Dilated cardiomyopathy

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Dilated cardiomyopathy (DCM) refers to chronic disease of the heart muscle leading to cavity enlargement and reduced systolic function of the left or both ventricles. The diagnosis follows on from the exclusion of specific causes including hypertension, coronary artery disease, rapid supraventricular arrhythmias, congenital heart disease, pericardial disease, cor pulmonale, and systemic disease such as sarcoidosis. In addition, though alcohol excess can lead to a DCM, abstinence may lead to reversal, so it is only included if a minimum of six months of complete abstinence still leaves a DCM at echocardiography.

The prevalence of DCM in the United States (adjusted for age) is 36 per 100,000 of the population. The etiology includes genetic transmission (estimated at 30–40%) indentifying familial DCM, cytotoxic agents (e.g., anthracycline derivatives), malnutrition (e.g., protein deficiency), myocarditis (viral etiology), and autoimmune disease.

The five-year survival averages 30–40% and has been improved by modern heart failure therapy, but 20% will die within a year of the diagnosis due to sudden death in most instances (64%) and heart failure deterioration in the remainder. Therefore not everyone responds and some patients rapidly deteriorate no matter the therapeutic efforts, and for them, if suitable, heart transplantation is the only option. Beta blockade and angiotensin-converting enzyme (ACE) inhibitors or angiotensin II antagonists improve left ventricular function in 50% of cases, and in 16% it returns to normal, but the remaining 34% suffer long-term disease progression.

In this issue we focus on DCM, opening up the door to metabolic mechanisms and potential therapies that may improve symptoms and prognosis. Metabolic agents such as trimetazidine can optimize myocardial energy metabolism, leading to increased energy from glucose rather than free fatty acids. The link between understanding energy metabolism and selecting therapy using pharmacology or devices is thoroughly explored and offers therapeutic options that may be complimentary rather than alternatives. The value of imaging in diagnosis and monitoring therapy reveals once more a potential benefit from the metabolic agent trimetazidine (a partial free fatty acid beta-oxidation inhibitor) both symptomatically and potentially prognostically, and the need for large-scale multicenter outcome studies.

Cardiac amyloidosis is almost certainly under diagnosed so it is timely that it is reviewed as part of this DCM issue. Though the prognosis is poor, determining the precise etiology can lead to targeted therapy and improved survival.

Throughout this issue of *Heart and Metabolism*, DCM is recognized as a challenging condition. By combining an understanding of the etiology, metabolic mechanisms, detection and therapeutic options, we explore the means of rising to the challenge

Further reading

Key references are provided with each article. A comprehensive review is provided in Hess OM, McKenna W, Schultheiss H-P (2009) Myocardial Disease. In: Camm AJ, Luscher TF, Serruys PW (eds.) ESC Textbook of Cardiovascular Medicine. Oxford University Press, Oxford, pp 665–716.