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The association between TNFRSF1B rs1061624 polymorphism and hypertension in end-stage kidney disease patients on dialysis

Susana Coimbra[1,2], Susana Rocha[3], Cristina Catarino[3], Maria João Valente[4], Petronila Rocha-Pereira[3,5], Maria Do Sameiro Faria[3,6], José Gerardo Oliveira[7,8], João Fernandes[9], Vasco M. P. Miranda[10], Luís Belo[3], Elsa Bronze-Da-Rocha[3] and Alice Santos-Silva[3]

[1]UCIBIO—Applied Molecular Biosciences Unit, Department of Biological Sciences; Associate Laboratory i4HB, Institute for Health and Bioeconomy; Faculdade de Farmácia, Universidade do Porto, Porto, Portugal
[2]TOXRUN—Toxicology Research Unit, University Institute of Health Sciences, CESPU, CRL, Gandra, Portugal
[3]UCIBIO—Applied Molecular Biosciences Unit, Department of Biological Sciences, Faculdade de Farmácia, Universidade do Porto; Associate Laboratory i4HB, Institute for Health and Bioeconomy, Faculdade de Farmácia, Universidade do Porto, Porto, Portugal
[4]National Food Institute, Technical University of Denmark, Kgs Lyngby, Denmark
[5]Health Science Research Centre, University of Beira Interior, Covilhã, Portugal
[6]Hemodialysis Clinic Hospital Agostinho Ribeiro, Felgueiras, Portugal
[7]Center for Health Technology and Services Research (CINTESIS), Faculty of Medicine, University of Porto, Portugal
[8]Hemodialysis Clinic of Porto (CHP), Porto, Portugal
[9]NefroServe Hemodialysis Clinic of Viana do Castelo, Viana do Castelo, Portugal
[10]Hemodialysis Clinic of Gondomar, Gondomar, Portugal

Background and Aims: In chronic kidney disease (CKD), hypertension (HT) is commonly found during its development and is a *leading cause* for its progression. HT, dyslipidemia and visceral obesity are among the components of metabolic syndrome (MetS) and seem to associate with progression of CKD to end-stage kidney disease (ESKD).

Enhanced levels of soluble tumor necrosis factor receptor 2 (sTNFR2) are known to associate with progressive CKD; and this biomarker was reported as an independent predictor of all-cause mortality in ESKD patients under dialysis. Concerning the *TNFRSF1B* gene, the polymorphism rs1061624 (A > G), located in the 3′-untranslated region (3′-UTR), was associated with the development of HT in men and with increased risk of MetS.

The aim of this study was to determine the genotype frequencies of *TNFRSF1B* rs1061624 in ESKD patients with and without HT, and to evaluate its relationship with the circulating levels of some MetS biomarkers.

Method: We studied 277 ESKD patients on dialysis (recruited between 2016-2019), 170 diagnosed with HT and 107 without HT (no-HT group); age, sex, body mass index (BMI), systolic blood pressure (SBP), diastolic blood pressure (DBP) and statin therapy were recorded. Real time PCR TaqMan SNP genotyping assay was used to assess genotype frequencies of *TNFRSF1B* rs1061624. We also evaluated the conventional lipid profile and the levels of oxidized low-density lipoprotein, adiponectin and leptin.

Results: The genotype frequencies in patients with and without HT were different ($P = 0.002$); the AA genotype was less frequent in HT than in no-HT group, while GG and AG genotypes were more frequent. Considering the AA genotype carriers, HT ESKD patients ($n = 23$), compared to the no-HT group ($n = 33$), presented lower adiponectin levels ($P = 0.025$); for the AG genotype carriers, HT group ($n = 89$) presented a trend towards lower adiponectin concentrations ($P = 0.055$), and were younger than no-HT patients ($n = 48$; $P<0.001$); in both comparisons, groups were matched for sex, BMI and number of patients under statins treatment, and in the first case also for age. Considering GG genotype, HT patients ($n = 58$) only differed from no-HT subjects ($n = 26$) in SBP, that was higher ($P<0.001$). Among HT patients, AG genotype carriers presented higher DBP ($P = 0.004$), lower values of leptin ($P = 0.012$) and leptin/adiponectin ratio ($P = 0.022$) and were younger ($P<0.001$) than GG genotype patients; compared to AA carriers, cholesterol levels were lower ($P = 0.012$). For the no-HT group, AG genotype patients presented lower DBP ($P = 0.008$) than AA genotype carriers.

Conclusion: The genotype frequencies were different in ESKD patients with and without HT, being G allele more frequent in HT. The HT ESKD patients with the AA or the AG genotypes seem to present lower adiponectin levels than the ESKD no-HT patients with the same genotypes.

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