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Morphological Changes in the Blood Vessels of the Femoral Head in Rats Under the Influence of Glucocorticosteroids

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Abstract: This study examined morphological changes in the vascular tissues of the femoral head in rats following the administration of glucocorticosteroids (GCS). Dexamethasone was selected as the GCS agent. Histological and histochemical methods were used for morphological analysis. The results revealed vasculopathic changes in the microcirculatory system of the femoral head, endothelial degradation, and microthrombosis under the influence of GCS.

Keywords: glucocorticosteroids, dexamethasone, femoral head, morphology, vasculopathy, rat.

Introduction

Glucocorticosteroids (GCS) are widely used in clinical practice due to their anti-inflammatory, antiallergic, and immunomodulatory effects. They are employed in the treatment of conditions such as osteoarthritis, bronchial asthma, autoimmune diseases, and post-transplantation reactions (Weinstein, 2012; Liu et al., 2020). However, prolonged high-dose use of GCS can lead to adverse effects on various organs and tissues, particularly bones and blood vessels.

Avascular necrosis of the femoral head (osteonecrosis) is one of the common and serious complications in patients receiving GCS. This condition usually arises due to impaired bone trophism, damage to the microcirculatory system, and restricted blood supply to bone tissue (Mankin, 1992; Kerachian et al., 2009).

Experimental studies, including those on rats, provide deeper insights into these pathological processes. Research using synthetic GCS such as dexamethasone has shown that they exert toxic effects on endothelial cells, causing vasculopathy, microthrombosis, narrowing of capillary lumens, and perivascular fibrosis (Zhang et al., 2019; Liu et al., 2016).

These processes significantly impact both the physiological condition of bone and its regenerative capacity. Therefore, studying the morphological changes in the vascular structure of the femoral head under the influence of GCS is essential for understanding the early stages of osteonecrosis pathogenesis.

The relevance of this topic lies in its potential to identify vasculopathic changes caused by GCS and develop preventive and therapeutic strategies accordingly.

Objective of the study. To assess the morphological and morphometric changes in the blood vessels of the femoral head in rats following dexamethasone administration.

Materials and methods. The study used 30 sexually mature laboratory rats (weighing 180–200 g), divided into 3 groups: group 1 (n=14): received intraperitoneal injections of dexamethasone at 1 mg/kg for 14 days, group 2 (n=16): received dexamethasone at the same dose for 30 days**,** control group (n=10): received normal saline

Methods: After euthanasia, the femoral head was extracted. Tissues were fixed in formalin and embedded in paraffin blocks. Hematoxylin-eosin, Van Gieson, and PAS stains were used. Microscopic, morphometric studies and photography were conducted using a light microscope.

Results and discussion. In the control group, the vessels of the femoral head had a normal structure, with intact endothelial cells and normal basement membrane thickness. In the experimental groups, endothelial cell swelling, microthrombosis, perivascular fibrosis, and microaneurysms were observed.

Prolonged exposure to GCS leads to toxic effects on vascular endothelium, disrupting microcirculation. This may contribute to the development of femoral head osteonecrosis.

Histological analyses revealed dystrophic changes, sclerosis, fibrosis, and increased collagen fibers in vessel media. Focal necrotic areas, leukocyte infiltration, and inflammatory responses in perivascular zones were also noted. Histochemical examination showed weakened staining of glycoproteins in the adventitial tissues, indicating trophic impairment.

Microthrombi, formed due to endothelial damage, block vessel lumens, leading to hypoxia — a key factor in the pathogenesis of osteonecrosis.

Morphometric Indicators of Femoral Head Vessels under GCS Influence

Comparison of morphometric data showed that in rats treated with dexamethasone, capillary diameters significantly decreased and arteriolar wall thickness increased. Additionally, there was an increase in the number of microvessels and percentage of perivascular fibrosis.

Table

(n=10) (n=14)

6.2 ± 0.2 4.5 ± 0.3** (n=16)

2.1 ± 0.3 3.5 ± 0.4* 4.8 ± 0.6**

0.0 6.0 ± 0.5* 7.2 ± 0.3**

3.0 ± 0.8 18.0 ± 2* 27.2 ± 2.2**

Morphometric Parameters	**Control n=10**	**Experimental group 1 n=14**	**Experimental group 2 n=16**
Capillary diameter (μm)	6.2± 0.2	4,5±0.3**	4,1±0.3*
Arteriolar wall thickness (μm)	2.1± 0.3	3,5± 0.4*	4,8± 0.6**
Number of microvessels (per mm²)	0,0	6,0± 0,5*	7.2± 0,3**
Perivascular fibrosis (%)	3.0± 0,8	18,0± 2*	27,2± 2,2**

Note: * – statistically significant difference at $P \leq 0.05$; ** – statistically significant difference at $P \leq 0.01$

Conclusion

Endothelial damage caused by GCS is a major factor in the development of pathological changes in vascular walls, leading to disrupted microcirculation, hypoxia, and impaired bone trophism. This, in turn, can trigger osteonecrosis.

Dexamethasone administration in rats causes significant morphological changes in the blood vessels of the femoral head, indicating the need to adjust the dosage and duration of GCS use to prevent degenerative changes in bone tissue.

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