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

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Abstract: The morphological effect of alloxan on the pancreas is fundamental for modeling diabetes in experimental medicine. Alloxan is a toxic substance that has a specific effect on pancreatic cells, especially on the beta cells of the islets of Langerhans, which are responsible for insulin production. Morphological changes in the pancreas of rats injected with alloxan tetrahydrate solution confirm the formation of diabetes in the animal's body. In addition, changes in biochemical parameters in the blood indicate the development of hyperglycemia. These processes indicate that a model of diabetes under the influence of alloxan has been successfully induced in the body of rats.

Keywords: Hypoglycemia, Langerhans, alloxan, acinar cell, beta cell.

Introduction: Under physiological conditions, alloxan degrades rapidly, especially in the presence of reducing agents (such as glutathione). Therefore, in experimental practice, its solutions are prepared immediately before use. When reacting with biomolecules, alloxan can modify protein amino groups, oxidize SH groups, and cause irreversible damage to biomembranes. The chemical properties of alloxan are listed in Table 1.

Alloxan is a highly active chemical compound that exerts its toxic effects primarily through the induction of oxidative stress. Its biochemical activity is due to its ability to generate reactive oxygen species (ROS) and interact with cellular components—proteins, lipids, and nucleic acids.

Table 1. Main physicochemical properties of alloxan

Parameter	Meaning
Chemical formula	$C_4H_2N_2O_4$
Molecular weight	142.07 г/моль
Appearance	White or pinkish crystalline powder
Melting point	~250 °C (разложение)
Solubility in water	It dissolves well

Parameter	Meaning
Solubility in organic solvents	Poorly soluble
pH of an aqueous solution	5.0–6.0 (slightly acidic environment)
Sustainability	Oxidizes quickly in air
Biological activity	Oxidative stress inducer

- After entering the cell, alloxan reacts with intracellular reducing agents such as glutathione (GSH), cysteine, ascorbate, and NADPH. These reactions result in the reduction of alloxan to dihydroalloxans, which are rapidly reoxidized, resulting in the formation of:
- Superoxide anion (O_2•–),
- Hydrogen peroxide (H_2O_2),
- Hydroxyl radicals (•OH).

AFK damage:

- Membranes (via lipid peroxidation),
- Proteins (oxidation of sulfhydryl groups, denaturation),
- DNA (strand breaks, mutations),
- Mitochondria (disruption of the respiratory chain, decreased ATP).

METHODS

Experimental studies were conducted on 36 male rats weighing 180-200 g. Diabetes was induced by subcutaneous administration of alloxan tetrahydrate solution at a dose of 15 mg per 100 g of body weight after one day of fasting.

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 rats of daily food, allowing them to fully satisfy their water needs.

RESULTS AND DISCUSSIONS

Morphological analysis of rats administered alloxan is being conducted to assess the pathological changes induced by this substance, which is used to model type 1 diabetes in laboratory animals. Alloxan selectively destroys pancreatic β-cells, causing hyperglycemia. The morphological effects of alloxan on the pancreas include a number of structural changes: degeneration and destruction of beta cells, alpha-cell hypertrophy, fibrosis, inflammation, microinfarctions, and changes in acinar cells. These changes lead to disruption of pancreatic endocrine function and the development of diabetes.

Alloxan, in turn, affects the beta cells of the islets of Langerhans. This is because they cannot be taken. Cell membranes and mitochondria are damaged by the incoming mechanisms of oxidative stress. Langerhans's islets are dehydrated, beta cells are deformed, and observation is not possible. These cells do not have empty space on the islets.

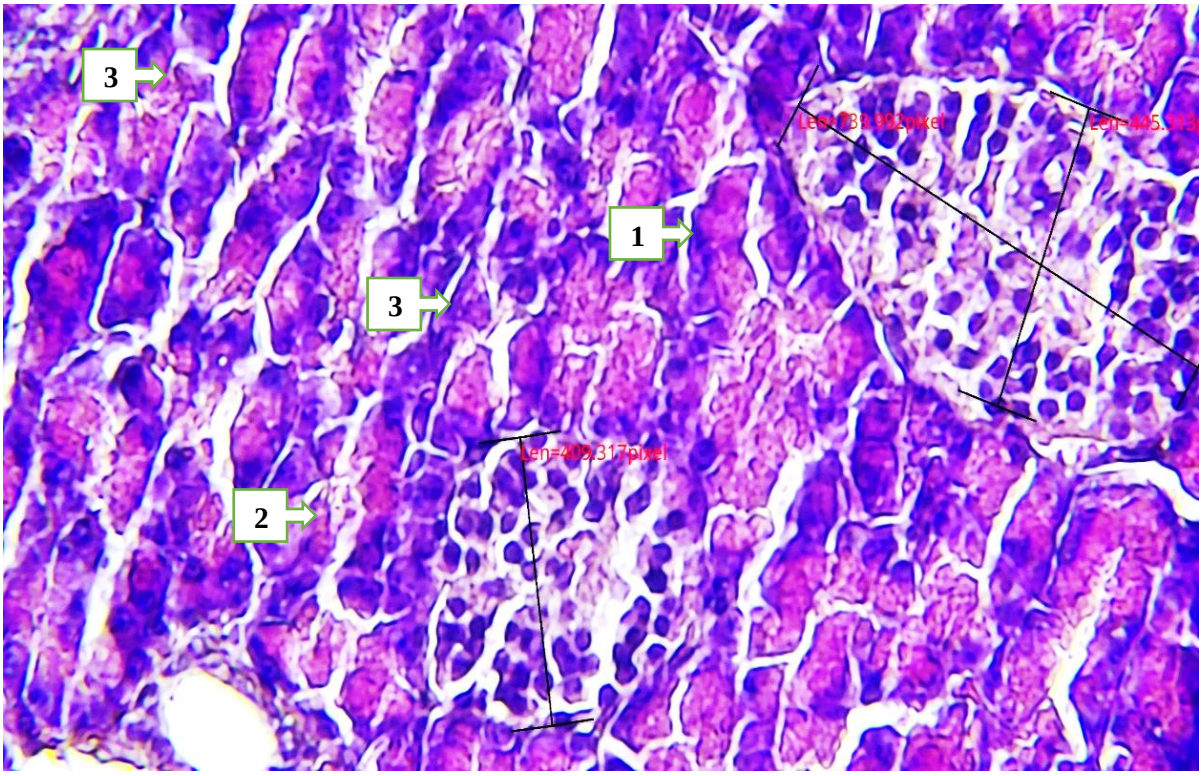

Figure 1. Microscopic view of the pancreas of a white outbred rat with experimental alloxan diabetes (21 days). Stained with hematoxylin and eosin. Magnified 100x20. 1-Edema around the islets of Langerhans; 2-Enlargement of the islets of Langerhans; 3-Death of acinar cells.

Alloxan's chronic effects on the stomach, including fibrosis and sclerosis, are possible. These fibroblast activation and collagen fiber accumulation, associated with simple glandular tissue, can replace the tissue. In response, damage to macrophages and lymphocytes can lead to inflammation. The same may occur with cells. The next step is to die.

From the outside of the islets of Langerhans, the stomach under the diaper contains enzymes that work to digest food, releasing enzymes that are absorbed by the cells. Alloxan and asinar can also cause changes in cells, such as degeneration or atrophy. This enzyme secretion can disrupt the cells (Figure 1).

Table 2. Alloxan diabetes rats in the blood of the substance the amount of change

	Glucose	Glycogen	Pyruvate	Lactic acid
Healthy	5,67 ±0,25	4,54 ±0,29	0,428 ±0,024	1,391 ±0,132
7 days	15,93 ±0,72	18,73 ±1,8	2,9 ±0,22	0,721 ±0,037
	180,90%	242,4%	39,2%	55%
14 days	13,63 ±0,91	15,87 ±1,69	2,75 ±0,23	0,814 ±0,058
	140.40%	170,3%	-44%	82.1%
21 days	11,95 ±1,07	13,82 ±1,24	2,54 ±0,15	0,933 ±0,061
	110.70%	159,3%	-47,5%	93,9%

CONCLUSION

In our observation, the diabetic effect of Alloxan was analyzed morphologically in the pancreas and blood. The results of the analysis are presented in Table 2. Blood sugar and acid levels increased. Conversely, glycogen and lactic acid levels decreased, indicating that diabetes has been present in animals since its onset.

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