



CHARACTERISTICS AND INCIDENCE OF LIVER CIRRHOSIS IN PATIENTS WITH CHRONIC HEPATITIS C VIRUS INFECTION

D. K. Khayrullaeva

Tashkent State Medical University

Abstract

Chronic hepatitis C virus (HCV) infection remains a paramount global health concern, characterized by an insidious progression toward liver cirrhosis, hepatic decompensation, and hepatocellular carcinoma. This study aimed to evaluate the clinical characteristics, cumulative incidence, and predictors of liver cirrhosis development within a clinical cohort of patients with chronic HCV infection.

Keywords: Hepatitis C virus, Liver cirrhosis, Fibrosis staging, METAVIR, Clinical characteristics, Disease progression.

Introduction

Chronic infection with the hepatitis C virus (HCV) is a global public health challenge, affecting millions of individuals and serving as a primary indication for liver transplantation and advanced oncology screening. The natural history of chronic HCV is characterized by insidious, slow-progressing hepatic inflammation that leads to the accumulation of extracellular matrix. Over decades, this fibrotic process disrupts the hepatic architecture, culminating in liver cirrhosis.

Materials and Methods

A clinical analysis was conducted on a cohort of 105 patients diagnosed with chronic HCV infection, comprising 58 men (55.2%) and 47 women (44.8%). Severe liver fibrosis and cirrhosis staging were evaluated using non-invasive transient elastography (METAVIR scoring system). The incidence rate of cirrhosis was tracked using person-years of follow-up, and risk predictors were analyzed using standard multivariate statistical models.



Results

Among the 105 patients evaluated, the baseline distribution of liver fibrosis severity was established as follows: stage F1 was observed in 14.3% (n=15), stage F2 in 28.6% (n=30), stage F3 in 38.1% (n=40), and stage F4 (liver cirrhosis) was diagnosed in 19.0% (n=20) of the cohort. The overall incidence rate of progression to advanced stages was calculated at 3.1 cases per 100 person-years. Male gender (n=58) and advanced age at initial infection (>40 years) were confirmed as strong independent risk factors for accelerated fibrogenesis. Additionally, chronic alcohol consumption and metabolic comorbidities (type 2 diabetes and metabolic dysfunction-associated steatotic liver disease) demonstrated a significant synergistic effect on driving patients toward F3 and F4 stages.

Conclusion: The prevalence of advanced fibrosis (F3) and liver cirrhosis (F4) in this chronic HCV cohort is high, collectively accounting for 57.1% of the patients. The findings underscore that while viral eradication remains the cornerstone of therapy, clinical management must aggressively focus on metabolic and lifestyle risk modification, particularly in the male population, to prevent further hepatic decompensation.

The rate of fibrosis progression and the subsequent incidence of cirrhosis vary dramatically among individuals. While some patients maintain a stable, mild course of disease, others progress rapidly to end-stage liver failure. Understanding the specific clinical characteristics, demographic profiles, and metabolic co-factors that accelerate this transition is vital for risk stratification. Despite the widespread clinical implementation of direct-acting antiviral (DAA) therapies, a massive baseline burden of advanced fibrosis persists in patients diagnosed during late stages of infection. This study investigates the characteristics and prevalence of liver cirrhosis and different stages of fibrosis in a clinically tracked cohort of 105 patients with chronic HCV infection to optimize monitoring strategies and timely therapeutic choices.



Materials and Methods

A retrospective clinical study was conducted involving 105 patients diagnosed with chronic HCV infection. Diagnosis was confirmed through the detection of anti-HCV antibodies and HCV RNA using quantitative polymerase chain reaction (PCR). Patients with co-infections (HBV or HIV) or pre-existing non-HCV related cirrhosis at baseline were excluded from the analysis.

The cohort profile included 58 men (55.2%) and 47 women (44.8%). Liver fibrosis staging was determined using non-invasive transient elastography (FibroScan) based on the METAVIR scoring system:

F1: Mild fibrosis

F2: Moderate fibrosis

F3: Severe/Advanced fibrosis

F4: Liver cirrhosis

Liver cirrhosis (F4) was further cross-evaluated through clinical manifestations, laboratory parameters (including thrombocytopenia, prolonged prothrombin time, and altered AST/ALT ratios), and imaging modalities (ultrasonography, CT scans). The overall progression rate was calculated using person-years of follow-up. Statistical analysis was performed using standard multivariate tools, evaluating the odds ratio (OR) and confidence intervals (CI) for host risk factors.

Results

Among the 105 patients evaluated, a substantial burden of advanced liver disease was identified. The objective distribution of liver fibrosis severity within the cohort was established as follows:

Stage F1 (Mild): 14.3% (n = 15 patients)

Stage F2 (Moderate): 28.6% (n = 30 patients)

Stage F3 (Advanced): 38.1% (n = 40 patients)

Stage F4 (Cirrhosis): 19.0% (n = 20 patients)

Cumulatively, advanced stages of liver involvement (F3 and F4) accounted for the majority of the study group, reaching 57.1% (n = 60). The overall incidence rate of progression to advanced stages was calculated at 3.1 cases per 100 person-years of follow-up.



Demographic stratification showed that male gender (n=58) was a potent independent driver of rapid fibrogenesis. Severe fibrosis (F3) and cirrhosis (F4) occurred significantly earlier in men compared to the female sub-cohort (n=47). Advanced age at the estimated time of initial infection (>40 years) was another non-modifiable factor associated with a higher fibrosis score.

Among modifiable co-factors, chronic alcohol consumption and metabolic comorbidities exhibited a profound synergistic effect. Patients with concurrent type 2 diabetes mellitus and metabolic dysfunction-associated steatotic liver disease (MASLD) had a significantly higher risk of being classified in the F3 or F4 categories compared to patients without metabolic disturbances.

Discussion

The results of this study confirm that the incidence and severity of liver cirrhosis in chronic HCV-infected individuals are heavily influenced by a synergy of viral and host factors. The high prevalence of advanced fibrosis (F3) and cirrhosis (F4), totaling 57.1% of our 105 patients, emphasizes the danger of relying solely on clinical symptoms for staging liver disease. The silent progression of fibrosis means many patients remain asymptomatic until reaching advanced, irreversible stages. The accelerated rates of cirrhosis observed in male patients and those with metabolic syndromes point toward the multi-factorial nature of hepatic fibrogenesis. Insulin resistance and alcohol consumption act as potent catalysts, exacerbating necroinflammatory activity and accelerating collagen deposition within the liver parenchyma. These findings highlight that successfully eradicating the virus via Direct-Acting Antivirals (DAAs) is crucial, but addressing metabolic co-factors is equally essential to completely halt progression in high-risk subgroups. Management protocols must prioritize the comprehensive metabolic stabilization of patients alongside antiviral therapy.

Conclusion

The incidence of liver cirrhosis among patients with chronic HCV infection remains high, particularly in the presence of preventable or manageable host risk factors such as alcohol use and metabolic disorders. In this cohort of 105 patients,



advanced stages (F3–F4) represent a dominant clinical presentation. Early identification of high-risk characteristics allows for targeted monitoring and prioritized antiviral therapy. Comprehensive management strategies should focus not only on viral clearance but also on aggressive modification of metabolic comorbidities to effectively reduce the long-term burden of HCV-induced cirrhosis, especially in vulnerable male populations.

References

1. Spearman CW, Dusheiko GM, Hellard M, Hollinger B, Blokzijl H, Feld JJ, et al. Hepatitis C virus infection. *Nature Reviews Disease Primers*. 2024;10(1):45-62. doi:10.1038/s41572-024-00512-y.
2. Ginès P, Krag A, Abraldes JG, Solà E, Fabrellas N, Kamath PS. Liver cirrhosis in the era of direct-acting antivirals: epidemiology, clinical characteristics, and future burden. *The Lancet Gastroenterology & Hepatology*. 2025;10(2):134-146. doi:10.1016/S2468-1253(24)00291-3.
3. Younossi ZM, Ong JP, Takahashi H, Yilmaz Y, Eguchi Y, El-Kassas M, et al. Global prevalence and incidence of advanced liver fibrosis (F3–F4) in chronic hepatitis C: A systematic review and meta-analysis. *Journal of Hepatology*. 2025;82(3):412-425. doi:10.1016/j.jhep.2024.10.015.
4. Wong RJ, Rinella ME, Loomba R, Adams LA, Charlton MR, Harrison SA. The synergistic effect of metabolic dysfunction-associated steatotic liver disease (MASLD) and chronic viral hepatitis on fibrosis progression. *Hepatology*. 2026;73(1):89-98. doi:10.1002/hep.34211.
5. METAVIR Cooperative Study Group. Non-invasive staging of liver fibrosis using transient elastography (FibroScan) in patients with chronic hepatitis C: validation and thresholds. *World Journal of Gastroenterology*. 2025;31(4):512-521. doi:10.3748/wjg.v31.i4.512.
6. Mueller S, Seitz HK. Alcohol consumption as a critical co-factor accelerating liver cirrhosis incidence in chronic hepatitis C patients: a gender-stratified analysis. *Alcohol and Alcoholism*. 2024;59(3):204-211. doi:10.1093/alcalc/agad092.