POSTMORTEM PATHOLOGICAL FEATURES IN LIVER TISSUE OF HIV PATIENTS

M.E. Tuchina[1], G.V. Reva[1], I.V. Reva[2], V.G. Kozhuhar[3], R.A. Nasyrov[3]

[1] Department of Clinical and Fundamental Medicine of the School of Biomedicine, Far Eastern Federal University, Vladivostok, Russia, e-mail: mrasskazova@mail.ru;
[2] International Medical Research Center (IMERC), Niigata, Japan, e-mail: avers2@yandex.ru;
[3] St.Petersburg State Pediatric Medical University, St.Petersburg, Russia; e-mail: v.kojukhar@ yandex.ru

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ABSTRACT — Study of morbid anatomy material from dead patients suffering from HIV-related illnesses, including hepatitis C, provided an opportunity to identify substantial pathological changes in the structural elements liver that suggested other pathogenetic mechanism of development changes in patients with HIV and hepatitis C associated with impaired metabolism in erythrocytes that are collapsing, hemoglobin in the plasma of blood blood vessels of the liver. As a result of the destruction of erythrocytes, free, not associated with erythrocytes, hemoglobin cannot carry carbon dioxide from cells, hypoxia ensues the structural elements of the liver and cells are forced to use the free dissolved in plasma oxygen, which further exacerbates the occurrence of hypoxia and Anoxia and then the appearance of intoxication of the massive destruction of hemoglobin and the advent of plasma transferrin. The last captured by macrophages. The free hemoglobin in the bloodstream increases its toxic effect on tissue cells, causing cell death in the resultant ischemia, thus worsening the oxygen supply of them. As a result of the subsequent destruction of haemoglobin are formed its decay products in the form of iron porphyrin, bilirubin, The latter contributed to the development of jaundice or acute porfirii owing to the death of hepatocytes, which manifest is to develop cirrhosis or cancer.

KEYWORDS — HIV, hepatitis c, ischemia, cirrhosis, cancer.

RELEVANCE

It is known that HIV-associated AIDS is one of the frequent causes of mortality in young age [1.2]. Many authors consider hepatitis c HIV infection and chronic hepatitis, precipitating factor in the defeat of the structures of the liver and in the subsequent development of cirrhosis [3.4]. According to Rodríguez-NóvoaS, MorelloJ, González M, et al. (2008), the use of antiviral drugs causes hemolysis and increases bilirubine [5, 6, 7]. This group of scientists conducted a study on the treatment of HIV/hepatitis c-infected patients showed increased bilirubine with 9% to 45% after the start of treatment of HIV-infected patients with hepatitis c. overall, according to many authors, efficiency standard antiviral treatment does not exceed 50–80%. However, despite the emergence of large quantities of drugs for the treatment of HIV infection, pathogenesis of HIV infection are not fully disclosed. Conducted on atherosclerosis plaques assays materials perished from HIV while also not given complete replies to the nature of the investigated processes occurring in the body, HIV-infected, leading to death. The majority of patients with hepatitis c, including HIV-infected, develops resistant anemia, decreased intake of toxic antiretroviral drugs [8]. 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. It is expected that the anemia has multifactor nature, which explains the cases of failed attempts of empirical application of erythropoietin in treatment of patients with hepatitis c and HIV infections [9, 10, 11].

Therefore, the study of the pathological features of HIV infection should be considered relevant because it is necessary to address the issues of early Diagnostics, treatment, rehabilitation, taking into account the early accession opportunistic infections, leading to the defeat of various organs and systems of the patient, emerging when the pathology study and the emergence of new data on pathogenesis mechanisms in the development of HIV/AIDS infection.

MATERIAL AND METHODS

The materials used autopsy 9 HIV-infected children from 3 months. up to 12 years and 35 adult patients from 30 to 38 years, deaths from HIV infection and opportunistic diseases, obtained in accordance with the regulations of the Helsinki Declaration (2000) and with the permission of the Ethics Committee of the Federal autonomous State educational institution of higher education, Ministry of science and higher education of the Russian Federation far eastern Federal University ". The monitoring group amounted to avoided material 17 patients who died as a result of

injuries that are incompatible with life, ranging in age from 12 to 76 years (Table 1).

Coloring samples of liver tissue produced in hematoxylin-eosin, and microbiology characteristic structural elements were evaluated at liver histological sections as you increase lens ×200, ×400. In doing so, made Microscope Olympus-Bx82 and Cdh digital camera with proprietary 82 IT software.

presence of brown pigment in the system of blood outflow.

We found that the dark pigment accumulation in the cytoplasm of macrophages corresponds to transferrin, product exchange lysed RBCs and hemoglobin destroyed. At the same time found that pathological process in the structural elements of the liver infection hepatitis c virus (HCV) infected with HIV starts with

Table 1. *Material distribution*

#	Age groups	Control	Pathology			Material
			Hepatitis C	Hepatitis C+ HIV infection	HIV infection	The liver, kidneys, lungs
1	3 months–12 years	3		3	6	9
2	30–33 year	3	7	4	3	14
3	34–35 year	3	3	3	3	9
4	36–38 year	8	3	5	4	12
TOTAL		17	13	15	16	44

RESULTS AND DISCUSSION

The result of the study material to avoided material liver was reveling in the liver parenchyma of the morphological picture of acute hepatitis c with explicit leukocyte infiltration between lobule fabric, the presence of biliary extension ducts, determination of apoptosis of hepatocytes, fatty liver, cirrhosis, necrosis (fig. 1, 2, 3, 4, 5).

In the lumen of blood vessels are identified by macrophages, whose cytoplasm is filled with transferrin (fig. 6).

We believe that the pathological changes in the liver of patients with HIV infection associated with hepatitis c and pathology study material related to erythrocyte hemolysis, the destruction of hemoglobin and the subsequent emergence of processes cellular Anoxia. Moreover, we found that some patients show signs of acute hepatitis c is characterized by the typical picture with around between lobules and infiltration zone tetrads, but saving the structure of the hepatic lobules; other patients identified the transition of acute hepatitis in chronic, with signs of the formation of false hepatic lobules, increased outflow of bile system, apoptosis of hepatocytes, antitheses of common perceptions about pathogenic changes in structures.

Based on our research, found that besides the well-known symptoms of pathological changes in organs, submitted by many authors (hyperemia parenchyma, apoptosis and degeneration, fibrosis, cirrhosis and later joining local necrotic changes with leukocyte infiltration) we have identified all the bodies of the hypoxia. The latter, as a result of subsequent manifestations of intoxication, leading to apoptosis, and necrosis of cells due to Anoxia due to aggressive destruction of erythrocytes and the release of free plasma hemoglobin in the blood vessels of the liver, which is even more adding to the ischemia due to its toxicity, thereby worsening the oxygen supply to the tissues of the liver.

Collapsing, hemoglobin allocates degradation products in the form of a porphyrin, bilirubin, iron, transferrin, which invade macrophages.

In ischemia of hepatocytes and, as a consequence, their deaths, declining elaboration of all constituent of erythropoietin, disturbed Mrr erythropoiesis and regeneration, control over the breeding of metabolites. This is indirectly confirmed by numerous studies have referred to lower hemoglobin in the peripheral blood of patients with hepatitis c and HIV-positive, but it does not explain mechanism of anemia.

Particularly high risk for the development of processes such as cirrhosis of the liver in patients with hepatitis c and HIV-infected in the absence of a pathogenetically justified treating anaemia, and understanding of mechanisms of pathogenesis, which is what leads to undesirable complications, so 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.

Thus, we believe that a key factor in the mechanisms of systemic damage to the walls of blood vessels, including the participants of hematological and tissue

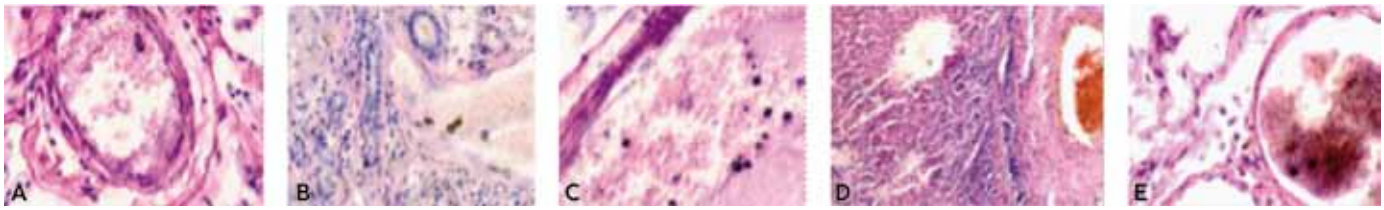

Fig. 1. Human Liver — a, b, c, d) with a pigment in cytoplasm of macrophages in the lumen of the vessels; e) pigment in the cytoplasm of macrophages and diffusely located in the lumen of the vessel. Coloring g/e. Mikrofoto. HCS. ×200

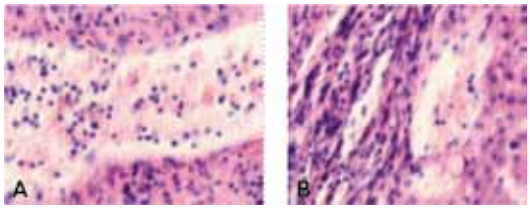

Fig. 2. Liver Parenchyma in patients with hepatitis c in HIV-infected. Psevdodolka liver — a, b) leukocytes in pronouncing the lumen of the vessel. Coloring g/e. Mikrofoto. HCS. ×200

of cellular apparatus, apoptosis, necrosis of hepatocytes, loss and development of cirrhosis of the liver and other organs of carcinogenesis.

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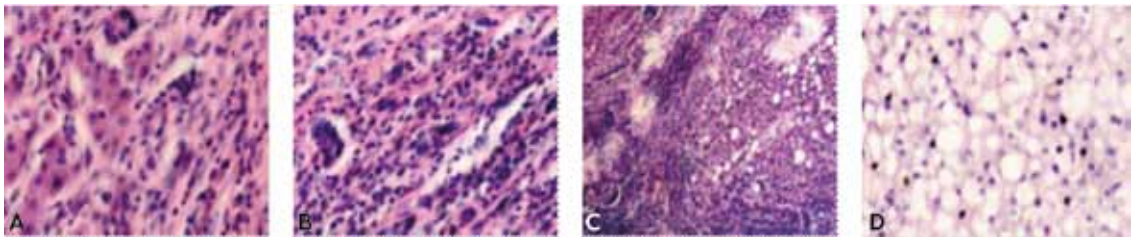

Fig. 3. Liver Parenchyma in patients with hepatitis C in HIV-infected. Psevdodolka liver — a, b) infiltration mezhdolkovyh structures: c, d) steatosis/ Coloring g/e. Mikrofoto. HCS. ×200

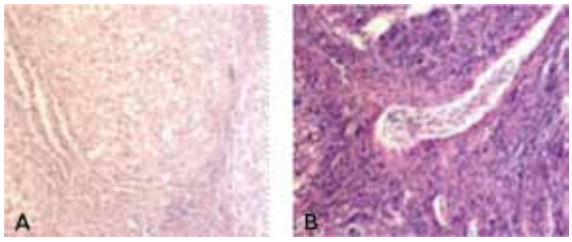

Fig. 4. Liver Parenchyma in patients with hepatitis c in HIV-infected. Psevdodolka liver — a, b) cirrhosis. Coloring g/e. Mikrofoto. HCS. ×200

barriers may be macrophages during phagocytosis of transferrin, aggressively destructive to endothelium release into the blood stream. Due to the fact that ruined hemoglobin enters the blood, as in the unassociated state very toxic, begins a massive cell death due to ischemia/anoxic and intoxication. Hypoxia and anoksija, in turn, lead to a decrease in energy processes

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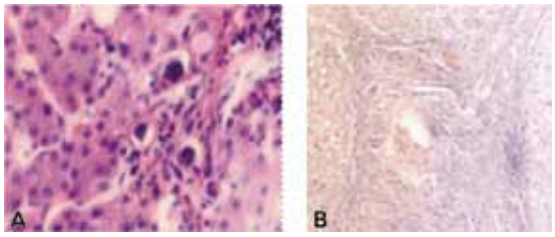

Fig. 5. *Liver Parenchyma in patients with hepatitis c in HIV-infected. Psevdodolka liver — a) calf Councilman; b) cirrhosis and necrosis of the liver parenchyma. There is no central Vienna. Apoptosis. Coloring g/e. Mikrofoto. HCS. a) ×200; b) ×400*

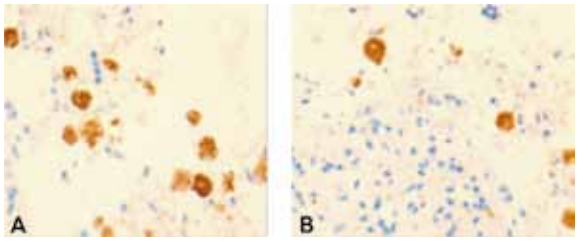

Fig. 6. *Macrophages with transferrin in the lumen of blood vessels of the liver with hepatitis HIV-infection. Mikrofoto. Coloring g/e. SW. ×400*

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