Association between PRKAG3 polymorphisms and sporadic Wolff-Parkinson-White syndrome

Ken-Pen Weng

**Introduction**: The aim of this study was to investigate whether mutation in AMPK subunit genes (PRKAG3-230) is associated with sporadic, isolated WPW syndrome.

**Methods**: This study consisted of 87 patients with symptomatic WPW syndrome, and 93 normal controls. PRKAG3-230 genotypes were determined by real-time PCR assay. Genotype and allele frequencies of PRKAG3-230 between patients with WPW syndrome and normal controls were analyzed.

**Result**: PRKAG3-230 were genotyped in 87 patients with WPW syndrome (M/F 53/34, age 24.4(18.0 yrs) and 93 normal controls (M/F 57/36, age 16.8(4.2 yrs). There were no significant differences in two groups in terms of age and sex. The patients with CG and CG+CC genotypes were with a significantly increased risk of WPW syndrome as compared to those with GG genotype (OR=1.99, 95% CI: 1.01-3.89, p=0.045; OR=1.99, 95% CI: 1.04-3.78, p=0.037, respectively). The allelic types were not associated with the risk of WPW syndrome. The patients with manifest type with CG and CG+CC genotypes were with a significantly increased risk of WPW syndrome as compared to those with GG genotype (OR=2.86, 95% CI: 1.16-7.05, p=0.022; OR=2.84, 95% CI: 1.19-6.80, p=0.019, respectively). The patients with right-side accessory pathways with CG and CG+CC genotypes were with a significantly increased risk of WPW syndrome as compared to those with GG genotype (OR=3.07, 95% CI: 1.25-7.51, p=0.014; OR=2.84, 95% CI: 1.19-6.80, p=0.019, respectively). The allelic types were not associated with the risk of WPW types and locations.

**Conclusion**: This study shows that PRKAG3-230 may be associated with sporadic WPW syndrome among the Taiwanese population. Further studies are warranted to elucidate the role of mutations in AMPK subunit genes other than PRKAG3-230 in sporadic WPW syndrome.