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Genotype-dependent Changes in Hepatic Steatosis and Fibrosis Following Sustained Virologic Response in Chronic Hepatitis C: A Prospective Cohort Study

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ABSTRACT

BACKGROUND. Chronic hepatitis C virus (HCV) infection remains a major global cause of hepatic steatosis, fibrosis, cirrhosis, and hepatocellular carcinoma. Mechanisms of steatosis vary by viral genotype, with genotypes 1 and 2 primarily associated with metabolic steatosis and genotype 3 with virus-induced steatogenesis. Although direct-acting antivirals (DAAs) achieve high sustained virologic response (SVR) rates, post-SVR changes in steatosis and fibrosis remain poorly characterized, particularly in populations with high metabolic burden, such as in Georgia.

OBJECTIVES. To evaluate genotype-specific changes in hepatic steatosis and fibrosis after SVR and assess the relative contributions of metabolic and viral factors.

METHODS. A prospective cohort of 200 patients with chronic HCV infection was followed from baseline through SVR24 in Tbilisi, Georgia, at the Infectious Diseases, AIDS and Clinical Immunology Research Center and the Hepatology Clinic "Hepa". Liver steatosis and fibrosis were assessed using transient elastography with controlled attenuation parameter (CAP) and liver stiffness measurement (LSM).

RESULTS. SVR was achieved in 100% of patients with genotypes 1/2 and 93.3% of patients with genotype 3. Liver enzymes normalized across groups. In genotypes 1/2, steatosis remained strongly associated with metabolic factors and persisted after SVR, with minimal fibrosis regression. In genotype 3, steatosis was more prevalent at baseline but significantly improved after SVR (mean CAP reduction −38 dB/m; $p<0.001$). BMI remained stable in genotypes 1/2 and showed a small but statistically significant decrease in genotype 3, although the magnitude of change was modest. Genotype 3 and baseline CAP independently predicted improvement in steatosis.

CONCLUSIONS. Hepatic recovery after HCV eradication is genotype-dependent. Steatosis persists in genotypes 1/2 due to metabolic drivers, whereas genotype 3 shows improvement following viral clearance. These findings support genotype-specific post-SVR management strategies.

KEYWORDS. Direct-acting antivirals; Fibrosis; Genotype 3; Hepatic steatosis; Hepatitis C virus; Sustained virologic response.

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BACKGROUND

Chronic hepatitis C virus (HCV) infection remains a major global health burden despite transformative impact of direct-acting antivirals (DAAs). According to the World Health Organization, approximately 58 million individuals worldwide are chronically infected, with 1.5 million new infections occurring annually. [1,2] Although DAAs have significantly reduced the incidence of cirrhosis, hepatic decompensation, and hepatocellular carcinoma (HCC), hepatic steatosis and fibrosis continue to pose important clinical challenges in the post-SVR era. [3,4]

Hepatic steatosis is a common manifestation of chronic HCV infection, affecting 40–70% of patients.[5,6] However, its pathogenesis differs by viral genotype. In genotypes 1 and 2, steatosis is predominantly metabolic and closely associated with insulin resistance, obesity, dyslipidemia, and type 2 diabetes.[6-8] The emerging concept of metabolic dysfunction-associated fatty liver disease (MAFLD) further supports the central role of metabolic dysfunction in hepatic fat accumulation and fibrosis progression.[7]Consequently, steatosis in these genotypes often persists after viral eradication unless underlying metabolic abnormalities are addressed.

In contrast, genotype 3 infection is associated with virus-induced steatosis, mediated by impaired very-low-density lipoprotein secretion, increased hepatic triglyceride accumulation, and activation of lipogenic

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pathways. Genotype 3 has also been linked to more rapid fibrosis progression and increased risk of hepatocellular carcinoma.[9,10]

Following the introduction of DAAs, there has been growing interest in the dynamics of hepatic recovery after SVR. Several studies suggest that steatosis tends to improve in genotype 3 following viral clearance, whereas improvement in genotypes 1/2 is limited following successful HCV eradication.[11] However, results remain heterogeneous and are influenced by metabolic comorbidities and baseline liver disease.

Fibrosis regression after SVR is also variable and influenced by baseline fibrosis stage, steatosis, metabolic factors, and viral genotype.[5,12,13] Persistent steatosis may act as a barrier to fibrosis regression, highlighting the importance of distinguishing between metabolic and virus-driven mechanisms.[14]

Georgia represents a unique clinical setting due to its national HCV elimination program and the high prevalence of metabolic risk factors. [15-17] Despite this, genotype-specific data on post-SVR hepatic outcomes remain limited.

We hypothesized that steatosis would improve predominantly in genotype 3 following viral clearance, while persisting in genotypes 1/2 due to underlying metabolic factors.

The objective of this study was to evaluate genotype-dependent changes in hepatic steatosis and fibrosis after HCV eradication in a Georgian cohort.

METHODS

Study design and setting

This prospective observational cohort study was conducted between January 2020 and December 2023 at the Infectious Diseases, AIDS, and Clinical Immunology Research Center and the Hepatology clinic “Hepa” in Tbilisi, Georgia. The study aimed to evaluate genotype-dependent changes in hepatic steatosis and fibrosis following hepatitis C virus (HCV) eradication with direct-acting antivirals (DAAs). Patients were assessed at baseline, at sustained virologic response 12 weeks after treatment (SVR12), and at 24-week follow-up (SVR24).

Study population

Participants were eligible if they met the following inclusion criteria:

- Age 18–75 years.
- Chronic HCV infection confirmed by detectable HCV RNA for at least 6 months.
- Eligibility for DAA therapy according to national guidelines.
- Ability to provide informed consent and comply with study procedures.

Patients were excluded if they had:

- Decompensated cirrhosis (Child–Pugh class B or C).
- HBV or HIV co-infection.
- Significant alcohol consumption (>30 g/day for men; >20 g/day for women).
- Other chronic liver diseases (autoimmune hepatitis, Wilson’s disease, hemochromatosis).
- Pregnancy or breastfeeding.
- Unreliable or missing elastography measurements.

Sample size and analytic population

A total of 200 patients were enrolled, including 125 with genotype 1/2 and 75 with genotype 3. Analyses of post-SVR outcomes were restricted to patients who achieved SVR. Sensitivity analyses excluding non-SVR patients were also performed.

Clinical and anthropometric assessment

All participants underwent standardized clinical evaluation at baseline, SVR12, and SVR24. Anthropometric measurements were performed according to international standards:

- Body weight measured to the nearest 0.1 kg.
- Height measured using a wall-mounted stadiometer.
- Body mass index (BMI) calculated as kg/m².
- Waist circumference measured at the midpoint between the lower rib and iliac crest.

Physical activity was assessed according to World Health Organization (WHO) recommendations and categorized into low, moderate, and high activity levels.

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Biochemical and virologic assessment

Fasting venous blood samples were collected at all study time points. Laboratory analyses included:

- Liver enzymes (ALT, AST, GGT).
- Metabolic markers (fasting glucose, HbA1c, lipid profile).
- Liver synthetic function (albumin, INR).
- Platelet count

HCV RNA levels were quantified using a real-time PCR assay (lower limit of detection: 15 IU/mL). Baseline viral load was expressed as $\log_{10}$ IU/mL. Sustained virologic response (SVR12) was defined as undetectable HCV RNA 12 weeks after completion of therapy.

Assessment of liver fibrosis and steatosis: transient elastography

Liver stiffness measurement (LSM) and controlled attenuation parameter (CAP) were assessed using FibroScan 502 Touch (Echosens, Paris, France). Examinations were performed by experienced operators blinded to genotype and clinical data.

Quality criteria included:

- At least 10 valid measurements.
- Interquartile range (IQR) <30% of the media.
- Use of M or XL probe according to BMI

Fibrosis staging thresholds:

- Significant fibrosis: >7.0 kPa.
- Advanced fibrosis: >9.5 kPa.
- Cirrhosis: >12.5 kPa

Steatosis grading (CAP):

- S0: <248 dB/m.
- S1: 248–267 dB/m.
- S2: 268–296 dB/m.
- S3: >296 dB/m.

Moderate-to-severe steatosis was defined as CAP >296 dB/m. When appropriate, BMI-adjusted CAP values were applied using manufacturer-recommended correction formulas. CAP thresholds and diagnostic performance were based on previously validated studies and meta-analyses. [15,17,18]

Antiviral treatment regimens

Patients received genotype-specific direct-acting antiviral (DAA) regimens in accordance with national treatment protocols available during the study period. Patients with genotype 1 were treated with ledipasvir/sofosbuvir, whereas those with genotypes 2 and 3 received ledipasvir/sofosbuvir plus ribavirin. The treatment duration was 12 weeks in all cases.

Treatment selection was guided by the availability of national programs and individual clinical characteristics, including fibrosis stage and eligibility for ribavirin. These regimens reflect real-world treatment practices in Georgia during the study period, when access to pan-genotypic antiviral therapies was limited.

Quality control and standardization

All elastography measurements were performed using the same device model, with regular calibration. Interobserver agreement was assessed in a random 10% sample, yielding a Cohen's kappa of 0.89. Laboratory analyses followed standardized internal and external quality control procedures.

Statistical analysis

Data were analyzed using SPSS version 27.0 (IBM Corp., Armonk, NY, USA). Continuous variables were tested for normality using the Kolmogorov–Smirnov test. Normally distributed variables are presented as mean ± standard deviation, while non-normally distributed variables are presented as median with interquartile range (IQR).

Between-group comparisons were performed using independent t-tests or Mann–Whitney U tests, as appropriate. Within-group changes over time were analyzed using paired t-tests or Wilcoxon signed-rank tests. Categorical variables were compared using the chi-square test.

Multivariable linear regression analysis was performed to identify independent predictors of change in CAP. Variables included in the models were selected a priori based on clinical relevance. They included age, sex, baseline BMI, diabetes status, dyslipidemia, baseline CAP, baseline LSM, genotype, and treatment regimen. Results are reported as β coefficients with 95% confidence intervals.

Missing data were handled using complete-case analysis. Sensitivity analyses were performed to assess the robustness of findings.

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Ethics statement

The study was approved by the Tbilisi State Medical University (TSMU) Biomedical Research Ethics Committee on February 20, 2017. Written informed consent was obtained from all participants prior to enrollment. The study was conducted in accordance with the principles of the Declaration of Helsinki. The study is reported in accordance with the STROBE guidelines for observational studies.

RESULTS

Study population

A total of 200 patients with chronic hepatitis C infection were included in the study. Of these, 125 (62.5%) were infected with HCV genotypes 1/2 (genotype 1: 84%, genotype 2: 16%) and 75 (37.5%) with genotype 3. The study flow and baseline characteristics of participants are summarized in **TABLE 1**.

TABLE 1. Baseline characteristics of the study population

Parameter	Genotype 1/2 (n=125)	Genotype 3 (n=75)	p value
Age (years)	57±12	52±10	0.003
BMI (kg/m²)	27.5±8.0	32.6±6.9	<0.001
Type 2 diabetes (%)	34	18	0.01
Dyslipidemia (%)	41	22	0.008
Viral load (log10 IU/mL)	6.1± 0.8	6.3±1.0	0.32
LSM (kPa) median (IQR)	7.2 (6.1–8.8)	6.9 (5.5–10.2)	0.18
CAP (dB/m) median (IQR)	279 (255–305)	295 (285–310)	<0.001

Abbreviations: BMI, body mass index; CAP, controlled attenuation parameter; IQR, interquartile range; LSM, liver stiffness measurement.

Patients with genotype 3 were significantly younger than those with genotypes 1/2 (52±10 vs. 57±12 years; p=0.003). Additionally, patients with genotype 3 had a higher baseline body mass index (BMI) (32.6±6.9 vs. 27.5±8.0 kg/m²; p<0.001).

Metabolic comorbidities differed between groups. Type 2 diabetes mellitus was more prevalent in genotype 1/2 than in genotype 3 (34% vs. 18%; p=0.01), as was dyslipidemia (41% vs. 22%; p=0.008). Baseline viral load did not differ significantly between groups.

Given the predominance of genotype 1 within the combined group, findings for genotypes 1/2 primarily reflect genotype 1 characteristics.

Virologic response

Sustained virologic response (SVR) was achieved in 100% of patients with genotypes 1/2 and 93.3% with genotype 3.

All post-treatment analyses were restricted to patients achieving SVR.

Significant improvements were observed (**TAB.2**):

- ALT decreased from 103.1±62.6 to 17.8±12.3 U/L, (p<0.001).
- AST decreased from 51.8±41.1 to 21.5±8.0 U/L, (p<0.001).
- GGT decreased significantly (p<0.001).

Normalization of liver enzymes occurred in more than 90% of patients.

TABLE 2. Biochemical and anthropometric changes after sustained virologic response (SVR)

Parameter	Group	Baseline	SVR24	p-value
ALT (U/L)	All	103.1±62.6	17.8±12.3	<0.001
AST (U/L)	All	51.8±41.1	21.5±8.0	<0.001
BMI (kg/m²)	GNT1/2	27.5±8.0	27.4±7.8	0.92
BMI (kg/m²)	GNT3	32.6±6.9	31.8±6.5	<0.01

Abbreviations: ALT, alanine aminotransferase; AST, aspartate aminotransferase; BMI, body mass index; SVR, sustained virologic response.

Anthropometric changes

In genotypes 1/2, BMI remained stable over the follow-up period (27.5±8.0 vs. 27.4±7.8 kg/m²; p=0.92).

In genotype 3, a small but statistically significant decrease in BMI was observed (32.6±6.9 vs. 31.8±6.5 kg/m²; p<0.01). Importantly, the magnitude of this change was modest and not clinically pronounced.

The proportion of patients achieving ≥5% weight loss was higher in genotype 3 (41%) compared to genotypes 1/2 (9%) (p<0.001). However, this finding should be interpreted cautiously, as overall weight changes after SVR are typically minimal and may reflect variability in individual metabolic or behavioral responses rather than a direct effect of viral clearance.

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Fibrosis assessment

Liver stiffness measurement (LSM) values are shown in **TABLE 3**. A numerical decline in liver stiffness was observed following SVR24 in both genotype groups. In genotype 1/2 patients, median LSM decreased from 7.2 (6.1–8.8) kPa to 5.8 (4.9–7.0) kPa. Similarly, in genotype 3 patients, median LSM decreased from 6.9 (5.5–10.2) kPa to 5.4 (4.5–6.8) kPa. The proportion of patients achieving ≥20% reduction in LSM was 18% in genotype 1/2 and 22% in genotype 3.

However, these changes did not reach statistical significance during the study follow-up period.

TABLE 3. Changes in liver stiffness and steatosis

Parameter	Group	Baseline (median IQR)	SVR24 (median IQR)	p value
LSM (kPa)	GNT1/2	7.2 (6.1–8.8)	5.8 (4.9–7.0)	0.08
LSM (kPa)	GNT3	6.9 (5.5–10.2)	5.4 (4.5–6.8)	0.11
CAP (dB/m)	GNT1/2	279 (255–305)	278 (250–295)	0.41
CAP (dB/m)	GNT3	295 (285–310)	258 (240–275)	<0.001

Abbreviations: CAP, controlled attenuation parameter; IQR, interquartile range; LSM, liver stiffness measurement.

Steatosis assessment

Baseline hepatic steatosis was more common in genotype 3 compared with genotypes 1/2 (76.4% vs. 46.4%; p<0.001).

After SVR:

- Genotypes 1/2: no significant change in CAP values.
- Genotype 3: significant reduction in steatosis (−38.2 dB/m; p<0.001).

Correlation analyses

In genotypes 1/2, CAP values correlated primarily with metabolic factors, including BMI, diabetes, and dyslipidemia.

In genotype 3, CAP values showed a significant association with baseline viral load.

Baseline steatosis was associated with reduced likelihood of fibrosis regression across all genotypes.

Multivariable regression analysis

Multivariable linear regression analysis identified independent predictors of improvement in CAP (**TAB.4**).

The overall model explained a substantial proportion of variance ($R^2 = 0.41$).

Significant predictors included:

- Genotype 3 (β=0.42; p<0.001).
- Baseline CAP (β=0.37; p=0.004).

Negative predictors included:

- Diabetes (β=−0.28; p=0.03).
- BMI was not a significant independent predictor (p=0.21).

TABLE 4. Multivariable regression for controlled attenuation parameter (CAP) improvement

Variable	β	95% CI	p-value
Genotype 3	0.42	0.28 to 0.56	<0.001
Baseline CAP	0.37	0.15 to 0.49	0.004
Diabetes	−0.28	−0.44 to −0.05	0.03
BMI	0.09	−0.08 to 0.20	0.21

Model R^2=0.41

Abbreviations: BMI, body mass index; CAP, controlled attenuation parameter.

DISCUSSION

The present study provides a prospective evaluation of genotype-dependent changes in hepatic steatosis and fibrosis following HCV eradication with direct-acting antivirals (DAAs) in a Georgian cohort. By integrating virologic, metabolic, anthropometric, and elastography data, our findings demonstrate that hepatic recovery after sustained virologic response (SVR) is heterogeneous and influenced by both viral genotype and host metabolic factors.

A key finding of this study is the high SVR rate, accompanied by rapid normalization of liver enzymes across all patient groups, reflecting effective resolution of hepatic inflammation following viral clearance. However, subsequent structural and metabolic changes differed significantly between genotypes, highlighting distinct mechanisms underlying hepatic steatosis.

In patients with genotypes 1 and 2, hepatic steatosis remained strongly associated with metabolic comorbidities, including diabetes, dyslipidemia, insulin resistance, and elevated BMI. These findings are consistent with the concept that steatosis in these

genotypes is predominantly metabolically driven. Notably, steatosis persisted after SVR despite viral eradication, suggesting that ongoing metabolic liver injury continues independently of the virus. These observations align with existing literature and emphasize the critical role of host metabolic factors in long-term liver disease progression.

These findings are also consistent with the emerging concept of metabolic dysfunction-associated steatotic liver disease (MASLD), which underscores the central role of metabolic dysregulation in hepatic fat accumulation. In this context, the persistence of steatosis following HCV eradication can be interpreted as a continuation of underlying metabolic pathology rather than a consequence of viral infection.[19]

Recent Georgian data have also demonstrated a substantial burden of NAFLD in the general population, supporting the relevance of metabolic contributors to persistent steatosis after HCV eradication. [21]

In contrast, genotype 3 infection exhibited a distinct phenotype, characterized by a higher prevalence and severity of steatosis at baseline, and a significant association between steatosis and viral load, supporting a virus-driven mechanism of steatogenesis. Following SVR, patients with genotype 3 demonstrated a marked reduction in hepatic steatosis, as reflected by a significant decrease in CAP values. While this finding is consistent with prior studies, it should be interpreted as an association rather than definitive evidence of a direct causal relationship between viral clearance and steatosis resolution.[17]

Despite improvements in steatosis, fibrosis regression was less pronounced across all genotypes. Liver stiffness measurements demonstrated a numerical decline following SVR24 in both genotype groups, suggesting partial hepatic recovery. However, these changes did not reach statistical significance. The relatively short follow-up period may have limited the ability to detect significant fibrosis regression. Furthermore, reductions in liver stiffness shortly after SVR may partly reflect resolution of hepatic inflammation rather than true histological fibrosis regression. Longer-term follow-up is required to determine whether these early improvements translate into sustained fibrosis regression.

Previous long-term studies have shown that clinically meaningful fibrosis regression may continue for several years after achievement of SVR.[16]

A modest decrease in BMI was observed in patients with genotype 3. Although statistically significant, the magnitude of change was small and unlikely to be clinically meaningful. This observation is consistent with previous evidence indicating minimal weight change following SVR and likely reflects variability in individual metabolic or behavioral responses rather than a direct effect of antiviral therapy. The absence of significant associations between dietary intake, physical activity, and hepatic outcomes in the sub study further supports this interpretation, although these findings should be considered exploratory.

Importantly, baseline steatosis was associated with a reduced likelihood of fibrosis improvement across all genotypes. This suggests that hepatic fat accumulation may act as a barrier to fibrosis regression, potentially through persistent metabolic and inflammatory pathways. These data highlight the interplay between steatosis and fibrosis and reinforce the importance of addressing metabolic risk factors even after successful viral eradication.

From a clinical perspective, these results support a stratified approach to post-SVR management. Patients with genotypes 1 and 2 may benefit primarily from targeted metabolic interventions, including weight management, glycemic control, and lipid profile optimization. In contrast, patients with genotype 3 may require closer hepatic monitoring due to the dynamic and partially reversible nature of virus-associated steatosis.

A limitation of this study is the combined analysis of genotypes 1 and 2. Although both genotypes are generally associated with metabolically driven steatosis, genotype 2 represented only a small proportion of the study population, limiting the statistical power for separate subgroup analyses. Future studies with larger genotype-specific cohorts are warranted to characterize potential differences between these genotypes further.

The relatively small number of genotype 3 patients may limit the statistical power of subgroup analyses. Additionally, the use of non-invasive methods

precludes direct histological confirmation of steatosis and fibrosis. Finally, the follow-up period was limited to SVR24, restricting the ability to assess long-term hepatic outcomes.

Further long-term studies are needed to evaluate the durability of steatosis improvement and the trajectory of fibrosis regression beyond SVR24.

CONCLUSIONS

Hepatic recovery after HCV eradication is influenced by viral genotype and metabolic context. While viral clearance results in normalization of liver enzymes across all patients, its impact on steatosis and fibrosis varies.

In genotypes 1/2, steatosis remains largely metabolic and persists after SVR, highlighting the importance of ongoing metabolic risk management. In genotype 3, steatosis improves substantially following viral clearance, although fibrosis regression remains limited.

These findings support a genotype-specific approach to post-SVR management, integrating virologic and metabolic considerations to optimize long-term liver outcomes.

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