Impact of Post-Traumatic Stress Disorder (PTSD) on Cardiovascular System

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- Introduction
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- Immune Dysregulation and Inflammation
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Introduction

Post-traumatic stress disorder (PTSD) is a psychiatric condition that develops after exposure to traumatic or life-threatening events such as war, violence, natural disasters, or serious accidents. It is characterized by symptoms including intrusive memories, nightmares, hyperarousal, emotional distress, and avoidance of trauma-related reminders. Beyond its psychological effects, increasing evidence suggests that PTSD also has important consequences for physical health, particularly the cardiovascular system.[1,2]

Recent studies have identified **PTSD as an independent risk factor for cardiovascular disease (CVD)**. Individuals with PTSD have a higher likelihood of developing coronary artery disease, myocardial infarction, stroke, heart failure, atrial fibrillation, and venous thromboembolism. The disorder is also associated with a greater prevalence of traditional cardiovascular risk factors, including hypertension, diabetes mellitus, obesity, and dyslipidemia.[3] Furthermore, a meta-analysis of longitudinal studies reported that PTSD increases the risk of coronary heart disease by approximately 55–61%, highlighting the significant impact of chronic psychological stress on cardiovascular health.[3]

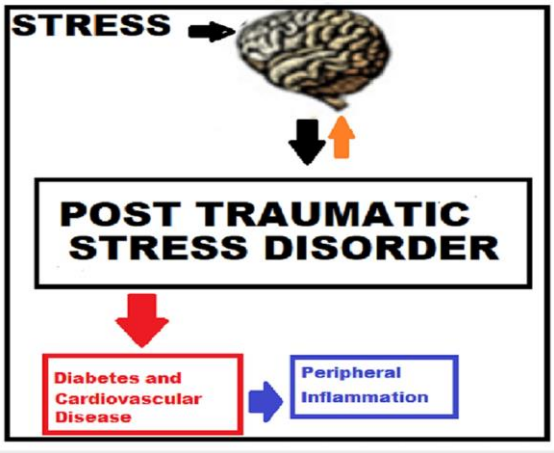


Figure 1: *Relationship between PTSD and cardiovascular disease through inflammatory pathways.*[8]

Pathophysiological Link Between PTSD and Cardiovascular Disease

PTSD affects the cardiovascular system through several interconnected biological mechanisms involving the autonomic nervous, immune, and neuroendocrine systems. Persistent psychological stress disrupts the body's normal stress-response pathways, particularly the hypothalamic–pituitary–adrenal (HPA) axis and the sympathetic–adrenal–medullary (SAM) system. These changes contribute to cardiovascular dysfunction and increase the risk of developing cardiovascular disease.[2,3]

In addition, individuals with PTSD often experience prolonged activation of the sympathetic nervous system (SNS) together with reduced parasympathetic activity. This imbalance can lead to an increased heart rate, elevated blood pressure, and reduced heart rate variability. Over time, these physiological changes may contribute to hypertension, atherosclerosis, metabolic syndrome, and other cardiovascular complications.[2,4]

Autonomic Dysfunction

Hyperarousal is a hallmark of PTSD and is associated with persistent activation of the sympathetic nervous system (SNS). Individuals with PTSD commonly exhibit increased resting heart rate and blood pressure, reduced heart rate variability, and elevated catecholamine levels, reflecting sustained sympathetic overactivity. This autonomic imbalance contributes to hypertension and other cardiovascular disorders, increasing the overall risk of cardiovascular disease.[2,4]

Immune Dysregulation and Inflammation

Chronic inflammation is considered one of the key mechanisms linking PTSD to cardiovascular disease. Autonomic imbalance in PTSD promotes persistent immune activation, leading to the release of pro-inflammatory cytokines such as IL-1β, IL-2, IL-6, and TNF-α, while reducing anti-inflammatory cytokines like IL-4. This sustained inflammatory response contributes to endothelial dysfunction, atherosclerosis, and an increased risk of cardiovascular disease.[2,3]

In addition, elevated levels of C-reactive protein (CRP), a marker of systemic inflammation, have been reported in many individuals with PTSD. Persistent inflammation and immune dysregulation may therefore accelerate the progression of cardiovascular disease and contribute to poorer clinical outcomes.[2,3]

Oxidative Stress

Oxidative stress is an important mechanism linking PTSD to cardiovascular disease. It results from an imbalance between reactive oxygen species (ROS) and the body's antioxidant defenses, leading to cellular damage and dysfunction. Persistent oxidative stress also interacts with chronic inflammation, creating a cycle that further increases cardiovascular risk.[3,7]

Oxidative stress contributes to endothelial dysfunction and atherosclerosis, both of which play key roles in the development of hypertension, coronary artery disease, and heart failure. Individuals with PTSD have been shown to exhibit increased markers of oxidative stress, supporting its role in the progression of cardiovascular disease.[3,7]

Mitochondrial Dysfunction

Mitochondria play a central role in cellular energy production and are an important source of reactive oxygen species (ROS). Chronic stress associated

with PTSD may impair mitochondrial function, disrupting energy metabolism and increasing oxidative stress. These changes can contribute to endothelial dysfunction, inflammation, and the development of cardiometabolic conditions such as hypertension, obesity, and atherosclerosis.[3]

Although research on mitochondrial dysfunction in PTSD is still limited, current evidence suggests that it may represent another biological pathway linking PTSD with an increased risk of cardiovascular disease.[3]

Renin–Angiotensin System (RAS) Dysregulation

Chronic psychological stress in PTSD can activate the renin–angiotensin system (RAS), an important neuroendocrine pathway involved in regulating blood pressure and cardiovascular function. Persistent RAS activation increases the production of angiotensin II (ANG II), which promotes vasoconstriction, inflammation, oxidative stress, and endothelial dysfunction.[3,4]

These changes contribute to hypertension, cardiac remodeling, and atherosclerosis, ultimately increasing the risk of cardiovascular disease in individuals with PTSD. Although the exact mechanisms continue to be investigated, current evidence supports RAS dysregulation as one of the key pathways linking PTSD to adverse cardiovascular outcomes.[3,4]

Conclusion

Post-traumatic stress disorder (PTSD) is increasingly recognized as an independent risk factor for cardiovascular disease. Chronic psychological stress contributes to cardiovascular dysfunction through multiple interconnected mechanisms, including sympathetic nervous system overactivity, persistent inflammation, oxidative stress, mitochondrial dysfunction, and renin–angiotensin system activation. Understanding these biological pathways highlights the importance of early recognition and comprehensive management of PTSD to reduce its long-term cardiovascular consequences.[3,4]

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