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ASSOCIATION BETWEEN A-ADDUCIN GENE, ANGIOTENSIN CONVERTING ENZYME GENE POLYMORPHISMS AND HYPERTENSIVE KIDNEY LESIONS IN HYPERTENSIVE PATIENTS

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Objective To investigate the association between polymorphisms of α-adducin (ADD) gene Gly460Trp, angiotensin converting enzyme (ACE) gene I/D and kidney lesions in patients with essential hypertension respectively and jointly.

Methods The case-control study was performed in 169 hospitalised hypertensive patients and 169 normal subjects (group A). On the basis of creatinine clearance calculated by Cockcroft–Gault equation, hypertensive patients were divided into two groups: normal renal function group (group B: Ccr≥80 ml/min) with 63 subjects and abnormal renal function group (group C: Ccr<80 ml/min) with 106 subjects. Basic clinical data such as sex, age, body mass, height, hierarchy and course of hypertension were collected; biochemical parameters such as serum creatinine, urea nitrogen, uric acid, fasting plasma glucose, lipids were measured; PCR was used to characterise ACE genotypes (ID, II, DD); PCR and restriction fragment length polymorphism (RFLP) were used to characterise α-adducin genotypes (GT, GG, TT). Both of them were confirmed by DNA sequencing.

Results (1) The levels of age, serum creatinine, urea nitrogen and uric acid were higher in group C by comparison with group A and B, and had significant difference ($p<0.05$). There was no significance of sex, body mass index, fasting plasma glucose and lipids among group A, B and C ($p>0.05$). (2) The expected frequencies of the ACE and ADD genotypes were under the assumption of the Hardy-Weinberg equilibrium in hypertensive group and normal group respectively ($p>0.05$). (3) Chi-square test demonstrated that the distributions of ACE -DD, ADD-TT homozygote and ACE-DD+ADD-TT genotype (25.47%, 30.19% and 13.21% respectively) were higher in group C than in A (the distribution was, 10.06%, 14.79% and 1.59%, respectively) and in B (the distribution was 7.94%, 11.11% and 1.59% respectively); and had significant difference ($p<0.05$), while there was no significance of ACE-DD, ADD-TT homozygote and ACE-DD+ADD-TT genotype between group A and B ($p>0.05$). (4) Binary logistic stepwise regression demonstrated that hypertensive course and ACE-DD+ADD-TT genotype entered the model at last, and was

significantly associated with abnormal renal function in group B and C. The OR were 1.006 (95% CI 1.004 to 1.008) and 4.977 (95% CI 1.465 to 16.914).

Conclusion It was suggested that both α-adducin-TT genotype and ACE –DD genotype were significantly associated with kidney lesions in patients with essential hypertension, respectively or jointly. ACE-DD+ADD–TT genotype can be the independent risk factor of hypertensive kidney lesions.