ALCOHOL ABUSE FOR LIVER DISEASE

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Alcohol is a rich source of compounds from renewable sources, however it has to be activated in order to allow the modification of their carbon backbone. The latter can be achieved via oxidation to the corresponding aldehydes or ketones (1). Alcohol dehydrogenases (ADH) is ubiquitous in higher organisms and participate in metabolizing a wide variety of alcohols and aldehydes, as an important “detoxification” mechanism (2).

Alcoholic and alcohol abuse are due to many interconnected factors, including genetics, how you were raised, your social environment, and your emotional health. Lower emotional intelligence is predictive of alcohol-related problems (3). Aromatic alcohols i.e. phenethyl alcohol, tyrosol and tryptophol are produced by C. albicans, especially under nitrogen poor conditions. The effects of ethanol on the innate immune system (4). People who have a family history of alcoholism or who associate closely with heavy drinkers are more likely to develop drinking problems (5). Finally, those who suffer from a mental health problem such as anxiety, depression, or bipolar disorder are also particularly at risk, because alcohol may be used to self-medicate.

It is no secret that alcohol consumption can cause major health problems, including cirrhosis of the liver and injuries sustained in automobile accidents. However, only a small portion of heavy drinkers develop disease, indicating that other factors (genetic, environmental or dietary) contribute to disease initiation (6). But alcoholic can make hallucinosis is a pathological mental state characterized by an acute onset of predominant auditory hallucinations that occur either during or after a period (7) of heavy alcohol consumption. Alcohols interact with lipid bilayers (8) with the OH group positioned in the bilayer interfacial region and with the hydrocarbon chains facing the hydrophobic core of the bilayer if you think liver disease and car crashes are the only health risks posed by drinking, think again: Researchers have linked alcohol consumption to more than 60 diseases. Heavy alcohol intake has been known for centuries to impair lung defenses (9). Alcohol exerts a huge toll on the nation's physical, social, and psychological health. Consumption doubled between 1950 and 1980, during which time the relative price of alcohol halved. Since then consumption has flattened off (10) recognising people with alcohol related problems is difficult probably less than 20% are known to their general practitioner, and a large proportion...
are missed in accident and emergency departments. Recognition is particularly difficult among teenagers, elderly people, and doctors. About half of the doctors reported to the General Medical Council for health difficulties liable to affect professional competence have an alcohol problem. Doctors may be alerted to an alcohol problem by the presenting complaint. The essential first stage in improving recognition is taking a drinking history, and this should be combined with selected investigations. Amount of alcohol consumed in units. Always inquire about quantity and type of drink. Many doctors are unaware of the unit values for common descriptions of daily intake. Time of first alcoholic drink of the day. Pattern of drinking: problem drinking is characterised by the establishment of an unvarying pattern of daily drinking (10).

Alcohol abuse is associated with a spectrum of pancreatic disease clinical manifestations, from acute self-limiting pancreatitis to chronic unremitting pancreatitis leading to exocrine and endocrine pancreatic insufficiency. The risk of developing alcohol-induced pancreatitis increases with the amount and duration of drinking. A minimum of 6–12 years of approximately 80 grams or more of alcohol per day is considered necessary for the development of clinically significant disease. However, less than 10% of heavy drinkers develop clinical pancreatitis suggesting that there are contributing genetic and environmental factors involved in disease expression. On the other hand, findings consistent with pancreatitis have been reported in up to 75% of autopsies performed on alcohol abusers. The volunteers marked the location that corresponded to the intensity of their sensation with a vertical line. We tested the items: agitation, alterations in motor coordination, hearing, walking and speech, sensation of well-being, tiredness, headache, dizziness, tremor, weakness, muscular tension, nausea, salivation, perspiration, visual disturbances, tachycardia and difficulty in breathing. (11). There are wide ranges in the reported incidence and prevalence of the disease between countries and sometimes within countries. However, there are patterns indicating that the incidence of alcoholic pancreatitis is more common among men, while pancreatitis caused by gallstones is more common among women. Another pattern is related to ethnicity: the studies of discharge data from Los Angeles County and New York hospitals and one in Portugal demonstrated that for both men and women, black patients are more likely, compared to other ethnic groups, to be hospitalized for chronic pancreatitis than alcoholic cirrhosis. In the US, Native Americans and Alaskan natives have the highest rates of alcoholic cirrhosis of any ethnic/racial group but have rates of pancreatitis similar to those of whites. Both smoking and dietary factors may contribute to the risk of alcoholic pancreatitis and Alcohol is a well-known risk factor for breast cancer (BC) (12).

Although the issue is complicated by the interrelationship between smoking and drinking, recent studies have demonstrated that cigarette smoking is an independent risk factor for alcohol-related pancreatitis and that smoking accelerates the disease progression. Further, one report provides evidence that there is possibly a synergistic association between alcohol and smoking in the development of pancreatitis. Different diets may also affect the development of the disease. Diets high in
fat and protein may be associated with the development of alcoholic pancreatitis while saturated fats and vitamin E may decrease the toxic effect of alcohol. The information provided above suggests that alcohol is a contributing factor to the initiation and development of both acute and chronic pancreatitis; and that alcohol alone may not cause pancreatitis unless accompanied by additional genetic and/or environmental factors. Thus, it is believed that alcohol “sensitizes” or “primes” the pancreas to pancreatitis. Additional factors, such as cigarette smoking, genetic factors (e.g., ethnicity) and/or diet, then act to initiate pancreatitis in the “alcohol-sensitized” pancreas. An extension of this hypothesis is that the pancreas has adaptation systems that protect it from insults caused by alcohol abuse and that pancreatic disease occurs when these adaptive systems are either disordered or insufficiently robust. In this scenario, genetic and/or environmental factors could increase the likelihood of disease development in alcohol abusers by altering key adaptive responses in the pancreas (6).

Chronic alcohol consumption has deleterious effects throughout the entire gastrointestinal system, including the liver, pancreas, esophagus, gastric mucosa, and malabsorption syndromes involving the small intestine. Cirrhosis of the liver is the characteristic organ dysfunction induced by longstanding unhealthy use of alcohol (13).

Alcoholic liver disease (ALD) is the most common liver disease in the Western world. For many reasons, it is underestimated and underdiagnosed. An early diagnosis is absolutely essential since it helps to identify patients at genetic risk for ALD; can trigger efficient abstinence namely in non-addicted patients; and initiate screening programs to prevent life-threatening complications such as bleeding from varices, spontaneous bacterial peritonitis or hepatocellular cancer. Alcoholic liver disease (ALD) is, either alone or in association with other comorbidities such as obesity or viral hepatitis, the leading cause of liver disease. The liver is also the most common target organ of chronic alcohol abuse. The early and exact diagnosis of ALD and namely of fibrosis/cirrhosis is important since patients receive an explanation for their symptoms and complaints and get the opportunity to control disease progression through change of life style, avoidance of alcohol and other potentially harmful factors such as obesity.

The diagnosis of ALD has first to establish the consumption of alcohol as cause of the liver disease. Beside serum alcohol concentration measurements as indicator for alcohol consumption within the last 20 hours, no serologic marker can be used to monitor chronic alcohol consumption on its own (14).

While many species have been used to study ALD including baboons, pigs and rats, mice have been used predominantly in current ALD research. This is due to the availability of numerous transgenic and knockout mice that can easily help scientists determine the role of a particular molecule or signaling pathway in the pathogenesis of ALD.

Alcoholic hepatitis (AH) is a clinical syndrome among chronic alcohol drinkers who often also binge drink, resulting in hospitalization. Approximately 30%-40% of AH patients die within one month of diagnosis, and current treatment options, which are use of corticosteroids or pentoxifylline, only provide about a 50% survival benefit. Therefore, there is an
urgent need for development of new targeted therapies for AH. As previously mentioned, most mouse models only mimic early ALD pathogenesis. The advantages of this model are obvious because it is easy to perform and is more relevant to the human AH condition because it generates more liver injury and inflammation than other frequently used models. However, it should be noted that the phenotype in this model does not progress beyond liver steatosis, inflammation, and injury. Therefore, improved animal models that more accurately reflect the human condition are still needed for studying ALD progression (15).

References