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Effect of telmisartan on metabolic syndrome components and cardiovascular protection: A prospective observational study.

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Abstract

Background

Metabolic syndrome (MetS) is a cluster of cardiovascular risk factors including central obesity, hypertension, dyslipidaemia, and insulin resistance, which substantially increase the risk of diabetes mellitus and cardiovascular disease. .To evaluate the effect of telmisartan on the components of metabolic syndrome and its role in cardiovascular protection.

Methods

This prospective observational study included 100 patients with MetS, treated with telmisartan (40–80 mg daily) for 24 weeks. Blood pressure, fasting glucose, fasting insulin, HOMA-IR, lipid profile, waist circumference, and BMI were measured at baseline, 12 weeks, and 24 weeks. Cardiovascular protection was assessed by pulse wave velocity (PWV) and flow-mediated dilation (FMD). Data were analysed using paired t-tests, with $p<0.05$ considered significant.

Results

Telmisartan significantly reduced systolic (152.8 ± 12.6 to 132.6 ± 8.7 mmHg; $p<0.001$) and diastolic blood pressure (94.6 ± 8.1 to 82.3 ± 6.1 mmHg; $p<0.001$). Fasting glucose (118.6 ± 16.4 to 104.8 ± 12.7 mg/dL; $p<0.01$), fasting insulin (16.2 ± 4.8 to 11.7 ± 3.9 µU/mL; $p<0.01$), and HOMA-IR (4.8 ± 1.5 to 3.1 ± 1.1; $p<0.01$) improved significantly. Lipid profile showed reduced triglycerides and LDL-C, and increased HDL-C. Waist circumference and BMI decreased modestly ($p<0.05$). Cardiovascular protection markers improved, with reduced PWV (10.2 ± 1.8 to 8.7 ± 1.5 m/s; $p<0.01$) and increased FMD (6.4 ± 1.3% to 9.1 ± 1.6%; $p<0.01$). Telmisartan was well tolerated with only mild, transient adverse events.

Conclusion

Telmisartan effectively improves multiple components of metabolic syndrome while enhancing vascular function, thereby offering both metabolic and cardiovascular protection. Its dual ARB and PPAR-γ activity make it a valuable therapeutic option in high-risk MetS patients.

Recommendations

Telmisartan should be preferred in metabolic syndrome for simultaneous blood pressure control, improved insulin sensitivity, and enhanced cardiovascular protection.

***Keywords**: Telmisartan, Metabolic Syndrome, Insulin Resistance, Cardiovascular Protection, Peroxisome Proliferator-Activated Receptor Gamma (PPAR-γ)*

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Introduction

Metabolic syndrome (MetS) represents a constellation of interrelated metabolic and cardiovascular risk factors central obesity, hypertension, dyslipidaemia, and insulin resistance that collectively increase susceptibility to type 2 diabetes mellitus (T2DM) and cardiovascular disease

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(CVD) [1]. Its rising prevalence worldwide constitutes a major public health concern, given its strong association with atherosclerosis, myocardial infarction, and stroke, thereby contributing significantly to global morbidity and mortality [2].

The renin–angiotensin–aldosterone system (RAAS) plays a central role in both hemodynamic regulation and metabolic dysfunction. Beyond its vasoconstrictive and sodium-retaining effects, activation of RAAS promotes oxidative stress, endothelial dysfunction, adipocyte inflammation, and insulin resistance, all of which underpin the pathophysiology of MetS [3]. Consequently, pharmacological inhibition of RAAS using angiotensin receptor blockers (ARBs) has emerged as a rational therapeutic strategy that not only lowers blood pressure but may also exert beneficial metabolic effects.

Telmisartan, a long-acting ARB, stands apart within its class due to its dual mechanism of action. In addition to blocking angiotensin II at the AT1 receptor, telmisartan functions as a partial agonist of peroxisome proliferator-activated receptor gamma (PPAR-γ), a nuclear receptor involved in glucose and lipid metabolism [4]. This unique pharmacodynamic property imparts additional metabolic advantages, including improved insulin sensitivity, reduced visceral adiposity, and favourable modulation of lipid parameters.

Clinical and experimental evidence supports telmisartan's pleiotropic benefits extending beyond antihypertensive efficacy. Studies have demonstrated that telmisartan improves endothelial function, reduces vascular inflammation, and attenuates the progression of atherosclerosis thereby providing cardiovascular protection in patients with MetS [5].

Given this dual profile, telmisartan is increasingly recognized as a promising therapeutic agent offering synergistic antihypertensive, metabolic, and vasculoprotective benefits in individuals with metabolic syndrome.

Aim

To evaluate the effect of telmisartan on the components of metabolic syndrome and its role in cardiovascular protection.

Objectives

To assess the impact of telmisartan on blood pressure control in patients with metabolic syndrome.

To evaluate the effect of telmisartan on insulin resistance and fasting serum insulin levels.

To study the influence of telmisartan on lipid parameters, including triglycerides, HDL-C, and LDL-C.

To determine telmisartan's role in reducing cardiovascular risk through its effects on endothelial function and arterial stiffness.

Material and Methods

Study Design and Setting

This was a hospital-based cross-sectional study conducted in the Departments of Pharmacology and General Medicine at Konaseema Institute of Medical Sciences & Research Foundation (KIMS & RF), Amalapuram, Andhra Pradesh, India. KIMS & RF is a tertiary-care teaching hospital with more than 700 beds, offering undergraduate and postgraduate medical education. The institution caters to rural and semi-urban communities of the Konaseema delta region and provides advanced diagnostic and clinical services across major specialties. The study was conducted over six months, from February 2025 to July 2025

Study Population

A total of 100 patients diagnosed with metabolic syndrome, as defined by the International Diabetes Federation (IDF) criteria, [6] were recruited.

Inclusion criteria

Age between 30–65 years.

Presence of central obesity (waist circumference ≥90 cm in men, ≥80 cm in women) plus at least two of the following:

Elevated triglycerides (≥150 mg/dL).

Reduced HDL cholesterol (<40 mg/dL in men, <50 mg/dL in women).

Raised blood pressure (≥130/85 mmHg or on treatment).

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Fasting plasma glucose ≥100 mg/dL or known diabetes.

Exclusion criteria

Secondary hypertension.

Severe hepatic or renal impairment.

History of myocardial infarction, stroke, or heart failure within the last 6 months.

Current use of thiazolidinediones, insulin sensitizers, or other ARBs/ACE inhibitors.

Pregnancy and lactation.

Study Size Justification

A sample size of 100 patients was chosen based on feasibility and the average monthly outpatient attendance of metabolic syndrome cases at the study site. Using a prevalence estimate of 40% for metabolic syndrome features requiring ARB therapy, with a 95% confidence level and 10% allowable error, the minimum sample size calculated was 92. To ensure adequate representation and account for incomplete data, the sample size was rounded to 100.

Intervention

All patients received telmisartan 40–80 mg once daily as per clinical requirement. Standard lifestyle modification advice regarding diet, exercise, and smoking cessation was also provided.

Data Collection and Measurements

Baseline demographic details, anthropometric measurements, and clinical history were recorded. Patients were followed up at baseline, 12 weeks, and 24 weeks.

The following parameters were assessed:

Blood pressure – measured using a calibrated sphygmomanometer (mean of two readings after 5 minutes rest).

Fasting blood glucose (FBG) – measured by glucose oxidase-peroxidase method.

Fasting serum insulin – measured using ELISA. Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) was calculated using the formula:

HOMA-IR=Fasting Insulin (µU/mL) × Fasting Glucose (mg/dL)/405

Lipid profile – total cholesterol, triglycerides, HDL-C, LDL-C measured using enzymatic methods.

Waist circumference and BMI – measured using standard protocols.

Cardiovascular protection markers – arterial stiffness assessed by pulse wave velocity (PWV) and endothelial function assessed by flow-mediated dilation (FMD).

Safety assessment – monitoring for adverse drug reactions, changes in serum creatinine, electrolytes, and liver function tests.

Outcome Measures

Primary outcomes: Improvement in blood pressure, HOMA-IR, fasting insulin, and lipid parameters after 24 weeks of telmisartan therapy.

Secondary outcomes: Improvement in markers of cardiovascular protection (PWV, FMD), safety, and tolerability profile of telmisartan.

Bias Control

To minimise selection bias, all eligible adult patients meeting the IDF criteria during the study period were consecutively recruited. Measurement bias was reduced by using standardised procedures for blood pressure recording, anthropometry, and laboratory investigations. Information bias was controlled by obtaining clinical data directly from patient records and laboratory reports rather than self-reported values. All laboratory tests were performed in the same NABL-accredited hospital laboratory using calibrated equipment.

Statistical Analysis

Data were analysed using SPSS version 20. Continuous variables were expressed as mean ± SD, and categorical variables as percentages. Paired t-test and ANOVA were used to compare baseline and follow-up values. A p value <0.05 was considered statistically significant.

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Ethical Considerations

Ethical approval for the study was obtained from the Institutional Ethics Committee of KIMS & RF, Amalapuram. Written informed consent was obtained from all participants prior to enrolment. Confidentiality of patient information was maintained throughout the study.

Results

Participant Flow

During the study period, 128 individuals were screened for metabolic syndrome. Of these, 112 patients met the initial eligibility criteria and were examined further. Twelve individuals were excluded because they did not satisfy the IDF diagnostic requirements (n = 8) or declined participation after counselling (n = 4). A total of 100 eligible patients were recruited and included in the study.

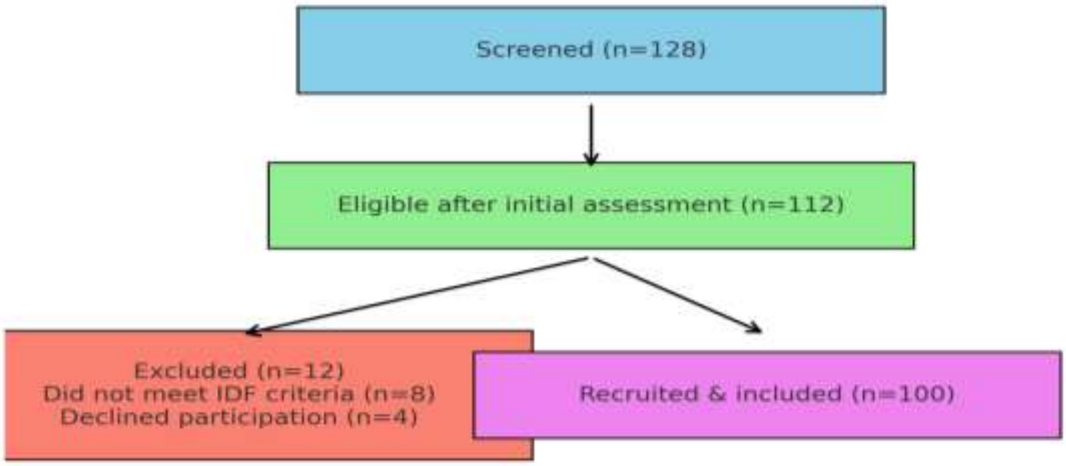

Figure 1: Participant Flow Diagram

All 100 participants completed baseline evaluation and initiated telmisartan therapy. During follow-up, five participants discontinued the study: three were lost to follow-up after the 12-week visit, and two withdrew consent for personal reasons. The remaining 95 patients completed the 24-week follow-up assessments and were included in the final analysis.

Baseline Characteristics

A total of 100 patients with metabolic syndrome were enrolled. Of these, 58 were male and 42 were female. The mean age was 52.4 ± 8.3 years. At baseline, the majority of patients had elevated blood pressure (100%), central obesity (100%), hypertriglyceridemia (72%), and impaired fasting glucose (64%).

Effect of Telmisartan on Blood Pressure and Metabolic Parameters

At 12 and 24 weeks, significant reductions were observed in systolic blood pressure (SBP), diastolic blood pressure (DBP), fasting blood glucose, fasting serum insulin, and HOMA-IR. In addition, lipid parameters showed improvement, with reduced triglycerides and LDL-C, and increased HDL-C. Waist circumference and BMI showed modest but significant reductions after 24 weeks (Table 1).

Effect on Cardiovascular Protection Markers

Telmisartan significantly improved vascular parameters. Mean pulse wave velocity (PWV) decreased from baseline 10.2 ± 1.8 m/s to 8.7 ± 1.5 m/s at 24 weeks ($p<0.01$). Flow-mediated dilation (FMD) improved from 6.4 ± 1.3% to 9.1 ± 1.6% ($p<0.01$), indicating better endothelial function.

Adverse Events

Telmisartan was generally well tolerated. Mild dizziness was reported in 6 patients and transient hyperkalaemia in 2 patients, which resolved without discontinuation. No severe adverse effects were noted.

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Table 1. Effect of Telmisartan on Metabolic Syndrome Components and Cardiovascular Parameters

Parameter	Baseline (Mean ± SD)	12 Weeks (Mean ± SD)	24 Weeks (Mean ± SD)	***p*** value (Baseline vs 24 wks)
SBP (mmHg)	152.8 ± 12.6	138.4 ± 10.2	132.6 ± 8.7	<0.001
DBP (mmHg)	94.6 ± 8.1	86.2 ± 6.9	82.3 ± 6.1	<0.001
Fasting glucose (mg/dL)	118.6 ± 16.4	110.2 ± 14.3	104.8 ± 12.7	<0.01
Fasting insulin (µU/mL)	16.2 ± 4.8	13.9 ± 4.2	11.7 ± 3.9	<0.01
HOMA-IR	4.8 ± 1.5	3.9 ± 1.3	3.1 ± 1.1	<0.01
Triglycerides (mg/dL)	186.7 ± 42.5	168.2 ± 36.8	152.9 ± 32.6	<0.01
HDL-C (mg/dL)	38.6 ± 6.7	41.2 ± 7.1	44.5 ± 7.6	<0.05
LDL-C (mg/dL)	132.4 ± 28.6	122.8 ± 26.7	115.2 ± 24.9	<0.05
Waist circumference (cm)	102.6 ± 8.4	101.2 ± 7.9	99.3 ± 7.6	<0.05
BMI (kg/m²)	28.6 ± 3.2	28.1 ± 3.0	27.6 ± 2.9	<0.05
PWV (m/s)	10.2 ± 1.8	9.3 ± 1.6	8.7 ± 1.5	<0.01
FMD (%)	6.4 ± 1.3	7.8 ± 1.5	9.1 ± 1.6	<0.01

Discussion

The present study demonstrates that telmisartan significantly improves metabolic parameters, insulin resistance, and vascular function in patients with metabolic syndrome (MetS). These findings are in concordance with prior evidence indicating that angiotensin II receptor blockers (ARBs), particularly telmisartan, exert pleiotropic actions beyond their antihypertensive effects [6,7].

Several clinical and experimental investigations have substantiated the vascular benefits of telmisartan. In hypertensive patients, telmisartan enhanced flow-mediated dilation (FMD) and reduced pulse wave velocity (PWV), reflecting improved endothelial function and diminished arterial stiffness [10]. Similar outcomes were confirmed in meta-analyses of randomized controlled trials, which demonstrated consistent improvements in endothelial function and arterial compliance compared with other antihypertensives [9,11]. In individuals with type 2 diabetes and essential hypertension, telmisartan effectively reduced carotid-femoral PWV, indicating its role in ameliorating vascular stiffness independent of blood pressure reduction [10,12].

The observed improvements in fasting insulin levels and HOMA-IR in this study corroborate earlier research showing that telmisartan enhances insulin sensitivity in both diabetic and non-diabetic hypertensive patients [6,7]. This effect has been attributed to its unique partial activation of the peroxisome proliferator-activated receptor gamma (PPAR-γ), which promotes glucose uptake and mitigates adipose tissue inflammation [8]. Through PPAR-γ modulation, telmisartan also influences adipokine balance and reduces leptin and inflammatory cytokine secretion, thereby contributing to metabolic homeostasis [8,9].

Furthermore, telmisartan has demonstrated long-term benefits on vascular remodeling. Experimental models revealed that telmisartan attenuates elastin degradation and collagen accumulation in arterial walls, thereby preventing vascular stiffening [6]. Clinical studies similarly reported significant reductions in arterial stiffness indices and improvements in endothelial-dependent vasodilation following prolonged telmisartan therapy [10,11].

Collectively, these findings support the notion that telmisartan exerts comprehensive cardiometabolic protection by combining hemodynamic control with metabolic and vascular modulation. Its dual mechanism angiotensin II receptor blockade and selective PPAR-γ activation positions telmisartan as a particularly valuable therapeutic option in patients with metabolic syndrome. Further large-scale longitudinal studies are warranted to

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evaluate its sustained impact on cardiovascular morbidity and mortality.

Generalizability

The findings of this study are primarily applicable to individuals attending a tertiary-care hospital in the Konaseema region, where patients often represent a mix of rural and semi-urban populations with diverse lifestyle patterns and variable access to healthcare services. Because the study was conducted in a single center, the results may not fully represent metabolic syndrome patterns in other regions with different dietary habits, occupational exposures, or socioeconomic backgrounds. Nevertheless, the diagnostic criteria used (IDF guidelines) are internationally recognized, which enhances the external validity of the observations. The demographic profile and clinical characteristics seen in this cohort mirror trends reported in similar Indian studies, suggesting that the overall patterns may be reasonably generalizable to comparable healthcare settings. Further multicentric studies involving heterogeneous populations would strengthen the ability to extrapolate these findings at a broader national or global level.

Conclusion

Telmisartan demonstrated significant improvement across multiple components of metabolic syndrome, including blood pressure, insulin resistance, lipid profile, and central obesity, indicating both metabolic and cardiovascular benefits. The reduction in HOMA-IR and triglyceride levels, along with improved HDL-C, pulse wave velocity, and flow-mediated dilation, underscores its potential to enhance vascular health and endothelial function. These beneficial effects likely stem from its dual mechanism as an angiotensin II receptor blocker with partial PPAR-γ agonist activity. The drug was well tolerated, with only mild, transient adverse effects. Overall, telmisartan represents a valuable therapeutic option for comprehensive management and cardiovascular risk reduction in patients with metabolic syndrome.

Limitations

The study was limited by its single-center design, modest sample size, and a relatively short follow-up duration of 24 weeks. Exclusion of other comparative ARBs restricts the broader applicability of the findings. Long-term outcomes, including cardiovascular morbidity and mortality, were not evaluated, leaving the sustained clinical impact of the intervention unknown.

Recommendations

Future research should include large-scale, multicentric randomized controlled trials to validate these findings and assess telmisartan's long-term cardiovascular benefits. Comparative studies with other ARBs and insulin-sensitizing agents are warranted to delineate its relative efficacy. Evaluation of inflammatory markers, adipokines, and endothelial biomarkers may further clarify its pleiotropic mechanisms. Incorporating lifestyle interventions such as structured exercise and dietary modification alongside telmisartan therapy could enhance outcomes. Clinicians should consider telmisartan as a preferred ARB in metabolic syndrome, especially among patients with hypertension, insulin resistance, and increased cardiovascular risk, given its favorable metabolic and vascular profile.

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Abbreviations

MetS – Metabolic Syndrome
ARB – Angiotensin II Receptor Blocker
PPAR-γ – Peroxisome Proliferator-Activated Receptor Gamma
BP – Blood Pressure
SBP – Systolic Blood Pressure
DBP – Diastolic Blood Pressure
FBG – Fasting Blood Glucose
HOMA-IR – Homeostatic Model Assessment for Insulin Resistance
HDL-C – High-Density Lipoprotein Cholesterol
LDL-C – Low-Density Lipoprotein Cholesterol
PWV – Pulse Wave Velocity
FMD – Flow-Mediated Dilation
BMI – Body Mass Index
CVD – Cardiovascular Disease

T2DM – Type 2 Diabetes Mellitus
RAAS – Renin–Angiotensin–Aldosterone System

Source of funding

The study had no funding.

Conflict of interest

The authors declare no conflict of interest.

Author contributions

AA-Concept and design of the study, results interpretation, review of literature and preparing first draft of manuscript. Statistical analysis and interpretation, revision of manuscript.MPK-Concept and design of the study, results interpretation, review of literature and preparing first draft of manuscript, revision of manuscript. NRT-Review of literature and preparing first draft of manuscript. Statistical analysis and interpretation.

Data availability

Data available on request

Author Biography

Dr. Anand Acharya, MBBS, MD (Pharmacology), currently serves as Dean and Professor, Department of Pharmacology, at the Konaseema Institute of Medical Sciences & Research Foundation (KIMS&RF), Amalapuram, Andhra Pradesh, India. A distinguished academician, researcher, and medical education leader, he has been pivotal in transforming KIMS&RF from its formative phase into a premier medical institution with over 200 undergraduate and 100 postgraduate seats.

With more than 18 years of teaching and administrative experience, Dr. Acharya has held several leadership positions including Vice Principal, Principal, Chief Warden, Member Secretary of Institutional Ethics and Animal Ethics Committees, and is an approved PhD Guide under Dr. NTR University of Health Sciences, Vijayawada. His visionary leadership has significantly enhanced the institution's academic quality, clinical exposure, research infrastructure, and postgraduate training standards.

He has successfully completed prestigious national faculty development programs such as the Revised Basic Course Workshop (rBCW), Advance Course in Medical Education (ACME), and National Teacher Training Course (NTTC, JIPMER, Puducherry). He also serves as Coordinator for Pharmacovigilance and Materiovigilance Programs under IPC–PvPI and MoHFW, Government of India, contributing actively to national drug safety and regulatory initiatives.

A prolific academician, Dr. Acharya has authored and co-authored more than 100 scientific publications in reputed national and international indexed journals. His wide-ranging research covers toxicology, pharmacovigilance, antimicrobial resistance, endocrinology, neuropharmacology, and clinical pharmacology. His recent studies include long-term analyses of pyrethroid, paraquat, and chlorpyrifos poisoning, investigations into antimicrobial resistance trends, and predictive models for treatment outcomes in dermatological and toxicological emergencies.

Dr. Acharya's professional interests include clinical pharmacology, toxicovigilance, rational drug use, pharmacovigilance systems, and innovations in medical education technologies. He continues to mentor numerous postgraduate and undergraduate researchers while playing an integral role in curriculum reform, ethics governance, and institutional academic advancement.ORCID iD: https://orcid.org/0009-0000-7967-9092

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over 16 years of teaching experience in various medical institutions, Dr. Nageswararao has made significant contributions to the field of pharmacology. His academic work includes nine research publications in reputed national and international journals, including one article indexed in PubMed. His dedication to research and education continues to enrich the academic and clinical landscape of medical pharmacology. ORCID iD:https://orcid.org/0009-0005-1638-6897

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