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Modulation of Isoprenaline-Induced Myocardial Damage by Atorvastatin

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Abstract : Background: Isoprenaline (ISO) administration induces myocardial damage via oxidative stress and endothelial dysfunction. Atorvastatin (ATV) treatment improves both oxidative stress and endothelial dysfunction yet recent studies have reported a pro-oxidant effect upon ATV administration on both clinical and experimental studies. The present study was directed to investigate the effect of ATV pre-treatment and treatment on ISO-induced myocardial damage. Methods: Male rats were divided into five groups (n = 10). Rats were given ISO (5mg/kg/day, i.p.) for one week with or without ATV (10mg/kg/day, p.o.). ATV was given either as pre-treatment for one week before its co-administration with ISO for another week or as a treatment for two weeks at the end of the ISO administration. At the end of the experiment, the electrocardiographic examination was done and blood was isolated for the estimation of plasma creatine kinase MB (CK-MB) activity. Rats were then sacrificed and the whole ventricles were isolated for histological examination and the estimation of lipid peroxides as malondialdehyde (MDA) level, reduced glutathione (GSH) level, catalase activity, total nitrate-nitrite (NOx), as well as the estimation of both endothelial nitric oxide synthase (eNOS) and inducible nitric oxide synthase (iNOS) protein expression. Results: ISO-induced myocardial damage showed a significant elevation in ST segment, an increase in CK-MB activity, as well as increased oxidative stress biomarkers. Also, ISO-treated rats showed a significant decrease in myocardial NOx level and eNOS as well as degeneration in the myocardium. ATV pre-treatment didn't show any protection to ISO-treated rats. On the other hand, ATV treatment showed a significant decrease in both the elevated ST wave and CK-MB activity. Moreover, ATV Treatment succeeded to improve oxidative stress biomarkers, tissue NOx, and eNOS protein expression, as well as amelioration of the histological alterations. Conclusion: Pre-treatment with ATV failed to protect against ISO-induced damage. This might suggest a synergistic pro-oxidant effect upon administration of the pro-oxidant ISO along with ATV as demonstrated by the increased oxidative stress and endothelial dysfunction. On the other side, ATV treatment succeeded to significantly improve oxidative stress biomarkers, endothelial dysfunction and myocardial degeneration.

Keywords : atorvastatin, endothelial dysfunction, isoprenaline, oxidative stress
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