Thesis Summary

Introduction

Aluminum phosphide (AlP) poisoning is one of the most life-threatening emergencies. Phosphine gas (PH3) is a highly toxic gas, which is released when the tablet is exposed to humidity, causing multisystem organ failure. Cardiovascular complications are the main cause of death induced by aluminum phosphide which is related to oxidative stress and mitochondrial damages. Apart from the anticancer, antioxidant, anti-inflammatory effects, and inhibition of aromatase, chrysin is involved in the protection of cardiovascular disorders. For this purpose, we investigated the effect of chrysin as an antioxidant and mitochondrial protective agent against AlP-induced toxicity in isolated cardiomyocytes obtained from rat heart ventricular.

Material and method

Isolated cardiomyocytes of male wistar rat(s) were divided into seven groups including control, 20 μg/ml Aluminium phosphide, treatment group (containing different concentrations of chrysin (10, 20, 50 and 100 μg/ml) plus 20 μg Aluminum phosphide) and chrysine (100 μg/ml). Cell viability, reactive oxygen species (ROS) formation, mitochondria membrane potential (MMP), lysosomal damage, lipid peroxidation and oxidation and reduction of glutathione of each group were assessed using biochemical and flow cytometric methods. Results of each test were compared between groups, separately.

Results

Our results showed that the administration of chrysin (up to 10 μM) efficiently decreased ($P < 0.05$) cytotoxicity, oxidative, lysosomal, and mitochondrial damages induced by AlP, in isolated cardiomyocytes.

Discussion and conclusion

Since the logical treatment of AlP induced cellular dysfunction could be an agent to protect the cells from the oxidative stress, halting the manifestation due to progressive cellular damage, chrysin might be a promising agent in treatment of AlP poisoning.

Key words:

Aluminum phosphide, chrysin, cardiotoxicity, oxidative stress, mitochondrial damage, antioxidant