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Protective effects of telmisartan against acute doxorubicin-induced cardiotoxicity in rats

Muzaffar Iqbal[1], Kiran Dubey[1], Tarique Anwer[1], Agrawal Ashish[2], Krishna K. Pillai[1]

[1]Department of Pharmacology, [2]Department of Pharmaceutical Medicine, Faculty of Pharmacy, Jamia Hamdard, (Hamdard University), New Delhi-110 062, India

Correspondence: Muzaffar Iqbal, e-mail: muziqbal@gmail.com, muzaffariqbal2002@yahoo.co.uk

Abstract:
The therapeutic usefulness of doxorubicin (DXR), an anthracycline antibiotic, is limited by its cardiotoxicity. The present study investigated the effects of telmisartan, an angiotensin II receptor (AT1) antagonist against doxorubicin-induced cardiotoxicity in rats using biochemical and histopathological approaches. Doxorubicin (20 mg/kg) was injected intraperitoneally (*ip*) as a single dose and telmisartan (10 mg/kg/day) was administered orally for 7 days. Rats treated with DXR showed cardiotoxicity as evidenced by elevation of serum lactate dehydrogenase (LDH) activity, tissue malondialdehyde (MDA) level, catalase activity and a decrease in the level of glutathione (GSH). Pre- and post-treatment with telmisartan elicited a significant decrease in the activities of LDH and catalase in comparison with DXR-treated group. Furthermore, pretreatment with telmisartan also decreased lipid peroxidation (MDA level) and increased the GSH content in comparison with DXR group. However, the difference in lipid peroxidation and GSH content were not statistically significant in post-treated group. Histopathological studies showed disruption of cardiac tissues in DXR groups. Pre- and post-treatment with telmisartan reduced the damage of cardiac tissue in rats. These results suggest that telmisartan treatment provides a significant protective effect against acute-doxorubicin induced cardiotoxicity in rats.

Key words:
doxorubicin, telmisartan, cardiotoxicity, angiotensin II, oxidative stress