Abstract: P730

Decreased adiponectin levels and FDG uptake in visceral adipose tissue in familial combined hyperlipidemia compared to heterozygous familial hypercholesterolemia and normolipidemics

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Background

Adipose tissue regulates energy balance and glucose homeostasis via the secretion of circulating molecules, termed adipokines, such as leptin and adiponectin. Excess adiposity and adipose tissue dysfunction have been involved in the pathogenesis of dyslipidemias. Positron emission tomography/computed tomography (PET/CT) with F-18-Fluorodeoxyglucose (FDG) has been used for the assessment of adiposity.

Purpose

To compare abdominal adipose tissue function assessed by FDG uptake with serum indices, such as plasma adipokines’ levels in individuals with different subtypes of dyslipidemia and normolipidemics.

Methods

Seventy individuals (mean age 44±13 years, range 21-75, 43 men) with a clinical diagnosis of either heterozygous familial hypercholesterolemia (heFH) (n=38) or familial combined hyperlipidemia (FCH) (n=32), not under statins for at least one year, and 20 age and sex matched controls, were enrolled. Visceral (VAT) and subcutaneous adipose tissue metabolic activity (SAT) was assessed with FDG-PET/CT imaging and was quantified by calculating the target-to-background ratios (TBR) in consecutive axial fat images between the proximal (cephalic) end of the L1 and distal (caudal) end of the L3 vertebrae by dividing the average of the mean standard uptake value (SUV) to the mean SUV of the vena cava. Leptin and adiponectin were measured in all the subjects.

Results

There was no significant difference of plasma leptin values between FCH, heFH and non dyslipidemias subjects (p =0.204). FCH had reduced adiponectin values compared to heFH patients and controls [median 5.7 IQR (3.9-7.6) vs. 13.1 (9.2-23.3) vs. 10.9 (6.1-19.1) µg/mL, respectively, p<0.001]. There was no difference in FDG uptake in subcutaneous adipocytes (SATTBR) between FCH, heFH and controls (p=0.161). In contrast, patients with FCH had reduced VATTBR values compared to heFH patients and controls (0.63±0.14 versus 0.81±0.17 versus 0.86±0.28, p=0.005). This difference remained significant even after adjustment for age, sex and cardiovascular risk factors (b=-0.428, p= 0.001, adjusted R2=0.219). SATTBR was inversely correlated to leptin levels (r=-0.484, p<0.001), while no significant association was observed with adiponectin values (p=0.167). No significant associations were observed between VATTBR and either serum leptin (p=0.066) or adiponectin levels (p=0.254).
Conclusions

Visceral adipose tissue FDG uptake is reduced in patients with FCH compared to those with heFH and normolipidemics. In addition, serum adiponectin levels are lower in patients with FCH. These findings highlight the different pathophysiological role of visceral fat function in the two most common types of familial dyslipidemia and suggest that visceral fat could be an attractive target for the treatment of FCH.