EFFECTIVE CARE OF EXPERIMENTAL DIABETES DEPENDS ON REGULAR MONITORING OF GLUCOSE AND GLYCATED HEMOGLOBIN LEVELS

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Abstract

Studying the morphofunctional state of pulmonary vascular endothelium in the alloxan diabetes model as a key aspect in evaluating the impact of diabetes on the pulmonary circulatory system

The study analyzed the morphometric changes in small and medium-diameter pulmonary blood vessels. It was found that alloxan diabetes leads to thickening of pulmonary capillary walls, narrowing of vessel diameters, and endothelial damage. These pathological processes are primarily associated with endothelial dysfunction, cellular degeneration, and the toxic effects of glucose.

The research results revealed that diabetes causes interstitial edema, an increase in collagen deposits, and microcirculatory disruptions in pulmonary blood vessels. These morphometric changes result in hypoxia and disturbances in pulmonary circulation as complications of diabetes. The findings of this study contribute to a better understanding of pathological processes in the pulmonary vascular endothelium in diabetes and aim to develop treatment strategies in this field.

Keywords: Alloxan diabetes, pulmonary blood vessels, morphometry, endothelial dysfunction, microcirculation.

Materials and Methods:

The study was conducted on male, outbred white laboratory rats weighing 170–185 g. The animals were kept in standard vivarium conditions with natural feeding and free access to water. An experimental diabetes model was induced using the alloxan preparation (Lachema, Czechoslovakia) administered intraperitoneally at a dose of 130 mg/kg body weight. The animals were fasted for 24 hours to facilitate the development of diabetes. The onset of diabetes was confirmed by measuring blood glucose levels using a Contour Plus glucometer.

Morphometric analysis of lung tissues and blood vessels was conducted on 116 rats at various time points. Micropreparations of lung tissues were obtained from decapitated rats. Tissue components, cellular structures, blood vessel diameters, wall thickness, and affected cells were examined in sections stained with hematoxylin and eosin. A NanoZoomer (REF C13140-21.S/N000198/HAMAMATSU PHOTONICS/431-3196 JAPAN) was used to scan the samples at 200x magnification, and correlations were analyzed using software-based formulas without human intervention.

Study Results:

The table above illustrates the morphometric changes in pulmonary blood vessels observed under the conditions of the alloxan diabetes model. It presents the changes in various parts of the pulmonary vasculature over 30, 60, 90, and 120 days, as well as comparisons with the control group. Below are the detailed findings:

Thickness of Alveolar Capillary Endothelium Layers. The thickness of the endothelial layer in pulmonary capillaries increased with the progression of diabetes. In the control group, the endothelial layer thickness was 9.2 ± 1.11 µm. A significant thickening was observed from 30 days, reaching 10.99 ± 1.15 µm by day 120. This thickening is attributed to the toxic effects of glucose on the endothelium and interstitial edema, which contribute to capillary wall thickening.

This study highlights the pathological changes in the pulmonary vascular endothelium associated with diabetes, providing valuable insights for developing therapeutic approaches.

Muscular Layer of Pulmonary Segmental Vessel Walls. The thickness of the muscular layer in pulmonary segmental vessel walls significantly increased with the progression of diabetes. In the control group, this parameter was 28.11 ± 1.10 µm, while it reached 32.02 ± 1.05 µm at 30 days and a maximum of 43.04 ± 1.01 µm at 90 days. This thickening is associated with hyperplasia of the muscular layer and an increase in collagen deposits in the vessel wall.

Average Diameter of Primary Branch of Pulmonary Lobule (mm). The diameter of vessels located around primary bronchioles increased under diabetic conditions. In the control group, this parameter was 3.35 ± 3.07 mm, while it reached 7.8 ± 2.05 mm by day 120. This change is explained by disruptions in microcirculation and the expansion of vessel walls.

Average Diameter of Secondary Branch of Pulmonary Lobule (mm). The diameter of secondary blood vessels also increased with the progression of diabetes. In the control group, this parameter was 2.2 ± 0.85 mm, rising to 3.1 ± 1.84 mm by day 120. These changes indicate morphological alterations in vessel walls and the toxic effects of glucose on blood vessels.

Average Diameter of Tertiary Branch of Pulmonary Lobule (mm). Changes in the diameter of tertiary distributing vessels were relatively less pronounced. In the control group, this parameter was 1.86 ± 2.01 mm, increasing to 2.2 ± 4.16 mm by day 120. These changes result from the effects of glucose on vascular endothelium and interstitial cells.

Conclusion: The study identified morphometric changes in pulmonary blood vessels under conditions of alloxan-induced diabetes, including thickening of vessel walls, endothelial damage, and the expansion of vessel diameters. These findings indicate the adverse impact of diabetes on the pulmonary circulatory system. Such data are essential for a deeper understanding of pulmonary complications in diabetes and for developing therapeutic approaches aimed at preventing these complications.