Introduction:

Trastuzumab is a human monoclonal antibody that targets human epidemic growth factor (HER2) receptor 2, which has dramatically improved the outcome of HER2-positive patients. One of the side effects of Trastuzumab is its cardiac toxicity, which leads to discontinuation of the drug in cancer patients. Studies have shown that Trastuzumab induces cardiac toxicity through the mitochondrial pathway associated with ROS production and oxidative stress. Therefore, the use of antioxidants and mitochondrial protectors can play an important role in reducing mitochondrial damage caused by Trastuzumab. The composition of chrysin as a potential protector of cardiovascular function in animal models. In this study, our aim was to evaluate the ability of chrysin to reduce mitochondrial disorders caused by Trastuzumab and cardiotoxicity.

materials and methods:

Using biochemical and flow cytometric methods of blood factors including (troponin, lactate dehydrogenase and CKME creatine kinase), oxidized glutathione or GSSG, mitochondrial factors (swelling - MTT, MMP membrane potential depletion) - active oxygen species and MDA in oxygen / ROS Cardiac cells isolated from rat hearts were measured.

Results:

The findings show that Trastuzumab (2.25 mg) can cause cytotoxicity, active radical formation, mitochondrial membrane potential collapse, lipid oxidation and oxidative stress, while Chrysin can prevent complications and Reduce cardiac toxicity caused by trastosumbe.

Discussion:

Based on the results, Chrysin is shown to be a promising agent in the treatment of patients with Trastuzumab-induced cardiac toxicity through active radical inhibition and inhibition of mitochondrial dysfunction of cardiomyocytes.

Keywords:

Trastuzumab - Chrysin - Cardiotoxic - Cardiomyocyte