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CLINICAL AND LABORATORY FEATURES OF THE BLOOD COAGULATION SYSTEM IN PATIENTS WITH CIRRHOSIS OF THE LIVER ASSOCIATED WITH VIRAL HEPATITIS B, C AND D

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Abstract
Viral hepatitis is one of the most urgent problems of modern medicine and healthcare. According to WHO, more than 50 million people are infected with viral hepatitis every year [2, 6]. Of these, more than 10% develop chronic viral hepatitis (HCV). In 20% of cases, the outcome of HCV is cirrhosis, and in 5% - hepatocellular carcinoma (HCC) of the liver, which determines high levels of mortality and mortality [1, 2, 6]. Viral liver cirrhosis (CP) is one of the final stages of the morphogenesis of chronic viral hepatitis.

Introduction

The main difference between CP and HCG is the development of a diffuse inflammatory process with high fibroplastic activity and fibrosis of the organ. Cirrhotic changes developing in the liver progress and can cause the development of primary HCC at different times [2, 6]. In addition, viral hepatitis B, C and D are classified as "slow" infections, since 10-15 years pass from the moment of infection to the formation of CP and HCC. The frequency of annual occurrence of HCC in patients with manifest forms of CP is 3-6% [1, 3, 4, 7]. Every year in the world, about 2 million people infected with hemocontact hepatitis viruses die from liver cell failure and liver cell cancer. CP and HCC are included in the list of 130 main causes of morbidity and mortality of the population that require the development and maintenance of large-scale prevention and treatment programs [7-10]. In the Russian Federation, CP occurs in 1% of the population, which is more than 1 million people [1]. It should be noted that up to 40% of patients with CP do not make any complaints for a long time, they remain asymptomatic of the disease. However, as soon as complications develop (ascites, bleeding from varicose veins or encephalopathy), the symptoms of cirrhosis manifest [1, 3, 4, 7]. In the pathology of hemostasis in chronic liver diseases, an important factor is the imbalance between the coagulation and anticoagulation systems of the blood. On the one hand, the permanent inflammatory process in the liver directly affects the

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anticoagulant system (anti-thrombin III, protein C). Impaired functioning of hepatocytes, as well as an increase in cytokine levels, lead to activation of the system. On the other hand, these changes play an important role in the development of thrombotic complications, including in CP. Finally, the combination of thrombotic risk factors in the form of a decrease in the level of physiological anticoagulants with local inflammation forms a favorable background for thrombotic events in the hepatic microcirculatory bed, which subsequently contributes to the development of liver fibrosis [5]. Therefore, the study of the state of the blood coagulation system in this pathology is considered fundamentally important both for improving diagnosis and for predicting the likelihood of developing complications of the disease. The aim of the study was to study the clinical and laboratory manifestations and indicators of the blood coagulation system in patients with cirrhosis of the liver associated with viral hepatitis B, C and D.

Material and Methods

The object of the study was a group of patients (100 people, including 56 men, 44 women) with CP associated with viral tami B, C and D, who underwent clinical examination and treatment at the infectious diseases department of Clinical City Hospital No. 3 in Tomsk in 2004-2008. The age of the patients ranged from 29 to 65 years. Complaints were detected in all patients, and a detailed epidemiological history was collected. The viral etiology of CP was confirmed by the detection of markers of viral hepatitis by enzyme immunoassay and the determination of DNA and RNA viruses using polymerase chain reaction. The diagnosis of CP was confirmed by ultrasound examination (ultrasound) and static liver scintigraphy, and esophagogastroduodenoscopy was also performed according to the indications of the pits. The Child—Pugh scale was used to determine the stage of CP. All patients underwent a clinical and laboratory examination: a general blood and urine test, a biochemical blood test, and a coprogram. In addition, the blood coagulation system, including indicators of fibrinolytic, anticoagulant systems, coagulation and platelet hemostasis, was studied in 50 out of 100 patients with CP associated with viral hepatitis B, C and D. To register changes in the fibrinolytic system, the level of plasminogen in the blood serum was determined using chromogenic substrates. Similar methods have also been used in the study of the anticoagulant system (protein C, antithrombi III). The study of coagulation hemostasis parameters included the determination of the international normalized ratio (INR), the prothrombin index (PTI), partially activated thromboplastin time (APTT) using "Tech Plates" and "APTT tests", as well as the level of fibrinogen - the "Fibrinogen test". Platelet hemostasis was studied in two directions: the functional state of platelets was determined using an "Agrescreen test" and the quantitative content of the Willebrand factor. The level of blood coagulation markers was assessed using an orthophenoltroline test showing the content of soluble fibrin monomer complexes in blood serum. The studies were conducted in patients with CP in the subcompensation stage. In 25 out of 50 patients, CP was of a mixed nature (toxic and viral), and in 25 it was induced exclusively by hepatitis B, C and D viruses. Similar studies were also conducted on 15 healthy volunteers (control group, 7 men, 8 women). Statistical data processing was performed using the Statistic 7.0 for Win dows software package. Median Me, 25th and 75th percentiles (Me (25; 75)) were used to estimate the average values; nonparametric methods (Mann-Whitney test) were used to assess the level of statistical significance of p.

Results and Discussion:

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According to the data of the Tomsk City Clinical Hospital No. 3, 296 people with acute and 526 with chronic viral hepatitis B and C. During the period 2004-2008, 100 patients with cirrhosis of the liver were hospitalized in the infectious diseases department, which amounted to 12% of the total the number of patients with hepatitis and 19% of the number of patients with HCV. HCC diagnosis was established in 8 patients, which accounted for 1% of all forms of viral hepatitis and 1.5% of chronic forms. When collecting an epidemiological history, the main ways of infection of patients were identified: the parenteral pathway was the leading one (86 cases), 70 people noted the possibility of iatrogeny (blood transfusions, operations, dental services, endoscopic diagnostic and therapeutic medical manipulations). 5 patients out of all examined did not deny the possibility of a positive transmission pathway. Cases of infection of contact persons from the family environment of patients with acute and chronic viral hepatitis were observed in 15 people.; Infection associated with professional activity was determined in 10 cases. Out of 100 examined patients, 20 indicated the possibility of several ways of infection at the same time. Upon further examination, all patients were divided into three groups. Patients with CP associated exclusively with viral hepatitis B, C and D (40 (40%) people) formed group I. Patients referred to the infectious diseases department with a diagnosis of CP of mixed etiology (viral and toxic), who had markers of viral hepatitis and a history of alcohol abuse for several years (52 (52%) people) were included in group II. In group III, according to liver ultrasound and computed tomography, HCC was detected against the background of CP (8 (8%) people). Mixed infection prevailed in all patients with CP: a combination of HCV with HBV (34%) and HBV with HDV (3%). HBV infection was observed in 34%, HCV infection in 29% of cases (Fig. 1). It was found that out of 100 patients, 76% had concomitant pathology of the biliary tract, including 50% of patients with chronic opiorhosis. 56% of patients abused alcohol. All these factors aggravated the course of the main pathology. The smallest number of people with chronic opisthorchiasis (45%) and biliary tract diseases (62.5%) were found in group I. All representatives of the II group abused alcohol. An interesting fact is that 7 patients with HCC were diagnosed with concomitant chronic opisthorchiasis. At the initial examination, extrahepatic signs (erythema palmar, vascular asterisks) were found in all examined group I (Fig. 2). Icteric skin and sclera, hepatosplenomegaly were observed in 70% of patients; dilated subcutaneous veins of the anterior abdominal wall, signs of ascites — in 57%. Hemorrhagic syndrome was detected only in 26% of cases, peripheral edema — in 44%. Although there were no pronounced disorders of consciousness in this group, individual signs of hepatic encephalopathy (sleep inversion, lethargy, memory impairment, euphoria) were present in 17.5% of patients. The Child—Pugh criteria scale was used to assess the severity of CP: in 17.5% of patients of group I, the decompensated stage of CP was determined (10-11 points), in 55% — subcompensated (8-9 points), in 27.5% — compensated (6 points) (Fig. 3). The most common decompensation of CP it occurred with mixed infection with viruses B and C; B and D. Almost half (46%) of patients had complications during the course of the underlying disease: patients with anemia (20%), hepatic encephalopathy (13%), as well as with a combination of these complications (9%) prevailed. 1 patient had cachexia in combination with anemia, 1 patient had CP accompanied by sepsis. Complications were mainly observed in the groups with decompensated (63%) and subcompensated (37%) CP. In group I patients, complications were found only in 17.5% of cases in the form of anemia and in 15% in the form of nocturnal encephalopathy, mainly in mixed infections B and C; B and D. In group II, complications were detected in almost half of the patients, of whom 19% were diagnosed with anemia. Hepatic

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encephalopathy, cachexia and anemia were observed in 7 patients of group III. The study of biochemical parameters in patients with CP revealed the following changes: an increase in the level of bilirubin, transaminases, and thymol samples (Table 1). Glucose, creatinine, and motility remained within normal limits, while the content of total protein was reduced. Patients of group I had higher protein values and lower bilirubin values. This explains the greater number of patients in group II with icteric skin and sclera, edematous ascitic syndrome, encephalopathy. The indicators of the biochemical blood test do not contradict the obtained clinical data. Thus, with a deficiency of natural anticoagulants (protein C, antithrombin III), there is an increase in markers of the blood coagulation system (RFMC), as well as activation of the vascular platelet link of hemostasis due to an excess of Willebrand factor. This indicates a risk of thrombosis, although procoagulation tests (PTI, APTT) show a risk of bleeding. Such a divergence of procoagulation (PTI, APTT) and paracoagulation tests is a laboratory sign of consumption coagulopathy.

Conclusions

1.According to the infectious diseases hospital in Tomsk, cirrhosis of the liver associated with viral hepatitis B, C and D amounted to 19% of all chronic forms of viral hepatitis, whereas hepatocellular carcinoma — 1.5%.

2. In patients with cirrhosis of the liver associated with viral hepatitis B, C and D, combined liver infection with HCV and HBV viruses (34%) occurred with the same frequency as HBV infection (34%)

3. Alcohol abuse exacerbates the clinical symptoms, course and laboratory (biochemical) indicators of the underlying disease.

4. In patients with virus-induced cirrhosis of the liver, according to the detailed coagulogram, the development of consumption coagulopathy was noted.

5. There are statistically significant differences in the indicators of anticoagulants (antithrombin III, protein C) and fibrinolytic (plasminogen) the system depends on the etiology of the CPU.

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