Antioxidative effects of CardiofortIN in doxorubicin cardiotoxicity

Milana M. Bosanac[1], Bojana M. Andrejić Višnjić[1], Nikola B. Martic[2], Jelena P. Radic[3], Ivana N. Kolarov Bjelobrk[3], Jelena P. Amidžić[1]

[1]Department of Histology and Embryology, Faculty of Medicine, Univeristy of Novi Sad, Novi Sad, Serbia

[2]Department of Pharmacology and Toxicology, Faculty of Medicine, University of Novi Sad, Novi Sad, Serbia

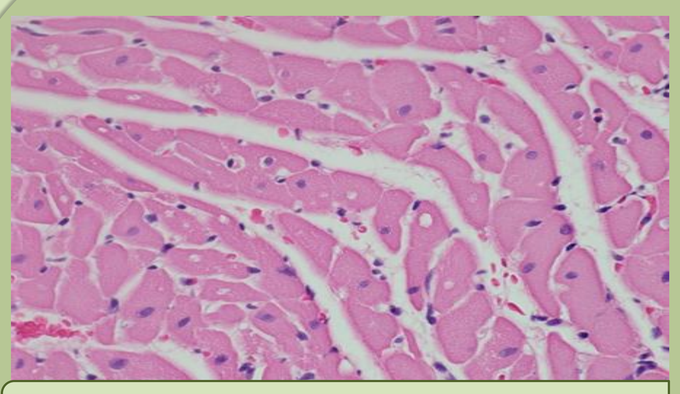
[5]Department of Internal Medicine, Faculty of Medicine, University of Novi Sad, Novi Sad, Serbia

XIV Nedelja bolničke kliničke farmakologije 24-25 decembar 2022. Sekcija za kliničku farmakologiju "Dr Srdjan Djani Marković" Srpsko lekarsko društvo

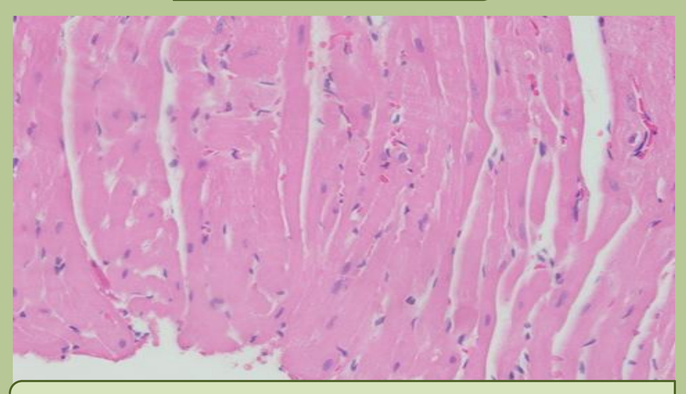
Introduction: Doxorubicin (adriamycin) is an anthracycline group antibiotic. Anthracyclines are commonly used antineoplastic agents in the treatment of a variety of malignancies. Cardiotoxicity remains an irreversible complication of anthracycline-based chemotherapy, which limits the use of doxorubicin. It is believed that doxorubicin cardiotoxicity is caused by DNA and myocardium damage through induction of oxidative stress.

Material and methods: In total, 36 animals, were randomly divided into 4 groups. According to study design, animals were given saline (C), CardiofortIN (CF, 19.25 mg/kg), doxorubicin alone (D, 1.5 mg/kg) or with CardiofortIN (CFD). We induced a model of acute cardiotoxicity, with a single, intraperitoneal dose of doxorybicin, on the 8th experimental day, while saline and CF were given from the 1st-8th experimental day. Heart tissue samples were processed by standard histological technique and stained with the hematoxylin-eosin method. Tissue slides were microscopically analyzed for the presence of indicators of myocardial damage. From the heart tissue homogenate were determined the intensity of lipid peroxidation (determined by the amount of malonyl aldehyde (MDA)) and specific antioxidative enzyme (AOE) activity (superoxide dismutase; catalase; glutathione S-transferase; glutathione peroxidase).

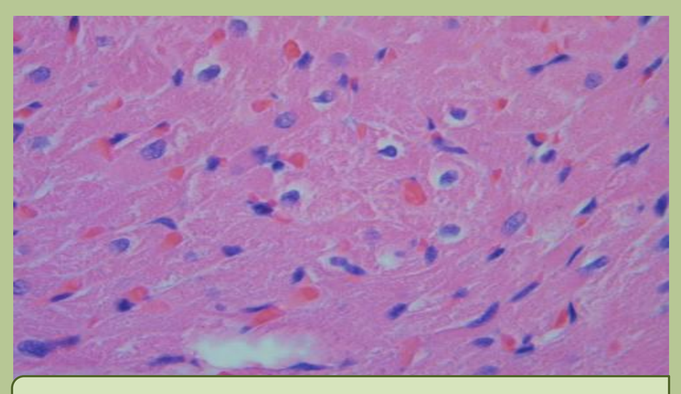
Results:



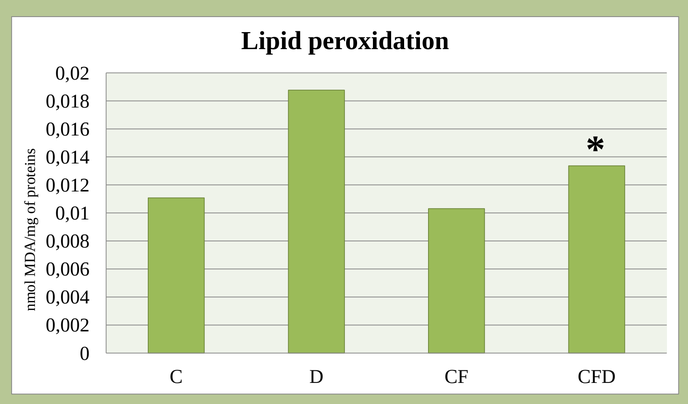
vacuolization

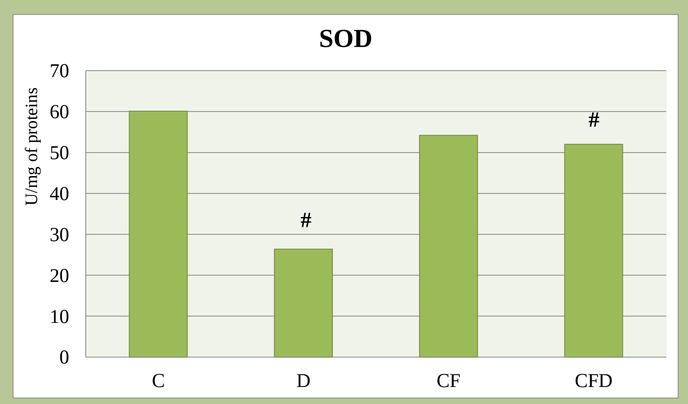


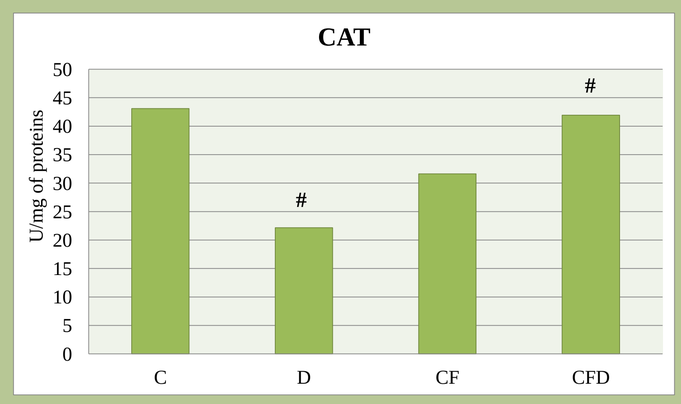
myofilaments disorganization



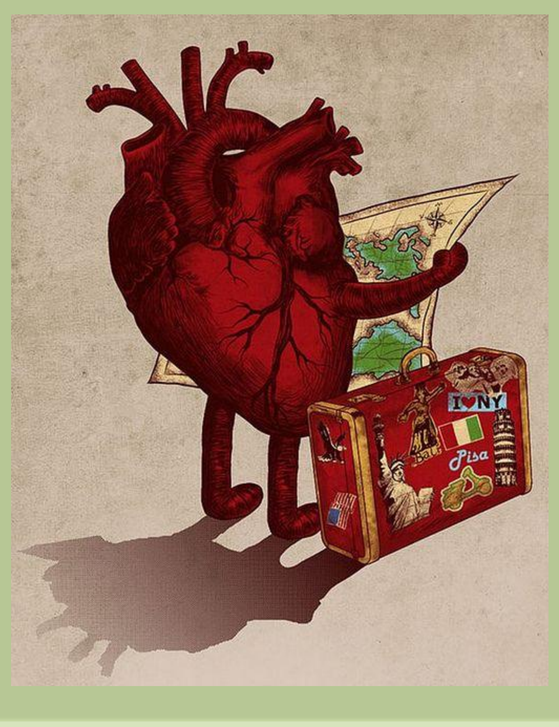
nuclear changes (perinuclear halo)

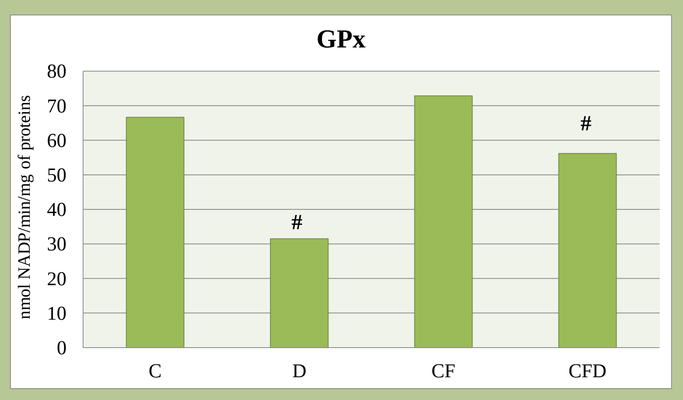


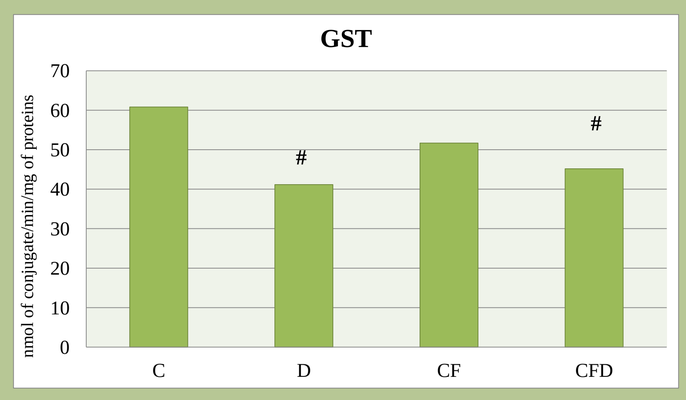




* - statistically significant difference compared to D group (p <0.01)







\# - statistically significant difference compared to K and D groups (p <0.05)

Conclusion: CardiofortIN has a high potential for preventing doxorubicin cardiotoxic effects, through lowering lipid peroxidation, inducing AOE activity, and leading to reduction of myocardial damage.

This study was supported by the Autonomous Province of Vojvodina (Project No 142-451-2543/2021-02)

milana.bosanac@mf.uns.ac.rs