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GIOVANNI LORENZINI
MILAN, ITALY



GIOVANNI LORENZINI
MEDICAL FOUNDATION
HOUSTON, USA



Weill Cornell Medical College

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Abstracts Issue



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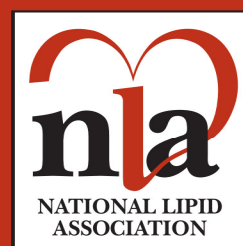


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ADIPONECTIN MODULATES THE EFFECT OF ATORVASTATIN ON PLASMA HDL CHOLESTEROL IN PATIENTS WITH TYPE 2 DIABETES

Mandy van Hoek, **Geesje M. Dallinga-Thie**, Hans Jansen, Eric J.G. Sijbrands, Arie van Tol. ErasmusMC, Rotterdam, Netherlands; AMC, Amsterdam, Netherlands.

Adiponectin plays an important role in lipid homeostasis and affects insulin sensitivity. We studied the effects of atorvastatin on plasma lipoproteins and adiponectin levels in patients with type 2 diabetes. In the DALI study, a randomized placebo-controlled study on the effects of aggressive atorvastatin treatment in patients with type 2 diabetes, plasma adiponectin levels, lipoproteins, as well as lipoprotein lipase (LPL) and hepatic lipase (HL) activities were assessed at baseline and after 6 months of treatment with placebo, 10mg (A10) or 80 mg atorvastatin (A80). At baseline, positive relationships were found between adiponectin and LPL activity ($r=0.19$ $p=0.012$) and HDL cholesterol ($r=0.46$, $P<0.001$). Negative relationships were present between adiponectin and HL activity ($r=-0.17$ $p=0.022$) and triglycerides ($r=-0.52$, $P<0.001$). Atorvastatin treatment had no effect on adiponectin levels. However, adiponectin levels at baseline significantly interacted with the effect of atorvastatin treatment on HDL-cholesterol ($p=0.007$), i.e. patients with the highest baseline plasma adiponectin concentration (tertile 3) displayed the largest increase in plasma HDL cholesterol during treatment (10%), while the increase in the lowest tertile group was negligible (1%). Adjustment for HL and LPL did not change the results. We conclude that plasma adiponectin is related to LPL (positive) and HL activity (negative). Furthermore, high adiponectin levels increase the HDL cholesterol response to atorvastatin treatment.

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THE METABOLIC SYNDROME DOES NOT ADD TO CAROTID ATHEROSCLEROSIS BEYOND THAT EXPECTED BY RISK FACTOR COUNTING OR RISK SCORING

José P. Werba, Samuela Castelnovo, Beatrice Frigerio, Mauro Amato, Alessio Ravani, Elena Tremoli, Damiano Baldassarre. IRCCS, Milan, Italy; Milan University, Milan, Italy.

Objective: The aim of this study was to assess if the Metabolic Syndrome (MS) has any add-on effect on subclinical atherosclerosis beyond that expected by risk factors (RF) counting or risk scoring. **Methods:** Intima-media thickness (IMT) of carotid arteries was assessed by using B-mode ultrasound in 1805 patients (56 ± 13 y; 52% women) attending a cardiovascular prevention program. Patients with (cases) or without (controls) MS according to NCEP ATP III criteria were 1:1 matched for sex, age and either the number of conventional RF (Analysis 1) or the Framingham risk score (Analysis 2). For Analysis 1 not more than 2 components of the MS were accepted as RF in the control group. **Results:** Case:control matches were 211 for Analysis 1 and 244 for Analysis 2. No significant differences in carotid IMTmean and carotid IMTmax were found between cases and controls in both analyses (Analysis 1: IMTmean 1.03 ± 0.38 vs 1.07 ± 0.37 ; IMTmax 1.90 ± 0.96 vs 1.95 ± 0.90 , cases and controls, respectively; Analysis 2: IMTmean 1.03 ± 0.36 vs 1.01 ± 0.33 ; IMTmax 1.91 ± 0.94 vs 1.83 ± 0.81 , cases and controls, respectively; all $p>0.1$). **Conclusions:** According to our results the metabolic syndrome does not add to the extent of carotid subclinical atherosclerosis beyond that expected by RF counting or risk scoring. These findings do not support any particular harmful synergism between components of the metabolic syndrome in determining carotid atherosclerosis.

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