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## SURGICAL TREATMENT OF BLEEDING FROM VARICOSE VEINS OF THE STOMACH AND ESOPHAGUS CAUSED BY PORTAL HYPERTENSION IN CHILDREN

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**Summary:** Treatment of portal hypertension syndrome and bleeding from varicose veins of the esophagus and stomach includes a therapeutic and preventive complex of measures: drug therapy, insertion of a Blackmore probe, endoscopic ligation and sclerotherapy, and, if absolutely necessary, surgical treatment. Timely diagnosis makes it possible to identify the disease in the compensated and subcompensated stages, which greatly facilitates treatment and reduces mortality.

**Keywords:** childhood, esophagus, stomach, bleeding, portal hypertension.

### Relevance

At present, the diagnosis and treatment of bleeding from varicose veins of the stomach and esophagus remain one of the most urgent problems in pediatric surgery [2,7]. This is due to the fact that the treatment methods for such patients are based on the topographic and anatomical specificity of the venous collaterals of the stomach and esophagus. In the literature, most authors believe that bleeding from varicose veins of the stomach and esophagus in children occurs in 95–98% of cases as a result of portal hypertension syndrome.

Currently, medical efforts are aimed at preventing the progressive development of portal hypertension in children and at finding therapeutic and surgical methods capable of radically reducing pressure in the portal venous system, thereby preventing the risk of bleeding from varicose veins of the esophagus and stomach [1,5]. Another approach to the prevention of gastroesophageal bleeding of portal origin is the use of local endoscopic therapy to prevent rupture of varicose veins.

A group of authors analyzed domestic and foreign studies in order to develop general approaches to the treatment of bleeding from varicose veins of the esophagus and stomach in children with this disease. The recommendations focus on unresolved problems and future prospects opened up by new research data [3].

When diagnosing varicose veins of the esophagus, they are detected in 30–40% of children with the compensated stage and in 60% of cases in the decompensated stage of the disease. The frequency of bleeding from esophageal varices in children averages 4% per year. In patients with medium and large vessels, the risk increases to 15% [6,8].

Despite progress made in recent decades, bleeding from the esophagus and stomach in portal hypertension is associated with 10–20% mortality within six weeks.

Recommendations for the diagnosis and treatment of patients with bleeding from varicose veins of the esophagus and stomach contain practical instructions for the management of such patients in hospitals of different levels. These recommendations should be reviewed regularly in light of new data in this field [7,9].

## Pathophysiology

In portal hypertension syndrome, blood pressure in the portal vein rises (up to 200 mm of water column, normally), leading to the development of the syndrome and an increased inflow of arterial blood to the unpaired abdominal organs and the portal vein (v. portae), depending on the imbalance between inflow and resistance to blood flow. The resistance (block) may be prehepatic, intrahepatic, or posthepatic.

Development of portal hypertension can result from developmental defects of the portal vein (venous atresia, obliteration) or its purulent processes in the abdominal cavity (umbilical sepsis), compression by a nearby tumor, and other extrahepatic blockages. Usually, the intrahepatic type of portal hypertension is more common (up to 80–90%) and is associated with liver cirrhosis. Obstruction due to hepatic vein thrombophlebitis (Chiari syndrome), occlusion of the inferior vena cava at the hepatic outflow level (Budd–Chiari syndrome), or congenital defects of the inferior vena cava belong to the suprahepatic blocks [4,6].

Patients with mixed cirrhotic block often have portal vein thrombosis. Increased pressure in the portal vein in idiopathic portal hypertension may arise from rapid blood inflow through an arteriovenous fistula.

Thus, the appearance of a barrier preventing the normal outflow of blood from the unpaired abdominal organs into the systemic venous system leads to portal hypertension, which in turn causes marked development (varicose enlargement) of natural portocaval anastomoses, through which blood is shunted.

## Diagnostic Methods

Diagnostic examination for portal hypertension involves determining the presence, size, and prevalence of esophageal and gastric varices using **esophagogastroduodenofibrosopy**, radiological contrast studies, and assessment of hepatic blood flow and its disorders to determine the cause and nature of the block.

## Treatment

The treatment of portal hypertension is closely related to the type of block, disease stage, and degree of compensation. The main course of treatment is aimed at eliminating the cause of the obstruction, restoring liver function, and preventing possible complications [3,6].

## Aim of the Study

To improve the effectiveness of treatment of bleeding from varicose veins of the stomach and esophagus in children.

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## Materials and Methods

The management strategy for bleeding from varicose veins of the stomach and esophagus in children was studied based on the analysis of treatment results of **45 children aged 1 to 18 years**, treated in the Department of Pediatric Surgery.

## Results

All patients were admitted to the hospital during or after a bleeding episode. Intensive hemostatic therapy and conservative methods were applied.

Endoscopy played a major role in diagnosis and treatment: 14 (31.1%) patients underwent endoscopic examination. After standard hemostatic therapy, in 20 (44.4%) patients bleeding stopped; in 7 (15.5%) older children, a Blackmore probe was inserted to stop bleeding from the esophageal veins; and in 4 (8.8%) patients with extrahepatic portal hypertension, an operation connecting subcutaneous veins of the esophagus and stomach was performed using the **Pasyora-Petrova** method.

In 2 patients with intrahepatic type, bleeding stopped during treatment, and gastric veins were sutured using the **Tanner-Bairov** method.

Among the many proposed operations for diagnosis and treatment of gastroesophageal bleeding in children with portal hypertension, in addition to portocaval anastomoses, procedures aimed at separating the portal system directly through the esophagus and gastric varices were used.

The most effective operation according to foreign researchers is the **Sugiura-Futagawa** procedure (1973), which requires both transthoracic and transabdominal approaches, including extensive devascularization of the esophagus with its transection, splenectomy, and pyloroplasty.

## Method of Suturing Gastric and Esophageal Varicose Veins

A midline laparotomy was performed. Near the anterior wall of the stomach up to the cardia, capron sutures were placed, and the stomach wall was incised 10–12 cm. The incision extended toward the lesser curvature. After opening the stomach, a retractor was inserted to lift the upper part of the anterior wall. The mucosa was dissected near the esophageal opening, exposing varicose veins of the cardial part (usually 3–5 trunks). Suturing of varicose veins began at the lesser curvature using separate interrupted stitches on the most prominent veins

## Study Findings

Among the 45 children (ages 1–18), the following pathologies were found: cavernous transformation (38 cases), atresia (5), splenic vein thrombosis (3), and umbilical vein and aortic anomalies (2).

According to **Sarin's (2001)** classification of varices before and after surgery

Type 1 – gastroesophageal veins along the lesser curvature

Type 2 – along the greater curvature

Type 3 – isolated gastric varices

Type 4 – ectopic varices (stomach and duodenum).

Endoscopic ultrasonography (Olympus UM-160) was performed according to standard methods. A mini-radial ultrasound probe was positioned in the esophagus. It was determined that:

No bleeding risk when the distance from the mucosa to the vein is  $\geq 0.9$  mm

High bleeding risk when mucosal thickness is 0.5–0.7 mm

Very high risk when thickness <0.5 mm with angioectasia present.

A **new non-invasive method** was developed to measure pressure in esophageal veins using endoscopic ultrasonography and the **Waldmann apparatus**. A balloon-tipped probe was used to compress esophageal vessels while Doppler monitoring determined cessation of blood flow, thus allowing direct measurement of venous pressure.

In 20 patients with grade III varices, portal gastropathy was a stable sign of increased portal pressure (>200 mm H<sub>2</sub>O). With vessel diameters and pressures exceeding 300 mm H<sub>2</sub>O, risk of rebleeding increased.

Depending on findings, **endoscopic sclerotherapy** was used in 24 cases, **endoscopic ligation** in 7, and a combination of both in 4 patients with type II varices.

Rebleeding prevention was required in 30 patients. In total, **41 patients underwent surgery:**

2 – mesentericocaval shunting

13 – H-shaped splenorenal

7 – distal splenorenal

2 – mesentericocaval anastomosis

and **Sugiura's operation** involving the left branch of the portal vein.

## Conclusions

In conclusion, endoscopic diagnosis and treatment of varicose veins of the stomach and esophagus remain leading methods in the management of portal hypertension in children. Considering their effectiveness in stopping active bleeding and the comfort of patients, these methods are essential in modern pediatric surgical practice.

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