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PORTAL HYPERTENSIVE GASTROPATHY AS A FACTOR OF ANEMIA IN PATIENTS WITH CIRRHOSIS

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Abstract

The authors provided information on portal hypertensive gastropathy and actual anemia in 58 patients with liver cirrhosis and sinusoidal portal hypertension. The first group (35) consisted of patients who had esophageal varices, PHG, and anemia, the second group (28) patients with varicose veins of the gastric varices, PHG and anemia. Endoscopic examination revealed the effects of PHG in patients in the first and second groups, which manifested as red and black spotting of the gastric mucosa with erosive defects. According to the Baveno scoring system for endoscopic appearance, severe manifestations of PHG in the first group occurred in 77% of patients and 13% in second. In patients with cirrhosis and portal hypertension without signs of acute bleeding anemia can be caused by PHG, which may be complicated by bleeding and/or development of pernicious anemia.

Keywords: Portal hypertensive gastropathy (PHG), cirrhosis, portal hypertension, esophageal and gastric varices.

Introduction.

PHG occurs in patients with portal hypertension regardless of its etiology, causes anemia, as a result of bleeding or B-12 deficiency, that requires correction. The prevalence of PHG in patients with liver cirrhosis ranges from 20 to 98%(1).

Most patients with PHG are asymptomatic, but some may have symptoms of chronic blood loss or chronic iron deficiency anemia, and significantly fewer

patients have active gastrointestinal bleeding. Chronic blood loss occurs in 30-60% of patients. (2)

The diagnosis of PHG is made during the endoscopic examination, which shows spotted mosaic mucosa, that resembles snakeskin, alternating red, white and black-brown macules, that resembles vascular ectasia.

In most cases ectasia is localized in the distal part of the stomach. Main histological changes are dilatation of capillaries and venules in mucosa and submucosa without significant inflammation. Portal hypertension is a progressive complication of cirrhosis (3) and the main pathophysiological mechanism for the development of complications in the gastrointestinal tract, such as esophageal and gastric varices, gastropathy, and enteropathy (4,5).

Methods.

The results of treatment of 58 patients with PHG, cirrhosis, the phenomena of sinusoidal portal hypertension and anemia were analyzed. Both groups had anemia of varying severity. The study excludes patients with acute gastrointestinal bleeding during the last 3 months. All patients were divided into two groups. The first group (35) consisted of patients with PHG who had varices of the esophagus, the second (23) patients with PHG and gastric varices.

Esophagogastroscopy allows us to reveal and evaluate the presence of varices, the severity of changes in the mucosa of various regions of the stomach by mosaic reconstruction, red macules, and telangiectasia. Depending on the spread and severity of the mucosa alteration, PHG assessment had been undertaken and measured on a Baveno score.

To reveal and assess the severity of PHG, sonographic Doppler ultrasound was used to measure hepatic blood flow, size of the portal and splenic veins. Sonographic examination allowed us to determine the presence of port systemic anastomoses. Obtained data allows identifying the relationship between portal vein flow velocity, the appearance of esophageal varices, changes in the gastric mucosa during portal hypertension. Data on sex, age, etiology of cirrhosis and laboratory parameters were taken into consideration. Laboratory and clinical data were included in the Child-Pugh scoring.

Results.

The development of PHG is prompted not only by portal hypertension but also by such factors as nonspecific inflammatory response, local vascular tone, endotoxins, insufficient liver function or gastric mucosal perfusion (6,7).

Endoscopic examination revealed changes in the gastric mucosa in patients of both groups. Thus in the first group 17(49%) patients with esophageal varices the cardiac gastric mucosa had a mosaic pattern, in 10 (29%) patients were covered with red and partially black macules, and in the distal part of the stomach were multiple erosions.

These changes in the gastric mucosa correlated with the size of the esophageal varices and were most severe in patients of the first group with 3rd-4th grade varices. In patients of the second group, the PHG phenomena were less pronounced. In 11 (48%) single erosions occurred in the distal part of the stomach, while 9 (39%) mosaic changes were localized mainly in the body of the stomach in the form of macules. According to the Baveno score in 27(77%), the severity of changes in the mucous membrane of patients in the first group was more than 4, which corresponds the severe form of PHG, while in 20 (87%) patients of the second group this index did not exceed 2- 3 points, indicating a mild degree of PHG.

Using the Child-Pugh score, cirrhosis severity in the first group Child A-grade was found in 2(6%) patients, Child B - in 23 (66%) and Child C - in 10 (28%) patients, while in second group Child A in 3 (13%) and Child B in 20 (87%) patients, respectively. This way, PHG was revealed in 78% of patients of the first group with Varices of the esophagus, and 87% in the second.

The average portal vein diameter in patients of the first group was 14.4 ± 1.2 mm and 12.3 ± 0.9 mm in the second group.

Duplex portal vein blood flow velocity measurements in patients of the first group were $12.7 \pm 2,9$ whereby arterial blood flow was preserved in 22(63%), and in patients of second group $14,3 \pm 5,1$ with preserved arterial blood flow in 19 (78%) patients.

Average spleen size in first group patients 15.5 ± 1.3 cm, while in the second group 13.1 ± 1.4 cm.

The average hemoglobin in the first group was $85 \pm 10,7$ g / l, in women $81 \pm 5,5$ g / l and men $79 \pm 11,3$ g / l, in the second group were $80 \pm 7,9$ g / l, in women - $75 \pm 6,4$ g / l and in men - $82 \pm 3,3$ g / l.

In the first group, first-degree anemia occurred in 10% patients, second - in 84%, and third in 6% of patients. In the second group first-degree anemia occurred in 7% patients, the second - in 87%, and third in 6% of patients.

Discussion.

The most common cause of portal hypertension is liver cirrhosis, which causes intrahepatic sinusoidal portal hypertension, which, like cirrhosis, needs different methods of diagnosis and treatment (8).

Portal hypertension with a high probability causes hemodynamic changes in the mucous membrane of the entire gastrointestinal tract (9).

Approximately 50% of patients with liver cirrhosis have gastrointestinal varices (10). The average diameter of the portal vein is considered to be the best indicator for assessing the severity of portal hypertension (11).

Endoscopic diagnosis in cirrhosis and portal hypertension in most cases is aimed at detecting varices in the esophagus and assessing the severity of their development, and less attention is paid to assessing the condition of the gastric mucosa in order to detect PHG and determine the severity of its development.

Pathological changes in gastric mucosa in patients with cirrhosis and esophageal varices, which accounted for the first group, showed a severe course of PHG in 77% patients, while patients in the second group observed a severe course of PHG in 13%. They had corresponding sonographic changes in measuring the velocity of blood flow in the portal vein, which indicated its slowdown. Arterial blood flow was mainly preserved in both groups. Spleen size was increased in patients of the first group and exceeded 15.5 cm.

Thus, the development of severe PHG was observed in patients with delayed portal blood flow, esophageal varices, and was the most severe in patients with C-class on the Child-Pugh score. In addition, the

decreased hemoglobin level corresponded to PHG severity in patients in both groups, which could indicate chronic blood loss in the absence of acute bleeding.

Conclusions

1. In the absence of a source of esophageal varices bleeding, the phenomenon of anemia in patients with PHG may be due to chronic bleeding from the gastric mucosa and the development of iron deficiency anemia.

2. The severity of PHG correlates with the severity of portal hypertension, as evidenced by increased portal vein size, slowing blood flow, enlargement of spleen and anemia.

3. PHG can be a prognostic factor for the development of liver cirrhosis and, depending on severity, is the base for predicting the need for invasive diagnostic methods.

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