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Commentary

The Role of Non-Alcoholic Fatty Liver Cirrhosis as a Risk Factor for the Severity of Acute Cholangitis

Jing Xue*

Department of Medicine, Stanford University, Boston, USA

DESCRIPTION

One of the most common chronic liver conditions globally is Nonalcoholic Fatty Liver Disease (NAFLD). This term refers to a broad spectrum of liver disease that starts with steatosis and inflammation and develops to Non-Alcoholic Steatohepatitis (NASH), fibrosis, cirrhosis, and finally hepatocellular cancer. Patients with NAFLD have been documented to exhibit biliary and pancreatic symptoms. NAFLD has been demonstrated to be positively linked with pancreatitis and the severity of the condition. NAFLD is regarded as the hepatic manifestation of the metabolic syndrome and is linked to it. Furthermore, there is a connection between NAFLD and a number of metabolic syndrome symptoms, particularly obesity and diabetes mellitus. It's significant to note that an imbalance in the gut flora has been linked pathogenetically to NAFLD. Recent research has shown that changes in the gut microbiome are related to NAFLD and the development of NASH. Inflammation and stasis in the biliary tract cause Acute Cholangitis (AC), a clinical condition that manifests as fever, jaundice, and abdominal pain. A different name for it is ascending cholangitis. The severity of cholangitis can range from moderate to life-threatening, notwithstanding Charcot's initial description of it as a serious and potentially fatal condition.

Inflammasome activation in cholangiocyte pathophysiology and the intriguingly recently identified Gut-Liver Axis play key roles in the pathogenesis of acute cholangitis, much as they do in NAFLD. There are no statistics on the prevalence, prognosis, or clinical course of cholangiocarcinoma in this rapidly expanding NAFLD population. The impact of the altered gut flora in NAFLD on cholangitis susceptibility is still unknown. The purpose of this study is to establish whether NAFLD poses a risk for severe AC. The link between NAFLD and bacterial infections has been demonstrated in a number of recent researches. The most likely explanation, though the precise mechanism is still unknown, seems to involve changes in the way the immune system functions and involves the dynamic gut-liver axis. This is likely due to the activation of the NLRP3 inflammasome and its recently described role in Cholangiocyte pathophysiology. The gut barrier failure and intestinal immunological abnormalities, which may overtax the liver's defences and let germs enter the bloodstream freely, are further potential contributors to this recently identified axis. The processes by which severe cholangitis may be brought on by NAFLD are still unknown. A growing body of research suggests that people with NAFLD and non-alcoholic steatohepatitis have greater levels of pro inflammatory cytokines, higher levels of oxidative stress, and altered lipoprotein metabolism. Furthermore, due to the discovery that adipocytes secrete a number of cytokines including IL-6 and TNF-alpha, which support inflammation, NAFLD is intimately linked to metabolic syndrome and obesity, both of which are now recognized as chronic low-grade inflammatory states. Intriguingly, regardless of BMI, our study found that NAFLD was independently linked to the occurrence of severe cholangitis. Additionally, it has been demonstrated that NAFLD independently increases the risk of severe pancreatitis through likely related processes. The lack of liver biopsies for the diagnosis of NAFLD is another significant constraint. As a result, we found no correlation between NAFLD participants' levels of inflammation and hepatic steatosis or NASH. It has been established that those with NAFLD are more likely to get severe cholangitis, as well as bacteremia and organ failure. More thorough research are urgently required to describe this significant link and effectively lower morbidity and mortality in this vulnerable patient population due to the high prevalence of NAFLD and the significant morbidity and mortality attributed to severe cholangitis.

Correspondence to: Jing Xue, Department of Medicine, Stanford University, Boston, USA, E-mail: Xue@gmail.com

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