Homocysteine status and polymorphisms of methylenetetrahydrofolate reductase are not associated with restenosis after stenting in coronary arteries

Werner Koch, Gjin Ndrepepa, Julinda Mehilli, Siegmund Braun, Marc Burghartz, Klaus Kölling, Albert Schömig & Adnan Kastrati

OBJECTIVE: We investigated the influence of elevated homocysteine plasma levels and 2 polymorphisms, 677C/T and 1298A/C, of the methylenetetrahydrofolate reductase (MTHFR) gene on the risk of restenosis after stenting in patients with symptomatic coronary artery disease. METHODS AND RESULTS: Homocysteine levels and MTHFR genotypes were determined in 800 consecutive patients treated with coronary artery stenting. Angiographic restenosis (> or =50% diameter stenosis at 6-month follow-up) was present in 25.8% of the patients with low homocysteine levels (at or below the median of 11.6 micromol/L; n=400) and 24.1% of the patients with high homocysteine levels (>11.6 micromol/L; n=400; P=0.62). Rates of angiographic restenosis were 26.0%, 23.5%, and 26.9% in carriers of the 677CC, 677CT, and 677TT genotypes (P=0.75), respectively, and 24.4%, 25.9%, and 24.0% in patients with the 1298AA, 1298AC, and 1298CC genotypes (P=0.90), respectively. The need for restenosis-driven reintervention (clinical restenosis) was 18.8% in subjects with low homocysteine concentrations and 19.0% in subjects with high homocysteine concentrations during the first year after the intervention (P=0.93). Rates of clinical restenosis were 19.5%, 17.1%, and 23.3% in carriers of the 677CC, 677CT, and 677TT genotypes (P=0.37), respectively, and 17.6%, 18.6%, and 24.7% in patients with the 1298AA, 1298AC, and 1298CC genotypes (P=0.27), respectively. CONCLUSIONS: Elevated levels of homocysteine and 2 polymorphisms of the MTHFR gene are not associated with restenosis after stenting in coronary arteries.