MECHANISM OF THE HEPATITIS C WITH GLOMERULONEPHRITIS AND CANCEROGENESIS

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ABSTRACT — The work presents morphological characteristic pathogenic structure changes in the kidneys, lungs and liver in patients with hepatitis c, including HIV-infected. Showing pathogenic mechanisms of hepatitis c associated with violation of the metabolism of hemoglobin, anoxic link revealed as well as apoptosis hepatocytes and cellular components of hem tissues barriers liver, lung and kidneys with Glomerulonephritis, cirrhosis and carcinogenesis in patients with hepatitis c, including HIV-infected.

KEYWORDS — ischemia, hepatitis, glomerulonephritis, carcinogenesis after low, apoptosis, cirrhosis, HIV infection, pneumonia, blastocysts, blood tissues barriers.

RELEVANCE

There is still a point of view that different pathogens are associated with increased risk of cancer.

However, the main culprits among the infectious causes of cancer, both for human and animal viruses that are associated with the 20% of cancers. Very strict criteria used to identify their oncogenic properties made it possible to classify as only six human oncogenic viruses.

At the same time Rodríguez-Nóvoa S, Morelloj, González M, et al. (2008) noted that the use of antivirals for HIV infection causes hemolysis and increases hyper bilirubin anemia. It has been proven that the majority of patients with hepatitis c, including people with HIV receiving antiretroviral therapy, develops resistant anemia, decreased intake of toxic drugs.

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It is known that anemia at the present stage is not an exhaustive justification pathophysiology as disclosure mechanisms reduce the hemoglobin in the blood of patients with hepatitis c and HIV infections. This assumes that the anemia has multifactor nature that explains cases of failed attempts of empirical application of erythropoietin in treatment of patients with hepatitis c and HIV infections.

Given the poor prognosis, study of mechanisms of development of renal diseases, cirrhosis and liver carcinogenesis on the background of hepatitis c, including HIV-infected in relation to anemia and anoxia is at the present stage, the most important.

MATERIAL AND METHODS

In the work material of the liver, kidneys and lungs of patients with hepatitis c, including HIV-infected was used. The age of patients was from 30-38 years. The monitoring group comprised 14 patients who died as a result of injuries that are incompatible with life, presumably without somatic pathology, ranging in age from 24 to 76 years. Biopsy is a multistage material tests on samples of writing in preparation for histological research of immediately after sampling.

Used classic histological research methods with hematoxylin-eosin staining to obtain total morphological pattern. Material analysis was carried out using a microscope Olympus-Bx82 and CDh82 digital camera with proprietary software.

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THE RESULTS OF THEIR OBSERVATIONS AND DISCUSS THEM

Found that in the liver of a patient with HIV, infected by hepatitis c virus in blood outflow system identified by macrophages containing cytoplasm phagocytes pigment Brown in color, with color pigment in cytoplasm of phagocytes or inside blood vessels varies from reddish to dark-brown. The endothelium of blood vessels can be saved partially, while misplaced, with cores arranged perpendicular to the basal membrane, but in most parts of the surface vessel destroyed. Against the backdrop of diffusely located brown pigment are identified by macrophages with dark inclusions in the cytoplasm.

In the liver parenchyma of the morphological picture of acute hepatitis with leukocyte infiltration expressed inter lobules tissue enlargement of the bile duct, apoptosis of hepatocytes, fatty liver dystrophy, and cirrhosis.

When microscopies are detected by the bodies Councilman, which some authors consider cluster star reticule endothelial cells, lymphocytes, macrophages, and neutrophils in necrotic masses formed in viral hepatitis in the liver resulting in apoptosis of hepatocytes, others microscopic homogenized eosinophil cells, detectable in around capillaries spaces with liver necrosis of hepatocytes, occurring as a result of apoptosis in different diseases.

In our studies in patients with HIV and hepatitis c process violations in the filtration barrier kidney associated with destruction of erythrocytes and the release of hemoglobin into the parenchyma of kidney, his capture by macrophages with subsequent evacuations across basils membrane barrier route. In this part of the parenchyma of kidney is characterized by flushing the system renal vascular glomeruli's, another part of the renal corpuscles is the picture of necrosis, and a large part of the kidney parenchyma presented kidney cells with signs of irregularities in the system of urine and urine and blood barrier, glomerulonephritis and extensions in which arteries filled with pigment and macrophages with phagocytes pigment inclusions in the cytoplasm.

We found that in the cortical parenchyma and Medulla of kidney vessels hyperemic. In vascular glomeruli's bringing arterioles and capillaries network expanded, taking away arterioles in extended gleams observed hemolysis cells, capture of iron by macrophages, as well as the presence of pigment in the proximal tubules. The presence of characteristic signs in morphological picture kidney damage manifested hyperemia, higher, the destruction of red blood cells, diffusion of transferrin and its capture by macrophages, in our opinion, can testify to the same mechanism of pathogenesis of renal and hepatic pathology of HIV and hepatitis C.

In the lungs of patients with HIV infection and hepatitis c amid pneumonia deaths is observed blastocysts advanced alveolar epithelium, hyperemia of the blood vessels, lung tissue infiltration blastocysts detection in the blood vessel lumen diffusely scattered pigment and macrophages with cytoplasm filled with dark pigment, alveoli or necrotic collapsed. The cartilage fragments revealed not only the process of apoptosis cartilage cells, but also pockets necrosis. In the parenchyma of the lung blastocysts are extended in the gleam of the alveoli, bronchi and lung gleams moves. At the same time in the lumen of blood vessels are identified by macrophages, cytoplasm which is filled with transferrin.

DISCUSSION OF THE RESULTS

We revealed an identical morphological picture of pathological changes in liver, kidney and lungs of patients with hepatitis c and hepatitis c HIV infection associated with the destruction of hemoglobin, erythrocyte hemolysis and advanced in the process of cellular anoxia.

Besides the already known and submitted by many authors for signs of pathological changes in organs associated with hyperemia parenchyma, apoptosis and degeneration, fibrosis and cirrhosis with local necrotic changes leukocyte infiltration, found all bodies of brown pigment accumulation in the system of blood outflow. Histological study of Renal Biopsy usually indicates the presence of infiltration glomerulus activated macrophages. When the macrophages loaded with pigment black, morphologically corresponding protein complex with iron-transferrin.

We assume that the dark pigment accumulation in the cytoplasm of macrophages corresponds to transferrin, a product of the metabolism of erythrocytes and hemoglobin destroyed hemolysis. This is indirectly confirmed by numerous studies have investigated the reduced hemoglobin in the peripheral blood of patients with hepatitis c and HIV infected but not shown mechanism of anemia.

In our opinion, the pathological process in the liver, lung, and kidney infection HIV infected hepatitis c virus begins with hypoxia, caused by an oxygen explosion, necessary for the induction of phagocytosis of pathogens, endings and then apoptotic and necrotic cells due to Anoxia associated with aggressive destruction of erythrocytes and the release of hemoglobin in the plasma of the blood vessels of the lungs, liver and kidneys. This process results in the release of transferrin with the subsequent seizure of his by macrophages. Because of the impossibility to transfer oxygen to the

tissues, as well as not fulfilling the second function of hemoglobin-the transfer of carbon dioxide, cell bodies were forced to switch to the use of free dissolved oxygen in plasma. With the arrival of hemoglobin in the blood, taking into account its high toxicity when intra vascular dissolution and released into the blood plasma, begins massive cell death due to ischemia/anoxic and intoxication.

The second link exposure to pathogenic virus hepatitis with vasa so that stem cells and early cells-predecessors of erythropoiesis in norm obey near regulation provided by their interaction with neighboring cells and stem bloods cells of the bone marrow stroma.

Anemia associated with adverse prognosis in chronic form of hepatitis C, HIV infection is marked by many authors. Found in the liver pronator erythropoietin submitted erythrogensin, together with erythrocytes poètinogen buds produced prenatally Kupfer cells , respectively, and kidney podocytes. When cerebral alveolar epithelium, hepatocytes and the UUT and, as a consequence, their loss, declining elaboration of all constituent of erythropoietin, Mrr erythropoiesis and regeneration, controlling the breeding of metabolites. Anoxia leads to a decrease in energy processes cells, apoptosis, necrosis and development of cirrhosis of the liver, glomerulonephritis. It can be assumed that this is the same mechanism of brain neuronal damage in hepatitis C, as there is evidence of hepatitis c virus detection in the endothelium of blood vessels in the brain.

A key factor in the mechanisms of systemic damage to the walls of blood vessels, including the participants of blood tissues barriers may be phagocytes transferrin with aggressively destroying macrophages endothelium to release into the blood stream. Regular measurements of hemoglobin of the blood of patients with hepatitis, especially HIV infected, could help identify which patients are at greatest risk of HIV disease progression to conduct therapeutic intervention.

Particularly high risk for the development of processes such as cirrhosis, Glomerulonephritis and carcinogenesis in patients with hepatitis c and HIV infected in the absence of a pathogenically justified the treatment and understanding of mechanisms of pathogenesis, which leads to unwanted complications.

Thus, there is a need to develop a randomized trials of high methodological quality assessment for strategic effects on anemia in persons infected with hepatitis c virus and human immunodeficiency.

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