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

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ABSTRACT

Portal hypertension (PH) is one of the major causes of life-threatening esophageal-gastric bleeding. The causes of PH in adults and children are fundamentally different. While in adults the cause of esophageal variceal bleeding (EVB) in 75% of cases is liver cirrhosis, in children in our country the most common form of UC is extra hepatic portal hypertension (EPH) - blockage of blood flow through the portal vein due to thrombosis or malformation. There is little change in liver function. The main aim of the treatment of patients with PHH is to prevent bleeding from the portal vein. The management of children with PG has changed in recent years due to new surgical treatment options[7,10].

Keywords: ***Portal hypertension, esophageal-gastric bleeding, pediatric surgery.***

INTRODUCTION

Study objective: Effective conservative treatment of portal hypertension and indication for surgery for esophageal-gastric hemorrhage in children.

Portal hypertension (PH) syndrome is a major problem in pediatric surgery, irrespective of the cause. In 80% of pediatric patients extra hepatic hypertension (EPH) is caused by a malformation or thrombosis of the portal vein. Every surgeon knows the danger of bleeding from varies (VV) of the esophagus and stomach, occurring in 80% of patients with portal hypertension (PG). Dramatic outcomes in the first hemorrhage 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 color over varies, 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 PH determines the disruption of the 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 unwarranted. PJD in PG in children is characterized by sudden onset, high intensity and low efficiency of conservative therapy. The issues of timely tactics of conservative therapy in the occurrence of this formidable complication remain unresolved and contradictory[8,9].

MATERIALS AND METHODS

All patients admitted with acute esophageal-gastric bleeding under emergency procedure

were subjected to EFGDS. Endoscopic examination of the esophagus and the stomach revealed first degree I bleeding in 2 children, second - in 11 patients, third - in 30 and fourth - 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 EFGDS it was found that their GIs were in the stomach.

Results of the study: In the therapeutic tactics of profuse PJD in children withPH, we adhered to the following principles:

1. Influence on the source of bleeding: we probed the stomach (with a conventional nasogastric tube), which allowed a constant evacuation of blood, gastric contents and control the intensity of bleeding. The stomach was flushed with cold physiological solution until clear. After that, lag oden 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 epigastria area. The intake of food and liquids by mouth was completely avoided until the bleeding had completely stopped;
2. Reduction of portal pressure: oxytocin 0.1 ml/year of life at 6-hour intervals, v/m, or pituitary at 1 in/kg/day was used to reduce portal pressure;
3. Effects on the clotting system: to increase platelet adhesion and reduce capillary permeability, we used 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 6-hour intervals, by IV drip. In order to stimulate the physiological mechanisms of the blood coagulation system, vicasol was administered v/m. To reimburse coagulation factors, single-group fresh frozen plasma was transfused - 5ml/kg fractionally, in patients with decreased PTI values below 60%;
4. Compensation of blood loss and control of hypoxia: plasma replacement solutions (reopolyglukin, etc.) were excluded from therapy to prevent a sharp rise in systemic BP, which is directly related to PD. 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, omeprozole or analogues, astringent and coating agents such as Almagel in an age-dependent dose of 10-15ml/day divided into 2-3 doses and sea buckthorn oil in 2.5-5ml 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 hemostasis was achieved in 31 patients.

Table 1.1
Dynamics of stopped bleeding over time according to the extent of the GVHD

	In 3 hours		In 6 hours		In 9 hours	
	WCPF n-30	WFTU n-6	WCPF мп-30	WFTU n-6	WCPF n-30	WFTU 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 1.3). 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.

Table 1.2
Degree of PHV in children with PH as measured by EFGDS

Childrenwith WCPF	**Degreeofvaricoseveins**						**Total**	
	I		**II**		**III**			
	Abs	**%**	**Abs**	**%**	**Abs**	**%**	**Abs**	**%**
Admitted with signs of HCC or post haemorrhagicanaemia	-	-	47	43,1	17	15,6	64	58,7

The disease in 64 (59.3%) children with PH started with HCC or post-hemorrhagicanemia from the LVH. In girls, the development of PH from the first bleeding episode was more common, 36 (56.2%), than in boys, 28 (43.8%). However, EGDS among 109 children with

PH showed that grade I PHV did not occur in children with HCC, but only in 8 (7.3%) children without HCC (Table 3.1). Grade II PHE was seen in 47 children (43.1%) admitted with signs of HCC or post-hemorrhagicanemia and 35 (32.1%) with no history of HCC. Third-degree PJV was seen in 17 (15.6%) patients with HCC and 2 (1.8%) without HCC. Given this, the greatest risk of hemostasis system abnormalities, we paid special attention to the study of relevant parameters in AFPPH and VPPH.

The results of the coagulation studies of the hemostasis system obtained in patients in the long-term period in relation to the control group are presented in

Table 1.3

Haemostasis indicators in patients with portal hypertension

Indicator	Control	VPPH	AFPPH
		n=6	n=21
Platelets	275,5±13,8	431,4±81,3	296,4±62,7
Heparintolerantplasma	437,7±45,1	478,2±73,4	450,6±49,5
Recalcificationtime	108,6±6,4	85,6±8,3*	105,0±7,9
Bloodclottingtime	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
Clotretraction	30,7±2,1	32,5±3,7	31,3±2,6
Fibrinolyticactivity	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 hemostasis 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 groups of patients with VPPG significantly indicates more pronounced violations of the platelet hemostasis than in the group with VPPG (P<0.05).

In the long-term period in patients after WFPH there is an activation of the coagulation component of the hemostasis system, as statistically significant shortening of plasma requalification 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 clotting process is activated: the level of fibrinogen in the blood increases and there is a significant increase in the fibrinolytic activity of the blood compared to controls.

CONCLUSION

Thus, in the group of patients after WFPG in the distant period the stable normal level of fibrinogen was marked; 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 that more expressed hemostatic disturbances in WFPH 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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