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Biochemical And Morphological Changes In The Blood And Lungs Of Rats With Alloxan Diabetes

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Abstract: Alloxan is a toxic chemical widely used in experiments to model diabetes, which primarily affects the pancreas. However, in addition to its direct effects on the endocrine system, alloxan also affects other organs, including the lungs. The morphological changes that can occur in the lungs under the influence of alloxan involve several mechanisms, including oxidative stress, inflammation, and general toxicity of the substance, which reflects the impact of diabetes on respiratory processes. Alloxan-induced diabetes had a significant effect on the lungs of rats, and the changes in the alveoli formed the basis of our findings.

Keywords: Alloxan, oxidation stress, alveoli, BALT, emphysema.

Introduction: Alloxan causes oxidative stress, which can damage lung cells. This leads to the formation of free radicals, which can damage cell membranes, lipids, proteins, and mitochondria. This leads to cellular dysfunction and also has a detrimental effect on lung tissue. Oxidative stress leads to damage to cell membranes, degeneration of cellular structures, and destruction of the alveolar epithelium. Both the alveolar epithelial cells and the cells of the pulmonary vascular walls are damaged. Alloxan activates inflammatory responses in the body, which, of course, also affects the lungs. Activated immune cells, such as macrophages, neutrophils, and lymphocytes, infiltrate lung tissue and cause chronic inflammation. Exposure to alloxan can damage alveolar epithelial cells, which play a vital role in gas exchange. Damage to the alveoli leads to disruption of normal lung function, which is especially important for the exchange of oxygen and carbon dioxide.

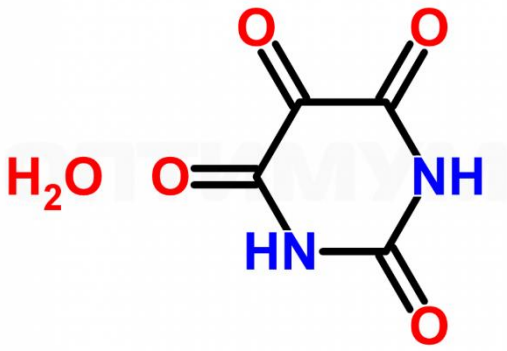

Figure 1. Structure of alloxan monohydrate

After systemic administration, alloxan is distributed throughout highly vascularized tissues, including the lungs. Alloxan is able to penetrate cells via transport systems such as GLUT1 and GLUT2, the glucose

transporters. These transporters are expressed not only in pancreatic β-cells but also in lung tissue, particularly in the epithelial cells of the bronchi and alveoli, as well as in the vascular endothelium. Thus, the lungs become vulnerable to its effects.

Alloxan has a high electron-withdrawing capacity, allowing it to actively participate in electron transfer reactions. In biological systems, it can be reduced to dihydroalloxans, which then spontaneously reoxidize to form reactive oxygen species (ROS), such as:

- superoxide anion (O_2•–),
- hydrogen peroxide (H_2O_2),
- hydroxyl radical (•OH).

It is this feature—cyclic ROS generation—that makes alloxan a potent inducer of oxidative stress, especially in tissues with high metabolism and rich blood supply, such as the lungs.

Alloxan-induced oxidative stress activates a number of intracellular signaling cascades, including:

- NF-κB, a transcription factor for inflammatory genes,
- MAPK/ERK, a cellular stress response cascade,
- Caspase, apoptotic pathways,
- Nrf2, an antioxidant response.

In lung tissue, activation of these pathways leads to:

- Increased secretion of cytokines (IL-6, TNF-α),
- Epithelial damage,
- Active infiltration of macrophages and neutrophils,
- Alveolar cell apoptosis.

METHODS

An alloxan solution was prepared by dissolving alloxan monohydrate in a 0.9% NaCl solution. The solution was filtered and bottled. Experimental studies were conducted on 30 male rats weighing 180-210 g. Diabetes was induced by subcutaneous administration of alloxan tetrahydrate at a dose of 15 mg per 100 g of body weight after a one-day fast.

The alloxan model was developed at the Scientific Laboratory of the Tashkent Medical Academy. Mortality with this method is approximately 20-30%. The method involves depriving the rats of daily food, allowing them to fully satisfy their water requirements.

RESULTS AND DISCUSSIONS

The induction of the alloxan-induced diabetes model can be understood based on blood test results (Table 1). The effect on the lungs became noticeable after 21 days (Figure 2). Morphological studies were conducted in the laboratory of the Department of Pathological Anatomy at the Bukhara State Medical Institute.

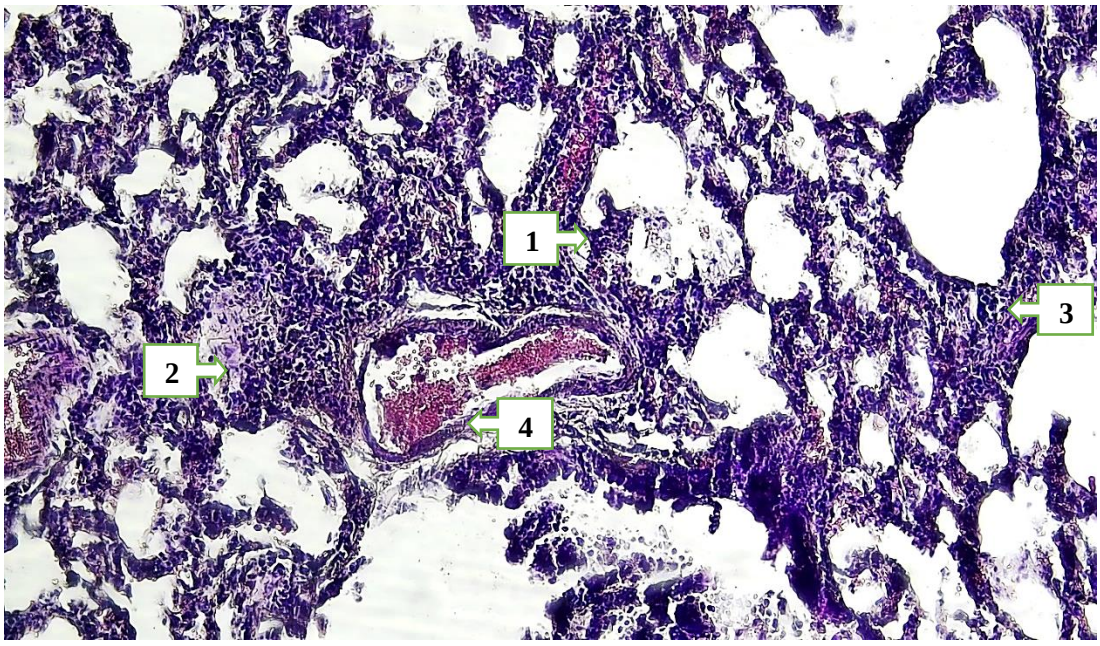

Figure 2. Microscopic view of lung tissue of a mongrel rat with experimental alloxan diabetes (21 days). Stained with hematoxylin and eosin. Magnification 10x20. 1 - formation of emphysematous alveoli (as a result of destruction of interalveolar septa) and accumulation of fluid in them; 2 - increase in BALT (bronchial-associated lymphatic tissue); 3 - thickening of the bronchial wall; 4 - intravascular perfusion.

Chronic inflammation and cellular damage lead to the development of fibrosis in the lungs. This condition is characterized by the replacement of normal lung tissue with connective tissue (collagen), which impairs the elasticity of the lungs and their ability to expand and contract. As a result, the interstitial tissue thickens, and fibrous bands form within the lung tissue, leading to respiratory failure and oxygen deficiency (Figure 2).

This injury can also alter the permeability of pulmonary vessels, which contributes to fluid accumulation in the interstitial and alveolar tissues and causes pulmonary edema. Impact on the endothelial cells of the pulmonary vessels leads to their damage, disruption of blood vessel integrity, and altered blood flow.

Biochemical changes in the lungs are primarily associated with the levels of C-reactive protein and surfactant A, as demonstrated by the blood tests of rats administered alloxan, as shown in Table 1.

Table 1. Changes in blood substance levels in rats with alloxan-induced diabetes.

	Surfactant A	C-reactive protein
healthy	5,47 ±0,5	0,73±0,02
7 days	9,93 ±0,72	0,39 ±0,06
	168,90%	46,58%
14 days	10,63 ±0,91	0,25 ±0,03
	136,40%	68,36%
21 days	11,75 ±1,07	0,15±0,01
	108,70%	79,45%

CONCLUSION

Exposure of rats to alloxan causes a wide range of biochemical changes in the blood, the most important of which are hyperglycemia, hypoinsulinemia, elevated ketone levels, dyslipidemia, and changes in protein and electrolyte levels. These changes reflect metabolic disturbances and the development of diabetes-like diseases accompanied by inflammation, metabolic stress, and damage to various organs, including the lungs and kidneys.

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