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Title :	Atorvastatin attenuates myeloperoxidase activity and regulates the Cyclooxygenase-2 receptors in the heart of Paraqut-exposed rats γ expression via PPAR
Authors :	Hassan Malekinejad, <u>MasoumiVerki Masoumeh</u> , Mona Khoramjouy, Sima Ahsan
Address :	Department of Pharmacology & Toxicology, Faculty of Veterinary Medicine, Urmia University, Urmia, Iran
Abstract :	Introduction: The main target tissue for paraquat (PQ) toxicity in mammalians is the lungs due to its accumulation against a concentration gradient. There are however, emerging data indicating that PQ exerts toxic effects in other organs such as liver, heart and kidneys. receptors in the γ The present study carried out to highlight the role of PPAR PQ-induced inflammatory alterations including the expression of cyclooxygenase-2 and also myeloperoxidase activity in the heart. The cardioprotective effect of atorvastatin also was examined. Materials and Methods: Forty two male Wistar rats (180-200 g) were exposed either against saline normal as control group or PQ (3.5 mg/kg, i.p.) as test groups for 14 consecutive days. The animals in the test groups received saline (PQ), pioglitazone (PGT, 10 mg/kg), Atorvastatine (STN, 10mg/kg), PGT + STN, PGT + GW 9662(0.6 mg/kg i.p.) and STN + GW9662 (0.6 mg/kg i.p.). Nitric oxide level and myeloperoxidase activity in the heart were determined. Expression of COX-2 at mRNA level also was studied. Results: Both PGT and STN were able to reduce the PQ-elevated myeloperoxidase activity significantly (P<0.05) and GW9662 pretreatment reversed the STN-reduced myeloperoxidase activity. STN but not PGT regulated the PQ- up-regulated COX-2 expression. The antagonistic effect of GW9662 on STN-related gene regulation also was documented. Conclusions: These data suggest that the PQ-induced myeloperoxidase activity and COX-2 up- regulation could be more effectively reversed by STN. Moreover, STN induced cardioprotective effects were antagonist suggesting that STN improves the γ significantly antagonized by PPAR receptors. γ PQ-induced damages in the heart via PPAR
Keywords :	Antagonist; Atorvastatin; γ Cyclooxygenase-2; Myeloperoxidase; PPAR