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Recurrent variceal bleeding in alcoholic liver cirrhosis (a case report)

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Abstract. *Alcoholic liver cirrhosis is widely known to doctors for its complications, including variceal bleeding from the esophagus and/or stomach. Recurrent variceal bleeding is considered a sign of decompensated portal hypertension. As fibrosis in the liver progresses, the gradient of portal pressure increases steadily, and each subsequent bleeding indicates a worsening survival prognosis. There are variety of measures available now to prevent upper gastrointestinal variceal bleeding. In the clinical case, the methods used to prevent repeated bleeding are described. Despite their use, numerous variceal bleedings were registered, which demonstrated the exceptional compensatory capabilities of the patient. In addition, long-term abstinence from the harmful factor, an alcohol, seems to be the most effective measure in this case.*

Keywords: *alcoholic cirrhosis; variceal bleeding; portal hypertension*

Introduction

It is difficult to determine the global frequency of acute variceal bleeding (VB) due to the polyetiological nature of the disease [1]. Epidemiological studies indicate that the most frequent cause of acute bleeding on the African continent is schistosomiasis, while in the eastern part of the world (India, China, Japan), it is more often provoked by viral hepatitis B, C and their combination with delta hepatitis [2]. Alcohol abuse is the most common cause of liver cirrhosis with the development of esophageal varices among the population of America and Europe [3, 4]. As of 2023, mortality from complications of portal hypertension in Ukraine is 47.3 cases per 100,000 population, which ranks third in the structure of total mortality, and has not had a significant downward trend since 2015 [20].

The prevalence of esophageal varices correlates with the degree of compensatory capacity of the liver according to the Child-Pugh scale: it is reported to be 42 % in the compensated stage, 71 % in the subcompensated stage and 76 % in decompensated cirrhosis [5]. The occurrence of acute VB estimated to carry a 10–20% mortality rate [6, 7].

However, recent research violates the concept of irreversible changes in the liver parenchyma in cirrhosis. The question of liver compensation in the absence of an impressive factor is being discussed more and more in general [11, 19].

We present a clinical observation of the treatment of a patient with decompensated portal hypertension (PH) caused by alcoholic liver cirrhosis, who had fourteen acute VB.

The purpose of the study was to demonstrate the regenerative potential of the liver parenchyma and rapid rehabilitation of the patient with multiple profuse variceal bleedings, provided there are abstinence intervals.

Materials and methods

The main methods of research we used were a scientific search and our own description of a clinical case. Laboratory methods, ultrasound examinations and spiral computed tomography were applied to confirm the diagnosis and carry out differential diagnosis. The endoscopic method was used for diagnosis and treatment (endoscopic ligation of esophageal varices with latex rings). Computed tomography was performed with intravenous enhancement of water-soluble contrast (iohexol) in the portal phase of blood flow followed by computer 3D modeling.

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Case presentation

In the first hours after hospitalization, the Child-Pugh score was within the limits of class B: total bilirubin — 25.3 μmol/l, albumin — 31 g/l, INR — 1.78, moderate ascites, encephalopathy of the second grade. Other blood parameters are given in Table 1.

Table 1 — Biochemical and general blood tests, coagulogram

Blood parameters	Value
Total protein, g/L	53
Direct bilirubin, μmol/L	6.3
Indirect bilirubin, μmol/L	19
ALT, mmol/L	0.92
AST, mmol/L	1.83
α-amylase, U/L	114
Creatinine, μmol/L	67
Urea, mmol/L	5.2
White blood cells, $\times 10^9$/L	2.8
Red blood cells, $\times 10^{12}$/L	2.03
Hemoglobin, g/L	72
Platelets, $\times 10^9$/L	46
Hematocrit, %	21.5
Prothrombin time, sec	16
Prothrombin index, %	80
Fibrinogen, g/L	2.4

Treatment with hemostatic agents (tranexamic acid, vitamin K), antibiotics (rifaximin), beta-blockers (carvedilol), octreotide, and placement of a Sengstaken-Blakemore tube in the esophagus for three days led to the cessation of bleeding, which resumed on the seventh day and required re-insertion of the tube with hemoglobin values < 40 g/l and continued treatment, including erythrocyte transfusion.

Abdominal and thoracic computed tomography with intravenous contrast enhancement was performed to assess the spread of portosystemic shunts.

Since 2018, the patient has had four episodes of gastrointestinal bleeding (GIB) due to the formation of alcoholic cirrhosis of the liver, which required inpatient treatment and massive blood transfusions. In 2020, he underwent operation due to recurrent bleeding from the esophageal varices of the third degree. The procedure included devascularization of the abdominal part of the esophagus and the proximal part of the stomach, stapler circular transection of the esophagus in the epicardial area.

An intraoperative biopsy in an enlarged liver with many macronodular nodes showed fibrotic-cirrhotic changes in the parenchyma.

There was an abstinence from alcohol during the three postoperative months. Further, alcohol abuse, despite the treatment for alcohol dependence, resumed, which was accompanied by the recurrent bleeding from the esophageal varices. Hemostatic therapy, use of beta-blockers, endoscopic ligation of the esophageal varices, as well as treatment for alcohol addiction had a short-term effect and accompanied by repeated VB. A restrictive strategy of blood transfusion was used, which did not worsen biochemical indicators under conditions of severe blood loss.

During five years, the patient had fourteen variceal bleedings, which required hospital treatment. Transjugular intrahepatic portosystemic shunt was not applied due to lack of technical possibility to perform the procedure. At the time of writing this case report, the patient is alive. He is recommended to strictly abstain from any alcohol consumption, to continue taking non-selective beta-blockers (carvedilol), to consider the possibility of endoscopic ligation of esophageal varices in order to prevent new cases of variceal bleeding.

Discussion

GIB is one of the most common causes of death in patients with cirrhosis and clinically significant PH. Gastroesophageal varices is the most common source of upper GIB in patients with PH [8]. The main factor in the development of esophageal VB is clinically significant PH [9]. In a sense, the acute loss of intravascular volume due to hemorrhage reduces splanchnic pressure and may stop hemorrhage. In contrast, sudden restoration of intravascular volume is associated with

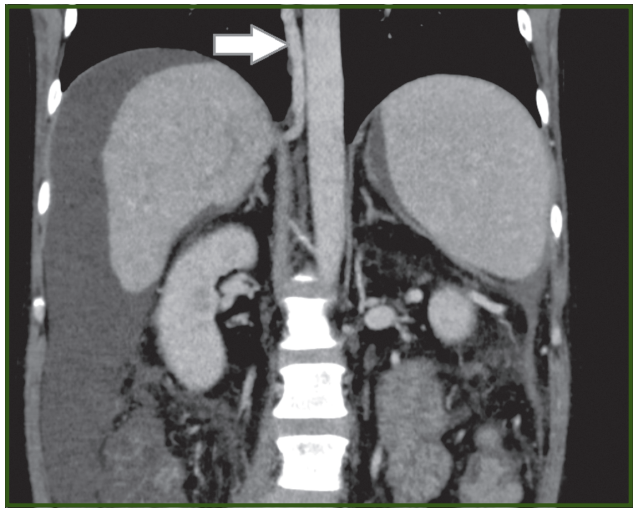

Figure 1 — Monochrome 2D image with contrast enhancement of the portal venous system. The arrow shows the portosystemic collateral to azygos vein

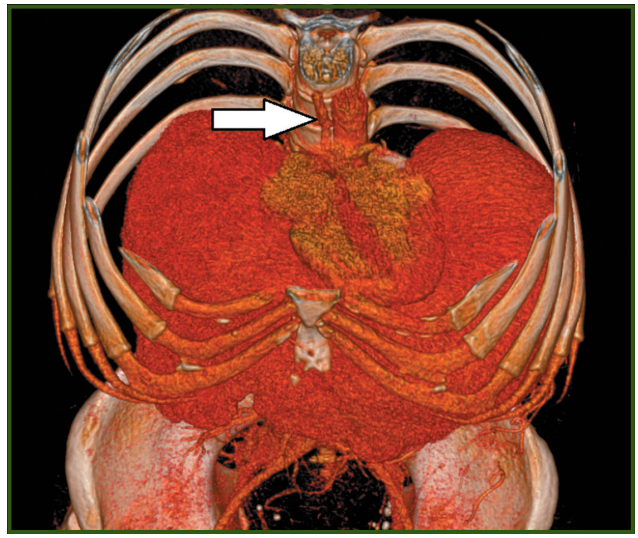

Figure 2 — 3D CT modeling. The arrow indicates the portosystemic collateral to the right of the esophagus

a sudden increase in portal pressure, which in turn may lead to failure to control bleeding and/or early recurrent bleeding [10]. According to Baveno VII, transfusion of fresh frozen plasma for acute VB is not recommended, because it will not correct the coagulopathy and may lead to volume overload and worsening portal pressure [11]. Hospitalized patients with end-stage liver disease have pronounced coagulopathy, but prolonged prothrombin time does not reflect an increased tendency to bleeding in these patients, so INR correction with fresh frozen plasma should not be performed [12, 13]. Somatostatin and vasopressin analogs (octreotide, terlipressin) exert their effect by reducing splanchnic blood flow, thus decreasing portal pressure [1, 5, 8, 14]. They are very effective, and a meta-analysis has clearly shown that the use of these vasoconstrictors is associated with a significantly higher probability of bleeding control and lower seven-day mortality [15]. In patients with uncontrolled bleeding, esophageal balloon tamponade with a Sengstaken-Blakemore tube is recommend, but it carries a high risk of complications, in particular respiratory infection [16]. Large international observational study has confirmed a beneficial effect on survival of preemptive transjugular intrahepatic portosystemic shunt only in patients with Child-Pugh C (score 10–13), but not in those with Child-Pugh B with active bleeding [17]. Alcohol-associated hepatitis usually progresses to liver cirrhosis if alcohol abuse continues, but in those who stop it, the hepatitis regresses within a few months, while the cirrhosis that has already occurred remains [18].

Conclusions

1. The main goal is to demonstrate the possibilities of hepatocyte regeneration in alcoholic cirrhosis and multiple profuse variceal bleedings under the condition of periodic abstinence.
2. Recurrences of bleeding from esophageal varices depend on the intensity of alcohol damage, and abstaining from alcohol in combination with pathogenetic treatment has positive results.
3. There are reasons to consider a restrictive strategy of blood transfusion decisive for the survival in this clinical case in combination with pathogenetic treatment, even after fourteen acute variceal bleedings.

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Повторні варикозні кровотечі при алкогольному цирозі печінки (клінічний випадок)

Резюме. Алкогольний цироз печінки відомий лікарям своїми ускладненнями, у тому числі кровотечами з варикозно розширених вен стравоходу та/або шлунка. Повторні варикозні кровотечі вважаються ознакою декомпенсованої портальної гіпертензії. У міру прогресування фіброзу в печінці градієнт портального тиску неспинно зростає, що вказує на погіршення прогнозу виживання з кожною наступною кровотечею. Сьогодні доступне різноманіття заходів для профілактики варикозних кровотеч. У клінічному випадку описано методи, які були застосовані для запобігання повторній кровотечі. Попри їх використання, зафіксовано численні варикозні кровотечі, що продемонструвало виключні компенсаторні можливості пацієнта. Крім того, найефективнішим заходом у цьому випадку було утримання від ушкоджуючого фактора — алкоголю.
Ключові слова: алкогольний цироз; варикозна кровотеча; портальна гіпертензія