Streszczenie w języku angielskim

Parameters of oxidative stress, nitrosative stress, and endoplasmic reticulum stress in chronic heart failure with preserved and reduced ejection fraction

Heart failure (HF) is a complex clinical syndrome and remains one of the greatest challenges in modern medicine. Research into the pathophysiological differences between various types of HF is becoming increasingly important. Such research can contribute to the development of new diagnostic methods and more precise, effective therapeutic strategies.

This doctoral dissertation aimed to assess the intensity of pathophysiological processes, such as oxidative stress, nitrosative stress, and endoplasmic reticulum (ER) stress, in different types of HF. The study was divided into two main areas. In the first, I compared the concentrations of markers of these processes in the plasma of patients with preserved ejection fraction HF (HFpEF) and reduced ejection fraction HF (HFrEF). The second area focused on analyzing the effects of myocardial infarction (MI) and a high-fat diet (HFD) on the intensity of these processes in a post-MI HF animal model.

The results of the first part demonstrated significant differences in the intensities of nitrosative stress and ER stress, suggesting different underlying pathophysiological mechanisms in each type of HF. I found that patients with HFpEF exhibited higher levels of nitrosative stress (elevated iNOS and 3-NT concentrations in plasma), indicating a critical role for this process in the pathogenesis of HFpEF. In contrast, ER stress appeared to dominate in HFrEF (elevated GRP78 levels in plasma). In the second part of the dissertation, I demonstrated that HFD combined with MI led to increased nitrosative stress (elevated iNOS and 3-NT levels) and ER stress (elevated GRP78 levels) in myocardial tissue. Both HFD and MI intensified inflammation and oxidative stress, as measured by MPO levels.

The study revealed that different pathophysiological mechanisms play a crucial role in HFpEF and HFrEF. Moreover, the findings suggest that MI in the presence of HFD exacerbates the aforementioned pathological processes in the heart. These observations open new possibilities for targeted diagnostic and therapeutic strategies and indicate potential benefits of dietary interventions in preventing post-MI HF progression.