Prevalence and predictors of abnormal alanine aminotransferase in patients with HCV who have achieved SVR

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Abstract
Chronic hepatitis C virus (HCV) is common. Treatment with direct acting antivirals (DAA) result in high sustained virologic response (SVR) associated with normalization of alanine aminotransferase (ALT). However, abnormal ALT after SVR has been observed. Since fatty liver disease can co-exist with HCV, its impact on abnormal ALT after SVR is unknown. This was a retrospective case–control analysis evaluating those with SVR and baseline fatty liver disease by transient elastography defined by controlled attenuation parameter (CAP) was performed. Abnormal ALT was defined as >1.5 ULN. The primary analysis compared abnormal ALT at SVR-12 and beyond in those with and without fatty liver disease. Six-hundred and ninety-three patients with SVR-12 were evaluated. Abnormal ALT at SVR-12 was present in 8.2% and was similar in those with and without fatty liver disease. Abnormal ALT at SVR-12 was associated with atrial fibrillation (p = .02), CAP (p = .047), age (p = .08), baseline ALT (p = .008), BMI (p = .002) and obesity (p = .02). On multivariate analysis, only BMI was associated with abnormal ALT at SVR-12 (p = .017). ALT at follow-up after SVR-12 was available in 264 patients. In those with initial normal ALT (n = 244), 11.5% had a delayed abnormal ALT and in those with initial abnormal ALT (n = 20), 47% remained abnormal while 53% normalized. Abnormal ALT after SVR following treatment with DAA is uncommon and related to increased BMI, but not related to underlying fatty liver disease assessed by CAP. The pattern of ALT can vary, and long-term follow-up is needed to assess the clinical impact of abnormal ALT after SVR.

KEYWORDS
abnormal ALT, CAP, HCV, steatosis, SVR

INTRODUCTION
Hepatitis C virus (HCV) infects about 57 million people worldwide.¹ Until 2011, the only treatment strategy was the combination of pegylated interferon and ribavirin-based therapy with significant side effects and suboptimal sustained virologic response (SVR), defined as undetectable HCV ribonucleic acid (RNA) 12 weeks after completion of therapy. By early 2015, however, all oral direct acting antiviral: DAALT, delayed abnormal ALT; DL, dyslipidaemia; DM, diabetes mellitus; DOC, department of corrections; FIB-4, Fibrosis-4 score; FLD, fatty liver disease; GT, genotype; HCV, hepatitis C virus; HTN, hypertension; NASH, nonalcoholic steatohepatitis; PAALT, persistently abnormal ALT; PNALT, persistently normal ALT; RNA, ribonucleic acid; SVR, sustained virologic response; TAALT, transiently abnormal ALT; TE, transient elastography; VCU Health, Virginia Commonwealth University Health System.

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antivirals (DAA) were approved for treatment of HCV with minimal side effects and rates of SVR exceeding 90%, usually with normalization of liver enzymes alanine and aspartate aminotransferase (ALT and AST). The high SVR rates with DAAs has resulted in a dramatic decrease in need for liver transplantation in those with HCV.

Those with chronic HCV can also have other liver diseases, including alcohol associated fatty liver disease (FLD) and nonalcoholic steatohepatitis (NASH) associated with obesity, type 2 diabetes mellitus (DM), hypertension (HTN) and the metabolic syndrome. With the high rate of SVR, alcohol and nonalcoholic fatty liver disease have become the leading indications for transplant. While underlying cirrhosis, elevated body mass index (BMI), DM, HTN, alcohol use and medications have been associated with AALT, the prevalence and risk factors for AALT in those with SVR in North America remain poorly understood. Furthermore, the pattern of AALT after SVR has not been described. Given the high prevalence of FLD in those with HCV, we hypothesized that those with underlying steatosis would be at higher risk for AALT after SVR. To address this gap in knowledge, we performed a retrospective analysis of patients treated with DAAs who achieved SVR-12 to determine (1) the prevalence of AALT among the population post-SVR, (2) the clinical characteristics and risk factors for AALT and (3) pattern of AALT after SVR.

2 | MATERIALS AND METHODS

2.1 | Patient population

A retrospective case–control analysis was performed of the HCV DAA Treatment Registry at Virginia Commonwealth University Health System (VCU Health) in treatment naïve patients with HCV who achieved SVR-12 after completing therapy with DAA. All subjects in the registry provided informed consent. We excluded those with prior liver or kidney transplant and underlying hepatocellular carcinoma. Those with human immunodeficiency virus (HIV) or chronic hepatitis B virus (HBV) surface antigen (HBsAg) positive were included as long as they were on suppressive therapy at the start of DAA and at the SVR-12 visit. Because we hypothesized that presence of steatosis would have a higher likelihood of AALT at SVR, we first identified all those with SVR and CAP ≥263 dB/m as cases. Then, we randomly selected subjects with SVR-12 without steatosis (CAP <250 dB/m) as controls. Those with CAP 250–263 dB/m were excluded to minimize any overlap.

2.2 | Data collected

Demographic, clinical and baseline laboratory data were prospectively collected. HCV RNA and genotype (GT) were determined by the Molecular Diagnostics Laboratory at VCU Health using commercial assays. The DAA regimen used for each patient was determined by the treating provider based on insurance coverage. Clinical data included history of DM, HTN or dyslipidaemia (DL). Metabolic syndrome was defined as having at least two of the following: DM, HTN, DL or obesity (body mass index [BMI] ≥30 kg/m²). Alcohol use (yes/no) at the SVR-12 visit was assessed by chart review. For those patients who were in the Department of Corrections (DOC) at the time of SVR-12, if not otherwise indicated, the assumption was made that there was no ongoing alcohol use. In all subjects, we also collected HCV RNA and ALT at a later time point (Week 24, 36 or 48 after completion of therapy) to confirm SVR (SVR-24, SVR-36 or SVR-48). Subjects were then classified as normal ALT at SVR-12 and subsequent visit (persistently normal, PNALT), those with abnormal ALT at SVR-12 and subsequent visit (persistently abnormal ALT, PAALT), abnormal at SVR-12 but normal at subsequent visit (transiently abnormal ALT, TAALT), and those with normal ALT at SVR-12 visit but abnormal at subsequent visit (delayed abnormal ALT, DAALT).

Liver fibrosis and steatosis were assessed by vibration controlled transient elastography (TE) by trained personnel with at least 10 valid measurements with IQR/med ≤30%. Advanced fibrosis (AF) and cirrhosis were defined as TE ≥9 kPa and 12 kPa, respectively. All those with baseline TE >12.5 or with FIB-4 >3.25 underwent ultrasound for hepatocellular carcinoma prior to starting DAA and every 6 months per guidelines. Those with HCC at any time were excluded. Steatosis was defined by a controlled attenuated parameter (CAP) of ≥263 dB/m. FIB-4 was calculated, and AF defined by a score of ≥3.25.

2.3 | Statistical analysis

Clinical and laboratory data are presented as mean and standard deviation (SD) or median and interquartile range (IQR) for normally and skewed continuous variables, respectively. Categorical variables are expressed as percent. We first compared baseline characteristics in those with (CAP ≥263 dB/m) and without (CAP <250 dB/m) steatosis. We then compared those with PNALT, PAALT, TAALT and DAALT at SVR-12 and beyond. We defined abnormal ALT 1.5 × ULN (normal 30 IU/L for men and 20 IU/L for women). Factors associated on univariate analysis (p < .2) were entered into a multivariable logistic regression model to identify independent variables associated with PAALT. Because the thresholds for steatosis of 248 dB/m and 275 dB/m have been suggested for ≥5% steatosis, sensitivity analyses for AALT at SVR-12 on those with CAP ≥248 dB/m, ≥275 dB/m and ≥300 dB/m were performed to minimize the impact of any subject with lesser degrees of steatosis (CAP <248, 275 and <300 dB/m).
3 | RESULTS

3.1 | Cohort

In total, 693 patients with SVR-12 were evaluated (Table 1): mean (SD) age of 51(12) years, 76% male, 57% Caucasian, 3% co-infected with HIV and 3% HbsAg+. In the cohort, 43% were obese (BMI ≥30 kg/m²), 19% had DM, 50% had HTN, 22% had DL and 16% had ongoing alcohol use. The median (IQR) ALT was 62(38–106) U/L, 81% had GT 1 and 8% had GT 3. AF was present in 76% and 46% had cirrhosis. Steatosis was present in 50% with an overall mean (SD) CAP of 257(63) dB/m.

3.2 | Impact of steatosis on baseline characteristics

Table 1 compares those with (CAP ≥263 dB/m) and without (CAP <250 dB/m). Those with (cases) and without steatosis (controls) were slightly older (52 vs. 49; p = .005) but otherwise similar by race, gender, HBV and HIV status, GT, alcohol use and baseline ALT. Those with steatosis, as expected, were more likely obese (61% vs. 23%; p = .0001), to have DM (24% vs. 14%; p = .0003), HTN (57% vs. 42%; p = .001) and DL (26% vs. 17%; p = .002) and less likely to have cirrhosis (42% vs. 55%; p = .0003). After adjusting for DM and HTN, BMI correlated CAP (p<.0001).

3.3 | AALT at SVR-12

Abnormal ALT at SVR-12 was observed in 56 patients. Table 2 compares those with and without AALT at SVR-12. AALT was associated with baseline AF (p = .02), increased CAP (p = .07), age (p = .08), baseline ALT (p = .008), BMI (p = .002) and obesity (p = .02) but not by presence of steatosis by CAP, alcohol use, DM, HTN, DL, HIV or HBV status, or GT (1 vs. 3). On multivariate analysis, only BMI was associated with AALT (p = .017). To account for the significant proportion of subjects in the DOC where alcohol use was presumed negative, we performed a sub-analysis excluding DOC subjects to assess the impact of alcohol use on AALT. When those in the DOC were excluded, alcohol use was not associated with AALT. While

| TABLE 1  Patient cohort with SVR-12 |
|-----------------------------------|
| Characteristics                  | Total population (N = 693) | CAP <250 dB/m (N = 323) | CAP >263 dB/m (N = 370) |
| Age (years), (mean±SD)           | 51 ± 12                    | 49 ± 12                   | 52 ± 11                   |
| Male gender, (%)                 | 76                         | 78                        | 74                        |
| Caucasian, (%)                   | 57                         | 59                        | 56                        |
| African American, (%)            | 43                         | 39                        | 43                        |
| DOC (%)                          | 61                         | 66                        | 57                        |
| Genotype (1/2/3/4)               | 81/9/8/1                   | 79/10/9/1                 | 82/8/8/1                  |
| Fibrosis (by kPa)                |                           |                           |                           |
| F0/1/2/3/F4 (%)                  | 1/20/23/20/33              | 3/23/26/20/27             | 1/19/19/20/39             |
| F4, (%)                          | 34                         | 27                        | 40                        |
| CAP dB/m, (mean±SD)              | 259 ± 62                   | 204 ± 34                  | 308 ± 34                  |
| HIV, (mean±SD), (%)              | 3                          | 4                         | 3                         |
| ALT (U/L), (median, IQR)         | 62, 38-106                 | 62, 38-107                | 61, 38-104                |
| FIB-4 (median, IQR)              | 1.60, 1.06-2.54            | 1.66, 1.1-2.53            | 1.57, 1.0-2.58            |
| Hepatitis B, (%)                 | 3                          | 3                         | 3                         |
| Baseline weight, (kg)            | 90.6 ± 18.7                | 83 ± 16                   | 97 ± 18                   |
| Baseline BMI kg/m²               | 29.5 ± 6.4                 | 27 ± 4.7                  | 31 ± 6.8                  |
| % Obesity (BMI ≥30)              | 43                         | 23                        | 61                        |
| %Diabetes                        | 19                         | 14                        | 24                        |
| %Hypertension                    | 50                         | 42                        | 57                        |
| %Statin/DL                       | 22                         | 17                        | 26                        |
| Final weight (kg), (mean±SD)     | 91.5 ± 19                  | 84 ± 16                   | 97 ± 19                   |
| Final ALT (U/L), (mean±SD)       | 23.28 ± 17.66              | 22 ± 18                   | 24 ± 17                   |
| %PAALT                           | 8.2                        | 6.69                      | 9.56                      |
| %Active EtOH                     | 16                         | 15.3                      | 16.85                     |
there was no correlation between baseline CAP and ALT, we did not observe a correlation between baseline CAP and ALT at SVR-12 ($r = .11; p = .0037$).

In sensitivity analysis of those with CAP <248 dB/m ($n = 381$) vs. ≥248 dB/m ($n = 313$), AALT at SVR-12 was similar (9.3% vs. 6.6%, $p = .19$). In those with CAP ≥275 dB/m ($n = 299$), AALT at SVR-12 was present in 10.7% of the cohort. When controlling for fibrosis, BMI was associated with AALT ($p = .01$). In those with CAP ≥300 dB/m ($n = 195$), AALT at SVR-12 was slightly higher (12%). However, BMI was no longer predictive of AALT in this cohort. Furthermore, there was no optimal cut-off BMI that predicted AALT (area under the receiver operating curve 0.55).

### 3.4 Pattern of AALT after SVR-12

Among those with SVR-12, 264 had available data on long term follow-up at a median of 36 (IQR 28–40) weeks after starting DAA. We observed significant differences in follow up data among our patient populations: 25% in DOC vs. 45% in non-DOC treated patients ($p < .0001$). During this extended period, abnormal ALT was observed in 38 patients (mean 57 U/L vs. 18 U/L in those with normal ALT; $p < .0001$). In those with normal ALT at SVR-12 ($n = 243$), 28 (11%) developed a delayed abnormal ALT (DAALT) (Figure 1). Conversely, among those with abnormal ALT at SVR-12 ($n = 21$), 10 (47%) remained abnormal at follow-up while 11 normalized (transient abnormal ALT) ($p < .0001$). A total of 49 subjects (18%) had an abnormal ALT at SVR-12 or beyond (3.4% with PAALT, 4% with TAALT and 10.6% with DAALT). While those with abnormal ALT at any point after SVR had higher baseline ALT (99 U/L vs. 74 U/L; $p = .036$) and high CAP (278 dB/m vs. 262 dB/m; $p = .079$), no factor including alcohol use was associated with abnormal ALT in longer term follow-up.

### 4 DISCUSSION

The introduction of DAAs has contributed significantly in the treatment of HCV and achieving SVR. The majority of patients who achieve SVR have normalization of aminotransferases, some patients have ongoing liver inflammation evidenced by PAALT. This large, retrospective case-control study involved patients with HCV who underwent treatment with DAA and achieved SVR. We observed AALT at SVR-12 in 9.6% of those with steatosis and 6.7% of those without and identified that AALT was related to increased BMI but not ongoing alcohol use. While increased BMI strongly correlated with CAP score, an indirect measure of hepatic steatosis, AALT was not directly associated with increased CAP itself. Importantly, followed out, approximately half (53%) of those will normalize their ALT at next visit while a smaller proportion (10%) will develop newly increased ALT in the absence of HCV RNA, steatosis or alcohol use.

The achievement of SVR correlates with improvement in underlying fibrosis, and in the majority results in normalization of liver enzymes. In those without baseline advanced fibrosis, treatment guidelines indicate no further liver follow up is needed. However, some patients have increased liver enzymes after SVR; the cause and management of those patients, particularly, in the absence of fibrosis remain a challenge. We identified that AALT at SVR was rare and only 8.2% of this United States based cohort had AALT at SVR. AALT at SVR was identified to be associated with increased BMI but was not significantly different between those with and without steatosis.

In a study based out of Spain by Olveira et al, among a patient cohort of 1112 patients, 130 were found to have persistent alteration of liver tests (11.7%) and similar to our cohort. Furthermore, they identified a significant difference in those who were co-infected with HIV having a higher rate of AALT than those mono-infected with HCV. In contrast, our study revealed similar and low co-infection rates with and without HIV. Olveira et al, identified a clinical diagnosis of NAFLD, high alcohol consumption and drug toxicity to be etiologies of AALT.

Hsieh et al demonstrated a prevalence of persistent liver inflammation of 9.8% based on their East Asian based cohort of 461 patients with HCV treated with DAA who had advanced fibrosis/cirrhosis. This study identified risk factors for ongoing liver inflammation and concluded that impaired baseline liver function tests (including elevated bilirubin and INR), a higher baseline ALT, and the presence of steatosis were associated.

Nonalcoholic fatty liver disease (NAFLD) affects more than one quarter of the adult population and is associated with underlying metabolic syndrome including obesity and DM. Sanyal et al identified that in patients with NAFLD, fibrosis stages F3 and F4 were associated with increased risk of liver related complications and increased mortality. Therefore, identifying those with NAFLD, regardless of HCV infection is important. Our cohort with FLD were more likely to have DM, HTN, increased BMI and alcohol use suggesting that use of CAP correctly identified FLD. However, CAP is not perfect, and some patients may have been misclassified as cases or controls.

As opposed to prior studies, we had additional follow-up that showed that the ALT at SVR-12 does not tell the whole story. We observed that approximately half improved at follow up while a smaller proportion with normal ALT at SVR-12 develop abnormal ALT at this later visit. While we hypothesized that advance fibrosis, underlying steatosis or ongoing alcohol use would be associated with abnormal ALT after SVR, this was not the case in our cohort suggesting that other factors, such as underreported alcohol use, medications or other unrecognized liver disease is may be present. In the absence of liver histology, the clinical significance of an abnormal ALT after SVR is unknown.

Our study has several strengths. We prospectively included a large population of North American subjects with HCV and SVR of both baseline and SVR-12 data to minimize bias. In addition, because all subjects underwent TE with CAP prior the therapy, we were able to select both cases and controls across three CAP thresholds (263, 275 and 300 dB/m). However, our study also has several limitations. First, our patient cohort was comprised a large proportion (61%) from department of corrections (DOC). However, we have previously shown that the DOC HCV population does well.
with DAA with high SVR-12. While in the DOC a patient's lifestyle is different, such as the consumption of little, if any, alcohol which may have limited our ability to assess alcohol use as a risk factor for AALT. Another limitation of including DOC patients was lack of follow-up after SVR which limited our post-SVR-12 data. Additionally, the diagnosis of fibrosis and steatosis were determined by TE-CAP and not confirmed with biopsy. Furthermore, we used baseline TE-CAP values and did not perform TE at the SVR-12 visit which could have affected our results. Finally, we also only had a small number with HCV GT 3, a known risk factor for steatosis. However, in those with GT 3 and SVR, the steatosis usually resolves.

In conclusion, we observed a low rate of AALT in those with SVR-12 and beyond. While AALT was observed more in those with increased CAP suggesting that it was related to underlying steatosis, CAP itself was not associated with AALT. The multivariate analysis
among those with CAP ≥275 dB/m and CAP ≥300 dB/m demonstrated the associated between higher CAP score and higher AALT, however, BMI was found to be less predictive. Furthermore, our data show that ALT patterns can vary after SVR-12 independent of obesity or underlying steatosis suggesting other factors may be at play such as unrecognized or unreported medications including over the counter medications and supplements, steatosis missed by CAP, other unrecognized liver disease (such as alpha-1 antitrypsin or iron overload), or underreported alcohol use. Longitudinal assessment with follow-up TE and CAP is underway to confirm this observation.

AUTHOR CONTRIBUTIONS
Chadha, Nikita involved in data retrieval, manuscript drafting, revisions and approval of submitted version of the manuscript. Turner, Alan involved in data retrieval and approval of submitted version of the manuscript. Sterling, Richard is the principal investigator who conceived, supervised, reviewed and approved the final manuscript.

CONFLICT OF INTEREST
Chadha, Nikita has no disclosures. Turner, Alan has no disclosures. Sterling, Richard has research support from Gilead, Roche, AbbVie and Abbott.

DATA AVAILABILITY STATEMENT
The data that support the findings of this study are available from the corresponding author upon reasonable request.

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How to cite this article: Chadha N, Turner A, Sterling RK. Prevalence and predictors of abnormal alanine aminotransferase in patients with HCV who have achieved SVR. J Viral Hepat. 2023;30:73-78. doi:10.1111/jvh.13763