NOVATEUR PUBLICATIONS
INTERNATIONAL JOURNAL OF INNOVATIONS IN ENGINEERING RESEARCH AND TECHNOLOGY
[IJIERT] ISSN: 2394-3696 Website: ijiert.org
VOLUME 8, ISSUE 4, Apr.-2021

FEATURES OF HEART DAMAGE IN PATIENTS WITH VIRAL CIRRHOSIS OF THE LIVER

Khusainova Munira Alisherovna,
Samarkand State Medical Institute, Samarkand, Uzbekistan

Gaffarov Xudoyor Xudoyberdiyevich
Samarkand State Medical Institute, Samarkand, Uzbekistan

ABSTRACT
The study analyzed the results of a survey of 96 patients (51% men, 49% women) with viral cirrhosis of the liver of class A, B, C according to Child-Pugh criteria, who were treated at the City Infectious Diseases Hospital in Chita. The median age of the patients was 42.1 [36;44] years, the duration of the disease was 3.5 [2.8;6.7] years. The diagnosis of CL was confirmed morphologically (laparoscopy with targeted biopsy) in 9 people, the rest of them were exposed on the basis of clinical, laboratory and instrumental data. The viral genesis of liver damage was confirmed by the presence of markers of viral hepatitis B in the blood serum [HBsAg, antibodies (AT) of classes M and G to HbcorAg, HBV DNA), C (AT of classes M and G to HCV, HCV RNA]. Depending on the presence of ascites, the patients were divided into 2 groups: 59 (61%) patients did not have ascites (group 1), 37 (38%) patients were diagnosed with ascites of varying severity (group 2), the control group consisted of 21 healthy volunteers of the corresponding age without signs of liver pathology. The study did not include: patients older than 52 years with essential and symptomatic arterial hypertension, heart and lung diseases, with chronic alcoholism and severe concomitant pathology. Thus, in patients with ICP without ascites, an increase in the mass of the left ventricular myocardium, the left atrium cavity, and the pulmonary artery was found, there is a violation of the heart rhythm and an elongation of the corrected QT interval. These changes were more pronounced in patients with ascites, in addition, they had increased pressure in the pulmonary artery and decreased systolic function of the heart's ventricles. Antiviral therapy has a positive effect on some cardiohemodynamic parameters.

Keywords: left ventricle, viral cirrhosis of the liver, antiviral therapy.

INTRODUCTION
Cirrhosis of the liver (CL) is one of the most pressing medical problems of the last decade. This is due to a large increase in viral liver diseases, in particular, caused by hepatitis B and C viruses. Disorders of portal blood circulation trigger a cascade of autonomic, neurohumoral and metabolic reactions that cause changes in central hemodynamics, which exacerbates not only disorders of intrahepatic blood flow, but also leads to multi-organ extrahepatic disorders, including cirrhotic cardiomyopathy. Currently, heart damage in patients with viral cirrhosis of the liver (VCL), the mechanisms of development of the main symptoms of cirrhotic cardiomyopathy, biochemical and electrophysiological changes in the heart, the conditions for the occurrence of diastolic and systolic dysfunction, and the features of structural and functional changes in the myocardium are actively studied. Meanwhile, the syndrome of cirrhotic cardiomyopathy has not yet been definitively classified, and many mechanisms for the development of myocardial dysfunction in patients with CL are unknown. The absence of these data indicates a lack of awareness of practitioners about changes in the cardiovascular system. Reports of deaths due to heart failure during liver transplantation, transjugular intrahepatic portosystem bypass surgery, and surgical portocaval shunts in patients with cirrhosis suggest that myocardial dysfunction may progress with increased circulating blood volume. It is known that antiviral therapy in patients with chronic viral hepatitis and cirrhosis of the liver serves as a prevention of the development of liver failure and hepatic cell carcinoma. Currently, more convenient endpoints are used to evaluate the effectiveness of the therapy, which include suppression of virus replication, disappearance of the virus antigen, normalization of alanine aminotransferase activity, improvement of the liver histological picture, prevention of reactivation of infection after liver transplantation, and improvement of the quality of life of patients. The only drug with proven effectiveness in the treatment of viral hepatitis and compensated viral cirrhosis of the liver is interferon, used in

combination with nucleoside analogues. The literature describes studies in which antiviral therapy leveled most of the structural and functional abnormalities of the cardiovascular system, improved the parameters of the functional state of the endothelium, and also contributed to the normalization of the number and frequency of increased levels of antimyocardial antibodies, with the exception of patients with mixed infection. The aim of our study was to study the structural and functional parameters of the heart, as well as the effect of antiviral therapy on some cardiohemodynamic parameters in patients with VCL.

MATERIALS AND METHODS

The study analyzed the results of a survey of 96 patients (51% men, 49% women) with viral cirrhosis of the liver of class A, B, C according to Child-Pugh criteria, who were treated at the City Infectious Diseases Hospital in Chita. The median age of the patients was 42.1 [36;44] years, the duration of the disease was 3.5 [2.8;6.7] years. The diagnosis of CL was confirmed morphologically (laparoscopy with targeted biopsy) in 9 people, the rest of them were exposed on the basis of clinical, laboratory and instrumental data. The viral genesis of liver damage was confirmed by the presence of markers of viral hepatitis B in the blood serum [HBsAg, antibodies (AT) of classes M and G to HbcorAg, HBV DNA), C (AT of classes M and G to HCV, HCV RNA]. Depending on the presence of ascites, the patients were divided into 2 groups: 59 (61%) patients did not have ascites (group 1), 37 (38%) patients were diagnosed with ascites of varying severity (group 2), the control group consisted of 21 healthy volunteers of the corresponding age without signs of liver pathology. The study did not include: patients older than 52 years with essential and symptomatic arterial hypertension, heart and lung diseases, with chronic alcoholism and severe concomitant pathology. There were no pronounced signs of heart failure in the groups of examined patients. Standard and tissue myocardial doppler echocardiography was performed according to the standard method on the device "AQQUVIX QX". Tissue Doppler echocardiography was performed from the apical approach at the level of two or four chambers, the Doppler spectrum was recorded from the fibrous rings of the mitral, tricuspid valves and ventricular segments, the indices were calculated: Sm-systolic myocardial contraction, the maximum rate of the first negative peak Em, the maximum rate of the second negative peak Am, the Em/Am ratio, the time before myocardial contraction Ivs, the relaxation time Ivr [8, 9]. Systolic pressure in the the pulmonary artery was measured by the rate of tricuspid regurgitation. Regional longitudinal deformity and the rate of left ventricular (LV) myocardial deformity were studied by the Neppler mode of two-dimensional seroscale deformity. The study was performed from the apical access in the long axis position, and the LV myocardium was recorded with optimal visualization of all segments, with a frame rate from 50 to 80 per second, with stable ECG recording. The endocardium was clearly traced, and the epicardial surface was traced automatically. The program calculated from frame to frame the displacement of the pattern of spots within the zone of interest throughout the entire cardiac cycle. After optimizing the area of interest, the software generated strain curves for each of the 6 segments. Regional and global (by averaging the values of all segments) amplitude and time indicators were obtained from these curves.

Xolter ECG monitoring (HM) was performed using the CardioSens complex. The variance of the QT interval was calculated as the difference between the mean maximum and minimum values of the QT interval in six precordial ECG leads. To correct the variance of the QT interval depending on the heart rate (variance of the corrected QTc interval), a modified H. Bazett formula was used: where dQTc is the variance of the corrected QT interval; dQT is the variance of the QT interval; RR is the duration of the cardiac cycle. The QT interval variability coefficient (QTvar) was calculated by the formula: QTvar=(QTc)/(QTcp)x100%, where QTsp is the average value of the QT interval. Statistical data processing was carried out using the statistical software package Statistica 6.0 (Statsoft Inc., USA). The distribution of almost all variation series did not comply with the criteria of normality, so the analysis used methods of nonparametric statistics. To assess the differences between the groups, the nonparametric Mann-Whitney test was used.The correlation analysis was performed using the Spearman rank correlation coefficient.

RESULTS

According to the XM ECG data, ventricular extrasystoles of various gradations – from I to IVB class according to the Laun-Wolf classification-were detected in 8 (13%) patients of group 1 and 12 (33%)

patients of group 2. When comparing the corrected QT interval in patients with viral cirrhosis of the liver, its lengthening was noted, this indicator in patients of group 1 was 457.9 [442;468], in group 2-478[433;502] in the control – 427.9 [406;438] ($p<0.001$). Supraventricular arrhythmias were represented by supraventricular extrasystoles in 15 (25%) patients of group 1 and in 18 (50%) patients of group 2; paroxysmal atrial fibrillation in 2 (3%) and 7 (19%) patients, respectively. When comparing the average values of the tissue Doppler spectrum in patients with viral CL, it was found that the peak systolic velocity (Sm) of the side wall (segments 3, 9) in patients with ascites was lower by 23% and 25%, respectively, compared with group 1. It can be assumed that the global longitudinal systolic function of the LV in patients with ascites, in contrast to patients without ascites, was reduced. The Shadow myocardial performance index in group 2 increased at the level of the tricuspid ring, which characterizes a decrease in the global function of the right ventricle in patients with ascites in comparison with the control and 1st groups. The conducted echocardiographic analysis showed that in patients with CL without ascites, there is an increase in indicators that characterize the mass left ventricular myocardium (interventricular septum, posterior LV wall, LV myocardial mass and LV myocardial mass index), dilated left atrium, pulmonary artery. These disorders increased in patients with ascites, where systolic pressure in the pulmonary artery also increased.

When assessing myocardial deformity in patients with CP, the index of the maximum systolic strain for the anterior-septal segment in patients with ascites was statistically significantly different from that in the control group. The parameters of the lower-lateral, lower, and lower-septum segments of the LV were lower in patients with ascites compared to patients in the control group and patients without ascites. The global systolic strain (in the position on chambers 2 and 4, and along the long axis from the apical access) in patients with CP in the presence of ascites was statistically significantly different from those in patients without ascites and in the control group. Thus, patients with ascites showed a decrease in segmental and global LV systolic function.

It is known that antiviral therapy in patients with viral cirrhosis of the liver serves as a prevention of the development of liver failure and hepatic cell carcinoma. 16 patients with viral CP received antiviral therapy with interferon drugs in combination with ribavirin with the formation of a persistent virological response time within 11.8 months. Specific treatment had a positive effect on some morpho-functional parameters of the heart: there was a decrease in left ventricular mass by 7%, left atrial volume index by 10%, and systolic pressure in the pulmonary artery by 12%.

DISCUSSION

The study confirms the data on the presence of myocardial dysfunction in patients with viral CL [1-5]. In compensated patients, the left ventricle is remodeled with an increase in its mass, the volume of the left atrium, the diameter of the pulmonary artery, and there is a decrease in the maximum systolic velocity of the mitral valve fibrous ring. In addition, there was a violation of the heart rhythm by the type of extrasystole, paroxysmal form of atrial fibrillation, as well as an elongation of the corrected QT interval. In patients with ascites, there is a more pronounced remodeling of the left ventricle with an increase in its mass, the left atrium and the pulmonary artery expand even more, the systolic function of the ventricles decreases, heart rhythm disorders are more common, and the QT interval is even longer. The leading role in the development of changes in the architectonics of the heart in patients with viral CL belongs not only to mechanical factors (an increase in the volume of circulating blood and its viscosity, total peripheral resistance, systolic tension of the walls of the left ventricle, etc.) and neurohumoral factors (the influence of the renin-angiotensin and sympathetic systems), but also to the influence of the damaging effect on the cardiomyocytes of the inflammatory factors and procoagulants circulating in the blood. There is evidence of a direct effect on the myocardium of the core protein of the virus, and there is a possibility of settling of circulating immune complexes both in the myocardium and in the vascular wall with the formation of vasculitis. It is also known about the cardiotoxic effect of endotoxins, in particular, bile acids, due to their ability to inhibit the automatism of the sinus node, which is one of the factors of vagal dysfunction of the reduced reactivity of the sympathetic nervous system. Hypocalcemia, developed as a result of increased activity of aldosterone, inhibits the processes of depolarization in the myocardium. A clear marker of these dielectrolyte disorders is the extended QT interval, which can form electrical instability of the myocardium with the development of

intraventricular conduction disorders and life-threatening arrhythmias. In response to overload, the structure and function of the heart changes, the left atrium expands, the mass of the myocardium increases, and due to compression of the intramural coronary arteries, the coronary reserve decreases, which contributes to the development of cardiac arrhythmias. In turn, a decrease in coronary blood flow impairs the pumping function of the ventricles. As a result of morphological changes in the myocardium, the shortening of the interval between contractions leads to a decrease in LV filling. The deterioration of its filling is compensated by an increase in pressure in the small circle. A decrease in extensibility also contributes to a decrease in contractile function. The insufficiency of one half of the heart causes the other half to be overstressed, and the increased preload leads to an increase in the residual volume of blood, increases the pressure in the right parts and veins of the large circulatory circle, splanchnic fullness is formed, which contributes to an increase in pressure in the LA. Antiviral therapy in patients with viral CP had a positive effect on cardiohemodynamic parameters and was accompanied by a decrease in left ventricular myocardial mass, left atrial volume index, and systolic pressure in the pulmonary artery. It is possible that the phase of virus integration due to antiviral therapy is accompanied by a decrease in portal hypertension, resulting in a decrease in LV mass and LP volume, which play an important role in the formation of cardiac arrhythmias. In all patients with CP, the relationship between the global maximum strain (Avg) and the parameters of hepatoportal hemodynamics, structural and functional parameters of the heart was studied. A strong correlation was found between Avg and increased viral load, portal vein diameter. From this, it can be concluded that a possible cause of myocardial damage may be direct exposure to hepatitis viruses.

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CONCLUSION

Thus, in patients with CP of viral etiology without ascites, an increase in the mass of the left ventricular myocardium, the left atrial cavity, and the pulmonary artery was found, there is a violation of the heart rhythm and an elongation of the corrected QT interval. These changes are more pronounced in patients with ascites, in addition, they increased the pressure in the pulmonary artery and decreased the systolic function of the ventricles of the heart. Antiviral therapy has a positive effect on some cardiohemodynamic parameters.

REFERENCES:

1) Peshkova S. V., Chistyakova M. V., Govorin A.V., etc. Clinical and pathogenetic features of cirrhotic cardiomyopathy. Zabaikalsky medical Bulletin. 2017;2:63-71. (In Russian). [Peshkova S. V., Chistyakova M. V., Govorin A.V., et al. Clinical and pathogenetic features of cirrhotic cardiomyopathy. Zabaikalsky Medical Bulletin. 2017;2:63-41].
2) Malinovskaya Yu. O., Moiseev S. V., Moisyuk Yu. G. Cardiomyopathy in cirrhosis and liver transplantation. Almanac of Clinical Medicine. 2016;44:775-84. (In Russian). [Malinovskaya Yu. O., Moiseev S. V., Moisyuk Ya. G. Cirrhotic cardiamiopathy and liver transplantation. Almanac of Clinical Medicine Medicine. 2016; 44: 775-84]. doi: 10.18786 / 2072-0505-2016-44-6-775-784.
3) Levitan B. N., Kasyanova T. P. Prevalence of the main diagnostic criteria of cardiomyopathy in patients with cirrhosis of the liver. Doktor.Ru. 2014;7(95):9-13. (In Russian). [Levitan B. N. Kasyanova T. R. Frequency of occurrence of the leading diagnostic criteria of cardiamiopathy in cirrhosis of the liver. <url>. 2014;7(95):9-13].
4) Kasyanova T. R., Astatsin A.V., Levitan B. N., etc. Evaluation of structural and functional parameters right heart in patients with cirrhosis of the liver. Modern problems of science and education. 2011;6:67. (In Russian). [Kasyanova T. R., Astakhin A.V., Levitan B. N., et al. Evaluation of structural and functional parameters of the right heart in patients with cirrhosis of the liver. Modern Problems Science and Education. 2011;6:67].

5) Kalacheva T. P., Chernyavskaya G. M., Beloborodova E. I., etc. Endothelial vascular regulation disorders and echocardiographic evaluation of myocardial dysfunction and pulmonary circulation hemodynamics in liver cirrhosis. Cardiology. 2016;56(1):41-7. (In Russian). [Kalacheva T. P., Chernyavskaya G. M., Beloborodova E. I., etc. Disorders of the vasoregulatory function of the endothelium and echocardiographic assessment of myocardial dysfunction and hemodynamics of the small circle of blood circulation in cirrhosis of the liver. Cardiology. 2016;56(1):41-7]. doi: 10.18565/cardio. 2016. 1. 41-47.
6) Lopatkina T. N., Strizhakov L. A., Konysheva A. A., et al. Variants of heart damage in chronic hepatitis. Clinical pharmacology and therapy. 2014;23(4):90-3. (In Russian). [Lopatkina T. N., Strizhakov L. A., Konysheva A. A. (In Russian). Variants of heart damage in chronic hepatitis C. Clinical Pharmacology and Therapy. 2014;23(4):90-3].
7) Ratti L., Redaelli E., Guidi S. et.al. Diastolic dysfunction in cirrhosis of the liver. Gastroenterol. Hepatol. 2005;28(10):649-55.
8) Govorin A.V. Non-coronary myocardial injury. Novosibirsk: Nauka, 2014. (In Russian). [Govorin A.V. Non-coronarogenic myocardial lesions. Novosibirsk: Nauka; 2014].
9) Sokolova N. A., Danshova M. S., Govorin A.V., Zaitsev D. N. Association of Polymorphism matrix metalloproteinase genes (9, 12, and 20) with early left ventricular remodeling in patients with acute myocardial infarction. Heart. 2017;16(4):268-73. (In Russian). [Sokolova N. A., Danshova M. S., Govorin A.V., Zaitsev D. N. Association of matrix metalloproteinase gene polymorphism (9, 12, and 20) with early left ventricular remodeling in patients with acute myocardial infarction. Heart. 2017;16(4):268-73]. doi ID: 10.18087 / rhj. 2017. 4. 2362.
10) Palmieri V., Russo S., Palmieri E. A., et al. Changes in the components of left ventricular mechanics in patients with selective beta-1 blockade: a look from traditional and new technologies in echocardiography. Eur J Echocardiogr. 2009; 10: 745-52. doi:10.1093 / ejechocard/ jep055.
11) Teske A. J., De Boeck B. W., Olimulder M. Et al. Echocardiographic evaluation of regional function of the right vein: comparison of two-dimensional and tissue Doppler strain analysis. J Am Soc Echocardiogr. 2008;21(3):275-83. doi: 10.1016/j.echo.2007.08.027.