Long-term Management of Alcoholic Liver Disease

Garmen A. Woo, MD*, Christopher O'Brien, MD, AGAF, FRCMI

KEYWORDS

• Alcoholic liver disease • CAGE • AUDIT-C • Long-term management • Nutrition

KEY POINTS

- Alcoholic liver disease is a major cause of end-stage liver disease worldwide.
- Prevalence of alcoholic liver disease is strongly correlated to the cumulative lifetime consumption of alcohol. Genetic and environmental factors, such as gender, genetic polymorphisms, obesity, and viral hepatitis, also affect the development of cirrhosis from alcohol use.
- Early detection of alcoholism with screening tools and abstinence is important for both prevention and management of alcoholic liver disease. Abstinence is best achieved with concomitant psychological and pharmacological therapies.
- Treatment of alcoholic liver disease includes discontinuation of alcohol consumption, nutritional support, and management of the sequelae of cirrhosis. Liver transplantation should be considered in those patients with decompensated cirrhosis as early as possible.

Chronic consumption of alcohol in large amounts remains the hallmark for hepatic damage and subsequent development of alcoholic liver disease.[1] The spectrum of this disease ranges from fatty liver to hepatic inflammation, necrosis, and progressive fibrosis, and is a major cause of morbidity and mortality worldwide.[2] Alcohol accelerates the progression of other liver diseases, such as hepatitis C virus, hepatocellular carcinoma, and hemochromatosis. Alcoholic liver disease is also a major cause of end-stage liver disease that requires transplantation in most developed countries.[3]

The cumulative lifetime alcohol consumption is strongly associated with the prevalence of alcoholic liver disease.[1] The type of beverage and pattern of drinking also affects the risk for developing alcoholic liver disease.[1] Yet, it remains unclear why, despite reaching the required 'threshold' for alcohol intake, only 10% to 35% of heavy, long-term alcohol drinkers will develop alcoholic hepatitis and only 8% to 20% will develop cirrhosis.[4,5] Both environment and genetic factors have been explored.

Supporting funds: None.
Disclosures: The authors have nothing to disclose.
Center for Liver Diseases, Miller School of Medicine, University of Miami, 1500 Northwest 12th Avenue, Suite #1101, Miami, FL 33136, USA
* Corresponding author.
E-mail address: gwoo@med.miami.edu

Clin Liver Dis 16 (2012) 763–781
http://dx.doi.org/10.1016/j.cld.2012.08.007 **liver.theclinics.com**
1089-3261/12/$ – see front matter

Environmental risk factors, such as the dose and pattern of alcohol intake, and dietary and lifestyle factors are noted to be important in determining disease risk.[6,7] Coexisting external factors, such as obesity and hepatitis C infection, combined with daily alcohol use increase the likelihood of developing associated liver disease by as high as 100-fold.[8]

Host factors predisposing to the development of alcoholic liver disease are not as well known. There are several studies demonstrating that women develop liver disease after exposure to lower quantities of alcohol and over shorter periods.[3,9,10] This gender difference may be attributable to several factors, such as differences in gastric alcohol dehydrogenase (ADH) levels and a higher proportion of body fat in women.[11] The rates of development of cirrhosis and mortality are also found to be higher in African American and Hispanic individuals compared with their White counterparts; but this may be more attributable to the longer and heavier drinking patterns seen in African American and Hispanic individuals.[12,13] There may be a genetic predisposition in the development of alcoholic liver disease. Polymorphisms of genes encoding for ADH and cytochrome P-450 enzymes have been associated with higher occurrences of liver disease.[14] In a study by Marcos and colleagues,[15] carriers of genotype TT and GT from the –330T>G interleukin (IL)-2 gene polymorphism were significantly higher in alcoholic individuals with cirrhosis compared with alcoholic individuals without liver disease. In addition, the presence of a deletion allele in NFKB1 polymorphism may be associated with a higher risk of developing alcoholic liver disease.[16] Further studies in this area are needed.

OVERVIEW OF MANAGEMENT

Detection

Alcoholism is essentially another medical disease; yet, there is a large stigma associated with having an alcohol-related diagnosis. Although there is a need to intervene early to prevent liver disease, fewer than 13% of those with an alcohol problem actually get diagnosed and even fewer than 6% of these diagnosed patients receive medical treatment.[17] Based on a national survey, few people are questioned about alcohol use when they visit a general practitioner and when an alcohol problem is identified, most do not receive appropriate follow-up.[18] Screening for alcohol problems by physicians in the primary care setting with various tools, including paper-and-pencil questionnaires, should be performed regularly to promote detection as well as to initiate brief counseling and use of medical treatment when appropriate.[19]

Screening tools

The US Preventive Services Task Force recommends screening for alcohol misuse in all adults.[20] A variety of screening tools are available; but the 2 most commonly used are the CAGE questionnaire and the Alcohol Use Disorder Identification Test (AUDIT).

CAGE is the mnemonic for the 4 questions listed in **Box 1**. One positive response to any of these questions suggests the need for further evaluation; whereas, positive responses to 2 or more suggest the possibility of alcohol misuse.[21,22] Although the brevity of the CAGE questionnaire makes it easy to incorporate into routine practice, it does not distinguish between current and past alcohol use and is insensitive in detecting heavy drinking, especially in women.[23,24]

AUDIT uses 10 questions to assess the quantity of alcohol consumed and the individual's experience with using alcohol.[25] Responses to these questions generate an additive score that correlates with risk for alcohol dependence. Although the AUDIT is not affected by gender or ethnic bias, its major limitation is the length of the questionnaire.[26] A modified version, the AUDIT-C, comprises only 3 questions (**Box 2**). Its relative sensitivity and specificity is 0.86 and 0.81, respectively, and has been