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LETTER TO THE EDITOR

Endothelial dysfunction in patients with non-alcoholic steatohepatitis

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Dear Editor,

I read with great interest the paper by Arinc et al. (1). They investigated the presence of endothelial dysfunction in a group of patients with non-alcoholic steatohepatitis (NASH). They found that patients with NASH have impaired flow-mediated dilatation (FMD) and increased carotid artery intima-media thickness (CIMT) when compared with healthy controls. And serum concentrations of gamma glutamyl transferase (GGT) and alanine transaminase (ALT) were associated with FMD and CIMT. The authors concluded that serum concentrations of GGT and ALT might have a predictive value for FMD and CIMT in patients with NASH.

A relationship between endothelial dysfunction and NASH has previously been established (2,3). Targher et al. showed that NASH predicted CIMT independently of potential confounders (2). Villanova et al. measured the vasodilatory response of the brachial artery in response to ischemia as well as cardiovascular risk profiles in non-alcoholic fatty liver disease (NAFLD)/NASH patients and controls (3). FMD was 6.3% in NAFLD versus 12.2% in controls ($p < 0.0001$), and higher in pure fatty liver (9.9%) compared with NASH patients (4.9%) ($p = 0.01$). Among NAFLD patients, low FMV was associated with NASH. The 10-year probability of cardiovascular events was moderately increased in NAFLD, and particularly so in NASH. This study provided evidence of endothelial dysfunction and increased risk of cardiovascular events in NAFLD and NASH (3). I appreciate the efforts of Arinc et al., but I think there are some points to be clarified related to their study.

First, the study was suggested to be unique in that CIMT and FMD were previously studied in patients suffering from NAFLD only but not NASH. A clear definition of NASH was not included in the methods section. How was the tissue diagnosis of NASH made in these patients? Severe steatosis, necroinflammatory activity (hepatocyte ballooning, necrosis), and stainable iron were previously proposed to be risk factors for advanced liver disease (2,3). I think that a detailed presentation of the histopathological findings and a search for a correlation of tissue parameters with FMD and CIMT would have improved the power of their study. Second, serum ALT concentrations of more than twice the upper limit of normal is a risk factor for advanced liver disease in patients with NAFLD (4,5). Could the authors provide a similar cut-off point for GGT and ALT to predict endothelial dysfunction?

Declaration of interest: The author reports no conflicts of interest. The author alone is responsible for the content and writing of the paper.

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