

Reverse myocardial remodeling in hypertrophic cardiomyopathy: Little explored benefit of physical exercise

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Introduction: Hypertrophic cardiomyopathy (HCM) is an autosomal dominant disease that causes myocardial remodeling. Physical exercise (PE) is a resource that promotes reverse myocardial remodeling (RMR). However, the scientific literature is not clear about this aspect in patients with HCM. Objective: To report how a Supervised Cardiovascular Rehabilitation Work (SCR), using a personalized PE prescription, promoted RMR and an improvement in the QoL of a patient with HCM. **Case description:** ANPC, female, sedentary, 43 years old. Has Asymmetric Septal Type HCM. New York Heart Association (NYHA) grade III/IV Heart Failure (HF), treated with 40 mg of Propranolol Hydrochloride (PHcl). In the Doppler echocardiogram, an end-diastolic volume (EDV) of 130 ml, end-systolic volume (ESV) of 44 ml, with left ventricular mass (LVM) of 236 g, interventricular septum thickness of 14 mm, left ventricular posterior wall thickness = 9 mm, left atrium diameter 46 mm, left ventricular end-diastolic diameter (LVEDD) 52 mm, septum/left ventricular wall ratio 1.55 mm, ejection fraction (EF) 66% (Teicholz). The ECG-Holter demonstrated the presence of ventricular arrhythmias, of which 5 were isolated and 1 was an episode in pairs; supraventricular arrhythmias, 115 isolated, 19 paired and 3 tachycardias. After SRC, a 26% improvement in QoL was achieved on the Minnesota Living with Heart Failure Questionnaire (MLHFQ). 50% reduction in PHcl; increase in EF 66 vs 69%, decrease in VDF 130 vs 102 ml, decrease in SFV 44 vs 32 ml. Decrease in MVE 236 vs 201 g. **Discussion:** The ability of PE to induce RMR for dilation and concentric hypertrophy (CH) is well-established in the literature. Although we did not see regression of CH, we did see a reversal of dilation observed through the reduction of VDF and VSF, LVEDD and LVM. Experimental studies demonstrate that EF induces the action of microRNAs, improving cardiac function. In this case, there was an improvement in cardiac function and consequently in the patient's clinical context. The increase in QoL assessed by the MLHFQ points to this. The evolution in ADLs was the points highlighted by the patient. Other factors, such as the improvement in the activity of the renin-angiotensin system and adaptations in the peripheral muscles, may have had as strong an impact on QoL as the central improvements. As this is a case report, the results presented here must be analyzed with caution and impartiality. Furthermore, this work advances something new: the possibility of PE causing a reversal of dilation in HCM, something that to our knowledge, has not yet been reported in the literature. **Conclusion:** An RCS program is a treatment capable of promoting RMR and, consequently, improving the QoL of a patient with HCM.