Dig Dis Sci (2012) 57:1109
DOI 10.1007/s10620-012-2066-y

CORRESPONDENCE

Mechanisms Linking Nonalcoholic Fatty Liver Disease with Coronary Artery Disease

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Received: 18 December 2011 / Accepted: 16 January 2012 / Published online: 7 February 2012

Nseir et al. [1] discuss the association between non-alcoholic fatty liver disease (NAFLD) and coronary artery disease (CAD). Some comments may be of interest.

A post hoc analysis of the Greek Atorvastatin and Coronary Heart Disease Evaluation (GREACE) study included 437 patients with CAD, dyslipidemia (low-density cholesterol >2.6 mmol/l) and NAFLD [2]. NAFLD was assumed in patients with moderately elevated transaminase activities (<3× the upper limit of normal) together with relevant ultrasonographic findings, after excluding other causes of abnormal liver function tests (LFTs) [2]. Patients with abnormal LFTs treated with statins had fewer cardiovascular (CV) events compared with those not on statins (68% relative risk reduction, $p < 0.0001$) [2]. Interestingly, the CV benefit from statin treatment was greater ($p = 0.0074$) in patients with abnormal than in those with normal baseline LFTs (39% relative risk reduction, $p < 0.0001$) [2]. Transaminase activities were reduced in patients treated with statins while further rises were noted in those not on statins [2]. However, these promising findings have limitations, including the post hoc analysis and small number of patients. Furthermore, liver biopsy, the "gold standard" for the diagnosis of NAFLD, was not performed.

We agree with Nseir et al. [1] that non-esterified fatty acids (NEFAs) may play a role in atherogenesis. In this context, NEFAs inhibit the synthesis and accelerate the degradation of prostacyclin (PGI_2) [3, 4]. PGI_2 is a vasodilator and platelet activation inhibitor. NEFAs also decrease vascular adenosine diphosphate (ADP)ase activity, thereby adversely affecting another platelet inhibitory process [5].

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