

Increased Risk of Cardiovascular Disease in Combat-Related Post-Traumatic Stress Disorder

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Abstract

Combat-related post-traumatic stress disorder (PTSD) carries consequences that extend beyond psychological distress. A growing body of evidence has associated PTSD with increased cardiovascular disease (CVD) risk among veterans, yet the mechanisms underlying this relationship remain incompletely defined. This review examines several pathways that may connect PTSD with adverse cardiovascular outcomes, including smoking and high-risk substance use, comorbid anxiety and depression, autonomic dysregulation, endothelial dysfunction, myocardial ischemia, and atherosclerotic disease. The available evidence suggests that the relationship is multifactorial rather than attributable to a single pathway. These findings have practical implications for clinicians caring for veterans with PTSD, particularly in cardiovascular risk assessment, behavioral risk reduction, and early identification of vascular disease. The review concludes by identifying limitations in the existing literature and areas in which further research is needed to clarify mechanisms and determine whether targeted screening or treatment can improve clinical outcomes.

Keywords: *post-traumatic stress disorder; cardiovascular disease; veterans; combat exposure; cardiovascular risk*

Introduction

Post-traumatic stress disorder (PTSD) can develop after exposure to traumatic events, including combat. In DSM-5, PTSD is classified among trauma- and stressor-related disorders and is characterized by symptoms involving intrusion, avoidance, negative alterations in cognition and mood, and changes in arousal and reactivity.[1] Combat-exposed veterans represent a population in which PTSD has been repeatedly documented, with prevalence estimates varying by conflict, study design, and diagnostic method.[2]

One of the more troubling observations concerning PTSD is the range of physical illnesses that accompany the disorder. Of particular interest is the growing body of evidence establishing an association between PTSD and cardiovascular disease (CVD). Researchers have described multiple pathways that may contribute to this relationship, including tobacco use, substance misuse, autonomic dysregulation, endothelial dysfunction, myocardial ischemia, atherosclerotic disease, and comorbid psychiatric illness. At the time of the original review, PTSD was not generally treated as a conventional cardiovascular risk factor, but the accumulation of evidence suggested a relationship that warranted clinical attention.

The goal of this review is to analyze evidence of PTSD-associated cardiovascular dysfunction across psychological, behavioral, autonomic, and vascular pathways. The clinical relevance is direct: patients with PTSD may benefit from more deliberate cardiovascular risk assessment and from attention to modifiable behaviors that compound risk. The review also identifies limitations in the literature and proposes directions for research needed to clarify mechanisms and improve clinical outcomes.

Pathophysiologic Background

PTSD-associated cardiovascular disease is unlikely to arise from a single mechanism. Psychological and behavioral factors may interact with autonomic dysregulation and measurable changes in vascular and cardiac function. The relative contribution of each pathway varies among patients and over time, which makes the association difficult to define with precision. For that reason, the following sections consider these pathways as an interconnected framework rather than as isolated causes.

Psychological and Behavioral Pathways

Patients with PTSD are at increased risk for behavioral and psychiatric comorbidity, including tobacco use, substance misuse, depression, and additional anxiety disorders. Smoking is particularly relevant

because it represents both a major cardiovascular risk factor and a behavior associated with PTSD symptom severity. In a sample of Iraq and Afghanistan veterans referred for mental health services, Cook and colleagues found that greater overall PTSD symptom severity was associated with heavy smoking. Emotional numbing was the symptom cluster most strongly associated with heavy smoking, while the other clusters were not independently related to smoking status.[3] This pattern raises the possibility that smoking may function, at least in part, as a form of affect regulation in veterans with prominent emotional-numbing symptoms.

Prescription opioid exposure is another clinically important pathway. Advances in battlefield medicine have allowed more service members to survive injuries that may result in persistent pain, and some of these veterans also carry a diagnosis of PTSD. In a large cohort of Iraq and Afghanistan veterans with pain diagnoses, Seal and colleagues found that veterans with PTSD were more likely to receive opioid prescriptions and, among those prescribed opioids, more likely to receive higher doses, concurrent opioids or sedative-hypnotics, and early refills. Opioid treatment was also associated with a greater risk of adverse clinical outcomes, with the most pronounced risk among veterans with PTSD.[5] These findings do not establish that PTSD itself causes unsafe opioid use, but they identify an important intersection of trauma, pain, prescribing, and preventable harm.

Depression and anxiety further complicate this relationship. Cardiovascular disease has been associated with depressive and anxiety disorders, and anxiety appears to contribute meaningfully to cardiovascular burden. In a large cohort studied by Vogelzangs and colleagues, cardiovascular disease was more prevalent among people with anxiety disorders, including those with comorbid depression and anxiety.[12] Because PTSD includes persistent stress and arousal symptoms and frequently coexists with other psychiatric disorders, these comorbidities may amplify cardiovascular risk even when they do not fully explain it.

Autonomic Reactivity and Endothelial Function

PTSD is also associated with alterations in autonomic regulation. Heightened sympathetic activity and reduced heart rate variability have been proposed as mechanisms through which chronic stress may influence cardiovascular health.[6] In a pilot study of physically healthy combat veterans, Clausen and colleagues examined autonomic reactivity during emotionally evocative scripts and measured vascular health with brachial artery flow-mediated dilation (FMD). Although PTSD symptom severity itself was not directly associated with FMD in that small sample, autonomic reactivity to negative emotional scripts was related to impaired FMD.[7] The findings support the broader hypothesis that stress-system dysregulation may contribute to vascular dysfunction.

FMD should be understood as a measure of endothelial function rather than as a direct measure of coronary blood flow. That distinction is clinically important. Impaired endothelial function can precede overt cardiovascular disease and may provide one pathway through which chronic psychological stress becomes biologically consequential.

Changes in Cardiovascular Structure and Function

The strongest evidence connecting PTSD with cardiovascular disease comes from studies using objective measures of vascular and cardiac pathology.[10,11] These include coronary artery calcium, endothelial function, myocardial perfusion, and exercise-induced ischemia. Such measures move the discussion beyond self-reported symptoms and demonstrate that the association can be observed in cardiovascular physiology and structure.

Ahmadi and colleagues evaluated coronary artery calcium in veterans without known coronary artery disease and found that PTSD was associated with a greater prevalence and severity of coronary atherosclerosis. PTSD also predicted mortality independently of several conventional risk factors.[13] A prospective twin study by Vaccarino and colleagues strengthened this evidence. Among Vietnam-era twin veterans, the incidence of coronary heart disease was more than twice as high in twins with PTSD as in those without PTSD. Objective positron-emission tomography measures also demonstrated worse myocardial perfusion among twins with PTSD, and the association persisted after adjustment for major depression and conventional cardiovascular risk factors.[9]

Endothelial dysfunction has also been demonstrated in a larger veteran cohort. Grenon and colleagues found that veterans with PTSD had significantly worse brachial artery flow-mediated vasodilation than veterans without PTSD, even after multivariable adjustment.[4] In a separate study, Turner and colleagues used standardized exercise treadmill testing to evaluate myocardial ischemia. Ischemia was present more frequently among patients with PTSD, and the association remained significant after adjustment for traditional cardiac risk factors, inflammatory markers, depression, and several behavioral and psychosocial variables.[8]

Taken together, these studies support a meaningful association between PTSD and adverse cardiovascular outcomes. Their importance extends beyond establishing correlation. First, the limitations of the existing evidence provide direction for future research, particularly studies designed to clarify causal pathways. Second, the available data identify practical clinical targets for risk assessment and prevention in veterans with PTSD.

Clinical Implications

Although this review is not exhaustive, the available evidence suggests that clinicians caring for veterans with PTSD should think beyond psychiatric symptoms alone. A comprehensive assessment should include conventional cardiovascular risk factors, smoking and substance use, chronic pain and medication exposure, relevant family history, and symptoms that may suggest cardiovascular disease. The goal is not to treat PTSD as a stand-alone cardiac diagnosis, but to recognize that it may identify a population in whom multiple cardiovascular risks cluster.

Several of the studies reviewed also demonstrate tools that can objectively characterize cardiovascular health, including coronary artery calcium imaging, flow-mediated dilation, exercise treadmill testing, electrocardiography, and measures of myocardial perfusion. These tests are not appropriate as universal screening tools simply because a patient has PTSD. Rather, the evidence argues for deliberate risk stratification and for research that can determine which patients benefit from additional cardiovascular evaluation.

The most immediately actionable targets remain familiar ones: tobacco cessation, careful opioid and sedative prescribing, identification of substance-use disorders, management of hypertension and metabolic risk, and appropriate evaluation of cardiovascular symptoms. What PTSD adds is a reason to recognize that these risks may coexist, interact, and accumulate in a vulnerable population.

Future Directions

Several limitations of the literature point toward future research. Much of the evidence reviewed here was derived from veteran populations, often composed predominantly of middle-aged men. That focus is understandable in research on combat-related PTSD, but it limits generalizability. Future studies should

include more women and more civilian populations exposed to trauma so that the relationship between PTSD and cardiovascular disease can be evaluated across a broader range of patients.

The underlying mechanisms also remain incompletely defined. Future work should distinguish the contributions of autonomic dysregulation, inflammation, endothelial dysfunction, health behaviors, psychiatric comorbidity, medication exposure, and other pathways. Longitudinal studies are particularly important because they can clarify whether changes in PTSD severity precede changes in cardiovascular function and whether successful treatment of PTSD alters cardiovascular risk over time.

Finally, the clinical utility of targeted cardiovascular screening in patients with PTSD should be tested rather than assumed. It is necessary to determine whether earlier identification of cardiovascular risk changes management, improves outcomes, or just increases testing. Similarly, research should examine whether effective treatment of PTSD or its associated behaviors produces measurable improvement in cardiovascular health. These questions move the field from recognizing an association to determining what clinicians can meaningfully do about it.

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