

Abstract

Nutr Metab Cardiovasc Dis. 2010 Nov;20(9):641-6.

Vitamin D deficiency in the spontaneously hypertensive heart failure [SHHF] prone rat.

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BACKGROUND AND AIMS: Vitamin D deficiency has been associated with the etiology and pathogenesis of heart disease including congestive heart failure. We previously observed cardiac hypertrophy in vitamin D deficient rats and vitamin D receptor knockout mice. These studies indicate that the absence of vitamin D-mediated signal transduction and genomic activation results in increased sensitivity of the heart to inotropic stimuli and cardiomyocyte hypertrophy. This study's aim is to investigate the relationship between vitamin D status and the heart failure phenotype in the rat.

METHODS AND RESULTS: Vitamin D status was assessed by measuring 25-hydroxyvitamin D levels and related to heart weight in young, middle-aged and aging spontaneously hypertensive, heart failure (SHHF) prone rats. We also measured the effects of the vitamin D hormone, 1,25(OH)(2)D(3), on cardiac function in SHHF rats. Cardiac hypertrophy in this model of the failing heart increased with age and related to decreasing vitamin D status. Vitamin D deficiency presented after cardiac hypertrophy was first observed. Additionally, we found that 1,25(OH)(2)D(3) treatment between 4.0 and 7.0 months of age prevented cardiac hypertrophy and permits decreased workload for the heart while allowing adequate blood perfusion and pressure, resulting in reduced cardiac index.

CONCLUSIONS: Our findings suggest that low vitamin D status is associated with the progression and final terminal phase of the heart failure phenotype and not with initial heart hypertrophy. Also, we report that in the vitamin D sufficient SHHF rat, 1,25(OH)(2)D(3) treatment provided protection against the progression of the heart failure phenotype.

PMID: 19836216