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## Risk factors and complications during operations on abdominal organs in patients with cirrhosis of the liver

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### ABSTRACT

This review presents an analysis of risk factors and characteristic complications of extrahepatic abdominal surgery in patients with cirrhosis of the liver. Portal hypertension, edematous-ascitic syndrome, spontaneous bacterial peritonitis, hepatorenal syndrome, and hepatic encephalopathy are poor predictors. Consideration and analysis of which will predict and prevent complications, as well as improve the effectiveness and results of treatment. This review represents a set of practical algorithms whose basis is the most effective methods to diagnose, treat, and prevent complications of cirrhosis.

### Keywords:

liver cirrhosis, surgery on the abdominal cavity

For physicians supervising patients with severe liver disease, especially in the presence of signs of complications of cirrhosis of the liver (CP), it is important to rely on diagnostic and treatment algorithms from the point of view of evidence-based medicine, i.e. use only methods whose effectiveness has been proven in randomized trials. Despite the fact that patients with cirrhosis of the liver, as a rule, fall into the field of view of surgeons due to bleeding from varicose veins of the esophagus, or when hepatocellular carcinoma is detected, the presence of another, especially emergency surgical disease may raise the question of the need for its surgical intervention. . At the same time, liver dysfunction, multi-organ hemodynamic disorders characteristic of portal hypertension (PG), leading to complications characteristic of it, as well as a number of other adverse circumstances, do not always allow it to be solved unambiguously, which makes the

problem relevant. Currently, to determine the tactics of treatment of a patient with CP, including when deciding on conservative and surgical manuals, hepatologists use prognostic scales and indices. The indices given in this review are the most frequent in application [1,3].

Scale assessment of liver function before surgery.

### *Шкала Child-Turcotte-Pugh*

The "gold" standard for assessing the severity of the disease, the risk of death during surgical operations (in the original - PG surgery) and prognosis in patients with CP was the Child-Turcotte-Pugh scale (CTP) [6]. Its original version, described in 1964 (Child-Turcotte scale), included two quantitative (serum bilirubin and albumin levels) and three qualitative (ascites, encephalopathy and nutritional status) indicators (Table 1).

**Table 1.**  
**Шкала Child-Turcotte**

Index	Deviation of indicators from the norm, points		
	1	2	3
Bilirubin, $\mu\text{mol/L}$	<34	34-51	>51
Albumin, g/l	>35	30-35	<30
Ascites	No	Controlled	Resistant
Encephalopathy	No	Minimum	Coma
Nutritional status	Good	Satisfactory	Bad

*Note.* A: 5-8 points (low operational risk); B: 9-11 points (medium operational risk); C: 12-15 points (high operational risk).

In the subsequent modified version (Child-Pugh scale), the nutritional status was replaced by prothrombin time, and the lower albumin level was reduced to 28 g / l (Table 2).

**Table 2.**  
**Шкала Child-Pugh**

Index	Deviation of indicators from the norm, points		
	1	2	3
Bilirubin, $\mu\text{mol/L}$	<34	34-51	>51
Albumin, g/l	>35	28-35	<28
Ascites	No	Controlled	Resistant
Encephalopathy	No	Minimum	Coma
Prothrombin	<4	4-6	>6

*Note.* A: 5-6 points (low operational risk); B: 7-9 points (medium operational risk); C: 10-15 points (high operational risk)

The main disadvantages of the TDB scale are the empirical selection of its main components, the arbitrary use of threshold values for quantitative indicators, the possible ambiguous interpretation of qualitative variables, as well as ignoring other important critical factors, for example, the severity of renal dysfunction. However, it is widely used to predict treatment outcomes, as well as in retrospective and randomized clinical trials. In addition, the TDB scale, together with other indicators, in particular the assessment of the patient's condition according to the American Association of Anaesthetists (ASA) and the

determination of preoperative serum sodium levels, is convenient "at the patient's bedside" as a simple descriptive indicator of surgical risk, especially in emergency situations [13].

### **The role of portal hypertension in the development of postoperative complications in patients with cirrhosis of the liver**

#### **Methods for assessing portal hypertension**

Many complications of CP are the result of an increase in portal pressure, the degree of which correlates both with the severity of hepatocellular insufficiency and with a violation of the histological structure of the

liver. The most accurate reflection of the severity of PG is the hepatic venous pressure gradient (GPVD), which is the difference between the wedged and free hepatic venous pressure. It has been established that its normal indicators are in the range of 1-5 mm Hg, and a level exceeding 10 mm Hg indicates the presence of pronounced PG, despite the clinical manifestations [4]. Unfortunately, the direct measurement of GPVD in most observations cannot be applied in normal clinical practice, so alternative methods for assessing the severity of PG are currently being considered.

As one of them, it is proposed to use various Doppler sonographic indices, which are calculated based on the parameters of portal, hepatic and splenic arterial blood flow. It has been shown that the resistance index in the hepatic artery (the ratio of the difference in the maximum systolic and final diastolic velocity of blood flow to the maximum systolic velocity of blood flow) and the pulsation index in the hepatic artery (the ratio of the difference in the maximum systolic velocity of blood flow and the minimum systolic velocity of blood flow to the average speed during the cardiac cycle), as well as the hepatic vascular index (the ratio of the average linear blood flow rate in portal vein to pulsation index in the hepatic artery) correlate with the magnitude of portal pressure measured during surgery [17].

#### *Multi-organ hemodynamic disorders in cirrhosis of the liver*

PG not only leads to the development of well-known and common complications, such as bleeding from esophageal varicose veins and ascites, but also underlies the complex circulatory disorders characteristic of CP. It is accompanied by a hyperdynamic circulatory status, which is manifested by an increase in cardiac output, a decrease in peripheral vascular resistance and the opening of arteriovenous communications. Splanchnic fullness due to vasodilation and intra-organ venous stagnation reduces the effective volume of blood, contributing to arterial hypotension and the development of functional insufficiency of almost all organs [2].

Despite the increase in portal venous inflow,

as a result of the formation of collateral circulation, the portal blood flow supplying the liver decreases [15], and the constancy of hepatic perfusion is maintained due to the so-called "hepatic arterial buffer response". This phenomenon, first described in 1981 by W. Lauth has been identified both under physiological conditions and in various pathological conditions, including CP. It allows you to maintain the delivery of oxygen to the liver, providing protection for its structure and function [7].

Developing *cirrhotic cardiomyopathy* is characterized by a decrease in the contractility of the heart with left ventricular systolic and diastolic dysfunction, as well as an extension of the Q-T interval on the electrocardiogram. Its morphological basis is cardiac hypertrophy, focal fibrosis and subendothelial edema. Due to a decrease in afterload due to a decrease in peripheral vascular resistance and increased extensibility left ventricular insufficiency in patients with cirrhotic cardiomyopathy can occur latently and manifest itself only with physical exertion or treatment with vasoconstrictors. Left ventricular diastolic dysfunction is accompanied by a violation of the ability of the myocardium to accept a sufficient volume of blood by the left ventricle during diastole, despite normal final diastolic pressure and a high stroke volume of blood. [16].

Left ventricular systolic dysfunction and severe systemic vasodilation lead to vasoconstriction and a decrease in renal blood flow, reducing the value of glomerular filtration, which underlies *hepatorenal syndrome*. The development of hyperdynamic circulatory status activates renin-angiotensin-aldosterone and sympathetic nervous system, inducing the production of vasopressin. Secondary hyperaldosteronism and tubular hypersensitivity to aldosterone increase reabsorption of sodium in the distal parts of the nephron, while the sympathetic nervous system stimulates its reabsorption in the proximal tubules and henle loop. Angiotensin II causes a spasm of predominantly efferent arterioles and significantly reduces glomerular filtration. This contributes to a further decrease

in sodium release, even with stable blood pressure.

The criteria for diagnosing hepatorenal syndrome in patients with CP are a serum creatinine level of more than 133  $\mu\text{mol} / \text{l}$  without normalizing its content after at least 2 days of diuretic withdrawal and albumin administration, no shock and data on the use of nephrotoxic drugs, exclusion of any parenchymal kidney diseases manifested by proteinuria, microhematuria and / or corresponding ultrasonic picture. Doppler sonography with the study of the renal artery resistance index can allow to assess the severity of hemodynamic disorders to the clinical signs of hepatorenal syndrome [10].

*Porto-pulmonary hypertension* is defined as pulmonary arterial hypertension associated with liver disease and PG. It is characterized by an increase in pressure in the pulmonary artery of more than 25 mm Hg, an increase in pulmonary vascular resistance of more than 240 dipgsm-5 and a magnitude of the pressure of jamming of pulmonary capillaries below 15 mm Hg. The first and most accurate signs of portopulmonary hypertension can be detected by Doppler echocardiography. One of the they are the disappearance of the presystolic flow in the pulmonary artery. In addition, the form and duration of pulmonary regurgitation will be typical. An increase in pressure in the pulmonary artery leads to a prolongation of the period of isovolumic contraction and relaxation of the right ventricle, a shortening of the acceleration time and the period of expulsion of the flow in the exit tract of the right ventricle and pulmonary artery. [11].

Specific signs of *hepato-pulmonary syndrome* are arterial hypoxemia and intrapulmonary vascular dilatation. Hypoxemia occurs as a result of a low ventilation-perfusion ratio in the case of capillary expansion (ventilation with excessive perfusion) and anatomical shunting in the presence of direct arteriovenous anastomoses (perfusion without ventilation). Hepatopulmonary syndrome is characterized by a decrease in alveolar  $\text{pO}_2$  to less than 80 mmHg. and an increase in the alveolar-arterial oxygen gradient to a level of more than 15 mm Hg. Like portopulmonary hypertension,

hepatopulmonary syndrome most often develops in patients with CP with PG. The relationship between the severity of impaired liver function and the degree of hypoxemia is weak, but the risk is more significant in patients of class TDB C.

With PG, the gastric mucosa is morphologically and functionally different from normal. Its increased susceptibility to damaging agents, such as non-steroidal anti-inflammatory drugs, alcohol, bile acids, etc., as well as acid-peptic factor, contributes to the development of insidious, difficult to control bleeding. Currently, *gastropathy associated with PG* is considered as a unique pathological condition, different from other forms of gastritis, in which macroscopic disorders of the gastric mucosa are combined with the expansion of the vessels located in it without any significant histological signs of inflammation. The changes identified during the endoscopic examination are in the form of small polygonal areas of pink color, slightly protruding to the center and surrounded by the whitish-yellow contour of the mosaic type is defined as light, the presence of flat red spots in the center of the pink areola, not reaching its outer contour, as moderate, and in the case of diffuse red coloration of the areola - as pronounced. Hemorrhagic manifestations on the gastric mucosa in patients with PG and everything that is associated with them are unfavorable prognostic signs. Hemorrhagic manifestations are directly dependent on the severity of gastropathy, the magnitude of GPVD, the degree of liver dysfunction and inversely depending on the severity of gastric varices. It should be noted that similar changes in the mucous membrane in patients with PG can also be observed in the small and large intestine [5].

#### *Coagulopathy in cirrhosis of the liver*

The traditional notion that patients with CP a priori have an increased risk of hemorrhagic complications is not confirmed by the results of recent studies. Of course, their characteristic thrombocytopenia, a decrease in the level of blood coagulation factors II, V, VII, IX, X, XI and hyperfibrinolysis contribute to hypocoagulation, which is confirmed by

routine laboratory tests, but it is usually compensated by adaptive changes in the hemostasis system. First, a decrease in the number and functional activity of platelets is accompanied by a significant increase in the level of thromboplastic plasma factor - von Willebrand factor, which is synthesized by activated endothelial cells as a result of hemodynamic changes and exposure to various humoral substances in PG. Secondly, a decrease in the level of blood coagulation factors is offset by a lack of natural anticoagulant proteins C, S and antithrombin, as well as significant resistance to the inhibitory effect of thrombomodulin. Finally, hyperfibrinolysis can be balanced by a concomitant decrease in profibrinolytics. At the same time, this balanced state of hemostasis in patients with CP is unstable and easily disturbed when any complications occur, for example, uncontrolled bleeding from large vessels during surgery, infection, renal failure, etc., which leads to a shift in balance towards either hypo- or hypercoagulation [12].

It was found that prophylactic transfusion of blood products, in particular freshly frozen plasma, not only does not reduce intraoperative blood loss in patients with CP, but can also contribute to it due to an increase in the volume of circulating blood. In this regard, there is increasing evidence that the main role in the development of bleeding during surgical interventions in such patients is played not so much by hemostasis defects as by hyperdynamic circulatory status and the formation of a powerful collateral vascular bed due to PG [14].

Refractory ascites, SBP, GDS, severe hyponatremia are the most frequent complications of the natural course of CP, which significantly worsen the prognosis in the patient. The medical and social significance of ascites is evident in view of the high probability of death in patients with CP and the listed complications, so the basis for the development of national guidelines for the management of patients with CP was based on the principles of evidence-based medicine, describing the most effective methods of diagnosis and treatment of developing complications. The formation of

practical recommendations for doctors has undergone many changes over the past few years, and the data, given in this article, summarize the principles of differential diagnosis, the rules for prescribing therapy, the criteria for the effectiveness of treatment and issues related to the ineffectiveness of conservative measures. [4,5].

Thus, reducing the risk, as well as minimizing postoperative complications, when performing extrahepatic abdominal operations in patients with cirrhosis of the liver, is possible through the qualitative preparation of patients with CP for surgical intervention. The risk can be significantly reduced due to the competent treatment of ascites. Sometimes conservative therapy is sufficient, however, in case of its ineffectiveness, it is possible to use volumetric paracentesis with albumin infusion and TIPS [9]. With conventional drainage of the abdominal cavity, the threat of infection of ascitic fluid increases. In this regard, it is proposed either to avoid drains or to use sterile aspiration systems [8].

Thus, the literature indicates that the main critical factors affecting the development of complications and mortality after surgery in the presence of CP are the severity of liver failure, as well as the severity of complications of PG. It was found that class C on the TDB scale with a high degree of probability makes it possible to predict the risk of an unfavorable outcome. Careful selection of patients and preoperative correction of these disorders using modern techniques will contribute to both increasing the effectiveness of treatment and minimizing the threat of possible complications. In particular, the use of TIPS in patients with CP with PG before surgical interventions on the abdominal organs looks promising. Reduction of portal pressure will reduce the risk of bleeding, reduce ascites and correct complex multi-organ hemodynamic disorders. Additional aggravating circumstances are the type and urgency of extrahepatic abdominal operations. Here the further development of minimally invasive is optimal. technologies and the maximum possible limitation of the scope of surgical intervention in patients with decompensated

CP in emergency situations.

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