Asian Journal of Technology and Applied Sciences
ISSN: 2301-xxxx (online) | IF: 10.01 | Vol: 1, issue 1
Available: http://asiansciencejournal.org

CHANGES THAT OCCUR IN CHRONIC HEART FAILURE AND THEIR CAUSES

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Abstract: This article examines the functional and structural changes that occur in the body as a result of the development of chronic heart failure, as well as the factors contributing to their occurrence. The gradual decline in the heart's pumping function negatively affects the circulatory system, leading to various clinical manifestations and complications. The study analyzes the mechanisms underlying these pathological processes and their impact on the functioning of the organism.
Keywords: chronic heart failure, cardiac function, circulatory disorders, hemodynamic changes, tissue hypoxia.

Introduction

Heart failure is a pathological condition in which the heart is unable to pump enough blood to meet the body's metabolic needs. As a result, the delivery of oxygen and essential nutrients to tissues becomes impaired. This condition does not mean that the heart has completely stopped functioning; rather, its pumping capacity is weakened or its normal function is compromised. As the disease progresses, fluid accumulation may occur in the body, leading to symptoms such as shortness of breath, fatigue, and swelling of the legs, ankles, or abdomen. Heart failure commonly develops as a consequence of hypertension, myocardial infarction, diabetes mellitus, and other cardiovascular diseases. Although it can affect individuals of any age, it is more frequently observed in older adults. Early diagnosis and appropriate treatment can help control disease progression. Proper medical care, regular monitoring, and adherence to a healthy lifestyle can significantly improve patients' quality of life. Several diseases and risk factors contribute to the development of heart failure. The most important of these include: **Coronary Artery Disease (CAD).** The accumulation of atherosclerotic plaques within the coronary arteries reduces blood flow to the heart muscle. Insufficient oxygen supply weakens the myocardium and impairs its function. **Arterial Hypertension.** Persistently elevated blood pressure places excessive strain on the heart. Over time, the heart muscle becomes thickened and eventually loses its ability to contract effectively, which may lead to heart failure. **Myocardial Infarction.** Damage to or death of a portion of the heart muscle reduces the heart's pumping efficiency. Consequently, the risk of developing heart failure increases significantly following a heart attack. **Cardiomyopathies.** Structural and functional abnormalities of the heart muscle, including those caused by genetic factors, viral infections, excessive alcohol consumption, and exposure to toxic substances, can weaken the myocardium and interfere with its normal function.

Method

In the preparation of this article, methods of scientific literature review, comparative analysis, and data synthesis were employed. The theoretical foundation of the study was based on national and international scientific sources, clinical guidelines, textbooks, and recent medical publications related to chronic heart failure. Relevant information was

collected and evaluated according to its scientific significance and reliability. During the study, the pathophysiological changes, clinical manifestations, and underlying causes associated with chronic heart failure were analyzed. The collected data were systematically examined to identify the relationship between impaired cardiac function and the functional and structural changes occurring within the body. In addition, major risk factors contributing to the development of chronic heart failure and their effects on the organism were discussed based on evidence from scientific literature.

Results

Acute heart failure usually develops as a consequence of myocardial infarction, acute myocarditis, or severe cardiac arrhythmias such as ventricular fibrillation and paroxysmal tachycardia. In this condition, the heart's pumping capacity declines abruptly, resulting in a significant reduction in cardiac output and arterial blood flow. Clinically, acute heart failure resembles acute vascular insufficiency and is sometimes referred to as cardiac collapse. In **chronic heart failure**, the body initially compensates for impaired cardiac function through a variety of adaptive mechanisms that help maintain adequate circulation over an extended period. These compensatory responses include an increase in the force of cardiac contractions, acceleration of the heart rate, dilation of peripheral blood vessels, and improved tissue perfusion. Such mechanisms temporarily offset the decline in cardiac performance. As the disease progresses, cardiac output gradually decreases, the volume of residual blood within the ventricles increases, and the cardiac chambers become excessively filled during diastole. Consequently, myocardial fibers undergo excessive stretching. Continuous overload on the heart initially results in compensatory myocardial hypertrophy. However, over time, dystrophic and sclerotic changes develop within the myocardium, leading to the onset of the decompensation stage. At this point, the heart muscle itself begins to experience deficiencies in oxygen and energy supply. In the later stages of the disease, neurohumoral regulatory mechanisms become activated. Stimulation of the sympathetic-adrenal system causes peripheral vasoconstriction, which helps maintain arterial blood pressure. At the same time, constriction of the renal blood vessels reduces renal perfusion, promoting the retention of sodium and water within the body. Furthermore, increased secretion of antidiuretic hormone enhances water reabsorption in the kidneys. As a result, circulating blood volume rises, venous and capillary pressures increase, and fluid movement from the blood vessels into surrounding tissues becomes more pronounced. This process contributes to the development of edema. Thus, chronic heart failure leads to complex hemodynamic, neurohumoral, and metabolic disturbances that adversely affect the function of multiple organs and body systems.

Discussion

The reviewed scientific literature and analytical findings indicate that chronic heart failure leads to numerous functional and structural alterations within the body. During the early stages of the disease, the impairment of cardiac function is partially compensated by the body's adaptive mechanisms. However, as the pathological process progresses, the effectiveness of these compensatory responses gradually declines, resulting in more pronounced clinical manifestations.

A reduction in the heart's pumping capacity decreases the amount of blood delivered to organs and tissues. Consequently, cellular oxygen deficiency develops, leading to disturbances in metabolic processes. In addition, neurohumoral responses activated by the body initially help maintain circulatory function, but over time they impose additional stress on the myocardium and contribute to disease progression. In advanced stages of chronic heart failure, fluid retention, venous congestion, and the development of edema become evident. These pathological changes negatively affect not only the cardiovascular system but also the

function of the kidneys, lungs, and other organs. As a result, patients experience reduced physical capacity and a decline in overall quality of life. Therefore, early detection of chronic heart failure, identification and control of contributing risk factors, and the implementation of individualized treatment strategies are essential. Effective disease management requires not only pharmacological therapy but also adherence to a healthy diet, appropriate physical activity, and regular medical supervision. Such a comprehensive approach can help slow disease progression and improve patient outcomes.

Conclusion

Chronic heart failure is a serious pathological condition characterized by impaired cardiac contractility, leading to hemodynamic and metabolic disturbances throughout the body. Electrocardiography, echocardiography, and laboratory investigations play an important role in the diagnosis of the disease. Treatment involves not only pharmacological therapy with medications such as ACE inhibitors, beta-blockers, and diuretics, but also lifestyle modifications and rehabilitation measures. A comprehensive approach not only alleviates clinical symptoms but also improves patients' quality of life and increases life expectancy.

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