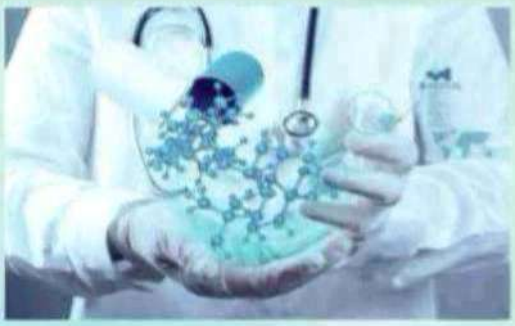
New Day in Medicine
Новый День в Медицине
NDM

TIBBIYOTDA YANGI KUN

Ilmiy referativ, marifiy-ma'naviy jurnal



AVICENNA-MED.UZ

ISSN 2181-712X.
EiSSN 2181-2187

3 (77) 2025

ТИББИЁТДА ЯНГИ КУН
НОВЫЙ ДЕНЬ В МЕДИЦИНЕ
NEW DAY IN MEDICINE

Илмий-рефератив, маънавий-маърифий журнал
Научно-реферативный,
духовно-просветительский журнал

УЧРЕДИТЕЛИ:

БУХАРСКИЙ ГОСУДАРСТВЕННЫЙ МЕДИЦИНСКИЙ ИНСТИТУТ
ООО «ТИББИЁТДА ЯНГИ КУН»

Национальный медицинский исследовательский центр хирургии имени А.В. Вишневского является генеральным научно-практическим консультантом редакции

Журнал был включен в список журнальных изданий, рецензируемых Высшей Аттестационной Комиссией Республики Узбекистан (Протокол № 201/03 от 30.12.2013 г.)

3 (77)

2025

март

www.bsmi.uz
https://newdaymedicine.com E:
ndmuz@mail.ru
Тел: +99890 8061882

Received: 20.02.2025, Accepted: 03.03.2025, Published: 09.03.2025

UDC 616.36-002.

CLINICAL AND LABORATORY CHARACTERISTICS OF CHRONIC HEPATITIS C

Shamsiyeva Muattar Axmadovna https://orcid.org/0009-0003-1895-3352
E-mail: shamsiyeva.muattar@bsmi.uz

Bukhara State Medical Institute named after Abu Ali ibn Sina Uzbekistan Bukhara, Gijduvon street 23, e-mail: info@bsmi.uz

✓ ***Resume***

In terms of fibrosis and disease progression, the relationship between HCV and steatosis is unclear. HCV genotype 3 is clearly associated with steatosis. In some patients, steatosis may be associated with the development of fibrosis. The mechanisms underlying this association are unknown. This article describes and discusses the main and clinical-laboratory and instrumental aspects of the relationship between the development of steatosis and fibrosis and the response to treatment in patients with chronic hepatitis C.

Keywords: chronic hepatitis C, hepatic steatosis, hepatotropic virus, hepatocytes, fatty acid, fibrosis tissue.

КЛИНИЧЕСКАЯ И ЛАБОРАТОРНАЯ ХАРАКТЕРИСТИКА ХРОНИЧЕСКОГО ГЕПАТИТА С

Шамсиева Муаттар Ахмадовна https://orcid.org/0009-0003-1895-3352
E-mail: shamsiyeva.muattar@bsmi.uz

Бухарский государственный медицинский институт имени Абу Али ибн Сино, Узбекистан, г. Бухара, ул. Гиждуванская. 23 e-mail: info@bsmi.uz

✓ ***Резюме***

Связь между ВГС и стеатозом с точки зрения фиброза и прогрессирования заболевания неясна. Генотип ВГС 3 явно связан со стеатозом. У некоторых пациентов стеатоз может быть связан с развитием фиброза. Механизмы, лежащие в основе этой ассоциации, неизвестны. В этой статье описываются и обсуждаются фундаментальные, клинико-лабораторные и инструментальные аспекты взаимосвязи между развитием стеатоза и фиброза и ответом на лечение у пациентов с хроническим гепатитом С.

Ключевые слова: хронический гепатит С, стеатоз печени, гепатотропный вирус, гепатоциты, жирные кислоты, фиброзная ткань.

SURUNKALI GEPATIT C NING KLINIK VA LABORATORIYAVIY XUSUSIYATLARI

Shamsiyeva Muattar Axmadovna https://orcid.org/0009-0003-1895-3352
E-mail: shamsiyeva.muattar@bsmi.uz

Abu Ali ibn Sino nomidagi Buxoro davlat tibbiyot instituti, Ozbekiston, Buxoro sh. G'ijduvon ko'chasi 23 – uy, e-mail: info@bsmi.uz

✓ ***Rezyume***

Fibroz va kasallikning rivojlanishi jihatidan HCV va steatoz o'rtasidagi bog'liqlik aniq emas. HCV genotipi 3 steatoz bilan aniq bog'liq. Ba'zi bemorlarda steatoz fibrozning rivojlanishi bilan bog'liq bo'lishi mumkin. Ushbu assotsiatsiya asosidagi mexanizmlar noma'lum. Ushbu maqolada steatoz va fibrozning rivojlanishi va surunkali gepatit C bilan og'rigan bemorlarda davolanishga javob o'rtasidagi munosabatlarning asosiy va klinik-laboratoriya va instrumental jihatlari tavsiflanadi va muhokama qilinadi.

Kalit so'zlar: Surunkali gepatit C, jigar steatozi, gepatotrop virus, gepatotsitlar, yog' kislotasi, fibroz to'qima.

Relevance

About 170 million people in the world are infected with hepatitis C virus (HCV). Since the discovery of HCV in 1989 [1], the number of acute HCV cases has fallen by more than 80% [2-3].]However, hepatitis C is still a major health burden because 60–80% of infected people progress to chronic infection [4]. HCV is a single-stranded RNA virus belonging to the Flaviviridae family [5]. The major routes of transmission are injection drug use, blood transfusion, hemodialysis, organ transplantation and less frequently sexual intercourse. Six major genotypes (1–6) of HCV have been identified, and they have varying geographical distribution. Genotypes 1, 2 and 3 are distributed worldwide with genotype 1 accounting for 40–80% of all cases. Genotype 4 is found in the Middle East and Egypt, genotype 5 in South Africa and genotype 6 in South East Asia [6].

The mechanism of steatosis development occurs when the amount of fatty acids in the liver exceeds the level that can be excreted as very low-density lipoproteins or oxidized as an energy source [7]. The main causes of fatty acid accumulation in the liver are listed below. Increased absorption of fatty acids by the liver. Fatty acids are mobilized from peripheral adipocytes under the influence of hormone-sensitive lipoprotein lipase [8]. Insulin resistance leads to increased lipolysis with the accumulation of circulating fatty acids; this, in turn, causes their enhanced passive uptake by hepatocytes, leading to increased glycolysis [9]. Insulin resistance/hyperinsulinemia stimulates fatty acid synthesis, which in turn reduces fatty acid oxidation in mitochondria and decreases fatty acid excretion from hepatocytes in the form of low-density lipoproteins associated with apolipoprotein B, leading to the development of steatosis [10].

Currently, chronic hepatitis C is one of the leading causes of liver transplantation, and annually around 242,000 people worldwide die from complications associated with hepatitis C. HCV can cause hepatic steatosis through mechanisms related to viral genotype [11-12]. Both acute and chronic hepatitis C can independently cause liver-related complications, and their coexistence can have an additional impact on liver health. Chronic hepatitis C with steatosis is a chronic inflammatory liver process in which fat (steatosis) can accumulate in liver cells.

This disease is one of the common patterns of liver disease and requires a detailed study of its clinical, laboratory, and instrumental manifestations. Hepatitis C occurs mainly as a result of viral infection, as well as other factors such as obesity, excessive alcohol consumption, type 2 diabetes, and hyperlipidemia. In individuals with hepatitis C virus (HCV), genotype 3 is most commonly associated with steatosis. In vitro studies suggest that the HCV core protein (genotype 1) may cause lipid accumulation in hepatocytes. Chronic hepatitis C with steatosis may present with the following clinical symptoms: patients often experience fatigue, vague pains, and abdominal discomfort. In some cases, jaundice may appear on their skin.

The purpose of this study is to examine the clinical and laboratory characteristics specific to chronic hepatitis C.

Materials and methods

This study includes a retrospective review of clinical records from CHC patients diagnosed through serological and molecular testing. Parameters such as liver function tests, viral load, histopathological findings, and patient demographics were analyzed. Additionally, factors such as genotype distribution, co-infections (e.g., HIV, HBV), and treatment responses were evaluated to assess disease progression and therapeutic outcomes.

Results and discussion

We registered the following results among 100 patients with various diseases based on examinations:

Blood tests: In all 40 patients, antigen-antibody complexes against hepatitis viruses (e.g., anti-HCV, HBsAg) were detected, which allowed determining the level of infection.

Laboratory indicators: Transaminases (ALT and AST) - these enzymes indicate liver cell damage, and changes were observed in all 100 of our patients with hepatitis C accompanied by steatosis. Gamma-glutamyltransferase (GGT) - an increase in this enzyme level was also observed in steatosis.

Triglycerides - increased fat tissue in the liver can lead to elevated triglyceride levels. Hepatomegaly (enlargement of the liver) and, in some cases, splenomegaly were found in 22 (55%) of our patients.

In all 100 patients, ultrasound examination revealed fat accumulation.

Computed tomography (CT) was performed on 15 patients, and the results confirmed the ultrasound findings. Additionally, X-ray imaging of the liver provided information about the amount and distribution of fat in the liver.

Conclusion

A comprehensive understanding of the clinical and laboratory characteristics of CHC is vital for early diagnosis and effective treatment. Further research is required to explore novel biomarkers, resistance patterns, and personalized therapeutic approaches. Public health initiatives focusing on screening, harm reduction, and vaccination against hepatitis B can help control HCV transmission.

LIST OF REFERENCES:

1. Alberti A, Boccato S, Vario A, Benvegnu L. Therapy of acute hepatitis C. // Hepatology. 2002;36(5 Suppl 1):195-200.
2. Alter MJ, Kruszon-Moran D, Nainan OV, et al. The prevalence of hepatitis C virus infection in the United States, 1988 through 1994. // N Engl J Med. 1999;341:556-562.
3. Bacon BR. Treatment of patients with hepatitis C and normal serum aminotransferase levels. // Hepatology. 2002;36(5 Suppl 1):179-184.
4. Ibrokhimovna M.M. (2024). Improvement of Primary Prophylaxis and Treatment of Spontaneous Bacterial Peritonitis Complicated in Virus Etiology Liver Cirrhosis. Journal of Intellectual Property and Human Rights, 2024;3(4):19-25. Retrieved from http://journals.academiczone.net/index.php/jiphr/article/view/2506
5. Oblokulov Abdurashid Rakhimovich Mukhammadieva Musharraf Ibrokhimovna Sanokulova Sitora Avazovna Khadieva Dora Isakovna. (2023). Clinical and laboratory features of spontaneous bacterial peritonitis in patients with viral liver cirrhosis. // Journal of Advanced Zoology, 2023;44(S2):3744–3750. Retrieved from http://www.jazindia.com/index.php/jaz/article/view/1716
6. Di Bisceglie AM, Hoofnagle JH. Optimal therapy of hepatitis C. Hepatology. 2002;36(5 Suppl 1):121-127.
7. Dienstag JL, McHutchison JG. American Gastroenterological Association technical review on the management of hepatitis C. // Gastroenterology. 2006;130:231-264.
8. El-Zayadi AR, Attia M, Barakat EM, et al. Response of hepatitis C genotype-4 naive patients to 24 weeks of Peginterferon-alpha2b/ribavirin or induction-dose interferon-alpha2b/ribavirin/amantadine: a non-randomized controlled study. // Am J Gastroenterol. 2005;100:2447-2452.
9. Fattovich G, Giustina G, Degos F, et al. Morbidity and mortality in compensated cirrhosis type C: a retrospective follow-up study of 384 patients. // Gastroenterology. 1997;112:463-472.
10. Mukhammadieva M.I. (2023). Вирус этиологияли жигар циррози беморларида спонтан бактериал перитонит билан асоратланишнинг профилактикаси ва давосини такомиллаштириш // Oriental Renaissance: Innovative, educational, natural and social sciences. 2023; 947-953 pp.
11. Feld JJ, Hoofnagle JH. Mechanism of action of interferon and ribavirin in treatment of hepatitis // C. Nature. 2005;436:967-972.
12. Fried MW. Side effects of therapy of hepatitis C and their management. // Hepatology. 2002;36(5 Suppl 1):237-244.

Entered 20.02.2025