that homozygote mutant genotype ADRB1 1165G>C is associated with the increase in effectiveness of Metoprolol in treating hypertension, that suggest the selection of anti-hypertension according to genotype of patients.[45] Furthermore, in Korean patients suffering from heart failure, it was observed that people with ADRB1 Gly389X genotype, have shown a better response to Bisoprolol compared to Arg389Arg genotype. These results show that treatment with beta-blocker goes to treatment of each person separately, on the basis of genotype.[46] Another study indicates a 10 times difference in the effects of Esmolol on exercise-induced tachycardia in people with ADRB1 389 genotype.[47] Furthermore, according to previous studies, in people with ADRb1-389 Arg/Arg genotype, a higher dose of beta blocker is needed to achieve a treatment response rather than in people with Gly genotype carriers.[48]

According to the results of the above-mentioned studies, the Pharmacodynamic difference of Propranolol in the present study may be associated with polymorphism of beta-adrenergic receptors in the population under study and its effect on the increase in responsiveness to this drug. It is worth to conduct future studies about polymorphism of beta receptors in order to investigate Pharmacodynamic differences in our population.

Conclusion

According to the results of this study, it seems that pharmacokinetic differences are not able to explain over-responsiveness of our patients to propranolol. Pharmacodynamic differences in responding to beta blocker drugs by Renin secretion or having a different sensibility to beta receptors might play a role in making our population have a different response to propranolol.

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Ethical Issues

The approval number of the study was 2.3.84-458. The ethical standards were met according to Fifth revision of Declaration of Helsinki.

Conflict of Interest

The authors report no conflicts of interest.

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