Морфофункциональные изменения в некоторых панкреатомозных органах при экзо- и эндотоксикозе
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Резюме
Под воздействием хлорида кадмия в клетках печени происходят нарушения мембранотропного характера. При этом нарушаются, в первую очередь, аппараты синтеза белков и выработки энергии, а также развиваются явления энергетического дисбаланса. В лимфатических узлах печени развиваются отек капсулы и паренхимы, дистрофические и цитологические изменения, что нарушает детоксикационную функцию лимфоузлов.

Ключевые слова: печень, лимфатические узлы, клетка, капсула, паренхима, поджелудочная железа, почка.

Эксо-жем эндотоксикоз кезінде кейіп берілген ақпаларда дамитын морфофункционалдық өзгерістер Жаксылыкова А.К., Ткаченко Н.Л.

Түйін
Хлорлы кадмиймен созылмалы улануда бауыр төршаларында жарықтай өзгерістер дамыды. Оларда акуыз құрасының және энергияның өндіру құшметтері нашарлады. Онымдар қатар, энергия өндіретін құралымдардың құрамынан үйледі. Бауырдың лимфа түйіндерінде түйін қапшығы мен ұлпасы ісініп, үлұда дистрофиялық және цитологиялық өзгерістер дамыды. Сондықтан лимфа түйінінің детоксикациялық құшметі нашарлады.

Түйінді сөздер: бауыр, лима түйіндері, торша, капшық, ұлпа, ұйқы безі, бүйірек.

Introduction.
Salt of heavy metals take a lot role of bad ecology and form unfavorable ecological situation [1]. Cadmium - is the most dangerous chemical element from all heavy metals. It is accumulation in organism of human and it is not go out from body. [2,3]. Liver and kidney is the central organs of our homeostasis. They are very sensitivity to different exo- and endotoxic agents [4,5]. Toxic injury of liver accompany with permeabilized hepar cells, activation of peroxide oxidation of lipids, development of endotoxicosis and dysfunction of liver [6,7]. Chemical pollution of the environment is a factor, which make to injury of the liver.

Material and method of investigation.
Morphological and functional changes of liver and its regional glands under chronic exotoxicosis caused by cadmium chloride have been studied in 130 male rats. Experimental model of poisoning was based on two groups of white rats, for whom every day, over 2.5 months in the standard diet cadmium chloride added at dose of 1.5 mg and 3 mg / kg. Animals were decapitated through 1,7,14,21 night, after the end of the experiment.

Findings of investigation.
Chronic cadmium intoxication resulted in dystrophic change of hepatocytes in the kind of cytoplasm vacuolization, cell hypostasis and destruction as well as disorder of blood microcirculation (picture 1).

Destruction diesis in rat's hepatocytes. First day after beginning of the chronic cadmium chloride exotoxicosis at doses of 1.5 mg/kg. Colouring blue toluidine. Magnification ×600. Picture 1.

Besides, disorder in the structure of cell nucleus, cell organelle, cell interfaces, spatial configuration of microfibres of exchange poles and gall capillaries were registered. Signs of albumen synthesis disorder were registered in hepatocyte cytoplasm: widened spaces without chromosomes were found in channels of granular endoplasm reticulum. The number of ribosomes and poliribosomes was decreased. Signs of cell power supply insufficiency supported by change of mitochondria density and reduction of crista were found (picture 2).
Dilatation and fragmentation of GER canals, swelling of mitochondrions, redaction crists, ribosome and glycogen in hepatocyte of rats. First day after beginning of the chronic cadmium chloride exotoxicosis at doses of 3,0 g/kg


These changes were accompanied by reduction of glycogen level and accumulation of excess of lipid inclusions. Chronic exotoxicosis exited development of hypostasis of regional liver nodes, reduction of sinus areas, increase of cortical and medullar substance volume. Due to hypostasis of node parenchyma and decrease of sinus gaps drainage function of the nodes was lower and the lymph coming through the node had close contact with the parenchyma of the nodes. Areas of secondary lymphoid nodes were increased reliably. Cell changes in all examined zones of liver lymphatic nodes were based on decrease of small and medium lymphocyte number and reliable increase of big lymphocyte number. This is the indicator of development of immune-morphological proliferative reaction in the lymphoid tissue as the reaction to toxic substance impact. In medullar tension bars and secondary lymphoid nodes increase of the number of plasma-blasts, plasmocytes, macrophages, Mot cells, degenerated cells were registered (picture 3).

Redaction plasmoblasts, plasmocytes, macrophages, and mast cells in brain’s band of rat’s lymphatic knot in hepatic, which took tagan-sorbent. First day after beginning of the chronic cadmium chloride exotoxicosis at doses of 3,0 g/kg Colouring azur II-eosin. Magnification ×1000. Picture 3.

Thus, after 7 days after the poisoning, the positive developments across the nodes were reviled, in contrast to untreated animals. By the 21st day of observation the areas of all zones approached to a control values (picture 4).

Redaction small and middle lymphocytes in parenchyma of brain’s band
of rat’s lymphatic knot in hepatic, which took tagan-sorbet. 21 day after beginning of the chronic cadmium chloride exotoxicosis at doses of 1.5 g/kg. Colouring blue toluidine. Magnification ×600. Picture 4.

Pancreatitis in experimental conditions was reproduced in 30 dogs by injection of autogall in the pancreas duct. Animals dye within 24 hours without treatment. Main reasons of kidney affection during acute pancreatitis are: reduction of blood supply due to arterial hypotension and hypovolamia, toxic action of circulating pancreas enzyme, vasoactive agents and tissue albumin decomposition products to kidney parenchyma. During severe form of acute pancreatitis loss of 30% of circulating plasma is possible within several hours [8]. Hypostasis of paranephral tissue, multiple hemorrhages into the capsule were developing. Intervascular changes, massive glomerulo-thrombosis, unclear gap in the capillary, suppressed with hydropic liquid vascular glomerule prevailed. Aggregation of regular elements in venules was observed. Epithelium necrosis foci were found in tubulas adjacent to glomerula. Tubular epithelium in kidney medullar layer contained vacuoles of various size filled with cytoplasmic liquid. Sometimes the vacuole occupied the whole cell pushing the nucleus to periphery. In cortical layer many peritubular capillaries were empty and with unclear borders, in some places capillary blood flow was completely stopped. Plethora of juxtamedullar glomerules and direct vessels of medullar kidney layer was clearly seen. Changes in kidney tubulas were registered in the kind of epithelium dystrophy and necrosis (picture 5).

Dystrophic change of parenchyma: necrosis renal tubules.
Redistribution of inter-kidney blood flow occurred with apparent ischemia of kidney cortex. Most evident changes were registered in venular section that perform drainage and deposit function (picture 6).

Mass glomerulo-thrombosis and multiple hemorrhage in the capsule were registered in kidneys. Magnification×600. Picture 6.
In condition of developing plasmorrhagia, hemorrhagia and increased re-absorption venules with thin compensatory widened wall earlier than the other units reached the stage of decompensation with micro-aneurism and thrombus.

Summary
1. There are some disorders under the influence of cadmium chloride in hepatic cells - membranotrop, protein synthesis, energy production, disorder of albumen synthesis.
2. Change in regional node lymphatic elapse synchronously that change in liver.
3. Morphofunctional disorders in liver and in lymph nodes dose-related: reaction to toxic substance more, than more doses, recovery is slowly to.
4. Changes in kidney are nonspecific - fibrinoid necrosis and gialnosis of glomerulus capillary, necrosis renal tubules. Aggregation of regular elements in venules was observed.

Literature