

9. Kostylev A.N., Derin M.A., Kalinin S.A., Mayorov A.Yu. Complex approach to treatment of lumbosacral pain syndromes.//Siberian international neurosurgical forum: thesis report. – Novosibirsk, 2012. – P. 141.
10. Kozyrev S.V. The capabilities of magnetic resonance imaging in complex diagnosis of postoperative changes in microdiscectomy of spine lumbar division: dis. of candidate. med. sciences. – SPb., 2011. – 89 p.
11. Kuschaev G.B. Prevention of scar-adhesive epiduritis (epidural fibrosis) in the lumbar microdiscectomy.//S.V. Kudaev, E. G. Pedachenko//Book of abstracts III Congress of neurosurgeons of Russia. – SPb., 2002. – P. 262–263.
12. Nuraliev H.A. The Preventive maintenance of scar-adhesive epiduritis in surgical treatment of lumbar division of the spine osteochondrosis.//Traumatology and orthopedics of Russia. – 2009. – № 1 (51) – P. 32–34.
13. Sagun A.E., Lyubishev I.S. Prevention of postoperative scar adhesions in surgery of hernias.//all-Russian scientific-practical conf. “Polenov’s readings”: thesis report. – SPb., 2012. – P. 167.
14. Toptygin S.V., Parfenov V.E., Scherbuk Yu. A. The use of differentiated microsurgical approaches and intraoperative endovideomonitoring to prevent recurrence of lumbosacral radiculitis.//all-Russian. scientific-practical conf. “ Polenov’s readings”: thesis report. – SPb., 2002. – P. 287–288.

*Sarsekeyeva Nazgul Yesentaevna,
Karaganda state medical university,
doctoral student specialty 6D110100 Medicine
E-mail: sarsekeeva.04@mail.ru*

Liver fibrosis of patients with chronic viral hepatitis B+C

Abstract: The article deals with the problem of combined chronic viral hepatitis B and C. The paper presents the results of clinical and laboratory studies of patients with chronic viral hepatitis B+C. The author analyzes the results of liver elastomers in patients with chronic viral hepatitis B+C.

Keywords: liver fibrosis, liver cirrhosis.

At the present stage viral hepatitis remains one of the most important problems of infectious pathology and cause great damage for public health and economic development of our country. In the structure of hepatitis B viral hepatitis with parenteral transmission mechanism, namely, viral hepatitis B and C occupy a leading position.

Today there is no common theory of the pathogenesis of chronic viral hepatitis. Processes such as chronic inflammation, including the inflammatory repair as a mandatory component and the process of tissue repair in response to liver damage are opposed unreasonably. Some researchers have linked the progress of chronic viral hepatitis with the development of an immune inflammation; other authors consider the formation of liver cirrhosis as the result of stroma repair breach and the hepatocytes in the portal tracts and hepatic parenchyma damaged areas [1–4].

In the base of pathogenesis of inflammatory and fibrotic changes in the liver in chronic hepatitis B and C, leading to poor outcomes for these infections there are complex interactions between the virus and the host immune system, and any damage of the liver tissue under these infections is immune-mediated. However, most modern scholars focus on the study of the immune response specific mechanisms in the immunopathogenesis of chronic viral liver disease, which naturally appears as insufficient studied the role of the innate immune factors and has the actual aspect of the problem decision at all [4; 5].

Chronic viral hepatitis B often develops in patients with progressive course of acute hepatitis B, combined with discordantly

high replicative activity of HBV, and rarely with primary chronicity of HBV-infection. The chronic form of viral hepatitis in its turn may cause the changing of liver tissue to fibrotic, liver cirrhosis, and also hepatocellular carcinoma of liver [6–8].

In comparison with other viral pathogens of serum hepatitis, HCV has the highest chronic potential. Patients with hepatitis C, especially chronic ones and chronic latent patients of HCV, are the source of infection. Chronic hepatitis C is accompanied by a progressive increase in liver fibrosis that the outcome of the disease leads to cirrhosis — not reversible changes in liver at high risk of fatal complications, including primary liver cancer. Most patients with biochemical signs of chronic hepatitis C have a favorable course — mild or moderate inflammatory activity in the liver tissue and minimal fibrosis [9–11].

The main differences in the course of chronic hepatitis B and chronic hepatitis C are determined by the interaction of virus and the human immune system in the initial stages of hepatitis virus infection.

The main way of progression of chronic diffuse liver disease — is the development of successive stages of liver fibrosis with eventually formation of cirrhosis and liver cancer, which mainly determines bad life prognosis and short survival date of this patient category [1].

According to the results of morphological studies of the liver we can diagnose hepatitis of different stages of activity and on the basis of assessment of such factors as the severity of inflammation and fibrosis. The rate of liver fibrosis depends on the activity of the inflammatory process in the liver [12].

Liver fibrosis at an early stage is a protective reaction of the body, which is directed to maintaining the structure of the affected organ. The regeneration of the cells and the absorption of excess collagen tissue activate in this moment. However, due to chronic liver damage, these processes get broken, whereby the rate of tissue increasing exceeds the rate of its destruction. As a result liver architectonics gets disrupted [13].

Starting point for the beginning of liver fibrosis is damaged hepatocytes, and then stellate cells start getting activated. Activation indicates the beginning of the early stage of liver fibrosis. Fundamentally, the ability of noninvasive diagnosis of timely identification of the liver fibrosis early stages, which allow to assign the treatment quickly, and to prevent further progression of fibrosis, is important [14; 15].

Until today, the only way to assess liver fibrosis was to use the “gold standard” diagnosis of fibrosis — liver biopsy. However it is not always possible to use this invasive, expensive method with a high risk of complications. These disadvantages limit the phased conduction of liver biopsies to control the reaction to antiviral or anti-fibrotic therapy. This limits the biopsy, forcing to perform non-invasive diagnostic procedures [16; 17].

In recent years, methods of non-invasive diagnosis of liver fibrosis have been widely used. The benefits of modern methods of non-invasive diagnosis to liver biopsy include: simplicity, absence of contraindications, high diagnostic accuracy in various stages of liver fibrosis, the ability to assess the dynamics of fibrosis, usability screening method for patients at risk, and the use in the outpatient setting [18–20].

In chronic hepatitis B and C prognosis of illness and tactics of the patients are highly dependent on the severity of fibrotic process in the liver, which has an impact on the timing of antiviral therapy and dose of drugs; it is a criteria for the effectiveness of drug therapy; it determines the risk of adverse outcomes [7; 20].

The high prevalence of parenteral hepatitis B and C, the steady increase in the incidence of these infections, the tendency to poor outcomes of disease (cirrhosis, hepatocellular carcinoma) make this problem very relevant.

Aim of research: to study the clinical course in patients with chronic viral hepatitis B+C and assess the degree of liver fibrosis.

Materials and methods of research

We performed clinical and laboratory studies in 43 patients with chronic viral hepatitis B+C. The average age of the patients was 41 years.

It was found that among the examined patients by gender, males were more than females. Thus, among the total number of 43 individuals with chronic viral hepatitis B+C, were 31 men (72.1 %), women — 12 (27.9 %).

The diagnosis of chronic viral hepatitis B+C was verified after a detailed study of patient complaints, medical history, taking into account the epidemiological history, medical history, objective data.

The diagnosis of chronic hepatitis B+C was established based on determining the serum of patients with HBsAg, anti-HCV ELISA and detection of DNA, RNA in plasma by PCR.

All patients underwent clinical examination, biochemical blood analysis with determination of total bilirubin and its fractions, alanine aminotransferase (ALT), aspartate aminotransferase (AST). It was determined all patients had a viral load.

The degree of liver fibrosis was measured with Fibroscan apparatus on a scale METAVIR.

Analysis of laboratory data showed that chronic viral hepatitis B+C was accompanied by 21 (48.8 %) patients cholestatic, in 19 (44.2 %) — mesenchymal-inflammatory, and in 31 (72.1 %) — cytolytic syndrome, with this transaminase activity in 23 (74.2 %) patients had increased by 2 times in 8 (25.8 %) — 3 times (Table 1).

Table 1. – Laboratorial syndromes in patients with chronic viral hepatitis B+C

Laboratorial syndrome	N	%
Cholestatic syndrome	21	48.8
Mesenchymal-inflammatory syndrome	19	44.2
Cytolytic syndrome	31	72.1

11 (25.6 %) patients with chronic viral hepatitis B+C complained for a loss of appetite during the clinical examination. However, the most typical complaints were asthenic character (unmotivated weakness, fatigue). So, weakness troubled 27 (62.8 %) patients, 8 (18.6 %) patients noted fatigue. 17 (39.5 %) patients had non-persistent joint pain. In this case, 21 (48.8 %) patients revealed a small icterus of sclera and skin. 32 patients (74.4 %) had recurrent pain and heaviness in the right upper quadrant. The proportion of patients with hepatomegaly and splenomegaly was 28 (65.1 %) and 9 (20.9 %). It should be noted that several clinical manifestations of the disease at the same time were noted in patients (Table 2).

Table 2. – Results of clinical examination in patients with chronic viral hepatitis B+C

Clinical manifestations	N	%
Decreased appetite	11	25.6
General weakness	27	62.8
Fatigue	8	18.6
Arthralgia	17	39.5
Icterus of sclera and skin	21	48.8
Pain and heaviness in the right upper quadrant	32	74.4
Hepatomegaly	28	65.1
Splenomegaly	9	20.9

Note: – one patient had several clinical manifestations of the disease simultaneously.

According the elastomers of the liver in patients with chronic viral hepatitis B+C there were no signs of fibrosis (F0) in 4 patients (9.3 %), minimal fibrosis (F1) with stellate expansion of portal tracts without septa formation was observed in 7 patients (16.3 %), moderate fibrosis (F2) with the expansion of portal tracts with a single port-portal septa in 6 patients (14 %), fibrosis (F3) with numerous porto-central septa in 17 patients (39.5 %), cirrhosis (F4) in 9 patients (20.9 %) (Table 3).

Table 3. – Results of liver elastomers in patients with chronic viral hepatitis B+C

Liver fibrosis on a scale METAVIR	N	%
F0	4	9.3
F1	7	16.3
F2	6	14
F3	17	39.5
F4	9	20.9
Total	43	100

Analysis of the results of liver elastomers in patients with chronic viral hepatitis B+C showed that the proportion of patients with advanced fibrosis (F3, F4) was 60.5 %.

Thus, the study of the peculiarities of chronic viral hepatitis B+C showed that patients did not have bright symptoms of hepatitis and a moderate level of activity of the infectious process was observed. However, patients with chronic viral hepatitis infected with two viruses B and C at the same time have a greater risk of severe fibrosis and cirrhosis.

References:

1. Van der Meer A. J., Wedemeyer H., Feld J. J., Dufour J. F., Zeuzem S., Hansen B. E., Janssen H. L. Life expectancy in patients with chronic HCV infection and cirrhosis compared with a general population. *JAMA*. – 2014, Nov. 12. – 312 (18): 1927–8.
2. Lok A. S. F., McMahon B. J. AASLD Practice Guidelines Chronic Hepatitis B. // *Hepatology*. – 2007. – 2 (45).
3. Alberti A., Pontisso P., Chemello L. et al. The interaction between hepatitis B virus and hepatitis C virus in acute and chronic liver disease. // *J. Hepatol.* – 1995. – 22: 38–41.
4. Chisari F. V., Ferrari C. Hepatitis B virus immunopathogenesis. // *Immunol.* – 1995. – 13: 29–60.
5. Ferraioli G., Tinelli C., Dal Bello B., Zicchetti M., Filice G., Filice C. Liver Fibrosis Study Group. Accuracy of real-time shear wave elastography for assessing liver fibrosis in chronic hepatitis C: a pilot study. // *Hepatology*. – 2012, Dec. – 56 (6): 2125–33.
6. Xiao G., Yang J., Yan L. Comparison of diagnostic accuracy of aspartate aminotransferase to platelet ratio index and fibrosis-4 index for detecting liver fibrosis in adult patients with chronic hepatitis B virus infection: a systemic review and meta-analysis. // *Hepatology*. – 2015, Jan. – 61 (1): 292–302.
7. Mena Á., Pedreira J. D., Castro Á., López S., Vázquez P., Poveda E. Metabolic syndrome association with fibrosis development in chronic hepatitis B virus inactive carriers. // *J Gastroenterol Hepatol.* – 2014, Jan. – 29 (1): 173–8.
8. Lok A. S. F., McMahon B. J. Chronic Hepatitis B. // *Hepatology*. – 2001. – 6 (34): 1225–1241.
9. Ziol M., Handra-Luca A., Kettaneh A. et al. Non-invasive assessment of liver fibrosis by stiffness measurement: a prospective multicentre study in patients with chronic hepatitis C. // *Hepatology*. – 2005. – 1 (41): 48–54.
10. Sands C. J., Guha I. N., Kyriakides M., Wright M., Beckonert O., Holmes E., Rosenberg W. M., Coen M. Metabolic phenotyping for enhanced mechanistic stratification of chronic hepatitis C-induced liver fibrosis. // *Am J Gastroenterol.* – 2015, Jan. – 110 (1): 159–69.
11. D'Ambrosio R., Aghemo A., Fraquelli M., Rumi M. G., Donato M. F., Paradis V., Bedossa P., Colombo M. The diagnostic accuracy of Fibroscan for cirrhosis is influenced by liver morphometry in HCV patients with a sustained virological response. // *J Hepatol.* – 2013, Aug. – 59 (2): 251–6.
12. Blanc J. F., Bioulac-Sage P., Balabaud C., Desmouliere A. Investigation of liver fibrosis in clinical practice. // *Hepatol. Res* – 2005. – 27.
13. Bataller R., Brenner D. A. Liver fibrosis. // *J. Clin. Invest.* – 2005. – 115: 209–218.
14. Ferard G., Piton A., Messous D., Imbert-Bismut F., Frairi A., Poynard T., Lessinger J. M. Intermethod calibration of alanine aminotransferase (ALT) and gammaglutamyltransferase (GGT) results: application to FibroTest and ActiTest scores. // *Clin. Chem. Lab. Med.* – 2006. – 44: 400–406.
15. Castera L., Foucher J., Bertet J., Couzigou P. FibroScan and FibroTest to assess fibrosis in HCV with normal aminotransferases. // *Hepatology*. – 2006. – 2 (43): 373–374.
16. Castera L., Vergniol J., Foucher J., Le Bail B., Chanteloup E., Haser M., Darriet M., Couzigou P., Ledinghen V. de. Prospective comparison of transient elastography, Fibrotest, APRI and liver biopsy for the assessment of fibrosis in chronic hepatitis C. // *Gastroenterology*. – 2005. – 128: 343–350.
17. Beaugrand M. Liver stiffness measurement: new tool to assess liver fibrosis. EASL Single Topic Conference on The role of liver biopsy in diagnosis and management of chronic liver disease. – Torino, Italy, June 14–15, 2004.
18. Mendoza J., Gomez-Dominguez E., Moreno-Otero R. Transient elastography (Fibroscan), a new non-invasive method to evaluate hepatic fibrosis. // *Medicina Clinica*. – 2006. – 6 (126): 220–222.
19. Su L. N., Guo S. L., Li B. X., Yang P. Diagnostic value of magnetic resonance elastography for detecting and staging of hepatic fibrosis: a meta-analysis. // *Clin Radiol.* – 2014, Dec. – 69 (12): 545–52.
20. Calvaruso V., Bronte F., Conte E., Simone F., Craxi A., Di Marco V. Modified spleen stiffness measurement by transient elastography is associated with presence of large oesophageal varices in patients with compensated hepatitis C virus cirrhosis. // *J Viral Hepat.* – 2013, Dec. – 20 (12): 867–74.