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LETTER TO THE EDITOR

No association between the degree of liver steatosis and early signs of vasculopathy in T2DM

Non alcoholic fatty liver disease (NAFLD) is both an independent and an associated risk factor for cardiovascular (CV) disease in the general population [1]. Whereas the association between NAFLD, and early signs of vasculopathy, such as an increased intima-media thickness (IMT) and a decreased flow-mediated vasodilation (FMD), has been reported in the general population, such an association in type 2 diabetes mellitus (T2DM) is controversial. In T2DM patients with NAFLD FMD was decreased [2], whereas IMT was not different, with respect to patients without liver steatosis [3]. Should a (causative) relationship between hepatic steatosis and early signs of vasculopathy exists, the degree of liver fat should be associated with a worse endothelial function and morphology. However, despite the bulk of data generated on this complex association, insufficient reports exist on T2DM.

To this aim, we measured the extent of liver fat, average IMT, the presence and type of carotid plaques, and FMD, in sixty consecutive T2DM patients largely affected by features of the MS. Liver steatosis, IMT, and presence and types of carotid plaques, were evaluated by ultrasonography (using an HDI 5000 Philips Medical Systems apparatus, Bothell, WA, USA), with a broad-band width phased array transducer (2–5 MHz). Steatosis was divided into four classes following the traditional US classification (class 0: absence; classes 1–3: increasing degrees, of steatosis) [4]. IMT was assessed using standard procedures [5]. FMD was evaluated in 45 patients using an internationally validated approach [6]. Only six patients were current smokers, and seven had a positive history for CV disease (five for ischemic heart disease, and two for cerebrovascular disease). No subject was positive for hepatitis C virus infection.

The overall prevalence of steatosis was 88% (34% mild, 34% moderate e 20% severe). Average IMT was 0.88 $\pm$ 0.03 mm (Mean $\pm$ SE), significantly greater ($p < 0.0001$) than the mean value of a healthy, age- and sex matched population at our Institution (0.72 $\pm$ 0.03 mm). Fifty-eight percent of patients had carotid plaques. Average FMD in the patients (5.02 $\pm$ 0.81%) was lower ($p < 0.001$) than the normal values of healthy, age- and sex matched individuals from our Institution (6.56 $\pm$ 0.60%). Nevertheless, there was no difference, among the four classes of steatosis, in either FMD (class 0: 5.10 $\pm$ 0.89%; class 1: 4.97 $\pm$ 0.46%; class 2: 4.73 $\pm$ 0.40%; class 3: 5.25 $\pm$ 0.17%) ($p = 0.543$ by ANOVA), average IMT (0.82 $\pm$ 0.08; 0.93 $\pm$ 0.05; 0.85 $\pm$ 0.05; and 0.85 $\pm$ 0.06 mm, respectively; $p = 0.760$ by ANOVA), or the prevalence of carotid plaques (43; 70; 60 and 69% respectively, $p = 0.644$).

In conclusion, in T2DM patients largely exhibiting features of the MS, the degree of liver steatosis is not associated with early signs of (sub)clinical atherosclerosis and altered vascular function. These data question the role of liver fat as a direct determinant of early signs of vasculopathy in T2DM. Alternatively, it is possible that the burden of cardiovascular risk factors already present in these T2DM patients, obscure the possible contribution given by the degree of steatosis, on early signs of arteriosclerosis.

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