Pierik, E. G. Endoscopic versus open subfascial division of incompetent perforating veins in the treatment of venous leg ulceration: a randomized trial / E. G. Pierik, H. Van Urk, W. C. Hop, C. H. Wittens // J. Vasc. Surg. – 2017. – № 26. – P. 1049–1054.
Endoscopic interventions with insufficiency of perforant veins of the lower extremities / A. Pietravallo [at al.] //

Phlebolymphology. – 2003. – № 19. – P. 5–12.

УДК 616.36-004-036.8 Imran Mohamed Adam, Angage Madusha Menuwanthi Perera

Scientific supervisor: Ph.D., Associate Professor A. G. Skuratov Educational institution

«Gomel State Medical University» Gomel, Republic of Belarus

THE CAUSES OF DEATH IN LIVER CIRRHOSIS PATIENTS

Introduction

Liver cirrhosis (LC) and its complications remain relevant today, despite the progressive development of modern medicine. LC is a continuously progressive disease, the mortality rate of which remains high (up to 35 per 100,000 population per year). The methods of medical examination of these patients aimed at identifying severe forms of the disease and preventing life-threatening complications do not solve the problem; their improvement is required. At the outpatient and inpatient stages, patients receive only symptomatic palliative care and ineffective pathogenetic and etiotropic treatment. With the development of complications of LC during the decompensation of portal hypertension, patients are hospitalized in the surgical department or the intensive care unit. The only radical method of treatment for terminal forms of LC is liver transplantation, which remains inaccessible to most patients for a number of reasons [1, 2, 3].

Goal

The aim of the present article is to analyze the structure of deaths in patients with liver cirrhosis.

Material and Methods of research

A complete non-randomized study was conducted based on a retrospective analysis of 67 autopsy protocols of patients with a postmortem diagnosis of LC. The following indicators were evaluated: gender and age of patients; duration of inpatient treatment; clinical and pathoanatomic diagnoses; and results of instrumental and laboratory research methods.

The results of the research and their discussion

The analysis of gender identity showed that there were no differences: of all the deceased men, there were 33 (49 %), and of all the deceased women, there were 34 (51 %). The average age for men was 56.1 years; for women, it was 60.5 years. The share of able-bodied age (35–65 years) accounted for the majority of male patients (75 %); among women of working age (from 40–63 years), there were 15 patients (44.1 %). The average duration of inpatient treatment was 7.9 days; mortality on the first day occurred in 25 cases (37.3 %).

The structure of the etiological characteristics of liver cirrhosis is represented by the following indicators: Toxic-alimentary LC was detected in 14 cases (20.9 %), of which alcohol addiction was confirmed in 5 patients; LC of viral etiology was detected in 13 cases (19.4 %), of which 12 people (92.3 %) had viral hepatitis C, and 1 person (7.7 %) had viral hepatitis B; cardiac LC on the background of heart failure was noted in 3 cases (4.5 %); LC on the background of Budd-Chiari syndrome (inferior vena cava syndrome) was detected in 1 case (1.5 %). However, in 35 patients (52.2 %), the etiological factor of LC was not confirmed, i.e., LC was cryptogenic.

According to the Child-Pugh LC classification, class C was present in most patients: 57 (85.1 %), i.e., in the vast majority of cases, the LC was in the decompensation stage.

LC was accompanied by complications in all patients: in 43 cases (62.9 %), portal hypertension with esophageal varicose veins (EVV) and bleeding from them, ascites, and splenomegaly; in 34 cases (50.1 %), hepatorenal syndrome; and in 18 (26.9 %) patients, hepatic encephalopathy.

The data from laboratory and instrumental research methods characterized the decompensated stage of LC and portal hypertension: dilatation of the portal vein up to 15–17 mm, according to ultrasound data, occurred in 64 (95.5 %) patients. Posthemorrhagic anemia of mild severity occurred in 6 (13.9 %) patients, moderate in 14 (32.5%), and severe in 13 (30.2 %) patients with the development of bleeding from EVV. Laboratory signs of liver failure: hyperbilirubinemia was detected in 55 patients (82.1 %); the maximum value was 310.4 mmol/l; hypoproteinemia (up to 39 g/l) was detected in 42 (62.7 %) cases; The level of aspartate aminotransferase (AST) was increased (up to 525 units/L) in 54 (80.1 %) patients; alanine aminotransferase (ALT) was elevated (up to 507 U/L) in 38 (56.7 %) patients; and a decrease in the prothrombin index (PTI) was detected in 56 (83.6 %) patients.

The medical care provided for bleeding from the EVV consisted of palliative measures: setting up a Blackmore's obturator tube in 8 cases, endoscopic hemostasis in 2 cases, replacement of the blood volume, and hemotransfusion.

When analyzing the structure of the causes of deaths in LC, it was found that of all the deceased, the diagnosis of "liver cirrhosis" as a cause of death without specifying complications was made in 5 (7.5 %) patients; posthemorrhagic anemia associated with bleeding from EVV was made in 13 cases (19.4 %); hepatorenal syndrome was the cause of death in 18 cases (26.9 %), liver failure – in 2 cases (3 %), alcohol addiction syndrome – in 1 case (1.5 %); posthemorrhagic anemia not associated with bleeding from the EVV (chronic duodenal ulcer with bleeding) – 1 (1.5 %). In this group, all patients had decompensated LC (Child-Pugh severity class C).

In 21 cases (31.3 %), if the patient had LC as a concomitant disease, the cause of death was another pathology: cerebral edema in 7 cases (28%), coronary atherosclerosis in 5 (20%), myocardial infarction in 2, leukemia in 1, liver cancer in 1, pulmonary embolism in 1, pneumonia in 1, peritonitis in 1, etc. At the same time, in this group, the LC for severity class A was in 2 patients, severity class B was in 8, and severity class C was in 11 patients.

Conclusions

Analysis of the structure of patients and causes of deaths according to autopsies showed that there were no differences in the number of deaths from LC among men and women by gender; patients of working age (from 35 to 65 years) prevailed. The etiology of LC was not confirmed in half of the cases (52.2 %), and the main causes of LC were toxic-alimentary and viral (mainly viral hepatitis C). In all cases in which LC was confirmed as the cause of death, the disease reached the terminal stage (Child-Pugh severity class C), in which complications developed that directly led to death: decompensated portal hypertension with bleeding from the EVV, acute hepatic-renal insufficiency, and liver encephalopathy. Mortality on the first day was noted in 37.3 % of cases. High numbers may indicate an insufficiently effective medical examination at the outpatient stage, as well as a low social level and low compliance among patients in this category. LC is a chronic, continuously progressive disease that can be asymptomatic for a long time with the development of irreversible changes in the liver. Thus, untimely diagnosis and limited effective ways of treating the disease often lead to fatal outcomes in people of working age.

LITERATURE

1. Ivashkin, V. T. Complications of portal hypertension in cirrhosis of the liver / V. T. Ivashkin // Russian Physiological Journal. $-2009. - N_{2} 10. - P. 74-76.$

3. Franchis, R. Expanding consensus in portal hypertension: Report of the Baveno VI Consen-sus Workshop: stratifying risk and individualizing care for portal hypertension / R. Franchis // J. Hepatol. – 2015. – N 63. – P. 743–752.

^{2.} Liver cirrhosis / P. Gines [et al.] // Lancet. - 2021. - № 398. - P. 1359-1376.