Melatonin controls oxidative stress and modulates iron, ferritin, and transferrin levels in adriamycin treated rats

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Abstract

Aim: Chemotherapy with adriamycin (ADR) is limited by its iron-mediated pro-oxidant toxicity. Because melatonin (MIT) is a broad spectrum antioxidant, we investigated the ability of MIT to control iron, its binding proteins, and the oxidative damage induced by ADR.

Main methods: ADR was given as single i.p. dose of 10 mg kg(-1) body weight into male rats. MLT at a dose of 15 mg kg(-1) was injected daily for 5 days before ADR treatment followed by another injection for 5 days. Biochemical methods were used for this investigation.

Key findings: ADR injection caused elevations in plasma creatine kinase isoenzyme, lactic dehydrogenase, and aminotransferases, iron, ferritin, and transferrin. These changes were associated with increases in lipid peroxidation and protein oxidation as well as decreases in glutathione (GSH) levels and glutathione-S-transferase (GST) activity, while glutathione peroxidase (GSH-Px), and catalase (CAT) activity were elevated in the heart and liver of ADR treated rats. In the MLT+ADR group, the cardiac and hepatic function parameters and the levels of iron, transferrin and ferritin in plasma were normalized to control levels. The rats that were subjected to MLT+ADR had normalized CAT and GSH-Px activity and decreased TBARS and protein carbonyl levels compared the group only treated with ADR. GST activity and GSH concentration in the heart and liver were normalized when MLT accompanied ADR treatment.

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Significance: MLT ameliorated oxidative stress by controlling iron, and binding protein levels in ADR treated rats demonstrating the usefulness of adriamycin in cancer chemotherapy and allowing a better management of iron levels.

Author Keywords: Adriamycin; Oxidative stress; Antioxidants; Melatonin; Heart; Liver; Iron; Ferritin; Transferritin; Iron binding proteins
KeyWords Plus: CENTRAL-NERVOUS-SYSTEM; BOUND IRON; INDUCED CARDIOTOXICITY; HEPATIC TOXICITY; MOLECULAR DAMAGE; IN-VIVO; CELLS; DOXORUBICIN; LEAD; INHIBITION
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