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List of Abbreviations

ACC ..... American College of Cardiology

AHA ..... American Heart Association

AC ..... Air Condition

CABG ..... Coronary Artery Bypass Graft

CAD ..... Coronary Artery Disease

CI ..... Cardiac Index

CMR ..... Cardiovascular Magnetic Resonance

CT ..... Computed Tomography

DCM ..... Dilated Cardiomyopathy

ECG ..... Electrocardiogram

EF ..... Ejection Fraction

GH ..... Growth Hormone

HCM ..... Hypertrophic Cardiomyopathy

HIV ..... Human Immunodeficiency Virus

ICM ..... Ischemic Cardiomyopathy

LV ..... Left Ventricle

LVEF ..... Left Ventricular Ejection Fraction

LVNC ..... Left Ventricular Non-compaction

MI ..... Myocardial Infarction

MR ..... Mitral Regurgitation

NYHA ..... New York Heart Association

SPECT ..... Single Photon Emission Computed Tomography

SRI ..... Stress Rate Imaging

SWMA ..... Segmental Wall Motion Abnormality

TDE ..... Tissue Doppler Echocardiography

WMSI ..... Wall Motion Score Index

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INTRODUCTION

Cardiomyopathy is an anatomic and pathologic diagnosis associated with muscle or electrical dysfunction of the heart. Cardiomyopathies represent a heterogeneous group of diseases that often lead to progressive heart failure with significant morbidity and mortality. Cardiomyopathies may be primary (i.e., genetic, mixed, or acquired) or secondary (e.g., infiltrative, toxic, inflammatory). Major types include dilated cardiomyopathy, hypertrophic cardiomyopathy, restrictive cardiomyopathy, and arrhythmogenic right ventricular cardiomyopathy ***(Wexler, et al., 2009).***

It is difficult to make an accurate clinical identification of heart failure resulting from poor ventricular function, but it is important to do so because of the need to relieve symptoms. A substantial number of patients with heart failure have normal ECG results. In other patients with apparent heart failure, echocardiography provides extra information on the nature of the cardiac disease that affects management. In addition, providing appropriate treatment is important in patients affected more severely (ejection fraction <35-40%), in whom treatment can significantly reduce the mortality rate ***(Burianova, et al., 2009).***

For most patients with heart failure, a search for underlying ischemia and viability would be an appropriate clinical strategy at some point in their evaluation. If substantial ischemia or viability of dysfunctional territories is found in the setting of vessels technically amenable to revascularization, the literature would suggest a clinical benefit from revascularization ***(Ficaro, et al., 2010).***

Myocardial structure, perfusion, and function are routinely assessed with echocardiography, nuclear imaging techniques, or cardiac magnetic resonance imaging. Two-dimensional echocardiography including Doppler flow study is an established technique for assessment of left ventricular (LV) size and function. Information on myocardial structure is gained from observation of abnormalities in end-diastolic myocardial wall thickness (e.g., too thin or too thick), and information on myocardial function is obtained via assessment for regional myocardial wall motion abnormalities ***(Chandarana, et al., 2010).***

Although SPECT tracers to image myocardial blood flow are commonly referred to as perfusion tracers, they require viable myocyte cell membranes for uptake and retention. Thus, the uptake and retention of these tracers do reflect regional flow differences, but myocyte cell membrane integrity is also a prerequisite ***(Dilsizian, et al., 2009).***

Aim of the Work

The aim of the study is to estimate the value of echocardiography versus myocardial nuclear imaging, in differentiating ischemic cardiomyopathy (ICM) from dilated cardiomyopathy (DCM), to define patients who would benefit from coronary intervention.

DILATED CARDIOMYOPATHY

Introduction

Dilated cardiomyopathy is characterized by dilation and impaired contraction of one or both ventricle. Affected patients have impaired systolic function and may or may not develop overt heart failure. The presenting manifestations can include atrial and/or ventricular arrhythmias, and sudden death can occur at any stage of the disease ***(Elliott, 2000).***

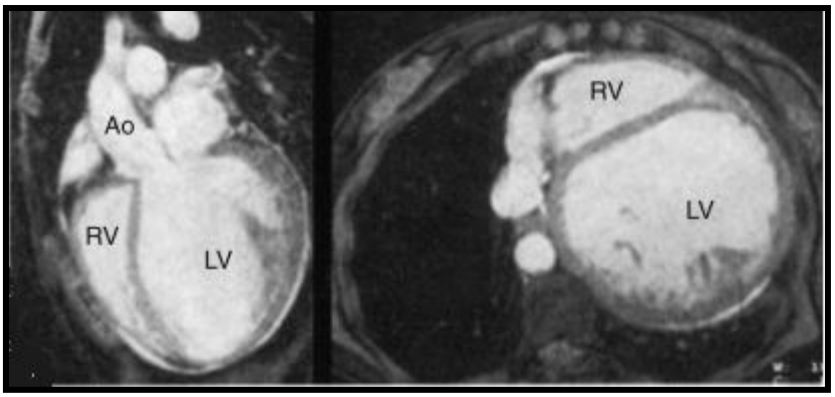

Fig. (1): Cardiac Magnetic resonance imaging of dilated cardiomyopathy, Hare JM: Etiologic basis of congestive heart failure. ***(In Colucci WS [ed]: Philadelphia, Springer, Current Medicine Group, 2008, pp 29-56)***

Definition

In 2008, the European Society of Cardiology defined cardiomyopathy as: "A myocardial disorder in which the heart muscle is structurally and functionally abnormal in the absence of coronary artery disease, hypertension,

valvular disease and congenital heart disease sufficient to explain the observed myocardial abnormality" ***(Elliott, et al, 2008).***

A diagnosis of dilated cardiomyopathy requires evidence of dilatation and impaired contraction of the left ventricle or both ventricles (e.g., left ventricular ejection fraction <40 percent or fractional shortening less than 25 percent) ***(Luk, et al, 2009).***

Clinical Presentation

Most patients present between the ages of 20 and 60, but dilated cardiomyopathy can occur in children and older adults. Affected patients can present in a number of different ways. Symptoms of heart failure-progressive dyspnea with exertion, impaired exercise capacity, orthopnea, paroxysmal nocturnal dyspnea, and peripheral edema-are most common. Other presentations include the incidental detection of asymptomatic cardiomegaly and symptoms related to coexisting arrhythmia, conduction disturbance, thromboembolic complications, or sudden death ***(Dec, 1994).***

Etiology

Dilated cardiomyopathy can be caused by a variety of disorders. In many cases, however, no etiology can be

found and the cardiomyopathy is deemed idiopathic. Where the relative frequency of the different causes in patients with initially unexplained cardiomyopathy was assessed in a review of 1230 patients in a study carried out by Johns Hopkins University in 2000 revealed:

- Idiopathic — 50 percent
- Myocarditis — 9 percent
- Ischemic heart disease — 7 percent
- Infiltrative disease — 5 percent
- Peripartum cardiomyopathy — 4 percent
- Hypertension — 4 percent
- HIV infection — 4 percent
- Connective tissue disease — 3 percent
- Substance abuse — 3 percent
- Doxorubicin — 1 percent
- Other — 10 percent

(Felker, et al., 2000)

A. Infectious Causes of Cardiomyopathy

A variety of infectious organisms can lead to myocarditis and dilated cardiomyopathy.

Viral Cardiomyopathy

Viral infection is the most common cause of myocarditis and has been implicated in the development of dilated cardiomyopathy. Viruses known to involve the myocardium include parvo virus B19, human herpes virus 6, coxsackie virus, influenza virus, adeno virus, echo virus, cytomegalo virus, and human immunodeficiency virus. The initial immune response limits the degree of viremia early during infection and protects against myocarditis. If, however, this response is insufficient, the virus may not be eliminated and myocyte injury may ensue ***(Bowles, et al., 2003).***

HIV Infection

Heart disease associated with HIV infection is being recognized with increasing frequency. The proposed mechanisms of cardiac damage include drug toxicity, secondary infection, myocardial damage by HIV itself, and an autoimmune process induced by HIV itself or in association with other cardiotropic viruses such as coxsackie virus, cytomegalo virus, or Epstein-Barr virus ***(Sani, 2008).***

Chagas Disease

A protozoan infection due to Trypanosoma cruzi, is the leading cause of dilated cardiomyopathy in Central and South America. It is characterized clinically by an acute myocarditis, cardiac enlargement, tachycardia, and nonspecific ECG abnormalities including right bundle branch block and premature ventricular contractions. Patients can develop left ventricular apical aneurysms that are pathognomonic for this disease ***(Sabino, et al., 2013).***

Lyme disease

Cardiac involvement with Lyme disease is usually manifested as a conduction abnormality. Cardiac muscle dysfunction can also occur; it is often self-limited and mild, leading to transient cardiomegaly or pericardial effusion on echocardiogram or chest x-ray. However, occasional patients develop symptomatic myocarditis and dilated cardiomyopathy ***(Fish, et al., 2008).***

B. Genetic causes of dilated cardiomyopathy

Among patients with idiopathic dilated cardiomyopathy, it is estimated that up to 50 percent have familial disease. No clinical or histologic criteria, other than family history and careful examination of relatives (including those who are asymptomatic), have been derived

to distinguish familial from non-familial disease. The mode of inheritance is usually autosomal dominant, although autosomal recessive, X-linked, and mitochondrial inheritance have also been described ***(Burkett, at al., 2005).***

Dilated cardiomyopathies in patients who do not have a known family history of disease may also have a genetic basis ***(Parks, et al., 2008).***

Inherited Syndromes

Dilated cardiomyopathy can be a common and important component of a number of inherited disorders, including a number of neuromuscular diseases, hereditary hemochromatosis, and the hereditary sideroblastic anemias and thalassemias ***(Modell, et al., 2008).***

Hypertrophic Cardiomyopathy

A small proportion of patients with HCM who survive the early risk of sudden death develop progressive myocardial wall thinning, a reduction in systolic performance, and an increase in left ventricular dimensions in the long term, resembling the morphologic and functional features of dilated cardiomyopathy ***(Biagini, et al., 2005).***