Calculation

The sample size was calculated to detect a significant change in weight, a primary continuous outcome, based on Kotan et al.'s reported mean weight gain of 0.8 kg.[1] Using an estimated standard deviation (SD) of 2.1 kg from Leucht et al.'s meta-analysis,[2] a paired t-test formula was applied: $n = (Z\alpha/2 + Z\beta)^2 \times SD^2 / d^2$, where $Z\alpha/2 = 1.96$ (95% confidence), $Z\beta = 0.84$ (80% power), $d = 0.8$ kg (effect size). This yielded n=54. Adjusting for 10% attrition due to potential data incompleteness, the final sample size was 60.

Data Collection

Data extraction was performed by two trained research assistants using a standardized proforma, with double-entry verification to minimize errors. Extracted variables included:

- Demographic details: age, sex.
- Clinical data: amisulpride dosage (mg/day), treatment duration (months), SBP, DBP.
- Metabolic parameters: weight (kg), BMI (kg/m^2, calculated as weight/height^2), fasting blood glucose (mg/dl, measured via enzymatic assay), HbA1c (%, via high-performance liquid chromatography), lipid profile (total cholesterol, LDL cholesterol, HDL cholesterol, triglycerides; all in mg/dl via enzymatic colorimetric methods), and prolactin levels (ng/ml, via chemiluminescent immunoassay). Baseline values were from the first month post-initiation; follow-up from ≥6 months (mean 14.4 months).

Laboratory tests were conducted in the hospital's accredited biochemistry lab, with quality controls as per standard protocols. No direct patient interaction occurred, ensuring the study's retrospective nature.

Statistical Analysis

Data were analysed using SPSS version 20.0 (IBM Corp.) and Microsoft Excel 2007. Descriptive statistics summarized demographics and clinical characteristics (means ± SD for continuous variables, frequencies and percentages for categorical). Paired t-tests compared baseline and follow-up metabolic parameters. Chi-square tests analysed categorical outcomes, such as hyperprolactinemia prevalence (defined as >20 ng/ml in males, >25 ng/ml in females). Subgroup analyses stratified by sex, age (<40 vs. ≥40 years), and dose (<600 mg/day vs. ≥600 mg/day). Assumptions of normality

(Shapiro-Wilk test), linearity, and homoscedasticity were verified. A two-sided P value <0.05 was considered statistically significant. No imputation was needed due to complete data in selected records.

Data Sharing Statement

De-identified data will be available upon reasonable request to the corresponding author, subject to institutional approval.

RESULTS

A total of 60 patients' Health records met the inclusion criteria and were analyzed, with no exclusions for attrition as all selected records had complete data. The cohort represented a typical psychiatric outpatient population in rural India, with balanced representation across age and sex.

Demographic and Clinical Characteristics

The mean age was 32.1±8.0 years (range, 18 to 58), reflecting a relatively young cohort common in schizophrenia onset. Males comprised 58.3% (n=35), and females 41.7% (n=25). Most patients had schizophrenia (80%, n=48), while 20% (n=12) had schizoaffective disorder. The mean treatment duration was 14.4±5.2 months (range, 6 to 24), exceeding the minimum 6 months to capture long-term effects. The amisulpride dose ranges from 400 to 900mg), consistent with therapeutic guidelines for maintenance therapy. Baseline characteristics are detailed in Table 1. No significant baseline differences were noted between sexes in age (P=0.45), diagnosis (P=0.32), or dose (P=0.28), ensuring comparability.

Changes in Weight and Body-Mass Index

Significant increases were observed in body weight and BMI. Mean weight rose from 71.6±15.5 kg at baseline to 72.8±15.3 kg at follow-up (mean change, +1.2 kg; 95% confidence interval [CI], 0.8 to 1.6; P<0.001). Correspondingly, BMI increased from 24.6±6.4 kg/m^2 to 25.0±6.4 kg/m^2 (mean change, +0.4 kg/m^2; 95% CI, 0.3 to 0.5; P<0.001). Subgroup analysis showed similar changes in males (weight: +1.1 kg, P<0.001) and females (+1.3 kg, P<0.001), with no sex difference (P=0.62). Age and dose did not predict weight change in regression models (β coefficients: age, 0.02, P=0.78; dose, 0.001, P=0.89). Clinically, 15% (n=9) experienced ≥5% weight gain, a threshold for metabolic concern, but none reached obesity (BMI ≥30 kg/m^2) from normal baseline.

Glucose Metabolism Parameters

No significant alterations occurred in glucose homeostasis. Fasting blood glucose decreased slightly from 91.0±9.9 mg/dl to 90.2±10.6 mg/dl (mean change, -0.8 mg/dl; 95% CI, -2.1 to 0.5; P=0.226). HbA1c remained stable at 5.0±0.6% (mean change, 0.0%; 95% CI, -0.1 to 0.1; P=0.165). Subgroups by sex, age, and dose showed no differences (all P>0.05). Regression analysis confirmed no predictors (e.g., duration β= -0.01, P=0.92). No patients developed hyperglycaemia (fasting glucose ≥126 mg/dl) or prediabetes (HbA1c ≥5.7%).

Lipid Profile Changes

Lipid parameters showed mixed results. Total cholesterol increased significantly from 170.1±37.2 mg/dl to 186.8±43.8 mg/dl (mean change, +16.7 mg/dl; 95% CI, 10.2 to 23.2; P<0.001). LDL cholesterol also rose from 110.6±28.5 mg/dl to 116.7±32.6 mg/dl (mean change, +6.1 mg/dl; 95% CI, 1.8 to 10.4; P=0.006). However, HDL cholesterol (50.7±8.3 mg/dl to 50.9±7.6 mg/dl; mean change, +0.2 mg/dl; 95% CI, -0.6 to 1.0; P=0.623) and triglycerides (129.1±49.8 mg/dl to 129.2±56.1 mg/dl; mean change, +0.1 mg/dl; 95% CI, -4.2 to 4.4; P=0.976) remained unchanged. Sex subgroups mirrored these patterns (females: total cholesterol +18.2 mg/dl, P<0.001; males: +15.6 mg/dl, P<0.001; no sex difference, P=0.51). Multivariate regression identified treatment duration as a weak predictor for total cholesterol increase (β=0.45, P=0.04), but not for LDL (P=0.12). Dyslipidaemia (total cholesterol ≥200 mg/dl or LDL ≥130 mg/dl) prevalence rose from 18% (n=11) at baseline to 35% (n=21) at follow-up, but no interventions were documented.

Prolactin Levels and Endocrinologic Effects

Prolactin elevation was pronounced, increasing from 17.5±5.9 ng/ml to 112.4±74.9 ng/ml (mean change, +94.9 ng/ml; 95% CI, 72.3 to 117.5; P<0.001). This was more marked in females (21.1±4.4 ng/ml to 189.2±55.3 ng/ml; mean change, +168.1 ng/ml; P<0.001) than males (14.9±5.5 ng/ml to 57.6±15.0 ng/ml; mean change, +42.7 ng/ml; P<0.001), with a significant sex difference (P<0.001). Hyperprolactinemia affected 95% (n=57) at follow-up, up from 0% at baseline. Clinical symptoms (e.g., menstrual irregularities in 3 females, decreased libido in 2 males and 1 female) were noted in 10% (n=6) of records with documentation. Regression models showed sex (female β=1.25, P<0.001) and dose (β=0.08, P=0.03) as predictors, but not age or duration.

No significant sex differences emerged in non-prolactin parameters (all P>0.05). Overall, no records indicated new-onset diabetes, dyslipidaemia requiring treatment, or other endocrinologic complications attributed to amisulpride.

DISCUSSION

This retrospective study of 60 Indian patients on long-term amisulpride monotherapy reveals a favourable metabolic profile overall, with minimal weight gain, stable glucose metabolism, and unchanged triglycerides and HDL cholesterol. However, significant elevations in total and LDL cholesterol and prolactin levels highlight areas for vigilance, particularly in females for hyperprolactinemia. These findings align with prior literature but provide novel insights into a rural Indian context, where metabolic risks are amplified by socioeconomic factors.[1-10]

The observed mean weight gain of 1.2 kg over a mean 14.4 months is consistent with Kotan et al.'s 0.8 kg over 24 weeks[1] and Leucht et al.'s meta-analytic estimate of 1.27 kg over 6 months,[2] positioning amisulpride as low-risk compared with olanzapine (3-4 kg) or clozapine (4-5 kg).[2] Taylor and McAskill's systematic review[7] and Russell and Mackell's epidemiology[9] further corroborate this, attributing amisulpride's limbic selectivity and lack of histamine H1 antagonism to reduced appetite stimulation. In our cohort, only 15% exceeded 5% weight gain, far below rates for high-risk SGAs (up to 50%).[5] BMI increases were modest, with no shifts to overweight categories, suggesting clinical insignificance for most patients. Subgroup analyses showed no predictors like age or dose, unlike Peuskens et al., where high baseline BMI amplified olanzapine effects but not amisulpride's.[5] This supports De Hert et al.'s guidelines recommending amisulpride for metabolically vulnerable patients,[8] especially in India, where baseline BMI (mean 24.6 kg/m^2) reflects a leaner population prone to visceral fat accumulation.[3,4]

Glucose stability—no changes in fasting glucose or HbA1c—mirrors Rettenbacher et al.'s 16-week findings[6] and Newcomer's review,[3] contrasting with clozapine's insulin elevations.[6] Tschoner et al.'s cluster analysis classified amisulpride as non-high-risk, with no insulin or glucose dysregulation.[4] Peuskens et al.'s RCT showed amisulpride even reducing glucose slightly (-2.82 mg/dl), akin to our -0.8 mg/dl.[5] The absence of new-onset diabetes (0% vs. 1-2% in olanzapine trials)[5] underscores amisulpride's safety, potentially due to minimal muscarinic M3 antagonism, which impairs insulin secretion in other SGAs. In Indian contexts, where diabetes prevalence is 10-15% and genetic factors (e.g., PPARG polymorphisms) heighten risk,[3] this profile is advantageous. Regression confirmed no predictors, suggesting amisulpride does not exacerbate latent glucose intolerance.

Lipid changes were selective: significant total and LDL increases without HDL or triglyceride shifts. Kotan et al. reported similar total cholesterol rises (from 176.7 to 194.4 mg/dl),[1] though our magnitude (+16.7 mg/dl) was comparable. Newcomer's review[3] and Bushe et al.[10] noted milder dyslipidaemia vs. risperidone, but our 17% rise in dyslipidaemia prevalence warrants attention. Duration predicted total cholesterol elevation, implying cumulative effects, unlike short-term studies.[1,6] HDL stability is beneficial, as low HDL is a South Asian metabolic hallmark.[3] Compared with olanzapine (triglyceride increases of 50-100 mg/dl in Peuskens et al.), amisulpride's profile is superior,[5] but monitoring is essential to prevent atherosclerosis.

Hyperprolactinemia's high prevalence (95%) and severity, especially in females (mean 189.2 ng/ml), echo Bushe et al.'s 89% rate[10] and Kotan et al.'s marked elevations (235 ng/ml in females).[1] Amisulpride's potent D2 blockade explains this, with females more susceptible due to oestrogen modulation of prolactin.[10] Symptoms in only 10% align with literature, where many elevations are asymptomatic but risk bone density loss long-term.[10] Sex and dose as predictors highlight need for female-specific monitoring and dose optimization.

Strengths include real-world Health records data from an underrepresented rural Indian setting, comprehensive parameters, and robust statistics. Limitations: retrospective design risks selection bias (e.g., only monitored patients included); single-centre limits generalizability; no control group or pre-treatment washout; potential confounding from undocumented lifestyle factors; shorter mean follow-up than ideal for chronic effects; lack of cardiac (e.g., QT interval) or additional endocrinologic (e.g., thyroid) assessments. Future prospective multicenter studies with controls, longer follow-up (>24 months), and biomarkers (e.g., insulin resistance indices) are needed. Incorporating lifestyle interventions could mitigate risks.

In conclusion, amisulpride offers a metabolically safer alternative for Indian psychiatric patients, with low risks for weight gain and glucose dysregulation but requiring lipid and prolactin surveillance. These data support tailored prescribing and monitoring protocols to optimize outcomes in LMICs.

Table 1. Baseline Demographic and Clinical Characteristics.

Characteristic			Value (N=60)		
Age (years)			32.1 ± 8.0		
Sex — no. (%)					
Male			35 (58.3)		
Female			25 (41.7)		
Duration of treatment (months)			14.4 ± 5.2		
Other Clinical Parameters					
Parameter	Baseline (Mean ± SD)	Follow-up (Mean ± SD)	t-value	p-value	Significance
SBP (mmHg)	118.45 ± 9.01	118.94 ± 10.45	0.791	0.432	Non-significant
DBP (mmHg)	80.74 ± 7.90	81.46 ± 8.90	1.359	0.179	Non-significant
Creatinine (mg/dL)	1.00 ± 0.21	1.01 ± 0.23	1.400	0.167	Non-significant
TSH (mIU/L)	2.06 ± 0.49	2.11 ± 0.66	1.097	0.277	Non-significant

Table 2. Metabolic and Endocrinologic Parameters at Baseline and Follow-up.

Parameter	Baseline	Follow-up	P Value
Weight (kg)	71.6 ± 15.5	72.8 ± 15.3	<0.001
BMI (kg/m²)	24.6 ± 6.4	25.0 ± 6.4	<0.001
Fasting blood glucose (mg/dl)	91.0 ± 9.9	90.2 ± 10.6	0.226
HbA1c (%)	5.0 ± 0.6	5.0 ± 0.6	0.165
Total cholesterol (mg/dl)	170.1 ± 37.2	186.8 ± 43.8	<0.001
LDL cholesterol (mg/dl)	110.6 ± 28.5	116.7 ± 32.6	0.006
HDL cholesterol (mg/dl)	50.7 ± 8.3	50.9 ± 7.6	0.623
Triglycerides (mg/dl)	129.1 ± 49.8	129.2 ± 56.1	0.976
Prolactin (ng/ml) — overall	17.5 ± 5.9	112.4 ± 74.9	<0.001
Prolactin Males	14.9 ± 5.5	57.6 ± 15.0	<0.001
Prolactin Females	21.1 ± 4.4	189.2 ± 55.3	<0.001

Values are means ± SD. P values from paired t-tests.

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