Rosiglitazone Attenuates Atrial Structural Remodeling and Atrial Fibrillation Promotion in Alloxan-induced Diabetic Rabbits

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INTRODUCTION
The pleiotropic effects of glitazones may favorably affect atrial remodeling. We sought to investigate the effects of peroxisome proliferator-activated receptor-γ (PPAR-γ) activator rosiglitazone on atrial structural remodeling and atrial fibrillation (AF) promotion in alloxan-induced diabetic rabbits.

METHODS
Twenty alloxan-induced diabetic rabbits were examined. Ten of these were treated with rosiglitazone for 4 weeks and the remaining were left untreated while 10 additional healthy rabbits served as controls. Isolated Langendorff perfused rabbit hearts were used to evaluate atrial electrophysiological parameters and vulnerability to AF which examined by burst pacing. We also performed histology examination to measure atrial fibrosis and measured plasma oxidative stress and inflammatory biomarkers.

RESULTS
The duration of induced AF was significantly prolonged in the alloxan-induced diabetic rabbits than controls (1.6±0.4 s vs 0 s). Rosiglitazone treatment significantly reduced the duration of induced AF in the treated rabbits (1.6±0.4 s vs 1.2±0.05 s). Moreover, rosiglitazone attenuated atrial structural remodeling reducing the interatrial activation time (35.4±12.1 ms vs 24.2±10.8 ms, P<0.05; control 23.3±10.4 ms) and the atrial interstitial fibrosis as well (collagen volume fraction: 5.6±3.9% vs 2.4±2.1%, P<0.05; control 1.6±0.8%). Rosiglitazone increased plasma superoxide dismutase (SOD) activity and on the other hand decreased malondialdehyde (MDA), hs-C reactive protein, and tumor necrosis factor-α levels.

CONCLUSIONS
Rosiglitazone attenuates arrhythmogenic atrial structural remodeling and AF promotion in alloxan-induced diabetic rabbits. Also, it seems to modulate oxidative stress and inflammation in this experimental model.

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