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The use of ACE inhibitors, statins and omega 3 may ameliorate cardio toxicity of doxorubicin

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Abstract

Doxorubicin, also known as hydroxydaunorubicin, is an anthracycline antibiotic, its structure is closely related to the natural product daunomycin, and like all anthracyclines, works by inhibiting DNA replication. Doxorubicin is very beneficial in treatment of different types of cancer such as liver or breast cancer, its importance is unquestionable in arresting the size of tumor but its cardio toxicity limits its usefulness. There are different mechanisms involved in its cardio toxicity such as the release of free radicals, apoptosis, necrosis and autophagy of cardio myocytes. Doxorubicin-induced heart damage may be due to an increase in cardiac oxidative stress, as indicated by Reactive Oxygen Species (ROS) induced damage such as lipid peroxidation, along with reduced levels of antioxidants and mercapto groups (SH). It was found that irregularities in myofibrillar and intracellular calcium are also important mechanisms for doxorubicin-induced cardiac toxicity. Additionally, the changes in the high-energy phosphate pool, endothelin-1 levels, and disturbances of myocardial adrenergic signaling are the most suggested causing factors of cardiac toxicity associated with doxorubicin administration. Therefore, there are different trials aiming to suppress its cardio toxicity to widen its use. Here, in this abstract, we discussed that how much the use of captopril, omega 3 and rosuvastatin could reduce the doxorubicin induced cardio toxicity.

Captopril is an example of angiotensin converting enzyme inhibitor (ACEI), and largely is used in treatment of hypertension, heart failure and diabetic nephropathy. Additionally, captopril has anti-inflammatory and antioxidant properties due to inhibition of angiotensin ii formation and because of its structure which contains sulfhydryl group (SH) with high antioxidant properties. Magda Nasr et al., (2013) found that captopril (25mg/kg for 21 days) inhibited the cardio toxicity of doxorubicin which induced by intra-peritoneal (IP) injection of (12 mg/kg) doxorubicin in male mice. Moreover, it succeeded into reducing the cardiac level of tumor necrosis factor (TNF) which is a valid parameter of inflammatory mediators and increasing the cardiac level of antioxidant parameters such as super oxide dismutase (SOD) and glutathione in reduced form (GSH).

Biography

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