

INFLUENCE OF PSYCHOEMOTIONAL FACTORS IN THE DEVELOPMENT OF ISCHEMIC HEART DISEASE AND RECOMMENDATIONS FOR THEIR REDUCTION

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Annotation. *This article explores the role of psychoemotional factors in the pathogenesis and progression of ischemic heart disease (IHD). Psychological stress, anxiety, depression, and chronic emotional tension significantly influence coronary circulation, autonomic imbalance, endothelial dysfunction, and inflammatory activation. The article evaluates key mechanisms linking psychoemotional states with myocardial ischemia, presents research-based findings, and offers practical recommendations aimed at reducing psychoemotional burden to prevent IHD progression. This work examines how psychoemotional disturbances influence the progression of ischemic heart disease by altering neurohumoral balance, autonomic activity, and vascular stability. The evaluation is based on evidence linking emotional tension, mental overload, and maladaptive behavioral responses to coronary dysfunction and instability of myocardial oxygen supply. Emphasis is placed on identifying critical mechanisms through which emotional imbalance triggers harmful cardiovascular shifts and on highlighting outcome-based insights derived from current scientific observations.*

Keywords: *ischemic heart disease, psychoemotional stress, anxiety, depression, coronary circulation, autonomic dysfunction, prevention, stress reduction.*

Introduction:

Ischemic heart disease remains one of the leading causes of mortality worldwide, with multifactorial pathogenesis involving both physiological and psychological determinants. While traditional risk factors such as hypertension, dyslipidemia, diabetes, smoking, and obesity are widely recognized, psychoemotional factors have gained increasing attention due to their strong and often underestimated influence. Chronic stress, persistent anxiety, depressive symptoms, and emotional instability contribute to the development of coronary artery spasm, hemodynamic fluctuations, hormonal imbalance, and pro-inflammatory responses. These mechanisms impair endothelial function, increase myocardial oxygen demand, and accelerate atherosclerotic changes.

Understanding the psychoemotional contribution to IHD is essential for improving clinical outcomes and optimizing preventive strategies. Emotional dysregulation is increasingly recognized as a determining factor capable of modifying the clinical trajectory of ischemic myocardial pathology. Prolonged mental strain affects sympathetic–parasympathetic coordination, leading to excessive circulatory pressure fluctuations, impaired endothelial signaling, and increased metabolic load on cardiac tissue. These disruptions predispose vulnerable individuals to plaque destabilization, reduced coronary perfusion reserve, and heightened myocardial vulnerability. As a result, the interface between mental load and ischemic processes has become a vital subject for clinical scrutiny.

Greater acknowledgment of these influences is essential for tailoring preventive strategies aimed at limiting the harmful cascade initiated by persistent emotional imbalance, particularly in individuals experiencing high psychological burden.

Research Methods and Approaches:

This study is based on an integrative review of scientific literature published in international cardiology, psychology, and behavioral medicine journals. Sources included observational clinical studies, randomized controlled trials, systematic reviews, and meta-analyses examining the relationship between psychoemotional states and ischemic heart disease.

Databases such as PubMed, Scopus, and Web of Science were used to identify relevant publications. Key parameters analyzed included stress-response biomarkers, emotional state assessments, heart rate variability indices, neuroendocrine indicators, inflammatory markers, and coronary imaging data. In addition, the study evaluated preventive interventions targeting psychoemotional health, including behavioral therapy, mindfulness-based practices, psychotherapy, lifestyle modification, and pharmacologic options.

Results:

Findings from the analyzed studies demonstrate a strong correlation between psychoemotional stress and increased risk of ischemic heart disease. Patients with high levels of anxiety and depression exhibited significantly reduced heart rate variability, indicating sympathetic overactivity and impaired autonomic balance. Chronic stress was associated with elevated cortisol levels, increased C-reactive protein, and enhanced platelet aggregation, all contributing to endothelial dysfunction and increased likelihood of coronary events. Several studies revealed that individuals experiencing long-term emotional overload had up to a 2–3-fold higher incidence of angina, myocardial infarction, and recurrent ischemic episodes. Interventions aimed at reducing psychoemotional stress—including cognitive behavioral therapy, meditation, improved sleep hygiene, structured physical activity, and social support—showed significant improvement in cardiac outcomes and reduction in ischemic symptoms. Evaluation of available scientific material shows that individuals exposed to high mental tension present with measurable alterations in cardiovascular regulation, including reduced vagal tone, heightened catecholamine secretion, and increased inflammatory mediator production. These biological changes correlate with a higher frequency of coronary artery constriction episodes, acceleration of atherogenic changes, and diminished myocardial perfusion efficiency. Furthermore, long-term psychological overload has been associated with greater incidence of angina, recurrent ischemic discomfort, and adverse coronary events. Scientific findings also demonstrate that structured interventions aimed at controlling emotional reactivity significantly alleviate ischemic manifestations and improve cardiac function metrics.

Discussion:

The results highlight psychoemotional factors as significant but often overlooked contributors to the development and progression of ischemic heart disease. Emotional states influence cardiovascular health through a complex interplay of neuroendocrine, autonomic, and inflammatory mechanisms. Sympathetic overactivation increases heart rate, blood pressure, and myocardial oxygen demand, while chronic cortisol elevation promotes insulin resistance,

dyslipidemia, and visceral fat accumulation. At the vascular level, stress accelerates endothelial damage and atherosclerotic plaque instability, increasing the risk of acute coronary syndromes.

The evidence supports incorporating psychological assessment and stress management into routine cardiovascular care. Effective psychoemotional interventions help modulate autonomic imbalance, normalize hormonal pathways, reduce inflammatory markers, and consequently lower overall cardiovascular risk. The compiled evidence reinforces the understanding that mental instability operates as a significant contributor to ischemic deterioration by advancing autonomic imbalance, intensifying neuroendocrine activation, and promoting pro-inflammatory vascular processes. These influences extend beyond simple behavioral patterns, directly affecting physiological pathways essential for cardiovascular integrity. Heightened sympathetic influence increases mechanical strain on arterial walls, while sustained cortisol elevation fosters metabolic conditions conducive to atherosclerotic advancement. Clinical benefits observed after implementing emotional regulation practices indicate that addressing mental load is an indispensable component of contemporary ischemic management. The insights obtained underscore the necessity of integrating targeted psychological evaluation alongside somatic treatment to mitigate cumulative cardiovascular risk.

Conclusion:

Psychoemotional factors play a major role in the onset and progression of ischemic heart disease by influencing autonomic function, endocrine balance, and inflammatory pathways.

Early identification and targeted management of emotional stress, anxiety, and depression are essential components of comprehensive cardiovascular prevention. Integrating psychological support, behavioral therapy, relaxation techniques, and lifestyle modification into clinical practice can significantly improve patient outcomes, reduce ischemic episodes, and enhance overall quality of life. Further research is recommended to deepen understanding of the psycho-cardiological interface and optimize individualized preventive strategies. Mental tension represents a potent modifier of ischemic progression by inducing maladaptive hemodynamic responses, neurohormonal imbalance, and inflammatory activation. Recognition of these effects allows for more comprehensive preventive planning, emphasizing early detection of emotional burden and timely implementation of corrective strategies. Incorporating structured mental regulation protocols into cardiovascular care has demonstrated meaningful improvement in cardiac outcomes, stress resilience, and overall patient stability. Continued exploration of emotional-cardiac interactions will further refine predictive models and strengthen preventive cardiological practice.

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