Surgical Treatment of Picevodogasteric Bleeding in Patients with Liver Cirrhosis

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Abstract: A little more than half a century has passed since the fundamental ideas about the morphological types of such liver lesions as hepatitis, hepatois, cirrhosis were formed (Bluger A.F., Novitsky J.N., 1984). Subsequent morphological studies have established that LC- it is the result of a pathological reconstruction of the lobular structure of the organ. From a clinical point of view, cirrhosis can be defined as a progressive (or slowly progressive) diffuse chronic polyetiologic liver disease. This is the final stage of adversely occurring forms of chronic hepatitis, due to obstruction of the outflow of bile or blood from the liver or genetically determined metabolic defects. The disease is characterized by a significant decrease in the mass of functioning liver cells, a pronounced fibrosing reaction, a restructuring of the parenchyma and the vasculature of the liver (Loginov A.S., 1987).

According to WHO, mortality from cirrhosis ranks 8th and ranges from 14 to 30 per 100,000 population in different countries. LaVecchiaet al. (1994) present a wider range of mortality from this disease from 3 to 40 people per 100,000 population based on 35 years of mortality studies in 38 countries on various continents. According to Dufour M.S. et al. (1993), about 900,000 people in the United States suffer from this disease. Moreover, the share of this disease among the causes of death has doubled in USA, France, Sweden, Canada. In the US, among the causes of death, CP has moved from the 7th place to 4th place. The structure of the etiological factors leading to the development of cirrhosis has quite pronounced differences in different regions of the world. So, in Europe, North America in 72-92% of cases of cirrhosis of the liver of alcoholic origin (Dufour M.C. et al., 1993, ZamanA. Et al., 1999), while in the states of South America, South and East Asia is etiologically dominated by viral lesions of the liver, liver schistosomiasis, malaria, exposure to hepatotropic poisons. In addition to liver cirrhosis in the formation of portal hypertension in these countries, a significant role is played by liver fibrosis, extrahepatic changes in the portal vein, the share of which in the structure of morbidity reaches 30-40% (Ahmad N. Et al.1999; Bhandarkar P.V. et al., 1999; Dhiman R.K. et al., 2002; Urbani C. Etal., 2002).

For the countries of Central Asia, LC can be considered a marginal pathology associated with epidemiological ill-health in relation to viral hepatitis, exposure to adverse environmental factors, some nutritional features (Karimov Sh.I. et al., 1987; Shamirzaev N.Kh. et al., 1989, Nazyrov F.G., 1999). In Kazakhstan, over 25 years, according to G.N., Andreeva et al. (1994), the proportion of patients with cirrhosis among those admitted with acute abdominal diseases increased 7 times from 0.17 to 1.2%. Chronic diffuse liver diseases are detected in 11-12% of residents of the Andijan region of Uzbekistan, examined on a random basis, of which 3% have already been diagnosed with latent cirrhosis (Dzhumabaev U.T. et al., 1996).
Of particular concern is the epidemiological situation of viral hepatitis in the region among children (A.I. Kamilov, Sh.T. Azimov, 2001). The share of chronic hepatitis of viral etiology in Uzbekistan reaches 95% in the total amount of liver pathology in children (F.I. Inoyatova 1999; H.Ya. Karimov...
and F.A. Ariphkhodzhaeva, 1999). According to D.V. Ruzmetova (2002) in 24.8% of "practically healthy children" of school and preschool age in Tashkent, echographic changes in the hepatobiliary system were detected, and in 9.4% of the examined, echocatological changes in the liver were recorded, corresponding to the echo picture of chronic hepatitis. Moreover, 45.5% of them had HBV markers, 11.5% had HCV markers, and 3.3% had a combination of HBV and HCV markers.

It is known that it is the prevalence of HBV markers that largely determines the regional features of the etiology of liver cirrhosis. In various countries of the world it is uneven, ranging from 4 to 95% of the population. It is customary to distinguish three degrees of prevalence of HBV markers in the population: high (70-95%), with a carrier frequency of 8-20%; intermediate (22-55%) (carrier - 2-7%) and low (4-6%), with virus carrier less than 2% (Mayer K.P., 1999; Sherlock S., Dooley D., 1999). In general, the countries of the Central Asian region are characterized by intermediate endemicity, although certain areas of our region can claim other high rates of endemicity with all the ensuing consequences.

Previously, it was generally recognized that cirrhosis is most often diagnosed in people over 40 years of age (Agzamkhodzhaev C.M., 1975). However, due to a number of unfavorable circumstances characteristic of the Central Dzinya region, the rapid formation of liver cirrhosis within 0.5-1.5 years significantly changed the age composition of patients towards rejuvenation (Nazirov F.G., 1997, Aliev M.M., 1999). Patients with cirrhosis much younger than 40 years old are admitted to hospitals already with distinct manifestations of complications of PH (Abdurakhmanov V.M., 1997). Moreover, the features of the development and progression of a particular complication and the life expectancy of patients are largely determined by the etiological factor, the stage at which the diagnosis was made, the degree of activity of the process, the adequacy of the treatment. According to long-term observations of 501 patients, the average life expectancy of a patient with cirrhosis is 8.56 years (Khazanov A.I., 1997). Taking the activity of the pathological process as a basis, the same author obtained the following indicators of the average life expectancy of patients with cirrhosis: with a subacute course of 5.5 months, with a rapidly progressing - 2.69 years, with a slowly progressing - 7.53, with a sluggish course - 10.3 and with a latent course - 12.25 years. More than 80% of all diagnosed cirrhosis are active (Loginov A.S., Blok Yu.E., 1987).

According to T.G. Zhukova 1983; IN AND Fomicheva, 1984, and others. It is especially important to identify cirrhosis at its early stages, because the prognosis of the disease depends on the time of diagnosis. At the same time, the diagnosis of the early stages of cirrhosis is very difficult, since the latency of their course often leads to a belated request for medical help, when there are already clear signs of the activity of the pathological process in the liver, signs of liver failure or PH (Mikhailovskaya R.P., 1983). The latter develops as a result of compression of the postsinusoidal microvasculature of the liver by regenerated nodes, which disrupts the drainage function of the hepatic veins, contributing to the development of an intrahepatic postsinusoidal block of portal blood flow. In addition, as a result of the replacement of the liver parenchyma with connective tissue, there is a narrowing and obliteration of the intrahepatic portal vein branches with the development of an intrahepatic presinusoidal block of the portal circulation. Here it is appropriate to clarify that, in accordance with modern concepts, an obstruction to portal blood flow can be localized at different levels of the microvasculature of the liver, depending on the etiology of the disease and the stage of the pathological process in the liver. Thus, viral hepatitis in the chronic stage affects the presinusoidal and sinusoidal sections with a gradual increase in the latter as cirrhosis develops (VanLeeuwenD.J. et al., 1991). On the contrary, hepatitis caused by alcohol abuse affects the sinusoidal and postsinusoidal sections of the liver microcirculation (Maer K., 1999). A detailed study of the level of block formation inside the liver in relation to the sinusoidal channel can determine many key positions of the therapeutic effect, primarily the prospects for operational decompression of the portal system using portosystemic shunting (Morgunov G.A., 2002).

A certain role in the development of hypertension is played by portal-hepatic anastomoses, newly formed shunts between the branches of the hepatic artery and the portal vein, which lead to an increase in hydrostatic pressure in the portal channel (Patsiora M.D., 1974, 1984).
In the last decade, the active-contrast properties of stellate cells and their role in increasing the tone of intrahepatic vessels (a functional component of portal hypertension) in liver cirrhosis have been actively studied (Pinzani M. et al., 1992).

Numerous options for substantiating the pathogenesis of PH can be conditionally reduced to a double effect on the pressure in the portal vein of the intensity of blood flow through it and vascular intrahepatic resistance, which led to the creation of two theories of the pathogenesis of PH (BenoitJ.N., GrangerD.N., 1986). The first of of them, called "backwardflow", explains the increase in portal pressure by an increase in vascular resistance caused by a decrease in the overall radius of the functioning portal tracts in the liver. According to the second theory, called "forwardflow", the increase in pressure in the portal system is due to excess blood flow.

With the progression of PH and diffuse compaction of the liver tissue, an increasing intrahepatic collateral shunting is noted, the specific blood flow to the liver decreases, the lability of the hepatic blood flow during breathing increases, which aggravates the degree of hepatic hypoxia and activates the pathological developing in it.


Regional hemodynamic disorders developing in PH are accompanied by the development of porto- caval and porto-hepatic anastomoses, the anatomical variants of which have been studied in detail (Sherlock Sh., Schuli J., 1999; Arakawa M. et al., 2002; Cottam D.R. et al., 2002). The reasons for the predominance of one or another variant of shunting are probably associated with the individual characteristics of the formation of the venous system. Portocaval anastomoses of the gastroesophageal region are of the greatest clinical importance as the cause and main source of bleeding in patients with portal hypertension syndrome.

With cirrhosis of the liver, EVIL is detected in 50-70% of patients (Cales R., Pascal J.P., 1988). According to S. Sherlock, J. Dooley (1999), this complication develops in 60% of patients with decompensated and 30% of compensated liver cirrhosis. More detailed data are presented by A. Zaman et al. (1999) - in 68% of patients with cirrhosis of the liver, varicose veins of the esophagus were detected, and in 15% - varicose veins of the stomach. O.G. Kotenko (1999) reports that the predominance of gastric varicose veins was detected in 57.10% of patients with decompensated liver cirrhosis, while isolated esophageal varicose veins predominated in patients with compensated liver cirrhosis.

Sh.Kh. Khashimov (2000), studying the severity of varicose veins in patients admitted to the hospital with esophageal-gastric bleeding, found that with the deterioration of the functional state of the liver, the diameter and length of varicose veins of the esophagus decreased. Thus, 22.2% of patients of functional class "A", 17.9% - class "B", and only 13.3% - class "C" had total esophageal vein dilatation. This agrees with the data of K.I. Bulanova et al. (1995), who calculated for 90 patients with cirrhosis of the liver, which in class A portal pressure was 435.1 mm w.c., B - 335.7 mm w.c., C - 283.7 mm w.c. Research by K.Ueno et al. (1996) also showed that patients with large varicose veins had a higher mean esophageal venous pressure than those with small varices.

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