

Safe new therapy for genetic heart disease

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A new clinical trial suggests that long-term use of candesartan, a drug currently used to treat hypertension, may significantly reduce the symptoms of genetic heart disease. The related report by Penicka et al, "The effects of candesartan on left ventricular hypertrophy and function in non-obstructive hypertrophic cardiomyopathy: a pilot, randomized study," appears in the January issue of *The Journal of Molecular Diagnostics*.

Hypertrophic cardiomyopathy, or HCM, is a genetic heart disease where the heart muscle is thickened, especially in the left ventricle. Although people with HCM usually display mild symptoms or are completely asymptomatic, up to 1% of affected people succumb to sudden cardiac death (SCD), often with no previous signs of illness. HCM can be caused by mutations in a number of different genes, and different gene mutations may result in more or less severe symptoms.

Researchers lead by Dr. Jiri Krupicka of Na Homolce Hospital, Prague conducted a double-blind, placebo-controlled, randomized study on the long-term administration of candesartan in patients with HCM. Dr. Krupicka's group found that candesartan reduced the symptoms of HCM, including decreasing the thickening of the left ventricle. This effect was found to be dependent on the underlying gene mutations in each patient. They did not observe any adverse effects of candesartan use over the course of their study.

These data suggest that effectiveness of angiotensin II receptor blockers, such as candesartan, on HCM may vary depending on the nature of the

causative mutation and that the treatment protocol, therefore, should be customized to individual patients. Future studies in Dr. Penicka's group will extend this pilot trial to more patients to confirm the current findings and to identify the mechanism by which candesartan improves left ventricle thickening.

Paper: Penicka M, Gregor P, Kerekes R, Marek D, Curila K, Krupicka J: The effects of candesartan on left ventricular hypertrophy and function in non-obstructive hypertrophic cardiomyopathy: a pilot, randomized study. J Mol Diagn 2009 11:35-41

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