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MORPHOLOGICAL FEATURES OF VARICOSE VEINS OF THE ESOPHAGUS ON THE BACKGROUND OF LIVER CIRRHOSIS

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Abstract: Varicose veins of the esophagus (vascular channels that connect the portal venous and systemic venous blood flow) are formed as a result of portal hypertension, most often in the submucosa of the lower esophagus. Although varicose veins can occur anywhere in the gastrointestinal tract (GIT), it most often appears on a few centimeters of the distal esophagus with cirrhosis of the liver.

Causes of varicose veins of the esophagus

The replacement of healthy liver tissue with scar tissue (fibrous) occurs as a result of chronic inflammation and damage to the liver caused by various causes, such as viral hepatitis, alcoholic liver disease, non-alcoholic fatty liver disease, biliary tract diseases, pancreatic lesions, autoimmune diseases, etc. The formation of fibrous tissue triggers the mechanism of development of varicose veins of the esophagus:

• the structure of the liver lobules is disrupted, where metabolism and blood filtration occur;
• scar tissue compresses blood vessels, including small vessels inside the liver lobules;
• there is an increase in pressure in the portal vein, which collects blood from the stomach, spleen, intestines and sends it to the liver (this condition is called portal hypertension);
• normal blood flow through the liver is disrupted – the blood searches for alternative pathways (collaterals) to return to the heart, bypassing the liver, and begins to bypass the damaged areas through smaller vessels.;
• one of the main alternative routes is the venous plexuses in the lower esophagus, which are significantly stretched, become thin–walled and convoluted, and varicose veins occur, especially in the esophagus and stomach;
• over time, as the volume of blood flow increases, the pressure in the varicose veins increases, which increases the risk of rupture and bleeding.

Varicose veins of the esophagus with narrowing (obstruction) of the superior vena cava (for example, with thrombosis, mediastinitis, or mediastinal tumors, including lung cancer) rarely develop due to impaired venous outflow. The cause of the disease in this case is not portal hypertension, but venous congestion in the superior vena cava system with the formation of alternative blood flow through the upper or middle part of the esophagus, where the veins dilate and form varicose veins.

The presence of enlarged gastroesophageal veins is directly correlated with the severity of liver disease:

• At the time of diagnosis, approximately 30% of patients with cirrhosis have varicose veins of the esophagus. This figure reaches 90% in about 10 years.;
• in some patients, varicose veins and bleeding occur at the onset of the disease, even in the absence of cirrhosis;
• Approximately every 5-6 patients with hepatitis C have varicose veins of the esophagus.

Classification of varicose veins of the esophagus

There are various classification systems for varicose veins of the esophagus. They are used to assess the risk of bleeding and to select patient management tactics. The classification of varicose veins according to severity is most widely used:

• 1st degree: the diameter of the veins is not more than 5 mm, elongated, located only in the lower third of the esophagus;

• 2nd degree: veins 5-10 mm in diameter, convoluted, located in the middle third of the esophagus;

• 3rd degree: diameter more than 10 mm, tense, with a thin wall, located close to each other, "red markers" on the surface of the veins.

However, many doctors classify varicose veins as large (5 mm or more) or small (up to 5 mm), because in practice it is important to identify the size of the veins that threaten the development of bleeding.

The Bavarian classification of varicose veins of the esophagus uses the following to assess the degree of varicose veins:

• small – minimally raised varicose veins above the surface of the mucous membrane of the esophagus;

• medium – sinuous varicose veins occupying less than a third of the surface of the esophagus;

• large – varicose veins occupy more than a third of the surface of the esophagus.

By localization, there are:

• varicose veins of the lower third of the esophagus, typical of portal hypertension in cirrhosis of the liver. Blood from the portal vein (from the intestine, spleen and stomach) is diverted through alternative routes, forming varicose veins in the submucosal veins of the lower esophagus;

• Varicose veins of the upper or middle thirds of the esophagus are characteristic of obstruction of the superior vena cava. Blood from the head, neck and upper extremities cannot enter the heart directly and bypasses through alternative routes – through the unpaired vein and veins of the upper and middle parts of the esophagus to reach the systemic blood flow.

Symptoms of varicose veins of the esophagus

Many patients with cirrhosis and initial portal hypertension have no obvious symptoms. Varicose veins are detected by chance during screening esophagogastroduodenoscopy.

Sometimes patients may feel slight discomfort or heaviness in the chest, especially after eating, due to the pressure of enlarged vessels on the esophagus. Heartburn and belching may occur, although these symptoms are nonspecific and may be associated with other diseases of the digestive system.

Typical symptoms that precede bleeding include weakness, dizziness, sweating, discomfort, or a feeling of swelling in the lower chest.

A few hours before bleeding, heartburn, belching, difficulty swallowing and other nonspecific manifestations may occur, which are also characteristic of esophagitis (inflammation of the esophageal mucosa), especially since esophageal varicose veins and esophagitis often combine in one patient.

The first minor bleeding may go unnoticed, as the blood immediately enters the intestines. However, at the same time, the stool acquires a dark or black tar-like stool (melena). With increased bleeding, loose stools may turn dark red. Vomiting of scarlet blood may also occur.

The alarming signs of acute blood loss and hypovolemic shock are tachycardia, sweating, weakness (up to loss of consciousness), and a sharp drop in blood pressure.

If there are any signs of bleeding, seek immediate medical attention.

Diagnosis of varicose veins of the esophagus

Esophagogastroduodenoscopy (EGDS) is the method of choice. The study makes it possible to determine the presence, size and shape of varicose veins, signs of a high risk of bleeding, "red

markers" on the surface of the veins, concomitant diseases (esophagitis, gastropathy with portal hypertension, ulceration).

Ultrasound examination of the liver and spleen with determination of blood flow in the portal vein.

Liver elastography determines the degree of fibrosis and fat content in the liver, and helps identify high-risk patients for endoscopy.

Varicose veins of the esophagus are a pathological condition caused by increased pressure in the portal vein of the liver. It can be considered as a compensatory mechanism: against the background of increased portal pressure, the body creates alternative routes for blood flow, forming new vessels connecting the portal venous system with the system of hollow veins. Their role is to drain blood from an overloaded portal vein into the systemic circulation, bypassing the liver. Newly formed vessels are most often located in the submucosal layer of the lower esophagus.

Pathogenesis and causes of portal hypertension

The portal vein (Latin vena portae) plays a key role in the blood supply to the liver, being its main blood vessel. It is through it that most of the blood from the digestive tract enters the liver.

The main cause of portal hypertension is cirrhosis of the liver, which progresses with the proliferation of connective tissue and the formation of regenerative nodes that disrupt the normal architecture of the hepatic vessels. Because of this, the lumen of the vessels narrows, their tortuosity and deformation increase. Even a slight decrease in the diameter of blood vessels provokes a sharp increase in resistance to blood flow inside the liver. To overcome this increased resistance, a significant increase in pressure in the portal venous system is required.

Resistance to blood flow can occur at different levels, forming subhepatic, intrahepatic, and hepatic portal hypertension. More detailed information about the causes of each type of pathology is presented in Table 1.

Table 1. Types of portal hypertension and its causes

Type of Reason

Subhepatic Pressure increase occurs due to obstacles to normal blood flow in the vessels of the hepatolienal system:

• Portal vein thrombosis is a partial or complete blockage of blood flow, causing blood stagnation and increased portal pressure. It can be the result of trauma, tumors, blood clotting disorders, inflammatory processes, and other pathologies.;

• thrombosis of the splenic vein, one of the main tributaries of the portal vein. Its occlusion prevents the outflow of blood from the spleen;

• splanchnic arteriovenous fistula is an abnormal junction between the arteries and veins in the abdominal cavity, in which blood bypasses the capillary bed.

Intrahepatic Intrahepatic portal hypertension occurs due to impaired blood flow inside the liver. The main reasons:

• Schistosomiasis is a parasitic invasion caused by blood flukes of the genus Schistosoma. The parasites migrate to the portal vein, causing inflammation and fibrosis of the liver, which impedes blood flow.;

• Primary biliary cirrhosis is an autoimmune disease in which the small bile ducts in the liver gradually collapse. This process is accompanied by inflammation, fibrosis, and eventually cirrhosis of the liver with impacted blood flow.; The above pathological conditions directly cause portal hypertension, which means that they can be considered the primary cause of the development of varicose veins of the esophagus.

Epidemiology

Vascular pathology due to portal hypertension can occur in any part of the gastrointestinal tract. However, the most characteristic localization is the lower third of the esophagus. According to statistics, approximately 50% of patients with cirrhosis of the liver have esophageal vessels affected, and 30% have stomach vessels.

The development of varicose veins in these parts of the digestive tract is due to anatomical features and the direction of venous blood flow. Under conditions of increased pressure in the portal vein, excess blood rushes through the collateral vessels in a bypass route through the veins of the esophagus and stomach. These vessels, which are not designed for intensive blood flow, gradually stretch and deform, forming varicose veins. In approximately 1-2% of cases, these newly formed veins reach critical sizes, posing an immediate threat of rupture and bleeding.

This complication of portal hypertension is progressive. The existing small varicose veins continue to increase in size over time in a significant proportion of patients — in 4-30%. The change in their size is directly proportional to the risk of rupture and bleeding.

Risk factors

The experts of The World Gastroenterology Organization (WGO) in their clinical recommendations identify 3 key factors indicating a high probability of varicose veins of the esophagus on the background of liver cirrhosis:

• the increased value of the international normalized ratio (INR) is more than 1.5. This indicator reflects disorders in the blood coagulation system characteristic of progressive liver failure;

• an increase in the diameter of the portal vein of more than 13 mm indicates the presence of severe portal hypertension, which leads to the formation of bypass blood flow through the veins of the esophagus and stomach;

• thrombocytopenia is a decrease in the number of platelets in peripheral blood. This condition is typical for patients with decompensated cirrhosis of the liver.

Interpretation:

• in the absence of the above factors, the risk of esophageal varicose veins is relatively low and is less than 10%;

• if a patient has one of them, the probability of developing varicose veins of the esophagus in cirrhosis of the liver increases significantly and reaches 20-50%;

• the presence of two of the three risk factors — the probability is estimated as high (in the range of 40-60%);

• in the presence of all three risk factors, the probability of pathological vascular changes becomes extremely high, exceeding 90%.

If a patient with cirrhosis of the liver has at least one of these risk factors, then the examination should be continued. In such cases, it is recommended to conduct an endoscopic examination in order to visually assess the condition of the vessels of the proximal digestive tract.

Classification

Determining the severity of the pathological process is important for assessing the risk of bleeding and choosing patient management tactics: the higher the degree, the higher the likelihood of complications. There are several classifications of esophageal varicose veins used in clinical practice. Their features are described in Table 2.

Table 2. Pathology classification

Title/Authors Description

ICD-10 In the International Classification of Diseases of the 10th revision (ICD-10), the disease has the I85 code. At the same time:

• pathology complicated by bleeding is encoded by I85.0. This includes cases of blood loss of any severity from pathologically dilated veins of the esophagus;

• I85.9 — without joined bleeding at the time of examination or hospitalization of the patient.

Scherzinger A.G. This classification makes it possible to assess the severity of the disease based on the diameter of the dilated venous vessels determined during endoscopic examination. There are 3 degrees:

• I is the initial stage, characterized by a slight expansion of the veins of the esophagus up to 3 mm in diameter. At this stage, varicose veins are small, soft, and without pronounced tortuosity. The risk of complications is relatively low, but regular monitoring and preventive measures are required.;

• II — moderate degree of pathology. The lumen of the vessels is 3-5 mm. Varicose veins become larger, may have a convoluted course, and a dense consistency. The risk of bleeding increases;

• III — severe course of the disease. The diameter of the nodes is more than 5 mm, they are multiple, form varicose conglomerates. Vessels with pronounced convolution, dense consistency, possible presence of blood clots or erosions on their surface. The risk of bleeding is extremely high, and massive, life-threatening blood loss often develops.

N. Soehendra, K. Binmoeller This classification system is designed for a comprehensive assessment of the severity of pathology, taking into account not only the diameter of varicose veins, but also their prevalence, shape and presence of bleeding risk factors. There are also III degrees of severity of varicose veins of the esophagus:

• I — vessel diameter < 5 mm. They have an elongated rectilinear shape and are localized mainly in the lower third of the esophagus.;

• II — the diameter varies from 5 to 10 mm. Varicose veins become convoluted and spread to the middle third of the esophagus;

• III — vessel diameter >10 mm. They are sharply expanded, swollen, tense, with a thinned wall, tightly adjacent to each other. It is characterized by spreading over the entire length of the esophagus. On the surface of varicose veins, so—called red markers are visualized - areas of erosion or blood clots, indicating a high risk of imminent bleeding.

ESGE Classification (European Society of Gastrointestinal Endoscopy) An important feature of this improved classification is the emphasis not only on the size and shape of varicose veins, but also on the presence of specific endoscopic signs — air injection during insufflation and predictors of bleeding. These additional criteria help to more accurately assess the severity and risk of complications.:

• Grade I (G1, small varicose veins) is the initial stage in which varicose veins are marked in the form of rectilinear, poorly developed vascular formations of small diameter. The key feature of this degree is that when air is injected (insufflated) during endoscopic examination, these altered vessels partially or completely collapse, decreasing in size. This is due to the relatively low intraluminal pressure at this stage of the pathological process.;

• Grade II (G2, moderate varicose veins) — the lumen of the esophagus narrows, but not more than 1/3 of the diameter. Unlike the previous degree, the vascular nodes do not subside with air insufflation, which indicates a higher intraluminal blood pressure.;

• Grade III (G3, large varicose veins) is the most severe form with marked vascular pathology. They are sharply convoluted, strongly inflated, looped, occupy more than 1/3 of the diameter of the lumen. It is characterized by the presence of so-called predictors of a high risk of bleeding: supervarixes (clusters of varicose veins) and "cherry" spots (areas of erosion on the surface of varicose veins).

WGO classification According to the recommendations of the World Gastroenterological Organization, a unified classification has been proposed to standardize the assessment of the severity of this pathology and patient management tactics. The classification uses 2 criteria:

• the size of newly formed vessels;

• the degree of narrowing of the esophagus.

There are:

• small nodes — their diameter does not exceed 5 mm. They protrude minimally above the surface of the mucous membrane of the esophagus, are represented in the form of rectilinear or poorly developed vessels of small diameter.;

• the middle nodes have a convoluted shape, the diameter is increased, but it is less than a third of the circumference of the lumen of the esophagus;

• large — have a diameter of more than 5 mm. Varicose veins occupy more than a third of the lumen of the esophagus, have a convoluted structure and a thinned wall. They fit snugly together, often merging into huge clusters, and their surface may be ulcerated.

Clinical manifestations

Varicose veins of the esophagus are often characterized by an asymptomatic course for a long time: patients do not complain until bleeding develops. It is massive blood loss that becomes the first clear clinical sign of varicose veins of the esophagus of the disease. It is manifested by:

• vomiting with an admixture of bright red, scarlet blood, often in large quantities;

• absence of pain or significant discomfort during bleeding, due to the absence of pain receptors in the walls of blood vessels.

With significant blood loss, a hemorrhagic shock clinic develops due to a sharp decrease in the volume of circulating blood. It includes:

• dizziness;

• general weakness;

• cold sticky sweat;

• pallor of the skin and visible mucous membranes;

• rapid heartbeat;

• reduction of blood pressure down to critical figures;

• disturbances of consciousness, up to coma;

• Multiple organ failure.

Bleeding, as a rule, develops suddenly, which requires increased caution from doctors when managing such patients.

Diagnostics

Comprehensive diagnosis of varicose veins of the esophagus pursues several key goals:

• elucidation of the root cause of portal hypertension, which led to the formation of varicose veins;

• establishment of the exact localization — the vessels of which part of the esophagus are involved in the pathological process, whether there are changes in the veins of the stomach;

• Size and prevalence assessment — endoscopic classification systems are used that take into account diameter, shape, and degree of tortuosity. The larger and more swollen the varicose veins, the higher the risk of rupture.;

• the presence of predictors of a high risk of bleeding — the characteristic appearance of vessels with "cherry spots", supervarixes (cluster-shaped conglomerates of varicose veins);

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