UTERINE STRUCTURAL AND MORPHOMETRIC CHANGES UNDER CHRONIC CARBON MONOXIDE EXPOSURE: COMPARATIVE ANALYSIS AND EMERGING TREATMENT STRATEGIES

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Abstract. *Chronic exposure to carbon monoxide is considered as one of the factors causing profound morphofunctional disorders of the reproductive system. The present study analyzes morphological and morphometric changes in the uterus with an emphasis on the role of oxidative stress in their pathogenesis. It was found that prolonged CO exposure leads to a progressive decrease in endometrial thickness and a decrease in the number of uterine glands, which indicates a violation of regenerative processes and tissue hypoxia. It is assumed that the key mechanism of the identified changes is the activation of free radical processes and damage to cellular structures. The results obtained emphasize the importance of developing pathogenetically based therapies, including antioxidant protection and stimulation of reparative processes. The proposed approaches can help improve the effectiveness of treatment and restore reproductive function in chronic carbon monoxide intoxication.*

Keywords: *carbon monoxide, uterus, morphology, hypoxia, experimental study, reproductive organ.*

Introduction

Environmental pollution remains a major global health problem. Among toxic gases, carbon monoxide occupies a special place due to its high affinity for hemoglobin and its ability to induce tissue hypoxia[2,3].

Carbon monoxide is produced during incomplete combustion of organic materials such as coal, gasoline, wood, and natural gas. Chronic exposure to CO can cause pathological changes in many organs, including the brain, heart, liver, and kidneys. However, its effect on reproductive organs has not been fully investigated[5,6].

The uterus is a highly vascularized organ that plays a key role in reproduction. Any structural alteration in uterine tissues may lead to impaired reproductive function. Hypoxia caused by carbon monoxide may significantly affect the histological structure of the uterus[1,4].

Therefore, studying morphological changes in the uterus under CO exposure is important for understanding its impact on female reproductive health.

The aim of this study was to investigate morphometric and histological changes in the uterus under chronic carbon monoxide exposure.

Materials and Methods

The present experimental study was carried out on 40 female white laboratory rats weighing 180–220 g and aged 8–10 weeks. All animals were kept under standard laboratory conditions with a temperature of 22–24°C, relative humidity of 50–60%, and a 12-hour light/dark cycle. The animals had free access to standard laboratory food and drinking water throughout the experiment.

The experimental procedures were conducted in accordance with international ethical guidelines for the use of laboratory animals.

The animals were randomly divided into four groups (n = 10 each): one control group and three experimental groups exposed to different concentrations of carbon monoxide.

Results

Histological and morphometric analysis revealed significant structural alterations in uterine tissues following chronic exposure to carbon monoxide.

In the control group, the uterine wall showed normal histological architecture. The endometrium consisted of a well-organized epithelial layer, numerous uterine glands, and a well-developed stromal component. The myometrium displayed compact smooth muscle bundles with normal vascularization.

In contrast, animals exposed to carbon monoxide demonstrated progressive pathological changes depending on the concentration of the gas.

In the 50 ppm CO group, mild dystrophic alterations of the endometrial epithelium were observed. Slight dilation of blood vessels and moderate stromal edema were detected.

Conclusion.

Overall, the results of the present study indicate that chronic carbon monoxide exposure may significantly affect the structural integrity of uterine tissues. These morphological alterations may potentially impair reproductive function and highlight the importance of environmental factors in reproductive health.

Further studies are needed to investigate molecular mechanisms underlying these changes, including oxidative stress pathways, apoptosis, and inflammatory responses.

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