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IDENTIFICATION AND OPTIMIZATION OF METHODS OF TREATMENT OF BLEEDING FROM VARICOSE VEINS OF THE ESOPHAGUS AND STOMACH IN CHILDREN

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Abstract:

Esophageal and gastric varicosity in children is a serious medical problem associated with a high risk of life-threatening bleeding. Despite the achieved successes in diagnostics and treatment of this pathology, the frequency of complications remains high, which determines the relevance of further improvement of therapeutic and diagnostic approaches. The development of optimal algorithms for management of pediatric patients taking into account anatomo-physiological features of the growing organism is of particular importance. Timely diagnosis and choice of optimal treatment tactics play a key role in prevention and treatment of bleeding from esophageal and gastric varices in children. In this connection it is urgent to improve existing and develop new methods of diagnostics and treatment of this pathology in pediatric patients.

Keywords: varicose veins, esophagus, stomach, bleeding, children, diagnosis, treatment, portal hypertension.

RELEVANCE. Currently, variceal bleeding from esophageal varices (VBEV) is recognized as the most dangerous complication of portal hypertension (PH) and ranks first among non-ulcer bleeding in children, characterized by extremely high mortality rates. PH syndrome in almost 80% of children initially manifests with bleeding from esophageal and gastric varices, and even after successful treatment, recurrence is observed in 70% of cases. The mortality rate in VBEV ranges from 20% to 60%, reaching up to 80% in cases of first-time bleeding [1].

With the consistent development of modern medicine, new surgical, endoscopic, and medical methods for treating PH syndrome are being widely introduced. The ultimate goal of almost all surgical treatment methods for this condition is the prevention and treatment of life-threatening VBEV [3].

The pathogenesis of VBEV is based on the development of collateral venous pathways and their varicose transformation in the digestive tract organs due to increased venous pressure in the portal vein system for various reasons. The development of venous collaterals occurs simultaneously with the formation of varicose veins in the submucosal layer of the esophagus and cardiac portion of the stomach. Expansion of esophageal nodes leads to thinning of vessel walls, resulting in hemorrhage even from minor injuries [8]. The risk of bleeding is particularly high when portal pressure exceeds 250-300 mm water column. Additionally, hypersplenism disrupts hemocoagulation and reduces the possibility of thrombus formation at the site of vein rupture. Changes in the blood coagulation system are correlated with the frequency and duration of gastroesophageal bleeding. The risk of rebleeding increases with large varicose veins [5].

According to X.A. Akilov et al. [2], the thinning of esophageal varicose vein walls is influenced by increased internal pressure in PH, as well as negative pressure that intensifies during breathing, as evidenced by esophagomanometry data in adult patients with grade 2-3 esophageal varices. This affects the further protrusion of dilated vessels in the esophageal space and increases the risk of bleeding [8].

Most specialists in the field believe that it is unclear exactly when and why bleeding from esophageal varices begins, as no correlation has been found between the degree of PH or the severity of potential peptic damage to the esophageal mucosa and bleeding. However, there is a relationship between the size of certain varicose veins and the presence of hemorrhagic signs on their surface [9]. Retrospective analysis of medical histories of adult patients with liver cirrhosis showed no correlation between most clinical and laboratory parameters and the degree of esophageal variceal dilation. However, it was noted that the presence of ascites was the only parameter associated with the maximum dilation of esophageal vessels.

Currently, even after widely used "shunting operations" aimed at forming artificial portocaval anastomoses, there has not been a significant reduction in complications such as liver failure, encephalopathy, shunt thrombosis, and associated decreased hepatic portal perfusion. Therefore, endoscopic methods for treating VBEV, including endoscopic sclerotherapy (ES), are considered more promising due to their minimal

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invasiveness, ability to selectively obliterate esophageal veins in the submucosal layer while preserving paraesophageal collaterals, and absence of negative effects on liver function. However, endoscopic hemostasis methods are mostly used in adult patients, while experience with these procedures in children is extremely limited, and individual results are poorly studied. Furthermore, clear indications for esophageal vein sclerotherapy in various forms of PH have not been established, and treatment tactics have not been developed. The radical correction method proposed by de Ville de Goyet for restoring hepatic portal perfusion is expected to open new perspectives in the surgical treatment of EHPVT, but data is still insufficient for a complete evaluation of this method's results.

The high prevalence of VBEV in children and high mortality rates necessitate in-depth study of risk factors and main etiopathogenetic mechanisms of this severe complication, as well as the development of prevention methods, early detection, and effective treatment approaches [10].

RESEARCH OBJECTIVE: To optimize methods for detecting and treating cases of bleeding from esophageal and gastric varices in children.

MATERIALS AND METHODS: This study is dedicated to the scientific analysis of examination and complex treatment results of 128 pediatric patients aged 1 to 18 years with various forms of portal hypertension, treated at the Republican Scientific Center for Emergency Medical Care (RSCEMC), RSCEMC Andijan Regional Branch, and Andijan Regional Children's Multidisciplinary Medical Center from 2005 to 2024.

The gender distribution of pediatric patients showed an approximately 1:1 ratio between boys and girls, with 60 boys (46.9%) and 68 girls (53.1%). Regarding age distribution, more than half of the patients, specifically 70 children (54.7%), were between 3 and 7 years old.

Extrahepatic portal hypertension (EHPH) associated with postnatal portal vein thrombosis or congenital defect was observed in 89 children (69.5%).

79 children (61.7%) with PH were hospitalized with VBEV. Boys showed a lower incidence of PH manifestation with bleeding compared to girls (46.1% and 54.0% respectively). Among children with PH, bleeding episodes were most frequently recorded in the 3-7 age group and were minimal after age 13 (Figure 1).

In addition to clinical criteria for PH diagnosis (splenomegaly, dilation of abdominal and thoracic veins, gastroesophageal bleeding, enlarged or reduced liver size, ascites, etc.), all patients were examined for additional clinical signs that determine disease severity and prognosis. All 128 children underwent detailed medical history collection, biochemical and complete blood count analysis, viral hepatitis marker testing, and esophagofibrogastroduodenoscopy (EFGDS) for targeted assessment of esophageal and gastric veins. Additionally, 115 children (89.8%) underwent ultrasound examination of the liver and hepatolienal vessels.

Laboratory-instrumental diagnostics: Along with routine clinical-biochemical blood testing, screening for viral hepatitis B and C markers was performed, preferring polymerase chain reaction (PCR) and coagulogram methods.

Ultrasound examinations were performed on 128 patients using Toshiba 370A Powervision 6000, ATL 5000, and Siemens "Elegra" ultrasound scanners operating in "real-time" mode with multifrequency sector probes with 4.2 MHz (2.5-6 MHz) preference. Convex probes from 2.5 to 5.0 MHz were used for liver examination. Quantitative parameters of basal blood flow in liver vessels were strictly measured on an empty stomach. Examinations were based on the following parameters: liver contour, lobe size, protrusion beyond the costal margin, parenchymal composition, reflection intensity, echo structure uniformity, condition of intrahepatic bile ducts and vessels, and portal vein status. Additionally, the gallbladder, spleen, kidneys, and their blood vessels were examined ultrasonically.

Esophagofibrogastroduodenoscopy (EFGDS): This method enabled verification of PH syndrome presence, which is an undoubted sign of VBEV common in children with LC and varicose veins.

Examinations were performed on all 128 children using "Olympus" and "Pentax" devices. The degree of phlebectasia in admitted children was assessed using A.A. Shavrov's method (1994).

Indicators were evaluated based on the following criteria:

- By color: pink strip-like veins - 1 point, red trunk or nodular appearance - 2 points, dark red-bluish - 3 points;
- By size: diameter up to 0.3 cm - 1 point, 0.4 to 0.5 cm - 2 points, 0.6 and larger - 3 points;
- By vein length: not exceeding the lower third of the esophagus from the cardioesophageal junction - 1 point, up to the middle third - 2 points, and to the upper third - 3 points.

Ultrasound examination of portal system parenchymal organs and vessels confirmed PH diagnosis and clarified its form based on changes in the main portal vein trunk and its lobar branches, presence or absence of portoportal collaterals ("spaces") in the liver parenchyma and gates, as well as the severity and extent of fibrotic processes in the liver.

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The main diagnostic criteria for PH included: narrowing or dilation of main portal vein trunk segments, narrowing or dilation of venous branches, presence and severity of periportal fibrosis, tortuosity and dilation of splenic veins, splenomegaly with increased echogenicity, presence of additional venous structures (collaterals) in liver and splenic gates, and dilation of esophageal-gastric veins.

Doppler examination of portal system vessels was considered a highly objective diagnostic method that enabled accurate assessment of portal circulation changes, PH form, as well as determination of collateral blood flow direction and severity (presence of functioning useful portocaval shunts such as mesenterocaval, splenorenal, etc.), and to some extent, prediction of bleeding probability in VV. Portal blood flow was examined using an "Aloka SSD-1700" ultrasound scanner with a linear format probe operating at 7.5 MHz frequency.

EGDS enabled detection of one of the main signs of PH-VV, which is the source of gastrointestinal bleeding. The severity of varicose veins (grades I-II-III) and their distribution were determined according to E.A. Paquet. The severity of PH was assessed based on the classification of congestive portal hypertensive gastropathy (CPHG) by T.T. McCormack et al. According to this classification, two degrees of gastric mucosal damage were distinguished: mild CPHG showed a mosaic pattern of the mucosa, while severe CPHG was characterized by submucosal hemorrhages and dark red diffuse spots. This classification was also useful in predicting the probability of bleeding.

Morphological examination of liver biopsies was used to confirm the diagnosis of chronic liver disease with complicated PH, determine the severity and localization of fibrotic processes, and differentiate between intrahepatic and extrahepatic forms of PH (cirrhosis or periportal fibrosis).

Among 55 patients who underwent emergency surgery at the peak of bleeding, 46 (83.64%) were diagnosed with extrahepatic portal hypertension (EHPH), and 9 (16.36%) with intrahepatic portal hypertension (IHPH).

RESEARCH RESULTS: Emergency EFGDS was performed on all patients admitted with acute esophageal and gastric bleeding, revealing grade I varices in 18 children (14.1%), grade II in 25 (19.5%), grade III in 40 (31.3%), and grade IV in 45 cases (35.2%). Evidently, this dangerous complication occurs in 2/3 of cases (85; 66.4%) with grade III and IV varices.

Among 79 patients hospitalized with ongoing bleeding, the bleeding source was located in the middle and lower 1/3 of the esophagus in 69 cases (87.3%) and in the cardiac portion of the stomach in 10 cases (12.7%). In patients presenting with stopped bleeding (n=49), bleeding sources at the above-mentioned locations were recorded in 45 (91.8%) and 4 (8.2%) cases, respectively.

Complex treatment measures for profuse gastroesophageal bleeding in children with PH included the following components:

1. Bleeding source control: The stomach is intubated with a standard nasogastric tube, and continuous aspiration of gastric blood and contents is performed. The stomach is irrigated with cold physiological solution until clear. The nasogastric tube is also used to assess ongoing bleeding presence and intensity and monitor for rebleeding. Subsequently, lagoden or logochilus decoction is administered at 10-30 ml three times daily depending on the patient's age, with the tube clamped for 30 minutes. Cold compresses are applied to the epigastric region. Oral intake of food and fluids is completely restricted until bleeding is fully controlled.
2. Portal pressure reduction: Oxytocin is administered intramuscularly at 0.1 ml/year of age every 6 hours, or pituitrin at units/kg/day.
3. Coagulation system intervention: Intramuscular administration of dicynone at 10-5 mg/kg divided into 3-4 doses to increase platelet adhesion and reduce capillary permeability. To reduce blood fibrinolytic activity, 5% epsilon-aminocaproic acid is administered intravenously in age-dependent doses at ml/kg every 6 hours. Intramuscular vicasol injection is given to stabilize physiological mechanisms of blood coagulation. Fresh frozen plasma (5ml/kg) of matching blood group is administered to patients with PTI below 60%.
4. Blood loss replacement and hypoxia management: Plasma substitute solutions (rheopolyglucin, etc.) were excluded from therapy to prevent the risk of sharp increases in systemic arterial blood pressure that directly affects portal pressure. 5-10% glucose solutions and balanced salt solutions are the main preparations for infusion therapy. Blood loss is partially replaced, maintaining controlled hypotension until bleeding is completely stopped. Erythrocyte mass transfusions of matching blood group are performed strictly when indicated, at 5-10 ml/kg body weight when hemoglobin levels fall below 60 g/l.
5. Reduction of gastric acid effect on bleeding source: Age-appropriate doses of antacid H2 blockers (ranitidine, famotidine), proton pump inhibitors (omez, omeprazole or its analogues), and

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binding/coating preparations like almagel are administered at 10-15 ml/day divided into 2-3 doses.

6. Reduction of blood resorptive effect: Cleansing enemas are performed every 4-6 hours. This procedure also has specific diagnostic value in monitoring bleeding intensity.

Shock index, determined hourly, serves as the main indicator for monitoring conservative therapy effectiveness. Children mainly presented to the clinic with 2nd and 3rd-degree shock index values.

The above conservative therapy was conducted for 12 hours and proved effective in 62 patients total (Table 3.1). In most cases, especially with bleeding grade III and IV VV, conservative treatment shows its effectiveness after 6-9 hours. During the first 3 hours of treatment, bleeding was stopped in only 9 (14.5%) patients with grades I and II, and 4 (6.5%) children with grade III.

CONCLUSION:

The ratio of boys to girls among children with PH complicated by variceal bleeding is approximately 1:1 (46.9% and 53.1%). In children, extrahepatic portal hypertension resulting from postnatal portal vein thrombosis or congenital defect occupies the primary position (69.5%) in the disease etiology. PH manifestation with VV bleeding in children is more common (54.7% of cases) between ages 3-7, and least common (8.6%) in children over 13 years. Esophageal bleeding complications in children with PH occur in 2/3 of cases (66.4%) with grade III and IV VV. In most patients (87.3%), the bleeding source is located in the middle and lower 1/3 of the esophagus, and in 12.7% in the cardiac portion of the stomach. Bleeding represents a serious condition for liver morphofunctional state, as post-hemorrhagic ischemia causes significant morphological and functional changes in liver parenchyma and its microcirculatory bed, which may negatively affect the outcomes of complex treatment of acute esophageal bleeding complications and disease prognosis in patients with PH.

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