A Tale of Two Diseases or A Story of Co-Existence: Are Obese Individuals' Immune To Alcoholic Liver Disease?

Dear Sir, I read with interest the article by Mehta et al. in recent issue of the journal and wish to congratulate the authors for their study.¹ This study may have implications in management of obese patients with alcoholic liver disease (ALD).

The association of non-alcoholic fatty liver disease (NAFLD) and ALD may initially seem implausible since hepatologists are used to identifying them as separate entities. However, the presence of one disease in a person does not exclude insult by another. The interesting aspect here is that these diseases affect a common organ. In fact, rather than merely co-existing with each other, presence of NAFLD seems to accentuate the progression of ALD.

Naveau et al. in an elegant study involving 1604 individuals showed that overweight individuals with alcoholism are more prone to develop (histologically proven) steatosis, alcoholic hepatitis and liver cirrhosis, as compared with their non-overweight counterparts.²

In a large population based study from Finland, it was shown that the presence of metabolic syndrome and type 2 diabetes mellitus was significantly higher amongst the cohorts with NAFLD and ALD as compared with those having normal liver function tests.³

The fact that the pathogenic mechanism of ALD and NAFLD have common factors involved such as induction of CYP2E1 and oxidative stress, further points toward the association. Because oxidative stress is associated with steatosis in patients with NAFLD, alcohol abuse may further exacerbate the oxidative stress, thereby lead to progression of liver disease.⁴⁻⁶

Co-existence of two types of liver disease was recently seen in our-outpatient clinic when we came across a 47-year-male in our out-patient clinic with ALT and AST more than twofold upper limit of normal, impaired fasting blood sugar, dyslipidemia and found to have extra-hepatic portal vein obstruction (EHPVO) with fatty liver. He denied history of alcoholism, there was no evidence of portal cavernoma cholangiopathy, other causes of hepatitis were ruled out, hence diagnosed as non-alcoholic

steatohepatitis (NASH), upper gastrointestinal endoscopy ruled out esophageal varices and elastography was 5.4 kPa. He is presently being treated as NASH.

To conclude, it is important to identify the presence of metabolic syndrome in patients with ALD, as nutritional therapy in this group of patients may have implications in their management. Further studies to look for outcome of treating aspects of metabolic syndrome in patients with ALD can establish this association even more firmly.

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