

Key Words: *Cardiac hypertrophy, myocardial infarction, oxidative stress, apoptosis*

Introduction

Myocardial infarction (MI) is a leading cause of death and disability worldwide. The pathogenesis of MI involves a complex interplay of hemodynamic, biochemical, and cellular factors. Following the onset of MI, the heart undergoes a series of adaptive and maladaptive changes. One of the key features of the infarcted heart is cardiac hypertrophy, which is characterized by an increase in the size of the heart and the thickness of the ventricular wall. This hypertrophy is primarily driven by the activation of the renin-angiotensin system (RAS) and the sympathetic nervous system (SNS). The RAS and SNS lead to the release of angiotensin II and norepinephrine, respectively, which stimulate the growth of cardiac myocytes. Additionally, oxidative stress and apoptosis play significant roles in the pathogenesis of MI. Oxidative stress is characterized by an imbalance between the production of reactive oxygen species (ROS) and the body's ability to detoxify them. ROS can damage cellular components, including lipids, proteins, and DNA, leading to cell death. Apoptosis, or programmed cell death, is a natural process that occurs in many cells, but it is dysregulated in the infarcted heart, leading to an excessive loss of myocytes. The combination of hypertrophy, oxidative stress, and apoptosis contributes to the progression of MI and the development of heart failure.

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