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Short Commentary

Cardiac weight in infiltrative cardiomyopathies

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Abstract

In Infiltrative Cardiomyopathies (IC), abnormal deposition of various substances may lead to significant cardiac weight gain. This paper examines several reports of this phenomenon, and the extent of this weight gain in various ICs.

Keywords: Infiltrative cardiomyopathy; Weight gain.

Introduction

Due to accumulation of various substances in the myocardium, ICs are known for thickened cardiac muscle, hypertrophy, diastolic dysfunction, congestive heart failure, and other significant complications [1]. There are relatively few reports, however, on the actual amount of cardiac weight gain in these conditions, and comparisons of weight gain between different ICs has not been extensively studied. This report summaries the available data on these topics.

Methods

A literature search was conducted. Data on cardiac weight in various ICs is taken from autopsy studies.

Results

The average adult normal male heart weighs 280-340 grams (g), while the average adult normal female heart weighs 230-280 g. Weights can vary depending on body size and age [2].

In amyloidosis there is extensive extracellular amyloid deposition with markedly increased cardiac weight. Values of >500-600 g are frequently reported [3,4].

In cardiac hemochromatosis, iron deposition in myocytes leads to fibrosis. The cardiac weight is often moderately increased, with weights as high as 610 g (mean 425 g), but generally not quite as high as is amyloidosis [5].

In cardiac sarcoidosis, the cardiac weight can be normal or increased, depending on the degree of granulomatous inflammation and fibrosis. Weights in the 500-565 g range have been reported [6].

In Fabry disease, a lysosomal storage disorder leads to glycosphingolipid accumulation in cardiomyocytes. Weight is often moderately increased, in the 400-500 g range [7].

In Danon disease, Lysosome-Associated Membrane Protein 2 (LAMP2) deficiency leads to glycogen accumulation, and often significantly elevated weight gain. Adult male hearts of 600-1000 g have been reported [8].

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In Friedreich's ataxia, frataxin deficiency leads to mitochondrial dysfunction causing hypertrophy, fibrosis, and weight gain. Weights of 400-600g have been reported, but generally less extreme than in Danon disease [9].

In scleroderma, there have been some reports of weight gain (315 g) due to myocardial fibrosis and scarring, but this is not universally seen [10].

In cardiac lymphoma, lymphocytic infiltration has been reported to increase cardiac weight. In one case the heart weighed 910 g [11].

In a case of metastatic pancreatic carcinoma to the heart, the heart weighed 780 g [12]. In secondary Oxalosis associated with renal failure, calcium oxalate deposition has been reported in the myocardium. No data on cardiac weight was reported. In Pompe disease, deficiency of lyposomal enzyme acid α-glucosidase (GAA) has been associated with glycogen accumulation in the heart. The extent seems to depend on the subtype [13].

Discussion

In ICs, cardiac weight can increase from a small to a very large amount, depending on the particular disease. Of interest is that as the infiltrative process takes place and the myocardium becomes heavier, it would take an increasing amount of force to initiate systolic ejection. Thus the increased weight causes an increase in afterload, which can worsen systolic function even further [14].

Also, since the heart sits atop the left hemi diaphragm, increasing weight would progressively impair left diaphragmatic excursion, and cause more compressive atelectasis at the left lung base. These factors may cause reduced pulmonary ventilation with abnormal gas exchange, causing more shortness of breath [15].

Conclusion

Progressive cardiac weight gain is an important feature of many infiltrative cardiomyopathies. Further studies may be of considerable interest.

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