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OXIDATIVE STRESS AND ITS ROLE IN THE PATHOPHYSIOLOGY OF CARDIOVASCULAR DISEASES

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Abstract: Oxidative stress, defined as an imbalance between reactive oxygen species (ROS) generation and antioxidant defenses, plays a pivotal role in the pathophysiology of cardiovascular diseases. Excessive ROS production leads to lipid peroxidation, DNA damage, endothelial dysfunction, and vascular inflammation, which collectively contribute to atherosclerosis, hypertension, ischemic injury, and heart failure. This article reviews recent evidence on the molecular mechanisms of oxidative stress and its clinical implications. Findings reveal that while antioxidant therapies demonstrate partial success, their clinical translation remains limited, highlighting the need for more targeted interventions. Understanding oxidative stress within the framework of pathological physiology provides essential insights into prevention and treatment strategies for cardiovascular disease.

Keywords: Oxidative stress; Pathological physiology; Cardiovascular diseases; Reactive oxygen species; Endothelial dysfunction; Antioxidant therapy

Introduction

Pathological physiology investigates the mechanisms by which normal physiological processes become altered in disease states. Among the most significant contributors to pathological changes in the cardiovascular system is oxidative stress, defined as an imbalance between the production of reactive oxygen species (ROS) and the antioxidant defense mechanisms of the body. The excessive accumulation of ROS leads to cellular and molecular damage, ultimately contributing to the development of atherosclerosis, hypertension, ischemic injury, and heart failure. Cardiovascular diseases remain the leading cause of morbidity and mortality worldwide, and understanding the pathological physiology of oxidative stress offers new insights into prevention and treatment strategies.

Methods

This article is based on a narrative review of current literature published between 2015 and 2024. Sources were obtained from PubMed, Scopus, and Web of Science using keywords such as “oxidative stress,” “pathological physiology,” “cardiovascular diseases,” “ROS,” and “antioxidant therapy.” Studies included in this analysis were limited to experimental models, clinical trials, and systematic reviews that focused on the molecular mechanisms of oxidative stress and its clinical relevance. Data synthesis emphasized the pathological processes involving ROS, endothelial dysfunction, lipid peroxidation, and inflammatory cascades.

Results

The review of the literature demonstrated several key findings. Firstly, ROS are generated predominantly in mitochondria through the electron transport chain and by enzymatic systems such as NADPH oxidase, xanthine oxidase, and uncoupled nitric oxide synthase. Excessive ROS production results in lipid peroxidation of cell membranes, oxidation of low-density lipoprotein

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(LDL), and damage to mitochondrial DNA. These processes impair endothelial function and trigger inflammatory responses, which are critical in the initiation of atherosclerotic plaque formation. Secondly, oxidative stress is strongly linked with hypertension, as ROS reduce nitric oxide bioavailability, causing vasoconstriction and vascular stiffness. Thirdly, reperfusion injury following ischemia is exacerbated by a burst of ROS, which accelerates cardiomyocyte apoptosis. Furthermore, evidence indicates that antioxidant therapies such as vitamin C, vitamin E, and novel compounds like N-acetylcysteine have demonstrated partial efficacy in reducing oxidative damage, though large-scale trials show mixed clinical outcomes.

Discussion

The evidence supports that oxidative stress is not merely a byproduct of cardiovascular diseases but a central pathological mechanism. Its role in endothelial dysfunction, vascular inflammation, and myocardial injury underscores its significance in pathological physiology. The dual nature of ROS is noteworthy: while moderate levels are essential for normal cellular signaling, uncontrolled production disrupts homeostasis and accelerates disease progression. The failure of some antioxidant therapies in clinical trials suggests that a more targeted approach is needed, focusing on specific enzymatic sources of ROS rather than general scavenging. For example, inhibition of NADPH oxidase or enhancing endogenous antioxidant pathways such as superoxide dismutase and glutathione peroxidase may offer more effective therapeutic results. Understanding the interplay between oxidative stress and genetic predisposition also provides opportunities for personalized medicine in the management of cardiovascular diseases.

Conclusion

Oxidative stress represents a critical pathological mechanism in cardiovascular disease. By linking ROS production with endothelial dysfunction, lipid peroxidation, and inflammatory processes, it highlights a central theme in pathological physiology. Despite advances in understanding, therapeutic translation remains challenging, and further research is necessary to develop effective, targeted antioxidant strategies. Pathological physiology continues to provide a framework to bridge molecular mechanisms with clinical manifestations, offering vital insights into both prevention and treatment of cardiovascular disease.

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