Abstract

introduction

Apart from the anticancer, antioxidant, anti-inflammatory effects and inhibition of aromatase, chrysin is involved in the protection of cardiovascular disorders. Cardiovascular complications are the main cause of death induced by aluminum phosphide (AIP) which are related to oxidative stress and mitochondrial damages. For this purpose, we investigated the effect of chrysin as an antioxidant and mitochondrial protective agent against AIP-induced toxicity in isolated cardiomyocytes and mitochondria obtained from rat heart ventricular.

Material and method

Using by biochemical and flowcytometry, mitochondrial toxicity parameters such as mitochondrial NADH/succinate dehydrogenase activity, mitochondrial swelling, reactive oxygen species formation, mitochondria membrane potential and lipid peroxidation were analyzed in isolated mitochondria.

Discussion and conclusion

our finding in isolated mitochondria showed that chrysin (up to 10 μM) significantly ($P < 0.001$) decreased AIP induced mitochondrial toxicity. These findings demonstrated that chrysin as an antioxidant and mitochondrial protective agent exert protective effect in wild-type cardiomyocyte treated with aluminum phosphide. It was concluded that chrysin significantly reduced the toxicity of AlP in isolated cardiomyocytes and mitochondria. Due to the very low toxicity of chrysin to humans it could be a promising agent in treatment of AlP poisoning.

key words:

Aluminum Phosphide, Crystalline, Cardiac Toxicity, Lipid Peroxidation, Mitochondrial Swelling, Reactive Oxygen Species, Mitochondrial Membrane Potential Fall