"DEVELOPMENT OF PORTAL HYPERTENSIVE ENTEROPATHY IN PATIENTS WITH CIRRHOSIS OF THE LIVER"

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Annotation. *the article describes in detail from the etiology to the differential diagnosis of portal hypertensive enteropathy. Changes in the ultrastructure of the mucosal epithelium in portal hypertensive bowel disease are mainly characterized by colorectal bleeding. Bleeding is mainly caused by varicose veins of the submucosa, a sudden increase in pressure in the portal vein, erosion or ulceration of the surface of the mucous membrane, obstacles in blood clotting and a decrease in the quality or quantity of platelets.*

Keywords: *portal hypertensive bowel disease, portal hypertensive enteropathy, bleeding, vascular ectasia, varicose veins.*

Portal hypertensive bowel disease (PGA) was proposed by Visor in 1991 to denote submucosal telangiectasia of the intestine, stagnation, increased blood flow, arteriovenous shorts, capillary endothelium and bleeding based on portal hypertension (PGT). Changes in the ultrastructure of the mucosal epithelium in PGC are mainly characterized by colorectal bleeding [1]. Bleeding is mainly caused by varicose veins of the submucosa, a sudden increase in pressure in the portal vein, erosion or ulceration of the surface of the mucous membrane, obstacles in blood clotting and a decrease in the quality or quantity of platelets.

Pathogenesis

1. PGC is a lesion characterized by dilation of intestinal vessels secondary to portal hypertension. The pathogenesis is basically the same as that of portal hypertensive gastropathy (PGG). It is also associated with hyperdynamic blood

circulation of the portal vein. Measurement of the gradient of venous pressure in the liver (HVPG) showed that cirrhosis of the liver with PHC was significantly higher than in patients without PHC. Cirrhosis of the liver and portal hypertension not only increases blood flow in the stomach by 1 time, but also increases blood flow in esophagus, small intestine and colon. Shanmen, who underwent the laser Doppler method, found that in addition to the cecum, the blood flow from the rectum to the mucous membrane of the ascending colon was significantly increased with PHC, and the change in the blood flow of the mucous membrane was followed by severe PHC [2]. Tezuka et al . organ reflection spectroscopy was used to measure the rectal mucosa in patients with cirrhotic portal hypertension and found that the blood flow of the mucosa increased, indicating that the high-power cycle prevailing in portal hypertension is involved in the occurrence of PGC. It was reported that the width of the inner diameter of the portal vein is proportional to the vascular malformation of the colon. Damage to the colon mucosa is more evident in patients with a history of bleeding and colon viscosity in chronic portal hypertension [3]. The membrane, submucosal arterioles expand, and blood flow in the colon increases, that is, "splanchnic hyperemia" occurs, which increases blood flow in the portal vein and is one of the mechanisms for maintaining chronic high portal pressure.

Observation of certain vasodilating substances in patients with portal hypertension, such as nitric oxide, glucagon, prostaglandins, vasoactive peptides of the intestine, peptides of calcitonin-related genes, adenosine and carbon monoxide, may increase with synthesis, decreasing inactivation associated with factors such as portal shunt. In addition, peripheral arterial vasoconstrictor decreases in patients with portal hypertension. It is known that a vasoconstrictor, such as norepinephrine, is higher than normal in portal hypertension, but the visceral blood vessels are dilated [4]. The main one, representing a high-power cycle, is associated with an increase in the mass of endogenous vasodilators of the patient, which leads to the expansion of peripheral arteries, which, in turn, leads to a high-power cycle.

It is believed that nitric oxide is a key mediator of vasodilation, powerful blood circulation and PHC in many mediators. Nitric oxide is an inhibitory neurotransmitter, which mediates vasoactive expansion of the mucous membrane and microcirculation disorders in the mucous membrane.

Pathogenesis of PHC, experimental observation of portal hypertension, increased levels of nitric oxide synthase in the gastrointestinal tract, nitric oxide acts on the smooth muscles of the vessels, causing the expansion of blood vessels, nitric oxide also suppresses the function of the smooth muscles of the gastrointestinal tract, causing obstructive constipation, intestinal motor dysfunction is involved in the occurrence of PHC. In addition, glucagon plays an important role in the occurrence of high-intensity circulation of portal hypertension. Glucagon can expand the intestinal vascular network and direct the intestinal blood vessels to the noradrenal gland [5]. The reactivity of vasopressor vasoconstrictor decreases, which ultimately leads to stagnation in internal organs, vasodilation, increased vascular permeability, plasma extravasation, extensive swelling of the gastrointestinal mucosa, vascular distortion, liver dysfunction and shunting.

2. Dilation of the intestinal mucosa and submucosal vessels and thickening of mucosal edema is a characteristic histological manifestation of PGC. Under a light microscope, swelling of the colon mucosa, congestion, massive telangiectasia may be associated. The mucosal tissue is slightly inflamed, a small number of lymphocytes of the intestinal mucosa proliferate, weak lymphocytes and plasma cells infiltrate the mucous membrane of the mucous membrane; epithelial cells of the mucous membrane are separated from necrosis, forming erosions and bleeding; in some cases, swelling and degeneration of neurons were observed in the submucosal or intermuscular plexus. Ultrastructural changes of capillary endothelium and mucosal epithelial cells were observed under an electron microscope.

Exam.

1. Violation of the mechanism of blood clotting, a decrease in the quality or quantity of platelets.

2. Prolonged chronic bleeding - erythrocytes andhemoglobin may decrease.

3. Fecal latent blood is positive.

4. Colonoscopy

1- Vascular ectasia: a characteristic change in the PHC, which is characterized by a spider-like, spiral-shaped, convex or flat red lesion of the intestinal mucosa with a frequency of 28.6% to 93%. Mucosal biopsy showed telangiectasia and mucosal atrophy [6].

2- Varicose veins: It can be seen that the thickened veins of the colon mucosa are significantly thickened. In severe cases, cysts can be enlarged, and the frequency ranges from 16% to 45.7%. In some special cases, extremely dilated varicose veins of the rectum may be colonized. The mirror is misdiagnosed as a colon tumor, and a biopsy can cause severe bleeding.

3- Others: Blood vessels of the intestinal mucosa can also be seen in a curved shape, frontal spherical shape, distorted snakes and other irregular shapes, as well as submucosal hemorrhages, such as diffuse or isolated red spots or erythema [7].

5. Endoscopic ultrasound (EUS) is usually administered through the anus. It can also be used to detect varicose veins around the rectum and rectum. Varicose veins on the ultrasound image shows a cystic anechoic dark area and varicose veins of the rectum are detected. The positive indicator is higher than that of the endoscope.

Differential diagnosis.

1. Isolated dilation of small blood vessels is the cause of bleeding from the lower gastrointestinal tract, more common in elderly patients, mainly limited to the right colon, the total number is small, limited to a few.

2. Hereditary telangiectasia occurs in the jejunum, but also in the colon.

3. Henoch-Schenlein purpura is more common in young people, and sometimes it can be observed as vasodilation.

4. Vascular dysplasia (angiodysplasia) is observed in patients with aortic stenosis, and the incidence is mainly limited to the right colon [8].

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