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Mechanisms of Glybenclamide-Mediated Anti-Arrhythmia and Ischemic Conditionining in a Rat Model of Myocardial Infarction: Role of yohimbine Treatment

Ömer BOZDOĞAN[1], Ersöz GONCA[1], Eylem SUVEREN[1], Fatih GÖKÇE[2]
[1]Department of Biology, Faculty of Art and Science Abant İzzet Baysal University, 14280 Bolu - Turkey
[2]Department of Physiology, Faculty of Medicine Abant İzzet Baysal University, Düzce - Turkey

Abstract: Glibenclamide (Glyburide), a widely used second-generation sulfonylurea hypoglycemic agent, is a known specific blocker of adenosine triphosphate (ATP) sensitive potassium (K+) channels. Although the blockage of the ATP dependent potassium channel reduces arrhythmia, it increases enfarct size. Yohimbine is an alpha-2 blocker that reduces infarct size as suggested by several studies. We hypothesized that a combination of yohimbine and glibenclamide may be more effective in reducing the arrhythmia following ligation. The arteria descendence (LAD) branch of the left coronary artery in conscious rats was ligated and arrhythmia was recorded during the 15 min following coronary ligation. Sixteen hours after coronary ligation, infarcted myocardium was determined by histological analysis. One and five mg/kg of yohimbine and 5mg/kg of glibenclamide were given intraperitoneally 25 min before ligation. Yohimbine in a 5 mg/kg dosage reduced both the occurrence and the duration of ventricular fibrillation (VF) and ventricular tachycardia. The combination of yohimbine and glibenclamide did not exhibit any additive or synergistic effects on the antiarrhythmic effect of each drug alone following coronary ligation. Moreover, yohimbine in 5mg/kg doses and a combination of 1 mg/kg of yohimbine and glibenclamide increased the survival rate after coronary ligation compared to the non-treated group. The combination of glybenclamide with yohimbine had no clear additive effects such as reducing arrhythmia following cooronary ligation. The antiarrhythmia produced by glibenclamide may not only depend on the blockage of the ATP dependent potassium channels, but also on adrenergic preconditioning.

Key Words: ATP sensitive potassium channels, Hypoglycemic sulfonylurea compounds, Myocardial ischemia, Arrhythmia, Yohimbine

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