Abstract

Diabetes mellitus type II (DM II) is a disease that affects a large number of individuals. One of the drugs used for the treatment is metformin. Metformin is delivered into hepatocytes by a transporter encoded by the SL-C22A1 gene. Gene variants with reduced activity may decrease the amount of metformin available in the liver and reduce the therapeutic response. Various biochemical parameters were evaluated in relation to the metformin dose and the presence of transporter variants. A total of 103 patients older than 18 diagnosed with DM II who were treated with 1700 mg/day of metformin for more than six months were studied. Five polymorphisms in the SLC22A1 gene were analyzed as well as glycemia, HbA1c level, liver function, and lipid and kidney profiles. HbA1c and glycemia levels were higher in patients with the R61C, G401S, M420del and G465R polymorphisms; although the difference was statistically significant only for HbA1c in patients with the M420del and G465R variants (p=0.0273 and 0.0018, respectively). Polymorphisms with reduced activity in the SLC22A1 gene affect blood glucose - cose levels and HbA1c in patients with DM II when they are treated with metformin.

Keywords

diabetes type II * pharmacogenetics * SLC22A1 gene * metformin * polymorphisms