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Effects of Stanozolol on Apoptosis Mechanisms and Oxidative Stress in Rat Cardiac Tissue

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Abstract

Stanozolol is a widely used 17 α -alkylated anabolic androgenic steroid (AAS) derivative. Despite stanozolol's adverse effects, its effect on oxidative stress parameters and mitochondrial apoptosis pathway is not clearly defined. In our study, thirty four male Sprague-Dawley rats were divided into 5 groups as control (C), vehicle control (VC), steroid (ST), vehicle control-exercise (VCE), and steroid-exercise (STE). Animals were subcutaneously administered stanozolol 5 mg/kg in steroid groups and propylene glycol 1 ml/kg in the vehicle-control groups. On the 28th day-after sacrifice, oxidative stress (MDA, GSH, PC, SOD, CAT) and apoptosis parameters (TUNEL, Cytochrome-c) in cardiac tissue were evaluated. Also, blood vessel morphology of cardiac tissue was evaluated with Verhoeff-van Giesen staining. It has been demonstrated that stanozolol administration triggers apoptosis by using TUNEL assay and cytochrome-c immunohistochemical staining intensity, while this effect is significantly reduced in the presence of exercise. In conclusion, the present study demonstrated that stanozolol administration induces apoptosis with increasing PC and CAT levels, while GSH, MDA and SOD parameters do not reveal any significant change. Exercise has a protective role in stanozolol induced oxidative stress and apoptosis. According to Verhoeff –van Giesen staining results for blood vessel morphology assessment, it has been seen that exercise has a protective role on cardiac blood vessels. This mechanism needs further investigations with long term exposure studies for clarifying possible pathways.

Key Words: Stanozolol, Oxidative stress, Cardiac tissue, Apoptosis, Immunohistochemistry

1. Introduction

Anabolic androgenic steroids (AASs) are the synthetic derivatives of male hormone, testosterone and they have been reported as the most frequently detected doping drugs [1]. Currently, more than 100 types of AASs are available on the market. AASs are known to be used widely at supraphysiological doses which are approximately 10-100 times higher than therapeutic doses. In recent years, several studies have been reported that supraphysiological doses of AASs are associated with cardiovascular, hepatic, endocrine, reproductive and neurologic adverse effects [2,3,4]. One of the most important adverse effect of AASs is ventricular hypertrophy,

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