



Hepatitis C virus infection and alcohol

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An interaction between hepatitis C and the consumption of alcohol was suspected before the discovery of the hepatitis C virus (HCV) in 1989.¹ Alcohol and HCV both cause liver injury. Although it is known that alcohol consumption is associated with apoptosis, oxidative stress, immune dysfunction (lower activities of cytotoxic T cells and helper T cells and decreased cytokine expression), increased HCV viral load, and greater numbers of HCV quasiespecies, the mechanism of the interaction between alcohol and HCV is unknown.^{2,3,9}

The progression of hepatitis C to liver cirrhosis varies from 4% in donors to 22% in liver clinics, after 20 years of evolution. Epidemiological studies carried out in the 90s showed that excessive alcohol consumption is one of the main determinants of the severity of HCV progression.^{2,4,8} The definition of excessive alcohol consumption and the minimum amount of alcohol required to induce liver damage in patients with HCV infection is an important issue. It should be borne in mind that low levels of alcohol ingestion are beneficial and are associated with decreased mortality in conditions other than hepatitis C.

Several studies demonstrated an association between a high level of alcohol consumption and a high prevalence of fibrosis and cirrhosis.^{4,6-8} The existing association between low and moderate levels of alcohol consumption and the progression or severity of liver damage caused by hepatitis C is less clear, and some results are contradictory. Two recent studies showed a direct relationship between a high level of alcohol consumption and severe fibrosis.^{4,6-8}

There is no doubt that a relationship exists between HCV and hepatocellular carcinoma and that alcohol, by promoting cirrhosis, is conducive to the development of hepatocellular carcinoma. Some studies have even found that alcohol would be a predictive factor independently of histological state and even a predictor of evolution after surgical resection.^{4,5,8}

The effects of drinking alcohol on responses to treatment are not well characterized, and existing data are derived from studies with small numbers of patients. Absti-

nence from alcohol is considered mandatory for preventing poor adherence to treatment and an increased incidence of adverse side effects. None of the studies on alcohol consumption and HCV is a randomized or blinded trial, more biochemical responses have been evaluated than virological responses, and almost all studies have been done using interferon monotherapy. Decreased responses to treatment have been associated with alcohol consumption, but whether alcoholics are the worst responders to therapy is unknown.^{4,7,8}

Based on the best available evidence to date, which has mainly been supported by case-control studies, evidence type is III, and recommendation Grade is C, for general applicability or in an individual case.

The pathophysiology of interactions between alcohol and hepatitis C remain to be elucidated by further research. Patients with HCV should avoid excessive alcohol consumption, because it induces progression of fibrosis to cirrhosis and because moderate or low alcohol consumption seems to contribute to liver damage. There is a relationship between alcohol consumption by patients with hepatitis C and the development of hepatocellular carcinoma in association to the interaction of alcohol and cirrhosis, even if an independent association has not been proven. The incidence of sustained responses to antiviral treatment seems to be inversely proportional to the amount of alcohol consumed before and during the treatment.

Recommendations of the consensus panel

Is there evidence of a lower viral response in patients who consume alcohol?

Yes.

Evidence quality: 2

Is abstinence from alcohol consumption recommended?

Yes.

Evidence quality: 3

Does the consumption of more than 30 g/day of alcohol accelerate the course of HCV?

Yes.

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Evidence quality: 2

References

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