Escitalopram-induced QTc prolongation and its relationship with KCNQ1, KCNE1, and KCNH2 gene polymorphisms

Zimu Chen¹, Zhi Xu¹, Chenjie Gao¹, Lei Chen², Tingting Tan¹, Wenhao Jiang¹, Bingwei Chen³, Yonggui Yuan¹, and Zhijun Zhang¹

¹Southeast University Zhongda Hospital
²General Hospital of Eastern Theater Command, Nanjing, China
³Southeast University School of Public Health

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Abstract

Aims: We investigated the influence of KCNQ1, KCNE1, and KCNH2 gene polymorphisms along with clinical factors on escitalopram-induced QTc prolongation. Methods: A total of 713 patients prescribed escitalopram were identified and had at least one ECG recording in this retrospective study. 472 patients with two or more ECG data were divided into QTc prolongation (n=119) and non-prolongation (n=353) groups depending on the threshold change in QTc of 30ms above baseline value (ΔQTcΔ≥30ms). 45 patients in the QTc prolongation group and 90 patients in the QTc non-prolongation group were genotyped for 43 single nucleotide polymorphisms (SNPs) of KCNQ1, KCNE1, and KCNH2 genes. Results: Patients with QTc prolongation (ΔQTcΔ≥30ms) still got higher escitalopram dose (10.3mg) than patients without QTc prolongation (9.4mg), although no significant relationship was found between QTc interval and escitalopram dose among all patients. Patients who were older, or with coronary disease, or with hypertension were significantly at risk for QTc prolongation (ΔQTcΔ≥30ms). Concomitant antipsychotic treatment was associated with a longer QTc interval (7.469ms). Additionally, the distribution of KCNE1 rs1805127 C allele, KCNE1 rs4817668 CC genotype KCNH2 rs3807372 AG genotype differed significantly between the QTc prolongation and non-prolongation groups. Conclusion: Patients with QTc prolongation got higher escitalopram dose. Meanwhile, Greater age, coronary disease and hypertension are risk factors for QTc prolongation. Patients prescribed escitalopram would have longer QTc interval when prescribed concomitant antipsychotics at the same time. Moreover, polymorphism of KCN1 and KCNH2 SNPs play a role in escitalopram-induced QTc prolongation.

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