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Title :	Atorvastatin attenuates myeloperoxidase activity and regulates the Cyclooxygenase-2 receptors in the heart of Paraqut-exposed ratsγexpression via PPAR
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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 theγThe 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 theγsignificantly antagonized by PPAR receptors. γPQ-induced damages in the heart via PPAR
Keywords :	Antagonist; Atorvastatin; γCyclooxygenase-2; Myeloperoxidase;PPAR