

EARLY CHANGES IN THE LUNGS OF WHITE RATS FOLLOWING INTRATRACHEAL ADMINISTRATION OF POTASSIUM DICHROMATE

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Abstract. The article presents a study of the early morphological changes in the lungs of white rats following intratracheal administration of potassium dichromate. The experiments were conducted on 41 white rats. Potassium dichromate was administered intratracheally as a single dose of 0.2 mL of a 5% solution. One day after administration, the predominant morphological finding was diffuse or focal pulmonary edema. One month later, the inflammatory lesions were progressively replaced by granulation tissue containing aggregates of lymphoid cells, as well as purulent inflammatory infiltrates located around blood vessels and bronchi.

Keywords. white rats; lungs; potassium dichromate; hematoxylin and eosin staining; inflammatory foci; purulent infiltrates.

РАННИЕ ИЗМЕНЕНИЯ В ЛЕГКИХ БЕЛЫХ КРЫС ПРИ ИНТРАТРАХЕАЛЬНОМ ВВЕДЕНИИ БИХРОМАТА КАЛИЯ

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Резюме: В статье изучены ранние изменения в легких белых крыс при интратрахеальном введении бихромата калия. Опыты были поставлены на 41 белой крысе. Бихромат калия вводился интратрахеально в дозе 0,2 мл 5% раствора однократно. Через сутки после введения бихромата калия морфологически на первый план выступает картина диффузного или очагового отека легочной ткани. Через месяц воспалительные участки все больше замещаются грануляционной тканью, в толще которой встречаются

скопления лимфоидных элементов, а также гнойные инфильтраты в области сосудов и бронхов.

Ключевые слова: белых крысы, легких, бихромат калия, методы гематоксилином и эозином, очаги воспаления, гнойные инфильтраты.

Introduction. The primary route of occupational exposure to chromium compounds is inhalation of airborne aerosols, which enter the respiratory tract during industrial activities [5,6]. According to most authors, the most common pathological changes in the respiratory tract during the first months of occupational exposure include catarrhal and ulcerative rhinitis, as well as perforation of the cartilaginous portion of the nasal septum [1,2]. The issue of early morphological changes in lung tissue following inhalation of chromium compounds has not been sufficiently investigated [3,4]. It is known that chromium entering through the respiratory tract reaches the bronchioles and alveoli, where it is partially dissolved and absorbed into the systemic circulation, while another portion accumulates within the lung tissue.

Objective. The aim of this study was to investigate the early morphological changes in the lungs and the rate of chromium accumulation following intratracheal administration of potassium dichromate.

Materials and Methods. The study was carried out on 41 white rats. Potassium dichromate was administered intratracheally as a single dose of 0.2 mL of a 5% solution, corresponding to 10 mg of potassium dichromate (dry weight equivalent). Histological examination of the lungs began one day after administration and continued for the following 40 days.

The lungs were fixed in 10% neutral buffered formalin, embedded in paraffin, sectioned, and stained with hematoxylin and eosin. Chromium concentration in the lung tissue was determined colorimetrically using the diphenylcarbazide method.

Results. Twenty-four hours after intratracheal administration of potassium dichromate, diffuse or focal pulmonary edema became the predominant

morphological finding. Marked vascular congestion accompanied by numerous small hemorrhages was observed within the edematous lung tissue.

During the subsequent 3–5 days, in addition to vascular disturbances, activation of reticuloendothelial cells within the interstitial tissue was detected together with necrotic changes involving the walls of blood vessels, bronchi, and peribronchial connective tissue. By days 10–12, against a background of pronounced venous congestion, inflammatory foci developed within the lungs, occasionally accompanied by abscess formation. In several areas, productive inflammation predominated with the development of granulation tissue.

One month after exposure, inflammatory lesions were progressively replaced by granulation tissue containing aggregates of lymphoid cells and purulent inflammatory infiltrates surrounding blood vessels and bronchi. Bronchial lesions were frequently associated with bronchiectasis filled with purulent exudate. Along with degeneration and desquamation of the bronchial epithelium, marked epithelial regeneration was observed. The vascular walls underwent purulent inflammation followed by hyalinization and sclerosis. Thus, one month after potassium dichromate administration, organizing (carnifying) pneumonia developed, occasionally complicated by pulmonary abscess formation.

To reduce the high mortality observed during the early stages of the experiment following administration of a relatively large single dose of potassium dichromate (10 mg dry substance per 300 g body weight), an alternative approach was investigated to prolong chromium retention within the lungs. This strategy was expected to produce the same toxic effect on lung tissue while using a considerably lower effective dose. In the second series of experiments, white rats received 0.2 mL of a 5% potassium dichromate solution mixed with colloidal Infusin, which served as an adsorbent. In addition to non-anaphylactogenic casein, Infusin contains sodium, potassium, and magnesium salts. For comparison, chromium concentrations and morphological changes in the lungs were evaluated at identical time points in both experimental groups. On day 20, chromium concentration in

the lungs of the first group was 1.7 mg%, whereas in the second group, which received potassium dichromate together with Infusin, it reached 4.6 mg%, nearly three times higher. Histological examination during the first and second days revealed pulmonary edema, vascular disturbances, and proliferation of cellular elements similar to those observed in the first experimental series. A distinctive feature of this group was the abundance of eosinophils forming nodular aggregates or diffusely infiltrating the interstitial tissue, which most likely reflected an allergic response to the degradation products of the protein component.

During the following days, proliferative processes were accompanied by progressively increasing tissue destruction involving the bronchial walls and blood vessels. Chronic obliterative bronchitis and peritracheal interstitial pneumonia developed. By day 26, numerous fibrotic areas were identified against a background of chronic productive inflammation, while bronchi and blood vessels demonstrated sclerosis and obliteration.

Comparison of these findings with morphological changes observed in white rats following gastrointestinal exposure to chromium demonstrated that the pathological patterns were generally similar. However, intratracheal administration of potassium dichromate resulted in a considerably more rapid onset and greater severity of pulmonary lesions.

Conclusion. Intratracheal administration of potassium dichromate, either alone or in combination with a colloidal adsorbent, results—after a brief initial period (1–2 days) of diffuse or focal pulmonary edema and circulatory disturbances—in the development of chronic obliterative bronchitis and productive interstitial pneumonia. By the end of the first month, the pathological process is characterized by marked sclerosis of the lung parenchyma, blood vessels, and bronchi, and in some cases by abscess-forming pneumonia accompanied by purulent bronchiectasis.

Prolonging the deposition of potassium dichromate in the lungs through co-administration with a colloidal adsorbent makes it possible to reduce the required dose of the toxic agent while maintaining its pathological effects.

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