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Research Article

Decrease in Alcohol Consumption Improves Survival in Hepatitis C Infected Heavy Drinkers: A Longitudinal Cohort Study

Shahriar Tavakoli Tabasi[1,2]* and Sheeba Ninan[1]

[1]*Michael E DeBakey Veterans Affairs Medical Center, USA*
[2]*Baylor College of Medicine, USA*

***Corresponding author**
Shahriar Tavakoli Tabasi, Staff physician MEDVAMC and assistant professor of medicine Baylor College of Medicine, 2002 Holcombe Blvd, 111PC, Houston, TX 77030, Tel: 713-794-5280; Fax: 713-794-7838; Email: Shahriar@bcm.edu

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Abstract

Background: Excessive alcohol consumption is common among patients with chronic hepatitis C and contributes to the progression of liver disease.

Aims: The objective of this study is to determine the effect of decrease in alcohol consumption on survival of patients with chronic hepatitis C and excessive drinking.

Methods: We conducted a retrospective cohort study of patients with excessive alcohol consumption enrolled in the hepatitis C clinic from August 15, 2000 through December 30, 2007 at the Veterans Affairs Medical Center in Houston. Survival was measured through August 30, 2011. Inclusion criteria consisted of hepatitis C infection and consumption of > 50 grams of alcohol per day, absence of complicated cirrhosis, and absence of malignant medical conditions. The primary outcome of the study was survival, analyzed by Cox proportional hazard model.

Results: Two hundred eighty patients were followed for a mean of 6.34 years (SD 2.94). One hundred twenty two patients (44%) continued to drink above the risky drinking threshold and hundred fifty eight patients (56%) decreased alcohol consumption after a median of 2.73 (interquartile range: 0.34-5.26) years. Sixty six patients died. In Cox proportional hazards model, risky drinking (HR 1.58; CI 1.10-2.28), cirrhosis at baseline (HR 4.11; CI 2.38-7.10), and presence of medical co-morbidities (HR 2.65: CI 1.53-4.60) were associated with death.

Conclusion: Cessation of, or decrease in, alcohol consumption improves survival in patients with chronic hepatitis C infection.

INTRODUCTION

More than 3 million Americans live with hepatitis C virus (HCV) infection, the most common cause of chronic hepatitis in the United States [1]. HCV infection can result in progressive liver fibrosis, cirrhosis of the liver, and hepatocellular carcinoma [2-3]. Alcohol consumption plays an important role in progression of HCV-related liver disease [4]. Excessive alcohol consumption by HCV-infected patients increases the rate of progression of fibrosis, and development of cirrhosis [5-8]. In addition, even moderate alcohol consumption may be associated with accelerated progression of liver disease among HCV-infected individuals [9-12]. Moreover, alcohol may increase HCV replication and decrease the response rate to antiviral treatment [13-15]. It is also recognized that excessive alcohol consumption is common among HCV-infected patients, especially those using department of veterans medical system [16]. Therefore, treatment of alcohol abuse and dependence has been recommended for patients with chronic HCV infection prior to starting antiviral therapy [17]. To date, however, no study has proven a survival benefit from interventions that decrease alcohol consumption in HCV-infected excessive drinkers. It is also not clear if patients with behavioral and medical co-morbid conditions, which are prevalent among HCV-infected subjects [18,19], and those with advanced liver fibrosis may benefit from such interventions.

We performed this study to evaluate the survival in a cohort of HCV-infected patients who were drinking excessively. We evaluated the survival, as well as survival free of complications of cirrhosis, in those who quit or significantly cut down on drinking, compared to those who continued risky drinking.

METHODS

Patients

We conducted a retrospective cohort study of patients with chronic HCV infection and excessive drinking, aged 18–80 years, who were evaluated from 8/15/2000 through 12/30/2007 in the MEDVAMC hepatitis C clinic in Houston, Texas. We defined excessive drinking as an estimated average of more than 50 grams of alcohol intake on a daily basis at the time of the initial visit to hepatitis C clinic [20]. Exclusion criteria included patients

with: (a) preexisting complicated cirrhosis prior to or on the date of the first visit to hepatitis C clinic; (b) hepatocellular carcinoma (patients with AFP values > 200 ng/ml were included in this category); (c) concomitant chronic hepatitis B infection, defined by positive hepatitis B surface antigenemia; (d) malignant medical conditions (excluding basal cell and squamous cell carcinoma of the skin, CLL and slowly progressive lymphomas, low grade/slowly progressive prostate cancer).

Definition of variables

Alcohol consumption was evaluated in each subject quantitatively, both the past and current use. During each visit to the hepatitis C clinic, patients were asked to quantify the daily and average weekly alcohol consumption. In addition, we reviewed alcohol screening data, which is part of the annual clinical reminders and has been widely implemented in the department of Veterans Affairs hospitals since 1997. The yearly data included CAGE questionnaire from 2000 through June 2003, and AUDIT-Consumption (AUDIT-C) data afterwards [22,23].

Three variables were used to evaluate the effect of alcohol on survival. Past use of alcohol was evaluated semi-quantitatively as the number of years subjects reported drinking on average 50 grams of alcohol (excessive drinking years). Excessive drinking at the time of initial visit to hepatitis C clinic (more than an average of 50 grams daily alcohol consumption) was an inclusion criteria. Time for effective decline in alcohol consumption was evaluated based on the date alcohol consumption fell below a threshold of 14 drinks per week for men and 7 drinks per week for women, or an AUDIT-C score of more than 3 for men and more than 2 for women (risky drinking) [24]. The date for cessation of risky drinking was the first date when subjects reported a decrease in alcohol intake below risky levels, and never exceeded that threshold during subsequent visits. An ordinal variable was constructed in 4 groups based on when patients stopped risky drinking; within 30 days from visiting HCV clinic (group 1), within 1 year from visiting HCV clinic (group 2), after one year from visiting HCV clinic (group 3), never (group 4). Since the effect of alcohol consumption on progression of liver disease is not expected to reverse immediately after cessation of drinking, we did not include this variable as a time-dependent variable.

Co-morbid medical conditions were defined as one of the following: (a) coronary artery disease with NYHA class III or IV angina, (b) heart failure NYHA class III and IV, (c) chronic obstructive pulmonary disease GOLD stage IV and GOLD stage III with functional capacity less than 4 MET, (d) uncontrolled diabetes with hemoglobin A1c > 9%, and (e) diabetic proliferative retinopathy.

Information regarding substance use was actively sought by interview and thorough review of medical records. Active substance use was considered present if patients used cocaine, benzodiazepines, opiates, barbiturates, amphetamines, or other illegal drugs of abuse, in the 6 months prior to visiting hepatitis C clinic. The use of opiates, methadone, benzodiazepines, and barbiturates were not included in this category if prescribed by a physician. Recreational use of cannabis was not considered as active substance use.

Patients were asked about past history of mental health disorders, suicidal ideation with/without plan, and any attempt at suicide. Patients completed a Zung self reported depression (SDS) questionnaire during the first visit to hepatitis C clinic [18,21]. Mental health disorders were considered active in any patient with a history of major depressive disorder, bipolar disorder, psychosis, suicide attempt, hospitalization for pervasive suicidal ideation, or a score of 60 or more on Zung depression screen.

Patients were not eligible for antiviral therapy if they had co-morbid medical, substance use, or psychiatric conditions. Instead, these patients were referred for management of these conditions and were followed in hepatitis C clinic. If the co-morbid conditions improved, patients became eligible for treatment.

We considered patient to have cirrhosis of the liver at the time of first visit to hepatitis C clinic if: (a) a liver biopsy within 6 months of the initial visit, showed METAVIR stage 4 fibrosis, (b) laboratory data showed significant liver synthetic dysfunction in the absence of other etiologies (albumin < 3.2 mg/ml and International normalized ratio > 1.4), or (c) imaging studies showed a nodular or coarse liver texture with irregular borders plus evidence of portal hypertension (presence of esophageal varices, splenomegaly, and/or portal vein diameter of > 1.5 cm) [25]. We calculated the estimated duration of HCV infection based on the risk factors for hepatitis C infection, as previously described [26,27]. Chronic hepatitis B infection was defined as positive hepatitis B surface antigenemia.

Outcomes

The primary outcome was death of any cause. Secondary outcome was a composite outcome of development of complicated cirrhosis and/or death. Complications of cirrhosis included any of the following: variceal bleeding, prophylactic banding/sclerotherapy for high risk varices, ascites, hepatic encephalopathy, hepatocellular carcinoma, and/or undergoing liver transplantation. Subjects who had any of these outcomes were censored on that date. Other subjects were censored on the last date they had a visit to the VA hospital system or on August 30, 2011. Patients were considered lost to follow up if they did not have a visit to one of the VA health-care facilities after December 30th 2010. Survival analysis was completed on August 30, 2011. To evaluate for survival and development of complications of cirrhosis, electronic medical records were reviewed for visits to any Veterans Health Administration hospital nationwide.

Statistical methods

For primary analysis we compared the survival curves between those who quit risky drinking versus those who continued to drink above risky drinking threshold. We used Cox proportional hazards model to determine hazard ratio (HR) and 95% confidence intervals (CI) for the effect of independent variables of interest on survival. The primary independent variable of interest was the duration of risky drinking after initial visit to our clinic (groups 1-4). Other independent variables included in this model were age, number of years of heaving drinking above average of 50 grams alcohol daily, estimated duration of hepatitis C infection, presence/absence of active substance use disorder at the time of first visit, presence/absence of active mental health disorder at the time of first visit, presence/absence of co-morbid medical conditions at the time of first visit, presence of cirrhosis at the time of first visit, receiving antiviral treatment for hepatitis C, and sustained viral response to HCV treatment. We then repeated the model for secondary composite outcome death and/or complicated cirrhosis. The

proportionality assumption underlying the Cox proportional hazard was evaluated by means of Schoenfeld residuals.

To test for the possibility that patients who decreased alcohol consumptions may have been more likely to receive antiviral treatment, we performed sensitivity analysis. We repeated Cox proportional hazard regression by deleting all patients who had undergone treatment and achieved sustained viral response.

To determine the differences between those who continued to drink and those who quit risky drinking, we performed multiple regression analysis. The outcome in this model was the duration of risky drinking after visiting HCV clinic. The independent factors analyzed were age, race, total years of heavy drinking, presence/absence of active mental health disorders, presence/absence of active substance use disorder, Zung SDS, risk factor for hepatitis C infection (substance use related vs. others), estimated duration of HCV infection, presence/absence of cirrhosis of the liver at baseline. We checked for normality using the Shapiro-Wilk normality test. We checked for the presence of multicollinearity by calculating variance inflation factor.

This study was approved by the institutional review board of the Baylor College of Medicine, Houston, Texas, and MEDVAMC research and development committee.

RESULTS

From August 15th 2000 through December 30th 2007 hepatitis C clinic in MEDVAMC evaluated 2866 unique patients for hepatitis C infection. Two hundred eighty three had excessive alcohol intake without complicated cirrhosis. One patient was excluded due to hepatitis B co-infection, and two patients were excluded due to the presence of malignancy. At the end, 280 patients were included in the study. Baseline characteristics are shown in Table 1. As outlined in Table 1, the majority of patients were men with a high prevalence of co-morbid medical, psychiatric, and substance use disorders. Thirty four patients (12%) had cirrhosis of the liver during initial evaluation.

Twenty one patients (7.5%) had no visits after December 30th 2010 and were considered lost to follow up.

The mean duration of follow up was 6.34 years (SD 2.94). One hundred twenty two patients continued to drink above the risky drinking threshold. One hundred fifty eight patients decreased alcohol intake below risky drinking threshold, after a mean duration of 3.20 years (SD 2.82) from the initial visit to hepatitis C clinic. The primary outcome was death from any cause which occurred in 66 patients (24%). The cause of death was related to liver disease in 22 cases, unknown in 20 cases, and unrelated to liver disease in 24 cases.

Secondary composite outcome of complicated cirrhosis and/or death occurred in 87 subjects, over a mean follow up of 4.09 years (SD 2.73). Complications of cirrhosis occurred in 49 patients (17%). Initial complication of cirrhosis was ascites in 24 patients, hepatocellular carcinoma in 16 patients, variceal bleeding in 7 patients, prophylactic ligation of varices in one patient, and hepatic encephalopathy in 2 patients. One of the patients initially diagnosed with ascites was later diagnosed with hepatocellular carcinoma, bringing the total number of diagnosed cases of hepatocellular carcinoma to 17.

In multivariate cox proportional hazard regression, stepwise delay in abstaining from risky drinking (analyzed in groups 1 through 4 as an ordinal variable) (HR 1.58; CI 1.10-2.28), cirrhosis of the liver at baseline (HR 4.11; CI 2.38-7.10), and presence of medical co-morbidities (HR 2.65: CI 1.53-4.60) were associated with mortality. Number of years of excessive drinking (prior to visiting HCV clinic) and receiving antiviral treatment for hepatitis C were associated with a non-significant trend towards decreasing and improving survival respectively (Table 2).

When the analysis was repeated, with death and/or complicated cirrhosis as the composite outcome, results remained mostly unchanged. However, the number of years of excessive drinking prior to visiting HCV clinic reached statistical significance in this analysis, and was inversely associated with survival ($P = 0.05$).

Sensitivity analysis confirmed the findings in the Cox proportional hazard. When patients who had sustained viral response were not included in the analysis of the primary outcome, results remained unchanged.

Multiple regression analysis showed that older patients and those with cirrhosis of the liver at baseline, were more likely to quit risky drinking ($P = 0.027$ and < 0.001 respectively). Race, total years of heavy drinking, presence/absence of active mental health disorders, presence/absence of active substance use disorder, Zung SDS, risk factor for hepatitis C infection (substance use related vs. others), and estimated duration of HCV infection were not predictive of early cessation/decrease in alcohol consumption.

Table 1: Patient characteristics.

Gender	
Male	274 (98%)
Female	6 (2%)
Age (mean, SD)	51.6 (5.5)
Race/ethnicity	
African American	149 (53%)
White	116 (41%)
Hispanic	8 (3%)
Pacific-islander	2
American-Indian	1
Unknown	4
Risk factor for hepatitis C infection	
Positive history of IDU and/or snorting cocaine	229 (82%)
Median estimated duration of hepatitis C infection	29 (25-75 percentile: 24-33) years
Mean duration of history of excessive drinking	28.2 (SD 9.4) years
Past history of mental health disorder	93 (33%)
Depression-related disorders	54
Bipolar mood disorder	5
Post-traumatic stress disorder and anxiety	9
Organic/substance-induced mood disorders	9
Psychosis	16
Zung self-reported depression score; mean (SD)	52.4 (12.7)
Active mental health problems at initial HCV visit	92 (32%)
Co-morbid medical conditions	46 (16%)
Active substance use disorder	37 (13%)
Cirrhosis at initial HCV clinic visit	34 (12%)
Antiviral treatment during follow up	36 (13%)
Sustained viral response	12/36 (33%)

Table 2: Cox proportional hazards model for predictors of death.

Variable	HR	95% CI
Age at initial visit	1.02	0.96-1.07
Duration of excessive alcohol intake prior to initial visit	1.03	0.99-1.06
Mental health co-morbidity	0.81	0.47-1.38
Substance use co-morbidity	1.01	0.47-2.16
Medical co-morbidity	2.65	1.53-4.59
HCV treatment	0.28	0.06-1.22
Sustained viral response	0.89	0.08-10.09
Cirrhosis at initial visit	4.11	2.38-7.10
Quit risky drinking (groups 1-4)	1.58	1.10-2.28

DISCUSSION

We have demonstrated that HCV-infected excessive drinkers were less likely to die, if they were to quit drinking, or decrease drinking below risky drinking threshold. In addition, we have demonstrated that the composite outcome of death and/or complicated cirrhosis is improved in those who decrease drinking below the risky drinking threshold. We have also demonstrated that in addition to continued drinking, cirrhosis of the liver and presence of co-morbid medical conditions predicts poor prognosis in these patients. Finally, we found that older patients and those with cirrhosis of the liver were more likely quit risky drinking.

In spite of its retrospective design our study has several methodological strengths. The loss to follow up, one of the main pitfalls of cohort studies, was only 7.5%, which is highly unlikely to affect the outcomes of our study [28]. Missing data were negligible owing to availability of nation-wide reliable electronic medical records. Potential for exposure misclassification was not significant since alcohol use has been assessed by mandatory computer-generated clinical reminders in the department of Veterans Affairs hospitals on a yearly basis, since 1997 [22]. The risk for outcome misclassification was minimized due to the presence of detailed medical records and choice of well-defined objective outcomes. However, there were limitations to our study. The great majority of our subjects were men, limiting the external validity of our results for generalization to populations of HCV-infected women. The role of liver biopsy at baseline, close to initial HCV clinic visit, could not be evaluated since most patients did not have a liver biopsy then. However, if the improved outcome in those who quit risky drinking were because they had less severe liver fibrosis, we would have expected to see a reverse associated between cirrhosis of the liver and quitting risky drinking. On the contrary, we found that patients who quit earlier were more likely to have cirrhosis of the liver. Therefore, it is unlikely that such a phenomenon could explain the difference in survival observed in our study.

Our study demonstrates that HCV-infected patients with excessive drinking will significantly improve their prognosis if they quit risky drinking. Experts have widely recommended cessation of alcohol intake for HCV-infected patients based on indirect evidence that shows rapid progression of liver disease and higher incidence of cirrhosis in HCV-infected drinkers [5-8]. However, there has been little direct evidence to suggest that prognosis will actually improve if these patients were to quit/decrease alcohol consumption. Drumright et al. showed that transition from alcohol use to abstinence among HCV-infected patients was associated with a decrease in liver enzymes, but did not study the protective effect of abstinence on mortality or development of cirrhosis [29]. Serra et al., studied the effects of abstinence from alcohol in patients with alcoholic cirrhosis, 72 of whom also had HCV infection [30]. They showed that, among all patients with alcoholic cirrhosis, the cumulative survival in abstinent alcoholics was significantly different from that of alcoholics who maintained the same pattern of alcohol consumption. However, the study was not designed, or powered, to evaluate the effect of abstinence in the subgroup of patients with chronic hepatitis C.

We were concerned that the protective effect of decrease in alcohol consumption may not be evident in all HCV-infected heavy drinkers, especially those with co-morbid conditions such as substance use disorder. However, we showed that decrease alcohol intake is protective irrespective of the diagnosis of substance use, mental health disorders, and medical co-morbidities. We also found that medical co-morbidities, as defined in our study, predicted poor prognosis independent of other factors. This finding was expected since by definition these patients mostly had serious end-organ disease.

Patients who quit/decrease drinking were different from others in terms of being more likely to have cirrhosis of the liver and being older. Previously it has been shown that the knowledge of liver disease encourages patients to quit drinking [31-33]. This suggests that educating HCV-infected patients about progression of liver disease and consequences of cirrhosis may serve as an important tool for encouraging heavy drinkers to quit/decrease alcohol consumption. In addition, the results of our study should provide further encouragement for HCV-infected drinkers to quit/decrease alcohol consumption.

In summary, this study shows that cessation/decrease in alcohol consumption in HCV-infected patients improves outcomes. The benefit seems to extend to all patients, irrespective of severity of liver disease and co-morbid conditions. Knowledge of severity of liver disease may be a strong motive for patients to limit alcohol consumption.

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CONFLICT OF INTEREST

Authors are not reporting any conflict of interest related to this study. The views expressed in this article are those of the authors and do not necessarily represent the views of the Department of Veterans Affairs or the National Institute of Diabetes Digestive and Kidney Diseases.

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