Abstract

Background and aims: Some studies have pointed to a role of UCP3 in the regulation of biochemical and fat parameters in overweight patients. The aim of our study was to investigate the influence of -55CT polymorphism of UCP3 gene (rs1800849) on histological changes and insulin resistance in patients with non-alcoholic fatty liver disease (NAFLD). Material and methods: A population of 39 patients with NAFLD was recruited in a cross sectional study. The inclusion criterion was the presence of biopsy-proven NAFLD. A biochemical analysis of serum (lipid profile, and adipocytokines) was measured. An anthropometric analysis was assessed, too. Genotype of UCP3 gene -55CT was studied. Results: Nine patients (23%) had the genotype 55CC (mutant type group) and 30 patients (77%) 55CT (wild type group). TT genotype was not detected. Insulin levels and HOMA were higher in mutant type group (insulin: 17.7 ± 10.9 mUI/L vs 11.9 ± 4.7 mUI/L; p < 0.05) and (HOMA: 3.2 ± 1.8 vs 4.5 ± 2.8; p < 0.05). Adiponectin levels were lower in mutants type group (36.5 ± 28.1 ug/ml vs 21.5 ± 18.6 ug/ml; p < 0.05). Moderate-severe inflammation and moderate-severe steatosis were more frequent in mutant type group, with higher levels of insulin and lower levels of adiponectin than mild stages. Conclusion: -55CT genotype is associated with high insulin resistance and low adiponectin levels than -55CC genotype. Patients with -55CT genotype have more frequently moderate-severe steatosis and inflammation than -55CC genotype.

Keywords

-55CT polymorphism of UCP3 gene, Biopsy, Insulin resistance, Steatosis, Obesity.