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Original Article

Comparative assessment of liver fibrosis in patients with HIV/chronic hepatitis C co-infection in different ethnic groups

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Abstract

Introduction: In recent years, the frequency of human immunodeficiency virus (HIV) and hepatitis C (HCV) co-infection has increased, which is due to their common routes of transmission. HIV/chronic HCV co-infection aggravates the development of fibrosis and increases the risk of cirrhosis. The aim of the study was to evaluate the results of liver elastometry in patients of different ethnic groups with HIV/chronic HCV co-infection.

Methodology: The study involved 49 Kazakh and 46 Russian patients with HIV/chronic HCV co-infection. The stage of liver fibrosis was assessed by the results of indirect ultrasonic liver elastometry according to the METAVIR scale using FibroScan 502. As an indirect marker of liver fibrosis, level of alanine aminotransferase and aspartate aminotransferase, as well as platelet counts, were determined.

Results: Analysis of the results with the evaluation of the dynamics of fibrotic process in 36 months revealed a prevalence of patients with advanced liver fibrosis (F3, F4) among Kazakh compared with Russian patients, accompanied by a significant increase of liver elasticity indices in Kazakhs and Russians ($p < 0.05$). Significant differences in the indices of transaminases in the patients with later stages of liver fibrosis (F3, F4) were found ($p < 0.05$).

Conclusions: The study of patients with HIV/chronic HCV co-infection revealed differences in the progression of liver fibrosis depending on ethnicity. Results of elastometry and indirect markers of liver fibrosis were used in the comprehensive assessment at different stages of liver fibrosis.

Key words: HIV/chronic hepatitis C co-infection; liver fibrosis; cirrhosis; liver elastometry; Kazakhs; Russians.

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Introduction

The common factors of human immunodeficiency virus (HIV) and hepatitis C (HCV) transmission determine their frequent combined identification, and there are about 10 million people co-infected with both viruses [1-3]. In Kazakhstan, 23,593 cases of HIV infection had been registered at the turn of 2014 (2,216 cases in Karaganda region), and HCV was diagnosed in 1,098 HIV-infected patients [4].

Compared with HCV mono-infection, more rapid progression of chronic liver disease, prevalence of inflammatory and fibrotic expressed processes, and formation of cirrhosis at HIV/HCV co-infection have been observed in HCV and HIV co-infection [5-9]. In addition, the basic factors in the progression of chronic HCV include the Asian race, co-infection with HBV and HIV, age over 40 years, male sex, and alcohol abuse [10-12].

To date, the literature we have available reveals research findings focusing on the study of epidemiological and diagnostic criteria for viral hepatitis. Also, it suggests ethnic characteristics in the course of the disease among patients of different races and nationalities. The studies point to ethnic differences in the progression rate of liver fibrosis and hepatocarcinogenesis, as evidenced by a high incidence of liver cirrhosis and hepatocellular carcinoma in particular areas of the world (Japan, China, Taiwan, South Africa). Among patients with chronic HCV, approximately 10%–15% of men and 1%–5% of women develop liver cirrhosis; 1%–4% of patients with cirrhosis subsequently develop hepatocellular carcinoma. In contrast, Asian countries record

development of cirrhosis in 30%–46% of infected people and development of hepatocellular carcinoma in 10%–19% [11,13,14].

Research analysis indicates that there is still no clear understanding of the causes that lead to different rates of development of liver fibrosis in patients co-infected with HIV/chronic HCV. This fact has been the subject of research in different countries [15-23]. Thus, in the course of a retrospective study on the impact of HIV infection on liver fibrosis among HCV-infected African Americans, it was found that 50% of African Americans surveyed had early stages of fibrosis, despite the long duration of the disease [24]. A similar study was conducted among women of different racial/ethnic backgrounds co-infected with HIV/chronic HCV. The study included 495 African-American women, 140 Caucasian women, and 159 Hispanic women. The study found that African-American women had a significantly lower mortality rate of liver disease compared to the rates of the other female groups, namely Caucasian and Hispanic. The researchers suggested that the unfavorable course of hepatotropic infections may be due to the ethnic features underlying their progression [25].

Modern non-invasive methods of evaluation of hepatic fibrosis degree include liver elastometry, which is characterized by high information content, the possibility of the monitoring of staging process, dynamic assessment, and timely therapy administration to prevent the further progression of fibrosis [26-34].

Treating patients co-infected with HIV/chronic HCV requires a comprehensive assessment of liver fibrosis (elastometry of the liver and determination of the serum markers of fibrosis). To investigate the activity of fibrogenesis in the liver, the study uses a number of panels of biochemical parameters (aspartate aminotransferase, alanine aminotransferase, alkaline phosphatase, γ-glutamyltranspeptidase, total bilirubin, etc.) and acute-phase proteins (α-2-macroglobulin, haptoglobin, ferritin, etc.). Discriminant functions, derived on the basis of changes in the levels of these indices, reflect the activity of inflammatory process in the organ tissue and impaired state of its synthetic function, thereby allowing indirect determination of the stage of fibrosis [35-37].

In clinical practice, a panel of diagnostic tests is undertaken based on the indirect markers of fibrosis: FibroTest (α-2-macroglobulin, γ-glutamyltranspeptidase, apolipoprotein A1, haptoglobin, total bilirubin, age and gender), Forns Index (age, platelet count, cholesterol, γ-glutamyltranspeptidase), Virahep-C model (aspartate aminotransferase, platelet count, alkaline phosphatase, age) [28,33,38-40], and others. Wai *et al.* [41] recommend using tests based on indirect markers of fibrosis, and indicated that the following combination of indices should be noted: aspartate aminotransferase, alkaline phosphatase, and platelet count. This combination of markers is effective for the diagnosis of patients with chronic HCV.

Thus, studying the possibility of using liver elastometry to assess liver fibrosis assessment in HIV/chronic HCV-co-infected patients of different ethnicities can be promising. There are several risk factors currently known to underlie the progression of chronic diffuse liver diseases, including HCV. Simultaneously, ethnicity is among the significantly less frequently studied factors in the development and progression of liver fibrosis in patients co-infected with HIV/chronic HCV, especially patients of Central Asian descent (Kazakhs).

The aim of the study was to evaluate the results of liver elastometry in patients of different ethnic groups with HIV/chronic HCV co-infection.

Methodology

This was an open, non-randomized, prospective study. It was approved by the ethics committee.

Patients' characteristics

The study included 95 patients with HIV and chronic HCV co-infection. The patients were follow-ups of the Karaganda Regional Center for AIDS Prevention and Control. They were divided into two groups. Group I (study) included 49 Kazakh patients with HIV/chronic HCV co-infection. Group II (control) included 46 Russian patients with HIV/chronic HCV co-infection.

The inclusion criteria were as follows: a verified diagnosis of HIV/chronic HCV co-infection, Central Asian (Kazakh) and Slavic (Russian) descent, the absence of clinical stage IV (AIDS), the absence of antiviral therapy at the time of the study, and informed consent to the examination. The exclusion criteria were age under 18 years, non-viral etiology of liver disease, alcohol abuse, cancer, and severe mental or neurological pathology.

This study was conducted before the start of antiviral therapy. The problem of providing HCV treatment of everyone who needs it is not limited to Kazakhstan; it is a concern in many other countries. Hence, this is one of the reasons for the high cost of medication. Patients cannot afford expensive treatment on their own, given not only their individual low socio-

economic level, but also the country's as a whole. According to European standards and recommendations, priority order of antiviral therapy is given to patients with stage F3–F4 liver fibrosis [42]. The number of surveyed Kazakh and Russian patients with advanced stage of liver fibrosis (F3, F4) was 20.4% and 15.2%, respectively. It should be noted that patients co-infected with HIV/chronic HCV require an individual approach that will take into account their state of immunodeficiency. Patients were followed up in view of their further treatment.

Descriptions of the patients are presented in Table 1. In general, the study groups were comparable ($p > 0.05$).

Analysis of HCV disease duration showed that the minimum and maximum periods of the disease ranged between 3 and 12 years (mean values of 5.3 ± 0.4 years among Kazakhs and 5.4 ± 0.4 years among Russians). The duration of the HCV disease and its co-infections could be set only provisionally on the basis of possible risk factors for infection. Carefully collected epidemiological history served as grounds to determine the duration of HCV disease. However, the approximate period from the date of acquiring HCV infection was established based on the results of clinical and anamnestic data.

When examining the routes of infection in the study groups, it was found that the largest share of incidents corresponded to intravenous drug use, while a significantly smaller proportion was related to sexual transmission. For example, 69.4% of Kazakhs and 67.4% of Russians acquired infection through the route of injecting drugs, and 30.6% and 32.6%, respectively, acquired infection through sexual contact. These patients were not injecting drug users. The data on injecting drug users were recorded in Karaganda Regional Center for AIDS Prevention and Control. After careful collection of epidemiological history, it was revealed that female patients constituted the majority and had sexual contacts with HIV-infected patients. In Kazakhstan, a high level of HCV infection in HIV-infected patients can be explained by a significant prevalence of the intravenous drug route over the sexually transmitted route. This fact is explained by the drug epidemic that swept Kazakhstan in the late 90s, which led to HIV infection as a result of intravenous administration of psychoactive substances in 70%–80% of cases. In this regard, the use of intravenous drugs was the dominant cause of HIV infection and its combined course with HCV.

At the time of the study, patients did not need highly active antiretroviral therapy (HAART) given their

Table 1. Characteristics of patients of different ethnicities co-infected with HIV/chronic hepatitis C.

Indices	Group I (Kazakhs) n = 49	Group II (Russians) n = 46	p
Age, years (26–52 years)	40.3 ± 1.0 (median 40.0)	42.2 ± 1.0 (median 43.0)	0.1924
Gender			
Male	33 (67.3%)	32 (69.6%)	0.7955
Female	16 (32.7%)	14 (30.4%)	0.8613
HCV duration, years (3–12 years)	5.3 ± 0.4	5.4 ± 0.4	0.8491
Method of transmission			
Intravenous drugs	34 (69.4%)	31 (67.4%)	0.8634
Sexual intercourse	15 (30.6%)	15 (32.6%)	0.9074
HCV genotype			
Genotype 1	21 (42.9%)	18 (39.1%)	0.8016
Genotype 2	3 (6.1%)	4 (8.7%)	0.8887
Genotype 3	25 (51.0%)	24 (52.2%)	0.9445
Clinical stage of HIV infection			
Stage I	26 (53.1%)	23 (50%)	0.8348
(CD4-lymphocytes, cells/μL)	557.4 ± 23.0 (median 541.0)	567.3 ± 32.5 (median 576.0)	0.8013
Stage II	20 (40.8%)	19 (41.3%)	1.0000
(CD4-lymphocytes, cells/μL)	484.4 ± 24.0 (median 466.0)	476.5 ± 24.8 (median 494.0)	0.8201
Stage III	3 (6.1%)	4 (8.7%)	0.8887
(CD4-lymphocytes, cells/μL)	411.3 ± 8.7 (median 412.0)	418.0 ± 19.4 (median 425.5)	0.7919
Stage IV	-	-	-

stable levels of CD4 lymphocytes. In accordance with international recommendations, the main criterion for initiation of therapy in the Republic of Kazakhstan is the number of CD4 lymphocytes (fewer than 350 cells/μL) and clinical stage of HIV infection [43]. At the time of inclusion in the study, most patients (46 [93.9%] of patients in group I, and 42 [91.3%] in group II) were diagnosed with an early clinical stage of HIV infection. Sampling was carried out by an electronic database of patients with initially elevated levels of CD4 lymphocytes.

During the study, patients co-infected with HIV/chronic HCV in both groups were under dynamic observation, which included clinical examination as well as determination of transaminase activity, the number of CD4 lymphocytes, and viral load every six months.

Laboratory methods

Diagnosis of HIV (ICD-10, B20) was verified by the immune blotting method (Innogenetics, Ghent, Belgium) on the basis of the commercial test system New LAV Blot (BioRad Laboratories, Steenvoorde, France). The stages of HIV infection were determined according to the clinical classification of the World Health Organization (WHO) [44]. CD4 lymphocyte count was determined by the flow cytofluorometry method FACSCount using monoclonal antibodies (Becton-Dickinson, Franklin Lakes, USA).

Diagnosis of HCV (ICD-10, B 18.2) was verified by enzyme-linked immunosorbent assay (ELISA) using commercial kits (JSC Vector-Best, Novosibirsk, Russia) for detection of the HCV antigen. Quantification of HCV RNA was performed by real-time polymerase chain reaction (PCR) using commercial reagents with hybridization-fluorescence detection (AmpliSens HCV-FL, InterLabService, Moscow, Russia). In order to identify, quantify, and differentiate genotypes 1, 2, and 3, a commercial reagents kit (RealBest RNA HCV-genotype, Novosibirsk, Russia) was used.

Indirect markers of fibrosis were determined for the study of the activity of fibrogenesis in livers of patients with HIV/chronic HCV co-infection and included alanine aminotransferase (ALT), aspartate aminotransferase (AST), and platelets. Along with this, the rate of cholestatic syndrome (*i.e.*, alkaline phosphatase [ALP]) was determined. The severity of the cytolysis syndrome on the content of serum transaminases (ALT, AST) was determined in all the patients and included values within the normal range (0.028–0.19 μmol/[s-L]), minimal cytolysis (up to 3 standards), and moderate cytolysis (from 3 to 10 standards). The values of ALP were considered within the normal range at 278–830 nmol/(s-L), and platelets at 180–320 × 10^9/L. Biochemical parameters (ALT, AST, ALP) were determined by the colorimetric method on an analyzer (ERMA Inc., Tokyo, Japan) using commercial kits. Using the unified dinitrophenylhydrazine method of Reitman-Frenkel, the number of platelets was counted on a hematological analyzer (Cell-Dyn Ruby, Abbott, Holliston, USA).

The stage of liver fibrosis was assessed by the results of indirect ultrasonic elastometry according to the METAVIR scale using FibroScan 502 (Echosens, Paris, France). The results of the assessment of liver elasticity were expressed in kilopascals (kPa). The liver elastometry was performed twice, at an interval of 36 months.

Statistical methods

The control of the distribution was carried out using indicators of asymmetry and kurtosis. The distribution of indices in two groups was normal, so the t-test was used to assess the differences. The Pearson's linear correlation coefficient (r) was used to estimate the statistical dependence of two series of observations. Statistical processing of the results was performed using the software package Statistica 6 (Dell Statistica, Tulsa, USA), determined by the mean, standard deviation, median, 95% confidence interval (CI), and odds ratio (OR). Differences were considered significant at $p < 0.05$.

Results

Comparative analysis of liver elastometry according to the METAVIR scale showed that initially, stage F0 (no fibrosis) was not registered in Kazakhs and Russians, and the transition (border) stage F0–F1 was diagnosed in 8.2% and 10.9% of cases, respectively. Minimal fibrosis with stellate expansion of portal tracts without septa formation (F1) was found in 38.8% and 41.3% of Kazakhs and Russians, respectively. Moderate fibrosis with the expansion of portal tracts with a single port-portal septa (F2) was found in in 32.6% and 32.6% of cases; expressed fibrosis with numerous port-central septa (F3) in 12.2% and 8.7% of cases; and cirrhosis (F4) in 8.2% and 6.5% of cases, respectively (Table 2).

The changes of the indices of the liver elasticity at different stages, starting from stages F0–F1, were registered during the assessment of the dynamics of liver fibrosis at an interval of 36 months on the results of repeated elastometry in Kazakhs and Russians.

Table 2. Distribution of patients of different ethnicities with HIV/chronic hepatitis C co-infection according to the stages of liver fibrosis based on baseline data and dynamic monitoring.

Stages of fibrosis (reference values, kPa)	Baseline data				p
	Group I (Kazakhs) n = 49		Group II (Russians) n = 46		
	n (%)	M ± m, kPa	n (%)	M ± m, kPa	
F0–F1 (F0: 1.5–5.8; F1: 5.9–7.2)	4 (8.2)	6.1 ± 0.1	5 (10.9)	6.0 ± 0.1	0.4803
F1 (5.9–7.2)	19 (38.8)	6.8 ± 0.1	19 (41.3)	6.7 ± 0.04	0.1320
F2 (7.3–9.5)	16 (32.6)	8.4 ± 0.1	15 (32.6)	8.1 ± 0.1	0.0765
F3 (9.6–13)	6 (12.2)	11.0 ± 0.2	4 (8.7)	10.8 ± 0.1	0.4755
F4 (> 13)	4 (8.2)	26.6 ± 3.7	3 (6.5)	24.1 ± 4.9	0.6943
	Control in 36 months				
F0–F1(F0: 1.5–5.8; F1: 5.9–7.2)	3 (6.1)	6.2 ± 0.1	5 (10.9)	6.1 ± 0.1	0.2199
F1 (5.9–7.2)	16 (32.7)	7.0 ± 0.04	18 (39.1)	7.0 ± 0.1	1.0000
F2 (7.3–9.5)	10 (20.4)	9.0 ± 0.1	13 (28.3)	8.6 ± 0.2	0.0837
F3 (9.6–13)	11 (22.4)	12.4 ± 0.2	6 (13.0)	11.3 ± 0.3	0.0026*
F4 (> 13)	9 (18.4)	33.3 ± 1.1	4 (8.7)	26.6 ± 2.9	0.0213*

* Significance of differences in the two groups, kPa ($p < 0.05$); M: mean; m: standard error of the mean.

Table 3. Comparative analysis of laboratory data on patients of different ethnicities co-infected with HIV/chronic hepatitis C based on baseline data.

Stages of fibrosis	Group I Kazakhs n = 49	Group II Russians n = 46	p	Group I Kazakhs n = 49	Group II Russians n = 46	p
	ALT, µmol/(s-L)			AST, µmol/(s-L)		
F0–F1	0.14 ± 0.05	0.13 ± 0.04	0.8905	0.11 ± 0.05	0.11 ± 0.04	1.0000
F1	0.31 ± 0.03	0.35 ± 0.04	0.4616	0.29 ± 0.03	0.27 ± 0.03	0.6735
F2	0.44 ± 0.05	0.37 ± 0.04	0.2630	0.34 ± 0.03	0.32 ± 0.03	0.6602
F3	0.53 ± 0.10	0.42 ± 0.11	0.4661	0.43 ± 0.07	0.33 ± 0.05	0.3249
F4	0.59 ± 0.07	0.45 ± 0.14	0.3816	0.48 ± 0.09	0.35 ± 0.05	0.2816
	ALP, nmol/(s-L)			Platelets, 10^9/L		
F0–F1	404.5 ± 60.4	449.2 ± 101.3	0.7343	210.3 ± 12.2	207.0 ± 10.2	0.8409
F1	480.5 ± 38.4	461.3 ± 42.5	0.7394	193.3 ± 3.0	201.4 ± 4.1	0.1176
F2	533.9 ± 35.6	535.3 ± 40.5	0.9794	196.4 ± 5.7	196.7 ± 6.2	0.9718
F3	591.8 ± 63.3	561.0 ± 116.5	0.8061	190.0 ± 6.9	194.5 ± 12.4	0.7387
F4	545.8 ± 122.8	593.7 ± 134.3	0.8047	183.5 ± 12.4	186.0 ± 9.2	0.8864

ALT: alanine aminotransferase; AST: aspartate aminotransferase; ALP: alkaline phosphatase.

Table 4. Comparative analysis of laboratory data on patients of different ethnicities co-infected with HIV/chronic hepatitis C based on dynamic monitoring.

Stages of fibrosis	Group I Kazakhs n = 49	Group II Russians n = 46	p	Group I Kazakhs n = 49	Group II Russians n = 46	p
	ALT, µmol/(s-L)			AST, µmol/(s-L)		
F0–F1	0.17 ± 0.06	0.18 ± 0.04	0.8990	0.15 ± 0.06	0.16 ± 0.03	0.8624
F1	0.42 ± 0.05	0.39 ± 0.04	0.6309	0.31 ± 0.04	0.29 ± 0.03	0.7000
F2	0.54 ± 0.05	0.42 ± 0.05	0.1081	0.41 ± 0.06	0.37 ± 0.04	0.5781
F3	0.71 ± 0.06	0.45 ± 0.09	0.0276*	0.57 ± 0.04	0.36 ± 0.08	0.0196*
F4	0.76 ± 0.07	0.47 ± 0.10	0.0365*	0.65 ± 0.04	0.41 ± 0.10	0.0237*
	ALP, nmol/(s-L)			Platelets, 10^9/L		
F0–F1	554.0 ± 34.4	524.6 ± 84.0	0.8064	208.0 ± 4.2	201.8 ± 4.7	0.4038
F1	558.1 ± 43.6	540.7 ± 44.3	0.7823	197.3 ± 6.7	196.1 ± 3.4	0.8685
F2	548.2 ± 45.6	545.3 ± 54.5	0.9691	191.8 ± 4.3	194.2 ± 5.6	0.7505
F3	636.7 ± 39.4	622.7 ± 76.4	0.8583	187.6 ± 11.1	188.2 ± 15.0	0.9748
F4	611.7 ± 52.2	602.5 ± 63.4	0.9201	176.8 ± 11.6	180.0 ± 22.5	0.8906

* Significance of differences in the two groups ($p < 0.05$); ALT: alanine aminotransferase; AST: aspartate aminotransferase; ALP: alkaline phosphatase.

Figure 1. Enzymatic activity of alanine aminotransferase (ALT) depending on the stage of liver fibrosis based on dynamic monitoring.

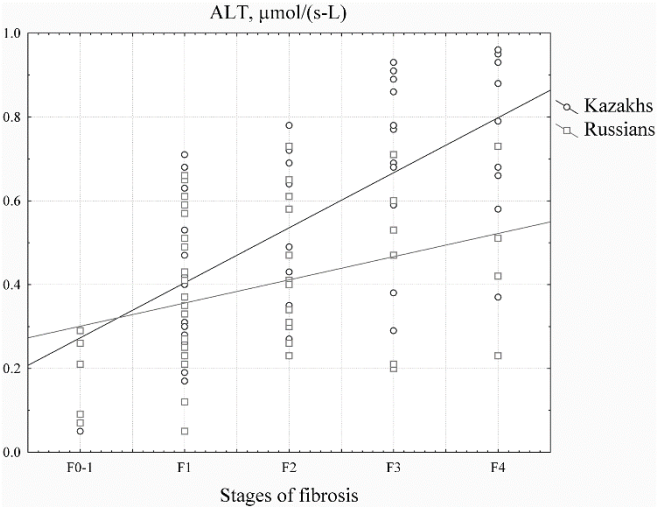
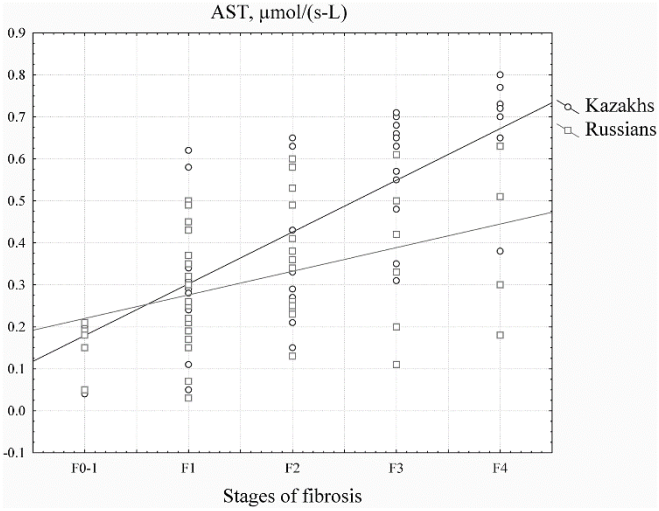


Figure 2. Enzymatic activity of aspartate aminotransferase (AST) depending on the stage of liver fibrosis based on dynamic monitoring.



As can be seen in Table 2, increasing numbers of patients with severe fibrosis (F3) (22.4% and 13.0%) and liver cirrhosis (F4) (18.4% and 8.7%) was registered in Kazakh and Russian groups, respectively, compared with the original data. This is apparently associated with more profound involvement of hepatocytes in the background of immunodeficiency of patients that contributed the development of liver fibrosis. However, the prevalence of patients with advanced liver fibrosis (F3, F4) was higher in Kazakhs than in Russians, and it was accompanied by a significant increase of liver elasticity indices in Kazakhs and Russians ($p = 0.0026$ for F3, $p = 0.0213$ for F4). This trend of rapid progression of liver fibrosis manifested also at the level of more intensive shift of fibrosis stages in Kazakhs (OR = 2.5; 95% CI = 1.0–6.1).

Table 3 shows the comparative characteristics of indirect markers of liver fibrosis (ALT, AST, platelets) and the index of cholestatic syndrome (ALP) depending on the stage of fibrosis in Kazakhs and Russians with HIV/chronic HCV co-infection. At the same time, significant differences based on the initial data on indirect markers of liver fibrosis were not found ($p > 0.05$).

Significant differences in the average level of transaminase activity were found in the two groups according to the results of the study in 36 months: ALT ($p = 0.0276$ for F3, $p = 0.0365$ for F4) and AST ($p = 0.0196$ for F3, $p = 0.0237$ for F4) (Table 4).

The analysis of indirect markers of liver fibrosis showed that in Kazakhs, the values of transaminases (ALT, AST) were higher compared with those in Russians. In Kazakhs, the average ALT was 3 times and the average AST was 2.3 times above the upper limit of norm in the Russian group (2.1 and 1.7 times, respectively). In this case, there was a development of the syndrome of cytolysis with the violation of hepatocyte integrity, manifested in the increased levels of ALT and AST, which are indirect markers of fibrosis (Figures 1, 2).

Significant differences in the level of alkaline phosphatase and platelet found were not found ($p > 0.05$). At the same time, the proportion of patients with thrombocytopenia was 8.4% in both groups that had been conditioned by HIV/chronic HCV co-infection (Figures 3, 4).

Figure 3. Alkaline phosphatase (ALP) indices depending on the stage of liver fibrosis based on dynamic monitoring.

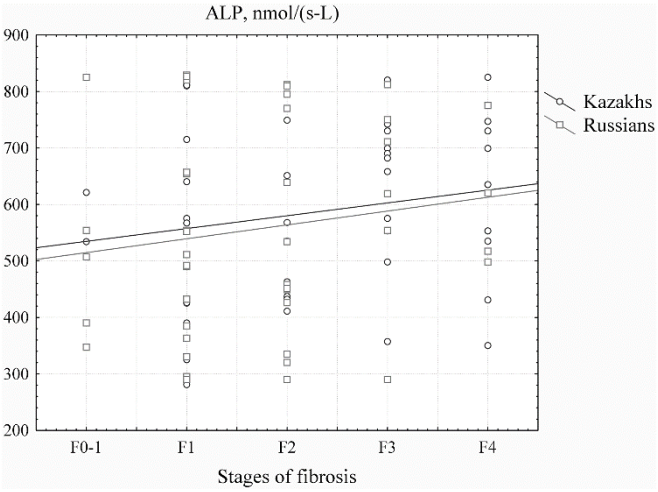
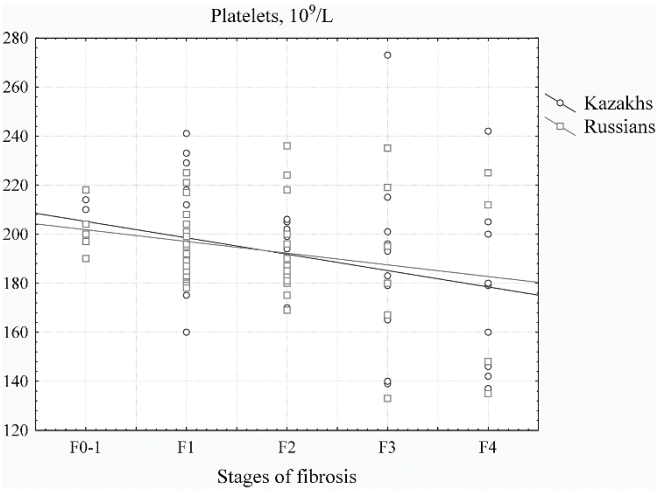


Figure 4. Platelet indices depending on the stage of liver fibrosis based on dynamic monitoring.



Discussion

In recent years, there has been increased interest in research related to the development of liver fibrosis in HIV/HCV co-infection in patients of specific racial/ethnic groups, which requires objective methods of assessment [24,25,45,46].

It was found, during the study, that in the Kazakh group, the progression of liver fibrosis occurred in 18.4% of patients with normal and minimal content of transaminases, and in 22.4% of patients with moderate process activity; in Russians it occurred in 8.7% and 4.3% of patients, respectively. It should be noted that liver fibrosis remained unchanged in 59.2% of Kazakhs and in 87.0% of Russians. This suggests that the progression of liver fibrosis occurred more frequently in Kazakh patients with HIV/chronic HCV co-infection than in Russian patients.

The conducted correlation analysis in patients with HIV/chronic HCV co-infection in the two groups revealed the direct relationship between liver elasticity on the elastometry results and values of hepatic transaminases. The direct moderate relationship between liver elasticity and ALT values ($r = 0.485$; $p < 0.05$) and direct appreciable relationship with the AST values ($r = 0.583$; $p < 0.05$) were registered in Kazakhs. Such relationships had not been revealed in Russians, but the direct weak correlation was noted ($r = 0.254$ and $r = 0.263$ respectively; $p > 0.05$).

Thus, analysis of the elastometry results and indirect markers of liver fibrosis allowed for a comprehensive assessment of liver fibrosis in different stages. In addition, differences in the progression of liver fibrosis due to ethnicity was found among Kazakhs and Russians.

Conclusions

The progression of liver fibrosis in patients with HIV/chronic HCV co-infection was observed more frequently in Kazakhs than in Russians (OR = 2.5; 95% CI = 1.0–6.1). The number of patients with later fibrosis stages F3 (22.4%) and F4 (18.4%) was greater in Kazakhs, and the liver elasticity indices were statistically significant in Kazakhs and Russians ($p < 0.05$).

On the basis of the dynamic observation of Kazakhs according to indirect markers of liver fibrosis, the transaminases indicators (ALT, AST) in the later stages of fibrosis (F3, F4) were significantly higher compared with those found among Russians ($p < 0.05$). According to the average level of platelets, significant differences were not found in the two ethnic groups ($p > 0.05$).

The correlation analysis revealed the direct relationship between liver elasticity based on elastometry results and values of hepatic transaminases. A direct moderate relationship was found between liver elasticity and ALT values ($r = 0.485$; $p < 0.05$), and a direct appreciable relationship with the AST values ($r = 0.583$; $p < 0.05$) was found in Kazakhs. A direct weak correlation in Russians ($r = 0.254$, $r = 0.263$ respectively; $p > 0.05$) was found.

Liver elastometry in patients of different ethnic groups with HIV/chronic HCV co-infection allowed identification and assessment of liver fibrosis in the early stages, as well as the monitoring of its development.

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