

Protective effect of Coenzyme Q10 on oxidative ovarian and uterine damage induced by methotrexate in rats

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Abstract

Methotrexate (MTX) has toxic effects on the uterus and ovaries via oxidative stress. Coenzyme Q10 (CoQ10) is an important component in electron transport in the mitochondria and an antioxidant in cellular metabolism through the inhibition of lipid peroxidation. The aim of this study was to investigate the preventive effects of CoQ10 on MTX-induced utero-ovarian damage and oxidative stress in rats.

In this experimental study, 30 albino Wistar female rats were divided randomly into three groups. Once a day for a month, 10 mg/kg of CoQ10 was orally administered to the rats in the MTX+CoQ10 group, while the same volume of olive oil was administered orally to the other two groups. One hour thereafter, 20 mg/kg of MTX was injected intraperitoneally into the rats in the MTX and MTX+CoQ10 groups; the remaining group was the control. At the end of the month, biochemical and histopathologic examinations were performed on the extracted uteri and ovaries. In the uterine ovarian tissues of the animals in the MTX group, there was an increase in oxidative stress mediators and a decrease in antioxidant and anti-inflammatory mediators, but these trends were reversed in the MTX+CoQ10 group, demonstrating the antioxidant effects of CoQ10. MTX leads to oxidative stress-related ovarian and uterine injury, and CoQ10 may be useful for protecting ovarian and uterine tissue from such injury.

Keywords

Antioxidants, Coenzyme Q10, lipid peroxidation, methotrexate, oxidative stress

Introduction

Methotrexate (MTX) is a chemotherapeutic folic acid antagonist that is used in the treatment of diseases^{1–3}—particularly inflammatory and autoimmune diseases^{2–4}—but, at high doses, it has serious toxic effects on organs and tissues. Increased levels of reactive oxygen species, oxidative stress, and inflammatory processes have emerged as key players in the pathogenesis of MTX-induced damage to organs and tissues,^{5–7} including ovarian functional and structural disorders and, at high doses, infertility.^{8–10} It has been reported that MTX reduces levels of the antioxidant glutathione (GSH) and increases myeloperoxidase (MPO) and malondialdehyde (MDA),¹¹ the latter of which is an important marker of lipid peroxidation and an indicator of oxidative tissue damage.¹² It has also been reported that MTX causes serious damage to uterine tissue.¹³

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