

MORPHOLOGICAL CHANGES IN THE CENTRAL NERVOUS SYSTEM UNDER EXPERIMENTAL EXPOSURE TO METHYL VINYL PYRIDINE

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Abstract. The effect of methylvinylpyridine on the brain was studied in rats after oral administration of the solution for 15 days. Brain tissue was fixed in Carnoy's solution and 10% formalin. Histological examination (hematoxylin and eosin, Nissl method) revealed congestion of the meninges and brain parenchyma, capillary congestion, degenerative changes in neuroglia, and pronounced swelling of neurons.

Keywords: experiment, rats, methylvinylpyridine, brain, hematoxylin and eosin, Nissl method, neurons.

МОРФОЛОГИЧЕСКИЕ ИЗМЕНЕНИЯ ЦЕНТРАЛЬНОЙ НЕРВНОЙ СИСТЕМЫ ПОД ВОЗДЕЙСТВИЕМ МЕТИЛВИНИЛПИРИДИНА В ЭКСПЕРИМЕНТАЛЬНЫХ УСЛОВИЯХ

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Аннотация. В эксперименте на крысах изучено влияние метилвинилпиридина на головной мозг при пероральном введении раствора в течение 15 дней. Ткань мозга фиксировали в жидкости Карнуа и 10%-ном формалине. Гистологическое исследование (гематоксилин-эозин, метод Ниссля) выявило полнокровие оболочек и паренхимы мозга, стаз в капиллярах, дистрофические изменения нейроглии и выраженное набухание нейроцитов.

Ключевые слова: эксперимент, крысы, метилвинилпиридин, головной мозг, гематоксилин-эозин, метод Ниссля, нейроны.

Introduction. In recent years, there has been a significant increase in interest in pyridine bases, including methylvinylpyridine (MVP), which are used in the production of synthetic rubber, latexes, plastics, ion-exchange resins, and other materials. The widespread use of pyridines in the national economy leads to contact with these substances by a large number of people; therefore, under certain conditions, toxic effects of these compounds on workers are possible [1,2,3]. Pyridine is known to have a dual action: local, irritating the mucous membranes and skin, and general toxic, manifested primarily in damage to the central nervous and vascular systems. In clinical settings, depression of the central nervous system has been described, resembling poisoning by narcotic substances [4,5,6,7]. Data from morphological studies of the nervous system in pyridine poisoning are scarce and unsystematized, and with regard to MVP, they are completely absent. The task of our work was to study the biological effect of this pyridine base on the brain of animals.

Aim of the study. To investigate morphological changes in the central nervous system under the influence of methylvinylpyridine in experimental conditions.

Materials and methods. The experiment involved 55 white rats, divided into three experimental groups and one control group. For 15-day oral intoxication, aqueous solutions of MVP were prepared by calculation, containing 1/7, 1/4, and 1/21 of the LD₁₀₀ (LD₁₀₀ of MVP = 1.4 g/kg body weight). This corresponded to 200 mg/kg, 100 mg/kg, and 66 mg/kg body weight for the first, second, and third groups, respectively. Control animals received tap water at the same morning hours. Experimental and control animals were euthanized by decapitation. The brain was fixed in Carnoy's fluid and 10% neutral formalin, followed by embedding in celloidin, paraffin, and gelatin. Sagittal and frontal sections were stained with hematoxylin-eosin and by the Nissl method.

Results. Microscopic examination of brain preparations from animals of the experimental groups revealed similar changes of varying severity. Therefore, a summary description of the entire material is provided. The brain substance and pia mater were congested with blood. Significant hyperemia with capillary stases was more pronounced in the base of the brain, midbrain, and medulla oblongata. Occasionally, leukocytic stases were observed in such vessels, as well as diapedesis of erythrocytes and plasmatic impregnation of the surrounding brain tissue. Uneven blood filling with alternation of paralytically dilated, blood-filled, and collapsed vessels was noted. This was particularly evident in the frontal cortex of the cerebral hemispheres, Ammon's horn, and basal nuclei of the brain. Rarely, pronounced tortuosity of capillaries was observed, indicating decreased tone of their walls. The endothelium of blood vessels appeared altered: some endothelial cells became pale and increased in volume. Nuclei in such cells were barely discernible, and their internal structure was indistinguishable. Desquamation of endothelium was sometimes observed. In the choroid plexus, paralytically dilated and collapsed blood vessels were also noted. In places, diapedetic hemorrhages were present. The epithelium of the choroid plexus and the ependyma lining the walls of the cerebral ventricles were significantly altered in some areas. Swelling of individual cells was observed, with alternation of pale, enlarged ependymal cells and flattened cells with pyknotic hyperchromic nuclei. In neurons of various cortical regions, chromatolysis phenomena were noted, occasionally vacuolization. Very rarely, shrunken, intensely staining cells with sharply twisted apical dendrites were encountered. In neurons of the thalamo-hypothalamic region, chromatolysis was noted, in places total; ghost cells appeared, neuronal vacuolization with significant reduction in Nissl substance reaction. Here, deformed hyperchromic cells were more frequent than in the cortex. In large neurons of the midbrain and medulla, "skeletonization" of neuronal cytoplasm was observed. In some nuclei, the majority of nerve cells were in a state of severe chromatolysis, with the nucleus almost undetectable. Around such cells, more glial elements than usual

accumulated, and pictures of neuronophagia were observed. Among Purkinje cells of the cerebellum, neurons in a state of almost complete chromatolysis were noticeable, as well as shrunken nerve cells that had lost their usual pear-shaped form and were totally stained with thionine. Phenomena of dropout of entire groups of Purkinje cells were encountered. In the studied subacute form of intoxication, a reaction of neuroglia was also noted. Mainly solitary fibrous, pale-staining astrocytes appeared. These had a small number of processes, sometimes undergoing fragmentation. Single microglial cells were found in the white matter of the cerebellum and near the basal nuclei of the brain. They weakly took up the stain, and their processes lacked secondary branching. Near the stroma of the choroid plexus, coarse, hyperimpregnated microglial cells appeared. Very rarely, small microglial nodules were encountered.

Conclusion. Thus, in subacute poisoning of the body with MVP, severe vascular disorders and dystrophic changes in the neuroglial apparatus occur. With the diffuse nature of these changes, their greatest degree was noted in the cerebral cortex, thalamo-hypothalamic region, and brainstem. The circulatory disturbances observed by us during MVP intoxication undoubtedly contribute to the spread and aggravation of pathological changes in neurons and glia. In nerve cells, phenomena of chromatolysis, vacuolization, appearance of shrunken hyperchromic neurons, as well as ghost cells were noted. Parallel to the neurons, changes also occurred in the glia. The observed fact of combined changes in neurons and glia during subacute MVP poisoning indicates their physiological unity. Significant damage to the glial apparatus during poisoning undoubtedly hinders the development of compensatory protective reactions on the part of neurons.

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