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MALLORY WEISS SYNDROME IN DIFFUSE LIVER LESIONS

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Annotation: Esophageal-gastric bleeding is one of the most common complications of diseases of the gastrointestinal tract caused by liver pathology with the development of portal hypertension. The paper presents the results of complex diagnostics and treatment of patients with bleeding from the upper gastrointestinal tract of varicose and non-varicose character.

Keywords: Mallory-Weiss syndrome, portal hypertension, varicose veins of the esophagus, esophageal bleeding, endoligation

Relevance.

Varicose veins of the esophagus and stomach are not always the only source of bleeding in portal hypertension. In this regard, when conducting an emergency endoscopic examination, it is necessary to exclude hemorrhage of another etiology. In patients with portal hypertension in 10 -28% of cases, the source of bleeding is not associated with varicose veins of the esophagus and stomach. One of the causes of esophageal-gastric bleeding is Mallory–Weiss syndrome, which occupies about 10% of all causes of gastrointestinal bleeding. In Mallory–Weiss syndrome, the source of bleeding is linear ruptures of the mucous membrane in the area of the gastro-esophageal junction, as well as the walls of the stomach in the cardiac department. The rupture of the mucous membrane of the esophageal-gastric junction is facilitated by physical overstrain, more often vomiting, taking coarse food. Changes in blood supply to the mucous membrane of the esophagus and stomach, looseness of the submucosal layer, the formation of cracks and multiple erosions of the mucosa are directly related to portal hypertension against the background of liver cirrhosis.

84 patients with cirrhosis of the liver with portal hypertension syndrome were observed, of which 62 (73.8%) patients had esophageal-gastric bleeding. The combination of portal hypertension with Mallory-Weiss syndrome was found in 16 (25.8%) patients.

The aim of the study was to diagnose bleeding from varicose veins of the esophagus in portal hypertension in combination with Mallory-Weiss syndrome in patients with cirrhosis of the liver.

Material and methods. During the period from 2018 to 2023, 90 patients with cirrhosis of the liver with portal hypertension syndrome were under our supervision. Portal hypertension was complicated by esophageal-gastric bleeding in 72 (80%) patients. The combination of portal hypertension with Mallory-Weiss syndrome was found in 18 (20%) patients. Among them, there were 15 men, 3 women, and the age of patients ranged from 21 to 66 years. To diagnose esophageal-gastric bleeding, clinical and laboratory research methods and urgent esophagogastroscopy were used in the first hours after hospitalization. Subsequently, these patients were subjected to ultrasound, CT and MRI examinations according to indications. As a result of a comprehensive examination, cirrhosis of the liver of various etiologies with portal hypertension syndrome and a high frequency of concomitant diseases were revealed in all patients. Hernias of the esophageal orifice of the diaphragm were found

in 3.7% of the examined, chronic pancreatitis – in 48.8%, chronic gastritis – in 76.2%, gastric ulcer and duodenal ulcer - in 10.7%. In 9.5% of the observations, chronic alcohol consumption was detected, which, undoubtedly, could not but have a negative effect on the mucous membrane of the esophagus and stomach. Mild blood loss at admission was diagnosed in 52.7% of patients, medium – in 34.8% and severe – in 12.5%. Bleeding from varicose veins of the esophagus was also detected in 62 patients, and ruptures of the mucous membrane of the lower third of the esophagus and the cardiac part of the stomach were detected in 16 examined patients.

Results and their discussion. A comprehensive analysis of the literature review and retrospective observation showed that bleeding from varicose veins of the esophagus occurs in 73.8% of patients with cirrhosis of the liver, and with cracks in the mucous membrane of the cardiac esophagus and stomach - in 19% of the subjects. Recently, the frequency of CMV in the structure of acute esophagogastroduodenal bleeding has almost doubled. This is due to the widespread use of EGDS to diagnose the source of bleeding, the introduction of round-the-clock duty of endoscopy specialists, the accumulation of clinical experience, as well as the growth of patients with viral hepatitis and the category of patients with CP of ethanol genesis. In addition, an important factor in increasing the number of patients is the increase in the frequency of concomitant and background diseases, which were detected in 96.4% of cases. Chronic gastritis, duodenitis (84.5%), peptic ulcer of the stomach and duodenum (10.7%), diseases of the biliary tract and pancreas (54.8%), gastroesophageal reflux disease (19%), diseases of the cardiovascular system (15.5%) and other pathologies (13.1%) were most often detected. In patients with CP and varicose veins of the esophagus in combination with Mallory-Weiss syndrome, the source of bleeding was linear ruptures of the mucous membrane in the gastro-esophageal junction, as well as the walls of the stomach of the cardioesophageal region. These ruptures were facilitated by physical overstrain, more often indomitable vomiting after alcohol abuse and coarse food, concomitant diseases of the biliary tract, stomach, duodenum and pancreas. Red blood counts ranged from 2.1 to 4.1 x1012/l and hemoglobin from 49 to 120 g/l. Further examination revealed diffuse liver changes, moderate hepatomegaly in all patients on ultrasound, and splenomegaly in 29 patients. The study of biochemical parameters revealed bilirubinemia from 40-42 to 126 mmol/l; a decrease in the protein-forming function of the liver (52 g / l and below), an increase in the level of transaminases by 1.5-4 times, a coagulogram in the direction of hypercoagulation or hypocoagulation, depending on the stage of development of the underlying disease.

Taking into account these factors and accurate timely diagnosis of this disease, based on clinical and endoscopic data, served as the basis for choosing rational therapeutic tactics for this group of diseases. Treatment of Mallory-Weiss syndrome in combination with portal hypertension consisted of conservative therapy, endoscopic and surgical methods of stopping bleeding. Conservative methods of treatment consisted in restoring the volume of circulating blood by using various colloidal-crystalloid solutions. In case of severe blood loss, hemo- and plasma transfusion (erythrocyte mass and freshly frozen plasma) was used. In patients with CP accompanied by PG and combined with CMV, the use of the Blackmore-Sengstaken probe remains controversial. With CMV, after insertion of the Blackmore probe, increased bleeding from the esophagus and stomach was often noted, which is characteristic of their combination. With control EGDS, they show an increase in the size of the rupture of the esophageal and stomach cardia due to balloon dilation. Given the above facts, we have recently restricted the use of the Blackmore-Sengstaken probe. During endoscopy, three patients were pricked with epinephrine (1:50,000) as a vasoconstrictor. In 5 cases, the administration of adrenaline was combined with electrocoagulation and in 11 cases, sclerosing with ethoxysclerol was performed – 1-3%. A good hemostatic effect when combined with CMV with

varicose bleeding of portal genesis was given by endolygation of bleeding vessels, which we successfully applied in 43 patients. The formation of superficial ulcers at the sites of endoscopic interventions is a natural consequence of the procedure; at the same time, starting from the first day, proton pump inhibitors and enveloping drugs were prescribed that suppress gastric secretion and, thereby, reduce the risk of bleeding. Nevertheless, there are cases of bleeding from the above-mentioned ulcers, which should be reported to the patient. Due to the ineffectiveness of conservative therapy and endoscopic methods of treatment, surgical intervention was performed in 6 (11.3%) cases: transverse gastrotomy in the cardiac part of the stomach and stitching of bleeding vessels in combination with suturing of a deep rupture of the esophageal mucosa. In the postoperative period, 5 (8.1%) patients died. The cause of mortality in the postoperative period was mainly multiple organ failure and progressive encephalopathy. In 2 patients of Child-Pugh functional class C, mortality was associated with peritonitis, which developed as a result of suture failure after gastrotomy. Exacerbation of the underlying disease (chronic hepatitis and cirrhosis of the liver) with suppuration of the postoperative wound and eventration caused death in 1 patient. Thus, Mallory-Weiss syndrome can occur in patients with diffuse liver damage, manifested by portal hypertension from the dystrophically altered mucous membrane of the esophagus and stomach, due to duodenogastric and gastroesophageal reflux, microcirculation disorders.

Treatment of hemorrhagic syndrome in patients with CP in combination with Mallory-Weiss syndrome should be comprehensive, aimed at hemostasis, replenishment of circulating blood volume with mandatory inclusion of hepatotropic and hepatoprotective drugs. One of the ways to improve the treatment of Mallory-Weiss syndrome is early endoscopic methods of stopping bleeding, such as endosclerosis, endoligation and timely surgical intervention if the above methods are ineffective.

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