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Regional Characterization of Molecular Markers and Gene Products in Dilated Cardiomyopathy

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Abstract

Characterization of Molecular Markers and Gene Products in Dilated Cardiomyopathy Qussay Marashly1, Salma Awad2, Coralie Poizat2 1. College of Medicine, Alfaisal University, Riyadh, KSA. Contact info. qmarashli@alfaisal.edu, Mob. 0598106096 2. Cardiovascular Research Program, King Faisal Specialist Hospital & Research centre Dilated cardiomyopathy (DCM) is a form of cardiomyopathy that is characterized by progressive cardiac dilation and contractile (systolic) dysfunction, usually with concomitant hypertrophy. 20-50% of the cases are familial and caused by inherited genetic abnormalities; however, other causes (myocarditis, toxicities, and childbirth) exist. This study aims to evaluate the expression of some hypertrophy-related proteins, p53 (a tumor suppressor gene), and Mdm2 (negative regulator of p53). Human hearts were obtained from heart transplant patients or unmatched heart donors at King Faisal Heart Institute. The sample included 5 patients clinically suspected of having dilated cardiomyopathy due to a distinctly abnormal ECG, a family history of cardiac symptoms including DCM, and a severely reduced ejection fraction (<25%). Two normal controls were also included. Sample age ranges between 20-55 years old. Obtained hearts were dissected into left ventricle (LV), right ventricle (RV), and Septum (Sep). Each section was further dissected into (upper, middle, and lower) sub-sections. The expression of (ANP, β-MHC, SERCA-2, Actinin and Sc-Actin, p53, and Mdm2) in the different sub-sections was investigated by means of western blotting, Q-PCR and Immunofluorescence. This study provides significant insights into the regional effects of DCM on the human heart and the possible involvement of the p53 pathway in the pathogenesis of DCM.

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