showed the following median values: total bilirubin – 20.1 μmol/L (range: 4.9–40.4 μmol/L) and LDH–289.3 U/L (range: 174–469 U/L). Significant reductions in haptoglobin levels were observed in 70.5% of cases. Hemoglobin electrophoresis revealed the following average values of hemoglobin fractions: HbA – 52.6% (range: 0.292–91.3%), HbF – 2.9% (range: 0–9.2%), and HbA2 – 2.7% (range: 0.023–6.3%). These results played a crucial role in confirming the diagnosis of thalassemia. Most patients (70.6%) were diagnosed with ß-thalassemia minor, with only one case of severe thalassemia diagnosed at the age of 1 year in a girl with a family history of major beta-thalassemia (father).

Conclusion

In the structure of HA in children, ß-thalassemia accounted for 21.3%. Only one severe case was diagnosed. Initial diagnosis of thalassemia was characterized by morphological similarities to IDA (microcytic hypochromic type, in some cases – normoregenerative). A characteristic feature of thalassemia was the presence of target cells in 88.2% of peripheral blood smears and 100% of laboratory biochemical signs of hemolysis. The main confirmatory test was a decrease in HbA level with an average value of 52.6%, as determined by hemoglobin electrophoresis.

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CIRCLES OF HEALING: THE JOURNEY THROUGH LIVER CIRRHOSIS

Introduction

Cirrhosis is the outcome of chronic Liver disease of any etiology due to progressive Liver injury and fibrosis. Consequently, cirrhosis leads to portal hypertension and Liver dysfunction, progressing to complications like ascites, variceal bleeding, hepatic encephalopathy, hepatorenal syndrome, hepatopulmonary syndrome, cirrhotic cardiomyopathy, sarcopenia, hepatocellular carcinoma, and coagulation disorders. End-stage liver disease leads to an impaired quality of life, loss of social and economic productivity, and reduced survival [1]. Among the leading causes of death liver cirrhosis took the spot number 12 in 2021 when deaths from this disease reached 1.3 M and thus 1.9% in total. Liver cirrhosis is accompanied by complications such as portal hypertension (ascites, esophageal varices, portal hypertensive gastropathy), hepatic encephalopathy, hemorrhagic diathesis and bleeding, acute kidney injury, cardiac rhythm and conduction disorders [2–4]. Early detection and management of the underlying causes are crucial to slowing the progression of the disease and improving patient outcomes.

Goal

The purpose of this study is to analyze the complication of Liver Cirrhosis

Material and methods of research

A cross-sectional, single-center study involving 25 patients with liver cirrhosis was conducted at the gastroenterology department of Gomel State Clinical Hospital No. 3 in January 2025. This study aimed to understand the clinical presentations and complications associated with liver cirrhosis in the hospitalized population of Gomel. Through this detailed analysis, we sought to identify patterns and insights that could enhance the management and treatment of liver cirrhosis in similar clinical settings.

25 people of different age groups of Belarus has participated in this study, of which 11 participants belonged to 30–49 age group, 4 participants were from 50–59 age group and 10 participants were from 60–79 age group. Among them 60% were Male and 40% were Female (tab. 1).

Table 1 — Structure of patients with liver cirrhosis

Category	Subcategory	Number of patients
Viral Hepatitis	Hepatitis B, Hepatitis C	2(8%)
Alcohol Abuse	Long term Alcohol Abuse	6(24%)
Metabolic Disorders	Non Alcoholic Fatty Liver Disease (NAFLD) Hemochromatosis	2(8%) 2(8%)
Autoimmune Diseases	Autoimmune Hepatitis	2(8%)
Billiary Diseases	Primary Biliary Cirrhosis Primary Sclerosing Cholangitis	3(12%) 1(4%)
Cryptogenic		7(28%)

The results of the research and their discussion

Portal hypertension was found in all patients, with 8 patients (32%) in the Compensative Stage, 10 patients (40%) in the Subcompensative Stage, and 7 patients (28%) in the Decompensative Stage. Ascites affected 68% of the patients: 6 patients (24%) were in Stage 1, 2 patients (8%) in Stage 1–2, 5 patients (20%) in Stage 2, 1 patient (4%) in Stage 2–3, and 3 patients (12%) in Stage 3. Esophageal varices were seen in 64% of the patients: 3 patients (12%) in Stage 0–1, 7 patients (28%) in Stage 1, 2 patients (8%) in Stage 1–2, and 4 patients (16%) in Stage 2. Hepatic encephalopathy (HE) was present in 60% of the patients: 3 patients (12%) in Stage 0–1, 10 patients (40%) in Stage 1, and 2 patients (8%) in Stage 2. Splenomegaly was noted in 48% (12 patients), hepatomegaly in 12% (3 patients), and hydrothorax in 12% (3 patients), with 1 (4%) having right-sided hydrothorax and 2 (8%) having bilateral hydrothorax.

In this experience we encounter various symptoms, among these symptoms were swelling from the shin to the feet, making mobility difficult and causing discomfort. General weakness was prevalent, leaving patients fatigued and impacting their daily activities. A noticeable increase in the volume of the abdomen was observed, indicating advanced liver disease. Drowsiness was common, affecting alertness and cognitive function. Additionally, jaundice was evident, characterized by a yellowing of the skin and eyes, signaling severe liver impairment.

Other symptoms of liver cirrhosis included loss of appetite, nausea, muscle wasting and weight loss. Patients often experienced easy bruising and bleeding due to decreased production of blood clotting factors. Itchy skin was also a frequent complaint, caused by the accumulation of bile products in the skin. Furthermore, confusion and memory problems, known as hepatic encephalopathy, were seen in more advanced cases, affecting cognitive functions. Spider-like blood vessels on the skin, known as spider angiomas, palmar erythema, gynecomastia and caput medusae were also observed. These symptoms, when combined, lead to a range of complications (tab. 2).

Table 2 — The other symptoms in liver cirrhosis

Symptoms/Signs	Number of Patients	Symptoms/Signs	Number of Patients (%)
General Weakness	16 (64%)	Leg Edema	11 (44%)
Nausea	2 (8%)	Jaundice	10 (40%)
Right Hypochondrium Heaviness	3 (12%)	Spider Angiomas	2 (8%)

Conclusions

In light of these points, it is evident that 100% of the hospitalized patients with liver cirrhosis got Portal hypertension, 68% Ascites, 64% Esophageal varices, 60% Hepatic Encephalopathy, 48% Splenomegaly, 12% Hepatomegaly and 12% Hydrothorax (4% right sided, 8% bilateral).

Raising awareness and ensuring timely medical care are essential in improving outcomes for individuals with liver cirrhosis. And evidently, no disease arises without underlying causes that contribute to its development. Therefore, we recommend:

1. Implementing public health policies, such as increasing alcohol taxation, restricting sales, and promoting alcohol cessation programs

2. Widespread vaccination against hepatitis B (HBV) and early detection and treatment of hepatitis C (HCV)

3. Given the rising prevalence of non-alcoholic fatty liver disease (NAFLD) and non- alcoholic steatohepatitis (NASH), lifestyle interventions, weight management, and pharmacological treatments

4. High-risk populations (e.g., individuals with chronic HBV, HCV, or metabolic dysfunction) should undergo routine liver function tests, elastography, and non-invasive fibrosis assessments to enable early intervention before cirrhosis develops.

5. Identifying and minimizing exposure to hepatotoxic substances, including certain medications industrial toxins, and herbal supplements

6. Exploring novel antifibrotic therapies, stem cell treatments, and gene editing techniques holds potential in reversing liver damage at an early stage.

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