The impact of non alcoholic fatty liver disease (NAFLD) on carotid artery intima-media thickness as a risk factor for atherosclerosis

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Introduction
Nonalcoholic fatty liver disease (NAFLD) is a common and underdiagnosed chronic liver disease affecting up to one-third of the global population in Western countries. The prevalence of NAFLD is increased up to 70% to 90% among T2DM persons and makes it an important public health problem. NAFLD is considered a hepatic manifestation of metabolic syndrome and is closely associated with abdominal obesity, atherogenic dyslipidemia, and diabetes exposing subjects with NAFLD to an increased risk of developing cardiovascular disease. Carotid intima-media thickness (cIMT) is a reliable index of subclinical atherosclerosis and a mirror of atherosclerosis progression in NAFLD patients. Carotid IMT is higher for individuals with hepatosteatosis than for those with simple steatosis and the histological severity of NAFLD independently predicts cIMT (1,2).

The aim of this study was to determine the prevalence of NAFLD in diabetes mellitus (DM) patients, to evaluate the impact of clinical parameters and indices of body composition on the relation between NAFLD and carotid intima-media thickness (cIMT), in a type 2 diabetes mellitus population (T2DM).

Methods
We retrospectively enrolled 120 T2DM outpatients who regularly attended a Consulting Clinic in Cluj. Clinical, anthropometric and biochemical parameters were measured. NAFLD was diagnosed using fatty liver index (FLI, 3), an algorithm based on body mass index (BMI), waist circumference, triglycerides and gamma-glutamyltransferase (GGT). Hepatic steatosis (HS) and cIMT were evaluated by ultrasoundography (US). Body composition was assessed by bioelectric impedance (BIA, InBody 720) in all subjects.

Results
HS was correlated with NAFLD – FLI ($p < 0.0001$). Patients were divided into three groups: G1: FLI≤30 (n = 56); G3: FLI > 60 (n = 40), G2: intermediate group (n = 28). The prevalence of NAFLD using FLI ≥ 60 in DM patients was 40% (p = 0.035). cIMT increased with FLI (G3 = 0.54 ± 0.08 versus G1 = 0.40 ± 0.08 mm, $P < 0.0001$). FLI was associated with increased low-density lipoprotein cholesterol ($r = 0.38$), alanine aminotransferase ($r = 0.55$), BMI ($r = 0.34$), diastolic blood pressure ($r = 0.41$) and reduced high-density lipoprotein cholesterol ($r = -0.52$) and insulinemia ($r = -0.30$, all $P < 0.0001$). The correlations hold also in multivariate analysis after adjusting for age and gender. Behavioral variables (smoking, diet) and fasting plasma glucose, did not significantly differ between subjects with and without FLI.

Conclusions: The results of our study are suggestive for the fact that, in T2DM patients, fatty liver is not a direct mediator of early carotid atherosclerosis. Our data indicate that visceral fat accumulation and glycated hemoglobin are determinant factors of cIMT suggesting that controlling abdominal obesity and hyperglycemia might reduce atherosclerotic disease risk in NAFLD-T2DM subjects.

These findings support the hypothesis that the identification of NAFLD abdominal obese T2DM patients may help in CVD risk prediction, with important management implications.

Bibliography

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