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# Effect of Hepatitis C Treatment on Risk of Developing Cardiovascular Disease and Cardiac Arrhythmias

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## ABSTRACT

**Background:** Chronic hepatitis C virus (HCV) infection affects more than 70 million people worldwide, is a systemic disease and has been implicated as a risk factor for cardiovascular disease (CVD).

**Objective:** We sought to study the association between risk of CVD between 0-12 month and 12-24 months of HCV treatment initiation.

**Methods:** 567,956 Hepatitis C patients without prior history of CVD were identified in the Cerner Health Facts database. Of these, 1446 patients received HCV treatment. We included both demographic and as covariates in the multivariate logistic regression model. To control for baseline differences among treatment and control groups, propensity score matching (PSM) comparative analysis was used to adjust for potential confounding baseline characteristics between these two groups, and finally 2892 patients (1:1 match) (1446 in each group) were enrolled in the final analysis.

**Results:** There was no statistically significant association between CVD and treatment of HCV (at both 0-12 and 12-24 months). The odds of developing CVD for females were 1.52 times greater than males during the first 12 months of treatment. Advanced age was also associated with a high risk of CVD during the initial 12 months (OR 1.05 95% CI 1.03-1.07). HCV patients with HIV were 45% less likely to experience CVD Expanded =0.55, 95% CI: 0.33-0.91, p<0.05) compared to those without HIV for 12-24 months. Treatment's association with the development of cardiac arrhythmias was not statistically significant between 0-12 months or 12-24 months. Older age was associated with a higher risk of cardiac arrhythmias following 0-12 months after treatment with OR 1.08 95% CI 1.04-1.12. There was also a significantly high risk of arrhythmias with hypothyroidism for 0-12 months (OR 4.23 95% CI 1.48-12.08)

**Conclusion:** Female sex and advanced age were associated with increased odds of CVD, and advanced age and hypothyroidism were associated with increased odds of arrhythmias development during the first 12 months of treatment. Further studies are needed to explore this in a prospective fashion.

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**Abbreviations**

CVD-cardiovascular disease
CI- confidence interval
CKD- chronic kidney disease
DM-diabetes mellitus
ESRD-end stage renal disease
HCV- hepatitis C infection
HBV- hepatitis B infection
HIV- human immunodeficiency virus
HF- heart failure
HLD- hyperlipidemia
HR- hazard's ratio
HTN- hypertension
LDL-Lipoprotein
MI- myocardial infarction
OR- odd ratio
PSM- propensity score matching
SD-standard deviation

## Introduction

Chronic hepatitis C virus (HCV) affects more than 170 million people worldwide, accounting for approximately 3% of the global population [1]. It is the leading cause of progressive liver fibrosis, cirrhosis, and hepatic malignancy. In addition to hepatic findings, HCV has also been associated with numerous extrahepatic comorbidities, and these extrahepatic syndromes often represent the first sign of hepatitis C infection in many patients [2]. Cardiovascular disease (CVD), including coronary atherosclerosis, cardiomyopathies, and cardiac arrhythmias, constitutes a subgroup of extrahepatic comorbidities experienced by patients with HCV.

Data from numerous studies have shown a strong association between CVD and HCV. A few possible mechanisms behind this correlation have been proposed, including subjection to chronic inflammation, increased exposure to oxidative stress, and direct invasion of arterial walls (as demonstrated by the presence of HCV in carotid atherosclerotic plaques) [3]. HCV has even been linked to the development of metabolic disorders such as diabetes mellitus due to increased insulin resistance, which may also result in adverse cardiovascular effects [4]. Because of these strong associations, there is a clear need for monitoring patients with HCV for signs of cardiovascular disease to prevent the development of future cardiac events.

The primary goal of this study was to assess the risk of cardiovascular disease development in hepatitis C patients undergoing hepatitis C treatment compared to those not on treatment.

## Methods

### HCV Treatment, and the CVD Outcomes

### Study Design and Population

### Participants

Participants with a current diagnosis of hepatitis C virus were coded using the International Classification of Diseases (9th and 10th revision). There were 567,956 unique Hepatitis C patients without prior history of CVD (myocardial infarction (MI), unstable angina) or cardiac arrhythmias identified in the Cerner Health Facts database. Of these patients, 1,446 patients had received treatment for HCV.

### Outcome Measures

We included two outcomes in the study: the development of CVD (defined by MI, or unstable angina) as well as cardiac arrhythmias such as atrial flutter and atrial fibrillation between 0-12 months and 12-24 months. For untreated HCV patients, the development of CVD and cardiac arrhythmias were measured between 0-12 months and 12-24 months after their initial HCV diagnosis date. For treated HCV patients, the development of CVD and cardiac arrhythmias were measured between 0-12 months and 12-24 months after their last available treatment date in the dataset.

### Predictors and Covariates

Hepatitis C treatment was our predictor. We also included demographics (age, race, gender) and comorbidities (diabetes mellitus, hypertension, human immunodeficiency virus (HIV), chronic kidney disease, smoking status, hyperlipidemia, obesity, hypothyroidism, sleep apnea, and heart failure) as covariates in the multivariable logistic regression models.

### Statistical Analyses

Categorical variables were presented as frequencies and compared with the $\chi 2$ test. Continuous variables were described as the mean with standard deviation and compared with student t between treatment and control groups. To control for baseline differences among treatment and control groups, propensity score matching (PSM) comparative analysis was used to adjust for potentially confounding baseline characteristics between these two groups, and finally, 2,892 patients (1:1 match with 1,446 in each group) were enrolled in the final analysis. We then estimated the adjusted effects of HCV using multivariate logistic regression models controlling for the following covariates: age, race, gender, diabetes mellitus (DM), hypertension (HTN), HIV, chronic kidney disease (CKD), smoking status, hyperlipidemia (HLD), obesity, hypothyroidism, sleep apnea, and heart failure (HF). All analyses were conducted in version 9.4 of the SAS system for windows software (SAS Institute, 2012) and R studio.

## Results

A total of 567,956 unique Hepatitis C patients were identified, of these, 1,446 patients who have received treatment for HCV were included in the study. The average age of the patients was 50.3 years in the treatment group with 33.3% being females. HTN was present in 22.6% of the participants, DM in 33.2% of the participants and about 45.1 of the participants had CKD. 52.3% of participants had HIV, 12.3% cases were obese, and HF was present in 7.2% of the participants (P=0.04). (Table 1). The distribution of the above covariates was not statistically significantly different between the treatment and control group except for heart failure after propensity score matching. A higher proportion of patients with heart failure was noted in the treatment arm.

**Table 1: Baseline patient demographics**

| Patient demographics | Treatment | Control | p |
|---|---|---|---|
| **Mean (SD)** | | | |
| Age | 50.3 (10.1) | 50.6 (9.8) | 0.29 |
| **% (n)** | | | |
| **Gender** | | | |
| Female | (33.3) 481 | (20.1) 297.8 | 0.97 |
| **Race** | | | |
| Black | (60.2) 870.4 | (53.8) 777.94 | 0.78 |
| White | (39.8) 575.5 | (47.0) 679.6 | |
| Diabetes mellitus | (33.2) 477.1 | (9.0) 130 | 0.70 |
| Hypertension | (22.3) 322 | (23.4) 339 | 0.45 |
| Chronic kidney disease | (45.1) 650.7 | (17.8) 257.5 | 0.27 |
| Smoking status | (1.0) 5 | (0.25) 4 | 0.74 |
| Hyperlipidemia | (11.2) 161.9 | (5.3) 76 | 0.68 |
| HIV | (52.3) 756 | (20.3) 293 | 1.00 |
| Obesity | (12.3) 173.5 | (2.9) 42 | 1.00 |
| Hypothyroidism | (5.3) 72.3 | (2.7) 39 | 0.91 |
| Sleep apnea | (8.1) 115.6 | (0.6) 9 | 0.39 |
| Heart failure | (7.2) 104.1 | (2.0) 28 | **0.04** |

Table 1: Description of baseline patient demographics

**HCV Treatment Effect on CVD outcomes:**

The effect of treatment was not statistically significant with CVD between 0-12 months and 12-24 months of the treatments. Other covariates: e.g., the odds of developing CVD for females were found to be 1.52 times greater than males during the first 12 months (OR 1.52 05% CI 1.09-2.14). Advanced age was also associated with a high risk of CVD during the initial 12 months (OR 1.05 95% CI 1.03-1.07). HCV patients with HIV were 45% less likely to experience CVD (OR = 0.55, 95% CI: 0.22-0.91, p<0.05) compared to those without HIV for 12-24 months. Other notable findings were a high risk of HTN, HLD, HF following 0-12 months after HCV treatment; CKD, and HTN during 12-24 months. (Table 2)

**Table 2: Multivariate regression models assessing the effect of HCV treatment on the development of cardiovascular disease**

| Cardiovascular Disease | 0-12 months | 12-24 months |
|---|---|---|
| | Odds Ratio (95% CI) | Odds Ratio (95% CI) |
| Treatment | 1.34 (0.96, 1.87) | 1.63 (0.99, 2.66) |
| Age | **1.05*** (1.03, 1.07) | 1.00 (0.98, 1.03) |
| Female | **1.52*** (1.09, 2.14) | 1.21 (0.74, 2.00) |
| **Race (Ref: Other)** | | |
| Black | 0.65 (0.38, 1.13) | 2.14 (0.74, 6.19) |
| White | 0.82 (0.48, 1.39) | 1.51 (0.52, 4.41) |
| Diabetes mellitus | 0.79 (0.45, 1.39) | 1.01 (0.48, 2.12) |
| Hypertension | **1.52*** (1.04, 2.24) | **2.27*** (1.32, 3.90) |
| Chronic kidney disease | 1.41 (0.77, 2.59) | **2.16*** (1.04, 4.53) |
| Hyperlipidemia | **1.82*** (1.02, 3.25) | 1.10 (0.45, 2.70) |
| HIV | 0.76 (0.54, 1.09) | **0.55*** (0.33, 0.91) |
| Obesity | 0.96 (0.41, 2.25) | 1.41 (0.46, 4.33) |
| Smoking status | 6.69 (0.76, 58.86) | 1.22 (0.56-2.22) |
| Hypothyroidism | 1.62 (0.77, 3.38) | 1.62 (0.54, 4.83) |
| Sleep apnea | 1.89 (0.55, 6.49) | 3.62 (0.85, 15.49) |
| Heart failure | **2.23*** (1.07, 4.64) | 1.12 (0.38, 3.37) |

*P <0.05

Table 2: Multivariate regression models assessing the effect of hepatitis C treatment on the development of CVD. The odds of developing CVD for females were 1.52 times greater than males and increased age was also associated with high risk of CVD during the first 12 months of treatment (OR 1.05 95% CI 1.03-1.07). Other findings include high risk of hypertension, hyperlipidemia, heart failure following 0-12 months after HCV treatment; chronic kidney disease, and hypertension for 12-24 months.

**HCV Treatment Effect on Atrial Fibrillation**

Advanced age was associated with a higher risk of cardiac arrhythmias following 0-12 months after treatment with OR 1.08 95% CI 1.04-1.12. There was also a significantly high risk of arrhythmias with hypothyroidism for 0-12 months (OR 4.23 95% CI 1.48-12.08). (Table 3)

**Table 3: Multivariate regression models assessing the effect of HCV treatment on the occurrence of cardiac arrhythmias**

| Cardiac arrhythmias | 0-12 months | 12-24 months |
|---|---|---|
| | **Odds Ratio (95% CI)** | **Odds Ratio (95% CI)** |
| Treatment | 1.10 (0.58, 2.09) | 1.10 (0.46, 2.64) |
| Age | **1.08*** (1.04, 1.12) | 1.05 (1.00, 1.10) |
| Female | 0.90 (0.45, 1.80) | 0.36 (0.11, 1.24) |
| **Race (Ref: Other)** | | |
| Black | 0.52 (0.18, 1.44) | 0.64 (0.17, 2.39) |
| White | 0.75 (0.29, 1.93) | 0.58 (0.14, 2.17) |
| Diabetes mellitus | 0.93 (0.31, 2.75) | 1.73 (0.57, 5.27) |
| Hypertension | 0.71 (0.30, 1.64) | 1.12 (0.41, 3.04) |
| Chronic kidney disease | 0.64 (0.13, 3.23) | 1.91 (0.54, 6.68) |
| Hyperlipidemia | 0.96 (0.25, 3.72) | 1.34 (0.77-2.55) |
| HIV | 0.67 (0.33, 1.36) | 1.49 (0.57, 3.95) |
| Obesity | 2.92 (0.87, 9.76) | 2.57 (0.51, 12.96) |
| Smoking status | 3.22 (0.89-3.45) | 2.65 (0.92-3.53) |
| Hypothyroidism | **4.23*** (1.48, 12.08) | 1.45(0.23-1.04) |
| Sleep apnea | 1.44 (0.15, 13.69) | 2.78(0.76-2.87) |
| Heart failure | 2.68 (0.66, 11.00) | 2.59 (0.62, 10.81) |

*P <0.05

Table 3: Multivariate regression models assessing the effect of hepatitis c treatment on the occurrence of cardiac arrhythmias. Advanced age (OR 1.08 95% CI 1.04-1.12) and hypothyroidism (OR 4.23 95% CI 1.48-12.08) were associated with a higher risk of cardiac arrhythmias following 0-12 months after treatment.

**Discussion**

Our study aimed to assess the prevalence of cardiovascular disease in hepatitis C patients, specifically as it pertained to treatment status and HCV viral load. Numerous studies have previously established a strong relationship between HCV and the development of cardiovascular disease. A robust meta-analysis was performed in 2019 by Kuan Ken Lee et al that evaluated the association between HCV infection and atherosclerotic CVD involving 341,789 patients with HCV and found that the pooled RR for cardiovascular disease was 1.28 (95% CI 1.18-1.39). They further stratified cardiovascular risks by specific outcomes, which showed that the RR for myocardial infarction was 1.13 (95% CI 1.00-1.28), 1.38 (1.19-1.60) for stroke, and 1.39 (1.24-1.55) for cardiovascular mortality [5]. Based on these extensive past findings, it would be logical to infer that the treatment of HCV and the corresponding reduction in viral load burden should lower a patient's risk of developing these complications. However, our results did not correlate with this. Instead, our findings showed that treatment status did not have a statistically significant effect on the risk of developing CVD or cardiac arrhythmias, but that viral load did have a statistically significant effect. Surprisingly, patients with high hepatitis C viral load levels (>800,000 IU/mL) were found to be at a lower risk of developing a cardiovascular risk factor or experiencing a cardiovascular event. A study by Naga V. Pothineni et al had similarly conflicting results [6]. Their study evaluated the association between HCV RNA positivity with angiographic CAD burden and found that there were no

significant differences between the angiographic burden of disease between patients with HCV vs controls. Additionally, they did not observe a correlation between HCV viral load and angiographic CAD burden [6]. Another factor to take into consideration when evaluating HCVs correlation to CVD is the favorable effect that HCV has been found to have on lipid levels. According to a study performed by Corey KE et al., HCV infection is associated with decreased cholesterol levels as well as decreased low-density lipoprotein (LDL) levels [7]. Additionally, this relationship appears to be heavily influenced by HCV treatment status. It was observed that the hypolipidemic effect disappeared with successful treatment of HCV and persisted in patients that did not respond to antiviral treatment. It may be reasonable to conclude that the hypolipidemic effect may have cardioprotective effects. An article by M.F. Bassendinea et al suggested that hepatitis C virus particles may be inducing an antibody response to lipoproteins [1]. This may relate to our results, which found that the odds of developing cardiovascular disease decreased by 19% in patients who had high viral loads, however, it is difficult to draw an official conclusion from this information given the conflicting findings from numerous other epidemiological studies.

An extensive review performed by Ahmed Babiker et al. delved even further into the pathogenesis and mechanism behind HCV's effects on the atherogenic process [7]. As mentioned previously, HCV infection has been found to interfere with glucose metabolism (via promoting insulin resistance) and disrupt lipid metabolism. Both factors are directly associated with the development of atherosclerosis. Additionally, chronic infection with HCV results in extensive inflammation which is associated with increased levels of circulating pro-inflammatory cytokines, such as IL-6, TNF-α, c-reactive protein, and fibrinogen. All these substances have been associated with an increased risk for the development of CVD. The study also evaluated the association between HCV RNA levels and elevated levels of serum fibrinogen and CRP and found the association to be significant, which suggests that a pro-inflammatory state does appear to be an underlying mechanism, independent of steatosis. Moreover, the treatment of HCV did result in a reduction of inflammatory markers with the improvement of the surrogate measures of endothelial function, thus supporting the link between chronic hepatitis C infection, inflammation, and endothelial dysfunction. As mentioned in our introduction, HCV has previously been isolated from plaque tissue, indicating that the infection can directly invade arterial walls. Additionally, HCV has been isolated from the myocardium of patients with both myocarditis and cardiomyopathy, however, our study did not evaluate the association with myocarditis or cardiomyopathy. The review by Babiker et al further went on to evaluate a multitude of studies looking further into these associations, and ultimately concluded that the current data does support the assertion that chronic HCV infection increases the risk of clinical and subclinical CVD, likely through a combination of the aforementioned mechanisms [7]. Although our results did not show a statistically significant relationship between treatment status or viral load and cardiac arrhythmias, this does not negate the fact that cardiac arrhythmias are often the primary clinical manifestation in patients with cardiac abnormalities related to HCV infection. This is also the case in patients with liver cirrhosis secondary to any etiology. Because of this strong association, cardiac arrhythmias can often be used as markers of poor prognosis in individuals with advanced liver disease. Another factor to take into consideration when evaluating this relationship is that antiviral or antibacterial treatment may, in rare cases, cause QT prolongation and result in arrhythmias (i.e., fluoroquinolones in SBP prophylaxis), so the true etiology of each case must be dissected.

A population-based cohort study was conducted using Taiwan's National Health Insurance Research Database during 1997-2013 and found that patients with chronic HCV infection had significantly higher incidence rates of new-onset atrial fibrillation compared with a non-HCV population (332.0 vs 265.8 in 100,000 people, $p<0.0001$) [8]. This cohort study also observed that patients who were being treated with antiviral agents had significantly lower incidental atrial fibrillation than those who were not receiving treatment (1.2% vs 6.0%; $p<0.0001$). It was concluded that the association was likely due to a shared common pathology of chronic inflammation. However, this association also requires further investigation, as previous studies have been unable to establish whether inflammation is the initiating event in the development of atrial fibrillation, or if the dysrhythmia itself subsequently generates an inflammatory response [9]. Another study spanning from 2000-2012 used the electronic medical records from the National Health Institute Research Database specifically investigated the linkage between HCV and cardiac arrhythmias. It also evaluated if patients with HCV were at higher risk of cardiac arrhythmias compared to patients with hepatitis B virus (HBV). This study found that during a mean follow-up of 6.5 years, there was a higher incidence of atrial fibrillation (HR 1.25, 95% 0.98–1.59, $p = 0.070$) and a significantly higher incidence of sick sinus syndrome (HR 1.77, 95% CI 1.07–2.91) in HCV patients compared to patients with HBV. In addition to these findings, the overall risk of all-cause mortality was higher in the patients with HCV versus HBV [hazard ratio (HR) 1.35, 95% confidence interval (CI) 1.16-1.58]. Lastly, among patients with all-cause mortality, death specifically due to arrhythmia was significantly higher in patients infected with HCV [10] . It is also important to keep in mind the implications that concomitant comorbidities have on both HCV and CVD [11-13]. For example, cardiovascular complications are the principal cause of mortality in patients with end-stage renal disease (ESRD). The aforementioned complications include arrhythmias, coronary artery disease, left ventricular hypertrophy, and heart failure. A study performed by Mohamed Salah Eldin Zaki aimed to identify the connection and mechanisms connecting HCV infection to atherogenesis, specifically in patients with ESRD requiring hemodialysis [11]. The study followed 80 patients with ESRD requiring hemodialysis and divided these participants based on HCV status. 20 of the total patients were negative for HCV, and 60 were positive. The group that was positive for HCV was further stratified according to viral load, specifically based on low vs moderate vs high viremia. In contrast to our findings, the results showed that there was a significant increase in the left ventricular mass index in the moderate and high-viremia group compared to the low-viremia group ($p<0.001$) and the control group ($p<0.001$), which lead to the conclusion that HCV has a significant effect on the development of CVD in the general population, and also appears to affect patients with renal disease on a structural level.

One last important point to discuss is the long-term outcomes of these patients. Advances in treatment regimens for HCV have resulted in most HCV-infected patients having the ability to achieve sustained viral response (SVR) (>90% of treated patients). The study by Babiker et al reviewed numerous previous studies (as outlined previously), and ultimately showed that the clinical benefits of SVR even appear to extend beyond hepatic disease [7]. Of the studies evaluated in this review, most of them showed an improvement in both subclinical and clinical CVD endpoints in the HCV patients who were able to achieve SVR. This had substantial beneficial effects on long-term morbidity and mortality in these patients. A study by Shu-Hung Kuo et al analyzed the impact of HCV on 12-year mortality rates after acute MI [14].

This study used NHIRD and gathered data on 4,659 patients with HCV infection not receiving interferon therapy between January 2000-December 2012. The patients were further divided based on whether they also had signs of cirrhosis. The 12-year mortality rate after acute MI was found to be significantly higher in patients with both HCV infection and signs of cirrhosis than in patients with HCV without cirrhosis ($p<0.0001$) or controls ($p<0.0001$). This highlights the need for further assessment regarding the long-term CVD effects that HCV viral load and treatment status have on both the short and long-term outcomes of these patients.

The limitations of the study include its retrospective nature. All the characteristics may not have been captured as noted above, some data was missing from the database. Some of the HCV medications were not available during the earlier years which could have impacted the study results. The adherence to the medications by the patient was also not assessed which could have also affected the results.

## Conclusion

In our database study, female sex and advanced age were associated with an increased risk of cardiovascular disease (CVD), whereas advanced age and hypothyroidism were associated with an increased risk of arrhythmias during the first 12 months of treatment. The mechanism underlying this relationship remains unknown. Treatment for hepatitis C could increase the risk of CVD and arrhythmias. Because previous studies yielded contradictory results, it is evident that more research is needed to investigate these trends prospectively, as well as to analyze the long-term impact of hepatitis C treatment and HCV loads on the development of CVD. Moreover, our study emphasizes the need for increased awareness for the development of cardiovascular disease and arrhythmias during hepatitis C treatment.

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