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EFFECT OF BITTER ALMOND OIL ON LIPID PEROXIDATION PROCESSES IN RATS WITH ALLOXAN DIABETES AND IDIOPATHIC PULMONARY FIBROSIS

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Abstract. *Chronic lung inflammation caused by tobacco smoke is characterized by intensification of POL in lung tissue, compensatory activation of GPO on the background of suppression of SOD and catalase enzymes activity. The main mechanism of development of progressive pulmonary fibrosis is repeated and persistent damages of alveolar epithelium with their subsequent dysregulated repair. Alloxan administration against this background further aggravates the imbalance in the pro- and antioxidant system. Experimental pharmacotherapy with Gerbion and MGM to some extent restored the balance in this system, it was more pronounced when using MGM. Thus, when exposed to alloxan on the background of existing lung damage leads to even greater damage to lung tissue, manifested by activation of free-radical processes and decay of elastin fibers of alveoli. In contrast to the Gerbion preparation widely used in pulmonology, bitter almond oil has a more pronounced therapeutic effect, inhibiting POL processes and activating the activity of SOD and catalase enzymes in lung homogenate of experimental animals. It demonstrates the most pronounced antioxidant effect, effectively normalizing markers of inflammation and lung tissue damage, increases insulin production and normalizes glycemia to a certain extent. This allows us to recommend its use in pulmonology.*

Keywords*: alloxan diabetes, lipid peroxidation, pulmonary fibrosis, oxidative stress, elastin, pulmonology, chronic inflammatory*

Introduction.

Despite significant progress achieved in the study of pathogenesis, diagnosis, prediction of lung disease progression, especially interstitial pulmonary fibrosis, its course in the presence of concomitant diseases, the mechanism of interrelation remain insufficiently illuminated. The main mechanism of development of progressive pulmonary fibrosis is repeated and persistent damages of alveolar epithelium with their subsequent dysregulated repair [3]. The main cells responsible for the development of pulmonary fibrotic remodeling are myofibroblasts and their precursors [18]. The mechanisms underlying the recruitment and proliferation of these cells are associated with the presence of a large number of mediators (cytokines, chemokines, fibrogenic factors, coagulation proteins, pro-oxidants and regulators of apoptosis [8]. The association of ILF with smoking, exposure to organic and inorganic dust species, pharmacologic therapy, and infectious factors has been shown. Genomic studies conducted by Peljto A.L. et al. (2013) showed an association of ILF with a single nucleotide allelic variant of the promoter of the MUC5B gene, which was present in 38% of patients' individuals [11]. Other genes involved in the development of familial pulmonary fibrosis include surfactant protein C and A2 genes [17]. Increased indices of vascular wall stiffness associated with hypoxemia and pulmonary fibrosis have been found in ILF patients [7]. The key point in the pathogenesis of both COPD and diabetes mellitus is the concept of chronic persistent systemic inflammation, which results in functional and structural changes in other organs and systems [16]. This is associated with the presence of such important mechanisms of pathological process as inflammation, oxidative stress and hypoxia.

Nowadays, free-radical mechanisms play an important role in the occurrence of many pathologies [1, 5, 12, 13]. This is manifested by the imbalance of oxidant and antioxidant defense systems, which necessitates the prescription of preparations with antioxidant action. The main mechanism of their action is due to the inhibition of lipid peroxidation (LPO) in biomembranes, stabilization of their structure and function, which restores homeostasis in the body and increases resistance to pathogenic factors [14]. Oxidized free radicals can perform a useful function, including neutrophils, macrophages, which, intensively producing them, phagocytize the bacterial cell. The concentration of malonoid dialdehyde (MDA) in blood serum serves as an indicator of the level of endogenous intoxication, which reflects the activity of lipid peroxidation processes in the patient's body [1, 5, 12].

In recent years, new directions of therapy of ILF, including pulmonary and extrapulmonary lung cell function, considered to be one of the most promising, have been actively developed. Experimental studies on the correction of pulmonary and extrapulmonary lung cell function in small laboratory animals with modeled pulmonary fibrosis have proved the efficacy and safety of herbal preparations. Nevertheless, many questions regarding the use of herbal preparations from local raw materials remain unresolved.

The aim of the study: to evaluate the efficacy of bitter almond oil in the correction of lipid peroxidation in IFL and alloxan diabetes.

Materials and methods of research.

Experimental studies were conducted in the scientific laboratory of Tashkent Medical University. Animal studies were performed in compliance with the requirements of GOST O´zDSt 2762:2018 (Good laboratory practice, GLP) [4]. Fifty white male mongrel rats, body weight 150-200 g, in which interstitial pneumonia was induced by chronic exposure to tobacco smoke in a special Kurlandsky chamber for 2.5 months were used for the study [5]. The rats were kept under standard conditions in the vivarium with natural light and free access to water and food. A special chamber designed to simulate chronic exposure to tobacco smoke provides controlled and repeatable exposure of rats to smoke from cigarette combustion. The Kurlandsky chamber is a device designed to conduct inhalation experiments with animals, specifically rats. Pall mall cigarettes (Uzbekistan) were burned in the chamber until ash was formed, which contained 0.7 mg nicotine. The combustion process took 20-30 minutes, during which the concentration of tobacco smoke increases and then remains at approximately the same level for 30-40 minutes with a slight downward change. After the procedure, the chamber is ventilated. Smoking sessions are performed once a day daily for 75 days. Rats inhaled tobacco smoke, resulting in irritation of the respiratory tract and development of inflammation in the lungs. After exposure to tobacco smoke, clinical signs of pneumonia were evaluated: change in respiratory rate, presence of cough, possible nasal and respiratory secretions. During the period of the experiment on day 21 of the study, lethal outcome was observed in one rat.

After 1 month, 30 rats were reproduced with DM by 2-fold administration of alloxan solution (AD) at a dose of 170 mg/kg of animal weight subcutaneously. After 1 month from the beginning of alloxan administration, the surviving animals were divided into groups to evaluate the efficacy of bitter almond oil (BAME). Gerbion produced by KRKA (Slovenia) was taken as a comparison drug. The preparations were administered for 15 days once a day per os: Gerbion - 3.2 ml/kg once a day, almond oil - 1.2 ml/kg once a day.

The rats were distributed into groups as follows:

Group I. Intact rats with which no manipulations were performed (8 rats); Group II. COPD without treatment (2.5 months) (10 rats);

Group III. COPD + alloxan diabetes without treatment (2.5 months) (10 rats), control;

Group IV. COPD+ alloxan diabetes+ herbion at the dose, 3.2 ml/kg (2.5 months) (10 rats), comparison;

Group V. COPD+ alloxan diabetes + bitter almond oil at a dose of 1.2 ml/kg (2.5 months) (10 rats) main.

During the whole experiment, the weight of rats, animal condition, pulse and respiration rates were determined. At the end of the experiment, the animals were decapitated, blood was collected, and serum was separated. Lung tissue was also used for the studies. Part of the lung tissue was taken on chloroform for morphologic studies, and from the rest homogenate was prepared in Tris-HCl buffer at the rate of 0.5 g/1 ml of buffer. Common biochemical parameters, glycemia level, lipid spectrum, and insulin content were determined in serum on a biochemical analyzer HumaLuzerPrimus 602828 (Germany) using a reagent kit of the same company. The content of malonic dialdehyde (MDA) in lung homogenate was determined according to the method of L.I. Andreeva et al. [2], based on the interaction of 2-3 diene bonds of unsaturated fatty acids with thiobarbituric acid to form MDA. The activity of catalase was determined according to the method of M.I. Koralyuk et al. (1986), the principle of determination of which is based on the interaction of H_2O_2 with molybdenum, resulting in the formation of yellow staining, the intensity of which depends on the concentration of H_2O_2 in the medium [6]. The activity of SOD was determined according to the method of V.G. Mkhitaryan and G.E. Badalyan (1978) [10]. Glutathione peroxidase activity was determined by the rate of oxidation of reduced glutathione in the presence of tretbutyl hydroperoxide [9]. The obtained data were processed using the program "STATISTICA", by paired Student's criterion [15].

Results and their discussion.

The conducted studies showed that 2-month inoculation in the Kurlandsky chamber led to the development of interstitial fibrosing alveolitis confirmed morphologically. The condition of rats sharply deteriorated, apathy developed, weight loss, hair acquired a dirty color and fell out in some places. The study of body weight of experimental animals showed a tendency to decrease, whereas in intact rats it statistically significantly increased by 42.5% ($p < 0.01$). In the group of rats with COPD+AD after administration of alloxan to rats the condition of rats deteriorated even more, polyphagia, polydipsia were noted. Weight loss amounted to 31,4% relative to the initial level (p< 0,05), compared to the values of the 1st group - 55,7% (p< 0,01) and relative to the values of the 2nd group - 33% (p< 0,05). Lethality in this group amounted to 30%.

Experimental pharmacotherapy for 15 days with Herbion promoted some improvement of the rats condition. Weight loss was 28.8% (p< 0.05) relative to the initial level, 51.1% (p< 0.01) relative to the values of the 1st group and 25.9% (p< 0.05) relative to the values of the 2nd group. No animal lethality was observed in this group. Experimental pharmacotherapy for 15 days with bitter almond oil promoted some improvement of the rats condition. Weight loss amounted to 21,9% (p< 0,05) in comparison with the initial level, 46,8% (p< 0,01) in comparison with the values of the 1st group and 19,3% (p< 0,05) in comparison with the values of the 2nd group. No lethality of animals in this group was observed.

The analysis of serum glucose level showed that in animals with COPD has only a tendency to increase (initially 5,03±0,27 mmol/l, after 2,5 months - 5,58±0,27 mmol/l). In the group of rats with COPD+AD the glucose content statistically significantly increased by 92,6% in comparison with the initial level and amounted to 10,11±0,45 mmol/l (p< 0,001). This index was higher than the values of the intact group of rats by 80.2% (p< 0.001) and by 81.2% (p< 0.001) compared to the values of the group of rats with COPD. Experimental pharmacotherapy of COPD+AD in rats with Herbion for 15 days promoted decrease of serum glucose level by 23% (p< 0.05) relative to the values of rats of the control group, amounting to 8.30±0.47 mmol/l (p< 0.001). However, the studied index remained higher than the initial values by 57.4% (p< 0.01), values of the intact group - by 47.9% (p< 0.01) and values of the group of rats with COPD - by 48.7% (p< 0.01).

The same dynamics, but more pronounced, was noted in the group of rats with COPD+AD treated with bitter almond oil. Experimental pharmacotherapy of COPD+AD in rats with bitter almond oil for 15 days contributed to the decrease of serum glucose level by 39,6% (p< 0,05) relative to the values of rats of the control group, amounting to 6,11±0,24 mmol/l. However, the studied index remained higher than the initial values by 19,8% (p< 0,05), values of the intact group - by 8,9% and values of the group of rats with COPD - by 9,5%.

To confirm hypoglycemic properties of the preparations we determined insulin content in blood serum of experimental animals. The studies showed that in intact animals the insulin content in blood serum amounted to 118.5±1.2 μUed/mL. Reproduction of COPD in experimental animals promoted statistically insignificant increase of insulin in blood serum up to 124,7±3,3 μED/mL that, in our opinion, is connected with adaptive reorganization of the organism of experimental animals. Alloxan administration against the background of toxicant exposure contributed to a sharp decrease in the insulin content in the blood serum of rats. Its values decreased 1.6 times (p< 0.001) relative to the values of intact rats and 1.68 times (p< 0.01) - compared to the values of the group of rats with COPD, amounting to 74.2±3.3 μED/mL. The mechanism of toxic effect of alloxan on pancreatic β-cells is associated with increased formation of free oxygen radicals with low activity of antioxidant defense enzymes. This leads to destruction of cell biomembranes, degradation and suppression of their functional defense. This coincides with loss of control of glucose homeostasis, weakening of the functioning of insulin-sensitive Glut-4-transporter and development of hyperglycemia.

Experimental pharmacotherapy of COPD+AD with Herbion for 15 days promoted some increase of insulin level in blood serum of this group of animals up to 84.1±3.2 μED/mL. We observed a 1.13 (P< 0.05) fold increase in insulin content relative to the values of the group of rats with COPD+AD. However, it should be said that these indices remained lower than the values of the group of rats with COPD in 1.48 (P< 0.01) times and the values of the intact group - 1.41 (P< 0.01) times.

Experimental treatment of COPD+AD with bitter almond oil also promoted the increase of insulin content in the blood serum of rats and amounted to 88,3±2,7 μED/mL, i.e. 1,19 (P<0,05) times relative to the values of the group of rats with COPD+AD. However, as in the previous group, these indices remained below the values of the group of rats with COPD by 1.41 (P<0.01) times and those of the intact group - by 1.34 (P<0.05) times. It should be noted that the insulin-stimulating effect of bitter almond oil slightly exceeded the values of the group of COPD+AD rats receiving Gerbion. Apparently, this is due to the higher content of polyunsaturated fatty acids and other biologically active compounds in the composition of bitter almond extract.

Thus, the combination of such pathologies as COPD and AD leads to a more severe course of diabetes with marked hyperglycemia on the background of hypoinsulinemia. Experimental pharmacotherapy of COPD+AD rats with Herbion or bitter almond oil led to a decrease in the severity of hyperglycemia and hypoinsulinemia. Bitter almond oil was more effective.

The study of POL indicators and activity of antioxidant defense enzymes showed that in rats with COPD in lung homogenate the content of MDA statistically significantly increased in 2.56 times relative to the values of intact rats, against the background of decreased activity of SOD and catalase enzymes in 1.43 and 1.58 times;

increased activity of GPO in 2.58 times (Table). This was apparently associated with the presence of chronic interstitial inflammation and activation of neutrophil-macrophage system. Alloxan administration against this background promoted even greater induction of POL. Thus, MDA content significantly increased 3.27 and 1.28 times relative to the values of the 1st and 2nd groups, respectively. At the same time, the activity of GPO increased 4.13 and 1.6 times in the above groups, respectively. At the same time, low SOD and catalase activities continued to decrease: 1.31 and 1.17 times relative to the values of the 2nd group; 1.87 and 1.85 times relative to the values of the intact group, respectively, for the enzymes. Probably, it was caused by the increase of free-radical processes in lung homogenate of rats with COPD under the influence of alloxan.

Table.

MDA content and activity of GPO, SOD and catalase enzymes in lung tissue homogenate of experimental animals

Groups	MDA, nmol/0.05 g of tissue	GPO, IU/mg protein	SOD, u.u./min*mg protein	CAT, nmol H(2) o2/min*mg protein
Group I	1,09 ± 0,11	0,52 ± 0,04	1,44 ± 0,11	5,21 ± 0,36
Group II	2,79 ± 0,26	1,34± 0,11	1,01 ± 0,10	3,29 ± 0,23
	$P_1>0.002$	$P_1>0.001$	$P_1>0.05$	$P_1>0.01$
Group III	3,56 ± 0,27	2,15 ± 0,19	0,77 ± 0,07	2,82 ± 0,32
	$P_1>0.001$	$P_1>0.001$	$P_1>0.01$	$P_1>0.01$
	$P_2>0.05$	$R_2>0.02$	$R_2>0.05$	$R_2>0.05$
Group IV	2,12 ± 0,19	1,09 ± 0,19	1,04 ± 0,08	3,86 ± 0,24
	$P_1>0.01$	$P_1>0.05$	$P_1>0.05$	$P_1>0.05$
	$P_2>0.05$	$R_2>0.05$	$R_2>0.05$	$R_2>0.05$
	$P_3>0.01$	$P_3>0.001$	$P_3>0.05$	$R_3>0.05$
Group V	1,41 ± 0,13	0,79 ± 0,07	1,24 ± 0,08	4,87 ± 0,32
	$P_1>0.05$	$P_1>0.05$	$P_1>0.05$	$P_1>0.05$
	$P_2>0.01$	$R_2>0.01$	$R_2>0.05$	$R_2>0.01$
	$R_3>0.001$	$R_3>0.001$	$R_3>0.01$	$P_3>0.01$

Note: P1 - reliability relative to the values of group 1; P2 - reliability in relation to the values of the 3rd group; P3 - reliability in relation to the values of the 4th group.

Experimental pharmacotherapy of rats with COPD+AD with Gerbion for 15 days resulted in a significant decrease of MDA content 1.68 times relative to the values of the 3rd untreated group and 1.32 times - to the values of the 2nd group of animals. However, it should be noted that these values still significantly exceeded the values of intact rats by 1.94 times. It should be said that administration of Gerbion to this group of rats suppressed superactivation of GPO in lung homogenate. As can be seen from the table, high activity of GPO in rats with COPD+AD decreased by 1.97 and 1.23 times relative to the values of the 3rd and 2nd groups, respectively, but still exceeded by 2.1 times the activity of GPO of intact rats. Low activities of SOD and catalase in the process of pharmacotherapy with Gerbion increased 1.35 and 1.37 times relative to the values of the 3rd group, tended to increase relative to the values of the 2nd group, but still remained below the values of the intact group of rats in 1.38 and 1.35 times, respectively, the activities of SOD and catalase enzymes.

Experimental pharmacotherapy of rats with COPD+AD with bitter almond oil for 15 days resulted in a significant decrease of MDA content in 2.52 times relative to the values of the 3rd untreated group and in 1.98 times relative to the values of the 2nd group of animals. However, it should be noted that these values still significantly exceeded the values of intact rats by 1.29 times. The antioxidant effect of MGM was more pronounced, as the content of MDA in the lung tissue of rats treated with MGM was 1.5 times lower than in rats treated with Gerbion. It should be said that administration of MGM to rats with IFL+AD suppressed high values of GPO in lung homogenate by 2.72 and 1.7 times relative to the values of the 3rd and 2nd groups, respectively, but still exceeded by 1.52 times the GPO activity of intact rats. Comparative analysis of GPO activity in lung tissue homogenate of IFL+AD rats treated with MGM was 1.38 times lower than the values of the group of rats treated with Gerbion. Low activities of SOD and catalase in the process of MGM pharmacotherapy increased 1.61 and 1.73 times relative to the values of the 3rd group, 1.23 and 1.49 times exceeded the values of the 2nd group, but still remained statistically insignificantly lower than the values of the intact group of rats in 1.16 and 1.07 times, respectively, the activities of SOD and catalase enzymes. It should be said that the activity of SOD and catalase in the group of rats treated with MGM were 1.19 and 1.26 times higher than the values of the group of rats treated with Gerbion.

Thus, chronic lung inflammation caused by tobacco smoke is characterized by intensification of POL in lung tissue, compensatory activation of GPO on the background of suppression of SOD and catalase enzymes activity. Alloxan administration against this background further aggravates the imbalance in the pro- and antioxidant system. Experimental pharmacotherapy with Gerbion and MGM to some extent restored the balance in this system, it was more pronounced when using MGM.

Analyzing the obtained data it should be said that the composition of Gerbioh includes extracts of roots of spring primrose and thyme in the ratio [14]. The drug is used as an expectorant in the complex therapy of inflammatory diseases of the respiratory tract accompanied by cough. Gerbion has expectorant, anti-inflammatory, antimicrobial action, helps reduce the viscosity of sputum and better expectoration of sputum.

In contrast, bitter almond oil is a dietary supplement and is recommended as a natural source of biologically active substances that help normalize the functioning of the gastrointestinal tract. Almond oil contains a lot of fats, organic acids, tannins and vitamins. The oil is rich in omega-9, omega-6, palmitic acid, vitamins B, E, K, magnesium, calcium, zinc, copper. It contains phytosterols, tocosterols, amygdalins. It has anti-inflammatory, wound-healing, enveloping, laxative and anticonvulsant action. It can be recommended for gastric and duodenal ulcers, gastritis, nephritis, chronic inflammatory diseases of the upper respiratory tract.

Conclusion.

Thus, when exposed to alloxan on the background of existing lung damage leads to even greater damage to lung tissue, manifested by activation of free-radical processes and decay of elastin fibers of alveoli. In contrast to the Gerbion preparation widely used in pulmonology, bitter almond oil has a more pronounced therapeutic effect, inhibiting POL processes and activating the activity of SOD and catalase enzymes in lung homogenate of experimental animals. It demonstrates the most pronounced antioxidant effect, effectively normalizing markers of inflammation and lung tissue damage, increases insulin production and normalizes glycemia to a certain extent. This allows us to recommend its use in pulmonology.

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