

**PREVALENCE AND CAUSES OF LIVER CIRRHOSIS IN DIFFERENT REGIONS  
LITERATURE REVIEW**

**Ravzatov Jasurbek Bahromovich.**

PhD, Associate Professor, Department of Family Physician Training, Andijan State Medical Institute.

**Nabieva Dildora Abdumalikovna.**

Head of the Department of Occupational Pathology, Faculty No. 1 and Hospital Therapy, Tashkent Medical Academy, Doctor of Medical Sciences, Professor.

**Annotation.** Chronic liver diseases, including fibrosis and cirrhosis, can be associated with chronic malnutrition. Malnutrition, i.e. less than the required amount of food intake (hypoalimentation), is often caused by a misunderstanding of the meaning of the term "liver diet", slowing down of food digestion processes (including secretion of bile acids), metabolic disorders consisting of limitation of protein synthesis and increased catabolism and acceleration of processes.

**Key words:** Liver cirrhosis, decompensated cirrhosis, steatohepatitis, hepatitis B, hepatitis C.

Liver cirrhosis (LC) is a diffuse process, manifested by a change in its normal structure as a result of fibrosis and nodule formation, and in most cases occurs in the final stages of chronic diffuse diseases of the organ [5: 245-266]. In the early stages of the disease, it proceeds asymptotically (compensation stage), and later the process is accompanied by an increase in pressure in the portal system and deterioration of liver function. This is accompanied by the appearance of clinical signs in the form of complications of LC (decompensation stage). In the compensation stage, most patients, as a rule, do not experience significant changes in their quality of life and can remain latent for several years. In the decompensation phase, patients develop a variety of clinical signs, including ascites (fluid accumulation with or without infection), bleeding from esophageal and gastric varicose veins, hepatic encephalopathy, hepatorenal and hepatopulmonary syndrome, cirrhotic cardiomyopathy, and secondary adrenal insufficiency and infection-related complications (spontaneous bacterial peritonitis, urinary tract infection, pneumonia, soft tissue infection, and bacteremia).

The most recent available data on the global prevalence of CVD are provided in the 2017 Global Survey of Diseases, Injuries and Risk Factors.

This study, based on pooled epidemiological data from 195 countries and regions, reports the incidence of cirrhosis by cause, age, and sex from 1990 to 2017. The results are presented as prevalence rates and age-standardized or age-specific rates per 100,000 population, with 95% confidence intervals (CIs) and relative risks. In 2017, 112 (CI 107-119) million people worldwide had compensated cirrhosis and 10.6 (CI 10.3-10.9) million people in decompensated cirrhosis. In 1990, these figures were 65.9 (CI 63.4-68.7) and 5.20 (CI 5.08-5.32) million, respectively. The figures show that the number of patients with cirrhosis has almost doubled during these years. The age-standardized prevalence of compensated cirrhosis increased from 1354.5 (CI 1300.6-1411.7) per 100,000 population in 1990 to 1395.0 (CI 1323.5-1470.5) in 2017, while the decompensated stage of the disease was 110.6 (CI 108.0-113.0) and 132.5 (CI 128.6-136.2), respectively. According to the 2017 study, 58.8% of compensated cirrhosis and 60.3% of decompensated cirrhosis were observed in men. These figures give reason to assume that men are more likely to have cirrhosis. When analyzed in

age-standardized terms among patients with advanced disease, the prevalence of compensated cirrhosis increased by 2.9% from 1990 to 2017. The prevalence of decompensated cirrhosis increased by 21.1%. In women, these figures were 3.5% and 18.1%, respectively. Overall, the prevalence of cirrhosis increased by 74.53% from 1990 to 2017 [13: 279-300].

The Global Diseases, Injuries and Risk Factors Study describes the epidemiologic characteristics of LC at the regional level [5: 245-266]. In 2017, the highest age-standardized prevalence of compensated and decompensated hepatitis was observed in the economically developed countries of the Asia-Pacific region, with 2455.0 (CI 2344.9-2575.8) and 267.4 (CI 259.8-275.1) cases per 100,000 population, respectively. It should be noted that most of this negative change was observed in the Japanese population and is associated with a high incidence of hepatitis C among citizens of this country. On the contrary, data on the low prevalence of compensated and decompensated hepatitis in Australia are presented, but they also indicate that hepatitis C is the main cause of the disease. The lowest standardized prevalence of compensated hepatitis (mainly due to hepatitis C) was observed in economically developed countries of the North American region. The lowest prevalence of decompensated hepatitis was observed in South Asia. When examined across countries, the highest prevalence of compensated HCV was observed in patients living in Moldova, Taiwan, and Slovakia. The detection of those in the decompensated stage was lowest in the Philippines and highest in Slovakia.

Recent studies also provide statistical data on the prevalence of the disease, taking into account the etiology of HCV. A systematic review of 520 studies conducted in 86 countries and territories worldwide, including a total of 1,376,503 HCV patients (observations conducted until August 1, 2021), showed that 42% of patients identified worldwide had chronic hepatitis B and 21% had hepatitis C infection [7: 724-735].

The prevalence of hepatitis B virus infection was higher in Africa and Asia (8%-61%) than in Europe, the Americas, and Oceania (3%-14%). The prevalence of hepatitis C virus infection varied significantly across countries and regions (12%-83%). Overall, the prevalence of these two infections was greater than 50% in most parts of Asia and Africa. In China, 68% (95% CI 60%-74%) of patients with hepatitis B virus infection were infected with hepatitis C virus, while only 7% (95% CI 5%-9%) were infected with hepatitis C virus [7: 724-735].

When age-standardized analysis was performed, the prevalence of compensated hepatitis B virus-associated hepatitis in 2017 did not change significantly compared to 1990, but the prevalence of decompensated hepatitis increased from 30.9 (95% CI 29.3-32.2) to 36.6 (95% CI 34.7-38.4) per 100,000 population. When these indicators were examined in relation to hepatitis C, the prevalence of compensated hepatitis B virus-associated.

The highest prevalence rates of CVD due to alcohol abuse are observed in Europe (16%-78%), North and South America (17%-52%), and Oceania (15%-37%), with the lowest rates in Asia (0%-41%).

In 2017, the age-standardized global prevalence of alcohol-related compensated cirrhosis remained stable compared to 1990 (288.1 per 100,000 population in 2017 and 290 in 1990). However, the global prevalence of the disease in the decompensated stage due to this cause has increased since 1990 (increasing from 25.3 per 100,000 population in 1990 to 30 in 2017) [5: 245-266]. It should be noted that in 2017, 9.42 million (II 8.57-10.34) patients in the compensation stage of JTs developed due to non-alcoholic fatty liver disease and 917,000 (II 850000-986000) in the decompensation stage

due to steatohepatitis were recorded. These figures show that the disease has increased significantly compared to 1990.

The age-standardized prevalence of compensated NAFLD was 115.5 (CI 105.0-126.5) per 100,000 population in 2017, an increase of 33.2% compared to 1990. The prevalence of decompensated NAFLD was 11.3 (CI 10.4-12.1) per 100,000 population in 2017, an increase of 54.8% compared to 1990. Although NAFLD is often caused by adverse drug reactions, autoimmune diseases, metabolic disorders, and other causes, there is no information on its global or regional prevalence in the literature [5: 245-266].

A study conducted in the United States analyzed statistical data on the prevalence and severity of digestive diseases among the insured adult population between 2016 and 2018. Of the total population studied, 7,297,435 (24%) were diagnosed with digestive diseases, and 0.389% (0.090% of the total population) of them were diagnosed with non-alcohol-related CVD.

Gu et al. studied all hospitalizations associated with a diagnosis of CHD in Germany between 2005 and 2018. They found that a total of 248,085,936 patients were hospitalized during this period, of which 2,302,171 were diagnosed with CHD, representing a prevalence of 0.94%.

In a comparative study conducted in Japan in 2020, 6,000 citizens from 2 cities in the country were monitored, and 488 of them were screened for fatty liver disease and its fibrosis. Based on the detection of liver stiffness, the prevalence of cirrhosis among the subjects was 1%, with a significantly higher incidence in men than in women (1.6% and 0.4%, respectively) [5: 245-266].

Another observational study using a database of physician referrals found that the prevalence of LC in adults was 0.21% in 2018, with a population of 536,856. According to the US National Health and Nutrition Examination Survey (NHANES), a cross-sectional study conducted in 2017-2018 among 825 adults with type 2 diabetes who had reliable transient elastography results found a prevalence of CVD of 7.7% (95% CI 4.8%-11.9%).

#### **Literature:**

1. Aghdassi AA, Schneider A, Kahl M, et al. Analysis of lifestyle factors in patients with concomitant chronic pancreatitis and liver cirrhosis. *Pancreatology*. 2017;17(5):698-705
2. Aldana Ledesma JM, V6zquez Rodriguez D, Lazcano Becerra M, Garcia Jim6nez ES, Tapia Calderyn DK, Ibarra Estrada MB, F6lix T6llez FA, Velarde Ruiz Velasco JA. [Comparison of different tools for the evaluation of malnutrition and sarcopenia in patients with liver cirrhosis] *Nutr Hosp* 2023; 40: 340-346
3. Grungreiff K, Gottstein T, Reinhold D, Blindauer CA. Albumin Substitution in Decompensated Liver Cirrhosis: Don't Forget Zinc. *Nutrients* 2021; 13; Fernandez J, Angeli P. et al, Efficacy of Albumin Treatment for Patients with Cirrhosis and Infections Unrelated to Spontaneous Bacterial Peritonitis. *Clin Gastroenterol Hepatol* 2020; 18: 963-973.e14
4. Fagerstr6m C, Frisman GH. Living with liver cirrhosis: a vulnerable life. *Gastroenterol Nurs*. 2017;40(1):38-46
5. Hansen L, Leo MC, Chang MF, Zaman A, Naugler W, Schwartz J. Symptom distress in patients with end-stage liver disease toward the end of life. *Gastroenterol Nurs*. 2015;38(3):201-210.
6. Janota B, Krupowicz A, Noras K, Janczewska E. Evaluation of the nutritional status of patients with liver cirrhosis. *World J Hepatol* 2023; 15(7): 914-924
7. Janota B, Krupowicz A, Noras K, Janczewska E. Evaluation of the nutritional status of patients with liver cirrhosis. *World J Hepatol* 2023; 15(7): 914-924

8. Kalal C, Benjamin J, Shasthry V, Kumar G, Sharma MK, Joshi YK, Sarin SK. Effect of long-term aggressive nutrition therapy on survival in patients with alcohol-related cirrhosis: A randomized controlled trial. *Indian J Gastroenterol* 2022; 41: 52-62; Aldana Ledesma JM, V6zquez Rodriguez D, Lazcano Becerra M, Garcia Jim6nez ES, Tapia Calderyn DK, Ibarra Estrada MБ, F6lix T6llez FA, Velarde Ruiz Velasco JA. [Comparison of different tools for the evaluation of malnutrition and sarcopenia in patients with liver cirrhosis] *Nutr Hosp* 2023; 40: 340-346
9. Kim HJ, Chu H, Lee S. Factors influencing on health-related quality of life in South Korean with chronic liver disease. *Health Qual Life Outcomes*. 2018;16(1):142. doi:10.1186/s12955-018-0964-1.; Loria A, Escheik C, Gerber NL, Younossi ZM. Quality of life in cirrhosis. *Curr Gastroenterol Rep*. 2013;15(1):301.
10. Marra M, Sammarco R, De Lorenzo A, Iellamo F, Siervo M, Pietrobelli A, Donini LM, Santarpia L, Cataldi M, Pasanisi F, Contaldo F. Assessment of Body Composition in Health and Disease Using Bioelectrical Impedance Analysis (BIA) and Dual Energy X-Ray Absorptiometry (DXA): A Critical Overview. *Contrast Media Mol Imaging* 2019; 2019: 3548284
11. Łapiński TW, Łapińska M. Nutritional status in patients with liver cirrhosis. *Clinical and Experimental Hepatology*. 2019;5(1):30-34. doi:10.5114/ceh.2019.83154
12. Levitt DG, Levitt MD. Human serum albumin homeostasis: a new look at the roles of synthesis, catabolism, renal and gastrointestinal excretion, and the clinical value of serum albumin measurements. *Int J Gen Med* 2016; 9: 229-255
13. Loria A, Escheik C, Gerber NL, Younossi ZM. Quality of life in cirrhosis. *Curr Gastroenterol Rep*. 2013;15(1):301.
14. Luengpradidgun L, Chamroonkul N, Sripongpun P, Kaewdech A, Tanutit P, Ina N, Piratvisuth T. Utility of handgrip strength (HGS) and bioelectrical impedance analysis (BIA) in the diagnosis of sarcopenia in cirrhotic patients. *BMC Gastroenterol* 2022; 22: 159
15. Marra M, Sammarco R, De Lorenzo A, Iellamo F, Siervo M, Pietrobelli A, Donini LM, Santarpia L, Cataldi M, Pasanisi F, Contaldo F. Assessment of Body Composition in Health and Disease Using Bioelectrical Impedance Analysis (BIA) and Dual Energy X-Ray Absorptiometry (DXA): A Critical Overview. *Contrast Media Mol Imaging* 2019; 2019: 3548284
16. Nishikawa H, Yoh K, Enomoto H, Ikeda N, Takashima T, Aizawa N, Nishimura T, Nishiguchi S, Iijima H. Predictors for Grip Strength Loss in Patients With Chronic Liver Diseases. *In Vivo* 2021; 35: 363-371
17. Pazokian, Marzieh & Esmaeili, Maryam. (2019). Quality of Life in Patients With Liver Cirrhosis: A Systematic Review. *Hospital Practices and Research*. 4. 111-116. 10.15171/hpr.2019.23
18. Tsien C, Davuluri G, Singh D, et al. Metabolic and molecular responses to leucine-enriched branched chain amino acid supplementation in the skeletal muscle of alcoholic cirrhosis. *Hepatology* 2015; 61: 2018-2029
19. Volk ML, Kanwal F. Quality of Care in the Cirrhotic Patient. *Clin Transl Gastroenterol*. 2016;7:e166
20. Wang Q, Liu M, Xu L, et al. Transcriptome analysis reveals the molecular mechanism of hepatic fat metabolism disorder caused by Muscovy duck reovirus infection. *Avian Pathol* 2018; 47: 127-139
21. Wang ZG, Dou XB, Zhou ZX, et al. Adipose tissue-liver axis in alcoholic liver disease. *World J Gastrointest Pathophysiol* 2016; 7: 17-26.