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CHANGES IN PLATELETS IN PATIENTS WITH CHRONIC HEPATITES AND LIVER CIRRHOSIS OF VIRAL ETIOLOGY

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Summary: In liver cirrhosis, the normal functioning of hemostasis is disrupted, which leads to an increased risk of both thrombosis and bleeding. The study of platelet changes in such patients helps to understand the pathogenesis of these disorders and contributes to improved diagnostics and therapy.

Purpose of the study. To assess the dynamics of platelet levels in patients with chronic viral hepatitis and liver cirrhosis.

Materials and methods. The study included 140 patients with chronic diffuse liver diseases, including 70 patients with liver cirrhosis of viral etiology, 20 patients with liver cirrhosis of unknown etiology and 50 patients with chronic viral hepatitis of moderate activity.

Results and discussion. As can be seen from the table, in liver cirrhosis of HBV and HBV+HDV etiology there is thrombocytopenia, erythrocytopenia and leukopenia.

The study of platelet indices of the hemogram performed on a hematology analyzer showed that patients with cirrhosis have significant violations of the mean platelet volume (MPV), platelet distribution width (anisocytosis) (PDV) and platelet crit (PCT).
The performed hematological studies of platelets in patients with liver cirrhosis and chronic hepatitis of viral etiology showed significant disturbances in the number of blood cells of platelets in liver cirrhosis and unexpressed changes in chronic hepatitis.
Key words: chronic hepatitis, liver cirrhosis, platelet, blood test.

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The problem of viral hepatitis (especially B and C) is one of the most actual in infectology. Chronic viral hepatites can progress into liver cirrhosis which increases the risk to the health and life of patients [2; 5].

One of the characteristic complications of chronic hepatic disorders is a decrease in the platelet level, which is associated with several factors, including hypersplenism, impaired platelet synthesis in the liver and their increased destruction [4; 6]. Thrombocytopenia can contribute to an increased risk of bleeding, that significantly worsens the clinical condition of patients.

In liver cirrhosis, the normal functioning of hemostasis is disrupted, which leads to an increased risk of both thrombosis and bleeding. The study of platelet changes in such patients helps to understand the pathogenesis of these disorders and contributes to improved diagnostics and therapy. Understanding the mechanism of platelet changes in patients with chronic hepatitis and cirrhosis allows us to identify patients with a high risk of complications (for example, esophageal varices, gastric and intestinal bleeding). This requires regular monitoring of platelet counts and other hemostasis markers [1;3].

Modern treatment methods of chronic viral hepatites (e.g. antiviral therapy) and liver cirrhosis may affect platelet level. The researches in that area helps to develop the strategies to improve therapy and reduce the complications.

The changes in platelet level can also affect the prognosis of the disease and the quality of patients' life. Early detection of changes in the hemostasis system can help in more timely intervention and improvement of the condition of patients.

Thus, the study of platelet changes in patients with chronic hepatitis and liver cirrhosis of viral etiology is vital, since the study of these changes is important for improving the quality of diagnosis, treatment and prevention of complications in this category of patients.

Purpose of the study. To assess the dynamics of platelet levels in patients with chronic viral hepatitis and liver cirrhosis.

Materials and methods. Clinical studies were conducted in the infectious diseases department of the multidisciplinary clinic of the Tashkent Medical Academy from 2015 to 2024. The study included 140 patients with chronic diffuse liver diseases, including 70 patients with liver cirrhosis of viral etiology, 20 patients with liver cirrhosis of unknown etiology and 50 patients with chronic viral hepatitis of moderate activity.

When diagnosing liver cirrhosis and chronic hepatitis of viral etiology, anamnesis data (for example, indications of blood transfusion, treatment at the dentist, etc.), characteristic clinical syndromes (hemorrhagic, anemic, asthenoneurotic, icteric and others), as well as the results of laboratory and instrumental studies were taken into account. A mandatory condition for inclusion in the study was the presence of hepatitis virus markers determined by ELISA and PCR blood tests, with the detection of hepatitis B virus (HBV) DNA and hepatitis C virus (HCV) and D (HDV) RNA, as well as determination of their genotypes. In patients with chronic hepatitis and liver cirrhosis of viral etiology, the viral load exceeded 1,000,000 IU / ml.

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The diagnosis of liver cirrhosis and the degree of liver failure were established taking into account the recommendations of the World Health Organization (WHO, 2008) and the Child-Pugh classification based on diagnostic criteria.

Ultrasound examination (US), multi-layer computed tomography (MSCT) and liver fibroscan were performed to assess the degree of fibrosis in patients with liver cirrhosis.

Only patients with chronic hepatitis and liver cirrhosis who did not receive antiviral therapy were included in the study.All patients examined by us were divided into 6 groups: group I included 20 patients with decompensated cirrhosis of the liver of viral etiology HBV, class B according to the Child-Pugh classification, group II - 20 patients with decompensated cirrhosis of the liver of viral etiology HBV + HDV, class B according to the Child-Pugh classification, group III - 30 patients with decompensated cirrhosis of the liver of viral etiology HCV, class B according to the Child-Pugh classification, group IV - 20 patients with decompensated cirrhosis of unknown etiology, class B according to the Child-Pugh classification, group V - 25 patients with moderate chronic viral hepatitis B, group VI - 25 patients with moderate chronic viral hepatitis C.

Among the 140 patients included in the study, 78 were men (55.7%), 62 were women (44.2%). The age of patients ranged from 21 to 69 years, the average age was 48.2 ± 12.1 years. Among patients, 43.5% were people of working age.
The control group included 20 practically healthy individuals with no history of liver damage and fatty hepatosis, with negative results for hepatitis B and C markers.
Results and discussion. To study the vascular-platelet link of hemostasis, we performed a general analysis of peripheral blood with platelet counting, and also studied the adhesive and aggregation functions of platelets. The results showed that in groups with liver cirrhosis, there is a clear tendency to moderate thrombocytopenia. The average platelet count in patients of group I was 148 ± 25.8 x 10 ^ 9 /l, in group II - 146 ± 32.9 x 10 ^ 9 /l, and in group III the platelet count was significantly reduced and was 105 ± 33.5 x 10 ^ 9 /l. These data significantly differed from the control group indicator, which was 222 ± 21.21 x 10 ^ 9 /l. While in patients of group IV the number of platelets did not differ significantly from the control group and was 174 ± 48.6 x 10^9/l.
Thrombocytopenia was more often detected in patients with liver cirrhosis of HCV etiology than in liver cirrhosis of HBV and HBV+HDV etiology. Studies have shown that the main cause of thrombocytopenia in groups I and II was pancytopenia caused by hypersplenism in liver cirrhosis. This is confirmed by a decrease in the number of erythrocytes and leukocytes. As is known, with hypersplenism there is a delay and destruction of formed elements of the blood - erythrocytes, leukocytes and platelets - in the hypertrophied spleen.
The indicators of the red part of blood in patients were characterized by a moderate decrease in the number of erythrocytes in groups I and II, $2.88\pm0.16x10^{12}$/l and $2.83\pm0.21x10^{12}$/l respectively, which appeared to be significantly reduced compared to the control group. The number of erythrocytes in group III was $3.47\pm0.54x10^{12}$/l, while in the control group the number of erythrocytes was $4.22\pm0.27x10^{12}$/l, no significant differences were found. Severe anemia in patients with cirrhosis is more often diagnosed in patients who have suffered bleeding from esophageal varices.
Another indicator confirming hypersplenism in liver cirrhosis is a decrease in the number of leukocytes. Thus, in patients of the group I, the number of leukocytes averaged $3.54\pm0.32x10^{9}$/l, in the group II it was $3.49\pm0.19x10^{9}$/l, which is significantly lower than in

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the control. In group III, this indicator was within the normal range of 4.89±0.69x10^9/l. and in patients in the control group, the number of leukocytes was 5.95±1.01 x10^9/l (Table 1).

Table 1

Peripheral blood parameters in patients with cirrhosis

Groups	Control group (n=20)	I group, HBV liver cirrhosis (n=20)	II group, HBV+HDV liver cirrhosis (n=20)	III group, HCV liver cirrhosis (n=30)	IV group, liver cirrhosis of unclear etiology (n=20)
Platelets, x10^9/l	222 ± 21.21	148 ± 25.8*	146 ± 32.9*	105 ± 33.5**	174 ± 48.6
Erythrocytes, x10^{12}/l	4.22 ± 0.37	2.88 ± 0.16**	2.83 ± 0.21**	3.47 ± 0.54	3.53 ± 0.42
Leukocytes, x10^9/l	5.95 ± 1.01	3.54 ± 0.32*	3.49 ± 0.19*	4.89 ± 0.69	5.86 ± 1.05

Note: *-P<0.05, **-P<0.01 significant in relation to the control group.

As shown in the table, thrombocytopenia, erythrocytopenia and leukopenia are observed in liver cirrhosis of viral etiology HBV and HBV+HDV. These changes can be mainly explained by hypersplenism. At the same time, the phenomena of hypersplenism in liver cirrhosis of HCV etiology are manifested to a lesser extent than in liver cirrhosis of HBV and HBV+HDV etiology. In liver cirrhosis of unclear etiology, significant changes in the peripheral blood are not observed.

The results of the study of peripheral blood parameters of patients with chronic viral hepatitis of B- (group V) and C- (group VI) etiology showed that the number of platelets in these groups was within the normal range, which was 216±29.6x10^9/l and 187±32.9x10^9/l, respectively. There were no significant differences with the platelet count in the control group (222±21.21x10^9/l). The number of erythrocytes in these groups was slightly lower than in the control group: 3.47±0.53 x10^{12}/l and 3.46±0.35x10^{12}/l, respectively, and in the control group the number of erythrocytes was 4.22±0.27x10^{12}/l. A similar picture was observed when studying the number of leukocytes; in patients of groups V and VI they were within normal values – 6.06±1.99x10^9/l and 5.48±1.1x10^9/l, respectively (Table 2).

Table 2

Peripheral blood parameters in patients with chronic viral hepatitis

Groups	Control group (n=20)	V group, CVH B (n=25)	VI group, CVH C (n=25)
Platelets, x10^9/l	222 ± 21.21	216 ± 22.8	187 ± 32.9
Erythrocytes, x10^{12}/l	4.22 ± 0.37	3.47 ± 0.53	3.46 ± 0.35
Leukocytes, x10^9/l	5.95 ± 1.01	6.06 ± 1.99	5.48 ± 1.17

Note: *-P<0.05 significant in relation to the control group.

The study of platelet indices obtained using a hematology analyzer showed that patients with liver cirrhosis have significant changes in the mean platelet volume (MPV), platelet distribution width by volume (PDV) and platelet crit (PCT).

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In patients from group I, the mean platelet volume (MPV) was 13.27 ± 1.17 fl, in group II this figure was 13.57 ± 0.70 fl. In groups III and IV, the MPV values were 10.52 ± 0.76 fl and 10.48 ± 0.57 fl, respectively. In patients in the control group, the mean platelet volume (MPV) was 8.25 ± 0.64 fl. These data allow us to conclude that in liver cirrhosis, there is a reliable increase in the mean platelet volume, which indicates the predominance of young forms of platelets in the blood in response to their shortened lifespan. These changes are especially pronounced in patients with liver cirrhosis of HBV and HBV+HDV etiology.
The PDV index in group I was 29.3 ± 1.21%, in group II — 30.57 ± 0.82%, in group III — 22.91 ± 0.92%, and in group IV — 20.12 ± 0.68%. The platelet distribution width by volume in the control group was 12.43 ± 0.92%. This confirms that in liver cirrhosis, there is a reliable increase in the platelet distribution width by volume, which indicates pronounced platelet anisocytosis (Table 3).

Table 3

Platelet indices of hematological analyzer in patients with liver cirrhosis

Hemostatic profile	Control group (n=20)	I group, HBV liver cirrhosis (n=20)	II group, HBV+HDV liver cirrhosis (n=20)	III group, HCV liver cirrhosis (n=30)	IV group, liver cirrhosis of unclear etiology (n=20)
MPV, fl	8.25 ± 0.64	13.27±1.17***	13.57±0.70***	10.52± 0.76*	10.48± 0.57*
PDV, %	13.45± .51	29.30±1.21***	30.57±0.82***	22.91±0.92***	20.12±0.68***
PCT, %	0.28 ± 0.01	0.10±0.008***	0.10±0.008***	0.08±0.008***	0.16±0.01***

Note: *-P<0.05, ***-P<0.001 significant in relation to the control group.

When studying the platelet crit (PCT), it was found that it also significantly decreased in liver cirrhosis of viral etiology, especially in cirrhosis of HCV etiology. Thus, in the group I, PCT was 0.10±0.008%, in the group II it was 0.10±0.008% and in patients of the group III it was 0.08±0.008%. In patients of the group IV, PCT was 0.16 ± 0.01%. The platelet crit indicator in the control group was 0.28 ± 0.01%. The results of the PCT study confirm a decrease in the number of platelets in groups with liver cirrhosis of viral etiology, especially in liver cirrhosis of HCV etiology.
Thus, the hematological studies of platelets in patients with liver cirrhosis and chronic hepatitis of viral etiology revealed significant disturbances in the number of platelets in liver cirrhosis, while in chronic hepatitis the changes were less pronounced.

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