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Telmisartan Adverse Drug Reactions: A Clinical Pharmacist's Review with Case Insight

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ABSTRACT

Telmisartan is a long-acting angiotensin II receptor blocker widely used for hypertension management. Although generally well tolerated, it has been associated with various adverse drug reactions (ADRs) ranging from mild symptoms to serious events. To review telmisartan's ADR profile from a clinical pharmacist's perspective and illustrate key insights through a case of drug-induced acute kidney injury (AKI). A comprehensive literature review and analysis of the Pharmacovigilance Programme of India data were conducted. A 40-year-old male who developed pre-renal AKI following dose escalation of telmisartan is presented. Common ADRs include dizziness, headache, diarrhoea, and respiratory symptoms. Serious reactions encompass hyperkalemia, angioedema, hypotension, and AKI. PvPI reports have highlighted rare cutaneous reactions such as lichenoid keratosis. In the case study, serum creatinine rise from 1.3 to 1.8 mg/dL within 48 hours of increasing the telmisartan dose and normalized after discontinuation. Vigilant monitoring and pharmacist-led interventions are essential to detect and manage telmisartan-related ADRs, optimize therapy, and ensure patient safety.

Keywords: Telmisartan; Adverse Drug Reactions; Pharmacovigilance; Clinical Pharmacy; Acute Kidney Injury

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INTRODUCTION

Hypertension remains one of the most prevalent chronic conditions globally, affecting approximately one billion individuals worldwide and serving as a leading risk factor for cardiovascular morbidity and mortality. The effective management of hypertension is crucial for preventing complications such as stroke, myocardial infarction, heart failure, and chronic kidney disease. Among the various classes of antihypertensive agents, angiotensin II receptor blockers (ARBs) have emerged as first-line therapeutic options due to their efficacy in lowering blood pressure and their favourable tolerability profiles.

Telmisartan, introduced to clinical practice in 1998, represents a distinctive member of the ARB class with unique pharmacokinetic and pharmacodynamic properties. Unlike other ARBs, telmisartan possesses the longest elimination half-life of approximately 24 hours, the highest lipophilicity, and the largest volume of distribution, which contribute to its sustained antihypertensive effects and once-daily dosing convenience. Additionally, telmisartan demonstrates unique pleiotropic effects through partial activation of peroxisome proliferator-activated receptor-gamma (PPAR-γ), offering potential metabolic benefits beyond blood pressure reduction.

Despite its therapeutic advantages, telmisartan, like all pharmacological agents, is associated with various adverse drug reactions (ADRs) that can significantly impact patient safety and treatment adherence. Clinical studies have reported common adverse effects, including dizziness, headache, diarrhoea, and upper respiratory tract symptoms, while serious reactions such as hyperkalemia, acute kidney injury, and angioedema, though rare, pose considerable clinical concerns. The identification, monitoring, and management of these adverse effects are paramount for optimizing therapeutic outcomes while minimizing patient harm.

Clinical pharmacists play an increasingly vital role in pharmacovigilance activities, particularly in the detection, assessment, and reporting of ADRs in clinical practice. Their specialized knowledge of drug properties, mechanisms of action, and patient-specific factors positions them uniquely to identify drug-related problems and implement appropriate interventions. The integration of clinical pharmacy services in healthcare systems has demonstrated significant improvements in medication safety, reduction in ADR incidence, and enhanced patient care quality.

This review aims to provide a comprehensive analysis of telmisartan's adverse drug reaction profile from a clinical pharmacist's perspective, incorporating current evidence on its safety profile, risk factors, and management strategies. Through detailed examination of reported ADRs and presentation of a relevant clinical case study, this manuscript seeks to enhance understanding

of telmisartan-related safety concerns and guide clinical decision-making in its therapeutic use. [1-8]

Drug profile

The generic name of the drug is Telmisartan. Its well-known brand name is *Micardis*, with other available brands including *Telpres*, *Tazloc*, and *Cresar*.[9]

Dose: 20mg, 40mg,80mg

Mechanism of Action

Telmisartan belongs to the class of angiotensin II receptor blockers (ARBs). It selectively blocks the binding of angiotensin II to AT1 receptors, which mediate vasoconstriction and aldosterone secretion. Inhibiting this pathway promotes vasodilation, reduces sodium and water reabsorption, and lowers blood pressure.[10]

Indications

- Treatment of hypertension to reduce blood pressure.
- Prevention of cardiovascular events such as stroke and myocardial infarction.
- Cardiovascular risk reduction in patients intolerant to ACE inhibitors.

Pharmacokinetics:

Rapidly absorbed after oral intake with peak plasma levels within 0.5–1 hour. Bioavailability is around 50%, slightly reduced by food. Highly protein-bound (99.5%) with extensive tissue distribution. Metabolized by glucuronidation in the liver and mainly excreted in faeces. Elimination half-life averages 24 hours, enabling once-daily dosing.

Pharmacodynamics:

Provides selective AT1 receptor blockade, reducing vasoconstriction, aldosterone activity, and sodium reabsorption. Sustained receptor binding confers 24-hour antihypertensive action.

Dosing:

Usual starting dose is 40 mg once daily. Doses may be adjusted between 20–80 mg depending on patient response. For cardiovascular risk reduction, 80 mg once daily is standard.

Formulations:

Tablets available in 20 mg, 40 mg, and 80 mg strengths. Also available in fixed-dose combinations with hydrochlorothiazide or amlodipine.[11]

Expected Side Effects

Common: Dizziness, headache, diarrhoea, sinus congestion, and back pain.

Serious: Hyperkalemia, irregular heartbeat, swelling, respiratory difficulty.

Rare: Renal impairment, severe hypotension, and angioedema (facial and throat swelling).

Contraindications

- Anuria
- Pregnancy (second and third trimesters)
- Severe hepatic impairment, cholestasis, or biliary obstructive disorders
- Severe renal dysfunction
- Refractory hypokalemia or hypercalcemia

Drug-Drug Interactions

- Telmisartan + Aliskiren leads to high blood pressure, hypotension, hyperkalemia, and renal impairment.
- Telmisartan + Sparsentan leads to hypotension, syncope, hyperkalemia, and changes in renal function.
- Telmisartan + colchicine leads to increased risk of colchicine toxicity.
- Telmisartan + digoxin leads to increased risk of digoxin toxicity.

ADR overview

Under the Pharmacovigilance Programme of India (PvPI), telmisartan has been reported to cause lichenoid keratosis, a rare cutaneous adverse reaction, prompting multiple safety alerts and newsletters recommending healthcare professionals and patients to vigilantly monitor for and report similar events to enhance national signal detection and patient safety.

Reported ADRs

Telmisartan has been reported to cause lichenoid keratosis, prompting safety alerts and underscoring the importance of vigilant monitoring and reporting of this reaction. [12-18]

Case study

A 40-year-old male presented to the emergency department with progressively worsening breathlessness and cough. On admission, his blood pressure was 170/100 mm Hg, and he was hypoxic, though he reported no chest pain. Physical examination showed no dehydration or pedal oedema, and systemic examination was otherwise unremarkable. The patient denied fever and had no family history of cardiovascular or renal disease.

Past Medical History

- Congestive cardiac failure
- Type 2 diabetes mellitus (4 years)
- Hypertension (11 years)
- Dilated cardiomyopathy with severe mitral regurgitation (off medication)
- Ex-alcoholic (stopped 1 year prior), non-smoker

His BMI was 21 kg/m², consistent with a healthy weight. He followed a diabetic diet.

Current Medications

- Digoxin 0.25 mg (five days per week)
- Telmisartan (Telmer) 20 mg daily
- Human Mixtard insulin 10U (morning dose)

Clinical Findings and Investigations

Hemogram: Elevated blood urea (31.7% above normal), other parameters within normal range

Chest X-ray: Cardiomegaly

ECG: Sinus tachycardia and left ventricular hypertrophy

Echocardiography: Severe left ventricular systolic dysfunction, global hypokinesia, moderate pulmonary arterial hypertension, and dilation of the left atrium and ventricle

Hospital Course

The patient was admitted with acute decompensated heart failure, severe left ventricular dysfunction, hypokalemia, and suspected renal impairment. He was initially stabilized with IV furosemide and NTG infusion, leading to symptomatic improvement.

Due to uncontrolled hypertension, the Telmisartan dose was increased from 20 mg to 40 mg daily. Within 48 hours, serum creatinine levels rise from 1.3 mg/dL to 1.8 mg/dL, and routine renal function tests showed deterioration.

Interventions

- Potassium correction with oral potassium chloride (30 mL/day) and spironolactone (25 mg/day)
- Sodium correction with Tolvaptan (15 mg/day)
- Blood sugar remained controlled
- Nephrology consultation obtained

Telmisartan was discontinued considering the likelihood of drug-induced acute kidney injury (AKI) and was replaced with Prazosin 1.5 mg/day, later titrated to 2.5 mg/day. Blood pressure normalized, NTG infusion was reduced, and supportive therapy with sodium bicarbonate 500 mg BD was initiated. Loop diuretic therapy was continued for cardiac management, with careful hydration balance.

Outcome

The patient's clinical status improved following discontinuation of Telmisartan. Serum creatinine levels fell to near-normal values (1.5 mg/dL). He was discharged with:

- Prazosin 2.5 mg once daily

- Sodium bicarbonate 500 mg twice daily
- Furosemide (Lasix) 60 mg once daily

At one-week follow-up, renal parameters had normalized and recovery was ongoing. No other cause for the acute kidney injury was identified. The temporal association and resolution after drug withdrawal strongly implicated Telmisartan as the causative factor. A rechallenge with the drug was not attempted.

Final Diagnosis

- Acute decompensated heart failure with severe left ventricular dysfunction
- Telmisartan-induced acute kidney injury (pre-renal)
- Hypokalemia and hyponatremia (corrected during hospital stay)

Mechanism behind ADRs

Telmisartan's ADRs arise from its primary pharmacological actions and off-target effects. Efferent arteriole dilation reduces glomerular filtration pressure, which in patients with compromised renal perfusion such as those with heart failure or on aggressive diuresis—can precipitate pre-renal acute kidney injury. Hyperkalemia results from decreased aldosterone secretion and reduced potassium excretion in the distal nephron. Severe hypotension may occur when systemic vasodilation exceeds compensatory responses, particularly after dose escalation or in volume-depleted states. Angioedema likely stems from local bradykinin accumulation; although telmisartan does not inhibit bradykinin breakdown directly, ARB-induced endothelial changes may enhance vascular permeability. Lichenoid keratosis and sprue-like enteropathy appear immune-mediated, with late-onset T-cell–driven mucocutaneous injury, analogous to Olmesartan-associated enteropathy, suggesting a class effect involving cytokine dysregulation and epithelial apoptosis. Understanding these mechanisms informs risk stratification, early detection, and targeted management of telmisartan-related ADRs.

Risk Factors - Factors Contributing to Acute Kidney Injury

Pre-existing Cardiovascular Disease

The patient had acute decompensated heart failure with severe left ventricular dysfunction, conditions that inherently compromise renal perfusion and predispose to pre-renal AKI.

Use of Telmisartan (ARB Therapy)

Although Telmisartan is beneficial in hypertension and heart failure, its mechanism of dilating efferent arterioles can lower glomerular filtration pressure. In the setting of already reduced renal perfusion, this effect likely contributed to the onset of AKI.

Dose Escalation of Telmisartan

An increase in Telmisartan dosage from 20 mg to 40 mg daily was temporally associated with a swift creatinine rise (1.3 to 1.8 mg/dL within 48 hours), indicating a direct drug-induced impact on renal function.

Volume Depletion from High-Dose Diuretics

Administration of IV furosemide and continued diuretic therapy likely caused intravascular volume depletion, which further impaired renal perfusion and potentiated AKI.

Electrolyte Imbalances

The presence of hypokalemia and hyponatremia signalled renal dysfunction and possible over diuresis. These imbalances not only worsened renal outcomes but also increased cardiac risk.

Underlying Hypertension

Persistent, uncontrolled hypertension added additional strain on both cardiac and renal systems, complicating patient management and contributing to renal compromise.

Age and Comorbidities

The patient's diabetes, long-standing hypertension, and possible age-related decline in renal reserve increased vulnerability to medication-induced renal injury and heightened overall clinical risk.

Management and monitoring

The patient demonstrated significant symptomatic improvement, with serum creatinine levels decreasing to near-normal at 1.5 mg/dL. At discharge, his prescribed medications included Prazosin 2.5 mg once daily, Sodium Bicarbonate 500 mg twice daily, and Furosemide (Lasix) 60 mg once daily. He was instructed to attend a follow-up visit after one week. At follow-up, renal function tests had normalized and continued to show progressive recovery. In the absence of other contributing factors, Telmisartan was identified as the most probable cause of the pre-renal acute kidney injury.[19-26]

DISCUSSION

This case describes a middle-aged male with a background of congestive cardiac failure, long-standing hypertension, and type 2 diabetes mellitus who presented with acute decompensated heart failure (ADHF), severe left ventricular systolic dysfunction, and worsening renal function. On admission, he had elevated blood pressure, hypoxia, and imaging findings of cardiomegaly. Echocardiography revealed severe left ventricular dysfunction with global hypokinesia, chamber dilation, and moderate pulmonary hypertension, indicating advanced dilated cardiomyopathy.

Of particular concern was the rapid deterioration of renal function following escalation of Telmisartan therapy. His serum creatinine increased from 1.3 mg/dL to 1.8 mg/dL within 48 hours

of dose adjustment from 20 mg to 40 mg/day. Telmisartan, an angiotensin II receptor blocker (ARB), lowers glomerular filtration pressure by dilating the efferent arteriole. In individuals with compromised renal perfusion—such as those with ADHF on intensive diuretic therapy—this mechanism may precipitate acute pre-renal kidney injury.

Although other contributing factors were present, including loop diuretic use, potential volume depletion, and underlying cardiac dysfunction, the temporal association between Telmisartan dose escalation and renal deterioration strongly suggests a causal relationship. This is further supported by the patient's renal recovery, with creatinine returning to near-normal values (1.5 mg/dL), following withdrawal of Telmisartan. In line with clinical best practice, rechallenge was avoided to prevent re-exposure to a potentially nephrotoxic agent, and therapy was shifted to Prazosin, a safer antihypertensive alternative in this context.

Supportive measures such as careful diuretic titration, NTG infusion, correction of electrolyte imbalances, and nephrology consultation were essential for stabilization. Importantly, blood cultures were negative, ruling out sepsis as a potential contributing factor to the renal insult. The clinical course highlights the complexity of managing cardiorenal syndrome, where both therapeutic interventions and underlying pathophysiology can adversely impact renal outcomes.

This case underscores the necessity of cautious renal function monitoring in patients with advanced heart failure who are prescribed ARBs. It illustrates the fine balance required between optimizing cardiac function, ensuring effective blood pressure control, and minimizing renal complications. Individualized treatment approaches remain crucial for minimizing drug-related adverse events, especially in vulnerable populations with multiple comorbidities.

CONCLUSION

Telmisartan remains an effective and widely prescribed angiotensin II receptor blocker for the management of hypertension and cardiovascular risk reduction. Its favorable pharmacokinetic profile supports once-daily dosing and sustained therapeutic effects. However, despite its general tolerability, telmisartan use can be associated with a range of adverse drug reactions, ranging from common mild symptoms such as dizziness and headache to rare but serious events including hyperkalemia, angioedema, and acute kidney injury. The clinical pharmacist plays an essential role in early identification, monitoring, and management of telmisartan-related ADRs, thereby improving patient safety and treatment adherence. Pharmacovigilance data, including reports from the Pharmacovigilance Program of India, underscore the importance of vigilant safety surveillance, especially for rare cutaneous reactions like lichenoid keratosis.

The presented case of telmisartan-induced pre-renal acute kidney injury highlights the need for cautious dose titration, consideration of patient comorbidities, and prompt intervention when renal impairment is suspected. Individualized care, frequent renal function monitoring, and interdisciplinary collaboration between physicians and clinical pharmacists are critical for optimizing therapeutic outcomes and minimizing harm. This review emphasizes the need for ongoing education, awareness, and proactive pharmacovigilance to ensure the safe use of telmisartan in clinical practice.

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Conflict of Interest:

The authors declare that there are no conflicts of interest regarding the publication of this article.

AUTHORS CONTRIBUTIONS:

B.D.S.L., S.S.J., A.S.S.S.T., and B.B.S.S. conducted the literature review, performed data collection, and contributed to drafting the manuscript. J.S.V. assisted with reference compilation, formatting, and preliminary revisions. D.V.K. and D.N. provided critical academic guidance, supervision, and validation of the clinical content. A.V.K.S.G. conceptualized the study, coordinated the review process, finalized the manuscript, and ensured its overall intellectual integrity. All authors read, reviewed, and approved the final version of the manuscript.

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