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NEW BIOMOLECULAR MECHANISMS IN THE PATHOGENESIS OF MYOCARDIAL INFARCTION

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Abstract: This review presents data from modern scientific studies indicating the involvement of the immune system in the regulation of arterial pressure. The mechanisms of neuro-immune regulation of vascular tone, endothelial dysfunction, and modulation of cytokine status in the pathogenesis of myocardial infarction are discussed. The authors conclude that immune and inflammatory responses play a causal and predictive role in the development of myocardial infarction.

Keywords: myocardial infarction, immunity, cytokines, endothelium, cardiovascular diseases.

The immune system's first line of defense is the innate response, which is rapid; the second line, adaptive immunity, is slower to emerge but highly specific. In the context of myocardial infarction (MI), the crosstalk between these two arms appears critical [8,30]. T-cell–derived cytokines occupy a central role in the pathophysiology of cardiovascular disease (CVD) and hypertension and contribute to target-organ damage [2,3]. Among the earliest and best-characterized cytokines linked to hypertension is interleukin-17 (IL-17). T helper (Th) cells and their pro-inflammatory cytokine IL-17 are key drivers of hypertensive autoimmunity and endothelial dysfunction. Notably, $CD4^+$ T cells from hypertensive individuals produce more IL-17A than normotensive controls, while $CD8^+$ T cells from patients with hypertension produce more interferon-γ (IFN-γ) than those from normotensive controls [29].

Objective. To summarize and analyze current scientific data on the role of the immune system, cytokines, and molecular inflammatory mechanisms in the pathogenesis of myocardial infarction (MI), with particular attention to the interaction between innate and adaptive immunity, cytokine networks (IL-1, IL-6, IL-10, IL-17, TNF-α, IFN-γ, TGF-β), and activation of the renin–angiotensin–aldosterone system (RAAS) in the development of endothelial dysfunction, fibrosis, and myocardial injury.

Materials and Methods. This article is a narrative review of contemporary publications focusing on the molecular and immunological aspects of the pathogenesis of myocardial infarction and arterial hypertension.

A comprehensive literature search was conducted using the international scientific databases PubMed, Scopus, Web of Science, SpringerLink, ScienceDirect, and Google Scholar for the period 2015–2025. 46 publications were analyzed, including original research articles, meta-analyses, and systematic reviews addressing the role of cytokines, T lymphocytes, macrophages, the RAAS system, and endothelial dysfunction in cardiovascular diseases.

Results. Naive $CD4^+$ T cells polarize according to the cytokine milieu into Th1, Th2, Th17, or regulatory T (Treg) phenotypes. Th1 cells, induced by IL-12 and IFN-γ, mainly secrete IL-2, tumor necrosis factor-α (TNF-α), and IFN-γ. Th2 cells, induced by IL-4, predominantly secrete IL-4 and IL-10. Th17 differentiation requires IL-6, IL-21, IL-23, transforming growth factor-β (TGF-β), and IL-1β; it is potentiated by aldosterone and yields IL-17A, IL-17F, IL-21, and IL-22. Tregs arise in TGF-β1 with low IL-6 and exert anti-inflammatory effects via IL-9, IL-10, TGF-β, cytotoxic T-lymphocyte antigen-4 (CTLA-4), and contact-dependent mechanisms. Functional overlap among cytokines complicates attempts to ascribe hypertensive pathobiology to single mediators and likely explains variable benefits from selectively suppressing individual cytokines [36,37].

Whereas pro-inflammatory mediators (IL-17, IFN-γ, TNF-α) aggravate hypertension, IL-10 exerts protective effects. Adaptive immune responses mediated by T and B cells are engaged early, releasing cytokines and antibodies that alter renal sodium-transporter expression and vascular endothelium, and promote cardiac, renal, and perivascular fibrosis. Although robust in animal models, human data remain more limited; nevertheless, the evidence supports developing antihypertensive strategies targeting adaptive immune mechanisms [25].

Cytokines—small signaling proteins that can traverse the blood–brain barrier—serve as principal regulators of immunity and key messengers between the nervous and immune systems. Along with neuropeptides (e.g., substance P), they modulate CVD, likely participating in the onset and maintenance of hypertension. Their widespread expression in peripheral tissues and blood, and their BBB permeability, indicate they relay signals among the central nervous system, immune cells, and the cardiovascular system, thereby contributing to blood-pressure homeostasis [4].

A major advance over the past decade has been the recognition that both innate and adaptive immunity contribute to hypertension, a crucial piece in the mosaic leading to MI [12]. Angiotensin II, long known to elevate blood pressure and to trigger vascular and renal inflammation, is now understood to activate immunity as a mechanistic driver—not merely a by-product—of hypertension. The nervous and immune systems share gatekeeping roles at the interface of internal and external environments [6,32].

Immune organs are richly innervated by the autonomic nervous system; sympathetic noradrenergic fibers are prominent in primary (bone marrow, thymus) and secondary (spleen, lymph nodes) lymphoid tissues. Evidence indicates the sympathetic nervous system modulates immune and inflammatory responses, with both activating and dampening actions. The discovery of the inflammatory reflex provided a neurophysiological framework for this regulation, suggesting that fine-tuning sympathetic inputs to immune organs can shape inflammatory outputs [33].

These insights raise questions about MI and autoimmunity. Primary hypertension mirrors autoimmune mechanisms seen in systemic lupus erythematosus, psoriasis, systemic sclerosis, rheumatoid arthritis, and periodontitis—and hypertension is more prevalent in these conditions than in control populations. Inflammation and oxidative stress form a self-reinforcing loop that drives vascular dysfunction and hypertensive kidney injury. Infiltration of T and B cells, macrophages, and NK cells into kidneys and vessels is pathogenic. Effector cytokines (IFN-γ, TNF-α, IL-17) alter renal Na^+/H^+ exchange; in the vasculature, they induce endothelial dysfunction, reduce nitric-oxide bioavailability, and promote vasoconstriction. These effects are partly mediated by NADPH oxidase–derived reactive oxygen species and impaired antioxidant defenses. Accordingly, oxidative neoantigen formation (e.g., isolevuglandin adducts) has emerged as a therapeutic target [38]. Circulating levels of IL-6, TNF-α, IFN-γ, and IL-17A are elevated in hypertension, reinforcing this link [22,45].

Inflammation increases vascular permeability and releases potent mediators—reactive oxygen species, nitric oxide, cytokines, and matrix metalloproteinases (MMPs). Cytokines drive neo-intima formation, narrowing resistance-vessel lumens and fostering fibrosis, thereby raising vascular resistance and stiffness. They also enhance tubular angiotensinogen/angiotensin II synthesis and promote sodium and volume retention. MMPs degrade extracellular matrix, facilitating leukocyte transmigration, apoptosis, and collagen deposition, culminating in target-organ damage. While animal data are compelling, human evidence is largely associative (e.g., C-reactive protein, TNF, interleukins) [15,31].

Hypertension reflects a balance between pro-inflammatory T-cell responses and Treg-mediated suppression; adoptive transfer of Tregs ameliorates hypertension in several models. Dysregulation of both effector and regulatory T cells contributes to renal and vascular inflammation underpinning hypertensive end-organ injury [31]. Blood pressure severity correlates with C-peptide and lactate dehydrogenase (LDH) levels, suggesting their potential as prognostic biomarkers; in grade 2 hypertension, elevated LDH may signal evolving metabolic syndrome and apoptosis in visceral organs, including myocardium [28].

Inflamed vascular endothelium promotes leukocyte extravasation. Neutrophils adhere early to activated endothelium; IL-17 enhances neutrophil mobilization, while Th1-derived IFN-γ activates macrophages, and TNF and chemokines orchestrate broader leukocyte recruitment, with tissue injury resulting from lysosomal enzymes and pro-inflammatory mediators [1].

Renin–angiotensin–aldosterone system (RAAS) activation drives endothelial dysfunction, vascular remodeling, and hypertension. Hypertrophic remodeling enlarges vascular smooth-muscle cells and increases extracellular matrix proteins (e.g., collagen) via TGF-β activation [20]. Pattern-recognition receptors can also trigger inflammasome assembly, activating caspase-1 to mature IL-1β and IL-18. Cellular injury elevates extracellular ATP as a danger signal that further promotes IL-1 release [43].

Immunosenescence intersects with CVD and hypertension. Post-thymic involution reduces naïve T cells and expands memory—particularly $CD8^+$ effector—pools, partly due to recurrent/persistent viral infections. Senescent T cells (shortened telomeres, loss of CD27/CD28, gain of CD57) exhibit chronic pro-inflammatory activity (IFN-γ, IL-6, TNF-α) and increased cytotoxic granzyme. Such cells are found in atherosclerotic plaques and inflamed synovia. Young hypertensive individuals may harbor expanded CD28-deficient $CD8^+$ T cells producing excess IFN-γ, perforin, and granzyme. IL-6 correlates with blood pressure and decreases with angiotensin-receptor blockade; its capacity to skew T cells toward IL-17 production likely contributes to hypertension [23].

IL-1 augments sympathetic tone, causing systemic vasoconstriction and impairing natriuresis. Exogenous IL-1 infusion heightens hypertensive responses. IL-1R1 deficiency or blockade reduces sodium retention in the thick ascending limb and mitigates angiotensin II–induced hypertension; IL-1R1 activation also shapes myeloid maturation that influences NO-dependent sodium handling [42,46]. Strategies to blunt IL-1–mediated injury must avoid increasing infection risk [41].

IFNs produced by T cells and macrophages promote Th1 differentiation and myeloid/B-cell activation, constraining natriuresis. Blocking IFNGR1 does not attenuate angiotensin II–driven hypertension, implicating IFNAR2 in sodium regulation; however, IFNGR1 inhibition can limit tubulointerstitial inflammation in this setting [42; 7,19,35].

TGF-β is a central driver of renal fibrosis in RAAS-associated hypertension. By suppressing MMP activation, TGF-β enhances extracellular-matrix deposition. Exogenous TGF-β1/β2 infusion induces renal fibrosis, albuminuria, and hypertension—likely through vascular dysfunction and/or enhanced sodium retention. Chronic angiotensin II elevates circulating TGF-β. Conversely, Treg-derived TGF-β cooperates with IL-10 to dampen hypertensive responses by suppressing effector T-cell activation; the renal impact depends on source and concentration [36,42].

IL-17A, produced by $CD4^+$ T cells, amplifies pro-inflammatory cytokines and chemokines that drive cellular immune responses [13,16]. Serum IL-17 is elevated in hypertension; chronic angiotensin II increases IL-17 production and vascular expression. Exogenous IL-17 worsens hypertension and endothelial dysfunction, whereas IL-17A (but not IL-17F) deficiency or blockade lowers blood pressure and renal inflammation in angiotensin II–dependent models. Non-specific IL-17 inhibition, however, can be neutral or deleterious for renal function [35].

IL-10, an anti-inflammatory cytokine produced by Th2 cells, Tregs, mast cells, and monocytes, suppresses pro-inflammatory cytokines and chemokines [5]. In animal models of pregnancy-induced hypertension, IL-10 infusion reduces proteinuria, endothelial injury, and blood pressure; IL-10 deficiency exacerbates microvascular damage and hypertension via NADPH oxidase signaling [14,21,39].

Further supporting a role for $CD8^+$ T cells, these lymphocytes express the mineralocorticoid receptor (MR). MR complexes with NFAT1 and AP-1 to enhance IFN-γ production; T-cell–specific MR deletion markedly reduces angiotensin II–induced hypertension and renal/vascular injury, whereas MR overexpression worsens them. The MR antagonist eplerenone prevents IFN-γ production by $CD8^+$ T cells in hypertension [26].

Macrophages, key innate immune cells, activate NF-κB signaling, releasing cytokines such as IL-6 and TNF-α [3]. IL-6 drives C-reactive protein (CRP) production, a systemic inflammatory marker. CRP can promote Th1 differentiation (with liposomes) and, via FcγRI, support Th2 differentiation (with phosphatidylcholine). As cytokine secretion escalates in CVD, Th1 polarization predominates, fueling inflammation through pro-inflammatory cytokine release and immune-cell activation [40,44].

Conclusion

Cytokine-mediated sodium retention represents a key mechanism in the development of myocardial infarction (MI). Understanding the chain of cytokine-dependent events is therefore essential. Recent findings indicate that sodium retention itself stimulates the production of pro-inflammatory cytokines by T lymphocytes and macrophages. Consequently, combined therapy using diuretics and anti-inflammatory agents may reduce renal and cardiovascular damage in patients with hypertension. Endothelial dysfunction, recognized as a predictor of metabolic syndrome and cardiovascular disease, develops in response to a deficiency of nitric oxide (NO) produced by macrophages. Pro-inflammatory macrophages together with Th1 and Th17 cells intensify renal injury and hypertensive reactions by releasing TNF-α, IL-17A, IL-1, and IFN-γ. These mediators impair sodium transport in the thick ascending limb and reduce renal blood flow, thereby promoting endothelial dysfunction.

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