IJACT, Volume 3, Issue 4, 2025 ISSN: 2995-5378
http://medicaljournals.eu/index.php/IJACT

INTERNATIONAL JOURNAL OF ALTERNATIVE AND CONTEMPORARY THERAPY

Novel Approaches to Management and Treatment of Gastroesophageal Variceal Hemorrhage in Pediatric Patients

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Abstract: Gastroesophageal variceal bleeding represents one of the most severe and life-threatening complications of portal hypertension in pediatric patients. Despite advances in diagnostic technologies and therapeutic interventions, this condition remains associated with significant morbidity and mortality rates in children with portal hypertension of various etiologies. The multifactorial pathophysiology, unique anatomical considerations, and distinct hemodynamic features in children present particular challenges in the clinical management of these patients.

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

Relevance. Portal hypertension in the pediatric population differs significantly from adult manifestations, with biliary atresia, congenital hepatic fibrosis, and extrahepatic portal vein obstruction being the predominant underlying causes rather than cirrhosis secondary to viral hepatitis or alcohol consumption. This etiological distinction necessitates tailored therapeutic approaches specifically designed for pediatric patients. Furthermore, the smaller vessel caliber, developmental hemostatic differences, and physiological vulnerability of children require specialized adaptation of both diagnostic modalities and therapeutic interventions.

Current management strategies encompass a wide spectrum of approaches including pharmacological therapies, endoscopic interventions, radiological techniques, and surgical procedures. However, the optimal treatment algorithm for pediatric variceal hemorrhage remains inadequately defined due to limited high-quality evidence, with most recommendations derived from adult studies or small pediatric case series. The remarkable regenerative capacity of children's livers, alongside their potential for long-term survival, makes the identification of effective yet minimally invasive approaches particularly crucial.

This research aims to systematically evaluate existing therapeutic modalities for gastroesophageal variceal bleeding in children, identify the most effective protocols for acute management and secondary prophylaxis, and develop an optimized, evidence-based treatment algorithm specifically tailored to pediatric patients. Through comprehensive assessment of outcomes including hemostasis success rates, rebleeding incidence, procedure-related complications, and long-term survival, this study seeks to establish standardized guidelines that balance immediate efficacy with consideration for the child's long-term development and quality of life

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

Materials and Methods. This research examines the clinical outcomes of 128 pediatric patients (ages 1-18) with various forms of portal hypertension who received treatment at the Republican Scientific Center for Emergency Medical Care (RSCEMC), its Andijan Regional Branch, and the Andijan Regional Children's Multidisciplinary Medical Center between 2005 and 2024.

The study population comprised 60 boys (46.9%) and 68 girls (53.1%), with the majority (54.7%) being children between 3 and 7 years old. Extrahepatic portal hypertension (EHPH), resulting from postnatal portal vein thrombosis or congenital anomalies, was diagnosed in 89 patients (69.5%).

Variceal bleeding from esophageal veins (VBEV) was the presenting complaint in 79 children (61.7%), with a higher incidence among girls (54.0%) compared to boys (46.1%). The 3-7 age group demonstrated the highest frequency of bleeding episodes, while children over 13 years showed minimal occurrence.

All patients underwent comprehensive diagnostic evaluation including detailed medical history, biochemical and hematological analyses, viral hepatitis marker testing, and esophagofibrogastroduodenoscopy (EFGDS). Additionally, 115 children (89.8%) received ultrasound examination of the liver and hepatolienal vessels.

Laboratory-instrumental diagnostics included routine clinical-biochemical blood tests, viral hepatitis screening (preferentially using PCR), and coagulation profile assessment. Ultrasound examinations were performed using various high-resolution scanners (Toshiba 370A Powervision 6000, ATL 5000, and Siemens "Elegra") with multifrequency probes. Parameters evaluated included liver morphology, parenchymal composition, vascular status, and the condition of adjacent organs.

EFGDS enabled direct visualization of variceal severity, which was graded according to A.A. Shavrov's methodology (1994) based on color, size, and extent of the varices. Doppler ultrasound of the portal system provided objective assessment of portal circulation dynamics, collateral flow patterns, and potential bleeding risk.

Among the 55 patients requiring emergency surgical intervention during acute bleeding episodes, 46 (83.64%) had EHPH and 9 (16.36%) had intrahepatic portal hypertension (IHPH).

Results. Emergency EFGDS revealed 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%). Notably, two-thirds of bleeding episodes (66.4%) occurred in patients with grade III and IV varices.

The bleeding source was predominantly located in the middle and lower third of the esophagus (87.3% of active bleeding cases), with cardiac portion of the stomach involvement in the remaining 12.7%. Similar distributions were observed in patients with recently ceased bleeding.

The comprehensive treatment protocol for gastroesophageal hemorrhage included:

1. **Bleeding source control**: Nasogastric tube placement with continuous aspiration and cold saline lavage until clear return. The tube facilitated administration of hemostatic agents (lagoden or logochilus decoction) while monitoring for ongoing or recurrent bleeding. Complete restriction of oral intake was maintained until hemostasis was achieved.
2. **Portal pressure reduction**: Oxytocin or pituitrin administration in age-appropriate dosages.
3. **Coagulation system support**: Administration of dicynone to enhance platelet adhesion, epsilon-aminocaproic acid to reduce fibrinolysis, vicasol to stabilize coagulation mechanisms, and fresh frozen plasma when indicated by coagulation parameters.
4. **Volume replacement strategy**: Purposeful avoidance of plasma substitutes to prevent increases in systemic arterial pressure, with preference for glucose solutions and balanced electrolytes. Controlled hypotension was maintained until complete hemostasis, with limited erythrocyte transfusions only when hemoglobin fell below 60 g/l.
5. **Gastric acid suppression**: Administration of H2 blockers, proton pump inhibitors, and mucosal protective agents in age-appropriate dosages.
6. **Reduction of blood resorption**: Regular cleansing enemas, which also served diagnostic purposes in monitoring bleeding severity.

Treatment efficacy was monitored through hourly assessment of shock index, with most patients presenting with 2nd or 3rd-degree values. The described conservative approach proved effective in 62 patients total, with the majority of responses in grade III and IV varices occurring after 6-9 hours of treatment. Only 13 patients (21%) achieved hemostasis within the first 3 hours.

Conclusions: The gender distribution among children with portal hypertension complicated by variceal bleeding is approximately equal. Extrahepatic portal hypertension represents the predominant etiological form (69.5%) in the pediatric population. Bleeding complications most frequently affect children between 3-7 years (54.7%) and are least common after age 13 (8.6%).

Severe variceal disease (grades III and IV) accounts for approximately two-thirds of bleeding episodes. Anatomically, the middle and lower thirds of the esophagus represent the primary bleeding sites (87.3%), with gastric cardiac involvement in 12.7% of cases. Hemorrhagic episodes significantly impact hepatic morphology and function, as post-hemorrhagic ischemia induces substantial changes in liver parenchyma and microcirculation. These alterations may adversely affect both immediate treatment outcomes and long-term prognosis in patients with portal hypertension.

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