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Perspective

Effects of Alcohol Use on Non-alcoholic Fatty Liver Disease and Chronic Kidney Disease

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DESCRIPTION

Around 25% of the adult population worldwide now has Non-Alcoholic Fatty Liver Disease (NAFLD), making it the most prevalent chronic liver disease. Its incidence is anticipated to rise annually as urbanization develops and conditions like obesity and diabetes become more common. After removing excessive alcohol use and other prevalent causes of chronic liver disease, NAFLD is defined by the presence of 5% hepatic fat build-up (e.g., viral or autoimmune hepatitis). NAFLD may progress to cirrhosis, hepatocellular carcinoma, and liver fibrosis. Non-Alcoholic Steatohepatitis (NASH) or non-alcoholic fatty liver are the two categories that apply to it Non-Alcoholic Fatty Liver (NAFL). Since it affects multiple systems, NAFLD is regarded as a separate risk factor for cardiovascular disease. However, there is mounting evidence that it plays a pathogenic role in the onset of Chronic Kidney Disease (CKD), and the occurrence of fibrosis may be linked to an increased risk of CKD incidence.

Chronic kidney disease is a major global health issue because it increases the risk of end-stage renal disease, cardiovascular disease, and significant morbidity and mortality. Numerous cardiovascular risk factors, such as diabetes mellitus, obesity, dyslipidemia, and hypertension, are shared by both NAFLD and CKD. Additionally, numerous researches have demonstrated that drinking alcohol has an impact on the prognosis of CKD patients. Chronic alcohol use has grown to be a serious global health risk. However, there is proof that moderate alcohol use lowers all-cause mortality in NAFLD patients and is linked to a low incidence of intrahepatic fibrosis and NASH. When compared to non-drinkers and heavy drinkers, moderate drinkers in the general population have a lower prevalence of CKD, which may be related to the fact that alcohol can enhance insulin sensitivity, raise levels of protective lipoproteins, and lower inflammation.

Alcohol intake and CKD in NAFLD patients, however, has not been the subject of any research that is pertinent; therefore, it is unknown whether this protective effect also applies to NAFLD patients. Therefore, the purpose of this study was to assess how drinking alcohol affected individuals with non-alcoholic fatty liver disease who later developed chronic kidney disease. In this population-based observational investigation, they discovered a significant reduction in kidney function in individuals with non-alcoholic fatty liver disease was adversely correlated with moderate alcohol consumption.

After taking into account potential covariates in the model, the results were only marginally reduced, indicating that the relationship between alcohol consumption and CKD may not be fully explained. However, this link was not seen in models adjusted for diabetes mellitus, ALT, AST, BUN, and other laboratory markers. Among fact, unadjusted ORs were likewise considerably lower in binge alcohol drinkers. Binge drinkers did not have a lower risk of negative renal outcomes.

These results suggest that the relationship between alcohol consumption and the development of CKD in NAFLD is significantly influenced by metabolic conditions. However, in women with NAFLD, they were unable to detect any impact of moderate alcohol consumption on kidney function. CKD, which affects up to 10%-15% of the general population and more than 25% of people over 65, continues to be a global public health burden. It is challenging to depict a relative association between the diagnosis of fatty liver disease and the onset and progression of renal disease because the characteristics of the metabolic syndrome, particularly obesity, diabetes, and hypertension, are risk factors for NAFLD.

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