Archives of Anesthesiology and Critical Care (Sept-Oct 2026); 12(5): 539-549.

Available online at http://aacc.tums.ac.ir

Review Article

The Impact of Telmisartan on Inflammation and Oxidative Stress in Human Studies and Murine Models: A Narrative Review

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ARTICLE INFO

Article history:
Received 22 May 2026
Revised 13 June 2026
Accepted 28 June 2026

Keywords:
Angiotensin II type 1 receptor blockers;
Telmisartan;
Inflammation;
Oxidative stress;
Immunomodulation;
PPAR gamma

ABSTRACT

Chronic inflammation and oxidative stress play a critical role in conditions such as atherosclerosis, metabolic syndrome, and diabetic nephropathy. Telmisartan is a unique long-acting ARB due to its dual mechanism of action. In addition to blocking the AT1R, it acts as a partial agonist for peroxisome proliferator-activated receptor-gamma (PPAR-γ). It is also characterized by high lipophilicity compared to other drugs in its class, facilitating tissue penetration into the central nervous system. The objective of this narrative review is to bridge the gap between murine models of oxidative stress and inflammation and human clinical outcomes across the cardiovascular, renal, hepatic, neurological, and autoimmune systems. Across extensive human clinical trials and murine models, telmisartan demonstrates unique potential as a multi-system anti-inflammatory and antioxidant agent that actively suppresses pro-apoptotic and fibrotic cascades. Beyond these systemic effects, telmisartan confers localized vascular benefits by actively stabilizing coronary plaques and suppressing cardiac inflammation. While its cardiovascular and renal benefits are the most widely investigated, it also demonstrates profound extra-cardiac efficacy. In particular, telmisartan improves neuroinflammation in neurodegenerative diseases, metabolic profiles in hepatic steatosis, and protects against gastrointestinal and musculoskeletal inflammation. But its protective effects are limited for established HIV fibrosis and severe primary autoimmune conditions like psoriasis. In conclusion, telmisartan is a cardiometabolic and immunomodulatory drug, extending the clinical use beyond hypertension.

Introduction

The Renin-Angiotensin System (RAS) controls blood pressure, blood vessel tone, and salt-water homeostasis via the cardiorenal axis [1]. Overactivation of the RAS is critical in hypertension pathogenesis and is a major therapeutic target for standard antihypertensive therapies, including angiotensin-converting enzyme (ACE) inhibitors, angiotensin II receptor blockers (ARBs), and angiotensin receptor-neprilysin inhibitors (ARNIs) [2]. ARBs block the angiotensin II activation of the AT1 receptor, thus preventing its vasoconstrictive and aldosterone-stimulating effects [3]. RAS activation also enhances the production of reactive oxygen species (ROS),

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DOI: 10.18502/aacc.v12i5.22437

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inflammation, and fibrosis via activation of the angiotensin II type 1 receptor (AT1R) [4]. The pathogenesis of atherosclerosis, metabolic syndrome and diabetic nephropathy is mediated by chronic inflammation and oxidative stress [5-7].

Telmisartan is a long-acting ARB and is more lipophilic than other drugs of its class which can facilitate tissue penetration including the central nervous system [8]. Telmisartan is unique in its dual mode of action. In addition to blocking the AT1R, it also partly activates peroxisome proliferator-activated receptor-gamma (PPAR-γ) [9]. Telmisartan through this action down regulates the pro-inflammatory markers like nuclear factor kappa B (NF-κB), tumor necrosis factor-α (TNF-α), interleukin-1 (IL-1), and cyclooxygenase-2 (COX-2) [10]. In vitro studies have demonstrated that telmisartan, but not losartan, can selectively neutralize hydroxyl radicals and protect endothelial cells from oxidative damage [11]. The potential applications of telmisartan extend outside cardiology and are currently being investigated for neurodegenerative diseases, chemotherapy toxicity, and autoimmune disorders [12-20].

While previous research mainly focuses on cardiovascular effects, there is a need to evaluate both cardiac and extra-cardiac applications of telmisartan in reducing inflammation and oxidative stress. This narrative review connects murine molecular models of oxidative stress and inflammation with human clinical outcomes across the cardiovascular, renal, hepatic, neurological, and autoimmune systems.

Cardiovascular and Endothelial Protection

Atherosclerosis, Plaque Stability, and Stent Restenosis

Telmisartan's anti-inflammatory differs from other RAS blockers, largely due to its capacity to engage the PPAR-γ pathway. This difference is evident in clinical comparisons regarding stent restenosis. Both valsartan and telmisartan are capable of lowering high-sensitivity C-reactive protein (hs-CRP) levels after implantation of sirolimus-eluting stent in hypertensive patients, but telmisartan has a second anti-inflammatory effect through reduction of upstream cytokines, such as interleukin-6 (IL-6) and TNF-α. This upstream suppression might explain the reduced late lumen loss, protecting against stent restenosis [21]. This supports the theory that the mechanism of telmisartan, as a PPAR-γ-driven molecule, acts directly on cytokine production. Telmisartan also shows superior efficacy in reducing hs-CRP levels compared to the ACE inhibitor ramipril 20 days after an acute coronary syndrome [22]. Although both telmisartan and enalapril mitigate inflammation by decreasing hs-CRP in patients with coronary artery disease, telmisartan uniquely activates the PPAR-γ pathway to elevate protective high-molecular-weight adiponectin levels [23]. Telmisartan also confers localized vascular benefits by stabilizing coronary plaques and suppressing cardiac inflammation. Integrated backscatter intravascular ultrasound (IB-IVUS) reveals that this ARB significantly reduces local levels of inflammatory cytokines (MMP-3, TNF-α, and hs-CRP) in the coronary sinus, even when these markers remain largely unchanged in the peripheral venous circulation [24].

Murine models of atherosclerosis show how this reduction in lesion formation occurs at the cellular level. By inactivating NAD(P)H oxidase, telmisartan reduces superoxide production and prevents its localization within the aortic vessel wall, specifically at the endothelium, to reduce overall atherosclerotic lesion formation [25]. In advanced atherosclerotic lesions, while systemic inflammatory markers like serum amyloid A (SAA) remain unchanged, telmisartan reduces the absolute accumulation of macrophages, which are the primary inflammatory cells driving atherosclerotic plaque formation [25-26]. In contrast, ACE inhibitors like ramipril do not significantly alter macrophage content within the plaques [26]. This suggests that telmisartan's ability to suppress ROS indirectly inhibits the downstream inflammatory cascades responsible for plaque growth [25].

In addition to ROS suppression, telmisartan directly inhibits local aortic inflammation in apolipoprotein E-deficient mice by reducing the expression and DNA-binding activity of the pro-inflammatory transcription factors Egr-1 and NF-κB [26]. These findings parallel human clinical observations. For example, while both telmisartan and losartan effectively decrease the mRNA expression of IL-6 in the aorta of ApoE-deficient mice, only telmisartan increases local vascular hepatocyte growth factor (HGF) expression. This effect is likely mediated by telmisartan's partial activation of PPAR-γ [27].

Ventricular Remodeling, Hypertrophy (LVH), and Heart Failure

Telmisartan and enalapril successfully reduce left ventricular remodeling and matrix metalloproteinase (MMP)-2 and MMP-9 activities in patients recovering from acute myocardial infarction (MI) after percutaneous coronary intervention (PCI). Telmisartan provides an additional benefit of suppressing vascular inflammation by significantly lowering pentraxin 3 (PTX3) levels [28]. In patients with essential hypertension, telmisartan has shown a superior effect to amlodipine by reducing both monocyte chemoattractant protein-1 (MCP-1) mRNA

expression in peripheral monocytes and overall serum MCP-1 levels. This suppression of MCP-1 inversely correlates with increased PPAR-γ mRNA expression [29]. In experimental models, PPAR-γ activation allows telmisartan to directly block downstream leptin signaling (STAT3 phosphorylation) to improve myocardial remodeling [30].

Telmisartan inhibits vascular remodeling in hypertension through blood pressure regulation and reduction of oxidative stress. The effect might be due to the upregulation of superoxide dismutase (SOD) and glutathione peroxidase (GPx) activity and downregulation of MDA production. Telmisartan prevents tissue remodeling by blocking the profibrotic TGF-β1/Smad3 signaling pathway [30-31]. In models of diabetic cardiomyopathy, telmisartan combined with the protein Klotho protects myocardial structure and reduces fibrosis by modulating TGF-β1, pSMAD-2/3, MMP-9, and pFOXO3a signaling networks [32].

Ischemia/Reperfusion (I/R) Injury and Myocardial Infarction (MI)

Telmisartan has several different protective pathways against myocardial ischemia/reperfusion injury and infarction. It mainly suppresses oxidative stress and apoptosis through the activation of AMP-activated protein kinase (AMPK) [33]. Telmisartan dose-dependently upregulates the body's own antioxidant defense by increasing the levels of reduced glutathione (GSH) and SOD and decreasing the levels of MDA in isoproterenol-induced MI in diabetic rats [34].

Telmisartan inhibits the pro-apoptotic protein Bax and upregulates the anti-apoptotic protein Bcl-2 and reduces TNF-α via the PPAR-γ pathway in diabetic models [35]. At the same time, telmisartan protects the myocardium in an AMPK-independent manner, which enhances calcium homeostasis and activates LKB1 [33]. Telmisartan decreases IL-17 expression and disrupts cardiac electrical conduction in post-MI myocardial tissue. Such a decrease leads to an upregulation of connexin 43 (Cx43) expression, which stabilizes gap junctions and prevents arrhythmias [36].

Iatrogenic and Environmental Toxicity

Chemotherapy-Induced Cardiotoxicity

In clinical trials of cancer patients receiving epirubicin, telmisartan prevents elevations in serum IL-6 and ROS levels. Telmisartan reverses the early reduction in myocardial strain rate, protects against chronic anthracycline-induced cardiotoxicity, and preserves systolic cardiac function throughout an 18-month follow-up period [15-16].

Chemotherapy-Induced Nephrotoxicity

Telmisartan protects against doxorubicin-, daunorubicin- and cisplatin-induced nephrotoxicity in rat models. The drug upregulates the PPAR-γ expression to effectively reduce MDA, restore GPx activity and ameliorate renal function in daunorubicin treated rats [14]. Protective effects against oxidative stress and inflammation are seen in cisplatin-induced nephrotoxicity, where telmisartan suppresses MDA levels, preserves endogenous GSH, SOD, and CAT levels, and reduces serum TNF-α [18]. Via the inhibition of the MAPK pathway, telmisartan lowers elevated caspase-3 levels, reduces the number of apoptotic (TUNEL-positive) tubular cells, decreases pro-apoptotic Bax, and increases anti-apoptotic Bcl-2 [18]. Both telmisartan and captopril decrease doxorubicin-induced oxidative stress by lowering MDA levels, preventing GSH depletion, and reducing the compensatory elevation of GPx and SOD activities [17].

Drug-Induced Toxicity

Telmisartan shows robust protective efficacy against drug-induced ototoxicity and heavy metal toxicities. In models of aminoglycoside-induced ototoxicity, telmisartan reduces the markers of lipid peroxidation in cochlear tissue. Telmisartan alleviates the focal oxidative stress and protects the auditory hair cells from kanamycin-induced damage. At molecular standard, telmisartan dose-dependently suppressed mRNA expression of IL-1β, IL-6 and TNF-α. It suppresses the up-regulation of both inducible nitric oxide synthase (iNOS) and COX-2, thereby preventing the overproduction of nitric oxide (NO) and inflammatory tissue damage mediated by prostaglandins. Telmisartan blocks the downstream apoptotic cascade by reducing levels of cleaved caspase-3 and caspase-7 [37].

Heavy Metal and Environmental Toxicity

Against subchronic arsenic exposure, telmisartan suppresses superoxide anion generation, mitigating aortic lipid peroxidation and restoring depleted SOD, CAT, GPx, and glutathione reductase (GR) activities [38]. This restoration of redox homeostasis is accompanied by lowered aortic levels of IL-6, IL-1β, and TNF-α and counters COX-2 activity to reduce prostaglandin E2 (PGE2) expression [38]. Similar protective mechanisms are also observed in arsenic-induced hepatotoxicity, where telmisartan administration inhibits hepatic lipid peroxidation, prevents GSH depletion, downregulates TNF-α, and reduces apoptosis by decreasing caspase-3 expression [39]. Telmisartan prevents cadmium-induced nephrotoxicity by inhibiting lipid peroxidation and reducing the expression of TNF-α and NO, and by

preventing tubular cell apoptosis by reducing cleaved caspase-3 [40].

Renal Pathology and Protection

Diabetic Nephropathy and Microalbuminuria

Telmisartan demonstrated superiority over valsartan in reducing the urinary albumin-creatinine ratio (UACR) in hypertensive patients with metabolic syndrome. Both drugs are equally effective in managing blood pressure, plasma glucose, hemoglobin A1c (HbA1c), immunoreactive insulin (IRI), and the homeostasis model assessment of insulin resistance (HOMA-IR). The therapeutic advantage of telmisartan in reducing microalbuminuria appears independent of class-effect hemodynamics or glycemic control. Instead, it is driven by its anti-inflammatory properties and capacity to upregulate adiponectin via partial PPAR-γ activation. Unlike valsartan, telmisartan significantly reduces serum hs-CRP and IL-6 levels while increasing adiponectin. The reduction in hs-CRP is as an independent predictor of the reduction in UACR [41].

This ability to protect endothelial function extends into acute inflammatory settings. In patients with type 2 diabetes mellitus undergoing coronary artery bypass grafting (CABG), pretreatment with telmisartan weakens the severe postoperative systemic inflammatory response. Telmisartan decreases microalbuminuria by significantly reducing postoperative hs-CRP levels and preserves renal function during acute surgical stress [42].

In murine models of diabetic nephropathy, telmisartan monotherapy suppresses macrophage infiltration and reduces expression of CD68, MCP-1, intercellular adhesion molecule-1 (ICAM-1), and osteopontin in the diabetic kidney [43]. Hypothetically, these effects are mediated through several mechanisms including suppression of oxidative stress by reduction of 8-OHdG and Nox4 expression [43], mitigation of inflammatory signaling by reduction of phosphorylation of p38 MAPK, MAPKAPK-2, and Akt, and elevation of protective PPAR-γ protein expression [44]. Telmisartan also decreases the AT1R and the pro-fibrotic cytokine TGF-β [43-44].

Telmisartan reduces blood urea nitrogen (BUN) and serum creatinine and protects kidney function when combined with α-Klotho. The combination modulates apoptosis by upregulating Bcl-2 and downregulating Bax, and reinforces anti-inflammatory and anti-fibrotic defenses by suppressing phosphorylated NF-κB levels and downregulating both AT1R and TGF-β1 expression [45].

Chronic Kidney Disease (CKD), UUO, and Kidney I/R Injury

Telmisartan has a strong anti-inflammatory effect in cardiometabolic diseases via PPAR-γ activation and adiponectin up-regulation. It clears asymmetric dimethylarginine (ADMA) from the blood stream to restore NO bioavailability and to attenuate oxidative stress. Telmisartan is uniquely superior to other ARBs in ADMA reduction independent of blood pressure, and this effect is likely mediated by modulation of PPAR-γ [46-47].

In rat models of acute renal ischemia-reperfusion (I/R) injury, telmisartan was shown to afford structural protection by decreasing total oxidant status (TOS) and oxidative stress index (OSI). It inhibits tubular apoptosis as indicated by decreases in TUNEL-positive apoptotic cell counts and total apoptosis scores [48].

In models of chronic obstructive nephropathy, unilateral ureteral obstruction (UUO) induces severe activation of NADPH oxidase subunits (p22-phox, p47-phox, and p67-phox) within the renal tubules of acatalasemic mice. Telmisartan treatment dose-dependently suppresses the tubular protein and mRNA expression of these ROS-generating subunits and simultaneously reduces the accumulation of lipid peroxidation products, including HNE and HHE [49].

Metabolic and Systemic Immunology

Non-Alcoholic Fatty Liver Disease (NAFLD) and Steatohepatitis (NASH)

In murine models of nonalcoholic steatohepatitis (NASH), telmisartan reduces the number of hepatic infiltrating inflammatory cells by suppressing the infiltration of F4/80-positive macrophages and decreasing the formation of foamy macrophages [50]. It also downregulates the hepatic mRNA expression of MCP-1, its receptor CCR2, and TNF-α [50]. These anti-inflammatory effects such as decreasing serum alanine aminotransferase (ALT) and TNF-α levels are mostly through the activation of PPAR-γ responsive element-dependent transcription and the reestablishment of hepatic PPAR-γ expression [51-52].

The direct blockade or genetic deletion of the AT1R upregulates PPAR-α and enhances fatty acid oxidation, protecting against hepatic lipid accumulation [53]. Telmisartan decreases markers of oxidative stress, fibrosis, and apoptosis and restores hepatic autophagy [54].

Obesity, Insulin Resistance, and Adipose Tissue

In obese hypertensive patients, telmisartan treatment significantly decreases IL-6 levels (by 2.6 times) and induces a superior increase in serum adiponectin

compared to olmesartan [55]. In hypertensive patients with obesity, telmisartan improves insulin sensitivity (HOMA-IR), elevates serum adiponectin, and decreases TNF-α levels [56]. These metabolic improvements are accompanied by a reduction in the visceral fat area, without altering subcutaneous fat [56]. In patients with metabolic syndrome, high-dose telmisartan (160 mg) significantly induces the expression of the PPAR-γ target gene CD36 in circulating monocytes [57]. Ex vivo stimulation of human primary monocytes confirm that telmisartan significantly induces CD36 mRNA and protein expression and directly augments PPAR-γ DNA binding [57]. In high-fat diet (HFD)-fed mice, telmisartan shifts adipose tissue macrophages from a pro-inflammatory to an anti-inflammatory state. It downregulates the mRNA expression of M1 markers (CD11c, TNF-α) and upregulates the expression of M2 markers (CD163, CD209, Mgl2) [58].

The inflammatory profile of adipose tissue in obesity has implications for oncogenesis. In triple-negative breast cancer (TNBC) tissue, programmed death-ligand 1 (PD-L1) is highly expressed. Expression of PD-L1 is driven by obesity and metabolic syndrome through M1 macrophages releasing IL-6 that activates JAK/STAT, more specifically JAK1/STAT1, and STAT3 signaling pathways in cancer cells. This process is antagonized by telmisartan by inhibiting NF-κB p65 and activating PPAR-γ in M1 macrophage and reducing IL-6 secretion [59].

Immunomodulation and Systemic Inflammation

In patients with type 2 diabetes mellitus and newly diagnosed hypertension, 12 weeks of treatment with telmisartan or active control (amlodipine, cilnidipine or ramipril) led to a reduction in the levels of hs-CRP, TNF-α and IL-6, with no statistically significant differences among groups [60].

Neuroinflammation and Cognitive Preservation

Neurodegenerative Diseases

Telmisartan is more effective than amlodipine in reducing the cerebrospinal fluid (CSF) IL-1β and TNF-α levels in elderly hypertensive patients who have Alzheimer's disease. Telmisartan prevents amyloid-beta 1-42 (Aβ1-42) depletion and improves global cognitive function, despite similar blood pressure control between the two drugs [12]. Similarly, in a mouse model of amyloid-β (Aβ) injection, pretreatment with low-dose telmisartan significantly improves cognitive function and cerebral blood flow, probably by improved Aβ clearance and suppressed neuroinflammation via the PPAR-γ pathway [13].

In models of Parkinson's disease (MPTP-treated mice), it inhibits the increases in iNOS, ionized calcium-binding adapter molecule 1 (IBA1), a-synuclein and Bax and preserves tyrosine hydroxylase (TH) and p-Akt/Akt signaling. These effects are mediated by modulation of the Akt/GSK3β/PGC1α pathway preserving mitochondrial biogenesis and bioenergetics, which likely underlie the observed improvements in gait and motor performance [61]. α-synuclein overexpression upregulates AT1R expression and NADPH oxidase activity. This pro-oxidative and pro-inflammatory axis is inhibited by both candesartan and telmisartan. Telmisartan reduces OX6-positive microglial cells and suppresses CD68 mRNA expression and M1 pro-inflammatory markers (iNOS, TNF-α, IL-6, and IL-1β) [62]. The proposed mechanism in this case is that telmisartan can block AT1R while at the same time activate PPAR-γ and endothelial nitric oxide synthase (eNOS), thereby improving vascular and neuronal function independent of blood pressure [13].

Telmisartan improves muscle strength and motor function in a 3-nitropropionic acid (3-NP) using rat model of Huntington's disease through antioxidative, anti-inflammatory and anti-apoptotic mechanisms. It attenuated 3-NP-induced oxidative stress by decreasing striatal MDA content and NADPH oxidase expression and increasing GSH levels. It reduces the pro-inflammatory markers (TNF-α, IL-1β, PGE2, COX-2) and inhibits apoptosis by reducing active caspase-3. The effect is mediated by modulation of the PI3K/Akt/GSK-3β and ERK 1/2 signaling cascades, and induction of PPAR-γ expression [63].

Neuroprotection in Stroke, Intracerebral Hemorrhage and Hypoxia

Telmisartan, ertugliflozin, and omaveloxolone exert neuroprotection against cerebral ischemia-reperfusion injury by activating the Nrf2/HO-1 antioxidant pathway and inhibiting NF-κB-mediated neuroinflammation through a reduction in TNF-α, IL-6, and MMP-9 levels [64]. Telmisartan suppresses NLRP3 inflammasome pathway, thus reducing cerebral edema and suppressing the production and maturation of IL-18 and IL-1β after traumatic brain injury in mice [65]. Chronic intermittent hypoxia (CIH) can induce CD45-positive neurons in the hippocampal CA1 region, increase plasma levels of CRP and IL-6 and increase regional neuronal apoptosis. Telmisartan inhibits these responses and protects hippocampus [66]. It also significantly inhibits excessive NO production and suppresses CIH-induced increases in MDA [66].

Telmisartan reduces TUNEL positive cells in the perihematomal area and decreases the activity of caspases-3, -8 and -9 involved in intrinsic and extrinsic apoptotic pathways in a rat model of intracerebral

hemorrhage (ICH). It downregulates COX-2 and decreases mRNA levels of TNF-α, Fas and FasL. It relieves oxidative stress as shown by decreased ROS generation and decreased MDA levels in the ICH rat model. These anti-inflammatory and antioxidant effects are independent of blood pressure reduction and mediated by AT1R inhibition and PPAR-γ and eNO activation [67]. In focal brain ischemia using atherosclerotic apolipoprotein E-deficient (ApoEKO) mice, telmisartan significantly reduces the ischemic brain area and neurological deficits, restores cerebral blood flow, and mitigates superoxide production, which are all independent of changes in systemic blood pressure or serum cholesterol [68].

Psychiatry and Behavioral Stress

Telmisartan mitigates rapid eye movement (REM) sleep deprivation-induced memory impairment and cognitive deficits by mitigating neuroinflammation and oxidative stress and by modulating the GSK-3β/CREB/BDNF signaling pathway. In sleep-deprived rats, elevated IL-6, IL-1β, and TNF-α in the hippocampus and prefrontal cortex are decreased by telmisartan. It reverses severe oxidative stress by restoring SOD, CAT, and GPx activities and clearing elevated levels of MDA and NO [69].

Mixed results were observed with telmisartan in a phencyclidine (PCP)-induced mouse model of schizophrenia. Telmisartan prevented reductions in IL-6 and IL-10 and prevented down-regulation of the AT1R in the frontal cortex, but induced impairments in spatial working memory and prepulse inhibition. This may represent a safety issue in some psychiatric conditions [70].

Mucosal Protection and Gastrointestinal Inflammation

Telmisartan was found to attenuate the mucosal damage and preserve the structural integrity of colon by reducing the necrosis and leukocyte infiltration in rat models of ulcerative colitis. It exerts anti-inflammatory effects by reducing myeloperoxidase (MPO) activity and levels of TNF-α and IL-10. Telmisartan protects against oxidative stress by reducing the level of MDA and by increasing the level of GSH, total antioxidant capacity (TAC) and the activities of SOD and GPx [19-20]. Telmisartan increases PPAR-γ which decreases the expression of NF-κ B and its downstream proinflammatory targets, COX-2 and iNOS. It prevents colonic epithelial cell apoptosis by downregulating pro-apoptotic genes (caspase-3, cytochrome c, and Bax) and upregulating the anti-apoptotic gene Bcl-2 [19].

Musculoskeletal Trauma and Neuropathic Pain

In a post-surgical Achilles tendon injury model in rats, both telmisartan and enalapril significantly decrease the local aggregation of inflammatory infiltrates [71]. They both reduce MDA content and restore or elevate total thiol levels, CAT activity, and SOD activity, which reduces fibrosis and improves mechanical properties, such as ultimate load capacity and stress tolerance of the healing tendons [71].

Telmisartan reduces hypersensitivity and prevents cold allodynia, mechanical hyperalgesia, heat hyperalgesia, and dynamic mechanical allodynia in a rat model of peripheral neuropathic pain induced by sciatic nerve constriction [72]. At the molecular scale, this anti-allodynic efficacy is mediated by suppression of neuroinflammation, specifically, by decreasing the elevated levels of TNF-α in the injured sciatic nerve [72].

Inflammatory Arthritis

ARBs like olmesartan, candesartan, and telmisartan modulate the immune system by inhibiting T-cell and decreasing IFN-γ production. Telmisartan significantly protects joint structure by reducing inflammation, tissue overgrowth (pannus formation), and cartilage and bone destruction in murine models of collagen-induced arthritis (CIA) [73].

Limitations and Clinical Complexities

Viral Chronic Inflammation (HIV/ART)

In HIV-infected adults who are on suppressive antiretroviral therapy (ART), telmisartan shows mixed results when contrasting its effects on deep tissue fibrosis versus systemic metabolic and inflammatory profiles. Over 48 weeks, telmisartan does not significantly alter CD4+ T-cell counts and fails to reduce collagen I, fibronectin, and pSMAD3, in lymph nodes or subcutaneous adipose tissue when compared to ART alone [74].

However, six months of telmisartan therapy in hypertensive HIV-positive men significantly reduces the serum levels of erythrocyte sedimentation rate (ESR), IL-18, and endothelin-1 while improving insulin resistance [75]. 24 weeks of telmisartan significantly increases urinary prostaglandin E metabolite (PGE-M) levels in patients with central adiposity, correlating with favorable visceral fat loss and increased subcutaneous fat and a decreased thromboxane-to-prostacyclin (TxB-M/PGI-M) ratio. These findings show that while telmisartan offers metabolic benefits, it may fail to reverse established lymphoid and adipose fibrosis in the HIV-positive population [76].

Refractory Inflammatory States

In severe psoriasis, the cardiovascular benefits of telmisartan are limited by ongoing skin inflammation. In a murine model, high-dose telmisartan lowers systolic blood pressure but fails to reduce vascular dysfunction and systemic inflammation. It does not reduce increased blood levels of reactive oxygen and nitric species and inflammatory markers such as IL-6, IL-17A, IL-1β, and granulocyte-colony-stimulating factor (G-CSF). It fails to improve skin symptoms or prevent inflammatory cells from infiltrating the aortic wall. It can be hypothesized that lowering blood pressure alone is insufficient, and vascular dysfunction and inflammation remain resistant to standard antihypertensive therapy as long as the primary skin inflammation induced by IL-17A persists [77].

Human case reports demonstrate that telmisartan may trigger severe psoriatic flares. A 45-year-old female whose chronic plaque psoriasis was previously controlled on oral methotrexate experienced a severe disease exacerbation within a month of initiating telmisartan as she developed new, widespread desquamating plaques [78]. A 50-year-old male with stable pustular psoriasis developed acute, widespread pustular eruptions and severe burning pain shortly after starting the medication [79]. In both cases, the cutaneous lesions completely resolved within a month of discontinuing telmisartan [78-79]. In the latter case, when an inadvertent re-exposure to telmisartan six months later triggered a second rapid pustular flare, the causal relationship was assumed, which again resolved rapidly upon drug cessation [79]. These cases suggest that telmisartan can paradoxically induce or exacerbate localized flares in patients with psoriasis. This exacerbation may involve complex, disease-specific alterations in the kinin-kallikrein system or arachidonic acid pathways [78].

Conclusion

Across human clinical trials and murine models, telmisartan shows multi-system anti-inflammatory and antioxidant effect that suppresses pro-apoptotic and fibrotic cascades. Its cardiovascular and renal benefits are mainly investigated, but the drug also demonstrates neuroprotective and metabolic effects. However, its protective capabilities are limited in HIV fibrosis and severe primary autoimmune conditions like psoriasis. Telmisartan serves as a cardiometabolic and immunomodulatory agent, which can be used for more than a blood pressure medication.

Ethics approval and consent to participate

Not applicable.

Data availability

This study used no original data.

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