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Electrophysiological effects of rosiglitazone on heart ventricular papillary muscles of control and diabetic histidine decarboxylase knock-out and wild-type mice

Andrea Szebeni*, Ágnes Kovács, Valéria Kecskeméti

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Background

Rosiglitazone is a thiazolidinedione derivative oral hypoglycemic agent active in both diabetic animal models and type 2 diabetic patients. Rosiglitazone is a high affinity ligand for the peroxisome proliferator-activated receptor gamma, which is responsible for the insulin-sensitizing action of the compound. Recent large clinical trials found an association between the antidiabetic drug rosiglitazone therapy and increased risk of cardiovascular adverse events.

Methods

The aim of this report is to elucidate the cardiac electrophysiological properties of rosiglitazone on control and diabetic murine ventricular papillary muscles using conventional microelectrode technique.

Results

In control histidine-decarboxylase knock-out mice (HDC-KO) as well as in their wild-types (WT) rosiglitazone (1–30 μM) shortened AP duration at the 90% level of repolarization (APD_{90}) and increased the AP amplitude (APA) in a concentration-dependent manner. Moreover, rosiglitazone reduced the maximum velocity of depolarization (V_{max}). In diabetic animals we detected very similar effects.

Conclusions

The action potential changes caused by rosiglitazone probably can be explained by ion channel effects. The observed alterations may carry a serious proarrhythmic risk in case of overdose intoxication with rosiglitazone, especially in patients having multiple cardiovascular risk factors, like elderly diabetic patients.

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* Correspondence: szeband@pharma.sote.hu
Department of Pharmacology and Pharmacotherapy, Semmelweis University, 1089 Budapest, Hungary