J Clin Endocrinol Metab. 2015 Feb 24:jc20151063. [Epub ahead of print]

Long-term echocardiographic and cardioscintigraphic effects of growth hormone treatment in adults with Prader-Willi syndrome.

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Abstract

Context: In Prader-Willi Syndrome (PWS), an altered GH secretion has been related to reduced cardiac mass and systolic function compared to controls. Objectives: To evaluate the cardiovascular response to a 4 yr GH therapy in adult PWS patients. Study participants: Nine severely obese PWS adults (3 females, 6 males) and 13 age-, gender- and BMI-matched obese controls. Methods: In an open-label prospective study, assessment of endocrine parameters and metabolic outcome, whole body and abdominal fat scans, echocardiography, radionuclide angiography in unstimulated and dobutamine-stimulated conditions were conducted at baseline and after 1 and 4 yr of GH treatment. Results: GH treatment increased IGF-I (P<0.0001), decreased C-reactive protein levels (P<0.05), improved visceral fat mass (P<0.05), and achieved near-significant changes of fat and fat-free body mass in PWS patients. Left ventricle mass indexed by fat mass increased significantly after 1 yr and 4 yr of GH therapy (P<0.05) without evident abnormalities of diastolic function, while a trend toward a reduction of the ejection fraction was documented by echocardiography (P=0.054). Radionuclide angiography revealed stable values throughout the study both of the left and right ventricle ejection fraction, though being accompanied by a statistically not significant reduction of the left ventricle filling rate. A positive association between lean body mass and LVEF was evident during the study (P <0.05). Conclusions: GH therapy increased cardiac mass of PWS adults without causing overt abnormalities of systolic and diastolic function. While the association between lean mass and left ventricle ejection fraction during GH therapy corroborates a favorable systemic outcome of long-term GH treatment in adults with Prader-Willi syndrome, subtle longitudinal modifications of functional parameters advocate appropriate cardiac monitoring in the longterm therapeutic strategy for Prader-Willi Syndrome.

PMID:

25710568

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