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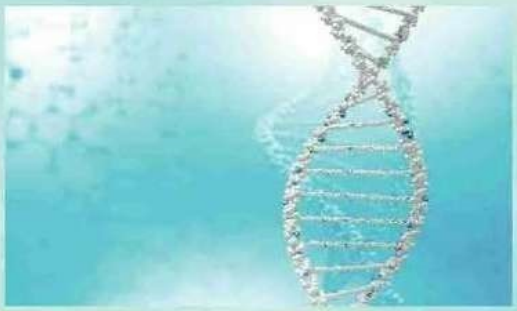

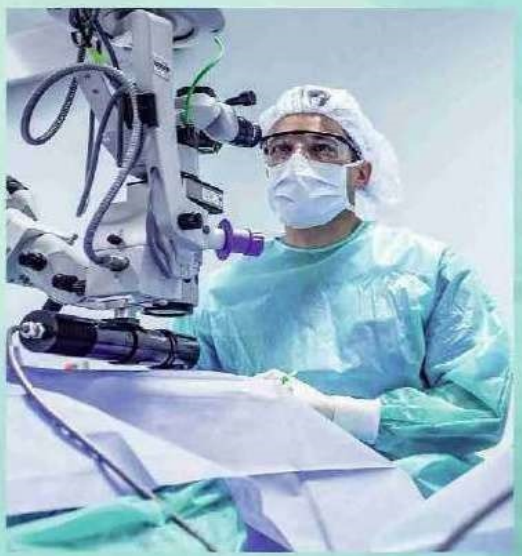

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NEW DAY IN MEDICINE

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АКТУАЛЬНЫЕ ВЗГЛЯДЫ И МЕТОДЫ ЛЕЧЕНИЯ КОНСЕРВАТИВНОЙ ТЕРАПИИ ПО ВИДАМ ПОРТАЛЬНОЙ ГИПЕРТЕНЗИИ И ПОКАЗАНИЯ К ОПЕРАЦИЯМ ПРИ ПИЩЕВОДНО-ЖЕЛУДОЧНЫХ КРОВОТЕЧЕНИЯХ У ДЕТЕЙ

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✓ ***Резюме***

Портальная гипертензия может быть вызвана целым рядом состояний. Часто проявляется кровотечением из варикозно расширенных вен пищевода. Подход к острому варикозному кровотечению у детей представляет собой пошаговое продвижение от наименее инвазивного к наиболее инвазивному. Лечение острого варикозного кровотечения несложное. Но данные о первичной профилактике и длительном лечении рецидивирующих кровотечений из варикозно расширенных вен у детей недостаточны, поэтому для установления передовой практики необходимы проспективные многоцентровые исследования.

Ключевые слова: Портальная гипертензия, кровотечения, дети, варикохно расширенные вены

CURRENT VIEWS AND TREATMENTS OF CONSERVATIVE THERAPY BY TYPE OF PORTAL HYPERTENSION AND INDICATIONS FOR SURGERY FOR OESOPHAGEAL-GASTRIC BLEEDING IN CHILDREN

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✓ ***Resume***

A number of conditions can cause portal hypertension. It often manifests as bleeding from varices in the oesophagus. The approach to acute variceal bleeding in children is a stepwise progression from the least invasive to the most invasive. The treatment of acute variceal bleeding is straightforward. However, data on primary prevention and long-term treatment of recurrent variceal bleeding in children are scarce, so prospective multicentre studies are needed to establish best practice.

Keywords: Portal hypertension, bleeding, children, varices

PORTAL GIPERTENZIYA TURLARI BO'YICHA KONSERVATIV TERAPIYANING HOZIRGI KO'RINISHLARI VA DAVOLASH USULLARI VA BOLALARDA QIZILO'NGACH-OSHQOZON QON KETISHI UCHUN JARROHLIK KO'RSATMALARI

Khalilov Sh.K., Abduvalieva Ch.M.

Андижон давлат тиббиёт институти

✓ ***Rezyume***

Portal gipertenziya bir qator holatlar tufayli yuzaga kelishi mumkin. Bu ko'pincha qizilo'ngach varikozlaridan qon ketishi sifatida namoyon bo'ladi. Bolalarda o'tkir vaskulyar qon ketishini davolash yengilroq. Ammo bolalarda takroriy varikulyar qon ketishining birlamchi profilaktikasi va uzoq muddatli davolash bo'yicha ma'lumotlar kam, shuning uchun ham yaxshi amaliyotni o'rnatish uchun bizning tadqiqotlar kata rol o'ynaydi.

Kalit so'zlar: Portal gipertenziya, qon ketish, bolalar, varikozlar

Relevance

Portal hypertension (PH) syndrome is a major problem in paediatric surgery, irrespective of the cause. In 80% of pediatric patients extrahepatic hypertension (EPH) is caused by a malformation or thrombosis of the portal vein. Every surgeon knows the danger of bleeding from varices (VV) of the oesophagus and stomach, occurring in 80% of patients with portal hypertension (PG). Dramatic outcomes in the first haemorrhage are as high as 60% (1.4.6). Bleeding recurs in 50-90% of cases with a mortality rate of 70% [5].The main endoscopic risk factors for bleeding from esophageal and gastric VVH are vein size, dominant mucosal colour over varices, angioectasia, esophagitis, presence of gastric VVH, gastropathy [3]. The frequency of bleeding depends on the severity of the inflammatory changes in the esophageal and gastric mucosa (4). The mass and duration of bleeding from esophageal and gastric varices (EVVV) and gastric mucosa (GML), as a complication of AI, determine the disruption of the blood clotting system due to the development of hypersplenism. The main task of surgeons has always been to find methods of predicting, diagnosing and treating this complication. To date, according to Razumovsky A.Y. (2018), there is no ideal way to treat children with CHD, as no methods have yet been developed to restore blood flow in the portal vein system while reducing portal pressure. It is generally accepted that splenectomy as a treatment option is not justified. PJD in children is characterised by sudden onset, high intensity and low efficacy of conservative therapy. The issues of timely tactics of conservative therapy at occurrence of this dangerous complication remain unresolved and contradictory.

Purpose of the study: Treatment of conservative therapy by portal hypertensis and recommendations for operations on gerd-groval breaths in children

Materials and methods

All patients, admitted with acute esophagogastric bleeding were subjected to EFGDS. Endoscopic examination of esophagus and stomach revealed first degree I incision in 2 children, second degree - in 11 patients, third degree - in 30 and fourth degree - in 35 patients. The source of bleeding in 69 children was detected in the esophagus (in c/3 and n/3) in 9 patients in the cardiac region of the stomach. All patients with bleeding of the 1st or 2nd degree had bleeding after increasing body temperature and taking NSAIDs and after EGGDS it was found that the source of the bleeding was in the stomach.

Result and discussion

In the treatment tactics of profuse RPL in children with PG we adhered to the following principles:

1. We probed the stomach (with the usual nasogastric tube), allowing a constant evacuation of blood and gastric contents and control of the bleeding intensity. The stomach was flushed with cold physiological solution until clear. After that, lagoden or logochilus broth was administered 10-30ml, depending on age, 3 times a day and the probe was closed for 30 minutes. Cold on the epigastric area. Food and liquid intake by mouth were completely excluded until the bleeding had stopped.
2. Reduction of portal pressure: oxytocin 0.1 ml/year of life at 6-hour intervals, i.m., or pitiutrin at the rate of 1 iu/kg per day was used to reduce portal pressure.
3. Effects on the blood coagulation system: to increase platelet adhesion and reduce capillary permeability, we administered decinone at 10-5mg/kg, divided into 3-4 doses v/m. Reduction of blood fibrinolytic activity was achieved by intravenous administration of epsilon-aminocaproic acid -5% in ml/kg, depending on age, at intervals of 6 hours, by IV, dropwise. In order to stimulate the physiological mechanisms of the blood coagulation system, vicasol was administered v/m. To replenish clotting factors, single-group fresh frozen plasma was transfused - 5ml/kg fractionally, in patients with a decrease in PTI values below 60%.

4. Blood loss compensation and hypoxia control: plasma replacement solutions (reopolyglucin, etc.) were excluded from therapy to prevent a sharp rise in systemic BP, which is directly related to AP. The basic preparations for infusion therapy were 5-10% glucose solutions and balanced saline solutions. Blood loss was partially replenished and the patients were kept in a state of controlled hypotension until the bleeding had stopped. Hemotransfusions with single-group erythrocyte mass were performed under strict indications, when the patient's Hb index was lower than 60 g/l,

fractionally, at the rate of 5-10 ml/kg.

Reducing the impact of gastric acidity on the source of bleeding: to reduce the impact of gastric acidity on BPV, antacids - H2 blockers- ranitidine, famotidine, proton pump inhibitors- omez, omeprosol or analogues, astringent and coating drugs such as Almagel in an age-appropriate dosage of 10-15 ml/day divided into 2-3 doses and sea buckthorn oil 2.5-5 ml 3 times/day were used.

Reducing the resorptive action of blood: every 4-6 hours the patients were given purging enemas. This procedure had a certain diagnostic value in controlling the intensity of bleeding. The main indicator to monitor the effectiveness of the conservative therapy administered was the shock index, determined hourly. Children were mostly admitted to the clinic with grade 2 and 3 shock indices. Conservative therapy reduced the index after 6 hours in 27 patients, but complete haemostasis was achieved in 31 patients.

Table 1.

Dynamics of stopped bleeding over time according to the degree of GVHD

	After 3 hours		After 6 hours		After 9 hours	
	GVHD n-30	GVHD n-6	GVHD n-30	GVHD n-6	GVHD n-30	GVHD n-6
I-degree	2		0	2	0	2
II-degree	7		4	1	0	1
III-degree	4		21		14	
IV-degree	0		1		3	

Conservative therapy was effective in 46 patients (23.2%).

According to our observation, for the first 3 hours of conservative therapy hemostasis was achieved in only 13 children (4.6%), these patients 9( ) cases of grade I-II 4 grade III on EFGDS. We can say the main cause of bleeding in PG of I-II degree is concomitant pathology (ARI etc.) with the intake of NSAIDs and increased body temperature, whereas after 9 hours of observation bleeding was stopped in 5 patients (11.6%), after 12 hours only in 3 (6.9%) patients it was possible to stop bleeding.

No particular correlation between the effectiveness of conservative therapy and the type of PH was found (Table 3.1). Thus, effective haemostasis after conservative therapy in patients with intrahepatic PH was achieved in 60% of cases, in children with CHD in 26.2% of cases. In 33 patients (76.7%) after 12 hours of conservative measures it was not possible to stop PJD, which was a direct indication for surgical intervention.

The disease in 64 (59.3%) children with PH started with HCC or post-haemorrhagic anaemia from the PH. In girls, the development of PH from the first episodes of bleeding occurred more frequently in girls, 36 (56.2%), than in boys, 28 (43.8%). (43,8%). At the same time, EGDS among 109 children with PH showed that stage I LUT did not occur in children with HCC, but only in 8 (7.3%) children without HCC (Table 3).Grade II FVC was observed in 47 children (43.1%) admitted with signs of HCC or post-haemorrhagic anaemia and 35 (32.1%) with no history of HCC or post-haemorrhagic anaemia, who had no history of HCC. The third degree of PJV was in 17 (15.6%) patients with HCC and 2 (1.8%) without HCC.Taking into account this, the highest risk of hemostasis system disorders, we paid special attention to the study of corresponding indices in CHF and HCC. The results of the study of the coagulation component of the haemostasis system, obtained in patients in the long-term period in relation to the control group, are presented in Table 3.

Table 2

Degree of PHV in children with PH as measured by EFGDS

Children with CFPDs	Degree of varicose veins						Total	
	I		II		III			
	Abs	%	Abs	%	Abs	%	Abs	%
Admitted withSigns of HCC or Post haemorrhagic	—	—	47	43,1	17	15,6	64	58,7

Table 3.

Haemostasis indicators in AI patients

Indicator	Control	WFPH	OPFPG
		n=6	n=21
Platelets	275,5±13,8	431,4±81,3	296,4±62,7
Heparin tolerant plasma	437,7±45,1	478,2±73,4	450,6±49,5
Recalcification time	108,6±6,4	85,6±8,3*	105,0±7,9
Blood clotting time	268,6±15,3	224,6±37,8	251,8±40,2
Prothrombin:	95,4±2,5	105,2±3,4*	97,0±3,7
Fibrinogen:	3285±287	3453,1±2и 49,4	3174±266
Clot retraction	30,7±2,1	32,5±3,7	31,3±2,6
Fibrinolytic activity	12,2±1,6	16,8±2,0	15,1±1,9

*Note: * - differences relative to control are significant (* - P<0.05), ^ - differences relative to post-SE group are significant (^ - P<0.05)*

As shown in Table 3.2, significant changes in coagulation parameters of the haemostasis system were registered in the group of WTPG and WTPG patients.

The patients in the WFPH groups had higher platelet concentrations than those in the donor and WFPH groups. At the same time thrombocytosis in the group of patients with VPPG significantly testifies to more expressed disturbances of thrombocytic hemostasis than in the group with VPPG (P<0,05).

In the distant period in patients after WFPH there is activation of the coagulation link of the hemostasis system, as statistically reliable shortening of plasma recalcification time, clotting time and increase of plasma tolerance time to heparin and prothrombin percentage are observed.

At the same time, the third phase of the blood coagulation process is activated: the level of fibrinogen in blood increases, a significant increase in blood fibrinolytic activity is noted compared with control.

Conclusion

In the group of patients after WFPH in the distant period there was a stable normal level of fibrinogen, its index and other indexes of the blood coagulation system didn't differ statistically reliable from the data of the practically healthy people. It follows from here, that more expressed hemostatic disturbances in WASH are connected with organic change of liver tissue as everybody knows that liver plays an important role in hemostasis and hemostasis in human organism**.**

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